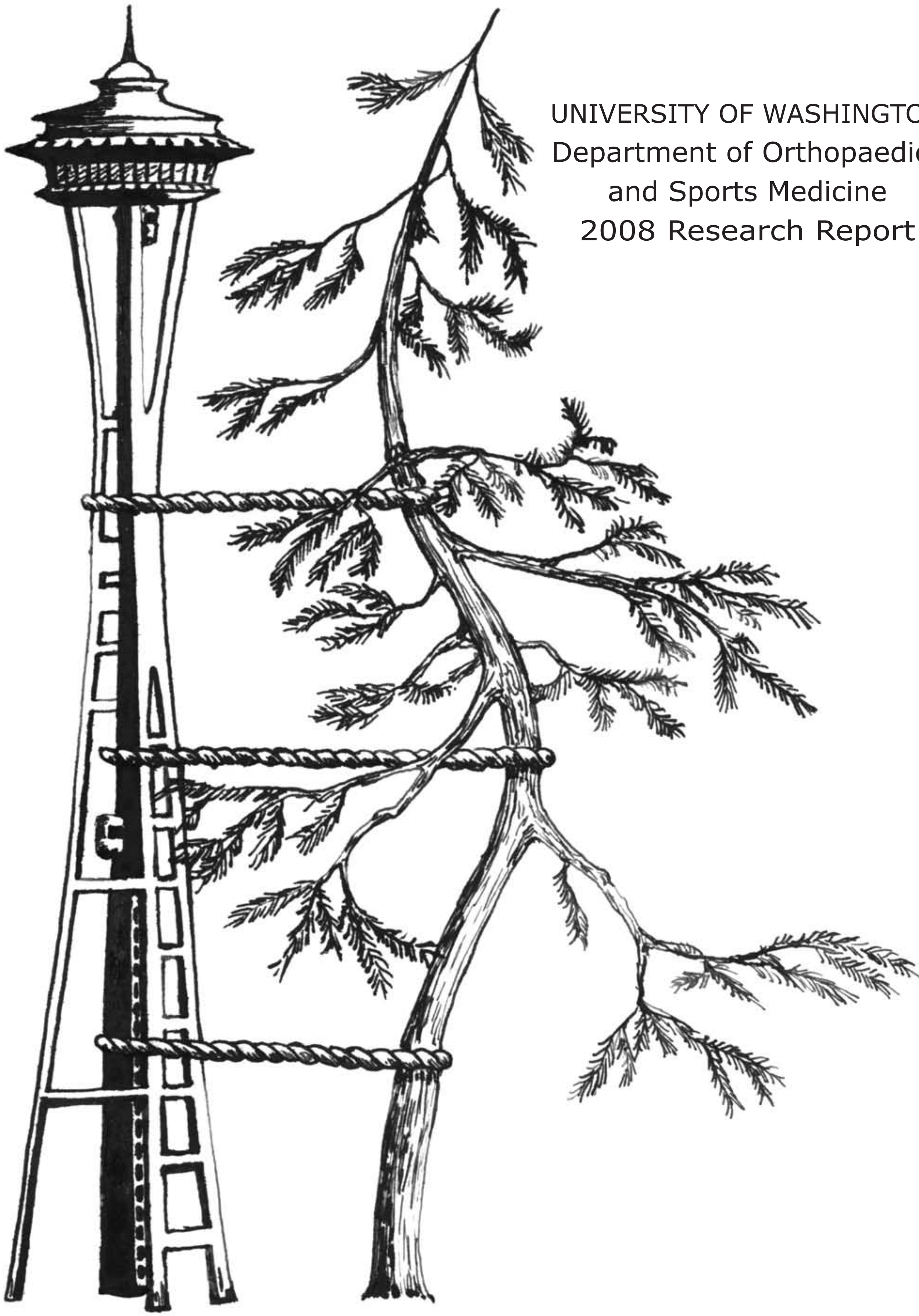
University of Washington

Department of Orthopaedics and Sports Medicine
2008 Research Report
Orthopaedic Discoveries Issue



UNIVERSITY OF WASHINGTON
Department of Orthopaedics
and Sports Medicine
2008 Research Report

UW Medicine
SCHOOL OF MEDICINE

Department of Orthopaedics and Sports Medicine
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Seattle, WA 98195

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Front Cover Illustration: "The Starry Night"
by Vincent Van Gogh, 1889.
The Museum of Modern Art, New York, NY, USA

Back Cover Illustration: V838 Mon: Echoes from the Edge. Credit: NASA and the Hubble Heritage Team (AURA/STScI). Variable star V838 Monocerotis. (See website apod.nasa.gov/apod/ap040305.html)

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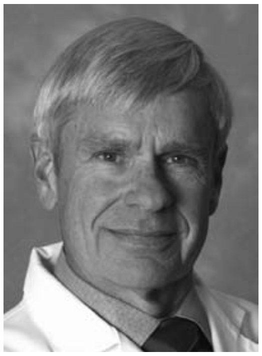

I am sure many of you will have no difficulty in identifying the art covering our 2008 Research Report. I thought it connected to our theme, "Discovery!" because of the bright swirling stars against a night sky. Everyone knows that discovery happens at unexpected times, whether it is Archimedes in his bathtub or Roentgen who accidentally put his hand in front of a cathode ray tube. Sometimes the discovery is so sudden that it causes us to run down the street clad in little but our enthusiasm, crying, "Eureka, I have found it!" For many of us these discoveries come in the night, against a dark sky, like the stars in Van Gogh's painting. Starry Night (painted in 1889 at Saint-Rémy) hangs in the Museum of Modern Art (MOMA) in New York City where it continues to inspire individuals like us and like Don McLean, who crafted the lyrics to Vincent: "Starry, starry night. Paint your palette blue and grey,...". Like a scientific discovery, Van Gogh brought together in this painting seemingly unrelated elements: in the center, the village of Saint-Rémy, to the right, the Alpilles, a notable geological feature of the Provence region of France, the cypress tree added to the left, and the constellation Ursa Major above. The swirl in the center even 'predicted' the image of the star, V838 Monocerotis, as photographed by the Hubble telescope in 2004 (see back cover). Starry Night is indeed about discovery.

In reflecting on the fact that it is often night that enables discovery, I would be remiss if I did not quote a bit of one of D.K. Clawson's favorite poems, the Ladder of St. Augustine by Henry Wadsworth Longfellow, one of Van Gogh's 19th century contemporaries

We have not wings, we cannot soar;
But we have feet to scale and climb
By slow degrees, by more and more,
The cloudy summits of our time.

The mighty pyramids of stone
That wedge-like cleave the desert airs,
When nearer seen, and better known,
Are but gigantic flights of stairs.

The distant mountains, that uprear
The solid bastions to the skies
Are crossed by pathways, that appear
As we to higher levels rise.

The heights, which great men reached and kept,
were not attained by sudden flight.
They, whilst their companions slept,
were toiling upwards, in the night

Ever since Dr. Clawson founded our Department in 1965, the faculty, residents and students of Orthopaedics and Sports Medicine have been all about discovery. These discoveries sometimes come unexpectedly, sometimes from toiling upwards in the night and sometimes they come from executing a deliberate research plan.

Our discoveries represent the full range of our core missions: innovations in basic science, clinical science, patient care, and education. We seek to make contributions in these domains because we are not content with today's orthopaedics, as good as it is. Today, one in four Americans has an orthopaedic problem needing medical attention; musculoskeletal conditions remain the leading cause of disability in our country (www.boneandjointburden.org). We know that the future holds better ways to understand, prevent, diagnose and treat the conditions that deprive so many millions of individuals each year of their ability to be active and to be able to participate comfortably in their work and play. If you would like to learn more about us and what we do, I invite you to visit us at www.orthop.washington.edu or to email me at matsen@u.washington.edu.

In conclusion I would like to thank all the private individuals and foundations that have enabled Orthopaedics and Sports Medicine to continue its commitment to ongoing discovery. May your days be bright and your nights starry.

Best wishes for good health,

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The Bob Behnke Endowed Chair for the Health of the Student Athlete

This issue of our research report concerns discovery in the Department of Orthopaedics and Sports Medicine and the relevance of our discoveries to the benefit of individuals across the United States and beyond. Our Department's dedication to the student athletes at the University of Washington enabled us to discover a previously unacknowledged challenge: the health of the student athlete is at risk. This risk arises not only from the possibility of injury from collisions in football or sudden changes of direction on the basketball court or falls in gymnastics, but also from the pressure on the athlete to try to optimize her or his performance. We discovered that this pressure comes from coaches, peers, parents, fans, and from inside the athletes themselves. The desire to excel can overwhelm good judgment. This pressure may tempt a woman cross country athlete to anorexia or the football athlete to take performance-enhancing medications. There is also pressure on those entrusted with the care of the student athletes to condone or to turn a blind eye to unhealthy means of improving their competitiveness.

Sally Behnke and her late husband Bob shared a deep love for the student athletes at the University of Washington.

Recognizing the need to ensure the well-being of athletes at the University of Washington and everywhere, Sally Behnke and her sons, Carl and John, have joined Bill and Lannie Hoglund, Gary and Alea Culpepper and Anne and Rick Matsen in endowing the Bob Behnke Chair for the Health of the Student Athlete. Bob (UW class of 1943), native of Yakima, former captain of the UW ski team, mayor of Medina and president of the UW Alumni Association is credited with persuading alumni to expand their interests to include not only athletics, but also academic and social concerns. Sally assures us that Bob would have been most pleased to have his dedication to UW students commemorated with this endowed chair so that its mission will be robustly supported in perpetuity.

We are delighted to announce that the first holder of the Bob Behnke Chair is John O'Kane, associate professor in the Department of Orthopaedics and Sports Medicine and Head Team Physician for the UW Department of Intercollegiate Athletics. John is a graduate of the UW residency in Family Medicine and was the first primary care sports medicine fellow at the UW. He is recognized by Seattle Magazine as a "Top Doctor" in sports medicine. For the past 12 years he has been known to all the UW varsity athletes as "Doc O'Kane". They know that he wants not only for them to be successful at the University of Washington, but also in their lives beyond college. He has organized gatherings of the Pac 10 team physicians to discuss the many issues they have in common, ranging from drug testing to anger management to nutrition. As a member of the staff of the UW Sports Medicine Clinic, John consults with athletes of all ages and degrees of excellence. The endowment will permanently assure that the UW always has someone like John whose primary concern is the health of the student athlete.

Congratulations, Doc O'Kane!

John O'Kane keeps a watchful eye on the student athletes at the University of Washington.

The Endowed Chair in Women's Sports Medicine and Lifetime Fitness

Helping Women Stay Fit For Life

Several years ago, Rick Matsen gathered together a group of friends of the Department of Orthopaedics and Sports Medicine to discuss the challenges of lifetime musculoskeletal health for active women. These challenges include the high rates of injury in young women athletes, the limitations that arthritis imposes on exercise in midlife, and the role of exercise in the treatment and prevention of osteoporosis in postmenopausal women. The compelling need for further research into the sex-specific aspects of bone and joint health led these generous friends of the Department to establish an endowed chair as the first step in building toward a permanent Center for Women's Sports Medicine and Lifetime Fitness. We are extremely pleased to introduce the first holder of this Chair, Peter R. Cavanagh.

Peter Cavanagh joins UWMedicine as the inaugural holder of the Endowed Chair in Women's Sports Medicine and Lifetime Fitness.

Peter comes to us from the Cleveland Clinic in Ohio, where he was the Virginia Lois Kennedy Chairman of Biomedical Engineering. Prior to that, he was the Distinguished Professor of Kinesiology, Orthopaedics and Rehabilitation, Medicine and Behavioral Health at Pennsylvania State University. Peter's past work in women's health across the lifespan has included a longitudinal study of the effects of exercise on bone health in middle-school girls, a definitive paper on sex differences in foot structure, studies of elite women distance runners, and experiments on the influence of visual factors and postural unsteadiness on falls in older women. He has done award-winning work on the foot complications of diabetes and musculoskeletal changes during long duration space flight. He is also an important figure in the development of the scientific study and design of athletic shoes.

Peter's move will open new avenues for research as he leads the Center for Women's Sports Medicine and Lifetime Fitness at UW Medicine. This Center will develop innovative approaches to injury prevention, treatment, research, and life-enhancement through sport and exercise for women throughout their lifespan. Peter has a busy first year ahead of him including setting up a new laboratory on campus, forming a research team, developing web-based material on women's health, and starting a lecture series that will bring nationally prominent figures to campus. He will give a keynote address to an NCAA conference entitled: Paying the Price: Is Excellence in Sport Compatible with Good Health? and he will also be the keynote speaker at the 2009 meeting of the National Osteoporosis Foundation.

The new Center will exert national and international leadership in research, education, and outreach in Women's Sports Medicine and Lifetime Fitness to complement the outstanding reputation of our clinical programs. You will soon encounter our "brand" on the Internet and in the news media as our reach grows and our research in this area is published. You will hear about our role in providing guidance on the bone health of women astronauts who may travel to Mars later in this century. You will see us using information technology to improve the lives of active women around the world. Welcome Peter!

New Faculty

Walter F. Krengel III, M.D.

Dr. Walter F. Krengel III joins our department at Children's Hospital as chief of spine.

Dr. Krengel was with Proliance Orthopedics and Sports Medicine in Bellevue from 2000 to 2008. He was at Northwest Spine Surgeons from 1994 to 2000.

Krengel grew up in Seattle, attending Lakeside School. He attended Stanford University for both his undergraduate degree and medical degree. He completed his internship, residency and fellowship at the University of Washington Medical Center.

Well recognized in the field of spine surgery, Krengel has published and presented widely. His current research project is an FDA phase 3 trial, Safety and Efficacy of Titanium Surgical Mesh and Moss-Miami Pedicle Instrumentation for Spinal Fusion.

A member of the American Academy of Orthopedic Surgeons, the North American Spine Society and the Scoliosis Research Society, he has also served as vice president of the Washington State Orthopedic Society since 2004.

James C. Krieg, M.D.

Dr. James Krieg is a native of New Jersey. He received his undergraduate degree in Biological Sciences at Northwestern University, and attended medical school at the University of Medicine and Dentistry of New Jersey-New Jersey Medical School. He completed surgical internships and residencies at University of Iowa Hospitals and Clinics.

Dr. Krieg went on to complete an Orthopaedic Trauma fellowship at Harborview Medical Center. He was Co-Editor of the Iowa Orthopaedic Journal. He has served for over ten years on the clinical faculty of Oregon Health and Science University as a Clinical Assistant Professor and was Orthopaedic Trauma Director of Legacy Emanuel Hospital in Portland, Oregon.

He joins our Orthopaedic faculty as an associate professor at University of Washington specializing in Trauma Surgery. His surgical interests include treatment of cervical degenerative disc disease, minimally invasive approaches to the spine, and cervical and lumbar disc replacement.

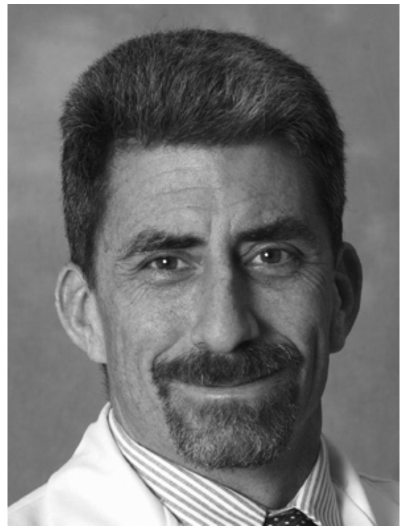
New Faculty

Michael J. Lee, M.D.

Dr. Michael Lee is a native of Ohio. He received his undergraduate degree in Biomedical Engineering at Northwestern University, and attended medical school there. He completed surgical internships and residencies at University Hospitals of Cleveland, and at Case Western Reserve University.

Dr. Lee went on to complete an Orthopaedic Research fellowship at Case Western, and an Orthopaedic Spine Surgery fellowship at Rush University, Chicago. He has received awards from the Ohio Orthopaedic Society, the Cleveland Orthopaedic Society, and the Cervical Spine Research Society. He was Editor-in-Chief of Case Orthopaedic Journal.

He joins our Orthopaedic faculty as an assistant professor at University of Washington specializing in Spine Surgery. His interests include treatment of cervical degenerative disc disease, minimally invasive approaches to the spine, and cervical and lumbar disc replacement.



Winston J. Warme, M.D.

Dr. Warme was educated at the University of Colorado at Colorado Springs and Uniformed Services University of the Health Sciences. His residency was completed at Madigan Army Medical Center. He completed fellowships in Sports Medicine at West Point and with the American Orthopaedic Society.

Beginning as a Special Forces Medic, he worked as an academic orthopaedist in the US Army, developing an interest in sports medicine with special focus on shoulder and elbow problems. His honors include the Association of Military Surgeons of the U.S. Award, and the Order of Military Medical Merit.

He joins the Orthopaedics & Sports Medicine faculty as an Associate Professor at University of Washington Medical Center. His philosophy of care is that successful treatment and rehabilitation requires a team approach. He has a particular interest in investigating different arthroscopic knots and the latest generation of high strength sutures. As a Shoulder and Elbow Team member, Dr. Warme will provide optimal care to patients while teaching orthopaedic surgeons-in-training and forwarding related research projects.

Eli Powell, M.D.
2008 Distinguished Alumnus
University of Washington School of Medicine

We are honored to salute Dr. Eli Powell as a distinguished alumnus of our residency. He was kind enough to speak both at our Resident Research Days and at the Chief Residents' Banquet.

Colonel Elisha T. Powell is the Commander, 3rd Medical Group, 3rd Wing, Elmendorf Air Force Base, Alaska, the Alaskan Command Surgeon, and the Senior Market Manager for the Anchorage Multi-service Market. The 3rd Medical Group serves the dynamically changing health needs of 81,000 Alaskan DoD and VA beneficiaries through TRICARE, DoD/VA Joint Venture, and the Alaska Federal Health Care Partnership. It does this by striving to deliver superior access, quality care, and service to its patients and other customers. The medical group is the primary Air Force referral center for the Pacific Theater.

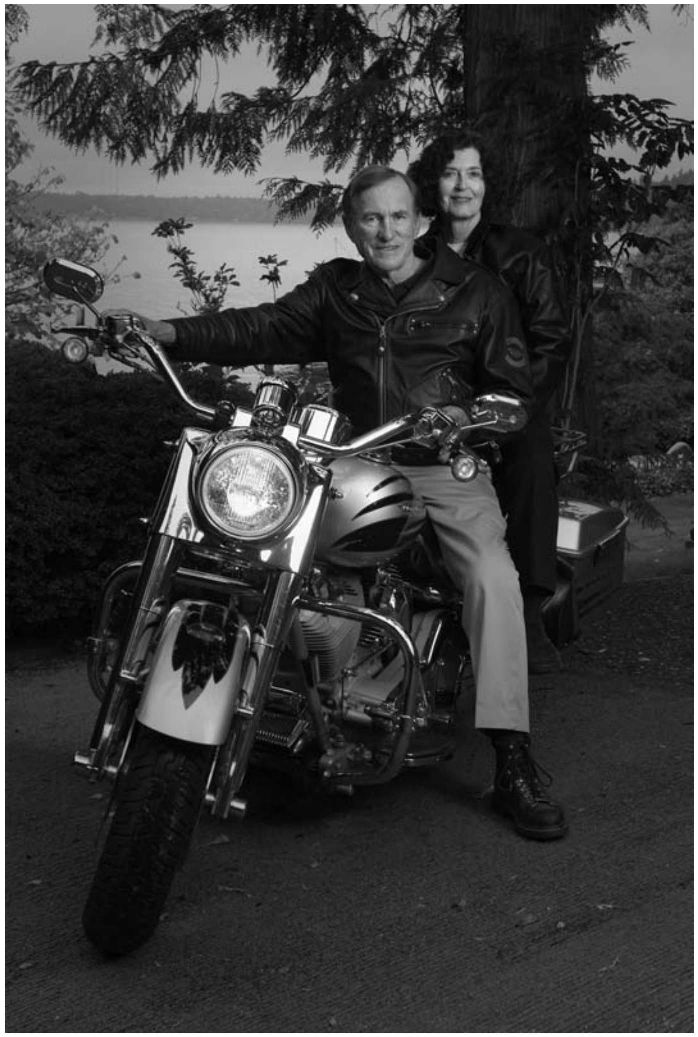
In June 1979, Colonel Powell entered military service as a basic cadet at the United States Air Force Academy. In 1983, Colonel Powell graduated from the Academy as a distinguished graduate, earning his Bachelor of Science degree and was selected as the outstanding cadet in biology. He entered medical school directly from the Academy and graduated with his Doctor of Medicine degree from the University of Florida in 1987. After a year of General Surgery Internship at Wright-Patterson Medical Center, he entered Orthopaedic Surgery Residency at the University of Washington.

He completed his Orthopaedic Surgery Residency in 1992 and was assigned to Yokota Air Base in Tokyo, Japan as an Orthopaedic Surgeon, followed by an assignment to Kirtland Air Force Base as Chief of Orthopaedics and Chief of Surgery. Colonel Powell served as the Orthopaedic Consultant to the Air Force Surgeon General from 1998 to 2003. He was Chief of Surgery at the Air Force Academy from 1998 to 2003 and served as head team physician for the Air Force Falcon Football, Hockey, and Basketball Teams during his assignment. Colonel Powell's previous command assignments include the Medical Operations Squadron and Chief of Hospital Services for the 363rd Expeditionary Medical Group, Prince Sultan Air Base, Saudi Arabia (2002), Medical Operations Squadron, United States Air Force Academy (2003-2004), 859th Surgical Operations Squadron Commander, Wilford Hall Medical Center, Lackland Air Force Base, Texas (2004-2006), and the 332nd Expeditionary Medical Group, Balad Air Base, Iraq (August 2005 to January 2006).

Colonel Powell is board certified in Orthopaedic Surgery by the American Board of Orthopaedic Surgery. He is past President of the 800-member Society of Military Orthopaedic Surgeons and a member of the American Academy of Orthopaedic Surgeons. He has over 100 flying hours in the F-4, F-15, F-16, KC-135, UH-60 Blackhawk and other aircraft.

We are indeed privileged to have Eli as one of our graduates and wish him well in his most promising career.

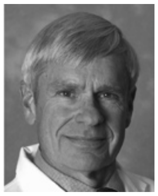
Marr P. Mullen, M.D.
2008 Grateful Alumnus
University of Washington School of Medicine



Dr. Marr Mullen and his wife Nancy. (Credit: photographer Karen Orders)

An obligation fulfilled. Washington native Marr P. Mullen ('55) went east for his undergraduate degree, then returned home to attend the UW School of Medicine. He trained in orthopaedics, eventually specializing in joint reconstruction and spine correction surgery. When Mullen retired in 1997, he didn't slow down. In addition to spending quality time with his Harley-Davidson motorcycle, he remains active in his profession through volunteer work on the Medical Quality Assurance Commission. Mullen is grateful for his education. "I owe my livelihood to the training I got from the UW Orthopaedics Department," he says. In turn, Mullen and his wife, Nancy, have made several gifts to the School of Medicine. One planned gift supports an endowed residency fund in orthopaedics; another provides unrestricted funds. The Mullens also support the Class of '55 scholarship fund. For the Mullens, giving is a pretty simple priority. "Everybody," Mullen says, "has some kind of obligation to give back to the community."

Department of Orthopaedics and Sports Medicine Faculty



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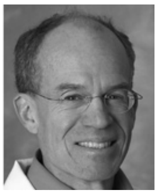

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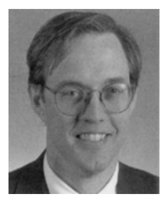

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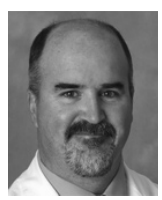

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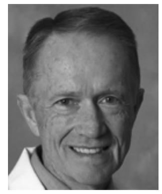

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Visiting Lecturers

Peter J. Stern, M.D.
2008 LeCocq Lecturer

This year at our annual LeCocq lecture on January 24th and 25th, we were honored to have Dr. Peter Stern as our 2008 LeCocq Lecturer. Peter J. Stern, M.D. graduated from Williams College and received his medical degree from Washington University School of Medicine. He completed his Orthopaedic residency at the Harvard Combined Orthopaedic Program followed by a hand surgery fellowship with Harold Kleinert.

In 1979, he was accepted as assistant professor and director of hand surgery at the University of Cincinnati College of Medicine. In 1991, Dr. Stern was promoted to Chairman of the Department of Orthopaedic Surgery. He is the Hill Professor and Chairman of the Department of Orthopaedic surgery at the University of Cincinnati.

Dr. Stern is a past President of the American Society for Surgery of the Hand and the American Board of Orthopaedic Surgery. He is the current president of the American Orthopaedic Association. He is a trustee of the Orthopaedic Research and Education Foundation, a Deputy Editor for the Journal of Bone and Joint Surgery and chairman of the Orthopaedic Residency Review Committee. He has educated 38 fellows and over 100 orthopaedic residents.

The diversity of Dr. Stern's interests is demonstrated by his recent JBJS publications: "Level-I Orthopaedic Trauma Care: A Model for Longevity", "Current Status of Orthopaedic Subspecialty Certification", "Proximal Row Carpectomy", and his magnificent AOA presidential speech "Hattage" (JBJS Dec 2007) that everyone should read.

Dr. Stern gave us great lectures on "Current Perspectives on the Management of Small Joint Injuries and Arthrosis", "My 10 Worst Wrist Procedures", and "Complications of Hinged Total Elbow Arthroplasty."

Michael A. Simon, M.D.
2008 OREF Hark Lecturer, Residents' Research Days

This spring we were honored to have Dr. Michael Simon as our OREF Hark Lecturer for Resident Research Days, June 26-27. He is Professor of Surgery and Associate Dean for Graduate Medical Education at the University of Chicago. Dr. Simon's clinical interest and expertise is in the field of orthopaedic oncology. Dr. Simon has published more than 100 publications on bone and soft tissue tumors. He is a past president of the Academic Orthopaedic Society, the American Board of Orthopaedic Surgery, the American Orthopaedic Association, the Orthopaedic Residency Review Committee of the Accreditation Council of Graduate Medical Education, and the Musculoskeletal Tumor Society. He is currently on the board of trustees for the Journal of Bone and Joint Surgery.

His college, medical school, and residency years were spent at the University of Michigan. Like our own Chappie Conrad, he took his research in Musculoskeletal Oncology and Pathology, at the University of Florida in Gainesville. Dr. Matsen had the pleasure of joining Dr. Simon for the American, British and Canadian traveling fellowship in Great Britain and South Africa in 1983.

His most recent articles in JBJS testify to his concern for the future of our field: "Future Physician Workforce Requirements: Implications for Orthopaedic Surgery Education", "Should There Be a Minimal Surgical Experience for a Graduating Orthopaedic Surgery Resident?", "The Clinician Educator", "Proposals to Improve Orthopaedic Surgery Graduate Medical Education".

Dr. Simon's Resident Research Days lectures are titled "How to improve orthopaedic surgery graduate medical education" and "Skeletal metastases of unknown origins."

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Health of the University of Washington Student-Athlete

- University of Washington Department of Orthopaedics and Sports Medicine is proud to provide team physician services to the UW Department of Intercollegiate Athletics.
- Athletic health care systems have evolved from a single team trainer to a full service medical clinic with teams of certified athletic trainers and physicians providing a range of medical services and administrative functions.
- UW Orthopaedics and Sports Medicine's ability to provide for the future health care of the Husky athletes has been insured by the recently established Bob Behnke Chair for the Student-Athlete.

The University of Washington boasts a long proud history of intercollegiate athletics dating back over a century. UW Football first took the field in 1889 and now 118 years later, the UW Department of Intercollegiate Athletics (UWICA) has grown to field 23 sports with 328 male and 346 female student-athletes. The University of Washington Department of Orthopaedics and Sports Medicine initiated its relationship with UWICA in the early 1970's when then President Charles Odegaard appointed as team physician Orthopaedic Chairman Dr. D. Kay Clawson. Dr. Clawson and Dr. Joe Kearney, the Director of Intercollegiate Athletics, established the UW Sports Medicine Clinic (UWSMC) in 1975. UWSMC was located in Hec Edmundson Pavilion, recognizing the essential link between the team physicians and the student-athletes. In the early '80s the care of the student-athletes moved into a private practice, and UW Orthopaedics re-assumed that role in 1996 for the Olympic sports programs, and re-assumed care for all of the student-athletes in 1999. Today there are 7 team physicians, 4 from UW Orthopaedics including the Head Team Physician and 3 from Hall Health Student Health Center.

Athletic health care and the field of sports medicine are fairly recent arrivals on the medical landscape. Sports Medicine is not a traditionally recognized medical specialty but an area of special interest, initially confined mostly to Orthopaedic surgery. Primary Care Sports Medicine has grown significantly in the past 15 years and university athletic health care programs now include both orthopaedic surgeons and primary care physicians with fellowship training and often a Certificate of Added Qualification (CAQ) in sports medicine. The thread that binds team physicians is a desire to work with active athletic people supporting their athletic aspirations within the larger context of health promotion. Those working as team physicians in university athletic health care systems are responsible primarily for insuring the health care of the student-athletes, assisting them in achieving their athletic objectives, and sometimes protecting their health from their competitive aspirations.

Athletic health care started with the "trainer". Athletic training dates back to ancient Greece while in the US, football teams dating back to the late 1800's would often identify a trainer who was the jack of all trades for the team. Part of the trainer's role was to assist injured players and gradually the trainer assumed increasing responsibility for the health care of the athletes on the team. In 1950 the National Athletic Trainers Association (NATA) was formed and in the years that followed, the Certified Athletic Trainer (ATC) with specific training requirements and job delineation was born. According to the NATA website, "Certified athletic trainers are health care professionals who specialize in preventing, recognizing, managing and rehabilitating injuries that result from physical activity. As part of a complete health care team, the certified athletic trainer works under the direction of a licensed physician and in cooperation with other health care professionals, athletics administrators, coaches and

parents." The partnership between the ATC and team physician is critical and the essential component of an athletic health care system. The UW athletic health care system currently includes 11 ATCs.

The team physicians are responsible for overseeing the medical care that takes place in the training room. The ATCs work independently in the fields of rehabilitation and injury prevention, but they are not trained or licensed to practice medicine so all of the medical care they provide requires team physician oversight. In addition to their oversight responsibilities, the team physicians annually provide over 2000 student-athlete medical visits in the UW training room, UW Sports Medicine Clinic, and Hall Health Primary Care Center to address injuries and illness incurred by the student-athletes. They also provide game coverage for home events, and away game coverage for football and other sports for certain high profile events.

The head team physician also has an administrative role, partnering with the head athletic trainer to oversee the training room operations and functioning as a liaison between the ICA administration, UW Medicine administration, and the other team physicians. The UW training room has evolved into a modern medical clinic with exam and procedure rooms, a small pharmacy, and an electronic medical record. UW Medicine and UWICA have developed a memorandum of understanding covering reporting lines, physician services and scope of practice in addition to an extensive policies and procedures manual covering issues ranging from operation of training room clinics, medical disqualification, hardship petitions, and drug testing to name a few.

One might ask why an athletic department requires such an elaborate health care system. Why are doctors needed on the playing field or in the training room? Athletic health care systems have evolved to provide on site medical services for a number of reasons. Pre-season medical screening is one critical function of the athletic health care team. A few years ago we identified a soccer player with critical narrowing of a heart valve. He had been cleared to play at a different school with a less rigorous screening

system. He had an operation, missed a year, came back, and in the last game of his senior year he scored the game winning goal. In his case a good screening system may have been literally the difference between athletic success and athletic sudden death.

Once someone is cleared to play, many of the sports in which the student-athletes participate are inherently risky. In addition, the risks may be amplified by pressures to play imposed by very competitive coaches, fans, and the student-athletes' own competitiveness. Fortunately sports are generally not life threatening, but occasionally potentially catastrophic events such as a gymnast falling and injuring her cervical spine or a football player collapsing from heat exhaustion do occur. On site medical staff can be the difference between life and death in these situations. Less serious but more frequent occurrences are injuries and a variety of medical conditions, situations where rapid diagnosis and treatment can significantly improve the outcome. Concussions are frequent occurrences in collision and contact sports. Unlike a broken leg where someone clearly can't play, concussions can be difficult to diagnose. There is increasing evidence that multiple concussions can result in long term brain damage and returning to play too soon after a concussion can result in "second impact syndrome" with lethal brain swelling. Immediately available physicians and ATCs are often all that stand between an eager but concussed athlete and the potentially life threatening decision to get back in the game. In the past decade, team physicians are also dealing much more frequently with psychological issues, a byproduct of the significant stressors associated with trying to excel as a big-time athlete and big-time student simultaneously.

A second reason University student-athletes require a high level of health care service is the media attention they receive both locally and nationally. Many Universities receive more exposure for their athletic endeavors than any other university program. Rarely does physics take place before a prime time national TV audience. In addition, coaches and athletic administrators whose livelihoods depend on winning games can easily be accused (fairly or not) of placing the success of a team above the health of a student-athlete. Team physicians charged with the task of insuring the health of the student-athletes and given the final authority to make play / no play decisions can insure that the competitive pressure of the moment does not result in a bad health care decision with consequences that can last a lifetime. Sadly, many athletic departments have experienced the negative publicity associated with providing sub-standard medical care to their student-athletes, or find themselves accused of placing the good of the program over the good of the participants. UW Medicine and UWICA, through the memorandum of understanding, partner to prioritize student-athlete health care above other concerns, free of the conflict of interest that can arise out of an employee/employer relationship.

UW Orthopaedics and Sports Medicine is extremely proud of its history with UW Intercollegiate athletics. UW Sports Medicine was founded with the goal of providing exceptional care for the Huskies, a charge highly valued, respected and enjoyed by the UW team physicians. The goal of UW Orthopaedics is to provide the highest level of care for the Husky Athletes forever. A major step toward reaching that goal was achieved this year through the endowment of a Chair to support the work of the UW Medical Coordinator, a position funded for the past 11 years through UW Orthopaedics. The Bob Behnke Chair was created through the generous support of Sally Behnke and her family in memory of her husband. The Chair has also received gifts from Bill and Lannie Hoglund, Gary and Alea Culpepper, and Rick and Anne Matsen. With some additional support, the Bob Behnke Chair just reached the level of an Endowed Chair, a first of its kind monumental statement elevating the heath care of the UW student-athlete to its appropriate place as a "highest" priority. UWICA's motto is, "Creating Winners, in the classroom, on the field, and in life". Great medical care is part of that mission and UW Orthopaedics and Sports Medicine is proud to be a permanent partner with UWICA to create winners in life.

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The Female Athlete

- Female athletes have more stress fractures than their male counterparts. Stress fractures are more common in women with abnormal menstrual cycles.
- Anterior cruciate ligament (ACL) tears are 4-6 times as common in female than in male athletes. Non-contact ACL tears in women are largely due to problems in their patterns of muscle use.
- The faculty of the Department of Orthopaedics and Sports Medicine at the University of Washington are dedicated to making physical activity safe and enjoyable for women of all ages and abilities.

Athletics, sports and physical activity provide substantial health benefits to women. Women who engage in physical activity have a more positive attitude, are more likely to be successful in school and business, and have lower risks of obesity, smoking, diabetes, depression, hypertension, osteoporosis and cancer. Athletic injuries may prevent women from enjoying the benefits of physical activity. While athletic women suffer from injuries similar to their male counterparts, some problems are more prevalent in the female athlete. These include stress fractures and tears of the anterior cruciate ligament.

With the passage of Title IX in 1972 came an increased participation of young women in collegiate sports. At that time the childhood of many women did not prepare them for intense sports participation at the collegiate level. While boys played football, baseball, and basketball starting at a young age, girls were less likely to have developed the strength and coordination to prepare them for vigorous practice and competition. As a result, female athletes often arrived at college less well prepared for the intensity of collegiate sports and more subject to overuse injuries than their male classmates. This was particularly well documented when the military academies were first opened to women. In the 1970s Dr. Kenneth Cooper began encouraging running as a means toward fitness for the general population and the aerobic dance craze soon followed. Women who had not been especially active as children or young adults and who had not had opportunities to be athletes in college began participating in intense and frequent athletic activities and many suffered consequences. They did not understand the principles and practice of athletic training, nor were their bodies conditioned for these types of physical stresses. Stress fractures became a common injury. A new syndrome called the Female Athlete Triad was defined as the combination of disordered eating (calorie intake insufficient for calorie expenditure), loss of menstrual periods (amenorrhea), and osteoporosis at a young age with resulting stress fractures. Young women who exercised intensely, burning more calories than they took in, were likely to delay starting their menstrual cycle or to stop having periods if they had previously started menstruating.

Injuries to dancers

Faculty from the U.W. Department of Orthopaedics and Sports Medicine investigated the relationship of activity, menses, and stress fractures in ballet dancers, who, because of their thin aesthetic, often developed amenorrhea. Drs. Nancy Kadel and Carol Teitz found that the length of time a dancer had gone without periods was also a significant factor in developing a stress fracture. Ballet dancers who had had no menstrual periods for 6 months or more were significantly more likely to develop stress fractures than dancers who had more regular menstrual cycles. In addition, 50 % of dancers who danced more than 5 hours per day developed stress fractures compared with 31% of those dancing less than 5 hours per day. All dancers with both risk factors had one or more stress fractures.

Drs. Kadel and Teitz, both with

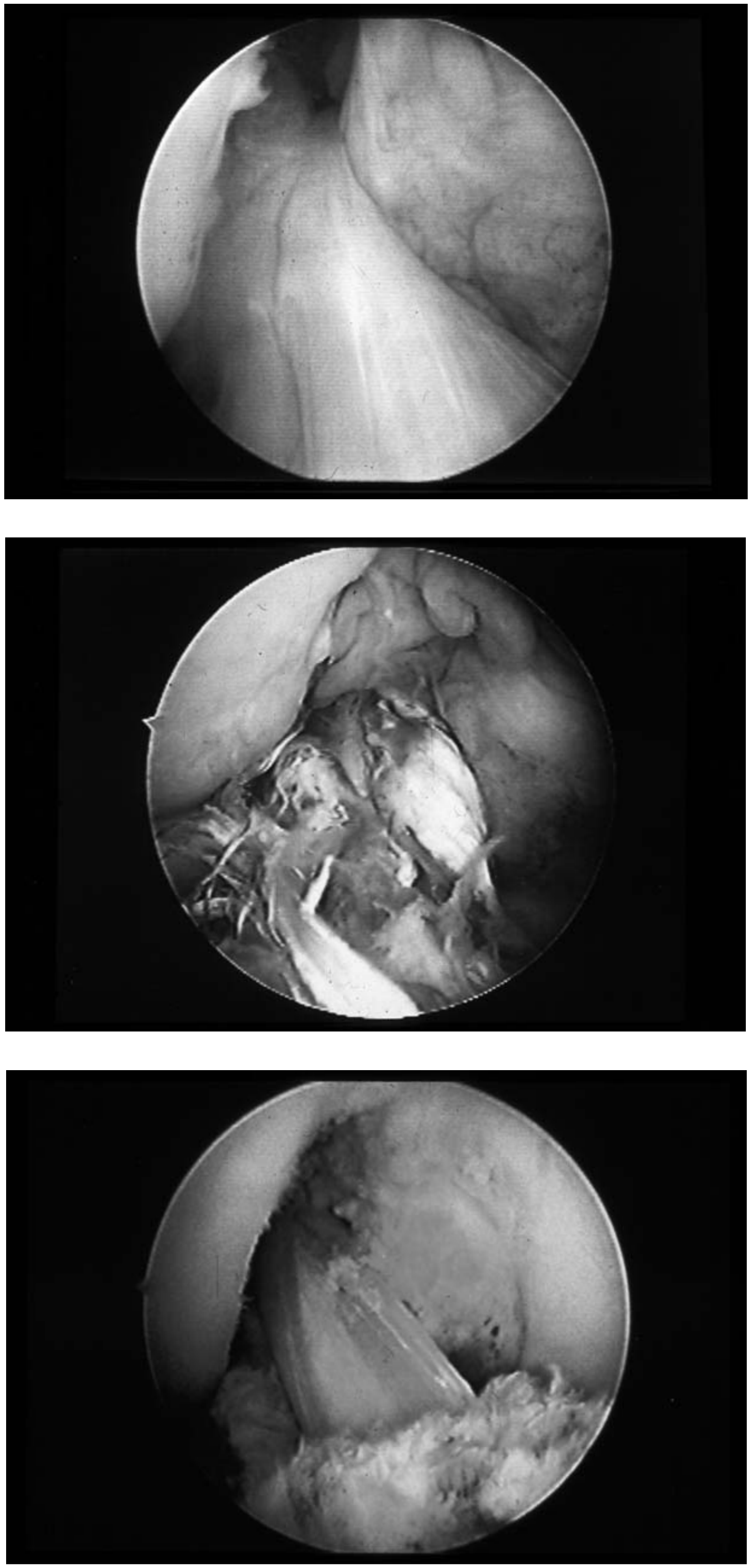

Figure 1: Anterior cruciate ligament pictures. Normal ACL (top), torn ACL (middle), and repaired ACL (bottom).

strong dance backgrounds, also studied some of the biomechanics of ballet to improve or prevent some of the foot and ankle problems found in ballerinas who dance "en pointe" on toe shoes. A particular injury sustained by these dancers is a stress fracture of the base of the second metatarsal. To understand this stress fracture better, Dr. Kadel's team measured in cadaver specimens the contributions of the ligaments and the pointe shoe to the stability of the joints in the midfoot. Cutting the ligaments attaching the base of the second metatarsal to its adjacent bones resulted in a small but significant change in relative alignment of these bones when loaded en pointe. Removal of the pointe shoe after the ligaments had been sectioned led to complete destabilization of the midfoot. These data supported the hypothesis that the pointe shoe is a major stabilizer of the midfoot and that the recessed position of the base of the second metatarsal along with the ligamentous and muscular attachments create a fulcrum at the site where dancers sustain second metatarsal stress fractures. The implication is that ballerinas need to gradually build up the strength of this part of the foot to avoid the stress fractures that may result from rapid increases in the frequency and duration of loads on it.

Drs. Teitz and Richard Harrington, investigated the pressures on the toes in pointe shoes and found that the pressures on the toes accounted for only 20 to 30% of the total pressure measured at the toe box (in the en pointe position); the rest of the load was distributed to the shoe by the rest of the foot. They also found that the forces on individual toes could be distributed more evenly depending on the type of pointe shoe, the stiffness of the shank of the shoe , the length of the shank, and by equalizing the lengths of the toes using caps. This knowledge was used to counsel young dancers in proper shoe purchase and to avoid not only toe injuries but also injuries to the rest of the body caused by imbalance resulting from uneven toe length and shifting weight from one side of the foot to another.

Anterior cruciate injuries

While dancers rarely tear the anterior cruciate ligament of the knee (ACL), female athletes in sports such as soccer, basketball, and team handball, tear their ACLs at 4-6 times the rate of their male counterparts. Factors that have been studied in an attempt to understand these differences include hormonal, anatomical, and muscular activation factors. A multi-center video analysis of 54 athletes tearing their ACLs was led by Dr. Teitz in 1998. We were particularly interested in the common features of ACL injuries occurring without contact from another player, "non-contact" ACL injuries. The most common event causing the non contact ACL tear was either suddenly stopping a run or awkwardly landing a jump. The center of gravity was found to be behind the knee in 2/3 of the injuries. Ground contact on the entire foot (rather than on the toes) was also noted in 2/3 of the female athletes and all of the male athletes. Finally the common knee position at the time of injury was bent less than 30 degrees, and in apparent valgus possibly secondary to internal rotation of the hip. These findings served as background for other investigators to confirm the importance of neuromuscular factors in the production of non-contact ACL injuries. Subsequently injury prevention programs have been designed to strengthen the hip abductors and external rotators to control the position of the knee as well as to train athletes to stay on their toes, to land on a bent knee and to stop in such a way that the center of gravity stays forward over the knee.

Future

Much of the research concerning ACL tears has been aimed at college age and older athletes, individuals in whom much of the strength and neuromuscular coordination has already been established. To better understand how injuries can be prevented, we are extending our investigations to explore the relationship between injuries and physical activity in younger girls. Dr. John O'Kane and Dr. Melissa Schiff have just completed a one year pilot study assessing injury incidence and risk factors among middle school girls playing select soccer in Seattle. The pilot study was designed mainly to confirm the methodology, and the NIH has funded the study for an additional 5 years. In the pilot study, acute knee injuries were infrequent and acute ankle injuries were the most common. For the knee and hip, overuse injuries were more common and overlapping sport seasons may be a risk factor. The 5-year study will better illustrate what types of injuries are most common in this age group and determine which baseline physical attributes are associated with a higher risk of injury. The study is also exploring psychological factors that may contribute to injury by evaluating how soccer injuries affect other aspects of young athletes' lives and the burden on their families.

Drs. Schiff, O'Kane, and Dr. Alan Tencer also plan to apply novel technology (originally designed to study car crash injuries-see related article in this volume) to better understand the biomechanical joint forces that occur with common soccer movements. One recent study suggests that the risk of knee ligament injury increases for women only after puberty. By studying girls' biomechanics before and after puberty, we hope to understand what occurs through maturation that increases the risk of injury. We believe this will lead us to an effective evidence-based injury prevention program.

There is nothing more elating than watching a young woman reach her full athletic potential on the field of play, and not many things more devastating than seeing it end with an injury. UW Department of Orthopaedics will continue to lead efforts to make sports and physical activity safe for women of all ages and abilities.

References

Griffin LY, Agel J, Albohm MJ, Arendt EA, Dick RW, Garrett WE, Garrick JG, Hewett TE, Huston L, Ireland ML, Johnson RJ, Kibler, WB, Lephart S, Lewis JL, Lindenfeld TN, Mandelbaum BR, Marchak P, Teitz CC, and Wojtys EM: Non-contact Anterior Cruciate Ligament Injuries: Risk Factors and Prevention Strategies. J Am Acad Orthop Surg 8(3): 141-150, 2000.

Griffin LY, Albohm MJ, Arendt EA, Bahr R, Beynnon BD, DeMaio M, Dick RW, Engebretson L, Garrett WE, Garrick JG, Gilchrist J, Hannafin JA,Hewett TE, Huston L, Ireland, ML, Johnson RJ, Lephart S, Mandelbaum BR, Mann B, Marks PH, Marshall SW, Mykelbust G, Noyes FR, Powers C, Shields C, Shultz S, Silvers H, Slauterbeck J, Taylor D, Teitz CC, Wojtys EM, and Yu, B: Update on ACL Prevention: Theoretical and Practical Guidelines. Review of Hunt Valley Meeting, February 2005. Am J Sports Med, 34 (9): 1512-1532, 2006.

Kadel N, Boenisch M, Teitz C, Trepman E.: Stability of Lisfranc joints in ballet pointe position. Foot Ankle Int. 26(5):394-400, 2005.

Kadel, N.J., Teitz, C.C., and Kronmal, R.: Stress Fractures in Ballet Dancers. Am J Sports Med. 20:445-449, 1992.

Teitz CC, ed: The Female Athlete, AAOS, Rosemont Ill, 1997.

Teitz CC, Hu SS, Arendt EA: The female athlete: Evaluation and treatment of sports-related problems. J Am Acad Orthop Surg 1997;5(2): 87-96.

Teitz, C.C., Harrington, R.M. and Wiley, H.: Pressures on the Foot in Pointe Shoes. Foot and Ankle. 5(5):216-221, 1985.

Teitz, C.C.: Diagnosing and Managing Common Dance Injuries. J Musculoskel Med. 13:46-57, 1996.

Teitz, C.C.: Non-contact ACL injuries in Dance and Skating. In Understanding and Preventing Non-Contact ACL Injuries. Human Kinetics, 2006.

Teitz, C.C.: Video Analysis of ACL Injuries. In Prevention of Noncontact ACL Injuries. American Academy of Orthopaedic Surgeons, 2001.

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Ewing's Sarcoma: A Molecular Model of Cancer Biology and Treatment

- Sarcomas are tumors that arise in the musculoskeletal system.
- Ewing's sarcoma is the second most common variety of bone cancer in children and young adults.
- Ewing's sarcoma is classified as a malignancy (cancer) due to its ability to invade and destroy local tissues and its propensity to spread (metastasize) to remote tissues, usually the lungs.
- Despite advances in chemotherapy, radiation therapy and limb-salvage surgery, only about 50% of children with Ewing's sarcoma survive beyond 5 years after diagnosis.
- Ewing's sarcoma is caused by a group of related genetic defects (mutations). The most common mutation involves an abnormal exchange (translocation) of DNA between chromosomes 11 and 22. This mutation results in production of an abnormal protein referred to as EWS/FLI1. EWS/FLI1 is referred to as a fusion protein since it consists of portions of two different proteins (EWS and FLI1)
- We have discovered that EWS/FLI1 in and of it self appears to be sufficient to cause Ewing's sarcoma and it related tumors.
- Similar fusion genes and proteins cause other cancers such as leukemia and sarcomas of cartilage and fat. Ewing's sarcoma serves as an ideal model to study all of these tumors.
- Therapies that specifically target these fusion proteins show promise as potential treatments for a variety of cancers. We are now testing one of these therapies, developed in our laboratory, in mice that have been implanted Ewing's sarcoma.

Orthopaedic research has slowly evolved from a purely biomechanical endeavor to a field heavily influenced by modern molecular technology. There have been great advances in orthopaedic surgical techniques and implants over the past 40 years. However, many if not most scientists and clinicians believe that the important orthopaedic advances over the next 40 years will be in the realm of molecular biology and nanotechnology. Progress is being made in areas such as tissue engineering and the detailed understanding of degenerative diseases. In this article, I will review the advances made in understanding the molecular basis of Ewing's sarcoma and our early attempts to model and treat this cancer in mice. The work in our laboratory and others has generated national and international interest and many feel that a new era of targeted molecular therapy for Ewing's sarcoma and other cancers is upon us. I will focus on the work done within the Department of Orthopaedics and Sports Medicine.

Historical Background

Sarcoma, derived from the Greek word "σαρκωμα", is a term that denotes fleshy cancers that arise within the connective tissues such as bone and cartilage. There are many subtypes of sarcoma. The one we now refer to as Ewing's sarcoma was first scientifically described in 1920 by Dr. James Ewing, the foremost pathologist in the country at that time, at what was then known as the Medical College of Cornell University. Little further progress was made in understanding the biology of Ewing's sarcoma until 1992 when Olivier Delattre from the Institut Curie in Paris, and a group of international collaborators, first described the chromosomal abnormality that is the sine qua non of a group of cancers now referred to as Ewing's family tumors. The particular chromosomal defect

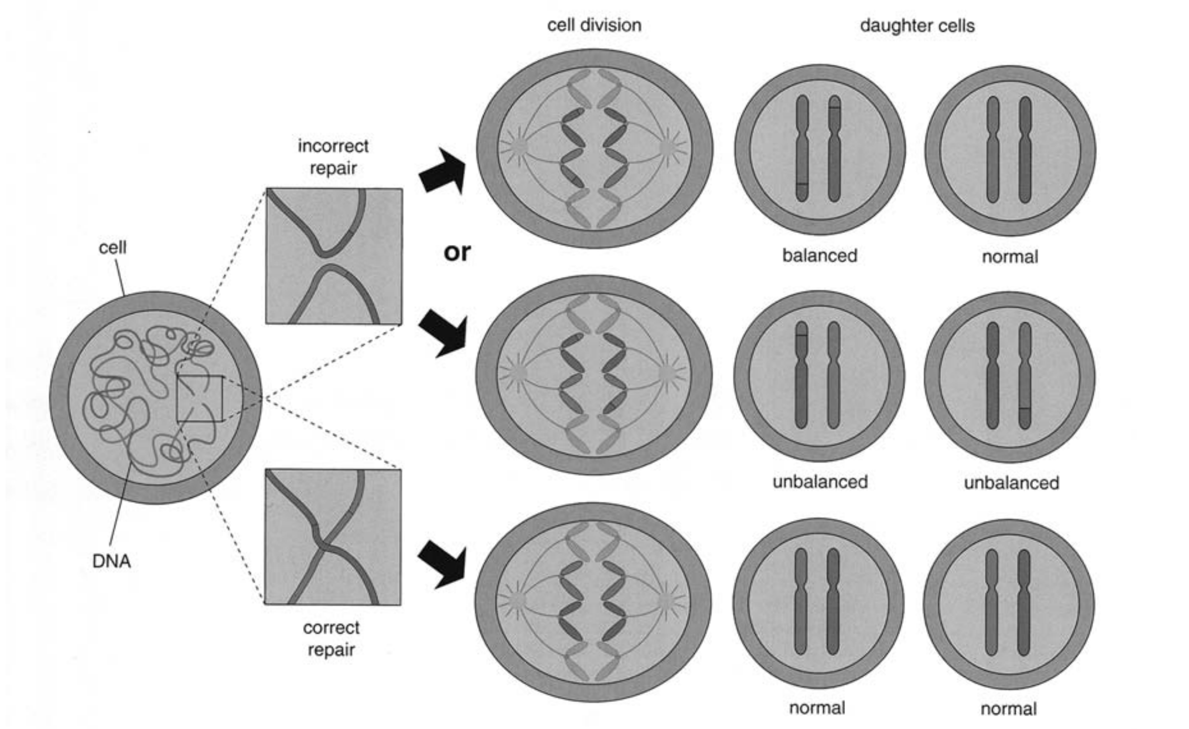

Figure 1: (From American Scientist, issue 731, p. 417, Figure 4): During cell division each chromosome must be duplicated so that each daughter cell receives a complete set of chromosomes. Occasionally DNA strands break (square boxes) during this process. When the damaged DNA is not repaired (top box), what are referred to as balanced or unbalanced translocations develop. In Ewing's sarcoma the translocation arises during development and there is no known inherited predisposition to developing Ewing's tumors.

discovered by Delattre involves a fusion of chromosomes 11 and 22.

Scientific Background

In over 85% of Ewing's sarcomas a specific chromosomal translocation t(11;22), results in an abnormal protein by combining a portion of the protein EWS with a portion of the transcription factor (transcription factors regulate the decoding of DNA) FLI1. These translocations occur when cells that are multiplying duplicate their 23 pairs of chromosomes in preparation for cell division. When a strand of DNA breaks during this process of duplication, occasionally two different chromosomes will fuse resulting in a translocation (Figure 1). An EWS/FLI translocation in just a single cell can result in a Ewing's sarcoma.

Most cancers involve multiple if not many mutations, this makes understanding them and developing molecular treatments ("magic bullets") against them a daunting challenge. In contrast, beause of their single mutation, many believe that Ewing's Family Tumors may be "ideal" models to study cancer biology and molecular therapy. Thus after its discovery about 15 years ago, the race was on to understand the mechansm by which a single protein, EWS/FLI1, could bring about such a deadly disease as Ewing's Sarcoma. The goal of understanding this mechanism is driven by the pure pursuit of knowledge but also by the belief that the molecular pathways that allow EWS/FLI1 to cause cancer are ideal targets for a focused molecular therapy, or "magic bullet".

Our laboratory has made important contributions to understanding the biology of Ewing's sarcoma. We have discovered pathways that mediate the cancer-causing properties of EW/FLI1 through effects on cellular senescence (the normal process by which older cells die before they can cause cancer and other diseases) and processing of RNA (the intermediary molecule that translates DNA into protein). As is typical for cancer, the initial concept of a simple mechanism has evolved into a more complicated picture. On the other hand, the discovery of multiple pathways that appear to be driven by this single mutation can also provide multiple targets to intefere with the cancerous effects of EWS/LI1.

Our ultimate goal has been to apply our discoveries about the basic biology of Ewing's sarcoma to the development of effective treatments. This is now the focus of our laboratory and below I will briefly review the current treatment of Ewing's sarcoma and then discuss some preliminary progress that we have made in biologically targeting (treating) Ewing's sarcoma in cell culture and in mice.

Treating Ewing's Sarcoma - Past, Present and Future

In the not too distant past, Ewing's sarcoma was treated by amputation of the involved extremity. Despite this radical surgery, the most common outcome after amputation was still death due to the propensity of Ewing's sarcoma to metastasize (spread) to the lungs. When Ewing's sarcoma would arise in locations such as the spine or pelvis, a slow and painful death would ensue as surgical techniques did not exist to completely remove tumors from these locations. This grim prognosis began change in the 1970's with the realization that chemotherapy administered before and after surgery would lower the likelihood of metastasis. In addition, the chemotherapy would shrink the tumor and thus make it more feasible to surgically excise the cancer without necessarily amputating the limb. In addition to chemotherapy, radiation therapy is also a mainstay of Ewing's treatment. Both chemotherapy and radiation therapy are blunt, powerful treatments that can be very debilitating as their effects on cells are non-specific - that is they damage and kill normal cells as well as cancer cells. As powerful as they are, these current treatments are not uniformly effective and many patients either do not survive or sustain serious treatment-related complications (Figure 2).

As eluded to above, EWS/FLI1 is an abnormal protein that is both responsible for the development of Ewing's sarcoma and never found in non-cancerous cells. In addition, EWS/FLI1 is similar to other fusion proteins that also cause cancers such as luekemia and a variety of other sarcomas. These facts make the inhibition or destruction of EWS/FLI1 a very tempting goal of targeted molecular therapies. Theoretically it should be possible to develop treatments that only effect the cancerous cells containing the EWS/FLI1 mutation. Future treatments of Ewing's sarcoma are likely to exploit this specificity and this has now become the main research interest of our laboratory.

RNA Interference and Molecular Treatment of Ewing's Sarcoma: RNA interference (RNAi) is a recently described endogenous cellular process that utilizes double-stranded RNA, referred to as short-interfering RNA (siRNA), to degrade or silence specifically targeted messenger RNAs. While quite complicated on the molecular level, the end result is that scientists and physicians now have a potent tool to eliminate the synthesis of unwanted proteins. The excitement over this biological process and the techniques to harness it garnered much press and in 2006 led to a Nobel Prize in Medicine and Physiology for the discoverers of RNA interference. We recognized that since EWS/FLI1 is present in all Ewing's cells but no normal cells, it would be an ideal target for RNAi (Figure 3).

We began our experiments by applying RNAi to Ewing's cells grown in culture in an incubator. These initial experiments were somewhat crude in

Figure 2: Top: This is an MRI of the pelvis of a young man who was diagnosed 5 years ago with Ewing's sarcoma in the left side of his pelvis (white star). Middle: After several cycles of chemotherapy to shrink his tumor, a portion of his pelvis was removed and replaced with cadaveric donor bone attached to his remaining pelvis with plates and screws. Bottom: Unfortunately, in a scenario not atypical for patients with Ewing's sarcoma, his tumor recurred in his right femur four years after his original surgery. This tumor was also removed and the hip and femur were reconstructed with a large oncologic prosthesis. He now has metastatic Ewing's sarcoma in his lungs.

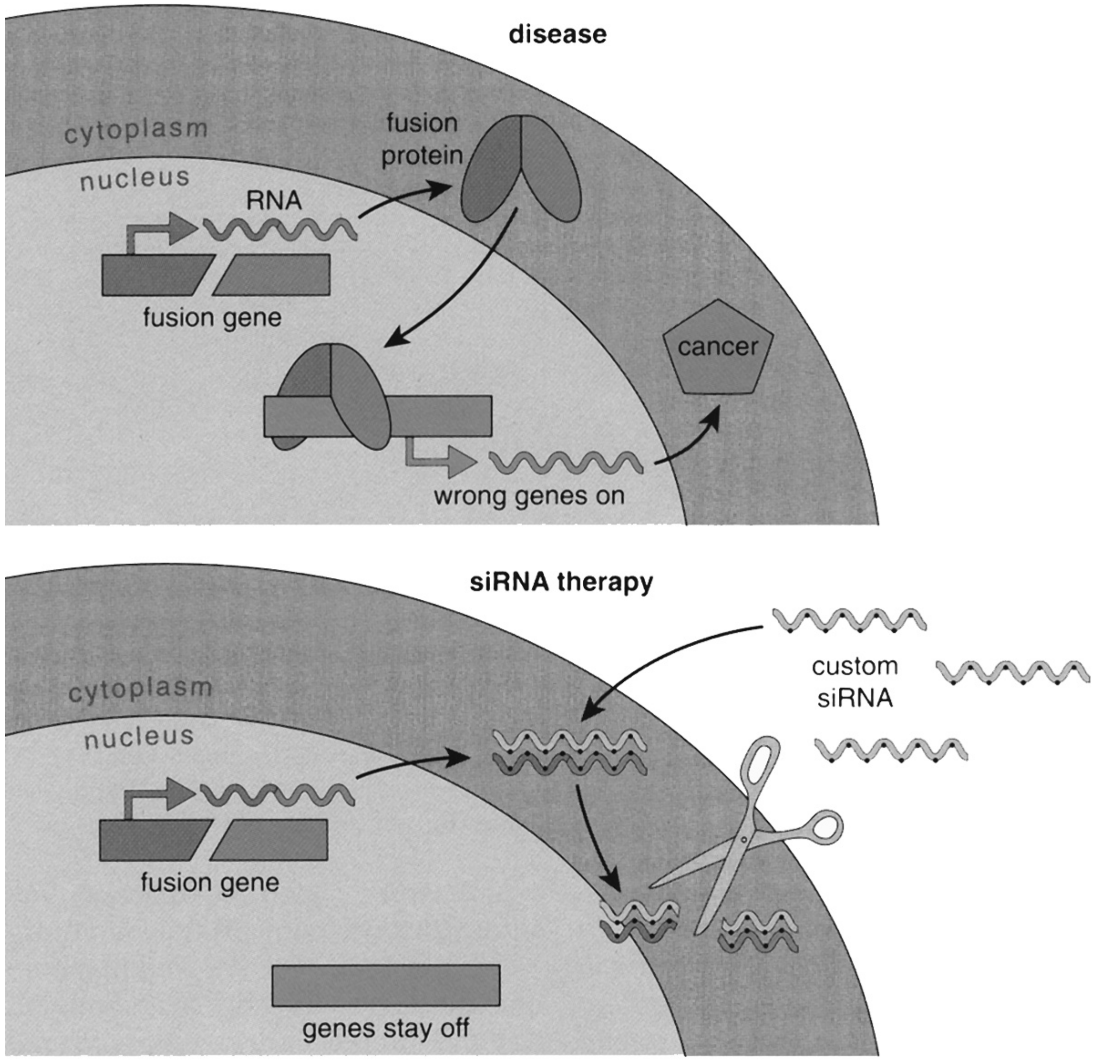

Figure 3: (From American Scientist, issue 731, p. 419, Figure 6): Top: Ewing's sarcoma is caused by a translocation that usually joins a portion of the EWS gene on chromosome 22 to a portion of the FLI1 gene on chromosome 11 (dark and light rectangles). The resulting fusion gene produces a fusion RNA (dark and light wavy lines) that in turn creates the EWS/FLI1 cancer fusion protein. Bottom: Using the technique of RNA interference (RNAi), short interfering strands of RNA (siRNA represented by light wavy lines) that specifically target the Ewing's fusion RNA. The cell recognized the bound Ewing's fusion RNA and the siRNA as abnormal and they are destroyed. Without the EWS/FLI1 RNA, there can be no production of the EWS/FLI1 cancer fusion protein.

that we basically coated our Ewing's cells with a mixture of pure dsRNA. We were very excited to see that not only did RNAi specifically eliminate EWS/FLI1 but this in turn led to the death of the Ewing's cells. We published these results but our enthusiasm was tempered by the realization that his technique would be impossible to use in patients with Ewing's sarcoma that had spread beyond a small focus of disease.

To overcome this hurdle, over the past several years we have been using gene therapy to deliver our siRNA. In our next set of experiments we continued to use Ewing's cells in culture but we employed an adenovirus to deliver the siRNA. The advantages of viral delivery are that theoretically the virus can deliver higher doses of the siRNA to cells anywhere in an actual patient. The downside is that the viral agent itself can cause disease. In our hands, the adenovirus vector was able to achieve an even greater reduction in EWS/FLI1 and more sustained inhibition of the growth of the Ewing's cells (Figure 4). These experiments led to further advances in understanding the biology of Ewing's sarcoma. Again, while these results were very promising, we were still treating cells as opposed to actual Ewing's tumors. In addition, there are certain technical limitations to using adenovirus in living organisms that made it unlikely to ever be used as actually therapy in humans.

Conclusion

We are attempting to circumvent these problems by using a lentivirus to deliver our siRNA into mice that have been implanted with Ewing's sarcoma. Lentivirus poses certain theoretical risks but it also harbors the ability to deliver even higher doses of siRNA for even longer periods of time. In addition, we are designing a lentivirus vector that will actually permit us to turn on and off production of the siRNA.

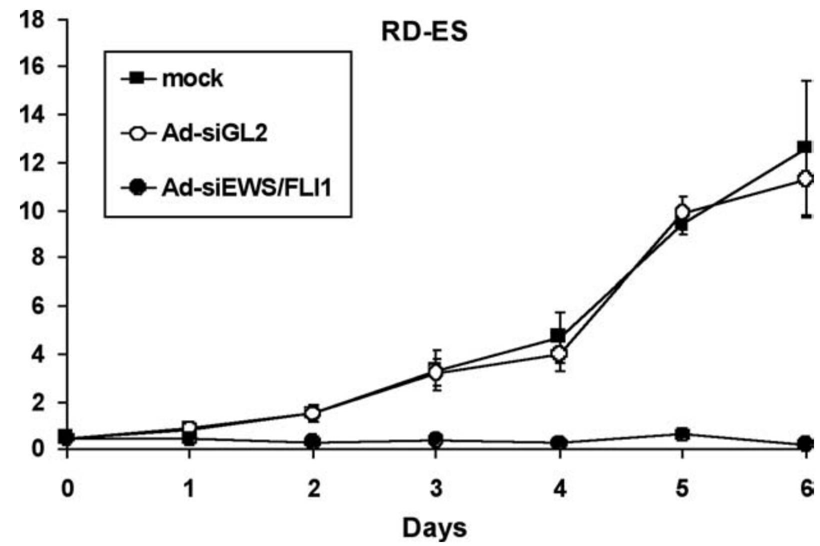

Figure 4: This graph demonstrates the profound effect of treating Ewing's sarcoma cells with virally delivered siRNA targeting EWS/FLI1. The vertical axis represents total cell number and the horizontal axis represents days after treatment with siRNA. The black squares and open round circles denote the control treatments. The black circles are the data points for the treated sarcoma cells. The treated Ewing's cells are unable to multiply while the cells treated with the control (sham) therapy proliferate unabated.

We believe this will also be an aid in deciphering more of the basic biology of Ewing's sarcoma.

We have designed our experiments to use as few mice as possible and to treat them as humanely as possible. Untreated mice will succumb to the disease as the Ewing's cells grow and spread to other organs, just as in humans. In our preliminary experiments, the lentiviral-delivered siRNA inteferes with production of the EWS/FLI1 fusion protein, dramatically shrinking or preventing the growth of the Ewing's tumors. Our preliminary experiments are very promising and we and others believe that as gene therapy vectors (viruses harnessed to deliver biological agents to cells) continue to improve, molecular targeting will be used in lieue of, or in addition to, chemotherapy and radiation therapy in treating Ewing's sarcoma and other cancers. These molecular agents should lead to more effective cancer treatments while sparing patients from some of the toxic effects of chemotherapy and radiation therapy.

Our research has been generously supported by the National Institutes of Health, the Orthopaedic Research and Education Foundation, the Veterans Administration Merit Review Awards, the Florence and Marshall Schwid Memorial Foundation as well as funds from the University of Washington Department of Orthopedics & Sports Medicine.

References

Anonymous (2007). The RNA Revolution. The Economist. June 16. London.

Chansky, H. A., F. Barahmand-Pour, Q. Mei, W. Kahn-Farooqi, A. Zielinska-Kwiatkowska, M. Blackburn, K. Chansky, E. U. Conrad, 3rd, J. D. Bruckner, T. K. Greenlee and L. Yang (2004). "Targeting of EWS/FLI-1 by RNA interference attenuates the tumor phenotype of Ewing's sarcoma cells in vitro." J Orthop Res 22(4): 910-7.

Chansky, H. A., M. Hu, D. D. Hickstein and L. Yang (2001). "Oncogenic TLS/ERG and EWS/Fli-1 fusion proteins inhibit RNA splicing mediated by YB-1 protein." Cancer Res 61(9): 3586-90.

Clinton, J. M., H. A. Chansky, D. D. Odell, A. Zielinska-Kwiatkowska, D. D. Hickstein and L. Yang (2002). "Characterization and expression of the human gene encoding two translocation liposarcoma protein-associated serine-arginine (TASR) proteins." Gene 284(1-2): 141-7.

Hu, H. M., A. Zielinska-Kwiatkowska, K. Munro, J. Wilcox, D. Y. Wu, L. Tang and H. A. Chansky (2008). "EWS/FLI1 Suppresses Retinoblastoma Protein Function and Senescence in Ewing's Sarcoma Cells." J Ortho Res in press.

Matushansky, I. and R. G. Maki (2005). "Fighting Cancer Through The Study of Sarcomas." American Scientist 93(761): 414-421.

Rapp, T. B., L. Yang, E. U. Conrad, 3rd, N. Mandahl and H. A. Chansky (2002). "RNA splicing mediated by YB-1 is inhibited by TLS/CHOP in human myxoid liposarcoma cells." J Orthop Res 20(4): 723-9.

Yang, L., H. A. Chansky and D. D. Hickstein (2000). "EWS. Fli-1 fusion protein interacts with hyperphosphorylated RNA polymerase II and interferes with serine-arginine protein-mediated RNA splicing." J Biol Chem 275(48): 37612-8.

Zou, J., H. Ichikawa, M. L. Blackburn, H. M. Hu, A. Zielinska-Kwiatkowska, Q. Mei, G. J. Roth, H. A. Chansky and L. Yang (2005). "The oncogenic TLS-ERG fusion protein exerts different effects in hematopoietic cells and fibroblasts." Mol Cell Biol 25(14): 6235-46.

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Understanding and Managing the Impact of Vehicle Crashes: Reconstructing the "Accident"

- A car crash may be an "accident" but the way in which forces are transferred to the occupants and their resulting injuries are not accidental - they are defined by the laws of physics and mechanics.
- Research into the biomechanics of injury and the incorporation of innovations such as frontal and side airbags, lap and shoulder belts, and booster seats for children have reduced injury and death from motor vehicle crashes.
- Today, however, significant numbers of people still suffer neck "whiplash" injuries in rear end impacts and serious head, chest, thigh, leg, foot and pelvic injuries in side "T-bone" crashes.
- Discoveries in the Department of Orthopaedics and Sports Medicine are defining the mechanics of these collisions and enabling improvements in vehicle safety equipment.

Despite significant improvements in vehicle safety equipment, including frontal and side airbags, significant numbers of people are still injured or killed every year in auto crashes. The new safety equipment was designed to meet federal regulations based on testing with crash dummies, usually with 'standard' hypothetical types of impact, such as a straight on crash into a wall. As such, they do not address such real world problems as out-of-normal driving positions of occupants and non-standard directions of crashes. The faculty of the Department of Orthopaedics and Sports Medicine have collaborated with the Harborview Injury Research and Prevention Center in a unique program that enables us to determine the actual injuries in real-world crashes. With the consent of the individuals involved, we document the injuries and investigate the specific direction and speed of the crash, the function and use of safety restraints, and the points of occupant contact in the vehicle to determine the mechanics of the injuries sustained. This information, combined with laboratory studies, has provided valuable insights into injury prevention. The following are short summaries of some of the findings.

Femur fractures in head on collisions

Just prior to a frontal collision, the vehicle and occupant are traveling at the same speed. At impact, the vehicle slows very rapidly, but the occupant continues forward at the original speed until encountering the restraint devices. The restraining devices, shown in Figure 1, include the airbag to stop the forward motion of the head, the shoulder belt to stop the torso, the lap belt to stop the pelvis, and the knee bolster to stop the legs. This system attempts to apply forces across the whole body so that the occupant "rides down" the collision. The knee bolster is the padding on the dashboard that absorbs impact forces as the occupant's knees move forward. These have been designed based on biomechanical impact testing of isolated femurs.

However, in a series of crashes, we were perplexed to find that normal adult occupants were sustaining femur fractures at crash speeds well below that at which femur fracture would be expected. To solve this mystery, we constructed computer models of each injury to determine the approximate forces acting. None of the occupants studied had any significant osteoporosis or other bone disease that might indicate weaker than normal bone that would predispose them to femur fracture. Almost all had been drivers, and all were in vehicles that had braked hard prior to impact, as indicated by skid marks from their tires. The forces we calculated did not reach levels expected to create femur fractures, yet each occupant had a documented femur fracture. We then postulated

Figure 1: A schematic diagram showing the forces acting on an occupant during "ride down" of a frontal collision. Force is required to slow the occupant's motion inside the vehicle, and is distributed between; head and airbag, chest and shoulder belt, pelvis and lap belt, and knees and dashboard. The external force on the knee during dashboard contact causes an axial compressive force acting on the femur, which is increased by internal muscles forces due to tensing the leg during braking. Dashboard stiffness has been designed based on the strength of the femur. Muscle contraction adds considerable compressive force to the external force from dashboard contact, resulting in occupants fracturing their femurs at lower than expected impact speeds. Therefore dashboards should be designed to be less stiff to accommodate the combined compressive force from external contact and internal force from muscle contraction.

that additional forces along the axis of the femur could have been applied by the occupant contracting muscles along the leg while bracing during the crash. When the maximum contractile forces applied were estimated based on muscle mass, and added to the calculated force of impact of the femur into the dashboard, the forces exceeded the average values necessary to create a fracture. We concluded that dashboard impact absorption systems, which are based solely on the external forces applied to the femur during contact, do not replicate the scenario in an actual frontal crash where large internal forces may also be applied by muscle contraction. Therefore, we are proposing that dashboard "knee bolsters" may be too stiff as currently designed and may need to be softened in consideration of the internal muscle loads that are associated with these crashes.

Chest and pelvic injuries in side impact (T-bone") collisions

In a side impact ("T-bone") collision, usually at an intersection when one vehicle runs a stoplight, the impacting vehicle hits the door of the struck vehicle, and deforms it inwards. (Alternatively, in a single vehicle crash, the car misses a curve, slides off the road, and contacts a tree with its door.) As the door bows inward, the inner surface of the door contacts the occupant, causing direct contact injuries and pushing the occupant towards the center of the car. In some cases, when the vehicle has a large center console, the occupant may be crushed against it, see Figure 2, causing additional injury. Recently, curtain airbags have become available which significantly protect the head from injury in these collisions, however other types of airbags such as those designed to protect the thorax, have been less effective. This is particularly the case when the occupant driver, sensing an approaching vehicle from the left, attempts to swerve to the right, which causes the driver to be thrown against the driver's side door, just as the airbag installed in the door deploys, which could result in airbag-induced rib fractures.

In reconstructing a series of side impact collisions, we discovered that the most significant factor related to the severity of side impact injuries to the chest and pelvis was the amount of intrusion of the door during the impact. (This is related to the weight and speed of the striking vehicle and the location of contact on the door). Further, in cars with center consoles, injuries were generally more severe. Therefore we are proposing a system that attempts to move the driver away from the incoming door during the impact. The elements of the system, shown in Figure 3, include a crushable center console, a seat with a track that allows it to move sideways towards the center of the car, and a stronger seat structure. In this system, the door hits the side of the seat, not the side of the occupant, and pushes the seat and occupant away from the incoming door into the center console. The console itself is soft enough to be crushed, absorbing the energy that otherwise would case injury. Our ultimate goal is to minimize the risk of serious injury from side impact by optimally managing the full range of forces that might be countered. A patent for this system is pending.

Whiplash injuries of the neck from rear impact

The most common injury in car cashes is "whiplash", which causes damage to the soft tissues of the neck from a low speed rear impact. The impact to the car from behind causes the occupant's body, Figure 4, to be thrust forward. Unless it is in firm contact with head rest, the head does not accompany the body in its forward thrust. Shear and extension loads are applied to the neck, resulting in unnatural distortion of the lower part of the cervical spine. The head then impacts the head restraint that limits the head's rearward motion. However, most head restraints are very elastic and during contact they bend backward, then propel the head forward like bouncing off a trampoline.

Figure 2: Side impact crash in which occupant was trapped between incoming door and the center console and sustained severe pelvic fracture.

An instant later, the forward motion of the torso is stopped by the shoulder belt, but the head continues to move forward, again inducing a horizontal or shearing force in the neck. Finally, as the head continues forward, it bends the neck into forward flexion. In our laboratory we found that these horizontal translations between adjacent vertebrae may be an important mechanism by which the soft tissues of the neck can be injured in a whiplash event.

Even though head restraints are required in all vehicles currently for sale, the incidence of whiplash injury has not diminished. By studying over 400 actual rear end impacts and the seated positions of over 700 drivers, we discovered that in the majority of cases head restraints are not adjusted properly. They are generally set too low and are too far behind the head. We are working to define ways in which the head restraints can be ideally positioned for each individual. We have designed and tested an improved head restraint that allows the user the ability to set its position both vertically and horizontally relative to the head, and that deforms during impact, catches the head, and eliminates most of the forward rebound. This design significantly reduces head to neck motions during rear impact. In the future the frequency of whiplash may be reduced by the application of such designs that protect the neck by keeping the head aligned with the body.

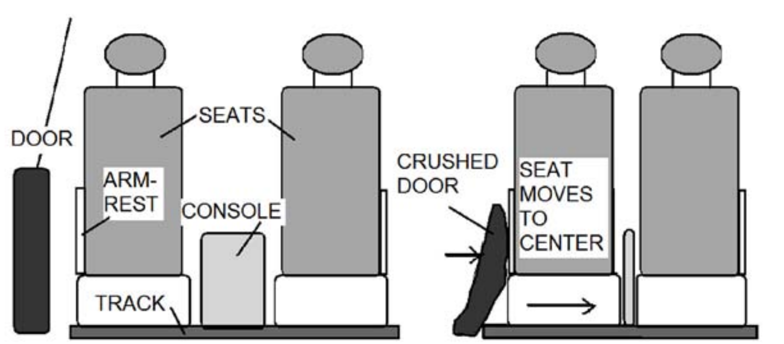

Figure 3: (left) schematic diagram of a (US patent pending) system to prevent or reduce severity of such injuries, the SEATS are on a lateral TRACK with a crushable center CONSOLE and a guard/ARMREST on the door side of the seat, (right) during a side impact, the crushed door impacts the seat and armrest/guard, displaces the seat and occupant towards the center, crushing the center console and moving the occupant away from the door. (Another modification would be that the seatbelts would be attached to the seat).

Conclusion

Investigation of the mechanics of how real occupants respond to the forces acting in real collisions (as opposed to studies limited to crash dummies) provides valuable new insights into the mechanisms of vehicular injuries. These insights will enable us to reduce the potential for occupant injuries in car "accidents". Our team will continue to study the real world of accidents in pursuit of further advances in the protection of the driver and passengers. We have a particular interest in reducing the risk of injuries to children in the rear seat during side impact collisions, recognizing that side impact airbags are designed and positioned for adults.

Acknowledgement

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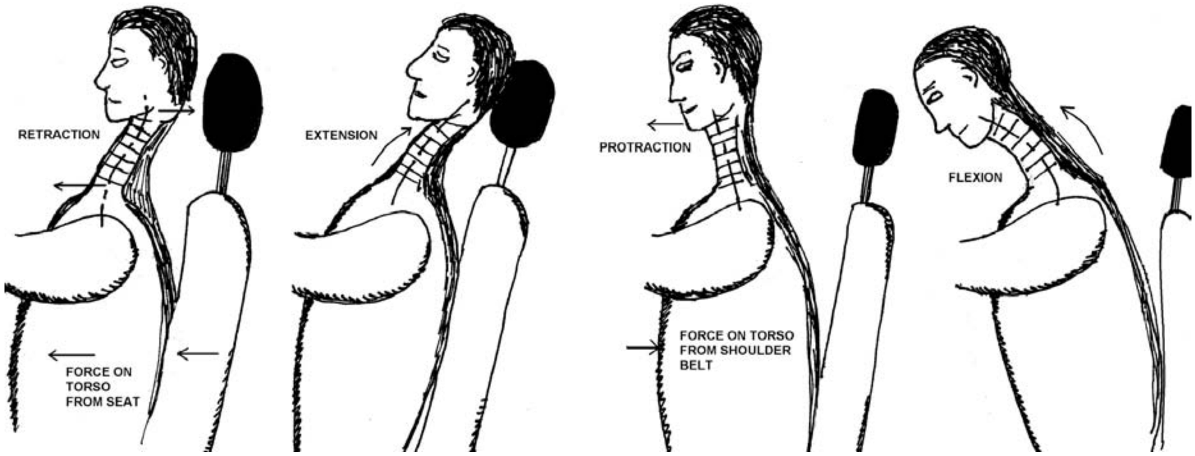
References

Tencer AF, Mirza SK, Whiplash mechanics in low-speed rear-end automobile accidents, J Musculoskeletal Pain 8:1/2:69-86,2000.

AF Tencer, R Kaufman, K Ryan, D Grossman, M B Henley, F Mann, C Mock, F Rivara, S Wang, J Augenstein, D Hoyt, B Eastman, Estimating the loads in femurs of occupants in actual motor vehicle crashes using frontal crash test data, Accident Analysis and Prevention, 34 (1): 1-11, 2002.

AF Tencer, SK Mirza, KD Bensel, The response of human volunteers to rear end motor vehicle impacts, The effect of head restraint properties, Spine



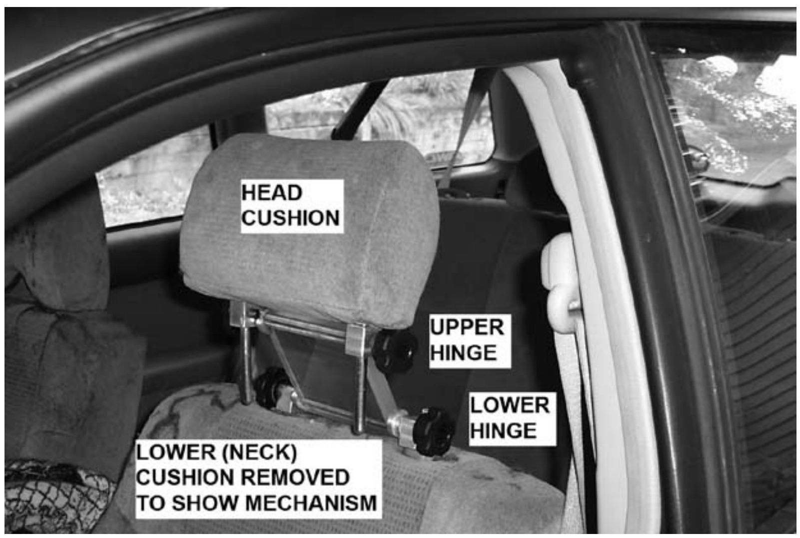
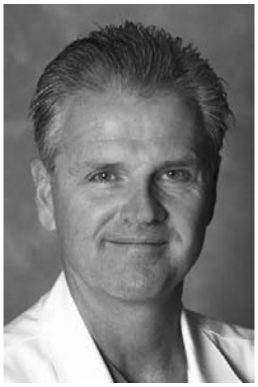


Figure 4: (upper) the four phases of "whiplash", left to right, torso is thrust by seat contact under head causing horizontal shearing or protraction of vertebrae, head falls back into head rest causing neck extension, torso stops due to shoulder belt acting on chest while head rebounds from head rest causing protraction or horizontal shearing, and finally, head continues forward causing neck flexion.

(lower) A device to reduce head to torso motion and neck forces, has a lower hinge to move head rest closer to head (reducing initial retraction), while upper hinge adjusts both a head cushion and neck cushion (not shown) to the occupant's posture, reducing extension. The hinges are "plastic" that is they move slightly and absorb energy from impact with the head and reduce head rebound, thus limiting protraction and flexion of the neck.

26:2432-2440, 2001.

AF Tencer, R Kaufman, P Huber, C Mock, ML Routt, Reducing primary and secondary impact loads on the pelvis during side impact, Traffic Injury Prevention, 8:101-106, 2007.



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Pelvic and Acetabular Surgery: Past, Present, and Future

- Unstable pelvic ring injuries are unusual and potentially fatal injuries.
- Death may occur early after pelvic injury due to associated injuries and/or uncontrolled pelvic related bleeding.
- Poor clinical outcomes in patients with unstable pelvic fractures are blamed on associated nerve injuries, pelvic deformity, and incomplete healing.
- Realignment (reduction) and stable fixation of unstable pelvic ring fractures optimizes early and long-term clinical results.
- Pelvic fracture surgery is complicated for many reasons.
- Stable fixation of pelvic ring injury is founded on both clinical and mechanical research carried out by the faculty of Orthopaedics and Sports Medicine.
- Acetabular (hip socket) fractures are difficult injuries to treat effectively because of their deep and complex anatomy.
- Displaced and unstable acetabular fractures are treated with fracture reduction (realignment) and stable fixation constructs.
- Accurate reduction and stable fixation of acetabular fractures avoids traction, allows early patient mobilization, and lowers the risk of post-traumatic hip arthritis.

Pelvic ring and acetabular injuries are uncommon but potentially devastating. Patients with pelvic and acetabular injuries typically have been involved in high-energy accidents such as automobile and motorcycle crashes. As a result, these patients often have other primary organ system damage including brain, chest, bowel, and other orthopedic injuries.

Pelvic ring disruptions are potentially lethal soon after injury due to uncontrolled pelvic bleeding. When the spherical structure of the pelvis is disrupted by fracture or dislocation, pelvic bleeding may proceed unchecked as the unstable pelvic bones displace and expand allowing continued bleeding and preventing stable clot formation. Early pelvic realignment and stability helps halt such bleeding and provides patient comfort. Definitive pelvic realignment and stable fixation improves healing and avoids disabling deformity and related chronic pain.

Acetabular (hip socket) fractures are similarly complex. These injuries result from excessive loading of the upper femur (thigh bone) onto the acetabulum causing the fracture. As the acetabulum fractures, the femoral head displaces the direction of instability causing a fracture-dislocation. The acetabular cartilage is severely damaged as a result. The femoral head may have decreased blood flow due to the displacement. Early restoration of the acetabular fracture-dislocation includes restoration of the smooth joint surfaces. Definitive fracture reduction and stable fixation are achieved using deep surgical exposures and unique equipment. Stable fixation allows the hip joint and patient to be mobile. The risk of post-traumatic hip arthritis is diminished by accurate repair of acetabular fractures.

Pelvic and acetablar fracture surgeries have evolved significantly, especially over the past 30 years. This evolutionary boom has been due to several factors including improved resuscitation techniques for injured patients, refined regional trauma care systems, increased clinical experience and devotion, advanced technologies such as imaging and surgical equipment, and reliable biomechanical research regarding fracture fixation strategies.

As the regional referral center for the Pacific Northwest, Harborview Medical Center is the perfect site to evaluate and advance the field of pelvic and acetabular surgery.

Biomechanical Laboratory Discoveries

Using departmental grant support

Figure 1 A: This morbidly obese 30 year old male was a struck by an automobile while walking. He had pelvic pain and instability. His plain pelvic radiograph demonstrates complete dislocations of the symphysis pubis and right sided sacroiliac joint dislocation. The left sacroiliac joint injury is associated with a posterior iliac fracture also.

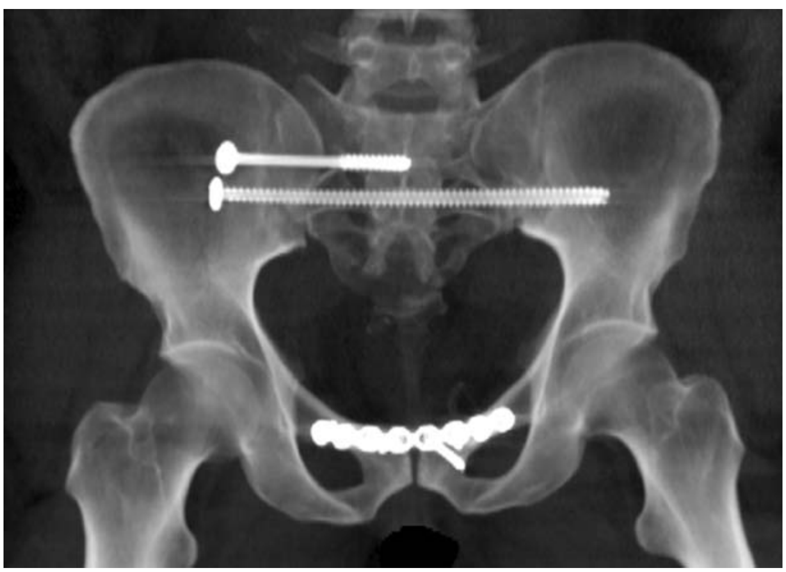


Figure 1 B: After resuscitation and completion of his medical evaluations, he was treated with open reduction of the symphysis pubis. Iliosacral screws were used to stabilize the posterior pelvic ring injuries. Each portion of the procedure was complicated by his obesity.

and under the direction of Dr. Allan Tencer, Richard Harrington and Dr. Peter Simonian, numerous pelvic and acetabular biomechanical investigations focused on fixation techniques. At that time, most surgeons believed that anterior pelvic fixation alone was sufficient to stabilize pelvic ring injuries. Operative treatment of posterior pelvic injuries had caused significant wound complications such as infections. As a result, clinicians sought treatment methods that would avoid open procedures on the posterior pelvis. Fixation failure resulted as the anterior pelvic fixation alone was insufficient for these unstable injuries. Our early pelvic research sought to solve this clinical conundrum. Using fresh cadaveric pelvic specimens and modern biomechanical research tools, we demonstrated that improved pelvic ring stability was easily achieved when each unstable pelvic injury site was realigned and stabilized. The overall pelvic stability gained by fixation of each instability site was impressive and changed our clinical practice. These findings also led to percutaneous (small incision) fixation strategies that avoided more extensive operative exposures and procedures.

Next our lab group evaluated symphysis pubis internal fixation methods. At the time, new internal fixation plates designed specifically for pelvic and acetabular fracture management were introduced but had not been evaluated beyond early clinical assessments. Some clinicians had noted implant and fixation failures, so we investigated a variety of plate fixation techniques using cadaveric pelvic specimens. The stiffest plate fixation construct consisted of a "box-plate" design where two separate plates were linked together using the same screws. Other pre-manufactured plates provided biplanar fixation yet showed no real mechanical benefit.

Armed with this early pelvic biomechanical information, this same research team proceeded to compare different percutaneous screw fixation techniques and compare them to standard plate-screw fixation implants. The subsequent biomechanical investigations focused on posterior (iliosacral) and anterior (medullary pubic ramus) screw fixation techniques. Again cadaveric pelvic specimens demonstrated that both iliosacral and medullary ramus screw fixations had similar stability when compared to the standard plate and screw fixation techniques. These findings supported clinical use of screw fixation techniques. These screws could be inserted using small incisions and therefore could avoid wound problems, lower operative blood losses, and decrease infection risks.

An important biomechanical evaluation of lumbopelvic fixation was also performed under the direction of Dr. Thomas Schildhauer. Lumbopelvic fixation is an extensive posterior pelvic operative procedure reserved for the most difficult and unstable posterior pelvic ring injuries. This cadaveric research showed that lumbopelvic fixation was more stable than previous fixation techniques.

Acetabular fracture fixation methods were also evaluated in the Biomechanical laboratory. Dr. Simonian and the other members of the research team evaluated different fixation

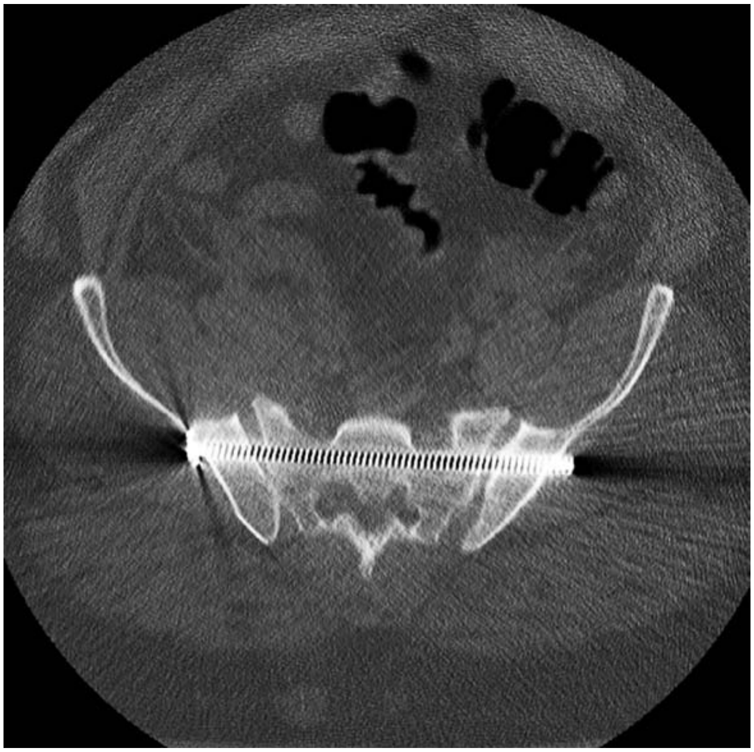


Figure 1 C: The pelvic computed tomography scan after surgery demonstrates the obesity, the second sacral segment screw location, and the posterior pelvic reductions bilaterally.

techniques for complex acetabular fracture patterns and, similar to the earlier pelvic stability lab studies found that fixation of each fracture component improved overall acetabular stability.

Clinical Discoveries

Clinical discoveries matched increasing patient volumes and their injury complexities. As more severely injured patients presented for management in increasing numbers, our management strategies and surgical techniques simultaneously became more advanced, refined, aggressive, and subsequently routine.

Simple techniques such as circumferential pelvic wrapping and two pin anterior pelvic external fixation were developed to decrease bleeding during patient resuscitation. The pelvic realignment in the circumferential wrap often allows percutaneous fixation to proceed using surgical portals cut from the wrap.

In collaboration with our general surgeon colleagues, we began to reduce and stabilize pelvic ring disruptions as soon as the patients were medically cleared after their injury. Such early pelvic fixation proved to benefit the patients. Similarly, we treated pelvic ring and genitourinary injuries in conjunction with our urology partners during the same anesthetic and often using the same surgical exposure. These coordinated operative procedures allowed pelvic fracture fixation and bladder or urethral repairs to be combined without complications.

Iliosacral screw techniques were evaluated critically at Harborview. The necessary pelvic radiology, manipulative reduction methods, technical details, and complications were described in our clinical series. As our clinical work expanded, we reported upper sacral dysmorphism, including its incidence, radiology, and impact on posterior pelvic fixation. Based on these findings, we then described the osteology and radiology of upper sacral dysmorphism and its relation to safe screw insertions within the upper and second sacral segments. Percutaneous posterior pelvic reduction and fixation methods were improved and made safer as a result of these clinical discoveries.

Acetabular fracture discoveries mirrored our pelvic findings. Based on the expanding patient volumes and acetabular fracture complexities, we developed predictable and reliable surgical exposures, reduction methods, and fixation techniques. Most recently, the acetabular intrapelvic plating procedure has been investigated, and its technical details and related complications have been detailed.

What Comes Next?

Unfortunately we have only scratched the surface of what we need to know about pelvic and acetabular injuries. Current educational efforts are helping clinicians to better understand these complex injuries, their treatments, and complications. This subspecialty field will continue to advance and benefit from careful clinical evaluations of our treatments and decision-making strategies. Our research efforts should focus on every detailed aspect including safety standards, accident prevention, resuscitation, evaluation, management, and recovery. It is vital that clinicians and investigators continue to integrate their research efforts.

Support

The biomechanical pelvic and acetabular research cited above was supported by several departmental grants from the University of Washington Department of Orthopedic Surgery. To assure ongoing discoveries in the management of pelvic and acetabular injuries, the Department of Orthopaedic and Sports Medicine is committed to establishing an endowed chair in pelvic and acetabular surgery.

References

Simonian, P.T., Routt Jr., M.L., Harrington, R.M., Tencer, A.F.: Internal Fixation of the Unstable Anterior Pelvic Ring: A Biomechanical Comparison of Standard Plating Techniques and the Retrograde Medullary Superior Pubic Ramus Screw. J Ortho Trauma, 8:476-482, 1994.

Simonian, P.T., Routt Jr., M.L., Harrington, R.M., Tencer, A.F.: Box Plate Fixation of the Symphysis Pubis: Biomechanical Evaluation of a New Technique. J Ortho Trauma, 8:483-489, 1994.

Routt Jr., M.L., Simonian, P.T., Grujic, L: The Retrograde Medullary Superior Pubic Ramus Screw for the Treatment of Anterior Pelvic Ring Disruptions: A New Technique. J Ortho Trauma,

Figure 2 A: This 52 year old male was injured in a fall. He complained of right hip pain and was unable to walk. This pelvic 3-D CT scan image demonstrate his irreducible acetabular fracture-dislocation.

Figure 2 B: He was treated with urgent open reduction using an ilioinguinal exposure. The fracture was reduced and stabilized using an intrapelvic plating technique.

9:35-44, 1995.

Routt Jr., M.L., Kregor, P.J., Simonian, P.T., K.Mayo: Early Results of Percutaneous Iliosacral Screws Placed with the Patient in the Supine Position. J Ortho Trauma, 9:207-214, 1995.

Teague D.C., Graney, D.O., Routt Jr., M.L.: Retropubic Vascular Hazards of the Ilioinguenal Exposure: A Cadaveric and Clinical Study. J Ortho Trauma, 10:156-159, 1996.

Routt Jr., M.L., Simonian, P.T., Agnew, S., Mann, F.: Radiographic Recognition of The Sacral Alar Slope Facilitates Optimal Placement of Iliosacral Screws: A Cadaveric and Clinical Study. J Ortho Trauma, 10:171-177, 1996.

Routt, M.L., Simonian, P.T., Mills, W.J: Iliosacral Screw Fixation: Early Complications of the Percutaneous Technique. J Ortho Trauma, 11(8):584-589, 1997.

Patel, N, Hunter, J., Weber, T., Routt, M.L.: Rotational Imaging of Complex Acetabular Fractures. J Ortho Trauma, 12(1):59-63, 1998.

Routt, M.L., Nork, S.E., Mills, W.J: Percutaneous Fixation of Pelvic Ring Disruptions. Clin Orthop, 375:15-29, 2000.

Switzer, J.A., Nork, S.E., Routt, M.L.: Comminuted and Unstable Iliac Fractures. J Ortho Trauma, Vol 14, No. 4, pp. 270-276, May, 2000.

Altman, G.T., Altman, D.T., Routt, M.L.: Symptomatic Hypertrophic Pubic Ramus Nonunion Treated With A Retrograde Medullary Screw. J Ortho Trauma, Vol 14, No. 8, pp.82-88, November, 2000.

Tucker M, Nork SE, Simonian PT, and Routt ML: Simple Anterior Pelvic External Fixation. J Trauma, 49(6):989-94, December 2000.

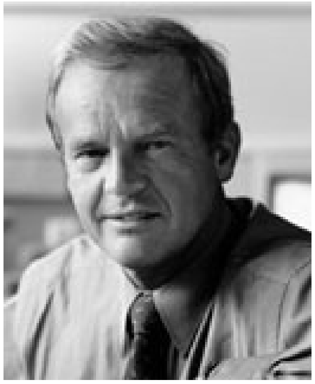
Nork SE, Jones, CB, Harding, SP, Mirza, SK, Routt, ML Jr,: Percutaneous Stabilization of U-shaped Sacral Fractures Using Iliosacral Screws: Technique and Early Results, J Orthop Trauma, 15 (4) 238-46; May 2001.

Barei, DP, Bellabarba, C, Mills, WJ, Routt, ML Jr.: Percutaneous Management of Unstable Pelvic Ring Disruptions, Injury, 32 Suppl.1: SA33-44; May 2001.

Russell GV Jr, Nork SE, Chip Routt ML Jr. Perioperative complications associated with operative treatment of acetabular fractures. J Trauma. 2001 Dec;51(6):1098-103.

Routt, ML Jr., Nork, SE, Mills, WJ: High-energy Pelvic Ring Disruptions, Orthop Clin North Am., 33 (1) 59-72; viii, Jan 2002.

Schildhauer, T.A., Ledoux, W.R., Chapman, J.R., Henley, M.B., Tencer, A.F., Routt, M.L.: Triangular Osteosynthesis and Iliosacral Screw Fixation for Unstable Sacral Fractures: A Cadaveric and Biomechanical Evaluation Under Cyclic Loads. J Orthop Trauma, 17(1) 22-31; Jan 2003.



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Collagen, the Skeleton's Scaffolding: From Basic Science to Clinical Applications

- Collagen, the protein polymer underlying the mechanical strength of essentially all tissues, comes in many different varieties. The highly evolved frameworks of bone and cartilage are examples.
- Defects in collagen, from genetic abnormalities to acquired proteolytic damage, are key to understanding many common human diseases, for example brittle bones (osteoporosis) or worn out joints (osteoarthritis).
- Our basic research on collagen has led to new approaches for monitoring bone metabolism clinically and advances in knowledge on the basic science of cartilage.
- Examples of such bench-to-bedside progress include the development of a whole new class of biomarkers for assessing bone breakdown in osteoporosis, a sophisticated test for a rare inherited form of brittle bones and insights on the unique structure of collagens in joint cartilages that explain their longevity.
- In the next decade a breakthrough is likely in understanding the mechanism by which bone cells mineralize collagen with potential new approaches for preventing and treating osteoporosis.
- Likewise progress defining mechanisms that underlie the destruction of the collagen fabric of articular cartilage in osteoarthritis and of intervertebral discs in the painful spine will lead to new therapeutic approaches.

In 1984 the Ernest M. Burgess Chair was created through the generosity of local donors to recruit and support a research scientist with a program of excellence central to the clinical challenges presented by musculoskeletal disorders. The Chair was named in honor of the many contributions of Ernest Burgess, a leading orthopædic surgeon in the Pacific Northwest, and I have been honored to be its first holder. In this departmental issue highlighting discoveries, it seems appropriate to relate the tale of how basic science from the early days of the Burgess Chair program translated into medical progress.

We were interested in understanding the protein chemistry responsible for the unique cross-linking properties of bone collagen and hence bone's remarkable strength. From this knowledge we found that cross-linked collagen fragments from resorbed bone were excreted as short peptides of a specific length in urine. This discovery suggested a new and specific way of quantifying how much bone was being broken down by any subject or patient on a daily basis. Since osteoporosis and other metabolic disorders of bone were suspected from indirect and relatively non-specific methods to be associated with accelerated bone breakdown, there was a need for more sensitive and specific biochemical assays for monitoring the process. We disclosed this potentially important invention through the UW technology transfer process in 1987 while continuing to pursue the basic science and sought mechanisms to fund further clinical development.

To cut a long story short a local company, Ostex, was founded in 1989 through local investors and a license to exclusive rights in order to commercialize products from the technology. In the early days the main effort was basic research to validate the clinical potential funded through a research grant to the department from the fledgling company. Within a year we had a working bone resorption assay and very promising pilot data in collaboration with Merck from

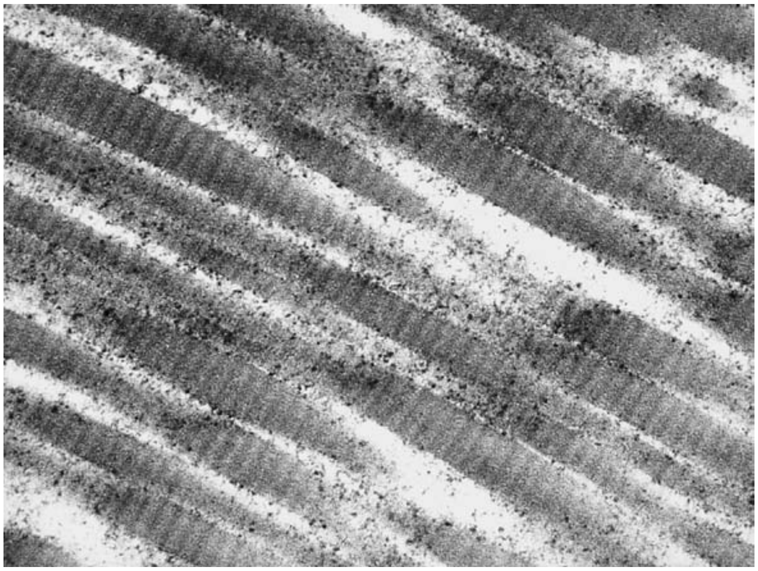

Figure 1 A: Typical banded fibrils of collagen seen by electron microscopy are the supporting fabric of all skeletal tissues.

monitoring patients in their trial of an osteoporosis drug under development. This drug was approved and became Fosamax. At that time Ostex opened their own doors to commercialize the pilot assay and evolve it to meet clinical needs and laboratory standards. To fast forward, Ostex was successful in doing this, in building pharmaceutical partners in the US, Europe and Japan and went on to become a public company in 1995. The company was eventually bought out in 2003.

Over the years, the material benefits to the Department and the UW from this bench-to-bedside example of technology transfer include several million dollars in research support as grants from Ostex, revenue in the form of license fees and sale of founders' stock awarded to the institution and substantial ongoing royalty payments from patents to the technology held by the UW / Washington Research Foundation. The original vision of the Department that the Burgess Chair should recruit and support a basic science program that could garner public and private research support and so catalyze clinical translation from basic discoveries has been amply borne out by this example. The pure science that led to this technology transfer (studying basic collagen biology) had no immediate or obvious commercial potential when initiated. In addition to material returns, the technology transferred produced many benefits in basic scientific progress. These included new clinical methods for probing the metabolism of bone and cartilage, vitalizing a field of research on bone and other skeletal tissue biomarkers and providing us with a rich collection of monoclonal antibodies and molecular tools that have opened new applications and research directions.

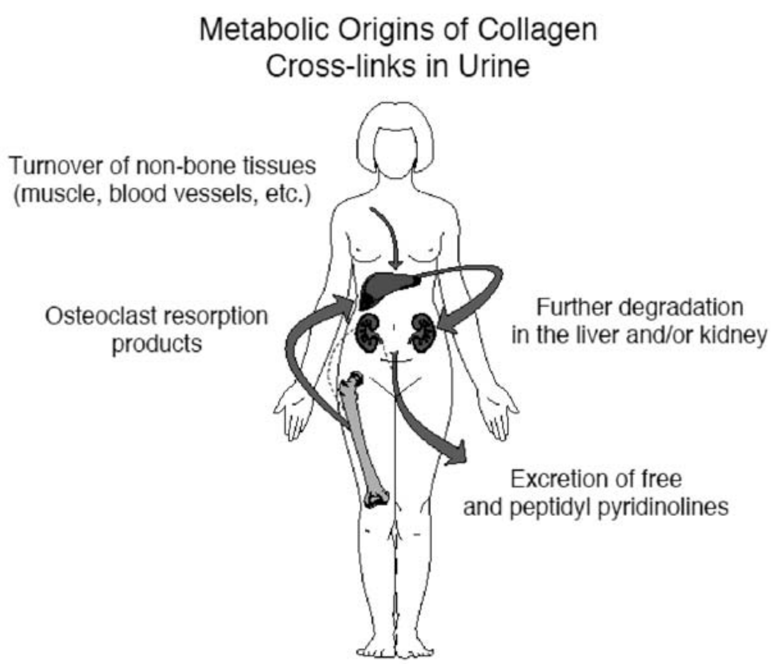

Figure 1 B: Collagen is broken down as part of normal tissue remodeling processes resulting in peptide fragments being excreted in urine. Bone is the major contributor to this pool, and the rate of bone turnover can be assessed by tests designed to measure the bone-specific collagen fragments selectively.

Beyond this specific example of a technology transfer, NIH-funded research on collagen in cartilage has also been productive in advancing scientific knowledge relevant to arthritis. Collagen in cartilage is unique in structure and function having evolved into a more complex polymer that incorporates different gene products to those in bone collagen. We have been pursuing the structure of cartilage collagen with a view to understanding how the fabric of cartilage is destroyed in osteoarthritis since, once the collagen framework has failed, the joint is doomed. The body has not evolved a mechanism for regenerating articular cartilage, unlike bone.

It turns out that a critical element in the formation of durable articular cartilage is a quantitatively minor form of collagen (collagen type IX). This component is linked by strong (chemical) bonds to the surface of the individual collagen fibrils that form the characteristic meshwork that frames cartilage (the strands extending from the surface of the molecular model of a fibril in Figure 2). We know that genetic disorders that prevent collagen

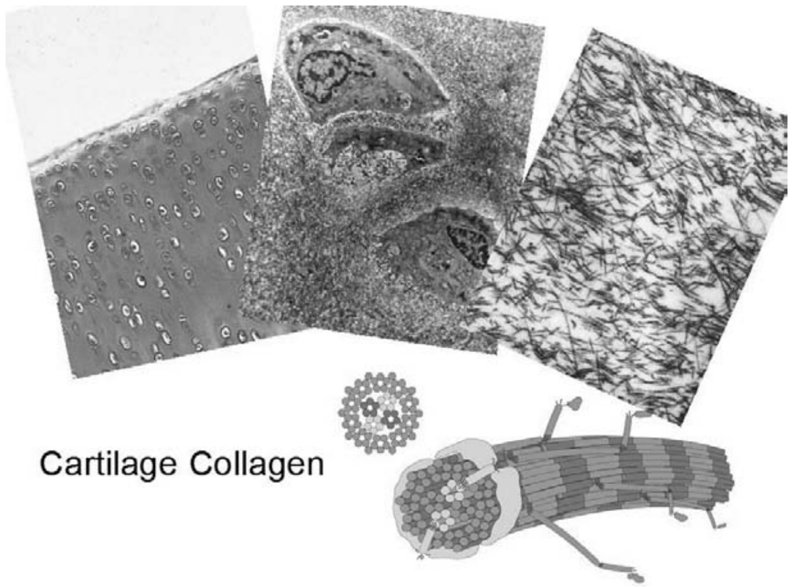

Figure 2: A montage showing progressively higher magnifications of articular cartilage from full thickness tissue (left), chondrocytes surrounded by matrix (center), and the underlying collagen fibril framework that forms the bulk of the tissue (right). Molecular models of an individual collagen fibril in cross-section and side-view are shown below.

type IX from forming normally cause prominent osteoarthritis of early onset. This unusual protein is required for the maintenance of long-lived joints including those formed by the intervertebral discs of the spine. We continue to pursue the basic mechanism by which this specialized type of collagen protects joint cartilages and how the knowledge might be translated into clinical advances.

Conclusions

There is no substitute for rigorous scientific method in the pursuit of medical breakthroughs. Basic research in the medical sciences is the foundation of long-term future cures and preventions.

Support

We have been fortunate in securing strong and sustained support from the NIH (NIAMS and NICHD) for this work since the inception of the Burgess Chair program in addition to the support from Ostex and other industrial sponsors.

Selected Publications

Eyre DR, Apone S, Wu JJ, Ericsson LH, Walsh KA. Collagen type IX: Evidence for covalent linkages to type II collagen in cartilage. FEBS Lett. 1987; 220:337-341.

Hanson DA, Weis M-A E, Bollen A-M, Maslan SL, Singer FR, Eyre DR. A specific immunoassay for monitoring human bone resorption: Quantitation of type I collagen cross-linked N-telopeptides in urine. J. Bone Miner. Res. 1992; 7:121-1258.

Gertz BJ, Shao P, Hanson DA, Quan H, Harris ST, Genant HK, Chesnut CH III, Eyre DR. Monitoring bone resorption in early postmenopausal women by an immunoassay for cross-linked collagen peptides in urine. J. Bone Miner. Res. 1994; 9:135-142.

Eyre DR. New Molecular Markers of Bone Metabolism. Ther. Res. 1994; 15:100-111.

Bollen A-M, Eyre DR. Bone resorption rates in children monitored by the urinary assay of collagen type I cross-linked peptides. Bone 1994; 15:31-34.

Diab M, Wu J-J, Eyre DR. Collagen type IX from human cartilage: A structural profile of intermolecular cross-linking sites. Biochem. J. 1996; 314:327-332.

Eyre DR. Bone biomarkers as tools in osteoporosis management. Spine 1997; 22(24S):17S-24S.

Eyre DR. biochemical basis of collagen metabolites as bone turnover markers. In: Raisz L, Rodin G, Bilezikian JP, eds. Principles of Bone Biology, Academic Press, London. 1996; 143-153.

Eyre DR, Atley LM, Wu J-J. Collagen Cross-links as Markers of Bone and Cartilage Degradation. In: Hascall VC, Kuettner KE, eds. The Many Faces of Osteoarthritis. Birkhäuser Verlag, Basel, Switzerland, 2002; 275-284.

Eyre DR. New biomarkers for bone resorption. J. Clin. Endocrinol. Metab. 1992; 74(3):470A-470C.

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Hip Arthritis and Replacement at the University of Washington: The Pursuit of Minimally Invasive Surgery

- Arthritis of the hip refers to the destruction of cartilage between the femoral head (ball of the thigh bone) and the acetabulum (hip socket of the pelvis). Hip arthritis affects millions, causing pain and stiffness, and results in difficulty performing normal activities, such as walking, dressing, and getting in and out of a car. Patients are often unable to enjoy recreational activities, such as golf, cycling, and hiking.
- Arthritis of the hip can result from several causes; the most common is osteoarthritis, while rheumatoid disease, injury or fracture, and avascular necrosis are other frequent causes. Many patients with hip arthritis can be managed with non-operative interventions, such as changing activity, medication, or physical therapy. However, when pain and disability become severe, patients can choose to have hip replacement surgery. A successful hip joint replacement can restore comfort and function, with long lasting and durable results. About 300,000 patients undergo hip replacement every year in the United States.
- The surgeons of the Department of Orthopaedics and Sports Medicine are using newer "minimally-invasive" approaches that appear to permit faster and less-painful recovery following total hip replacement surgery.
- Our arthritis surgery team is working to discover ways to prevent functional limitations for patients with arthritis, by identifying patients at risk, by minimizing the progression of the condition, and by speeding the recovery from hip reconstructive surgery.

Total hip arthroplasty (THA) is a common orthopaedic procedure performed for degenerative joint disease, other arthritic conditions, and avascular necrosis of the femoral head. Currently, approximately 300,000 hip replacements are performed in the United States each year, with this number expected to double over the next 20 years.

Initially, hip replacement was reserved for the elderly and infirm, with low demands and expectations. Increasingly, however, younger and more active patients with hip arthritis are seeking hip replacement to help them regain active lifestyle. The increased activity levels and longevity of these patients challenge us to discover more robust and durable approaches to hip joint replacement.

Hip Arthritis Research at UW

The University of Washington Department of Orthopaedics and Sports Medicine has a long history of active research into hip arthritis and hip surgery. Over 20 years ago, John Clark performed basic anatomic studies on the muscles around the hip as well as the shape and orientation of the bone. Other surgeons, such as William Lanzer, explored the use of core decompression for restoration of blood flow in the femoral head in avascular necrosis. Dr. Lanzer also looked at the results of joint replacement in obese patients and found, importantly, that they enjoy the same good results as other patients.

The goal of all medical research is to go "from the bench to the bedside." Dr. David Eyre, Research Professor, along with Dr. Howard Chansky, have been in the forefront of study of "biomarkers," or blood-borne molecules, which could be used to evaluate risk, presence of arthritis in a patient, or to tailor treatments for the disease in an individual.

University of Washington physicians have also been leaders in refining techniques of hip replacement, particularly revision. Dr. Seth Leopold has been active in studying the use of new surgical and diagnostic methods for complex cases with significant bone

Figure 1: Postoperative dressing for two-incision hip

loss, allowing safe reconstruction. In addition, he has studied how well physicians perform common noninvasive procedures, such as joint injections, with a view towards improving accuracy and teaching of these procedures.

While there are many areas of active inquiry in the subspecialty of hip replacement, perhaps the most controversial one - and the one that has had the most dramatic impact upon the clinical practice of hip replacement at UW and elsewhere that has arisen in the last decade or two - is "minimally invasive" hip replacement. Recent studies performed by our faculty have examined the use of a two-incision technique versus the standard approach for component placement, complication rate, and cost. There are three major categories of approaches: a standard approach, a single smaller incision, or a two-incision minimally invasive approach. Standard approaches utilize incisions that range from 15-30 cm, allowing direct visualization of both the proximal femur and the acetabulum. Single small incision approaches represent modifications of these, with special retractors and instrumentation to permit placement of components through incisions ranging from 6-10 cm. More recently, Mears developed a two incision minimally invasive surgery technique, popularized by Berger, which utilizes a 5 cm anterior incision for cutting the femoral neck and placing the acetabular component and a 2-3 cm posterior incision for reaming and placement of the femoral components. The smaller incisions are possible with use of lighted retractors and fluoroscopy to guide the cut of the femoral neck as well as to place the acetabular and femoral components.

Surgeons at the University of Washington were early adopters and researchers in the use of these minimally invasive approaches. Currently, our hip and knee team, Paul Manner, Seth Leopold, and Jason Weisstein, are continuing to evaluate these techniques, with followup studies to previously published reports on the accuracy of component placement, the complication rate, and the cost benefit of minimally invasive hip replacement. The research is based on the studies performed by Paul Manner while at the George Washington University before joining our faculty.

Preventing and managing hip joint infections

Another area of prime interest concerns the diagnosis and treatment of the infected joint replacement. Infection, though uncommon, is a devastating complication of joint replacement, and results in significant pain and suffering for the afflicted patient.

Diagnosis

Septic arthritis of adults in both native and prosthetic joints is associated with significant morbidity. The accurate and rapid diagnosis of a true infection is imperative because the best outcomes occur when infection is correctly diagnosed early. Although microbiological bacterial culture is currently the gold standard of diagnosis, many studies document significant false negative and false positive rates. Furthermore, the results of cultures are often not conclusive until days or even weeks after they are obtained. Misdiagnosis with standard microbiological testing has led to significant chronic complications, such as persistent infection, conversion of acute to chronic infections, and even death. The occurrence of false negative cultures, the inability to accurately diagnose infection in a patient on antibiotics, as well as long bacterial cultures incubation periods, undermine timely decision making for proper treatment of a suspected septic joint. These issues have societal as well as individual ramifications when considering potential unnecessary treatment and hospitalizations, longer hospital stays, more operations, increased cost, and increased antimicrobial resistance because of broad spectrum empiric treatment for unknown bacteria.

Recent investigations support the use of polymerase chain reaction (PCR) in the detection of bacterial DNA as a more sensitive method of identifying joint infection. PCR involves a process of selectively amplifying and detecting known target DNA or RNA sequences present in the suspected organism. The University of Washington Department of Orthopaedics and Sports Medicine is currently collaborating with the Cartilage Biology and Orthopaedics Branch of the National Institute of Arthritis, Musculoskeletal and Skin Diseases (NIAMS), at the NIH, on further evaluation of PCR as a potential rapid diagnostic tool for joint infection.

Treatment

In Europe and some parts of the United States, a one stage revision surgery (direct exchange arthroplasty) is employed to treat the infected hip arthroplasty. Higher reinfection rates have consistently been reported with

Figure 2: Diagram of hip resurfacing

the one stage approach when compared to the more commonly utilized staged treatment protocol. At present, in the United States, a staged revision is the treatment of choice for eradicating infection around a hip arthroplasty. However, with long term follow up and consideration for other etiologies of prosthesis failure, the two approaches are found to have similar overall rates of failure. The staged procedure also has significant morbidity with two major surgical interventions in patients who are often debilitated. Consideration must be made for the low quality of life imposed on patients treated with a prolonged staged approach for eradicating infection.

At the University of Washington, we are using decision analysis, a statistical tool based on Bayesian theory, to assess the value of these approaches. It has proved an invaluable method of interpreting clinical data while incorporating the patient's perspective of a given outcome, and we are completing a final analysis for publication.

The future of the management of hip arthritis at University of Washington

Hip resurfacing

Surface replacement for the hip was initially introduced in 1950s, then again in 1970s. However, durability of these implants was poor, with an unacceptable early failure rate, and hip resurfacing was abandoned by most surgeons. However, led by surgeons in the United Kingdom and elsewhere, there has been a resurgence of surface replacement over the past decade because of improvements in design, bearing surface, and instrumentation. As a result, surface replacement has become the fastest-growing segment of all total hip arthroplasties worldwide.

Surgeons at the University of Washington now perform hip resurfacing as well as total hip replacement. Over the course of the next several years, we will be creating and expanding a database and registry to follow the outcome of our hip resurfacing patients. Jason Weisstein, who also has an impressive background in complex tumor surgery, will be taking the lead on this new and promising approach to hip arthritis.

Biologic solutions to arthritis

In addition, collaborations with outside groups, such as the Cartilage Biology and Orthopaedics Branch at the National Institute of Arthritis Musculoskeletal and Skin Diseases, and the UWEB (University of Washington Engineered Biomaterials) have been ongoing. Prior to arriving at the University of Washington, Paul Manner was involved in the use of certain populations of adult stem

Figure 3: Hip resurfacing implant

cells, known as mesenchymal stem cells, for treatment of arthritis. Using these cells, obtained from the bone or bone marrow of patients undergoing hip or knee replacement, the NIH group was able to recreate cartilage surfaces. More recently, collaboration between UWEB and the Department of Orthopaedics has explored the use of a nanostructured tissue engineered composite as a framework for stem cells to recreate both bone and cartilage. Russell J. Fernandes has been working to discover ways to enable the body to regenerate the cartilage that normally covers the surfaces of the hip joint. The ultimate goal of these studies is to create a biologic reconstruction of the arthritic joint.

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2. The National Institutes of Health Intramural Research Program (Paul A. Manner, MD)
3. The Wallace H. Coulter Foundation (Paul A. Manner, MD)

References

Birmingham P, Helm JM, Manner PA, Tuan RS, Joint Infection Assessment by Rapid Detection of Live Bacteria via Real-Time Reverse Transcription Polymerase Chain Reaction. (Manuscript accepted for publication, Journal of Bone and Joint Surgery).

Braman JP, Bruckner JD, Clark JM, Norman AG, Chansky HA. Articular cartilage adjacent to experimental defects is subject to atypical strains. Clin Orthop Relat Res. 2005 Jan;(430):202-7.

Clark JM, Freeman MA, Witham D. The relationship of neck orientation to the shape of the proximal femur. J Arthroplasty. 1987;2(2):99-109.

Eyre DR, Pietka T, Weis MA, Wu JJ. Covalent cross-linking of the NC1 domain of collagen type IX to collagen type II in cartilage. J Biol Chem. Jan 23; 279(4):2568-74, 2004.

Eyre DR. Collagens and cartilage matrix homeostasis.Clin Orthop Relat Res. 2004 Oct;(427 Suppl):S118-22.

Leopold SS, Battista V, Oliverio JA. Safety and efficacy of intraarticular hip injection using anatomic landmarks. Clin Orthop Relat Res. 2001 Oct;(391):192-7.

Levine MJ, West K, Michelson JD, Manner PA. Retrospective Comparison of Two-Incision Total Hip Arthroplasty with a Standard Direct Lateral Approach: A Single Surgeon's Experience, Levine et al, Seminars in Arthroplasty 2007 Dec;18(4).

Lohmander LS, Atley LM, Pietka TA, Eyre DR. The release of cross-linked peptides from type II collagen into human synovial fluid is increased soon after joint injury and in osteoarthritis. Arthritis Rheum. Nov; 48(11): 3130-9, 2003.

Martell JM, Leopold SS, Liu X. The effect of joint loading on acetabular wear measurement in total hip arthroplasty. J Arthroplasty. 2000 Jun; 15(4):512-8.

Matyas JR, Atley L, Ionescu M, Eyre DR, Poole AR. Analysis of cartilage biomarkers in the early phases of canine experimental osteoarthritis. Arthritis Rheum. 2004 Feb;50(2):543-52.

McCarron J, Baumbusch C, Michelson JD, Manner PA. Economic evaluation of peri-operative admissions for direct lateral versus 2-incision minimally invasive total hip arthroplasty. (Manuscript accepted for publication, Seminars in Arthroplasty).

Morgan HD, McCallister W, Cho MS, Casnellie MT, Leopold SS. Impaction allografting for femoral component revision: clinical update. Clin Orthop Relat Res. 2004 Mar;(420):160-8.

Munjal S, Leopold SS, Kornreich D, Shott S, Finn HA. CT-generated 3-dimensional models for complex acetabular reconstruction. J Arthroplasty. 2000 Aug;15(5):644-53.

Powell ET, Lanzer WL, Mankey MG. Core decompression for early osteonecrosis of the hip in high risk patients. Clin Orthop Relat Res. 1997 Feb;(335):181-9.

Stickles B, Phillips L, Brox WT, Owens B, Lanzer WL. Defining the relationship between obesity and total joint arthroplasty. Obes Res. 2001 Mar;9(3):219-23.

Tuli R, Nandi S, Li WJ, Tuli S, Huang X, Manner PA, Laquerriere P, Nöth U, Hall DJ, Tuan RS. Human mesenchymal progenitor cell-based tissue engineering of a single-unit osteochondral construct. Tissue Eng. 2004 Jul-Aug;10(7-8):1169-79.

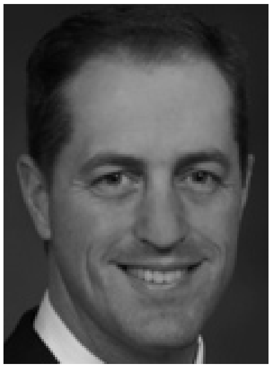
Tuli R, Seghatoleslami MR, Tuli S, Wang ML, Hozack WJ, Manner PA, Danielson KG, Tuan RS. A simple, high-yield method for obtaining multipotential mesenchymal progenitor cells from trabecular bone. Mol Biotechnol. 2003 Jan;23(1):37-49.

Tuli R, Tuli S, Nandi S, Huang X, Manner PA, Hozack WJ, Danielson KG, Hall DJ, Tuan RS. Transforming growth factor-beta-mediated chondrogenesis of human mesenchymal progenitor cells involves N-cadherin and mitogen-activated protein kinase and Wnt signaling cross-talk. J Biol Chem. 2003 Oct 17;278(42):41227-36.

Wang ML, Tuli R, Manner PA, Sharkey PF, Hall DJ, Tuan RS. Direct and indirect induction of apoptosis in human mesenchymal stem cells in response to titanium particles. J Orthop Res. 2003 Jul;21(4):697-707.

Wang X, Manner PA, Horner A, Shum L, Tuan RS, Nuckolls GH. Regulation of MMP-13 expression by RUNX@ and FGF2 in osteoarthritic cartilage. Osteoarthritis Cartilage. Dec; 12(12): 963-73, 2004.

Williams SL, Bachison C, Michelson JD, Manner PA. Component position in 2-incision minimally invasive total hip arthroplasty compared to standard total hip arthroplasty. J Arthroplasty. 2008 Feb;23(2):197-202.



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Hip of the Child

- Pediatric hip disease is major source of adult hip arthritis.
- Arthritis from pediatric hip disease can usually be prevented by proper screening and care of the child's hip.
- The faculty at Seattle's Children's Hospital and Regional Medical Center (CHRMC) are discovering new ways to prevent and manage pediatric hip problems in ways that enables children to grow up with comfortable and functional hip joints.

Untreated pediatric hip disease is a common source of arthritis in individuals as young as twenty years of age. Of the greater than 300,000 hip replacements performed each year in the United States, it has been estimated that up to 50% are performed for hip arthritis resulting from residual pediatric hip disease. The faculty of the Department of Orthopaedics and Sports Medicine at Children's Hospital and Regional Medical Center see hundreds of children each year for hip related disorders. These include:

(1) Developmental dysplasia (dislocation) of the hip (DDH) - an abnormal formation of the hip joint in which the ball on top of the thighbone (femoral head) is not held firmly in the socket (acetabulum) (Figure 1). When the femoral head is not in the acetabulum, neither mature to the proper shape.

(2) Legg-Calvé-Perthes disease - a condition in children in which the ball-shaped head of the thigh bone, referred to as the femoral head, loses its blood supply. As a result, the femoral head collapses. The body absorbs the dead bone cells and replaces them with new bone cells. The new bone cells will eventually reshape the femoral head of the thigh bone, but without proper treatment the shape will not be normal (Figure 2)

(3) Slipped capital femoral epiphysis (SCFE) - a hip problem in which the epiphysis (growing end) of the femur slips off the remainder of the femur (Figure 3). If the slip is not treated, a substantial and permanent hip deformity results.

(4) hip injury, such as a traumatic fracture or dislocation, either of which may lead to adult arthritis

(5) hip inflammation (synovitis), which may be due to a viral or bacterial infection. After such inflammation the anatomy of the hip may be permanently distorted.

Because these conditions of the child's hip can lead to a lifetime of disability, the faculty of the Department of Orthopaedics and Sports Medicine strive to discover better surgical and non-surgical means for preventing the adult consequences of these pediatric hip conditions. Research by our faculty has provided new treatments for DDH, slipped capital femoral epiphysis, and other conditions affecting the hip, such as arthrogryposis and cerebral palsy. Our current efforts are directed at new methods of risk assessment, diagnosis and treatment. We have established registries of children with hip conditions to perform longitudinal studies that will improve our understanding of the effectiveness of the treatments we perform now, and their consequences when these children become adults.

Much of our research centers on the evaluation of the natural history and functional results of treatment, using functional outcome measurements, including the StepWatch(r) activity monitor and the PODCI (pediatric outcomes data collection instrument) questionnaire. This represents a divergence from the past in which orthopedists have based their conclusions largely on the appearance of x-rays, or on ambiguous and inconsistent chart records. These more functionally oriented methods give us information about a child's true activity level, as well as insight into perceptions about their own well-being. We are using these tools to evaluate innovative new treatments for all types of hip disease. We are also applying these modern methods of clinical investigation to improve our understanding of rare genetic disorders and their relationship to hip disease.

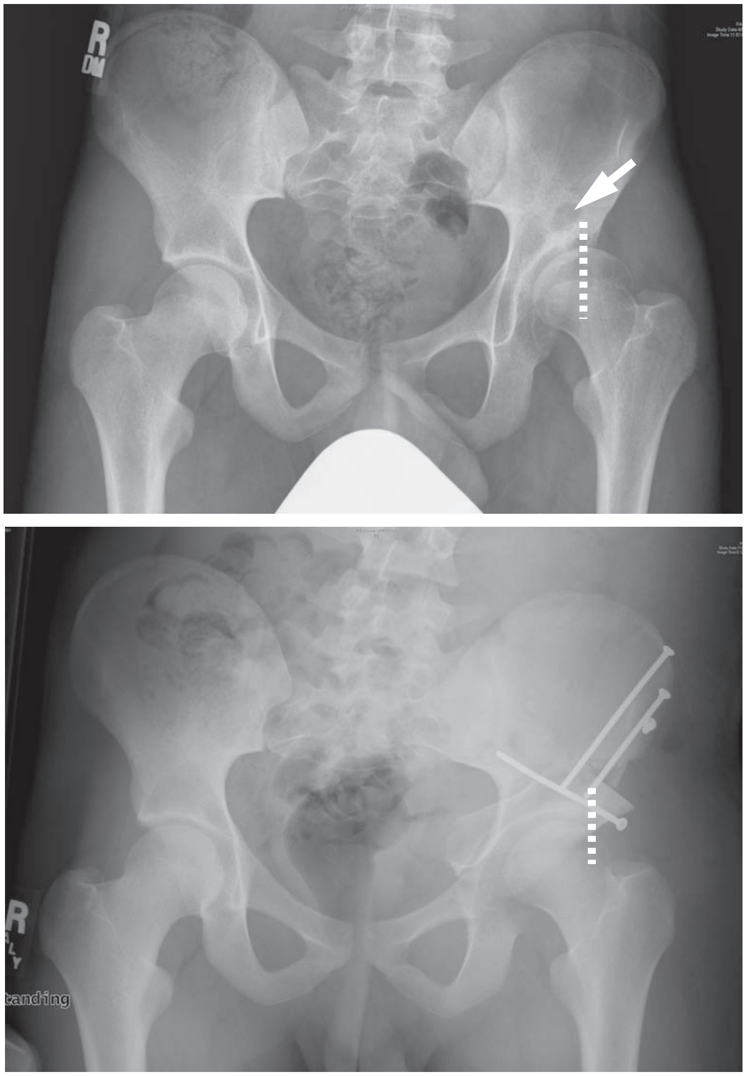

Figure 1: Developmental dysplasia of the hip represents a condition in which the ball and socket joint of the hip is underdeveloped (dashed line shows deficient coverage of head of femur by socket). In this case, a teenage male presented with previously undetected DDH and hip pain. He is already showing signs of early hip arthritis as demonstrated by the cyst highlighted by the arrow. The hip was reconstructed with a "periacetabular osteotomy" providing improved coverage of head of femur, thus prolonging the natural life of this hip and avoiding a hip replacement at a young age.

This information may be utilized to counsel families both prenatally and postnatally about expectations, as well as emerging treatment options, for their child afflicted by these often devastating diseases. Eventually, our understanding of these rare diseases may lead to genetic screening tools that will enable us to anticipate the risk of hip disease in other children.

We have discovered that modern imaging modalities, such as MRI and ultrasound, can improve our ability to characterize the growth and development of the child's hip, noting that conventional x-rays are not as revealing of the anatomy until the bones are formed later in childhood. Such methods are now helping us diagnose hip conditions and make decisions about treatment earlier, when treatment is easier on the child.

We are discovering more effective and less invasive methods for managing hip disorders in ways that prevent life-long consequences. We have learned that the earlier the diagnosis is made, the more likely that the condition can be managed without surgery. We have found that less invasive approaches for aligning the hip can enable the body to heal and redevelop a normal hip structure. Our research has enabled us to establish guidelines or pathways for the evaluation and management of the child's hip. These pathways are being shared with physicians and pediatric medical centers around the world. Because it is the obstetrician and the pediatrician that first sees the newborn child, it is essential that we equip them with the knowledge and tools for accurate evaluation of the hip and for the prompt diagnosis of pediatric hip disease.

We have established an interdisciplinary team of geneticists, pediatricians, radiologists, and orthopaedic surgeons who work together to minimize the impact of pediatric hip disease on the comfort and function of the adult. We will continue to explore new methods for risk assessment, early diagnosis and minimally invasive treatment using traditional means. As our understanding of the genetic underpinnings of pediatric hip disease increases, we will also be utilizing advances in genetics to identify children at risk for hip disease and to anticipate the need for treatment before the problem is clinically evident. Our goal is that every child grow up with a comfortable and functional hip joint.

References

Staheli LT, Chew DE, Elliott JS, Mosca VS. Management of hip dislocations in children with arthrogryposis. J Pediatr Orthop. 1987 Nov-Dec;7(6):681-5.

Staheli LT, Chew DE. Slotted acetabular augmentation in childhood and adolescence. J Pediatr Orthop. 1992 Sep-Oct;12(5):569-80.

Song KM, Halliday S, Reilly C, Keezel W. Gait abnormalities following slipped capital femoral epiphysis. J Pediatr Orthop. 2004 Mar-Apr;24(2):148-55.

Song KM, Bjornson KF, Cappello T, Coleman K Use of the StepWatch activity monitor for characterization of normal activity levels of children. J Pediatr Orthop. 2006 Mar-Apr;26(2):245-9.

Schmale GA, Eilert RE, Chang F, Seidel K. High reoperation rates after early treatment of the subluxating hip in children with spastic cerebral palsy. J Pediatr Orthop. 2006 Sep-Oct;26(5):617-23.

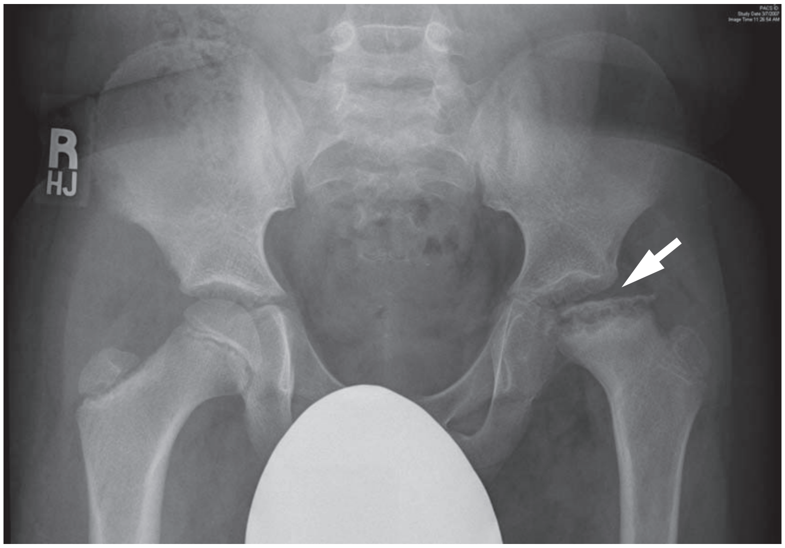

Figure 2: Nearly 100 years after its description, the etiology of Legg-Calvé-Perthes disease remains elusive. Legg-Calvé-Perthes commonly affect boys (in a 5:1 ratio) from age 4-13 years. The hip in this boy shows complete resorption and collapse of the left hip (arrow). Severe Legg-Calvé-Perthes such as this results in permanent deformity, and ultimately, the need for a hip replacement as an adult.

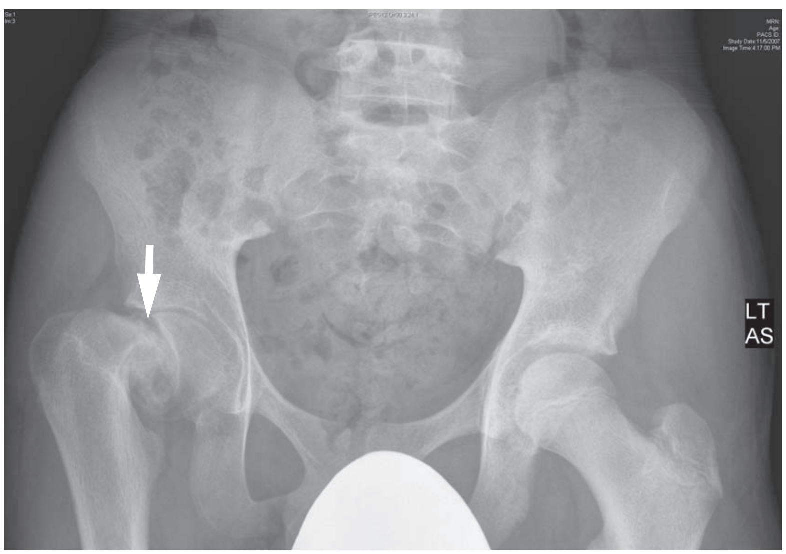

Figure 3: Slipped capital femoral epiphysis (SCFE) occurs when the cartilaginous growth plate at the upper end of the femur (thigh bone) gives way. SCFE most commonly occurs in children who are overweight, but can also occur in children with hormonal imbalances.

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The Role of Muscle Function in Fracture Healing: Good Muscle May Be a Key to Good Fracture Repair

- Impaired muscle function has been associated with delayed bone healing and non union of fractures.
- We have developed a novel *in vivo* model and used it to demonstrate that transient paralysis of the quadriceps muscle leads to delayed fracture healing.
- The development of this novel model provides the first opportunity to directly explore the cellular mechanisms underlying the relation between muscle impairment and compromised bone healing.
- We are working to translate this discovery into clinical practice.

In our early studies, we used an imaging approach (i.e., radiology and high resolution micro CT) to assess whether transient paralysis of a muscle physically adjacent to a fracture would alter normal fracture healing. Under general anesthesia, closed mid-diaphyseal femur fractures (CFx) were created in Sprague-Dawley male rats after inserting a Kirschner wire into the medullary canal to stabilize the fracture. Following radiographic confirmation of transverse, midshaft fractures, 11 rats were given a one time injection of Botox (BT) in their right quadriceps, while the remaining 12 rats received identical volume Saline (SAL) injections at the same sites. All rats were allowed to ambulate freely during the course of the experiment. At sacrifice, the quadriceps muscle groups were isolated from both the CFx and contralateral hind limbs and weighed. Post-necropsy microradiographs were then obtained from all fractured femurs and were evaluated for radiologic evidence of bone union. Subsequently, micro CT scans were obtained for all right and left femurs. For each animal, the callus (healing) bone volume was determined by subtracting the contralateral left femur bone volume within the scanned region with that determined for the experimental right femur.

Compared to the contralateral muscle group, quadriceps weight in the CFx + SAL controls was reduced by -14% and -5% at 2 and 4 wk post-fracture, respectively, although neither of these decreases reached statistical significance (Figure 1). By 6 wk post-fracture, the quadriceps weight in the CFx + SAL group had recovered to normal. In sharp contrast, intramuscular injection of BT induced decreases in quadriceps muscle mass of -39%, -60%, and -50% at 2, 4, and 6 wks post-fracture, respectively. Radiographic examination indicated an absence of bone union in 7 of 8 animals in the CFx + BT group at 4 or 6 wk, while all 8 animals in the CFx + SAL demonstrated evidence of bone union group across the fracture gap at 4 or 6 wk post-fracture (Figure 2). Visual examination of all micro CT images suggested that transient quadriceps paralysis led to diminished callus formation compared to animals with normal quadriceps function (Figure 3). The visual interpretation was confirmed quantitatively at the 2 wk time point; callus volume was nearly identical for CFx + BT rats compared to CFx + SAL rats. However, at 4 and 6 wks, fracture callus volume in CFx + BT group was reduced -44% compared to CFx + SAL rats (Figure 4).

In summary, we discovered that loss of quadriceps function severely inhibited normal fracture healing in the model and compromised both morphologic (i.e., absence of radiographic union and decreased callus volume) and structural aspects of fracture healing. Interestingly, although the CFx + BT group appeared to have initiated a normal healing response via rapid periosteal bone formation distal and proximal to the fracture site, progression toward bone union at the fracture site was clearly impaired. Given that the proximal and distal Botox injections corresponded with sites of normal periosteal bone response, it seems unlikely that the Botox itself was responsible for the impaired fracture healing. In total, these preliminary time course data

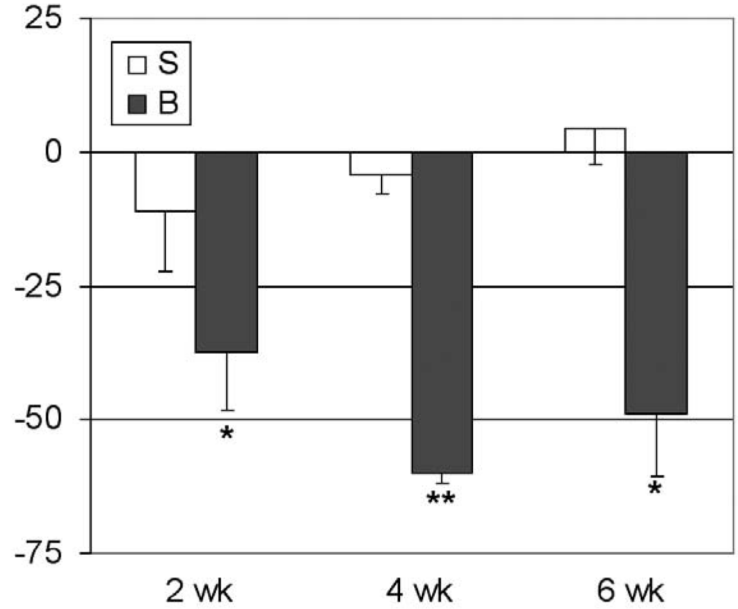

Figure 1: Quadriceps paralysis significantly decreases muscle wet weight. Muscle weight in Saline (S) injected quadriceps (vs. contralateral control), was decreased by -14.08% at 2 wk but the difference was not statistically significant. Subsequent time points indicated that muscle weight was recovering to normal levels in S rats. In contrast, quadriceps weight in the Botox (B) injected rats was significantly decreased at all time points. *P <0.05; and **P < 0.001 vs. contralateral.

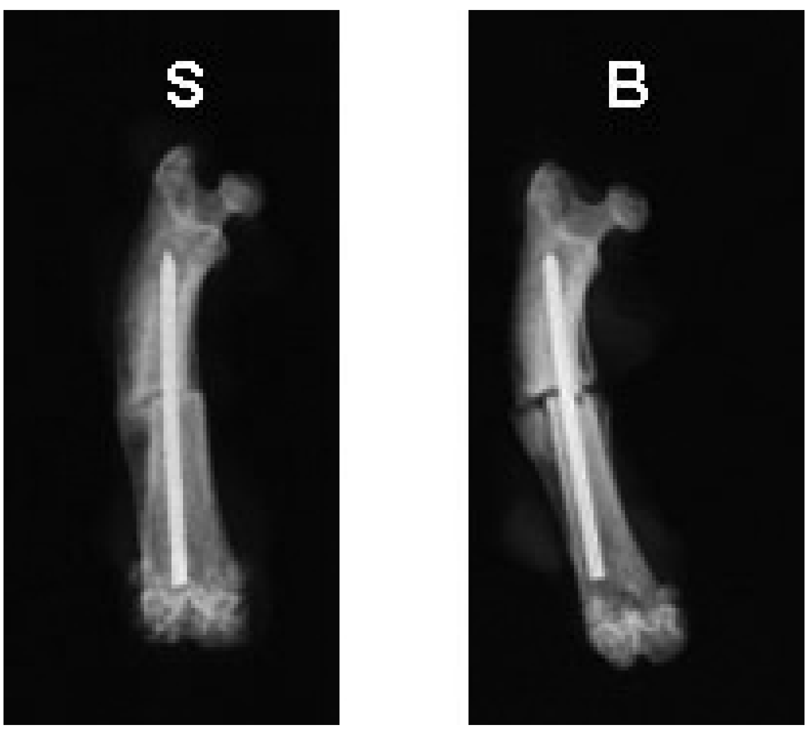

Figure 2: Radiographic evidence of delayed healing due to transient muscle paralysis is evident within 4 wk. Radiographs from representative Saline (S) and Botox (B) rats 4 wk following fracture. The Saline treated rats demonstrated clear progression toward bridging of the fracture site. In contrast, rats in which transient quadriceps paralysis was superimposed upon closed femur fracture demonstrated callus formation proximal and distal to the fracture site, but a lack of osteogenic bone bridging across the fracture gap.

strongly suggest that transient muscle paralysis is directly responsible for the observed effects on callus structure and radiographic nonunion.

Conclusion

In terms of health care significance, medical procedures associated with orthopaedic trauma are the second most expensive medical problem in the U.S., imposing an annual burden to the health care system of over $56 billion per year, of which 50% is attributed to the treatment of broken bones. The negative impact recalcitrant nonunions place on patient quality of life is amplified when repeated operative interventions are required to treat nonunions with loss of productivity and co-morbidities. As the population ages, fracture incidence will increase, which will further magnify the impact of these pathologies on the U.S. health care system and affected patient populations. An improved understanding of the pathways that influence the development of nonunions is an essential prerequisite for the prediction, management and/or treatment of nonunion pathologies. By coupling transient muscle paralysis with a well characterized model of fracture repair, we hope to decipher perturbations in the molecular pathways that lead to nonunion pathology in our novel model.

Our goal is to translate this laboratory work to the benefit of our patients, by finding ways to optimize muscle function in our patients with fractures.

Acknowledgements

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References

Gross, T.S., Poliachik, S.L.., Ausk, B.J., Nork, S.E., Bain, S.D. (2007). A novel model of impaired fracture healing. J Bone Min Res, 22(S1): S162.

Gross, T.S., Ruhlman, S., Ausk, B.J., Becker, B.A., Nork, S.E., Bain, S.D. (2008) Muscle function is critical for successful fracture healing. 54th Trans Orthop Res Soc, San Francisco, CA.

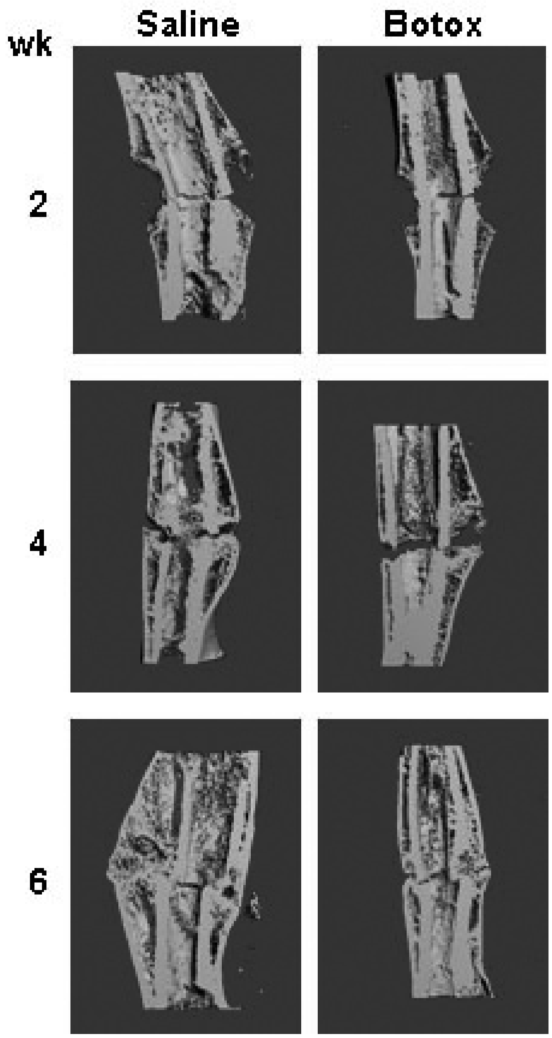

Figure 3: MicroCT images indicate that transient muscle paralysis delays the normal time course of fracture healing. Frontal plane sections of composite micro-CT imaging of representative rats injected with Saline or Botox and sacrificed at 2, 4, or 6 wk post fracture. As expected, Saline rats demonstrated a clear progression toward healing within 6 wk. When quadriceps paralysis was superimposed upon the model, early periosteal bone formation proximal and distal from the diaphyseal fracture site appeared minimally altered compared to Saline treated rats at 2 wk. However, at 4 wk and 6 wk, callus volumes were clearly reduced and the progression toward bridging of the fracture gap was halted.

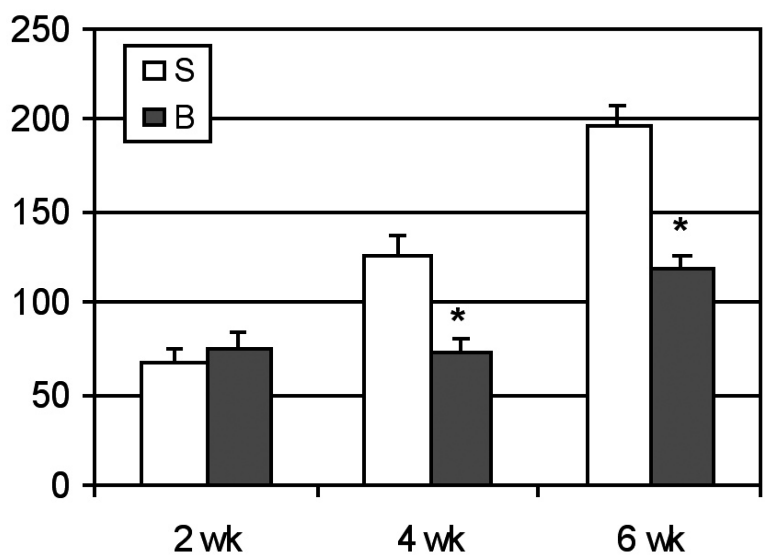

Figure 4: Muscle paralysis interferes with the development of callus morphology required for successful facture healing. Callus volume was significantly reduced at 4 and 6 wk in the Botox treated groups (B) compared to the Saline treated groups (S); *p < 0.003 and 0.05 for the 4 and 6 wk time points, respectively.

Bain, S.D., Ruhlman, S., Ausk, B.J., Becker, B.A., Nork, S.E., Gross, T.S. (in preparation). J Orthop Res.

Bonnarens F, Einhorn TA 1984 Production of a standard closed fracture in laboratory animal bone. J Orthop Res 2(1):97-101.

Bishop GB, Einhorn TA 2007 Current and future clinical applications of bone morphogenetic proteins in orthopaedic trauma surgery. Int Orthop 31(6):721-7.

Praemer A, Furner, S., Rice, D.P. 1999 Musculoskeletal Conditions in the United States. American Academy of Orthopaedic Surgeons, Rosemount, IL, pp 182.

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Femur Fractures: A Common Injury with Life Threatening Consequences

- Fractures of the shaft of the femur (thigh bone) typically occur as the result of high-energy trauma in young patients. The common mechanisms of injury are motor vehicle accidents, motorcycle crashes, and falls from height.
- Femur fractures are often associated with potentially life-threatening injuries to the chest, abdomen, head or pelvis.
- When femur fractures were treated by bed rest and traction, they were complicated by pneumonia, bedsores, and bowel and bladder problems.
- Treatment by traction often resulted in failure to heal (non-union), and poor restoration of the normal alignment (shortening, angulation and rotation).
- Our faculty adapted a German technique for treatment of fractures of the femoral shaft that consisted of driving a metal spike (intramedullary nail) down the inside of the bone. We further discovered that intramedullary nailing could be accomplished with only a small incision cosmetically satisfactory incision. This method of immediate stabilization of the fracture enables the patient to get out of bed, avoiding the complications of bed rest. This method dramatically reduces the risk of non-union and malunion.
- Our faculty have taught our approach to intramedullary nailing to surgeons around the world, resulting in a dramatic drop in the complications from this serious injury.
- We are striving to discover ways to further advance the care of femur fractures by developing approaches for the management of more complex injuries, such as those with many fragments, those that extend into the joint, and those that are associated with major skin and soft tissue damage.
- Our faculty, residents and fellow alumni have become contributors in the international community of academic surgeons pursuing improved management of the injured patient, including the Orthopaedic Trauma Association, the AO (Arbeitsgemeinschaft für Osteosynthesefragen) Foundation, the Orthopaedic Research Society, the American Academy of Orthopaedic Surgeons, and the American Orthopaedic Association.

The femur is the largest bone in the body; a huge amount of energy is required to break it. Femur fractures are associated with major loss of blood (up to half of the patient's total blood volume) in the thigh. Prior to World War II, the treatment of femur fractures was largely non-operative: bed rest, traction and casting. This 'conservative' treatment resulted in high rates of patient death and complications from bed rest. If the patient survived, limb shortening and angulation was often observed.

The origin of intramedullary nails is credited to Kuntscher who began placing a steel nail (a long metal post) down the canal in the center of the femur. This allowed for the femur to be realigned such that healing could proceed uneventfully. More importantly, this allowed for patients to be mobilized from bed during the healing process.

The introduction and use of intramedullary nailing techniques in the United States have largely been credited to our faculty members Ted Hansen, D.K. Clawson and Bob Winquist who reported their results beginning in 1971. Their pioneering work resulted in multiple publications on closed intramedullary nailing in complex injury patterns, that is, insertion of the nail without opening the fracture itself. This closed method of nailing

Figure 1,2: This 36 year-old male sustained multiple injuries after a motorcycle crash including a collapsed lung (pneuomothorax), abdominal trauma (a splenic injury), and this left femur fracture. The front (AP) and side (lateral) radiographic views of the femur demonstrate the segmental fracture with breaks just above the knee and in the middle portion of the thigh. A traction pin (small wire) with a traction bow was placed just above the knee and used to temporarily stabilize the femur during the ongoing resuscitation of the patient.

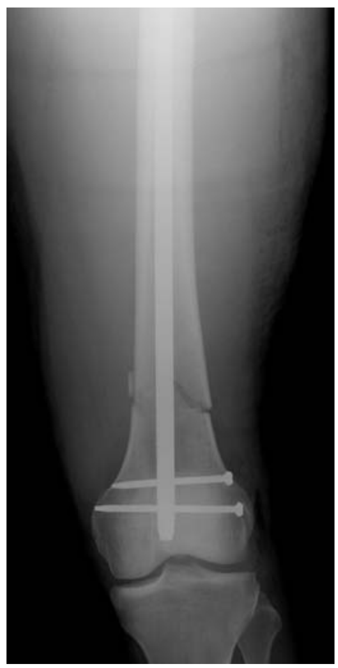

Figure 3,4: The patient was taken to the operating room urgently for stabilization of his multiple breaks in the femur. Through a small surgical incision (2 centimeters in length) placed just above the hip joint, reamers were passed down the canal of the femur to allow for placement of the intramedullary nail. The nail spanned both fractures and maintains the proper length, alignment, and rotation of the femur while healing progresses. Placement of the interlocked nail allows the patient to be immediately mobilized from bed and allows weight bearing on the left leg as comfort allows. (Case compliments of David P. Barei, MD, Harborview Medical Center, University of Washington.)

was important in that the fracture site was not disturbed; a small incision just above the hip was all that was required to place a nail down the femur as the fracture was reduced using external manipulation. This ultimately resulted in the largest series of femur fractures treated with this technique to date. In their review of 520 patients, they found a significant reduction in the commonly observed complications, and outlined the technique in detail.

Progress

While the technique for simple fractures of the femoral shaft is now well established, we continue to explore advanced methods for managing more complex injuries.

One innovation is the use of 'locking' nails that have screws that pass through the bone and the nail above and below the fracture. These locking nails provide additional stability for fractures with many pieces (comminuted fractures).

Fractures involving the joints at the knee and hip ends of the femur present special challenges because of the need to restore the contour and support of the joint. We are now using special nails and combinations of nails with screws and plates to restore the anatomy of these complex injuries.

Patients with bilateral femur fractures are at particularly high risk for early complications including death. This is largely due to number of injuries, the associated blood loss, and the impact of these injuries on other organ systems. Clinical research at our institution demonstrated the consequence of bilateral femur fractures. In a review of over 700 patients with femur fractures treated with reamed intramedullary nails, the death rate was four times as high for bilateral femur fractures as for unilateral femur fractures. This discovery pointed out the need for expert management of blood loss, respiration and associated injuries when both femurs are broken.

Conclusion

Although reamed, interlocked, intramedullary nailing for femur fractures is one of the most successful and predictable operations in orthopaedic traumatology, research into a number of areas can further improve patient outcomes. A further understanding of the injury itself and the associated response of the patient to injury will help to determine the optimal timing and method of definitive stabilization. Further, although fracture healing is observed in approximately 95% of patients, we are directing research at the causes of delayed union and nonunion to hasten recovery and patient mobilization. The traumatology team at Harborview* are dedicated to continuing to make major contributions to the management of femur fractures. We are proud of our newly endowed professorship for Fracture Fixation Biology and hope to grow this program to help solve the substantial remaining challenges in the care of the severely injured individual.

*Steve Bain, David Barei, Daphne Beingessner, Carlo Bellabarba, Steve Benirschke, Rick Bransford, Jens Chapman, Bob Dunbar, Ted Gross, Ted Hansen, Brad Henley, Jim Kreig, Sean Nork, Chip Routt, Bruce Sangeorzan, Doug Smith, Sundar Srinivasan, Lisa Taitsman, Allan Tencer

References

Clawson, DK; Smith, RF; and Hansen, ST: Closed intramedullary nailing of the femur. J Bone Joint Surg Am, 53(4): 681-92, 1971.

Winquist, RA, and Hansen, ST: Segmental fractures of the femur treated by closed intramedullary nailing. J Bone Joint Surg Am, 60(7): 934-9, 1978.

Hansen, ST, and Winquist, RA: Closed intramedullary nailing of the femur. Kuntscher technique with reaming. Clin Orthop, (138): 56-61, 1979.

Winquist, RA, and Hansen, ST, Jr.: Comminuted fractures of the femoral shaft treated by intramedullary nailing. Othop Clin North Am, 11: 633-47, 1980.

Winquist, RA; Hansen, ST, Jr.; and Clawson, DK: Closed intramedullary nailing of femoral fractures. A report of five hundred and twenty cases. J Bone Joint Surg [Am], 66(4): 529-39, 1984.

Swiontkowski, MF; Hansen, ST, Jr.; and Kellam, J: Ipsilateral fractures of the femoral neck and shaft. A treatment protocol. J Bone Joint Surg Am, 66(2): 260-8, 1984.

Webb, LX; Winquist, RA; and Hansen, ST: Intramedullary nailing and reaming for delayed union or nonunion of the femoral shaft. A report of 105 consecutive cases. Clin Orthop, (212): 133-41, 1986.

Lhowe, DW, and Hansen, ST: Immediate nailing of open fractures of the femoral shaft. J Bone Joint Surg [Am], 70(6): 812-20, 1988.

Barei, DP; Schildhauer, TA; and Nork, SE: Noncontiguous fractures of the femoral neck, femoral shaft, and distal femur. J Trauma, 55(1): 80-6, 2003.

Nork, S. E.; Agel, J.; Russell, G. V.; Mills, W. J.; Holt, S.; and Routt, M. L., Jr.: Mortality after reamed intramedullary nailing of bilateral femur fractures. Clin Orthop, 415(415): 272-8, 2003.

Nork, SE: Fractures of the Shaft of the Femur. In Bucholz, RW, Heckman, JD, Court-Brown, C, eds. Rockwood and Green's Fractures in Adults, 6th Edition. Philadelphia, Lippincott Williams & Wilkins, Chapter 47, pp. 1845-1914, 2006.

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Knee Arthritis and Knee Replacement Surgery

- Knee arthritis refers to the loss of the normal cartilage between the femur (thigh bone) and the tibia (leg bone). It is a common cause of difficulty with activities of daily living, such as walking, dressing, and getting in and out of a car as well as the inability to enjoy recreational activities, such as golf, cycling, and hiking.
- Most patients with knee arthritis can be managed with non-operative interventions, such as activity modifications, pills, or injections. When the knee arthritis becomes more severe, some patients elect to have knee replacement surgery. A successful knee joint replacement can restore comfort and function.
- Newer "minimally-invasive" approaches to the knee appear to permit faster and less-painful recovery following total knee replacement surgery.
- We are working to discover ways to prevent functional limitations for patients with arthritis of the knee, both by minimizing the progression of the condition as well as by speeding the recovery from reconstructive surgery.

Some of the more important early contributions made by UW orthopaedic faculty members were on the subject of anatomic topics that helped to form the core understanding that surgeons have about knees - including Victor H. Frankel, UW Orthopaedics' Chairman between 1976 and 1981 publishing on the relationship between meniscectomy and post-meniscectomy arthritis in '77, and John M. Clark, reporting on the mechanism by which popliteal ("Baker's") cysts fill with fluid in '75 -are two discoveries so deeply ingrained as to be part of the orthopaedic vernacular. Dr. Clark (who has a PhD in anatomy, in addition to his MD degree) made many other substantial contributions to our understanding of the structure of the human subchondral plate and its relationship to degenerative joint lesions, the ultrastructural response of cartilage collagen under static and cyclic loading, and surface topography.

More recently, one of UW Orthopaedics' newest faculty members, Dr. Paul A. Manner, has contributed to our molecular understanding of the progression of cartilage from the normal to the arthritic state in work done collaboratively with the National Institutes of Health. In terms of the basic science of cartilage biology, intimately relevant to the broader topic of knee arthritis, the greatest contributions have come out of the laboratory of Dr. David Eyre, Burgess Professor and Director, Orthopaedic Research Laboratories in the UW Department of Orthopaedics and Sports Medicine. Dr. Eyre's seminal work has focused on better defining the collagen structure of joint surface (articular) cartilage, and looking for collagen-related breakdown products as biomarkers of joint disease.

On the clinical side, former faculty member Dr. William Lanzer evaluated the relationship between obesity and patient-centered outcomes following total knee arthroplasty. While the literature on that topic certainly is controversial, Dr. Lanzer's work concluded that obesity did not result in decreased satisfaction or ultimate outcome following joint replacement surgery, and has been quoted along with studies of other investigators in substantiation of the decision to offer this procedure to a population of patients that stands to benefit from the intervention.

Not all interventions that surgeons perform are operative; prior to my arrival here, and in collaboration with another investigator who subsequently joined the UW Orthopaedics faculty, Dr. Winston Warme, I investigated the use of Synvisc (Hylan G-F 20, one of several FDA-approved "viscosupplement" products that were designed to palliate the pain associated with knee arthritis). In a randomized controlled trial against corticosteroid ("cortisone") injections, we learned that this expensive product, while effective for temporary relief of symptoms, is no better than a

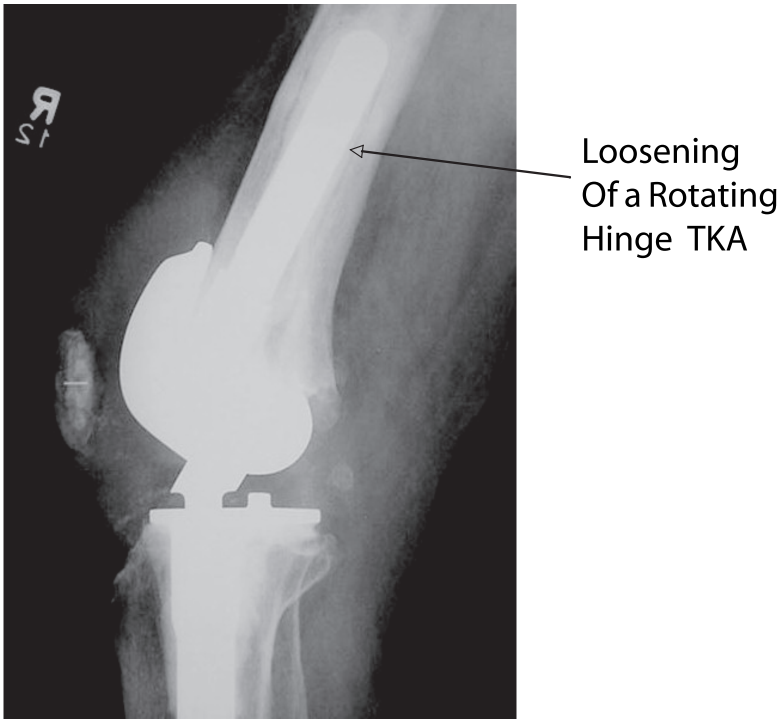

Figure 1: Research done at the University of Washington has focused on why some knee implants for tumor procedures fail prematurely. This x-ray shows loosening of the femoral (thigh bone) component; the black lines around the stem of the device indicate failure of the device's point of attachment to bone.

simple cortisone shot; this report was the first study on the subject that was not funded or supported by the manufacturer of the product in question, and was the first study that called into question what then was a trendy and popular approach. A subsequent study we performed identified that using repeated courses with this product increased the risk of painful reactions in the knee over eightfold, an observation that had not been published before, but which has since been accepted as accurate. Along with Dr. Warme and other colleagues, we've made important contributions to the understanding of the physiological stress cortisol response observed following minor and major knee surgery.

And not all important publications deal with primary "discoveries"; some of the more influential publications involve synthesis of known literature, dissemination and recommended practice guidelines. In partnership with Dr. Hannah Morgan, a past fellow in adult reconstruction and subsequently a teaching associate on the UW Orthopaedics faculty, we outlined an algorithmic approach to surgical decision-making regarding the use of constraint in implant selection for straightforward and complex knee replacement surgery. This report was published in the leading review journal of orthopaedic surgery and is considered an acceptable "board standard" approach to the topic. Along with Dr. Morgan and also Dr. Ernest "Chappie" Conrad, the UW Hip and Knee team partnered with the internationally known UW Orthopaedic Sarcoma Service to publish observations on factors leading to success or failure of tumor megaprosthetic replacement about the knee (Figure 1).

Some of the most important scientific contributions do not involve the sharing of success stories. Some of the most influential publications in the surgical literature are so-called "failure series." Along with colleagues at Rush-Presbyterian in Chicago, I reported on the clinical results of what then was the recommended technique for reconstructing the deficient extensor mechanism after total knee replacement. This technique failed utterly, but reporting on this failure led to subsequent evaluations of other approaches that have come much closer to solving this difficult problem.

While there are many areas of active inquiry in the subspecialty of adult knee reconstruction, perhaps the most exciting one - and the one that has had the most dramatic impact upon the clinical practice of knee arthroplasty surgery at UW and elsewhere that has arisen in the last decade or two - is the topic of "Minimally-Invasive" knee replacement approaches. These approaches offer the potential for faster, less-painful recuperation

Minimally-Invasive vs Traditional Total Knee Replacement

	MIS TKA	Traditional TKA
Pain	+	++++
Hosp Stay	2-3 days	3-4 days
Inpt Rehab	No	3 days
Walker	1 week	2-3 weeks
Cane	1 week	2-3 weeks
Phys Therapy	4 weeks	6-8 weeks

Figure 2: Research performed at the University of Washington and elsewhere has substantiated a faster recovery in patients who have undergone minimally-invasive (quadriceps sparing) total knee replacement compared with patients who have undergone traditional-approach total knee replacement.

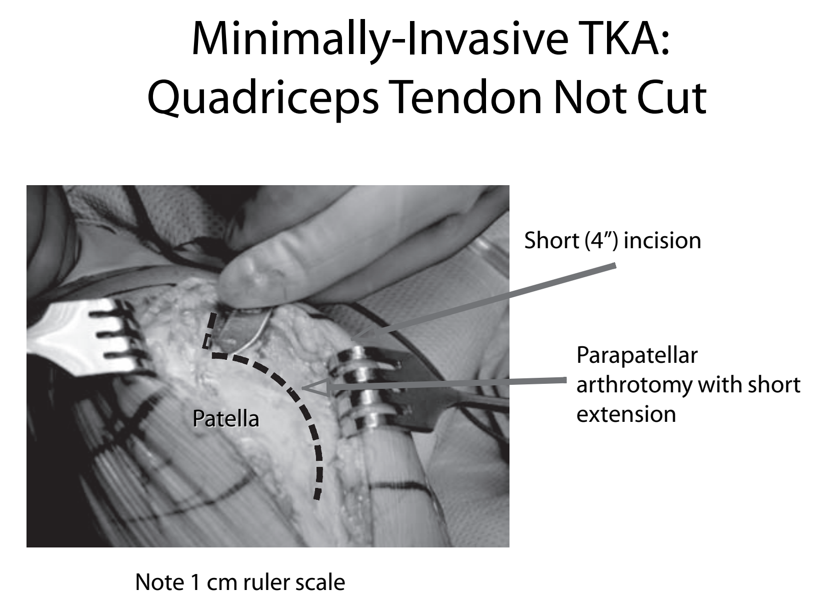

Figure 3: Some key advantages of the minimally-invasive (quadriceps-sparing) approach to knee replacement are shown here. In particular, the incision and surgical dissection are less extensive, and the quadriceps tendon is not cut.

following this major operation, but important questions have been raised about it: Can the implants be inserted with comparable accuracy to traditional "open" approaches to the knee? Does it result in wound complications? How hard are they to learn, and what compromises occur during the learning curve? Is the "hype" true - do people really recover more quickly? In a recent, controlled study, comparing the quadriceps-sparing approach to a traditional medial-parapatellar approach (the previous standard at UW and elsewhere) my colleagues at UW Orthopaedics and I addressed these questions, and concluded that indeed the recovery is faster with the quadriceps-sparing approach, but that there was a learning curve of 25-50 procedures, which might put the procedure out of reach for the occasional or low-volume joint replacement surgeon who might do 5-10 procedures per year. However, for the high-volume arthroplasty specialist, the technique offers the potential for significant benefits to accrue to the patients who have their knee implants inserted in this manner, including a shorter hospital stay (and essentially the elimination of inpatient rehabilitation), fewer days on crutches or a cane, and less pain during the recuperative period (Figure 2). The reasons for this may have to do with specific elements of the surgical approach to the knee, which, taken together, may make this approach gentler on the soft tissues around the knee joint (Figure 3, Figure 4). In progress now, with the collaboration of Dr. Manner as well as with our partners in the UW Department of Medical Education and Biomedical Informatics, are investigations on the medical-economic benefits of this surgical approach.

One of the most gratifying elements of being engaged in the practice of knee reconstruction is that it is seldom possible to see what the next exciting discovery will look like; that said, if history is any guide, it's a fair guess that UW Orthopaedics will have a hand in contributing to it.

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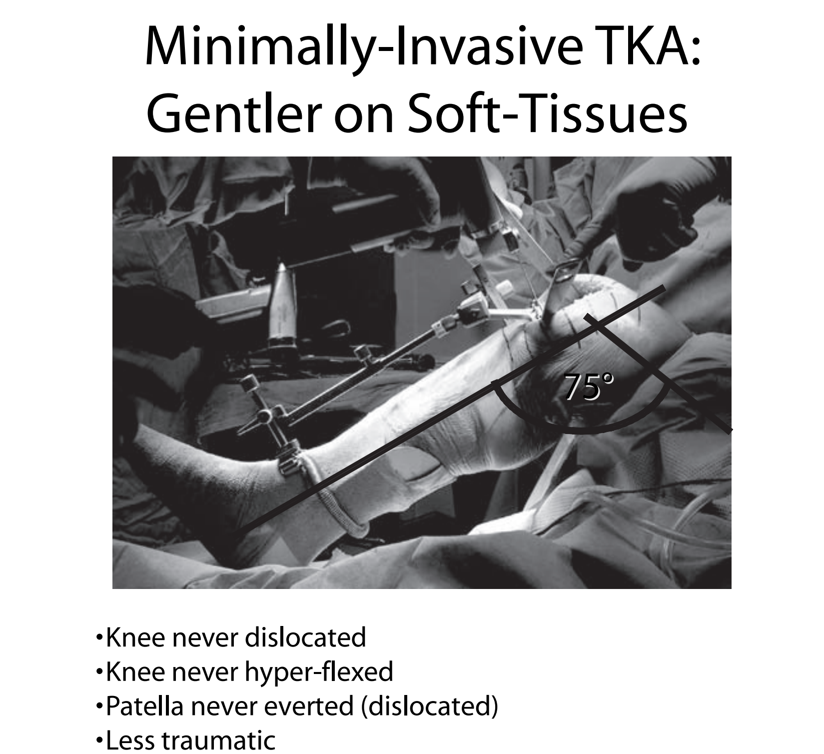

Figure 4: Other advantages of minimally-invasive (quadriceps-sparing) knee replacement are summarized in this photograph. The fact that the knee is not dislocated or put into severe flexion, and the fact that the kneecap is not everted (dislocated) all may contribute to the observation that this approach may be less traumatic to the soft tissues around the knee joint; this may result in the observed shorter recovery period.

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References

Convery FR, Clawson DK. The rheumatoid knee. Am Fam Physician. Aug; 4(2):52-9, 1971.

Clark JM, Norman AG, Kaab MJ, Notzli HP. The surface contour of articular cartilage in an intact, loaded joint. J Anat. Jul; 195 (pt 1): 45-56, 1999.

Clark JM, Huber JD. The structure of the human subchondral plate. J Bone Joint Surg Br. Sep; 72(5): 866-73, 1990.

Kaab MJ, Ito K, Clark JM, Notzli HP. Deformation of articular cartilage collagen structure under static and cyclic loading. J Orthop Res. Nov; 16(6): 743-51, 1998.

Wang X, Manner PA, Horner A, Shum L, Tuan RS, Nuckolls GH. Regulation of MMP-13 expression by RUNX@ and FGF2 in osteoarthritic cartilage. Osteoarthritis Cartilage. Dec; 12(12): 963-73, 2004.

Lohmander LS, Atley LM, Pietka TA, Eyre DR. The release of cross-linked peptides from type II collagen into human synovial fluid is increased soon after joint injury and in osteoarthritis. Arthritis Rheum. Nov; 48(11): 3130-9, 2003.

Eyre DR. Collagens and cartilage matrix homeostasis. Clin Orthop Relat Res. Oct; (427 Suppl): S118-22, 2004.

Eyre DR, Pietka T, Weis MA, Wu JJ. Covalent cross-linking of the NC1 domain of collagen type IX to collagen type II in cartilage. J Biol Chem. Jan 23; 279(4):2568-74, 2004.

King J, Stamper DL, Schaad DC, Leopold SS. Minimally-invasive total knee arthroplasty versus medial parapatellar total knee arthroplasty: new approach appears to facilitate recovery, but learning curve is lengthy. J Bone Joint Surg Am. 89:1794-1503, 2007.

Elmer RM, Moskowitz RW, Frankel VH. Meniscal regeneration and post-meniscectomy degenerative joint disease. Clin Orthop Relat Res. May; (124): 304-310, 1977.

Clark JM. Arthrography diagnosis of synovial cysts of the knee. Radiology. May; 115(2): 480-1, 1975.

Stickles B, Phillips L, Brox WT, Owens B, Lanzer WL. Defining the relationship between obesity and total joint arthroplasty. Obes Res. Mar; 9(3): 219-23, 2001.

Leopold SS, Redd BB, Warme WJ, Wehrle PA, Pettis PD, Shott S. Corticosteroid versus Synvisc (Hylan G-F 20) injections for knee osteoarthritis: a prospective, randomized trial. J Bone Joint Surg Am. 85-A: 1197-1203, 2003.

• Comment in: J Bone Joint Surg Am. 86-A: 874; author reply 874. J Bone Joint Surg Am. 86-A: 874; author reply 874-875.

Leopold SS, Warme WJ, Pettis, PD, Shott S. Increased frequency of acute local reaction to intra-articular Hylan G-F 20 (Synvisc) in patients receiving more than one course of treatment. J Bone Joint Surg Am. 84-A: 1619-1623, 2002.

• Comment in: J Bone Joint Surg Am. 85-A: 1618-1619; author reply 1619-1620. J Bone Joint Surg Am. 85-A: 1620; author reply 1620-1621. J Bone Joint Surg Am. 85-A: 2050; author reply 2050-2051.

Leopold SS, Casnellie MT, Warme, WJ, Dougherty PJ, Wingo ST, Shott S. Endogenous cortisol production in response to knee arthroscopy and total knee arthroplasty. J Bone Joint Surg 85-A: 2163-2167, 2003.

Morgan HD, Battista V, Leopold SS. Constraint in primary total knee arthroplasty: how much is enough? J Am Acad Orthop Surg. 13: 515-24, 2005.

Morgan HD, Cizik AM, Leopold SS, Hawkins DS, Conrad EU. Survival of tumor megaprostheses replacements about the knee. Clin Orthop Relat Res. 450: 39-45, 2006.

Leopold SS, Greidanus N, Paprosky WG, Berger RA, Rosenberg AG. High failure rate of allograft extensor mechanism reconstructions following total knee arthroplasty. J Bone Joint Surg Am. 81-A, 1574-1579, 1999.

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What To Do When a Good Knee Joint Suddenly Goes Bad: Tibial Plateau Fractures

- Healthy knees are something that we all take for granted, but getting a knee injury can often take us right off the ice and straight into the penalty box!
- The knee is made up of three bones, the distal femur (or bottom end of the thigh bone), the proximal tibia (or the top end of the shin bone), and the patella (more commonly called the knee cap).
- Tremendously strong ligaments hold the relationships between the distal femur and the proximal tibia, yet allow the knee to bend and straighten as a smooth, stable and comfortable hinge.
- Large fibrocartilagenous structures called menisci are located between the distal femur and proximal tibia. They help maintain joint stability and transmit loads between the two bones.
- Strong muscles and tendons cross the knee joint to give us power when doing activities as simple as getting out of a chair, or as complex as running through the defensive line toward the end zone.
- Knee injuries can range from a simple sprain of the ligaments as may occur after a small trip and fall, to complete ligament ruptures that are commonly seen during sporting events, to complex fractures that occur after falls from a height, high-energy sports activities like skiing, or car accidents.
- One of the many challenging fracture patterns that occurs around the knee involves the upper end of the shinbone know as the tibial plateau.
- Orthopaedic trauma surgeons at the University of Washington are discovering new and less invasive methods for reassembling and stabilizing knees with these complex fractures so that the patient can regain his or her lost comfort and function.

Fractures that involve the tibial plateau frequently demonstrate disruption of the smooth cartilage joint surface, misalignment of the leg, associated injuries to the surrounding ligaments and tendons, and often have serious injury to the skin such as marked swelling and/or blisters (Figures 1 and 2). The most challenging of these injuries is the bicondylar tibial plateau fracture, where the fracture has literally separated the smooth joint area from the rest of the lower leg. Unless the joint surface can be surgically restored and held securely in place, deformity and arthritis will result from these injures. Because of the potentially devastating outcomes of this injury, the surgeons of the University of Washington's Harborview Medical Center have been committed to optimizing their surgical management.

Historically, treating a bicondylar tibial plateau fracture with a single conventional plate/screw device demonstrated a high failure rate as the device was frequently unable to maintain the appropriate alignment of the leg until the fractures had healed. Alignment was improved when we began placing two plates to support both the inside (medial) and outside (lateral) aspects of the proximal tibia. Unfortunately, medial and lateral plating, particularly through a single anterior incision, was associated with a high rate of wound complications and deep infections. To decrease the incidence of these complications we have explored many different treatment methods, including isolated lateral plating with medial external fixation, hybrid external fixation, tension-wire fixation, and

Figure 1: (A) Plain anteroposterior (front view) radiograph of an uninjured left knee. (B) Plain anterioposterior (front view) radiograph of the left knee of a 55 year-old man involved in a motorcycle collision. This patient has sustained a significant fracture of the proximal tibia. Note the disruption of smooth joint surface, the separation of the tibial shaft from the joint fragments, and the malalignment.

Figure 2: Clinical photograph of the legs of a 25 year-old man that was involved in a car accident earlier in the day. He left leg has sustained a bicondylar tibial plateau fracture. Note that his left leg already demonstrates substantial swelling, bruising, and has formed blisters. The degree of soft tissue injury indicates that acute surgical intervention at this time may not be wise. His right knee has also sustained a deep laceration to the medial aspect.

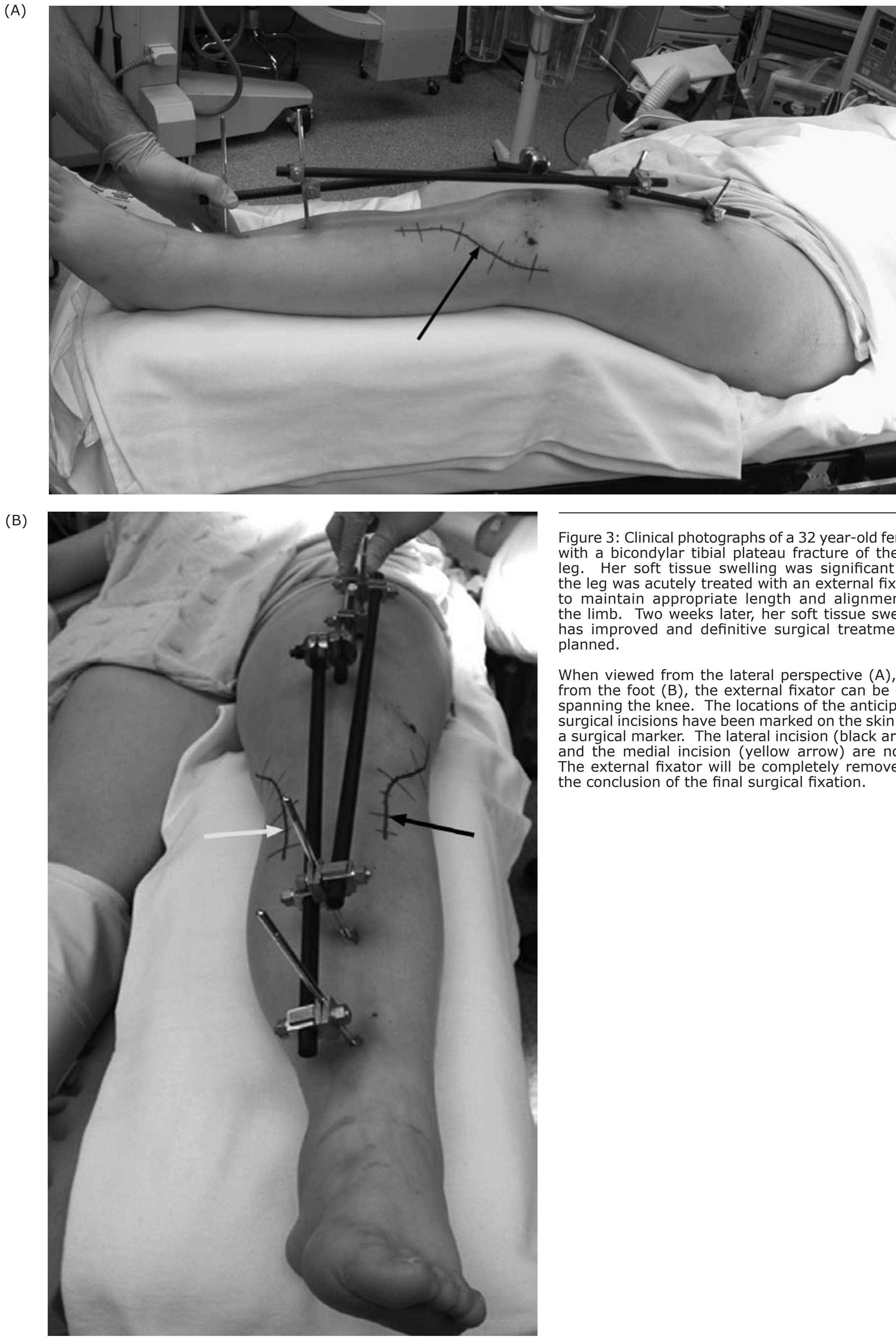


Figure 3: Clinical photographs of a 32 year-old female with a bicondylar tibial plateau fracture of the left leg. Her soft tissue swelling was significant and the leg was acutely treated with an external fixatior to maintain appropriate length and alignment of the limb. Two weeks later, her soft tissue swelling has improved and definitive surgical treatment is planned.

When viewed from the lateral perspective (A), and from the foot (B), the external fixator can be seen spanning the knee. The locations of the anticipated surgical incisions have been marked on the skin with a surgical marker. The lateral incision (black arrow) and the medial incision (yellow arrow) are noted. The external fixator will be completely removed at the conclusion of the final surgical fixation.

other percutaneous techniques. Unfortunately, complications still existed with these techniques and significant long-term problems were identified because important cartilage fragments that are required for good joint function could not be secured in their anatomically correct positions.

We next pioneered the use of open dual plating using a two-incision approach to manage these complex fractures. In the largest series reported to date, we demonstrated a dramatic decrease in wound complication rates using the two-incision technique. Furthermore, we have been able to demonstrate that achieving a good result is partly dependent on getting all the components of the fracture well-aligned, particularly the joint surface. We showed the importance of the handling the significant soft tissue injury that usually accompanies these fractures. Now, patients with these serious injuries are often initially treated with a device called an external fixator (Figure 3). This is a simple series of pins and bars that are placed shortly after the time of injury to help to realign the leg, provide comfort, and let the soft tissue swelling resolve. Once the swelling has decreased, the formal surgery can be carried out. Improvements in technology have allowed us to treat some of these fractures with special plates and screws that provide better hold of the fracture, and can be inserted through smaller incisions. Our faculty and graduates of our residency and fellowship programs at the University of Washington and Harborview Medical Center have closely examined results obtained with these implants and have found a low incidence of infection, excellent healing rates, and excellent alignment. While these advances in technology have been tremendous in improving the management of these injuries, they may not be appropriate for all situations. In particular, we have been able to identify an important, common component of these fractures, the posteriormedial fragment that has previously been under recognized and under treated.

Conclusion

By no means have we "figured out" this serious injury of the knee - our work remains incomplete. We continue to combine laboratory research with clinical investigation in our pursuit of more complete and less invasive methods for reconstruction of the severe tibial plateau fracture. We are striving to identify the effects that associated ligamentous injuries have in the outcome after these fractures, to determine the best way to regain the range of motion of the knee so critical to getting the best outcome, and to minimizing the risk of arthritis and pain in the knee after these injuries. We are starting investigations of methods for regrowing and replacing damaged cartilage. Finally, we are committed to finding a faster way to get patients back to the lives they lead before their injury. None of these answers will come easily, or quickly, but they need to be found. Here at the University of Washington's Harborview Medical Center, we will continue to ask the tough questions today, just like we have in the past, so that we can get the answers tomorrow.

References

Barei DP, Nork SE, Mills WJ, Henley MB, Benirschke SK. Complications associated with internal fixation of high-energy bicondylar tibial plateau fractures utilizing a two-incision technique. J Orthop Trauma 2004;18(10):649-57.

Barei DP, Nork SE, Mills WJ, Coles CP, Henley MB, Benirschke SK. Functional outcomes of severe bicondylar tibial plateau fractures treated with dual incisions and medial and lateral plates. J Bone Joint Surg Am 2006;88(8):1713-21.

Benirschke SK, Agnew SG, Mayo KA, Santoro VM, Henley MB. Immediate internal fixation of open, complex tibial plateau fractures: treatment by a standard protocol. J Orthop Trauma 1992;6(1):78-86.

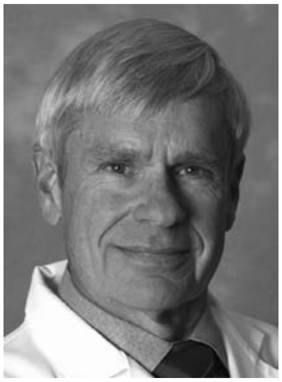
Cole PA, Zlowodzki M, Kregor PJ. Less Invasive Stabilization System (LISS) for fractures of the proximal tibia: indications, surgical technique and preliminary results of the UMC Clinical Trial. Injury 2003;34 Suppl 1: A16-29.

Cole PA, Zlowodzki M, Kregor PJ. Treatment of proximal tibia fractures using the less invasive stabilization system: surgical experience and early clinical results in 77 fractures. J Orthop Trauma 2004;18(8):528-35.

Barei DP, O'Mara TJ, Falicov A, Taitsman LA, Nork SE. The Tibial Less Invasive Stabilization System (LISS) and its Relationship to the Posteromedial Fragment in Bicondylar Tibial Plateau Fracture Patterns. In: Orthopaedic Trauma Association 21st Annual Meeting; 2005; Ottawa, Canada; 2005.

Gosling T, Schandelmaier P, Marti A, Hufner T, Partenheimer A, Krettek C. Less invasive stabilization of complex tibial plateau fractures: a biomechanical evaluation of a unilateral locked screw plate and double plating. J Orthop Trauma 2004;18(8):546-51.

Barei DP, O'Mara TJ, Taitsman LA, Dunbar RP, Nork SE. Frequency and Fracture Morphology of the Posteromedial Fragment in Bicondylar Tibial Plateau Fracture Patterns. J Orthop Trauma 2008;in press.



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Compartmental Syndromes: When the Pressure Gets Too Great

- A compartmental syndrome is a condition in which increased pressure in a confined space compromises the circulation and function of the contents of that space.
- Compartmental syndromes most often arise when swelling of muscle occurs in a part of the arm or leg that is confined by a tough tissue known as fascia.
- Compartmental syndromes are common causes of permanent disability following injuries such as fractures of the leg, fractures of the forearm and arm, dislocation of the elbow or knee, and prolonged pressure on the arm or thigh.
- Compartmental syndromes may also arise from excessive exercise, such as prolonged running or marching.
- The most practical and sensitive method for evaluating an arm or leg for possible compartmental syndrome is repeated clinical examination looking for (1) pain out of proportion to what would be expected from the clinical situation, (2) weakness of the muscles in the compartment, (3) diminished sensation in the distribution of the nerves running through the compartment, (4) pain on passive stretch of the muscles in the compartment, and (5) tenseness on palpation of the compartment
- If untreated, compartmental syndromes can lead to permanent deformity and loss of function, including both strength and sensation in the hand or foot.
- Because tissue has a very limited tolerance for increased pressure, the diagnosis of compartmental syndrome needs to be made promptly and surgical decompression needs to be carried out within hours of its onset.
- Once a compartmental syndrome is present, elevation of the limb above the heart further compromises the circulation to the compressed tissues.
- When a large amount of muscle dies as a result of a compartmental syndrome (rhabdomyolysis), there is a risk of kidney failure.
- In conditions where the risk of compartmental syndrome is high, preventative treatment, known as prophylactic fasciotomy is considered.

Thirty-five years ago there was a lot of confusion regarding the cause of weakness and deformity in the hands and feet after seemingly straightforward injuries, such as a fracture of the arm from roller skating or a fracture of the tibia from skiing. It was also noted that army recruits required to march long distances would develop similar problems in their legs and that sometimes this condition was complicated by failure of the kidneys. The names attached to these problems were confusing as well: march gangrene, Volkmann's ischemia, ischemic contracture, and claw foot. Opinion on the cause was divided: some attributed the problem to spasm of the arteries and others believing blockage of the veins was the problem. The diagnosis was often missed until it was too late because physicians could not understand how there could be inadequate circulation to the leg or arm when the pulses were normal and the digit tips were pink. This seeming paradox was ultimately explained by our group as shown in Figure 2.

Kay Clawson and Ted Hansen had a substantial interest in complications of trauma. They suggested that Rick Matsen take a year out of clinical residency training to work toward a better understanding of post-fracture swelling and its effects on limb function. Funding for this year was provided by

Figure 1: Alice: “I’ll just see what this bottle does. I hope it’ll make me grow large”…It did so indeed. (Reproduced from Matsen: Compartmental Syndromes.)

an NIH training grant. In the 1974-1981 era, substantial contributions to the field were made by an expanded investigative team that included Dick Krugmire, Geoff Sheridan, Bob Winquist, Anne Matsen, Keith Mayo, Rachael King, Cammy Mowery, Craig Wyss, Charles Simmons, Stan Newell and Rob Veith. These contributions can be grouped in categories as describe below.

Vocabulary and causation

We discovered that many clinical conditions shared the same underlying physiology: increased pressure within a limited space can compromise the circulation and function of the contents of that space. We defined a compartmental syndrome as any condition where this mechanism was in effect. We found that the leg had four compartments, any of which could be the site of a compartmental syndrome: the anterior, the lateral, the superficial posterior and the deep posterior (this latter was first described by our group in 1975). Compartmental syndromes of the forearm were most common in the deep and superficial volar compartments. While these compartments in the leg and forearm are limited by tough fibrous tissue known as fascia, compartmental syndromes can also occur within tight skin, tight dressings, and tight casts. One of the more interesting cases we encountered was a compartmental syndrome occurring within shrunk-fit jeans!

The increased pressure within these limited spaces can arise from a wide variety of causes, including fractures, contusions, crush injuries, surgery, arterial injury, infiltrated infusions, prolonged exercise and post-ischemic swelling (the swelling that occurs after circulation is restored following a period of poor circulation).

Pathophysiology

In each compartmental syndrome regardless of the initial cause, the mechanism of circulatory compromise is the same. Tissue is normally nourished by blood flowing through capillaries. The rate of capillary blood flow is determined by the perfusion pressure, i.e. the difference between the pressures at the arterial and venous ends of the capillaries. The pressure at the arterial end is the systemic blood pressure (unless the limb is elevated, in which case the local blood pressure is reduced by the amount of elevation). The pressure at the venous end of the capillary is determined by the local tissue pressure. In the normal recumbent person, the mean arterial pressure in the leg may be 80 mm Hg and the local venous pressure may be 10 mm Hg, so the perfusion pressure driving capillary blood flow might be 70 mm Hg. If, however, the local tissue pressure in the anterior compartment of the leg is increased by swelling in the closed space to 60 mm Hg, the perfusion pressure is reduced to 20 mm Hg, which may be insufficient to keep the tissues in that compartment functioning and even alive. Note that a tissue pressure of 60 mm Hg is not enough to cut off the arterial circulation (with its pressure of 80 mm Hg) so that the pulses at the ankle and the circulation to the toes may be completely normal. Note also that if this leg is elevated (in a vain attempt to reduce the swelling), the local arterial pressure will be reduced by the amount of the hydrostatic column between the leg and the heart, further reducing the capillary perfusion pressure. As a specific example, elevation of the leg 52 cm above the level of the heart reduces the local arterial pressure, as well as the tolerance of the leg for increased tissue pressure by 40 mm Hg! (1.3 cm

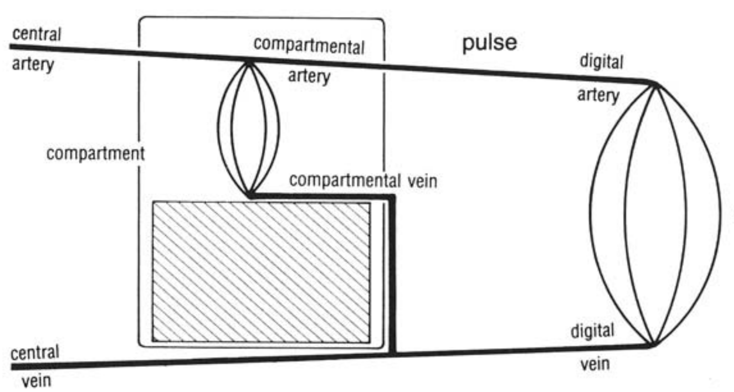

Figure 2: Pathophysiology and clinical findings in compartmental syndromes. Tissue perfusion in compartment (vertical rectangle) is compromised by the increased tissue pressure (shaded rectangle) that elevates the pressure in the compartmental veins. The pressure in the artery and in the extracompartmental veins is unaffected by the increase in tissue pressure. Thus the pulse and circulation to the digit remain normal in spite of impaired circulation to the tissues within the compartment. (Reproduced from Matsen: Compartmental Syndromes.)

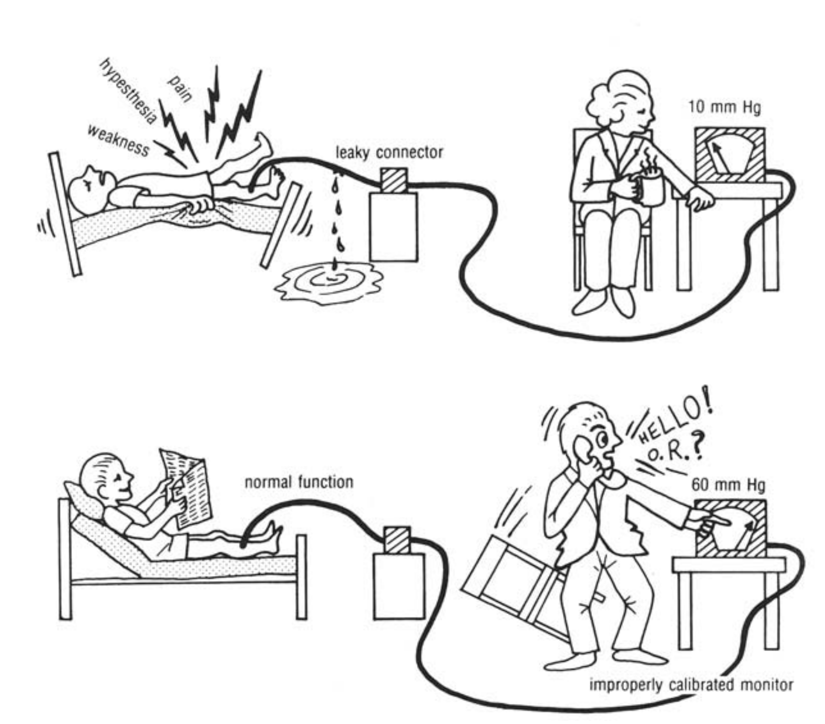

Figure 3: Dependence on pressure measurements alone can lead to delays in diagnosis (above) or over diagnosis (below) of compartmental syndromes. (Reproduced from Matsen: Compartmental Syndromes.)

whole blood = 1 mm Hg).

Tolerance of tissue for increased pressure

We were very interested in the question of how living tissue responded to increased tissue pressure. We developed a laboratory model of compartmental syndrome that consisted of a balloon that could be placed within a compartment of a small animal and then inflated to any desired pressure while the function of the muscle in the compartment was monitored. We then turned to a human model system in which pressure was applied to the leg using an air splint while the functioning of the nerves and muscles within the leg were monitored clinically and electrophysiologically. In both the animal and human models we were able to measure the tissue oxygen tension as well, using a special catheter connected to a mass spectrometer. Using the human model we were again able to demonstrate that the susceptibility of the tissue of the pressurized leg to ischemia (poor blood flow) was significantly increased by elevation of the leg above the level of the heart.

New efforts have been focused on a non-invasive, continual and real-time monitoring system for tissue perfusion. Near infrared spectroscopy (NIRS), a new technology which uses visible light to measure the percentage of oxygenated hemoglobin approximately 3 cm below the skin, has been FDA approved for monitoring cerebral perfusion during anesthesia. At Harborview Medical Center, Garr et al. applied this technology in the diagnosis of acute compartmental syndrome. Muscle oxygenation measured by NIRS was shown to be inversely related to increased intracompartmental pressures with high specificity and sensitivity in animals as well as human tourniquet studies.

Diagnosis of compartmental syndrome

Although we pioneered methods for the continuous monitoring of intracompartmental pressures, our clinical experience taught that individuals varied with respect to their tolerance for increased tissue pressure and that no absolute or relative value for intracompartmental pressure could be considered diagnostic of a compartmental syndrome or indicative of the need for surgical decompression (Figure 3).

Instead the most sensitive and specific approach for diagnosing compartmental syndromes is the repeated assessment of the physiological status of the tissue within the compartment by documenting the presence or absence of: (1) pain out of proportion to what would be expected from the clinical situation, (2) weakness of the muscles in the compartment, (3) diminished sensation in the distribution of the nerves running through the compartment, (4) pain on passive stretch of the muscles in the compartment, and (5) tenseness on palpation of the compartment. This clinical examination is most sensitive if it is performed serially (i.e. hourly) on limbs at risk for compartmental syndromes and if the findings are well-documented in the chart, where it is accessible to all those caring for the patient.

Treatment

Once the tissue within the compartment shows signs of ischemia from increased pressure, the treatment is surgical decompression by opening the fascia, skin, and any dressings that may potentially contribute to the increase in pressure.

One of our contributions to treatment was the description of the relatively function-preserving perifibular technique for decompression the four compartments of the leg (see Figures 5 & 6). In the management of individuals with compartmental syndrome it is important to keep an eye on the function of the kidneys, making sure the patient does not develop renal failure from the load of myoglobin released into the circulation by dying muscle.

Conclusion

In the future, research needs to be directed at anticipating when limbs will be at risk for compartmental syndrome so that they can be closely monitored and that prophylactic decompression can be considered. In addition, research needs to be directed at methods for preserving muscle and nerve function as well as joint range of motion after surgical treatment of compartmental syndromes.

Our research has been generously supported by the National Institutes of Health, the Orthopaedic Research

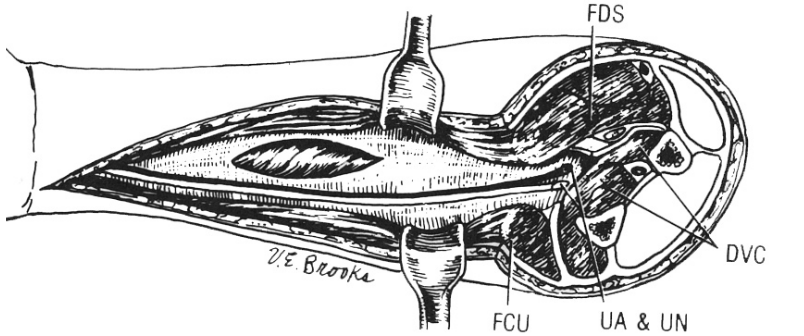

Figure 4: Surgical decompression of the deep compartment of the forearm from the relatively concealed ulnar incision. (Reproduced from Matsen: Compartmental Syndromes.)

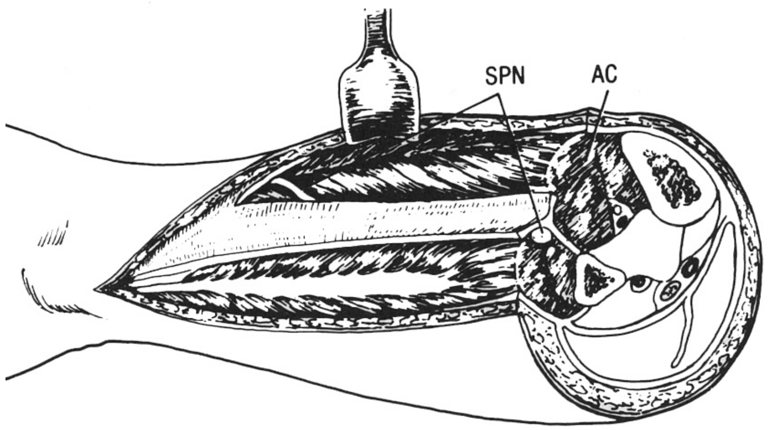

Figure 5: The perifibular approach to decompression of the four compartments of the leg: decompression of the anterior and lateral compartments. (Reproduced from Matsen: Compartmental Syndromes.)

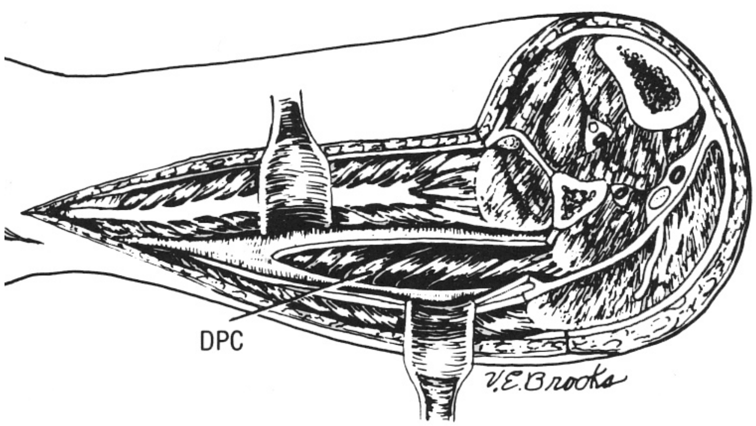

Figure 6: The perifibular approach to decompression of the four compartments of the leg: decompression of the superficial and deep posterior compartments. (Reproduced from Matsen: Compartmental Syndromes.)

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References

Matsen III, F.A., and Krugmire, R.B.: The Effect of Externally Applied Pressure on Post fracture Swelling. J. Bone Joint Surg., 56-A:1586-1591, 1974.

Matsen III, F.A., and Clawson, D.K.: The Deep Posterior Compartmental Syndrome of the Leg. J. Bone Joint Surg., 57-A:34-39, 1975.

Matsen III, F.A., Questad, K., and Matsen, A.L.: The Effect of Local Cooling on Postfracture Swelling--A Controlled Study. Clin. Orthop., 109:201-206, 1975.

Matsen III, F.A., and Staheli, L.T.: Neurovascular Complications Following Tibial Osteotomy in Children--A Case Report. Clin. Orthop., 110:210-214, 1975.

Matsen, F.A.: Compartmental Syndrome: A Unified Concept. Clin. Orthop., 113:8-14, 1975.

Sheridan, G.W., and Matsen III, F.A.: An Animal Model of the Compartmental Syndrome. Clin. Orthop., 113:36-42, 1975.

Sheridan, G.W., and Matsen III, F.A.: Fasciotomy in the Treatment of the Acute Compartmental Syndrome. J. Bone Joint Surg., 58-A:112-115, 1976.

Matsen III, F.A., Mayo, K.A., Sheridan, G.W., and Krugmire, R.B.: Monitoring of Intramuscular Pressure. Surgery, 79:702-709, 1976.

Sheridan, G.W., Matsen III, F.A., and Krugmire, R.B.: Further Investigation on the Pathophysiology of the Compartmental Syndrome. Clin. Orthop., 123: 266-270, 1977.

Matsen III, F.A., Mayo, K.A., Krugmire, R.B., Sheridan, G.W., and Kraft, G.H.: A Model Compartmental Syndrome in Man with Particular Reference to the Quantification of Nerve Function. J. Bone Joint Surg., 59-A:648-653, 1977.

Matsen III, F.A., Mayo, K.A., Sheridan, G.W., and Krugmire, R.B.: Continuous Monitoring of Intramuscular Pressure and Its Application to Clinical Compartment Syndromes. 9th European Conf. Microcirculation, Antwerp, Belgium, 1976. Bibl. Anat., 15:112-115 (Karger-Basel), 1977.

Matsen III, F.A., and Krugmire, R.B.: Compartmental Syndromes. Surg. Gynecol. Obstet., 147:943-949, 1978.

Matsen III, F.A.: Compartmental Syndromes. New England J. Med. (Editorial), 300:1210-1211, 1979.

Matsen III, F.A., King, R.V., Krugmire, R.B., Mowery, C.A., and Roche, T.: Physiological Effects of Increased Tissue Pressure. International Orthopaedics (SICOT), 3:211-291, 1979.

Matsen III, F.A., Krugmire, R.B., and King, R.V.: Increased Tissue Pressure and Its Effect on Muscle Oxygenation in Level and Elevated Human Limbs. Nicholas Andry Award. Clin. Orthop., 144:311-320, 1979.

Matsen III, F.A., Winquist, R.A., and Krugmire, R.B.: Diagnosis and Management of Compartmental Syndromes. J. Bone Joint Surg., 62-A:286-291, 1980.

Matsen III, F.A., Wyss, C.R., Krugmire, R.B., Simmons, C.W., and King, R.V.: The Effects of Limb Elevation and Dependency on Local Arteriovenous Gradients in Normal Human Limbs with Particular Reference to Limbs with Increased Tissue Pressure. Clin. Orthop., 150:187-195, 1980.

Veith, R.G., Matsen III, F.A., and Newell, S.G.: Recurrent Anterior Compartmental Syndromes of the Leg Due to Intensive Use of Muscles. Phys. Sportsmed, 8:80-88, 1980.

Matsen III, F.A.: Investigation of Viability of the Neuromuscular Structures of the Extremities. Bull. Prosthet. Res., 17:100-102, 1980.

Matsen III, F.A., Wyss, C.R., King, R.V., Barnes, D., and Simmons, C.W.: Factors Affecting the Tolerance of Muscle Circulation and Function for Increased Tissue Pressure. Clin. Orthop., 155:204-210, 1981.

Matsen III, F.A., and Veith, R.: Compartmental Syndromes in Children. J. Ped. Orthop., 1:33-41, 1981.

Matsen III, F.A.: Compartmental Syndromes. Grune & Stratton, New York, 1980.

Garr J.L., Gentilello L.M., Cole P.A., Mock C.N., Matsen F.A.: Monitoring for compartmental syndrome using near-infrared spectroscopy: a noninvasive, continuous, transcutaneous monitoring technique. J. Trauma, 46(4):613-618, 1999.

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Articular Fractures of the Distal Tibia: Pilon Fractures

- Fractures of the tibial pilon are injuries than involve the ceiling or top of the ankle joint. These are distinguished from "ankle fractures" which involve the sides or malleoli at the ankle joint.
- Tibial pilon fractures are usually the result of high energy trauma. Typical mechanisms of injury are falls from height (for example, falls from a ladder, roof, or building), motor vehicle crashes, or motorcycle crashes. Sometimes they occur from other activities such as snow skiing.
- These injuries are incredibly difficulty to manage due to the amount of damage to the joint, the unforgiving nature of operating in this region, and the large number of potentially devastating complications.
- Restoration of the normal anatomy following a fracture of the roof of the ankle joint is challenging. If left untreated, patients can expect to have poor function, deformity, and difficulty with walking.
- The goals of treatment include restoration of the normal osseus anatomy of the ankle, as well as reconstruction of the joint surface. Avoidance of complications is difficult.
- Multiple studies have shown that the treatment for these injuries has a high complication rate. This has resulted in alterations in treatment that have improved results.
- The faculty of the Department of Orthopaedics and Sports Medicine at Harborview Medical Center have played and continue to play a key role in discovering safer and more effective methods for managing these serious injuries.

Fractures of the tibial pilon (distal tibial articular surface) are associated with potentially devastating consequences, including arthritis, stiffness and pain in the ankle joint. This is largely due to the concentration of injury on the very small articular surface of the distal tibia, which is typically less than two inches in diameter. With high-energy fractures, the joint surface may be split in multiple pieces, often with 3 to 10 separate fragments of the normally smooth articular cartilage that is essential for normal joint function. The injury is further complicated by severe bony deformity and soft tissue swelling that can threaten the viability of the skin.

Without restoration of the normal anatomy of the joint surface, poor outcomes can be expected. This includes chronic pain, ankle joint arthritis, and difficulty with walking. Surgical strategies to restore the joint surface have been associated with problems. Swiss surgeons introduced implants and techniques for reduction and fixation approximately 40 years ago. However, these strategies, while successful in lower energy skiing injuries, where not applicable to most high-energy fractures seen at North American trauma centers. Complications included difficulty in re-establishing the anatomy of the joint, infection, and ultimately amputation in an unacceptable number of patients following surgical treatment of these injuries.

At Harborview, we have been developing protocols that increase the accuracy and safety of surgical management of pilon fractures. We are sharing our discoveries with the national and international community of surgeons who encounter patient with these injuries. Now many of these fractures can be treated surgically with an accurate restoration of the local anatomy, improving patient outcomes and allowing patients to return to activities.

Figures 1,2: This 26 year-old female injured her left ankle after a high-speed motor vehicle crash. The x-rays demonstrate the limb shortening, the joint injury, and the deformity of the leg that is commonly seen after these high-energy injuries.

Treatment Approach

After years of exploring different treatment options, we have developed a staged protocol for the management of pilon fractures. The first step is urgent operative fixation of the associated fracture of the fibula (the smaller bone of the leg) combined with temporary spanning external fixation using pins above and below the fracture and a frame to hold the leg out to length. This first step gives the leg some stability and allows for resolution of the soft tissue swelling in the region of the ankle joint. After two to three weeks, when the swelling has subsided, an open incision is used to reconstruct the joint surface and

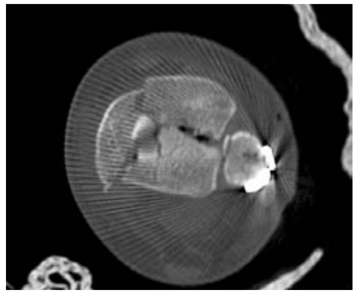


Figure 3: The axial computed tomography scan (radiographic “slice” of the tibia just above the ankle joint) shows the multiple pieces of the joint (five distinct fragments are demonstrated) as well as the articular impaction.

the supporting bone. At the time of this surgery, we must deal with the challenges of limited access to the joint, difficulty putting the pieces back in the normal anatomical configuration, difficulty holding the pieces in position for healing, and enabling healing of the soft tissues. Even with our extensive experience, pilon fractures remain a challenge because of their propensity for infection, lack of bone healing, persistent pain, and ankle arthritis.

Advances in Treatment

We have discovered that by using meticulous soft tissue techniques and strategic surgical incisions, the access to the joint and the risk of surgery can be optimized. This has allowed for improved reduction of the ankle joint and potentially, minimization of future post-traumatic ankle arthritis. Furthermore, we learned that if there are other fractures of the tibia, fixing these fractures at a preliminary surgery facilitates later reconstruction of the joint surface. Finally, we have developed methods for managing fractures in which there is loss of bone from the tibia using bone grafting to restore the anatomy of the leg.

Future Study and Conclusions

The treatment of tibial pilon fractures has improved significantly over the past decade, largely due to improvements in surgical technique, a better understanding of the injury patterns, and improved protocols for treatment. Many of these improvements emanated from work done at Harborview. Our current investigations include research in cartilage healing, prevention of cartilage death after injury, and

Figures 4,5: The patient underwent initial fixation of the fibula (the small bone) with temporary spanning external fixation. Several weeks later, following resolution of the soft tissue swelling, she underwent fixation of the joint. The front (AP) and side (lateral) views after healing demonstrate the accurate reconstruction of the joint surface as well as the relationship of the ankle joint with the remaining tibia.

strategies for maintaining ankle joint motion following these devastating injuries.

References

Barei, DP; Nork, SE; Bellabarba, C; Sangeorzan, BJ: Is the absence of an ipsilateral fibular fracture predictive of increased radiographic tibial pilon fracture severity? J Orthop Trauma, 20(1): 6-10, 2006.

Dunbar, RB, Barei, DP, Kubiak, EN, Nork, SE. Staged Management of Tibial

Pilon Fractures Effectively Transforming AO/OTA "C" Type Fractures into AO/OTA "B" Type Fractures. Manuscript Accepted to J Orthop Trauma.

Howard, JL, Agel, J, Barei, DP, Benirschke, SK, Nork, SE. A Prospective Study Evaluating Incision Placement and Wound Healing for Tibial Plafond Fractures. Manuscript Accepted to J Orthop Trauma.

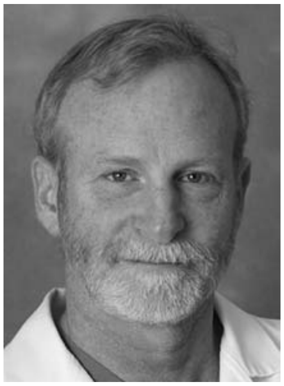
Gardner, MJ, Mehta, S, Barei, DP, Nork, SE. Treatment Protocol for Open Type C3 Pilon Fractures with Segmental Bone Loss. Manuscript Submitted to J Orthop Trauma.

Gardner, MJ, Nork, SE, Benirschke, SK, Barei, DP. Revision of Provisional Stabilization in Pilon Fractures Referred from Outside Institutions. Manuscript in progress.

Mehta, S, Gardner, MJ, Barei, DP, Benirschke, SK, Nork, SE. Reducton Strategies through the Anterolateral Exposure for Fixation of 43B and 43C Pilon Fractures. Manuscript in progress.

Nork, SE; Schwartz, AK; Agel, J; Holt, SK; Schrick, JL; Winquist, RA: Intramedullary nailing of distal metaphyseal tibial fractures. J Bone Joint Surg Am, 87(6): 1213-21, 2005.

Nork, SE: Distal Tibia Fractures. In Stannard, JP, Schmidt, AH, Kregor, PJ eds. Surgical Treatment of Orthopaedic Trauma. Thieme, Chapter 31, pp. 767-791, 2007.



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A New Level of Care for the Broken Foot

- Prior to the modern era of orthopedic trauma care, fractures of the foot were not anatomically aligned. Instead they were treated by immobilization in a plaster cast using crutches for protected weight bearing until the fractures were healed.
- Little attention was paid to the results of treatment in economic terms or in terms of quality of the patient's life.
- We learned that displaced fractures treated in this casual manner often result in a stiff, deformed foot with post-traumatic arthritis with great limitations in functions of daily living, recreation and work.
- We found that many injuries of the small bones in the foot (tarsal bones) were overlooked because of their odd shapes and overlying shadows on x-rays.
- We have discovered better methods for diagnosing foot injuries and better and less invasive methods for surgical treatment of fractures and dislocations of the foot.
- These methods reduce the risk of arthritis and deformity while allowing the foot to be mobilized more quickly.
- The improvements are yielding reduced healing times and better long-term function.

Several things occurred in the '70s that helped improve our understanding of the impact of foot fractures. First Harborview hospital developed into the regional trauma center for not only King County but for the state of Washington. This allowed concentration of injuries in one location with a better ability to study uncommon problems and outcomes. Secondly orthopedic trauma technology as a field began to evolve. Thirdly, Sigvard Hansen Jr. had completed his fellowship in pediatric orthopedics and developed in interest in the function of the foot. Combining an improved understanding of the foot, a growing population of patients with foot injuries, and principles of orthopedic fracture care paved the way for discovering approaches that are leading to much improved outcomes.

Understanding the foot

Foot injuries and deformities used to be treated by shoe modification, and/or activity modification. Doctors accepted disability after these injuries as being inevitable. When Dr. Hansen returned from his time in England treating childhood deformities such as clubfoot, he brought with him a better understanding of the basics of foot function. Lessons learned from the treatment of childhood clubfoot were applied to fractures and the deformities that resulted from them. These principles are simple: the foot needs to be able to come flat to the ground, the hindfoot bones need to be in the plumb line of the leg and the midfoot and forefoot bones need to form a straight platform. The key for the foot is to combine the flexibility needed to manage uneven surfaces with the solid support necessary to stabilize the loads encountered in standing, walking, work and sport.

The '70s also brought improvement in imaging technology. For the first time, computed tomography allowed 3-dimensional imaging of the musculoskeletal system. It allowed us to make sense of complicated injuries in the small bones that had previously been an unsolved puzzle. We are now developing methods for using dynamic three-dimensional imaging to better understand how the bones of the foot move under normal loading and after different injuries and surgical reconstructions. This has been a most exciting advance in that the complex motions of the foot could not be understood by imaging in only two dimensions.

Trauma as a specialty

In the 1970s medical delivery

Figure 1: The x-ray on the left shows a fracture of the talus with most of the injured bone pushed out the back of the ankle joint. All functions of the foot would be incapacitated if the fracture was not surgically reduced. The image on the right shows the bone back in place with screws and a plate holding it in the correct position.

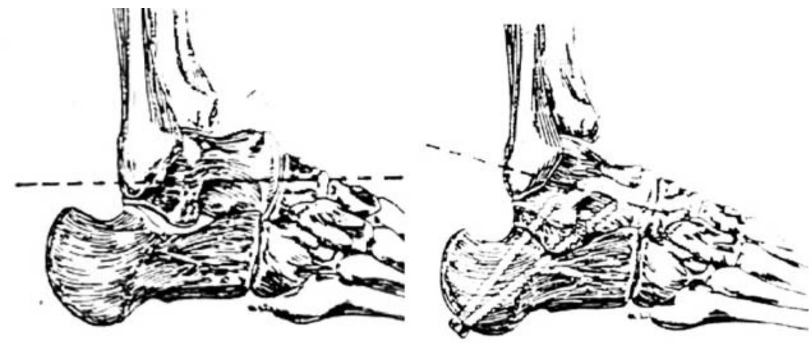

Figure 2: When calcaneus fractures are treated in a cast, they are left in a flattened position (left) that doesn't allow the ankle or major foot joints to function. Fusion of the calcaneus to the talus can establish a better position, but movement at the subtalar joint is sacrificed (right).

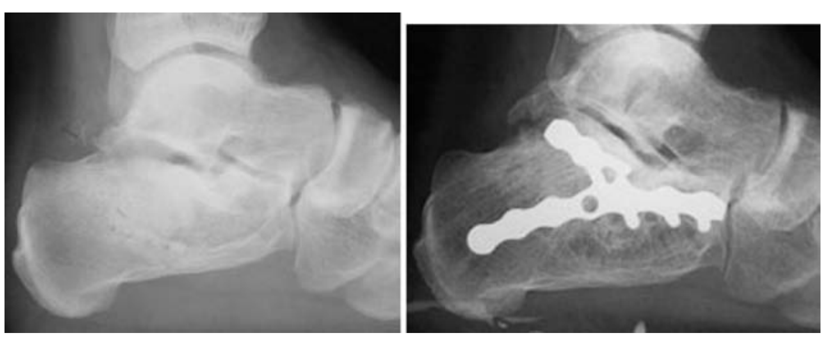

Figure 3: A better option is to repair the calcaneus fracture, restoring all the normal shape and preserving the joint. A side view of the calcaneus with a fracture (left) shows the joint disruption and the flattening of the foot as in the diagram above. The X-ray on the right shows the fracture fragments put back into place and held with a steel plate and screws. In this scenario the function is maintained in all the joints.

became sophisticated enough to realize that lives were lost un-necessarily to lack of immediately available care. At Harborview we were among the first centers to acknowledge trauma care is a specialty. The hospital along with Medic One began to develop protocols for early resuscitation of the severely injured. Now that many patients are surviving major injuries, we are challenged to optimize the management of complex foot and ankle injuries that accompany the previously fatal injuries to the head, chest and abdomen. We discovered that the principles for treatment of these severely injured 'polytrauma' patients included (1) early surgical intervention before swelling and skin problems appeared, (2) restoration of the normal anatomy of the injured foot, (3) stabilization of the injuries so that the foot could be mobilized early, and (4) prompt restoration of the patient's ability to get out of bed, reducing the risk of bedsores, lung, bladder and bowel problems.

The biomechanics lab at Harborview

At Harborview we have the unique combination of a high volume of injuries from all over our region and a state of the art Orthopaedic Science Laboratory. This allows us to pair researchers with treating physicians so that clinical problems can be brought to the lab and laboratory solutions can be brought to the operating room. The Orthopaedic Science Laboratory has tested many possible approaches for facture fixation and has worked to define the maximal amount of deformity that is consistent with good function. These results have enabled us to set standards for when and how to stabilize the bones of the foot.

Our current understanding of the foot

The foot is divided into three parts: the hind-foot, the mid foot, and forefoot. The hind-foot consists of two large bones the talus and calcaneus.

The talus is the most functional single bone in the foot. It participates in motions of the foot in all planes and is largely covered by articular cartilage. Because there are few places for the bone to receive its nutrient blood supply it is at risk for bone death (avascular necrosis) after injury or surgery. We have discovered new techniques using specialized bone screws for fixing fracture of the talus with limited surgical exposure. In some high-energy injuries, the talus is extruded in some high-energy injuries out of the joint and out of the patient altogether so that it is found lying on the street. In the past, surgeons were afraid to put an extruded talus back in the foot, leaving no opportunity for regaining comfort or function. At Harborview we pioneered the a method for cleaning the contaminated talus and putting it back in the foot. This has now become the accepted method for managing these injuries.

The calcaneus is the heel bone, the

Figure 4: This foot sustained an injury to Lisfranc's joint. Pins through the skin were used to hold it in place. When the pins had to be removed, the injury had not completely healed and the foot collapsed and turned outward.

one responsible for taking bodyweight at heel strike (when the foot hits the ground). It's very often fractured in a fall from a height. The calcaneus is often fractured in high-speed motor vehicle collisions. While improvements in airbags protect the body with a "crumple zone", this puts the feet at risk for severe injury. The foot does not have muscle around and is only covered by a thin layer of skin, surgery to correct calcaneus fractures used to carry prohibitively high risk. We discovered surgical approaches and principles that enabled the safe surgical management for the reduction and fixation of these common injuries. As a result the majority of patients with calcaneal fractures can return to normal activity after this injury.

The middle of the foot is the area of Lisfranc's joint. Injuries to this joint can have a substantial impact on long-term foot function. Previous treatment consisted of pushing the bones back into approximate alignment, placing small temporary wires through the skin and placing the foot into a cast. This method was often complicated by pin track infections and loss of the desired position of the bones.

We discovered methods for rigidly restoring the anatomy of the foot after an injury to Lisfranc's joint. These methods combined with aggressive rehabilitation have greatly improved the function and comfort of the foot after these injuries.

Summary

Prior to the modern era fractures of the foot were not considered important in the overall management of the injured individual. They were often placed in a cast and allowed to "heal." The result was too often someone who survived a crushed chest and abdomen but who had to spend the rest of their life limping on a painful foot. The dramatically increasing rates of survival after major trauma has prompted us to focus on better methods for restoring form, comfort and function to the injured foot. We have developed better methods for imaging, understanding, treating and rehabilitating these injuries. These discoveries were facilitated by the careful treatment and follow-up of the large numbers of these injuries we see at the Harborview trauma center and by the proximity of the Harborview Orthopaedic Science Laboratories. We also have the unique benefit of (1) the guidance of S. T. Hansen, Jr. as our senior foot surgeon, (2) the support of the Research Rehabilitation and Development Center for Excellence in Limb Loss Prevention and Prosthetic Engineering of which Bruce Sangeorzan is the Director, (3) the Jerome H. Debs II Endowed Chair and its holder Stephen Benirschke, (4) the Sigvard T. Hansen Jr. Endowed Chair and its holder Ted Gross, head of the Orthopaedic Science Laboratory, and (5) the terrific traumatology team of faculty, fellows, residents and staff at Harborview. We are especially grateful for the generous philanthropic support that has enabled much of our progress. Our work has also been supported by research grants from the federal government including:

"Contact Pressures in the Hindfoot" Veterans Administration RR&D Grant $201,000 Sangeorzan BJ: Principal Investigator; Tencer AF: Co-Investigator)

"Trauma Induced Changes in the Hindfoot" Department/AO North America Research Grant $20,000 Sangeorzan BJ: Principal Investigator

"Hindfoot Mechanics: A Cadaver Model" National Institutes of Health. Biomedical Research Grant $16,250 Sangeorzan BJ: Principal Investigator

"Subtalar and Transversal Joint Mechanics" Department of Veterans Affairs RR&D Grant A553-2RZ April 1993-March 1996 $290,000 Sangeorzan BJ: Principal Investigator (Tencer AF, Sidles JA: Co-Investigators)

Figure 5: The radiograph on the left shows a badly injured Lisfranc's joint. The bones are fractured and the joints are dislocated. The image on the right shows all the anatomy restored and held with screws and pins. The screws are under the skin, so they don't need to be removed. They hold the bones rigidly enough that the patient can begin to rehabilitate the foot before the bones are fully healed.

"Investigation of Midtarsal Joint Locking" American Orthopaedic Foot and Ankle Society $10,000 April 1, 2003 - March 31, 2004 Co-Investigator Ledoux W."

The Future

Our work is far from done. Our patients continue to be challenged by injuries to the skin that complicate the management of foot fractures, by slow healing times and by injuries that severely damage the surfaces of the critical joints in the future. Our research is directed at discovering more precise methods for reconstructing normal anatomy while using smaller incisions and less invasive approaches. We have a great interest in methods for speeding the healing of bone and for restoring damaged joint surfaces. Finally we will continue our work in dynamic imaging to learn more about the way the normal foot works so that we will better understand how to restore lost function.

References - Foot Function

Blackwood CB, Yuen TJ, Sangeorzan BJ, Ledoux WR. The midtarsal joint locking mechanism. Foot Ankle Int. 2005 Dec;26(12):1074-80.

Ledoux WR, Sangeorzan BJ.Clinical biomechanics of the peritalar joint. Foot Ankle Clin. 2004 Dec;9(4):663-83.

Sangeorzan BJ, Hansen ST JrEarly and late posttraumatic foot reconstruction. Clin Orthop Relat Res. 1989 Jun;(243):86-91.

References - Talus Injuries

Smith CS, Nork SE, Sangeorzan BJ The extruded talus: results of reimplantation. J Bone Joint Surg Am. 2006 Nov;88(11):2418-24.

Vallier HA, Nork SE, Benirschke SK, Sangeorzan BJ.Surgical treatment of talar body fractures. J Bone Joint Surg Am. 2004 Sep;86-A Suppl 1(Pt 2):180-92.

Vallier HA, Nork SE, Barei DP, Benirschke SK, Sangeorzan BJTalar neck fractures: results and outcomes. J Bone Joint Surg Am. 2004 Aug;86-A(8):1616-2.

Vallier HA, Nork SE, Benirschke SK, Sangeorzan BJSurgical treatment of talar body fractures. J Bone Joint Surg Am. 2003 Sep;85-A(9):1716-24.

Sangeorzan BJ, Mayo KA, Hansen ST. Intraarticular fractures of the foot. Talus and lesser tarsals. Clin Orthop Relat Res. 1993 Jul;(292):135-41.

Sangeorzan BJ, Wagner UA, Harrington RM, Tencer AF. Contact characteristics of the subtalar joint: the effect of talar neck misalignment. J Orthop Res. 1992 Jul;10(4):544-51.

References - Calcaneus Injuries

Schildhauer TA, Sangeorzan BJ Push screw for indirect reduction of severe joint depression-type calcaneal fractures. J Orthop Trauma. 2002 Jul;16(6):422-4.

Barei DP, Bellabarba C, Sangeorzan BJ, Benirschke SKFractures of the calcaneus. Orthop Clin North Am. 2002 Jan;33(1):263-85.

Harvey EJ, Grujic L, Early JS, Benirschke SK, Sangeorzan BJMorbidity associated with ORIF of intra-articular calcaneus fractures using a lateral approach. Foot Ankle Int. 2001 Nov;22(11):868-73.

Sangeorzan BJSalvage procedures for calcaneus fractures. Instr Course Lect. 1997;46:339-46.

Sangeorzan BJ, Ananthakrishnan D, Tencer AF Contact characteristics of the subtalar joint after a simulated calcaneus fracture. J Orthop Trauma. 1995 Jun;9(3):251-8.

Macey LR, Benirschke SK, Sangeorzan BJ, Hansen ST.Acute Calcaneal Fractures: Treatment Options and Results. J Am Acad Orthop Surg. 1994 Jan;2(1):36-43.

Benirschke SK, Sangeorzan BJ. Extensive intraarticular fractures of the foot. Surgical management of calcaneal fractures. Clin Orthop Relat Res. 1993 Jul;(292):128-34.

Benirschke SK, Kramer PA.Wound healing complications in closed and open calcaneal fractures. J Orthop Trauma. 2004 Jan;18(1):1-6. Review.

References - Lisfranc/Midfoot Injuries

Carr JB, Hansen ST, Benirschke SK.Subtalar distraction bone block fusion for late complications of os calcis fractures. Foot Ankle. 1988 Oct;9(2):81-6.

Schildhauer TA, Nork SE, Sangeorzan BJTemporary bridge plating of the medial column in severe midfoot injuries. J Orthop Trauma. 2003 Aug;17(7):513-20.

Pinney SJ, Sangeorzan BJ Fractures of the tarsal bones. Orthop Clin North Am. 2001 Jan;32(1):21-33.

Kuo RS, Tejwani NC, Digiovanni CW, Holt SK, Benirschke SK, Hansen ST Jr, Sangeorzan BJOutcome after open reduction and internal fixation of Lisfranc joint injuries. J Bone Joint Surg Am. 2000 Nov;82-A(11):1609-18.

Hunter JC, Sangeorzan BJA nutcracker fracture: cuboid fracture with an associated avulsion fracture of the tarsal navicular. AJR Am J Roentgenol. 1996 Apr;166(4):888.

Sangeorzan BJ, Swiontkowski MF Displaced fractures of the cuboid. J Bone Joint Surg Br. 1990 May;72(3):376-8.

Sangeorzan BJ, Veith RG, Hansen ST Jr. Salvage of Lisfranc's tarsometatarsal joint by arthrodesis. Foot Ankle. 1990 Feb;10(4):193-200.

Sangeorzan BJ, Benirschke SK, Mosca V, Mayo KA, Hansen ST Jr. Displaced intra-articular fractures of the tarsal navicular. J Bone Joint Surg Am. 1989 Dec;71(10):1504-10.

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Deformities of the Child's Foot

- Deformities of the child's foot are common:
  - Surgeons at the University of Washington Department of Orthopaedics and Sports Medicine have been discovering and applying effective methods for managing these deformities to optimize the chances for the child to develop a more normal foot.
  - Foot deformities may be congenital (genetically programmed and present at birth) or developmental (caused by nerve or muscle disorders and develop over time).
  - Sometimes the foot appears to be deformed, but the 'abnormal' appearance is only an anatomic variation that corrects spontaneously as the child matures. Identifying these variations is important to avoid over-treatment.
  - If surgery is required to manage the deformity, the distortions in each segment of the foot must be carefully defined and the treatment based on sound biological and biomechanical principles.
- Clubfoot is the most common congenital foot deformity:
  - Until very recently, the standard approach to clubfoot deformity correction was the application of a series of partially effective stretching casts, followed by extensive surgery in the infant. This treatment resulted in a foot that was stiff and became painful over time.
  - The present international standard treatment approach - the Ponsetti Method - is a series of 4-8 casts followed by simple, minimally invasive release of the Achilles tendon. This method results in a flexible, strong, well corrected foot that remains comfortable and functional for decades.
- Flexible flatfoot (FFF) is the normal shape of the foot in most babies and at least 20% of adults. It rarely, if ever, causes pain or functional disability and should, therefore, be considered an anatomic variation rather than a deformity:
  - The longitudinal arch of the foot increases in height spontaneously in most children during the first 10-12 years of life.
  - There is no evidence that special 'orthopaedic' shoes, orthotics, or any other intervention can create or elevate the arch in a child's foot.
  - FFF with a short, or contracted, Achilles tendon accounts for approximately 25% of FFF in adolescents and adults. This combination of deformities can cause pain and functional disability.
  - Joint preserving surgery that corrects the flatfoot shape and lengthens the Achilles tendon is used to relieve the pain and improve function.
- Cavus foot deformity refers to a longitudinal arch that is higher than normal. It frequently causes pain and functional disability:
  - Cavus is the result of a nerve or muscle disorder in almost all cases and is, therefore, often progressive (gets worse with time).

- Treatment for the pain and disability of cavus foot requires surgery that addresses both the shape abnormalities and the unbalanced muscle forces.

The height of the longitudinal arch of the foot has been a concern for parents and grandparents for generations. The presumed association of a flatfoot and pain has been implied rather than proven, yet this fear has generated an industry for treating what we now know to be an anatomic variation in foot shape. Lynn Staheli of the Department of Orthopaedics and Sports Medicine at the University of Washington School of Medicine and at Children's Hospital and Regional Medical Center, produced clinical and radiographic analyses of foot shapes in normal children, adolescents, and adults. He showed that the average (and normal range of) arch height is lower in babies than in older children and adolescents, the arch height increases spontaneously in most children, and flatfoot is in the normal range of arch heights for individuals of all ages. This normative data was used by others to show that special shoes and orthotics do not have an effect on the development of the longitudinal arch of the foot. Staheli showed that money could, therefore, be saved and negative long term psychologic impact could be avoided by resisting the temptation to use ineffective special shoewear for normal anatomic variations.

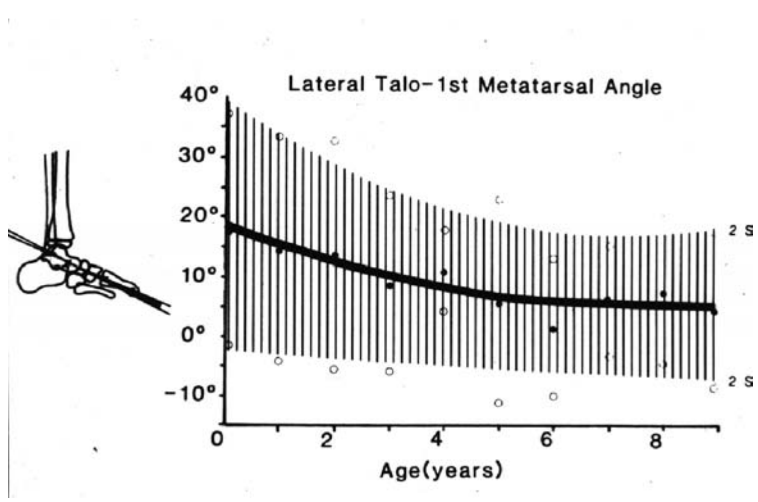

Figure 1: From: Vanderwilde R, Staheli LT, Chew DE, Malagon V: Measurements on radiographs of the foot in normal infants and children. J Bone Joint Surg 1988;70A:407-415.

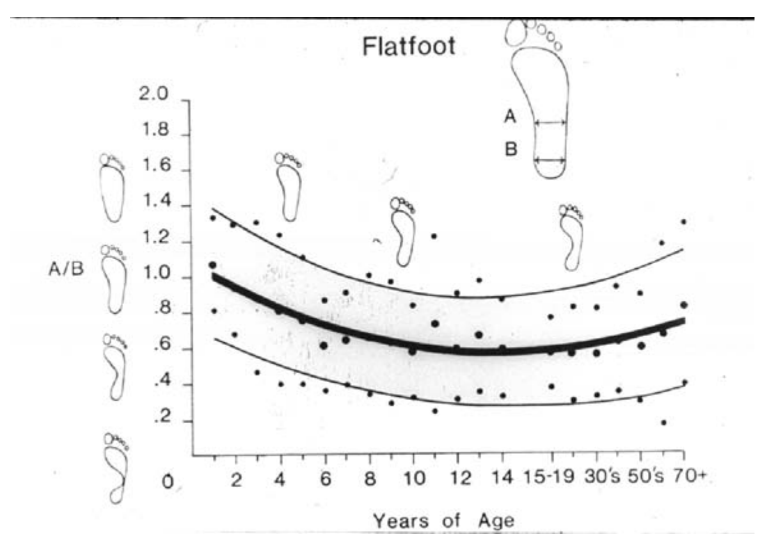

Figure 2: From: Staheli LT, Chew DE, Corbett M: The longitudinal arch. A survey of eight hundred and eighty-two feet in normal children and adults. J Bone Joint Surg 1987;69A:426-428.

It has been reported that approximately 20% of adults have flexible flatfeet and that these should be considered the normal shape of strong, stable feet. They do not cause pain or functional disability and do not need surgery. Some flexible flatfeet in adolescents and adults are associated with contracture, or shortening, of the Achilles tendon. Many of these will cause pain and disability. Surgery is indicated to treat these symptomatic feet. Many surgical procedures have been reported to treat flatfeet, but most reports do not provide strict indications for surgery or critical evaluation of the results over time. Lengthening of the lateral column of the foot by means of osteotomy of the anterior calcaneus was reported by a Welshman named Evans, in 1975, as a way to correct flatfoot deformity. Indications were not reported and the surgical description was terse, which often led to poor results by those who attempted the procedure. However, a long term follow-up article on Evans' patients indicated that the concept was correct. Vincent Mosca from the Department of Orthopaedics & Sports Medicine at the University of Washington School of Medicine and at Children's Hospital and Regional Medical Center, studied Evans' concept in the laboratory as well as in his patients. He developed the concept into a defined technique, reported his early clinical results, and has published clear and detailed descriptions of his interpretation of the technique for reliable outcomes by those who follow the outlined steps. Since Mosca's initial publication in 1995, the calcaneal lengthening osteotomy has become the standard for surgical correction of flatfoot deformity in the USA and in many parts of the world. His recognition of the need to surgically address the Achilles tendon contracture and the forefoot supination deformity concurrently has improved surgical outcomes for

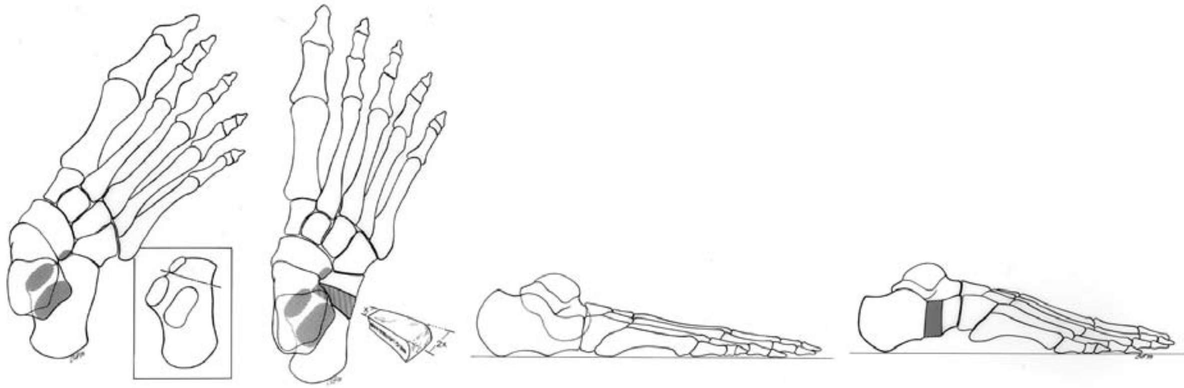

Figure 3: From: Mosca VS. Calcaneal lengthening for valgus deformity of the hindfoot. Results in children who had severe, symptomatic flatfoot and skewfoot. J Bone Joint Surg 1995;77A:500-512.

children. Reports from other centers around the world have confirmed the efficacy of his approach.

Vince Mosca has expanded the understanding and clarification of flatfoot deformity to an understanding and clarification of all deformities of the child's foot. His editorial in the Journal of Pediatric Orthopedics (JPO) on "The Child's Foot: Principles of Management" has become the foundation and reference for orthopedic surgeons to begin their own understanding of these principles. His subsequent JPO editorial on "The Cavus Foot" set the stage for understanding the principles for evaluation and management of this most complex foot deformity in children.

Mosca reported the first successful treatment for a rare, and occasionally symptomatic, deformity called skewfoot that is based on his principles of management. His soon to be published report on calcaneal lengthening for management of advanced rigid flatfoot deformity (do to tarsal coalition) describes an important change in the paradigm for managing this condition. His principle-based treatment of the acquired dorsal bunion deformity will also soon be published.

Clubfoot is the most common congenital foot deformity. The etiology is most likely multi-factorial, meaning that both genetic and environmental factors determine the development of the deformity. Elucidation of these factors is important. Vince Mosca and epidemiologists at the University of Washington reported a higher incidence of clubfoot in mothers who smoked cigarettes during their pregnancy than matched controls who did not smoke. Even more significant was the fact that there was a dose effect, with higher risk directly related to a greater number of cigarettes smoked per day. The same team of investigators found and reported a higher incidence of clubfoot in families with greater than average ligament laxity. Finally, Dr. Mosca and colleagues are completing a study that shows a consistently greater degree of femoral anteversion in a limb with a clubfoot than in a limb with a normal foot. The significance of this finding is forthcoming, but includes normative information about other parts of a limb with a clubfoot, and might include a genetic association that relates to the etiology of clubfoot deformity.

Future Work

Led by Dr. Mosca, the pediatric foot team of the Department of Orthopaedics and Sports Medicine at the University of Washington School of Medicine and at Children's Hospital and Regional Medical Center continues to discover and implement principles-based evaluation and management

Figure 4: Right foot appearance 6 months after a calcaneal lengthening osteotomy and Achilles tendon lengthening. Both feet looked like the unoperated left foot before the surgery on the right. Pain has been relieved and function improved. (From the private collection of Dr. Vincent Mosca.)

of foot deformities in children. His forthcoming book will describe these principles and the relevant surgical procedures.

Recommended Reading

Staheli LT, Chew DE, Corbett M: The longitudinal arch. A survey of eight-hundred and eighty-two feet in normal children and adults. J Bone Joint Surg 1987;69A:426-428.

Vanderwilde R, Staheli LT, Chew DE, Malagon V: Measurements on radiographs of the foot in normal infants and children. J Bone Joint Surg 1988;70A:407-415.

Driano AN, Staheli L, Staheli LT. Psychosocial development and corrective shoewear use in childhood. J Pediatr Orthop 1998;18:346.

Sangeorzan BJ, Mosca V, and Hansen ST Jr. The effect of calcaneal lengthening on relationships among the hindfoot, midfoot, and forefoot. Foot Ankle 1993;14:136-141.

Mosca VS. Skewfoot deformity in children: correction by calcaneal neck lengthening and medial cuneiform opening wedge osteotomies. J Pediatr Orthop 1993;13:807.

Mosca VS. Calcaneal lengthening for valgus deformity of the hindfoot. Results in children who had severe, symptomatic flatfoot and skewfoot. J Bone Joint Surg 1995;77A:500-512.

Mosca VS. Flexible flatfoot and skewfoot. An Instructional Course Lecture, The American Academy of Orthopaedic Surgeons. J Bone Joint Surg 1995;77A:1937-1945.

Niki H, Staheli LT, Mosca VS. Management of clubfoot deformity in amyoplasia. J Pediatr Orthop 1997;17:803-807.

Warme WJ, Mosca VS, Conrad EU. Comparison of structural bone allografts and autografts in pediatric foot surgery. Orthop Trans 1997;21(1):69.

Mosca VS. The Child's Foot: Principles of Management [editorial]. J Pediatr Orthop 1998;18:281-282.

Cappello T, Mosca VS. Metatarsus Adductus and Skewfoot. Foot Ankle Clin 1998;3:683-700.

Mosca VS. The Foot. In: Morrissy RT and Weinstein SL, editors. Lovell and Winter's Pediatric Orthopedics. 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2001;1151-1215.

Mosca VS. The Cavus Foot [editorial]. J Pediatr Orthop 2001;21:423-424.

Skelly AC, Holt VL, Mosca VS, Alderman BW. Talipes equinovarus and maternal smoking: a population-based case-control study in Washington state. Teratology 2002;66:91-100.

Olshan AF, Schroeder JC, Alderman BW, Mosca VS. Joint laxity and the risk of clubfoot. Birth Defects Research (Part A): Clinical and Molecular Teratology 2003;67:585-590.

DuMontier TA, Falicov A, Mosca V, Sangeorzan B. Calcaneal lengthening: Investigation of deformity correction in a flatfoot model. Foot Ankle Int 2005;26:166-170.

Skaggs DL, Flynn JM, Mosca VS, Noonan K. Foot problems in children. In: Skaggs DL and Flynn JM, editors. Staying Out of Trouble in Pediatric Orthopaedics. Philadelphia: Lippincott Williams & Wilkins, 2006;352-369.

Mosca VS. Flatfoot. In: Staheli LT, editor. Pediatric Orthopaedic Secrets. 3rd ed. Philadelphia: Mosby Elsevier, 2007;296-299.

Mosca VS. Cavus foot. In: Staheli LT, editor. Pediatric Orthopaedic Secrets. 3rd ed. Philadelphia: Mosby Elsevier, 2007;300-303.

Mosca VS, Bevan W. Calcaneal lengthening osteotomy for talocalcaneal tarsal coalition. J Child Orthop 2007;1(supp1):S24.

Mosca VS. Calcaneal lengthening osteotomy for valgus deformity of the hindfoot. In: Skaggs DL and Tolo VT, editors. Master Techniques in Orthopaedic Surgery: Pediatrics. Lippincott Williams & Wilkins, 2008 (in press).

Mosca VS, Ryan DD, Dias L. Dorsal bunion: a new corrective procedure. (in progress).

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- Musculoskeletal conditions are the #1 reason patients visit doctors. Over 28 million new cases of musculoskeletal impairment are reported each year. One out of 7 Americans is affected with a musculoskeletal condition. Musculoskeletal conditions cost an estimated $254 billion every year.
- Individuals with musculoskeletal conditions usually present themselves first to primary care physicians.
- Family medicine, primary care internal medicine and primary care pediatrics are the most common specialties chosen by medical students.
- Many primary care physicians are uncomfortable with the evaluation and ambulatory management of common conditions such as frozen shoulder, recurrent ankle sprains, or low back pain.
- The Department of Orthopaedics and Sports Medicine in concert with the Departments of Biological Structure, Physical Medicine and Rehabilitation, Family Medicine, and Internal Medicine have been national leaders in assuring that our medical students have a foundation in musculoskeletal medicine.
- Our research in musculoskeletal medicine education has helped us discover knowledge gaps and define curricular changes to better prepare students for careers in primary care medicine with enhanced competency in musculoskeletal medicine.

In the past, medical students were taught anatomy and physiology in the early part of their education and clinical application and skills in the later part. The University of Washington School of Medicine has taken a lead in the national movement towards an system based education to bridge the gap between early basic science instruction and the clinical instruction of the latter years of medical school training. The first subject for this integrated education at U.W. was musculoskeletal medicine. In the Winter of 1969, Chairman D. Kay Clawson of the Department of Orthopaedics and Cornelius Rosse of the Department of Biological Structure launched the Musculoskeletal Core Course, which was to become a flagship for the system-based approach. Course faculty included Peter Simkin of Rheumatology and Walter Stolov of Rehabilitation Medicine among others. Together Rosse and Clawson authored "Introduction to the Musculoskeletal System" in 1970, the text used to guide medical students through their introduction to the musculoskeletal system. Course emphases included basic science of connective tissues, applied anatomy, infections, fractures, neoplasia, and arthritis. Initially, dissections were not a part of this class, so students had to sign up for an elective in anatomy of the extremities with Dr. Dan Graney if they wanted to participate in dissection. When 90% of students were found to be signing up for the electives, the School of Medicine restored the hours necessary to include dissection in the musculoskeletal course. The system-based curriculum came to include a study of the musculoskeletal system built upon the anatomy, histology, physiology, rheumatology, and clinical vignettes including an introduction to physical exam.

The multi-state Washington, Alaska, Montana, Idaho system of medical education, or WAMI program, began in the early 1970's (Wyoming was added to the WAMI program, now WWAMI, in 1996.) In WWAMI, medical students received a substantial part of their education away from Seattle. So that all could benefit, the musculoskeletal core course was moved to the second year when all students returned Seattle. In 1980, Clawson and Rosse authored their second book on musculoskeletal medicine: "The Musculoskeletal System

Lisa practices her knee exam on classmate Jillian during one of four Living Anatomy sessions of the Musculoskeletal Core course.

in Health and Disease." New course emphases included applied anatomy of the spine and extremities, a study of gait, and common musculoskeletal and neuromuscular conditions.

The anatomy component of the course was turned over to Dr. Graney in the mid-1980's, and with the departure of Dr. Clawson, the clinical leadership moved initially to Dr. Walter Stolov of Rehabilitation Medicine, then back to the Dept. of Orthopaedics with Dr. Ted Greenlee, and Dr. Fred Lippert. The course included faculty in Orthopaedics, Rheumatology and Rehabilitation Medicine. From the 1983 through 2002, Dr. Carol Teitz of Orthopaedics and Sports Medicine chaired the course with Dr. Graney of Biological Structure. Major changes to the design of the course also included the integration of a Problem Based Curriculum in 1990, designed by Dr. Teitz of Orthopaedics and Dr. Bill Hammond of Hematology along with Dr. Roberta Pagon from Genetics and Dr. Peter Simkin from Rheumatology. In this innovation, a group of students is given a hypothetical clinical problem, for example a 67 year old person with shoulder pain and a fever, and challenged to muster the resources for the evaluation and management of this 'patient'. This component of the course was so successful that it became its own required course in the second year curriculum in 1997. Drs. Lippert and Teitz also wrote a textbook that was used for this course in its early years: "Diagnosing Musculoskeletal Problems. A Practical Guide."

In 1996, the Musculoskeletal Core Course was the first in the medical school to offer on-line resources (courses.washington.edu/hubio553/). The websites, designed by Carol Teitz and Michael Richardson, continue to provide great learning tools, including the musculoskeletal atlas (depts.washington.edu/msatlas/), the online musculoskeletal cases (courses.washington.edu/hubio553/cases/), "Totally Rad" (courses.washington.edu/hubio553/totrad/SCAR/index.html), "Son of Rad" (courses.washington.edu/hubio553/totrad/RadReference/index.html) and the multimedia musculoskeletal machine, or glossary (courses.washington.edu/hubio553/glossary/index.html). If you haven't visited these sites, you should!

In the early 1980s Drs. John Olerud and Steve Rice, then in the Orthopaedics Department Sports Medicine Division, began an elective in Sports Medicine for 2nd year medical students. Dr. Teitz's text "Scientific Foundations of Sports Medicine," was a regular resource for this course.

Third and fourth year students interested in musculoskeletal medicine and orthopaedics were traditionally enrolled in clerkships that primarily exposed them to in-patient services and orthopaedic surgery. Recognizing that the greatest benefit to most students would come from an outpatient or clinic experience, Dr. Teitz along with Drs. John Olerud and Steve Rice inaugurated an ambulatory rotation in Orthopaedics and Sports Medicine in the 1980's. This elective became a favorite elective among third and fourth year students. Now medical students at the U.W. have their choice of many different electives for clinical training in Orthopaedics and Sports Medicine, ranging from trauma to tumor, sports to spine, and pediatrics to adult reconstruction: www.washington.edu/students/crscat/orthop.html.

Using modern research methods, we have discovered that many challenges remain in preparing young physicians for the world in which musculoskeletal disorders are so common and so important. Despite generally improved levels of competency with each year at medical school, less than 50% of fourth-year students could demonstrate knowledge of basic facts and concepts in musculoskeletal medicine. Students who completed a musculoskeletal clinical elective scored higher and were more competent (78%) than students who did not take an elective. Thus the curricular approach toward teaching musculoskeletal medicine seems to be insufficient unless it is reinforced during the clinical years.

In another investigation, we evaluated the musculoskeletal knowledge and self-perceived confidence of fully trained, practicing primary care physicians. These physicians demonstrated greater confidence with medical issues (such as diabetes and heart disease) than with musculoskeletal issues. We discovered that a majority of primary care providers tested failed to demonstrate adequate musculoskeletal knowledge and confidence. Specifically, 64% of ninety-two practicing primary care physicians scored less than 70% on their musculoskeletal knowledge.

The Future

The impact of musculoskeletal conditions on the world's population is huge and growing. These conditions are now a primary cause of disability worldwide. As heart disease, cancer, and infectious diseases are being brought under better control, the impact of bone and joint problems continues to grow.

Katie examines classmate Abe for "too many toes" during a Living Anatomy session in the second year HuBio Musculoskeletal Core course.

The proper evaluation and management of individuals with these conditions requires an increased emphasis on the education of all physicians in musculoskeletal medicine. The faculty of the Department of Orthopaedics and Sports Medicine are committed to this education for medical students as well as for the primary care physicians who usually have the first opportunity to evaluate these patients.

Special thanks go to Drs. Carol Teitz, Dan Graney, Ted Greenlee, and Cornelius Rosse for their help in drawing up this short history.

References

Introduction to the Musculoskeletal System. Cornelius Rosse, MD and D. Kay Clawson, MD. Harper & Row, New York, 1970.

The Musculoskeletal System in Health and Disease. Cornelius Rosse, MD and D. Kay Clawson, MD. Harper & Row, Hagerstown, Maryland,1980.

Diagnosing Musculoskeletal Problems. A Practical Guide. Frederick G. Lippert, MD and Carol C. Teitz, MD. Williams & Wilkins, Baltimore, 1987.

Scientific Foundations of Sports Medicine, Carol C. Teitz, MD. Mosby-Year Book, Philadelphia, 1989.

More Evidence of Educational Inadequacies in Musculoskeletal Medicine. Gregory A. Schmale MD. Clinical Orthopaedics & Related Research. 437:251-259, August 2005.

Important Demographic Variables Impact the Musculoskeletal Knowledge and Confidence of Academic Primary Care Physicians. Joseph R. Lynch, MD1, Gregory A. Schmale, MD1, Douglas C. Schaad, PhD1 and Seth S. Leopold, MD1.The Journal of Bone and Joint Surgery (American). 2006;88:1589-1595.

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Discoveries in Hand Surgery: Smaller Operations, Better Results

- Carpal tunnel syndrome is a very common disease causing numbness and tingling in the fingers.
  - Patients often report a “pins and needles” sensation in their index and middle fingers. It commonly wakes them up at night.
  - Traditionally, the surgical treatment of carpal tunnel syndrome has been with a large open incision in the palm.
  - At the University of Washington Hand Center we have worked hard to perfect a minimally invasive surgery for the treatment of carpal tunnel syndrome.
  - Endoscopic surgery for carpal tunnel syndrome involves only a minimal incision at the wrist and provides a more rapid return to use of the hand.
- Arthritis of the base of the thumb is another common condition that affects our patient’s hands.
  - It can be a painful, often debilitating disease that prevents patients from using their hands normally.
  - At the University of Washington Hand Center we have developed a new form of joint replacement for thumb arthritis.
  - Our studies have shown that patients are very satisfied with this procedure.

The hand surgery team at the University of Washington strives to develop less invasive and more effective techniques for treatment of common hand problems such as arthritis and carpal tunnel syndrome. Our research ranges from studies of the basic mechanics and biology of the hand to clinical research evaluating the efficacy of different treatment methods. Recently we have focused on two of the most common and disabling problems of the hand: carpal tunnel syndrome and arthritis at the base of the thumb.

Endoscopic Surgery for Carpal Tunnel Syndrome

Carpal tunnel syndrome is a very common condition causing numbness and weakness of the hands. It effects both women and men between the ages of 30 -70 years. Patients complain of numbness of the hand that often wakes them from sleep and of weakness of the thumb. This condition is caused by compression of the median nerve as it travels through a tight tunnel at the wrist. Once established, the condition rarely improves without treatment. Patients may experience relief of symptoms with nighttime bracing or steroid injections into the carpal tunnel. The surgical treatment of carpal tunnel syndrome involves opening up of the tight tunnel by cutting the ligament that encloses it. Traditionally, the surgical treatment involved a large incision in the palm. The main problem with this approach is that the scar tissue can be quite sensitive resulting in a prolonged recovery time. We have learned that a minimally invasive carpal tunnel release can be accomplished using a small lighted tube known as an endoscope inserted through a very small incision at the wrist. When connected to a video camera, the endoscope can safely guide small surgical instruments that cut the ligament while protecting the nerve that is millimeters away. Our initial trial results were very successful. In 1996 we embarked on a second trial to evaluate the safety and effectiveness of endoscopic carpal tunnel surgery. We performed a prospective randomized trial of 47 patients, comparing the results of the traditional open carpal tunnel surgery with endoscopic

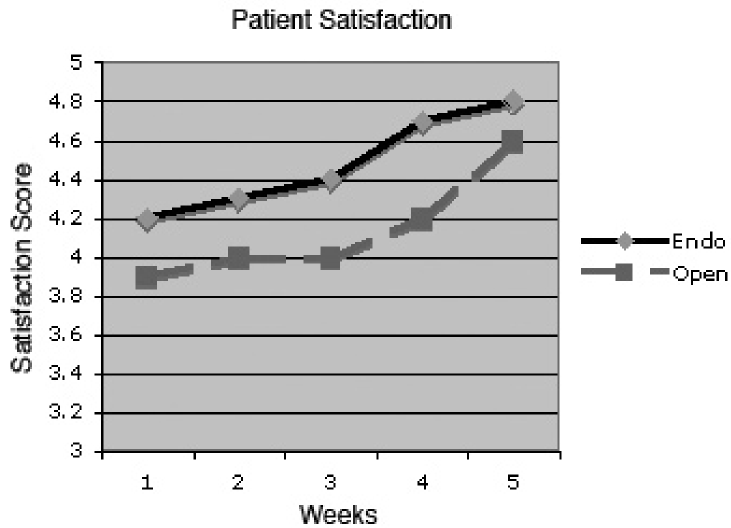


Figure 1: Patient satisfaction scores after endoscopic and open carpal tunnel release.

Figure 2: X-ray of a patient with thumb arthritis. The double red arrows point to the joint between the thumb metacarpal and the trapezium. Notice the lack of joint space, indicating significant arthritis. The white arrow points to the joint between the trapezium and the scaphoid. Notice how the joint space is preserved, there is no involvement of arthritis at this joint.

Figure 3: Post-operative x-ray of a patient after replacement of the basal joint with a costochondral allograft. The allograft cartilage is not visible on x-ray. However, note the thumb has maintained its normal alignment.

carpal tunnel release. In the first 3 months after surgery the patients who underwent endoscopic surgery were significantly more satisfied with their surgery and had greater grip strength, pinch strength and hand dexterity than those patients treated with an open incision. The scar tenderness was also dramatically decreased in the endoscopic group (Figure 1). We continue to focus on ways to improve our patient's satisfaction after carpal tunnel surgery.

Thumb Arthritis

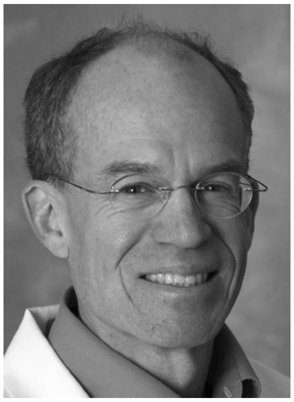
Thumb arthritis, also referred to as basal joint arthritis, is a common problem that affects mostly women between the ages of 50 and 70. The arthritis involves the bones at the base of the thumb, the trapezium, the metacarpal and the scaphoid (Figure 2). Patients with basal joint arthritis experience an achy pain at the base of their thumb, usually made worse with any pinching or gripping activity. Traditional treatment of this problem involves removing the trapezium and reconstructing the ligaments at the base of the thumb with a tendon taken from the patient's wrist. We have noticed that over time however, the tendon loosens and the thumb begins to subside. We have discovered an improved surgical procedure that involves removing only the arthritic portion of the trapezium and replacing it with a piece of allograft cartilage obtained from our tissue bank (Figure 3). Our patients have been very satisfied with this technique. And we have found less subsidence of the thumb over time. We continue to perfect this technique of thumb joint replacement.

The U.W. Hand Center continues to pursue move effective and less invasive approaches to the restoration of hand comfort and function.

References

Trumble, T. E.; Gilbert, M.; and McCallister, W. V.: Endoscopic versus open surgical treatment of carpal tunnel syndrome. Neurosurg Clin N Am, 12(2): 255-66, 2001.

Trumble, T. E.; Rafijah, G.; Gilbert, M.; Allan, C. H.; North, E.; and McCallister, W. V.: Thumb trapeziometacarpal joint arthritis: partial trapeziectomy with ligament reconstruction and interposition costochondral allograft. J Hand Surg [Am], 25(1): 61-76, 2000.



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Wrist Injuries: A Common and Serious Problem

- The wrist is one of the most complex joints in the body, including a total of ten bones and the ligaments joining them (Figure 1).
- Any one of these bones can be broken, and any one of the ligaments stabilizing these bones can be torn.
- Often these injuries occur in young, active persons who do not initially realize the severity of their wrist injury, and who do not seek medical attention until their pain does not improve.
- If fractures or complete ligament tears are diagnosed early, it may be possible to repair them directly and achieve a good outcome, with nearly normal wrist function.
- If left untreated, fractures or ligament injuries in the wrist can result in bones moving out of position and eroding wrist cartilage. This condition, known as post-traumatic arthritis, generally requires more extensive treatment than simple bone or ligament repair.
- Treatment for post-traumatic wrist arthritis may include removal of the arthritic bones or surgically fusing some of the bones together. Fusion may reduce the pain, but also reduces the motion of the wrist.
- We have discovered that early diagnosis and prompt treatment of wrist injuries are the keys to restoring comfort and function to the injured wrist.

Faculty of the Department of Orthopaedics and Sports Medicine are making major contributions to the evaluation and management of wrist injuries. We are focusing particular attention on an injury known as scapholunate dissociation. In this injury the ligament connecting two small wrist bones, the scaphoid and the lunate, is torn-often due to a fall on the outstretched hand. Normally, these bones are the link between the long bones of the forearm and the small bones of the hand. They undergo a tremendous amount of motion as the wrist goes through its various changes in position, from flexion to extension, palm down to palm up (pronation to supination), and side to side movement (radial and ulnar deviation). A ligament (the scapholunate interosseous ligament) stabilizes these two very mobile bones so that they stay seated in their socket at the end of the forearm bone (the radius) despite their great mobility. Injury to this ligament results in instability, so that the scaphoid bone moves in and out of joint, and the cartilage of this ball-and-socket is ground down (Figure 2). Persons with this condition experience popping of their wrist as the bones move out of position with motion. Once cartilage damage occurs, the exposed bone surfaces grind against one another, often causing pain and further limiting motion.

Scapholunate dissociation is diagnosed on physical examination and with various imaging studies such as plain radiograph (x-ray) (Figure 3) and MRI.

We have discovered that for patients with acute and complete scapholunate ligament rupture, primary repair of the tear may be sufficient. If additional stability is required, the surgeon can reinforce the repair with a flap of wrist capsule. When a new ligament needs to be constructed, a ligament graft with segments of bone graft at each end or by weaving a tendon through the scaphoid and lunate (Figure 4).

Conclusion

The surgeons at the University of Washington have developed a rational approach to persons with scapholunate injuries, emphasizing that early diagnosis and appropriate

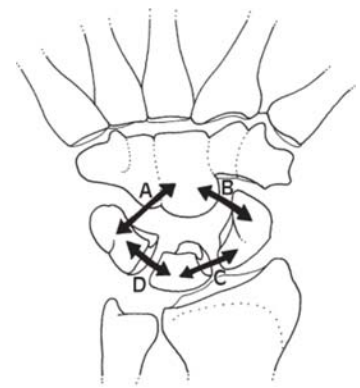

Figure 1: Normal wrist anatomy; motion occurs at sites A,B,C,D. C = Scapholunate interosseous ligament (from Green's Operative Hand Surgery).

treatment provide the best opportunity to restore wrist stability and minimize the long-term risk of wrist arthritis.

We continue to seek better methods for managing severe and chronic ligament injuries of the wrist. We are exploring entirely new strategies using our Department's expertise in the cutting-edge fields of tissue engineering and regenerative medicine. One such strategy in the early phase of development involves the use of mesenchymal stem cells to seed a biosynthetic scaffold held in place between the scaphoid and lunate, with the goal being a more biological restoration of the three-dimensional anatomy of the normal ligament and normal, stable and comfortable function of the wrist.

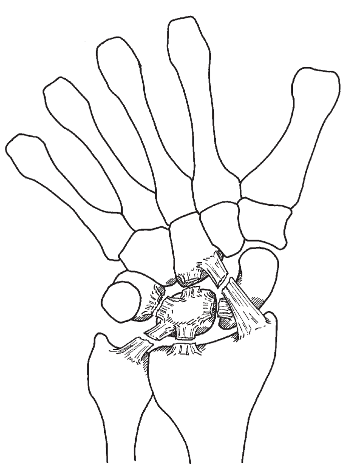

Figure 2: Disruption of scapholunate interosseous ligament (Trumble TE, Principles of Hand Surgery and Therapy, 2000).

Figure 3: Radiographic findings after SLIL injury: scaphoid and lunate bones no longer held together; early arthritis of joint between scaphoid and radius (Trumble TE, Principles of Hand Surgery and Therapy, 2000).

Acknowledgments

We gratefully acknowledge support from the Roberts Helping Hand Fund and the National Science Foundation through the University of Washington Engineered Biomaterials. These sources have made possible much of the Department's research in tissue engineering and regenerative medicine for hand and wrist injuries.

References

Allan CH, Trumble TE: Scapholunate Instability (chapter); AAOS Monograph: Carpal Fracture-Dislocations; Rosemont, IL 2002.

Coe M, Spitellie P, Trumble TE, Tencer AF, Kiser P.: The scapholunate allograft: a biomechanical feasibility study. J Hand Surg [Am]. 1995 Jul;20(4):590-6.

Harvey EJ, Hanel DP.: Bone-Ligament-Bone Reconstruction for Scapholunate Disruption. Tech Hand Up Extrem Surg. 2002 Mar;6(1):2-5.

Harvey EJ, Hanel D, Knight JB, Tencer AF.: Autograft replacements for the scapholunate ligament: a biomechanical comparison of hand-based autografts. J Hand Surg [Am]. 1999 Sep;24(5):963-7.

Howlett JPC, Pfaeffle HJ, Waitayawinyu T, Trumble TE: Distal Tunnel Placement Improves Scaphoid Flexion With The Brunelli Tenodesis Procedure For Scapholunate Dissociation. Submitted Jan. 2008 J Hand Surg.

Knoll VA, Allan CH, Trumble TE: Trans-scaphoid Perilunate Fracture Dislocations. J Hand Surg [Am]. 2005 Nov;30(6):1145-52.

Trumble TE: Principles of Hand Surgery and Therapy. Saunders. 2000.

Trumble T, Verheyden J.: Treatment of isolated perilunate and lunate dislocations with combined dorsal and volar approach and intraosseous cerclage wire. J Hand Surg [Am]. 2004 May;29(3):412-7.

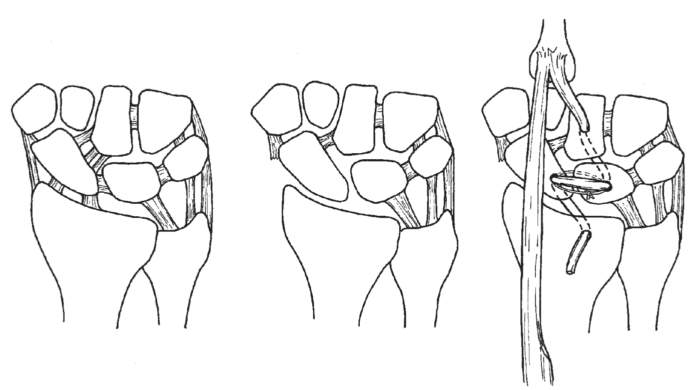

Figure 4: Tendon weave for scapholunate interosseous ligament disruption (Trumble TE, Principles of Hand Surgery and Therapy, 2000).

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New Insights into Fractures of the Elbow: The Biomechanics of Radial Head and Coronoid Fractures

- Fractures of the elbow often involve the upper ends of the forearm bones - the radius and the ulna.
- The upper end of the radius takes the form of a rotating joint surface called the radial head.
- The upper end of the ulna is in the form of a stabilizing concavity with the olecranon process at one end and the coronoid process at the other.
- The radial head and the coronoid process are primary bony stabilizing features of the elbow joint.
- Injuries of the radial head and coronoid process can lead to a painful and unstable elbow joint.
- The faculty of the Department of Orthopedics and Sports Medicine are discovering improved methods for evaluating, understanding and managing injuries to these key elements of the elbow joint.

The elbow joint is composed of three bones: the arm bone (humerus) and the two forearm bones (the radius and the ulna) (Figure 1). Each bone has important anatomic landmarks (Figure 2). The radial head is the upper end of the radius. The coronoid process forms the most anterior part of the socket in the upper end of the ulna. There are three important articulations of the elbow- the joint between the radial head and the humerus, the joint between the ulna and the humerus, and the joint between the radial head and the ulna. The radiohumeral joint and the radioulnar joint allow rotation of the forearm. The ulnohumeral joint allows flexion and extension (Figure 3).

Prominences on the inside and outside of the elbow (medial and lateral epicondyles of the humerus, respectively) are the origins of the medial and lateral collateral ligaments and forearm muscles. Elbow stability is provided by the soft tissue structures (muscles and ligaments) surrounding the joint as well as by the bony anatomy. Injury to any of these structures can lead to elbow instability.

Radial Head Fractures

Radial head fractures are common injuries and comprise one-third of all elbow fractures. They often arise from falls on the outstretched hand. Radial head fractures can occur in isolation or in conjunction with other injuries around the elbow. Untreated, they can lead to elbow instability, which ultimately may lead to arthritis and pain. Radial head fractures can be treated without surgery if they are not displaced, if they do not interfere with elbow motion and if they do not involve a significant portion of the joint (usually less than 1/3 of the diameter). When these conditions are not met, we have found that function is best restored by surgically aligning the fracture fragments and fixing them in place with screws or by replacement of the damaged radial head with a metallic prosthesis (Figure 4).

In the past, radial head fractures were often treated by removing the radial head altogether. Our biomechanical studies have demonstrated that radial head excision does not restore function as completely as the methods that restore the anatomy of the joint. In fact, contrary to previous beliefs, we found that even in the setting of intact ligaments, elbow biomechanics are adversely affected by radial head excision. As well, we found that even small displaced fracture fragments can adversely affect the function of the joint. Anatomical fixation of these small fragments helps to restore the joint's normal biomechanics. When severe radial head injury is accompanied by injuries to the ligaments of the elbow, we discovered that both a radial head replacement and ligament repair are required to restore elbow biomechanics to as close to normal as possible.

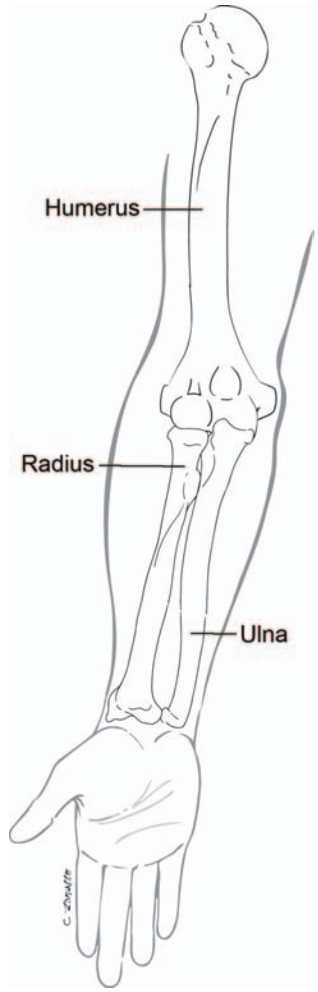


Figure 1: Anterior view of the right arm showing the three bones that constitute the elbow joint.

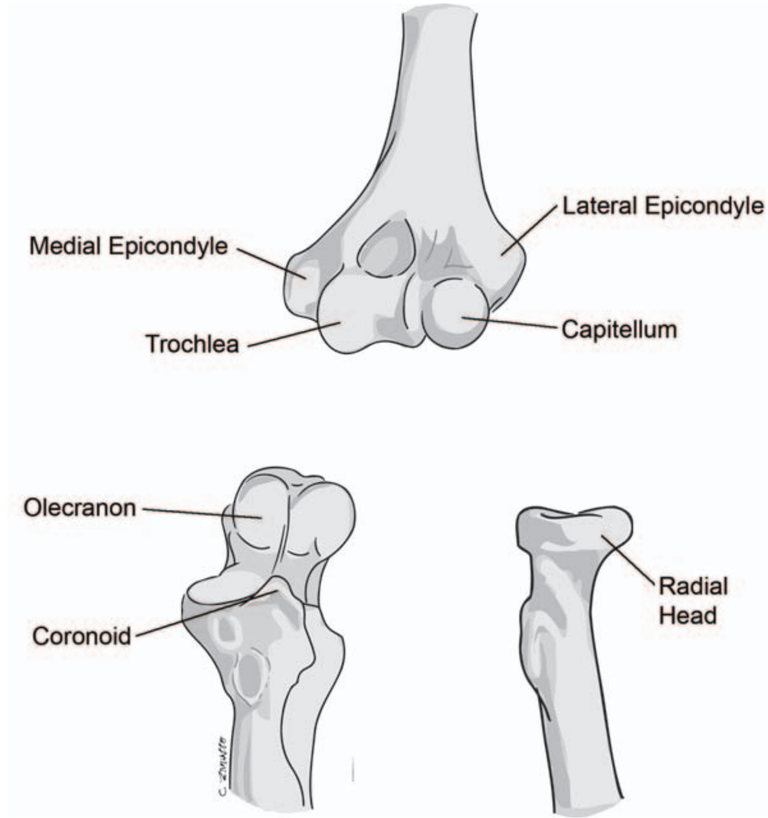


Figure 2: Anterior view of the distal humerus (top), proximal ulna (bottom left), and proximal radius (bottom right) showing the important anatomic structures around the elbow joint.

Coronoid Fractures

Fractures of the coronoid process are less common than radial head fractures. They are most commonly seen in conjunction with other elbow injuries, including elbow dislocations. Our research revealed that small coronoid process fractures can be treated non-operatively if they are not associated with elbow instability. Very small coronoid fractures with ligament injuries and instability can be effectively treated with ligament repair alone. Larger fractures require operative fixation with screws or sutures and repair of ligament injuries to restore elbow stability.

Conclusion

Our research has substantially increased the understanding of radial head and coronoid process injuries and the ligament injuries that often accompany them. Both of these bony structures are important to the stability of the elbow. Injury to them may result in instability of the joint which in turn can lead to pain, arthritis, and disability. Our current research is directed at learning whether subtle changes in the mechanics of the joint are likely to have long-term consequences. Finally, our clinical and laboratory research is continuing to explore new methods of fixation of these injuries that will more completely restore normal anatomy and function.

Financial Support

These studies were made possible by a grant from the Medical Research Council of Canada ($432,377). “Kinematics & Stabilizers of the Elbow”. Principal investigators: King GJW, Johnson JA.

References

Beingessner DM, Stacpoole RA, Dunning CE, Johnson JA, King GJW. The Effect of Suture Fixation of Type One Coronoid Fractures on the Kinematics and Stability of the Elbow With and Without Medial Collateral Ligament Repair. J Shoulder Elbow Surg, 16(2):213-17, 2007.

Beingessner DM, Stacpoole RA, Dunning CE, Johnson JA, King GJW. The Effect of Coronoid Fracture Size on Elbow Kinematics and Stability. Clin Biomech, 22:183-90, 2007.

Johnson JA, Beingessner DM, Gordon KD, Dunning CE, Stacpoole RA, King GJW. Kinematics and stability of the fractured and implant-reconstructed radial head. J Shoulder Elbow Surg, 14(1S): 195S-201S, 2005.

Beingessner DM, Dunning CE, Gordon KD, Johnson JA, King GJ. The Effect of Radial Head Fracture Size on Elbow Kinematics and Stability. J Ortho Res, 23(1): 210-17, 2005.

Beingessner DM, Dunning CE, Gordon KD, Johnson JA, King GJ. The Effect of Radial Head Excision and Arthroplasty on Elbow Kinematics and Stability. JBJS, 86A: 1730-1739, 2004.

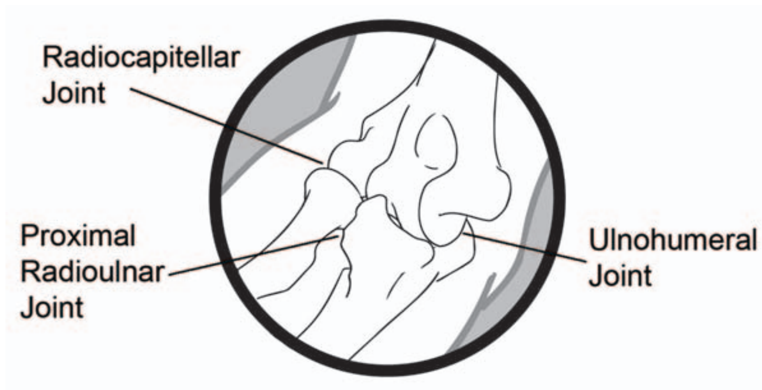


Figure 3: Anterior view of the right elbow joint demonstrating the three articulations.

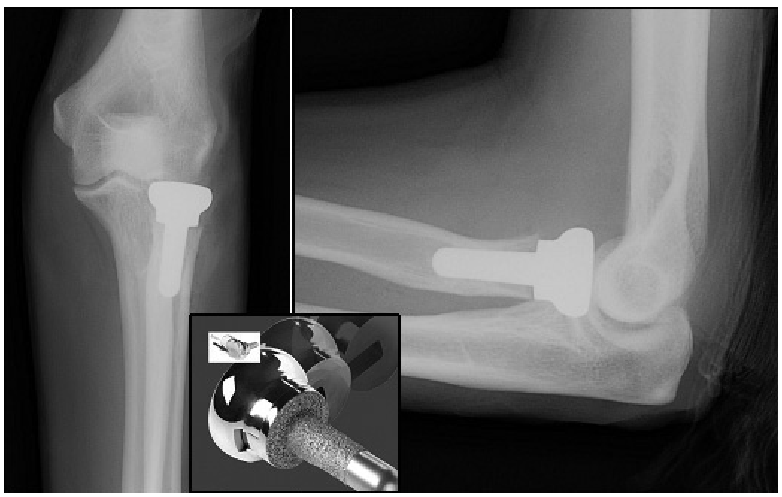
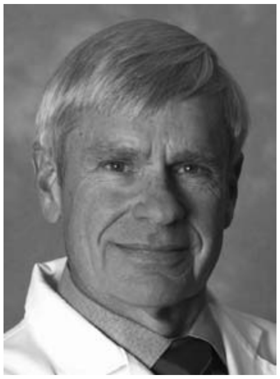


Figure 4: Anteroposterior and lateral views of the elbow demonstrating a radial head replacement. An example of a modular metallic radial head implant is shown (inset).

Beingessner DM, Dunning CE, Beingessner CJ, Johnson JA, King GJ. The Effect of Radial Head Fracture Size on Radiocapitellar Joint Stability. Clin Biomech. 18(7): 677-81, 2003.



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Discoveries in the Management of Shoulder Arthritis: New Solutions for Old Problems

- Shoulder arthritis is a disabling condition that prevents otherwise healthy individuals from sleeping, carrying out activities of daily living and from enjoying sports and other physical activities. In this condition, the normally smooth cartilage surfaces of the ball and socket of the shoulder are lost because of injury, degeneration, inflammation, or surgical misadventure. As a result, the joint loses the characteristics essential for normal function: flexibility, strength, stability and smoothness.
- As recently as fifty years ago, the treatment for shoulder arthritis was nothing more than exercises in which the patient leaned over swinging a heavy weight held in the hand to try to pull the damaged joint surfaces apart. The modern era of shoulder replacement began in the 1950's, when Charles S. Neer II introduced a smooth metal ball to replace the damaged joint surface with a stem to fix the ball to the arm bone.
- Since then, the art and science of joint replacement for the different types of shoulder arthritis has and continues to progress rapidly. Many of the key discoveries are taking place at the University of Washington, where a particular emphasis is placed on minimally invasive, biological approaches to joint reconstruction directed at the best possible return of the shoulder to comfort and function.

After two fellowships with Dr. Neer, Rick Matsen introduced the technique of shoulder joint replacement at the University of Washington in 1975. Since then, the U.W. Shoulder and Elbow team has played a major role in the ongoing development of more effective and less invasive approaches to the management of shoulder arthritis. In addition to Matsen, the team members have included the late Doug Harryman, John Sidles, Kevin Smith, Sarah Jackins and a legacy of outstanding shoulder fellows, residents and students. The team has recently been joined by Winston Warme and Alexander Bertelsen. In an important sense, the team also includes over 7000 individuals for whom we have performed shoulder reconstructions - these individuals have provided key observations on what works and what does not and especially on how to optimize the rehabilitation after shoulder replacement. A brief summary of some of our advances are listed below, along with the web sites where one can find more details.

Soft Tissue Management (www.orthop.washington.edu/totalshoulder)

We pursue the goal of minimally invasive shoulder replacement, which minimizes the amount of soft tissue dissection needed to perform the procedure. Presently, we perform shoulder replacement with the incision of only one tendon, the subscapularis, which is securely reattached at the conclusion of the procedure. The deltoid, rotator cuff and pectoralis muscles remain intact. An equally important goal is balancing the soft tissues of the shoulder to optimize both the mobility and stability of this joint. We achieve this by safely releasing motion-limiting contractures and scar tissue, while limiting excessive slack that may contribute to instability.

The Humeral Prosthesis (Replacement for the Ball of the Shoulder Joint) (www.orthop.washington.edu/totalshoulder)

An essential element of shoulder joint replacement is the insertion of a smooth metal ball to replace the joint surface damaged by arthritis. This requires proper component sizing, positioning and fixation. We have developed guidelines for selecting the proper ball dimensions to maximize range of motion and stability. The metal ball is attached to a stem that

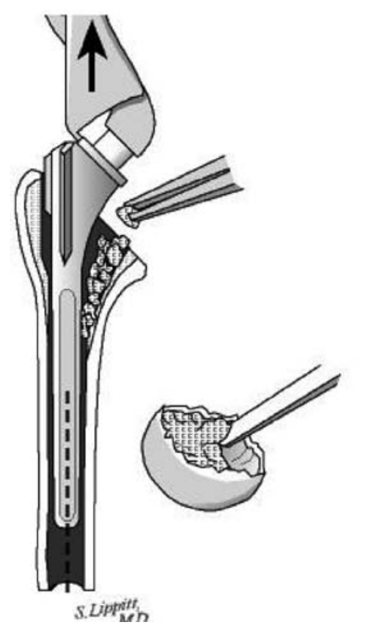


Figure 1: Impaction autografting. Bone harvested from the resected humeral head (right) is impacted into the canal of the humeral shaft providing safe and secure fixation of the humeral prosthesis.

must be secured safely and accurately to the inside of the arm bone. In the past, surgeons have relied on bone cement, which may crack and loosen. Other surgeons have tried to achieve a good fit with the prosthesis by reaming the inside of the bone to a tight fit, an approach that can weaken the bone, and predispose it to fracture. We have developed a method for using bone harvested from the patient's humerus to securely fix the prosthesis in the ideal position - a process known as impaction autografting (Figure 1). This procedure uses the patient's own bone material to support the implant, minimizing the risks of loosening and weakening the bone.

The Glenoid Prosthesis (Replacement for the Socket of the Shoulder Joint) (www.orthop.washington.edu/totalshoulder)

In total shoulder replacement arthroplasty, the plastic socket has traditionally been the weakest link. This is due in part to the difficulties in achieving durable fixation of the polyethylene prosthesis to the bone of the shoulder blade and due to deformation and wear of polyethylene from which the prosthesis is made. We discovered that using a socket with slightly greater diameter of curvature than that of the ball allowed for a greater range of motion and less risk to the rim of the component. We also learned that bone cement can damage the bone by its heat of curing. Thus we designed a system for fixation that uses precise preparation of the bone surface and fixation holes so that only minimal cement is required. We also developed a system for using carbon dioxide gas to clean and dry the bone for optimal cement fixation (Figure 2).

The Non-Prosthetic Glenoid Arthroplasty ("Ream and Run") (www.orthop.washington.edu/reamandrun)

Many of our more active patients with shoulder arthritis wish to avoid the risks associated with bone cement and a polyethylene glenoid socket prosthesis. These individuals encouraged us to pursue a solution that would enable them to return to unrestricted use of their shoulder. As a result we conducted laboratory research that demonstrated that the bone surface of the socket could heal over with a smooth layer of fibrous tissue and fibrocartilage if it was optimally shaped by reaming (Figure 3). We learned that early motion is the key to inducing the bone to heal and remodel to a durable joint surface. For this reason, we nicknamed the procedure 'ream and run'. We now offer the ream and run to active patients who are dedicated to the rehabilitation program that is essential to its success. In most cases the results have been truly exciting, with some individuals returning to unlimited physical activity with their new shoulder (Figure 4).

Failure analysis

The University of Washington shoulder service welcomes patients who have had unsuccessful shoulder replacement surgery elsewhere. This experience has helped us develop both an understanding of the causes of failure and an approach to managing failed shoulder joint replacements. We have learned that shoulder joint replacement is more likely to fail when it has been performed by a

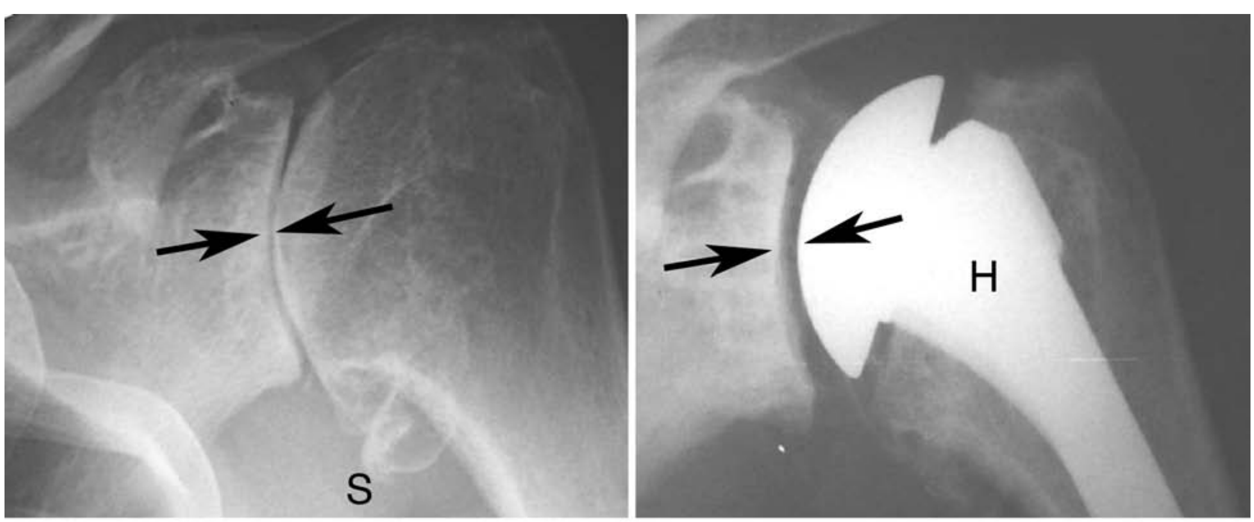


Figure 2: Total shoulder joint replacement arthroplasty. Arthritic shoulder (left) shows bone-on-bone contact (arrows) and bone spurs (S). Total shoulder (right) shows humeral component (H) contacting the dark space occupied by the plastic glenoid socket component (arrows).

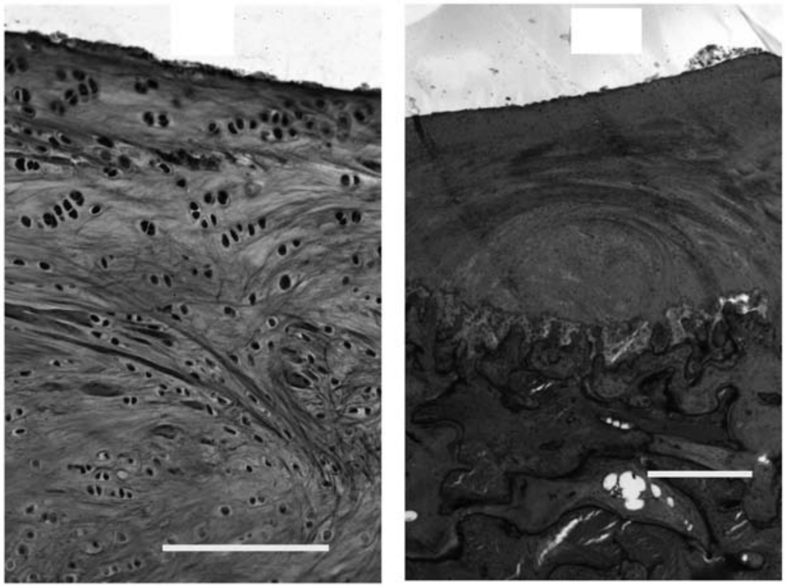


Figure 3: Regenerated biological joint surface 6 months after a ream and run procedure in a laboratory model. Note the smooth joint surface with living fibrocartilage tissue showing the black nuclei of the clear cartilage cells in a matrix of fibrous tissue (left). Note the arcades of fibers binding the regenerated tissue to the bone below (right).

surgeon who does this procedure infrequently. We have also learned that many individuals with painful shoulder joint replacements have previously unrecognized infections with low-virulence bacteria. With the collaboration of our infectious disease and pathology services, we are developing methods for preventing, diagnosing and managing these infections.

The Reverse Total Shoulder Arthroplasty (www.orthop.washington.edu/reverseshoulder)

Conventional approaches to shoulder joint replacement are ineffective when the shoulder is weak and unstable because of massive defects in the rotator cuff tendons. In these situations and in certain circumstances where conventional arthroplasty has failed, we have been able to restore substantial function with a reversed ball and socket prosthesis (Figure 5). We continue to discover improvements in this novel approach to shoulders where previously no reasonable options were available.

Conclusion

Now, over thirty years and over seven thousand shoulder joint replacements after our debut, the shoulder team at the University of Washington continues our pursuit of more effective and less invasive approaches to the management of shoulder arthritis. Our high-priority goals for the next few years are (1) to develop ways in which the ream and run procedure can be effectively applied to an increasing

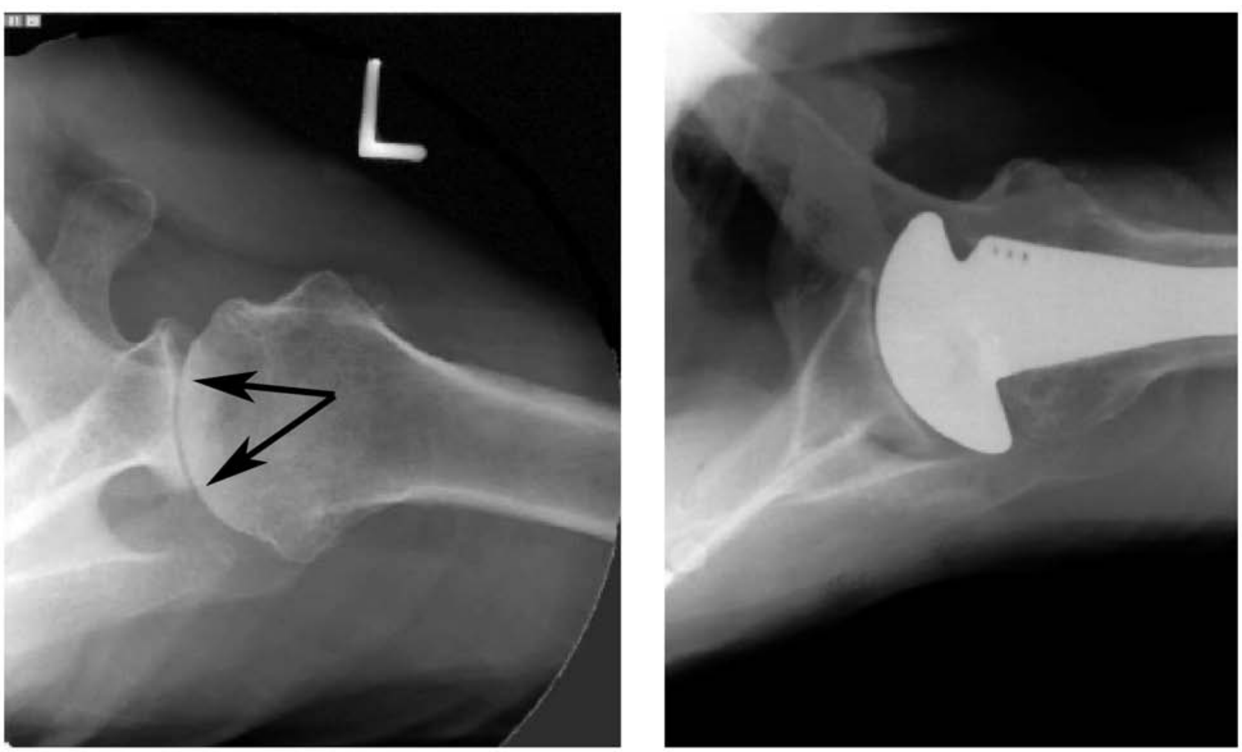


Figure 4: Restoration of glenoid concavity. Arthritic shoulder (left) shows wearing of the back part of the socket allowing the humeral heal (ball) to slip backwards. Ream and run arthroplasty (right) has re-established a normally configured socket that properly centers the humeral head.

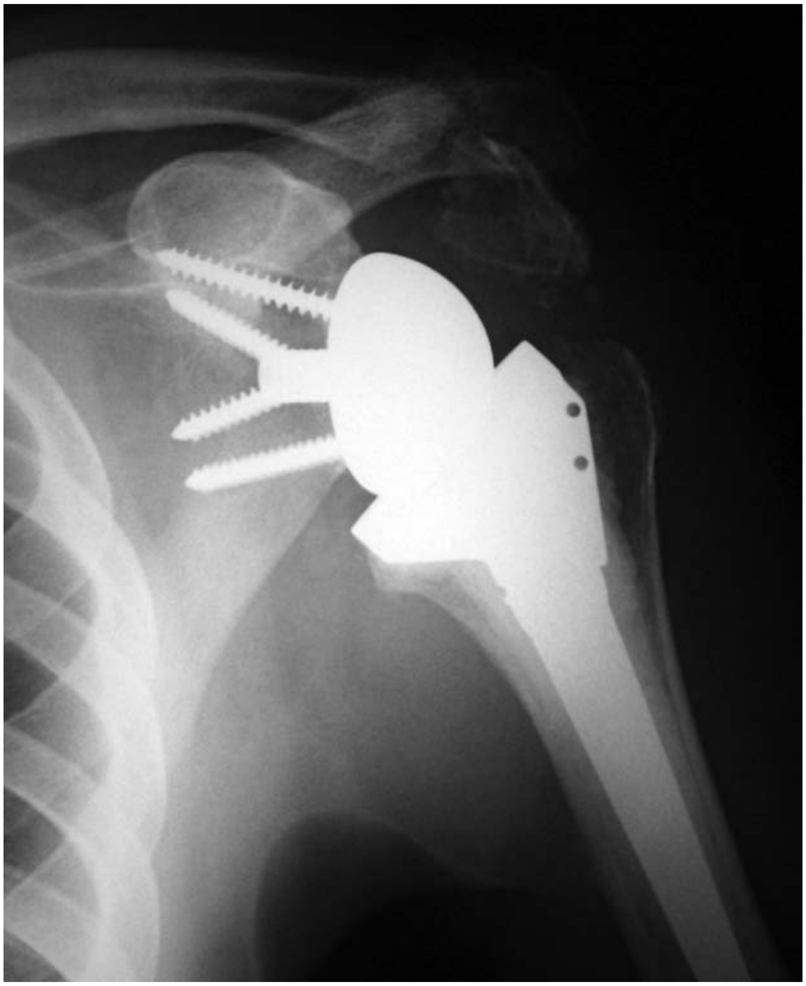


Figure 5: Reverse (delta) total shoulder arthroplasty. Note the ball is on the scapula (shoulder blade, left) while the socket is on the humerus (arm bone, right).

number of patients with shoulder arthritis, enabling them to avoid the risks associated with polyethylene and polymethylmethacrylate bone cement, (2) to speed the recovery of comfort and function after shoulder joint replacement and (3) to develop a better understanding of the ways in which 'stealth' organisms infect shoulder replacements so that these infections can be better prevented and treated.

Support

We have been supported by grant funding from the Orthopaedic Research and Education Foundation, the NIH, the Harryman/DePuy Endowed Chair, and most importantly by contributions from private individuals who wish to join in our quest to restore the arthritic shoulder. To all these entities and individuals, we express our profound gratitude and pledge our ongoing efforts.

References

Braman, J. P., A. Falicov, et al. (2006). "Alterations in surface geometry in retrieved polyethylene glenoid component." J Orthop Res 24(6): 1249-60.

Clinton, J., A. K. Franta, et al. (2007). "Nonprosthetic glenoid arthroplasty with humeral hemiarthroplasty and total shoulder arthroplasty yield similar self-assessed outcomes in the management of comparable patients with glenohumeral arthritis." J Shoulder Elbow Surg 16(5): 534-8.

Franta, A. K., T. R. Lenters, et al. (2007). "The complex characteristics of 282 unsatisfactory shoulder arthroplasties." J Shoulder Elbow Surg 16(5): 555-62.

Harryman, D. T., J. A. Sidles, et al. (1995). "The effect of articular conformity and the size of the humeral head component on laxity and motion after glenohumeral arthroplasty. A study in cadavera." J Bone Joint Surg Am 77(4): 555-63.

Lynch, J. R., A. K. Franta, et al. (2007). "Self-assessed outcome at two to four years after shoulder hemiarthroplasty with concentric glenoid reaming." J Bone Joint Surg Am 89(6): 1284-92.

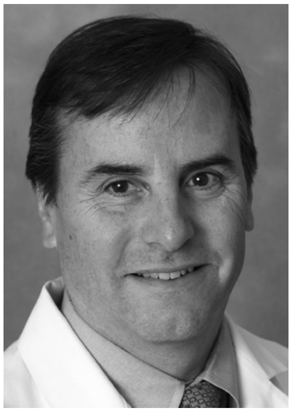
Matsen, F. A., 3rd, J. M. Clark, et al. (2005). "Healing of reamed glenoid bone articulating with a metal humeral hemiarthroplasty: a canine model." J Orthop Res 23(1): 18-26.

Matsen, F. A., 3rd, J. P. Iannotti, et al. (2003). "Humeral fixation by press-fitting of a tapered metaphyseal stem: a prospective radiographic study." J Bone Joint Surg Am 85-A(2): 304-8.

Matsen, F. A., 3rd, P. Boileau, et al. (2007). "The reverse total shoulder arthroplasty." J Bone Joint Surg Am 89(3): 660-7.

Matsen, F. A., 3rd, R. T. Bicknell, et al. (2007). "Shoulder arthroplasty: the socket perspective." J Shoulder Elbow Surg 16(5 Suppl): S241-7.

Matsen III, F. A., Rockwood, C. A., Wirth, M. A., Lippitt, S. B., Parsons, M.: Glenohumeral Arthritis and Its Management. In The Shoulder, Third Edition, Rockwood Jr., C. A., Matsen III, F. A., (eds), Wirth, M. A., Lippitt, S.B. (assoc. eds.), W. B. Saunders Company, Philadelphia, PA, pp. 879-1008, 2004.



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Improving the Function for Individuals with Amputation of the Arm Through Targeted Nerve Transfer Surgery

- Each year many individuals lose parts of their arms or legs because of tumors, injuries or infections.
- Surgeons of the Department of Orthopaedics and Sports Medicine are continuing to discover better methods for restoring function to individuals who have lost their limbs.
- Traditional upper extremity prostheses use power from straps attached to the shoulders to control the movement at the elbow and the grasping device that replaces the lost hand.
- Myoelectric prostheses use signals from active muscle contractions in the affected limb to control the movements of the prosthesis
- Individuals with below elbow amputations have always performed well with myoelectic prosthesis:
    - Amputation below the elbow retains some portion of volar and dorsal forearm muscle that contracts and generates an electrical signal when the brain thinks about closing or opening the hand.
    - This results in a more natural 'brain thought ⇒ forearm muscle contracture ⇒ myoelectric signal to the prosthesis ⇒ hand action' pathway.
    - Learning and operating the forearm level prosthesis is often fluid and logical.
- Individuals with above elbow amputations have struggled with myoelectic prosthesis:
    - While some muscle still remains to generate a signal when thinking elbow-up and elbow-down, these is insufficient muscle remaining to respond when the brain thinks "hand-open" or "hand-close".
    - As a result, the normal above elbow amputee has only a "2 signal arm", without the ability to generate signals for hand function.
    - Using alternative pathways to operate the prosthetic hand required alternative brain thoughts not previously associated with hand function. Often biceps and triceps function was used to operate the elbow in one mode , and then by co-contracting the muscles a switch could be made to a second mode to operate the hand.
    - Switching modes, using elbow thoughts to operate the hand in mode 2, and non-simultaneous elbow and hand function is slow and frustrating.
    - Learning and operating the forearm level prosthesis was difficult and the resulting mental calisthenics remained taxing and frustrating even after extensive training.
- Targeted nerve transfer surgery restores hand function signals in existing upper arm muscles:
    - We have discovered that we can 're-engineer' muscles remaining in the arm by transferring nerves to them so that they generate more naturally the signals necessary to control the prosthesis.

- Removal of the musculocutaneus motor nerve from the medial biceps, then transferring the median nerve to the motor point allows nerve in growth making the medial biceps a median nerve innervated muscle.
- After 3 to 4 months the residual medial biceps in the upper arm amputation will contract when the brain thinks about hand closing.
- Removal of the motor branch to the lateral triceps, and transferring the distal radial nerve to the motor point will likewise create a muscle segment to contract with hand open brain thoughts.
- Above elbow amputee now has a 4 signal arm that can generate elbow-up, elbow- down, hand-open and hand-close signals with normal brain thoughts.
- Simultaneous elbow and hand function is possible.
- Training and uses of a myoelectric prosthesis is more fluid, and natural.

Upper limb amputation, even more than lower limb amputation, creates a tremendous physical, functional, and emotional loss. Our hands are a vitally important part of who we are, how we see ourselves, and how we perform countless tasks. Our hands can be extremely gentle and precise: handwriting, painting a picture, threading a needle or playing a violin, for example. Our hands also enable us to perform heavy labor, such as digging with a shovel, swinging an ax, using a jackhammer to excavate through concrete, or pounding a railroad spike with a sledgehammer. We use our hands to feel whether something is rough or smooth, hot or cold, sharp or dull. We hold a child's hand as we cross the street. We caress the hand of a loved one. The loss of a hand can never really be replaced.

Prosthetic Limbs

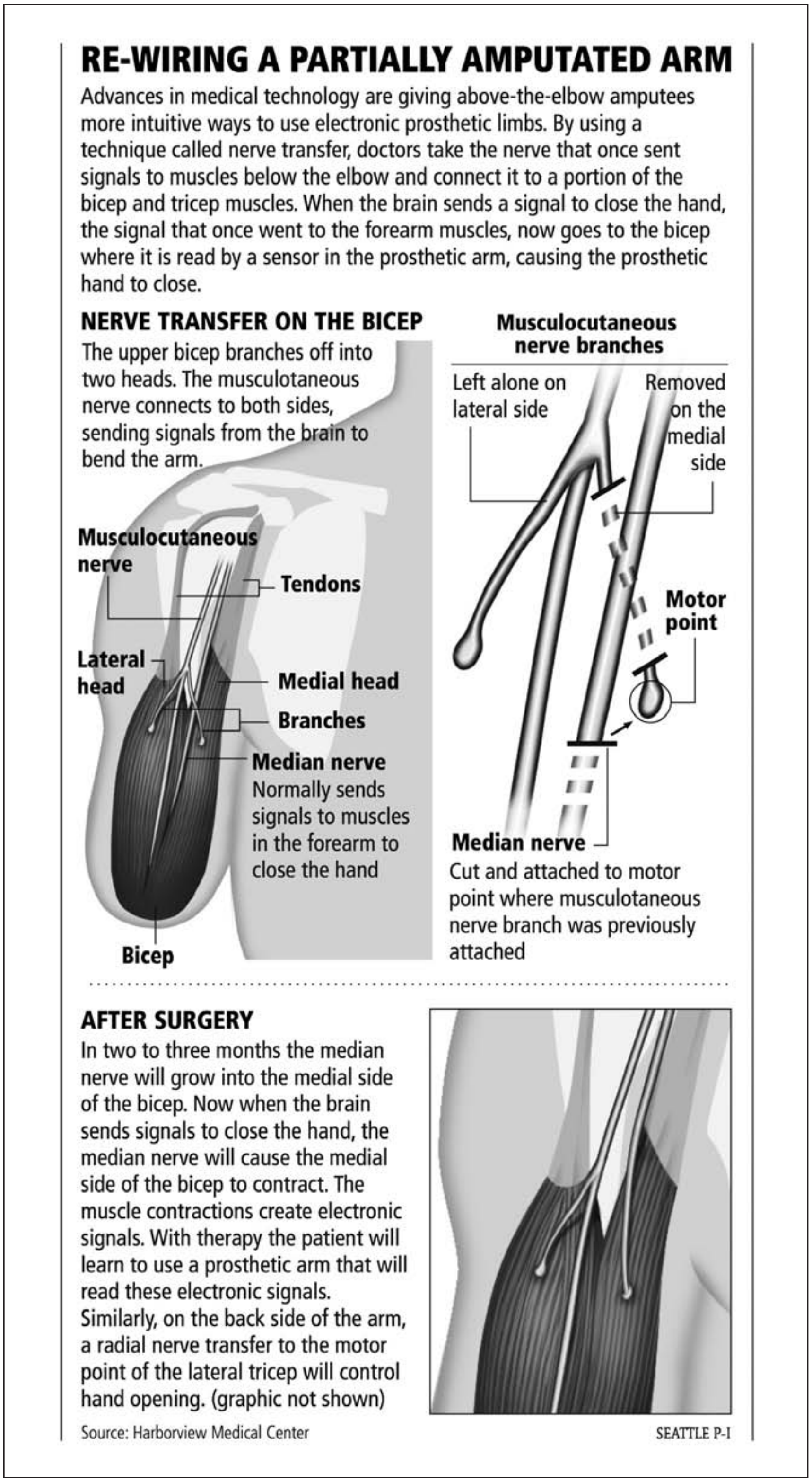
Upper limb prosthetics are, indeed, more complex and complicated than lower limb prosthetic devices. The primary function of our legs is walking. Lower limb prosthetics do an amazing job helping people to walk again. They really replace the primary function of walking and sometimes even running. But we demand more from our hands. Whether it's lifting a cup, moving a chair, scratching the back of your head, we use our hands in a different set of motions in a three-dimensional plane countless times per minute, literally, yet they resolve in a unified motion to complete the tasks. Despite many marvelous technological advances in prosthetic engineering, upper limb prosthetics just can't duplicate all these motions. We often don't need to actually see our hands while they work. But a person with an upper limb prosthesis does need eye contact with the device to make sure it is doing what he or she wants it to do, where it needs to be done.

Body powered prosthetic devices work in a relatively simple fashion. For a below elbow amputee, a cable runs from the prosthesis up the residual arm and across his back to a harness on the opposite shoulder. When the amputee rolls his shoulders forward, the distance between the shoulder blades widens and the cable stretches several inches, pulling the hook open against its springs or rubber bands. When he relaxes his shoulders, the cable goes slack and the springs or rubber bands snap the device closed. The amount of tension on the cable can be varied so the individual can open and close the hook quickly or slowly. The technology was developed during the first and second world wars and has survived because it's simple, functional and durable. It works.

For someone with amputation above the elbow, the cable system runs both the elbow and the hook, but not at the same time. The elbow bends when the shoulders are hunched forward pulling on the cable. Then, the person will lock the elbow in position by either a nudge of a switch on the socket with his chin or with a fast pull on the cable. After the elbow is locked in position, the same cable operates the artificial hand by opening the terminal device against a spring. Gravity lowers the elbow, and rubber bands close the hand or hook. The device works and is certainly useful, but because the same cable operates the elbow and hand they cannot work together at the same time. The user must change modes for each separate function. His prosthetic arm and hand motions were sequential and serialized, definitely not fluid and simultaneous. Only about 20% of unilateral, above elbow amputees use prosthesis on daily basis.

Myoelectric prosthetic devices work much differently. The muscle is a natural, physiologic tissue to receive brain signals and by contracting create a small electrical signal (EMG) in the range of 5 to 100 microvolts. This electrical signal is read transcutaneously an electrode touching the surface of the skin. The signal is amplified, and used to control the motors to run the prosthetic hand, wrist, or elbow to produce the desired movement and function. A below elbow amputee still has some of the volar and dorsal forearm muscles left behind. Fortunately, these residual muscles are still connected to the brain. An electrode sensor over the muscles on the volar residual forearm detects the EMG signal generated when his brain tells those muscles to contract like he was closing his hand. The sensor then sends a signal to the battery and motor to pull on a cable that makes the hand close. When the time comes to release the grip, the brain tells the residual dorsal forearm muscles to fire, the sensor reads that EMG signal, and sends it to the battery and motor to let the cable go slack and open the hand.

An above elbow amputee does not have any remaining muscle to contract when the brain thinks about hand opening or hand closing. Those muscles are gone. Myoelectric devices for this level must use alternative strategies to operate and elbow and the wrist. A hybrid device utilizes both mechanical and electrical components. Often the cable is used to bend the elbow and we let gravity extend it. The amputee can lock the elbow in a given position. A myoelectric sensor takes signals from his residual biceps to close the hand and from the triceps to open it. Doing this does require some mental calisthenics: as the amputee must think, "Biceps fire -- hand closes; triceps fire -- hand opens." A mechanical cable system operates the prosthetic elbow and electrical signals



from the sensor run the hand. This type of device takes a lot of patience, relearning and practice to learn to use. Alternatively, an all-myoelectric system can have two modes, and the biceps and triceps run the elbow in mode 1, and run the hand in mode 2. This also requires tremendous mental energy and results in fatigue and frustration.

Targeted Nerve Transfers - Reconnecting the Brain to New Muscles

Nerve transfer surgery has been around since the 1930s, but using nerve transfer to improve function of upper arm and shoulder level amputees is new. This remarkable surgical breakthrough was the vision, not of a surgeon, but of a physical medicine and rehabilitation physician named Dr. Todd Kuiken.

To create a 4 signal upper arm, with active muscle contraction for not just elbow-up and elbow-down, but also hand-close and hand-open nerve transfers are done to the medical biceps and lateral triceps muscle. The musculocutaneous motor branch to the medical biceps is identified and traced out to its motor point insertion into the muscle. The motor branch is resected paralyzing the muscle. The remaining medial nerve is then transferred onto the motor point, essentially the conduit or portal into the muscle. Posteriorly, the motor branch to the lateral triceps from the proximal radial nerve is identified, traced out to its motor point, and resected. The distal radial nerve is transferred onto the lateral triceps motor point. After 3 to 4 months of reinnervation the medial biceps has become a median nerve driven muscle contracting with hand-close thoughts, and the lateral triceps has become a distal radial nerve driven muscle contraction with hand-open thoughts.

The advanced prosthetic arm sensors can read the EMG signals for all four functions, and can simultaneously operate both the elbow and the hand with normal brain thoughts. No more mental calisthenics. No more slow sequential control.

In addition to upper arm amputees, nerve transfer surgery can also help shoulder level amputees. Amputation at the shoulder leaves an individual with no residual muscle to contract and create signals for either elbow or hand function. Prosthetic use at this level is complicated and frustrating. Through targeted nerve transfer surgery, the residual brachial plexus can be used to rewire the pectoralis major, pectoralis minor, serratus anterior or latissimus dorsi muscles. After nerve transfer and in growth these muscles will contract with brain thoughts of elbow and hand function creating EMG signals to run new advanced function prosthetic arms.

At the time of writing this article, only 18 individuals worldwide have had this unique surgery, and seven of them had their targeted nerve transfer at Harborview Medical Center.

Before performing the surgeries at Harborview, Dr. Smith visited and worked with Dr. Kuiken and plastic surgeon Gregory Dumanian in Chicago over a three-year period. Here in Seattle, the 2 shoulder level and 5 upper arm amputees at Harborview have all successfully demonstrated new hand and elbow signals.

Successful targeted reinnervation provides a physiologic conduit from the brain for controlling the arm and hand. There are no implanted microchips, no foreign devices and no internal electronics. When the nerve arborizes into the muscle through the natural portal of the motor point the axons connect to muscle units. Once the new brain-nerve-muscle pathway connects and works, it will keep working. There is nothing internally to break or wear out. We have created a new pathway for functional brain thoughts to operate a new muscle and talk to electric prosthetic devices.

An unexpected, but interesting byproduct of targeted nerve transfer surgery has been some sensory nerve in growth into the reinnervated muscles. Because a mixed motor and sensory nerve is transferred onto a motor point; both sensory and motor axons arborize and grow. Several of the amputees can actually feel part of the hand when the reinnervated muscle is rubbed or massaged. This has been a pleasant side effect allowing, in a very small way, the amputee to touch and have some feeling or connection the hand that was lost.

The Future

Targeted nerve transfer surgery has opened the door to improved function for above elbow and shoulder level amputees. It has also opened our minds to the fact that nerve management in all types of amputation surgery can probably be improved. Traditional nerve management has been to draw down the nerve, divide it, and allow the nerve to retract into a padded area away from scar, pressure and pulsing vessels. It did however create a dead-end nerve that always heals with axonal ending and scar, a neuroma. Creating a physiologic connection for a nerve with a segment of muscle gives the nerve an opportunity to grow, reconnect and remain physiologically active. I believe this will initially improve our management of symptomatic neuromas, and might eventually improve nerve management in many amputation surgeries.

References

Kuiken TA, Marasco PD, Lock BA, Harden RN, Dewald JP: Redirection of cutaneous sensation from the hand to the chest skin of human amputees with targeted reinnervation. Proc Natl Acad Sci U S A. 2007 Dec 11;104(50):20061-6. Epub 2007 Nov 28. PMID: 18048339.

Zhou P, Lowery MM, Englehart KB, Huang H, Li G, Hargrove L, Dewald JP, Kuiken TA: Decoding a new neural machine interface for control of artificial limbs. J Neurophysiol. 2007 Nov;98(5):2974-82. Epub 2007 Aug 29. PMID: 17728391.

Kuiken T, Miller L, Lipschutz R, Stubblefield K, Dumanian G: Prosthetic command signals following targeted hyper-reinnervation nerve transfer surgery. Conf Proc IEEE Eng Med Biol Soc. 2005;7:7652-5. PMID: 17282053.

Zhou P, Lowery M, Weir R, Kuiken T: Elimination of ECG Artifacts from Myoelectric Prosthesis Control Signals Developed by Targeted Muscle Reinnervation. Conf Proc IEEE Eng Med Biol Soc. 2005;5:5276-9. PMID: 17281440.

Zhou P, Lowery M, A Dewald J, Kuiken T: Towards Improved Myoelectric Prosthesis Control: High Density Surface EMG Recording After Targeted Muscle Reinnervation. Conf Proc IEEE Eng Med Biol Soc. 2005;4:4064-7. PMID: 17281125.

Ohnishi K, Weir RF, Kuiken TA: Neural machine interfaces for controlling multifunctional powered upper-limb prostheses. Expert Rev Med Devices. 2007 Jan;4(1):43-53. Review. PMID: 17187470.

Kuiken TA, Miller LA, Lipschutz RD, Lock BA, Stubblefield K, Marasco PD, Zhou P, Dumanian GA: Targeted reinnervation for enhanced prosthetic arm function in a woman with a proximal amputation: a case study. Lancet. 2007 Feb 3;369(9559):371-80. PMID: 17276777.

Hijjawi JB, Kuiken TA, Lipschutz RD, Miller LA, Stubblefield KA, Dumanian GA: Improved myoelectric prosthesis control accomplished using multiple nerve transfers. Plast Reconstr Surg. 2006 Dec;118(7):1573-8. PMID: 17102730.

Kuiken T: Targeted reinnervation for improved prosthetic function. Phys Med Rehabil Clin N Am. 2006 Feb;17(1):1-13. Review. PMID: 16517341.

Kuiken TA, Dumanian GA, Lipschutz RD, Miller LA, Stubblefield KA: The use of targeted muscle reinnervation for improved myoelectric prosthesis control in a bilateral shoulder disarticulation amputee. Prosthet Orthot Int. 2004 Dec;28(3):245-53. PMID: 15658637.

Kuiken TA, Childress DS, Rymer WZ: The hyper-reinnervation of rat skeletal muscle. Brain Res. 1995 Apr 3;676(1):113-23. PMID: 7796162.

Web Site Consumer Articles

OandP Edge: Targeted Muscle Reinnervation: The Future Is Now. By Miki Fairley. http://www.oandp.com/edge/issues/articles/2007-12_01.asp

SeattlePI.com: His Mind Controls His New Arm. By Carol Smith. http://seattlepi.nwsource.com/local/325630_arm30.html

King 5 TV.com: Bionic limb research making big strides in Seattle. By Jean Enersen. http://www.king5.com/health/stories/NW_112807HEB_bionic_limbs_LJ.4ab87471.html

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Ankle Arthritis: Nothing Ordinary About It

- Loss of the cartilage of the ankle joint causes severe pain and stiffness, making it difficult for the patient to stand, walk, and run.
- This loss of cartilage can be caused by
  - Injuries to the ankle joint or to the bones and ligaments that stabilize and support it. Such injuries are by far the most common causes of ankle arthritis, pointing to the need for their prevention as well as early and expert management so that the risk of this complication is minimized.
  - Congenital problems, such as clubfoot or Charcot-Marie-Tooth disease.
  - Systemic inflammatory disease, such as rheumatoid arthritis, gout, hemachromatosis, or psoriasis.
- The team at the S.T. Hansen, Jr. Foot and Ankle Institute is committed to discovering better ways to prevent ankle arthritis and to restore comfort and function to patients who are disabled by this condition.

The ankle and foot are at risk in sports, falls, motor vehicle accidents, work, and ordinary activities of daily living. These injuries may cause instability or abnormal loading of the surfaces of the ankle joint or may directly injure the joint cartilage that is essential to normal ankle function. Even an 'ordinary' sprain of the foot or ankle, if not properly treated, can result in permanent damage to the joint. It is rare for us to see severe arthritis in an ankle that has not been injured. However, the consequences of improper treatment may not be evident until decades later.

The characteristically long interval between the injury and the eventual severe arthritis means that we have an opportunity to do something about the chronic instability, prevent the ultimate arthritis, and avoid the possible need for an ankle fusion or total ankle arthroplasty - neither of which are equivalent to the function of a relatively normal ankle. When abnormal foot, ankle or leg anatomy underlies recurring ankle sprains, correction of the anatomy may prevent arthritis years later.

An expert, careful history and physical examination are necessary to define the static and dynamic factors that may contribute to the ankle instability. Once the cause of the ankle problem is identified, good orthopaedic care can make a major difference by stopping the inevitable march to end-stage arthritis. We discovered that once-popular non-anatomic repairs for recurrent sprains (usually with the peroneus brevis) cause abnormal biomechanics in the ankle. However, we now manage chronic ankle instability using a combination of ligament repair and anatomic reconstruction of the ligaments using the plantaris or toe extensor tendons.

In more complex cases, the instability may be associated with predisposing anatomy. The common problems are tight calf muscles (gastrocnemius equinus) or abnormal orientation of the heel (intrinsic heel varus, or forefoot-driven hindfoot varus from a plantar-flexed first metatarsal). Weak or torn muscles (e.g. the peroneus brevis) may contribute to foot malpositioning. Other factors that can contribute to ankle instability include abnormal external rotation of the thigh bone (femur) or shin bone (tibia) as well as generalized ligamentous laxity. Many of these predisposing factors can be treated. Gastrocnemius equinus can be managed with a procedure to relax this muscle (gastroc slide). Inward pointing of the heel (calcaneus varus) can be managed with surgical realignment (a lateralizing calcaneal osteotomy). A rigid downward pointing ball of the big toe (plantar flexed first metatarsal) can be addressed by surgical realignment (a dorsiflexion osteotomy or fusion through the first tarsometatarsal joint).

Figure 1: Three x-ray views of the right foot showing ankle and subtalar arthritis and collapse of the medial column of the foot.

A dynamic high arch (dynamic first metatarsal cavus), can be treated with muscle realignment (transfer of the peroneus longus to the attachment of the peroneus brevis to let it function as an evertor rather than a first ray plantar flexor). Bony abnormalities (for example, tibia vara) can be treated by cutting and straightening the bone (a high tibial osteotomy or closing lateral wedge supramalleolar osteotomy).

Treatment of Ankle Arthritis

In the 60s and early 70s, when I was in training and early practice, the only treatment we used for severe ankle arthritis was arthrodeses, that is, fusion of the joint so that it could not be moved up or down. Arthrodeses were done by an external fixator device called a Charnley clamp or by a Roger Anderson external fixation or, in children, by simply removing a very large amount of joint and placing them in a cast without any fixation. Many patients were too smart for this and did not want fusion. In these individuals we used bracing, rocker-bottom shoes, and anti-inflammatory medications. When we became more sophisticated, we used alignment corrections by varus or valgus, supramalleolar osteotomies, joint debridements, ligament repair, muscle transfers, heel cord lengthening, in an effort to avoid fusion.

My own experience was that many of these ankle fusions we did early on were unsatisfactory in terms of position of the ankle (equinus or varus or anteriorly displaced) leading to substantial disability. In addition, these feet had often been casted for a rather long time, which left a lot of stiffness in the joint below the ankle (the subtalar joint). My belief at the time was that if we did much better ankle fusions that healed quickly in anatomic position that the problem would be solved. In fact, we did get much improved position and a more rapid healing and early return of subtalar motion by using rigid AO compression screws and trying to maintain most the anatomy by removing very little bone. We thought for a while that the problem was solved.

However in the 90s, our ‘good’ ankle fusions began to return with symptomatic, sometimes severe arthritis of the joint below the ankle joint (the subtalar joint). Also, of course, there were other patients who presented with co-existing ankle arthritis and subtalar arthritis. In these situations, ankle arthrodesis would not work by itself because it would just increase the problem in the subtalar joint. In these cases, we fused the subtalar joints as well. Some of these patients did very well with this when wearing rocker-bottom shoes but others were so unhappy that they asked for an amputation.

Therefore, when Dr. Frank Alvine (one of the many outstanding graduate

Figure 2: Post surgical x-rays showing reconstruction of the angle using a custom stemmed total ankle and subtalar alignment and fusion as well as alignment and fusion of medial column.

of our residency program) developed a new ankle joint replacement in the early 1990's, we joined him in developing the early clinical experience. Within a couple of years, we were seeing one or more patients with end-stage ankle arthritis per week. Most recently the average is probably three or four.

The initial results were very encouraging with surprisingly little problems of subsidence or loosening. However, we discovered that good results are critically dependent on patient selection and exacting surgical technique. Specifically we found that the ankle is much more difficult to align than the hip or knee because (1) it has a companion joint, the subtalar joint, just below that may or may not be stable and (2) its alignment depends on stability of the midfoot and forefoot, all of which must be addressed before or during the ankle replacement. We also discovered the difficulties in fixing the new prosthetic joint surface to the talus. Failure to bond and subsequent loosening and change of position or subsidence of the talar component were all too common.

However, we soon found out that some of the preconceived notions about arthroplasty were not true. It was assumed that failures would have to be followed by fusion and that removal of a fair amount of bone for arthroplasty would be a bad idea. In fact, most all failures could be revised, usually successfully, and often just by redoing the talar component. Also, we discovered that fusions can be taken down and replaced by a prosthesis if the malleoli and deltoid ligament have not been removed; something that had never been suggested previously. In addition, over the first 10 years, the design of the talar component was revised at least three times to make it larger and more reliable and several years ago, a stemmed prosthesis was developed for revisions in patients who had subtalar arthritis or could sacrifice the subtalar joint. These stemmed components also proved useful in patients who were quite old with marked osteopenia or those with a failed fusion secondary to subtalar arthritis. The custom made nature of this talar component means also that the body can be supplemented to compensate for subsidence or erosion of the talus by adding some height or bulk to the component.

Our Current Approach

In early stages of instability, we identify and treat predisposing conditions including gastroc equinus, varus, and cavovarus feet and perform a stress test under anesthesia to see if ligaments need repair. Our goal is to prevent the long-term consequences of ankle instability through early intervention.

When a younger person has end-stage ankle arthritis, we consider re-alignment and ankle arthrodesis when there is a good subtalar joint. Patients accept this much more readily when they are informed that the subtalar joint will provide very good function for a period of time and when it no longer does, when they are older and total ankle arthroplasties are even more improved, they can then have a subtalar arthrodesis and take-down of the ankle fusion and insertion of a total ankle arthroplasty. In other words, the fusion is not forever.

In older patients who have ankle arthritis without subtalar arthritis and treatable malalignment, we use a standard total ankle arthroplasty and adjunct procedures for alignment and stabilization. If the patient has marked ankle and subtalar arthritis or is quite elderly and has osteopenia, we carry out ankle arthroplasty with a much more stable custom stemmed arthroplasty and simultaneous subtalar arthrodesis at the outset (see Figures 1 and 2).

In failed arthroplasty with talar component subsidence, we revise with a custom-stemmed talar component and an augmented body as needed. In the rare tibial component failure, we use a stemmed tibial prosthesis. In the rare total ankle that gets infected, either early or late, treatment is by irrigation and IV antibiotics in acute cases; in delayed cases with established soft-tissue infection or deeper infection, the prosthesis is removed and replaced by a block of antibiotic-infused methylmethacrylate and three to six months later, we reinsert a prosthesis. Only the rare patient is converted back to a fusion, and that is done if the arthroplasty fails by continual overgrowth of bone indicating that this patient's ankle 'wants' to fuse itself. In these cases, the ankle usually goes on to fusion fairly easily when the prosthesis is removed.

Conclusion

While these results are very encouraging, the fact remains that our current surgical approaches to ankle arthritis often do not last more than 10 years. An ankle fusion, even if done very well, may lead to subtalar arthritis and the need for another operation. Even a good total ankle arthroplasty will develop poly wear and the need for a revision.

Thus, one of the primary goals of the Foot and Ankle Institute is to discover better ways to anticipate and prevent ankle arthritis on one hand and more durable approaches to treating it on the other.

References

Knecht SI, Estin M, Callaghan JJ, Zimmerman MB, Alliman KJ, Alvine FG, Saltzman CL. The Agility Total Ankle Arthroplasty. Seven to Sixteen-Year Follow-up. J Bone Joint Surg Am. 2004;86:1161-1171.

Spirt AA, Assal M, Hansen ST Jr. Complications and failure after total ankle arthroplasty. J Bone Joint Surg Am. 2004 Jun;86-A(6):1172-8.

Haddad SL, Coetzee JC, Estok R, Fahrbach K, Banel D, Nalysnyk L. Intermediate and long-term outcomes of total ankle arthroplasty and ankle arthrodesis. A systematic review of the literature. J Bone Joint Surg Am. 2007 Sep;89(9):1899-905.

SooHoo NF, Zingmond DS, Ko CY. Comparison of reoperation rates following ankle arthrodesis and total ankle arthroplasty. J Bone Joint Surg Am. 2007 Oct;89(10):2143-9.

Pyevich MT, Saltzman CL, Callaghan JJ, Alvine FG.Total ankle arthroplasty: a unique design. Two to twelve-year follow-up. J Bone Joint Surg Am. 1998 Oct;80(10):1410-20.

Graduating Residents
Class of 2008

Drew Fehsenfeld, M.D.

In the coming year, Drew will complete a sports medicine fellowship at the University of Connecticut. Afterwards, he plans to practice in Texas.

Christopher Howe, M.D.

Upon graduation Chris will begin his spine fellowship here at Harborview Medical Center and the University of Washington. After the fellowship he and his wife plan on staying in the Pacific Northwest.

Michael Lee, M.D.

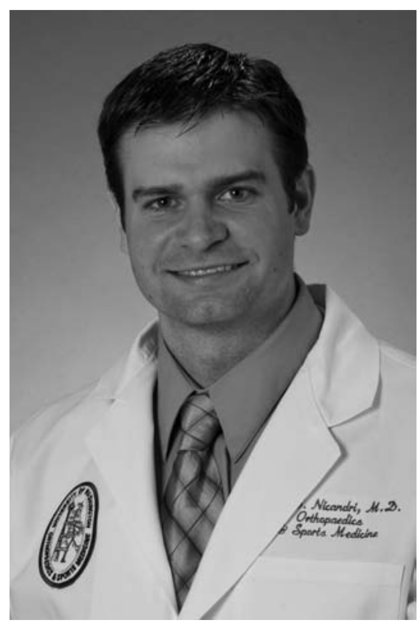
Next year Michael will be moving to San Francisco where he will begin a sports fellowship at the Palo Alto Medical Foundation in Palo Alto, California. He hopes to return to the Seattle area to practice.

Mark Freeborn, M.D.

Next year, Mark will be a fellow in spine surgery at Harborview Medical Center and the University of Washington. He and his wife (Jayme) plan to reside in the greater Seattle area after his training is complete.

John Howlett, M.D.

In the next academic year, John will complete a fellowship here at the UW in Hand and Microvascular Surgery. He then plans to remain in the Pacific Northwest to establish a practice specializing in the upper extremity.



Gregg Nicandri, M.D.

After residency, Gregg will be completing a one year fellowship in sports medicine at Duke University. He plans on pursuing an academic career in sports medicine and arthroscopy.

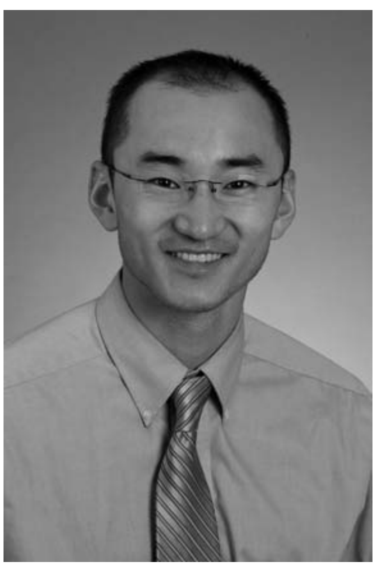
Incoming Residents

Benjamin Amis

Ben is from Plano, Texas. He attended Rice University as an undergraduate and received his medical degree from the University of Texas Southwestern. His current orthopedic interests include hand and sports. In his spare time, he enjoys skiing, wakeboarding, running, triathlon, and cooking.

Gregory Blaisdell

Starting in Maine where he grew up, Greg has made a slow pieced-together trek across the country. He attended the University of New Hampshire, where he studied Biochemistry and met is wife-to-be, Randi. He then got his medical degree from Penn State University. Greg's interests include trauma and sports medicine, and whenever possible, Greg enjoys spending time doing outdoor activities with his wife, family and friends.



Adam Bakker

Adam was born in Seoul, South Korea and raised in Walker, Minnesota. He went to Concordia College in Moorhead, Minnesota for undergraduate and went to the University of Minnesota for medical school. His Orthopaedic interests are hand surgery and sports medicine. His extracurricular interests includes road biking, snowboarding, cooking/eating with friends and family, international travel, and spending time with his Yellow Lab, Duke and his wife, Erin.

Joshua Lindsey

Josh was raised in Mineral Point, WI. He enrolled in undergraduate studies at the University of Wisconsin and subsequently enrolled in graduate studies at Drexel University in Philadelphia. His medical education was at the University of Wisconsin School of Medicine and Public Health. Josh and his wife Katie enjoy travel, cooking, skiing and independent movies. His interests are shoulder, sports and total joint replacement surgery.

Incoming Residents

Grant Lohse

Grant was born and raised in Indiana. He earned an engineering degree from Purdue University and attended medical school at Northwestern. His early research interests have focused on the biomechanical properties of orthopedic implants. In his free time, Grant enjoys spending time with his wife and exploring Seattle's natural beauty.

Andrew Merritt

Andrew was born and raised in Ojai, California. He graduated from California Polytechnic University with a degree in Biomedical Engineering. After a year of travelling the world, he attended the University of Southern California for medical school. His research interests include biomechanics and biomaterials, and he remains interested in all of the orthopaedic subspecialties. In his spare time he enjoys traveling and spending time in the mountains.

Matthew Lyons

Matt was born and raised in Walla Walla, Washington. He received both his undergraduate and medical degrees from the University of Washington. His current orthopaedic interests include Sports Medicine and Hand and Microvascular Surgery. In his free time, he enjoys weightlifting, running, snow and water skiing, as well as following Husky football and basketball.

Nels Sampatacos

Nels received his undergraduate degree from Arizona State University and his medical degree from The University of Arizona College of Medicine. Within orthopaedics, his areas of interest include sports medicine, joints, and trauma. Outside of work, he enjoys skiing, snowboarding, weight lifting, rappelling, cycling, and traveling.

ACEs

Foot/Ankle

Helen J. Rawlinson, M.D.

Tracy D. Rupke, M.D.

Ferras Zeni, M.D.

Spine

Paul E. Kraemer, M.D.

Anthony J. Russo, M.D.

Trauma

Michael L. Brennan, M.D.

Andrew R. Evans, M.D.

Jason M. Evans, M.D.

Michael J. Gardner, M.D.

Zachary V. Roberts, M.D.

Raymond D. Wright Jr., M.D.

ACEs

Shoulder/Elbow

Jeremiah M. Clinton, M.D.

Joseph R. Lynch, M.D.

Oncology

Philip J. Krueger, M.D.

Pediatrics

Jennifer W. Lisle, M.D.

Fellows

Hand

Jay Bridgeman, M.D.

Sarah Pettrone, M.D.

James D. Schlenker, M.D.

Michael Shuler, M.D.

Research Grants

National Institutes of Health (NIH)

Changes in the Characteristics of Plantar Soft Tissue with Diabetes
Bruce J. Sangeorzan, M.D.

Collagens of Cartilage and the Intervertebral Disc
David R. Eyre, Ph.D.

Collagen Cross-Linking in Skeletal Aging and Diseases
David R. Eyre, Ph.D.

Collagen Type II/IX/XI Heteropolymer Assembly
Russell J. Fernandes, Ph.D.

Disuse Induced Osteocyte Hypoxia
Ted S. Gross, Ph.D.

Organization of Mechanical Signals in Bone Cells via Membrane
Ted S. Gross, Ph.D.

Predicting Bone Formation Induced by Mechanical Loading Using Agent Based Models
Sundar Srinivasan, Ph.D.

Skeletal Dysplasias
David R. Eyre, Ph.D.

Veterans Affairs Rehabilitation Research and Development Service

Ankle Equinus and Plantar Pressure in Individuals with Diabetes
Bruce J. Sangeorzan, M.D.

Ewing's Sarcoma Fusion Proteins and mRNA Splicing Factors
Howard A. Chansky, M.D.

Dynamic Gait Simulation: Neutrally Aligned and Pathologic Feet
Bruce J. Sangeorzan, M.D.

Kinematic Models of Normal and Painful Osteoarthritic Feet
Bruce J. Sangeorzan, M.D.

Treatment Outcomes for Ankle Arthritis
Bruce J. Sangeorzan, M.D.

VA Center of Excellence in Amputation Prevention and Prosthetic Engineering
Bruce J. Sangeorzan, M.D.

VA Merit Review Functional Analysis of EWS/FLI-1
Howard A. Chansky, M.D.

Orthopaedic Research and Education Foundation (OREF)

Clinical Efficacy and Cost Implications of Acute BMP-2
David P. Barei, M.D.

Perioperative Economic Analysis of Minimally Invasive Versus Traditional Total Knee Arthroplasty
Seth S. Leopold, M.D.

The Effect of Obesity on Outcomes Among Trauma Patients with Lower Extremity Orthopaedic Injuries
Sean E. Nork, M.D.

A.O. North America

An Observational Study Assessment of Surgical Techniques for Treating Cervical Spondylotic Myelopathy (CSM)
Jens R. Chapman, M.D.

An Observational Study Comparing Surgical to Conservative Management in the Treatment of Type II Odontoid Fractures Among the Elderly
Jens R. Chapman, M.D.

AO North America Orthopaedic Trauma Fellowship
Bruce J. Sangeorzan, M.D.

AO Spine North America Fellowship
Carlos Bellabarba, M.D.

Amgen, Inc.

Contrasting the Ability of OPG and Alendronate to Inhibit Bone Loss
Ted S. Gross, Ph.D.

Bayer AG

A Multi-Center, Randomized, Double-Blind, Placebo-Controlled, Parallel Design, 2-Arm Study to Investigate the Effect of Aprotinin On Transfusion Requirements in Patients Undergoing Elective Spinal Fusion Surgery
Jens R. Chapman, M.D.

BioAxone Therapeutique, Inc.

Cethrin Trial
Jens R. Chapman, M.D.

CeraPedics, LLC

An Assessment of P15 Bone Putty In Anterior Cervical Fusion with Instrumentation
Jens R. Chapman, M.D.

Research Grants

Christopher Reeve Paralysis Foundation

Using Muscle Stimulation to Mitigate Bone Loss
Due to Muscle Paralysis
Ted S. Gross, Ph.D.

Foundation for Orthopedic Trauma

The Role of Muscle Function in Fracture Healing
Sean E. Nork, M.D.

Integra Lifesciences Corporation

Comparison of Bioabsorable Tubes for Repair of Nerve Injury
Thomas E. Trumble, M.D.

DePuy Spine, Inc. (Johnson & Johnson, Inc.)

Clinical Spine Fellowship Grant
Theodore A. Wagner, M.D.

Ostex International, Inc.

Molecular Markers of Connective Tissue Degradation
David R. Eyre, Ph.D.

Orthopaedic Trauma Association

The Effect of Obesity on Outcomes Among Trauma
Patients with Lower Extremity Orthopaedic Injuries
Sean E. Nork, M.D.

The Role of Muscle Function in Fracture Healing
Sean E. Nork, M.D.

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, M.D.

Smith & Nephew, Inc.

University of Washington Arthroscopy, Research and Training (ART) Lab
Christopher J. Wahl, M.D.

Synthes Spine Co.

PRODISC-C Versus Anterior Cervical Discectomy and Fusion (ACDF)
Jens R. Chapman, M.D.

Spine End-Results Research Fund
Sohail K. Mirza, M.D.

The Boeing Company

Randomized Clinical Trial of Open versus Endoscopic Carpal Tunnel Release and Hand Therapy Comparing Patient Satisfaction: Functional Outcome and Cost Effectiveness
Thomas E. Trumble, M.D.

US Army Research Office

UW Team-Advance on Single Nuclear Detection and Atomic-Scale Imaging
John A. Sidles, Ph.D.

US Department of Education

Advancing Orthotic and Prosthetic Care Through Research, Standards of Practice and Outreach
Douglas G. Smith, M.D.

Department Publications 2007-2008

1. Barei, D. P.; Agel, J.; and Swiontkowski, M. F.: Current utilization, interpretation, and recommendations: the musculoskeletal function assessments (MFA/SMFA). J Orthop Trauma, 21(10): 738-42, 2007.

2. Barei, D. P.; O'Mara, T. J.; Taitsman, L. A.; Dunbar, R. P.; and Nork, S. E.: Frequency and fracture morphology of the posteromedial fragment in bicondylar tibial plateau fracture patterns. J Orthop Trauma, 22(3): 176-82, 2008.

3. Barei, D. P.; Taitsman, L. A.; Beingessner, D.; Dunbar, R. P.; and Nork, S. E.: Open diaphyseal long bone fractures: a reduction method using devitalized or extruded osseous fragments. J Orthop Trauma, 21(8): 574-8, 2007.

4. Beingessner, D. M.; Dunning, C. E.; Stacpoole, R. A.; Johnson, J. A.; and King, G. J.: The effect of coronoid fractures on elbow kinematics and stability. Clin Biomech (Bristol, Avon), 22(2): 183-90, 2007.

5. Beingessner, D. M.; Stacpoole, R. A.; Dunning, C. E.; Johnson, J. A.; and King, G. J.: The effect of suture fixation of type I coronoid fractures on the kinematics and stability of the elbow with and without medial collateral ligament repair. J Shoulder Elbow Surg, 16(2): 213-7, 2007.

6. Birmingham, P.; Helm, J. M.; Manner, P. A.; and Tuan, R. S.: Simulated joint infection assessment by rapid detection of live bacteria with real-time reverse transcription polymerase chain reaction. J Bone Joint Surg Am, 90(3): 602-8, 2008.

7. Cabral, W. A. et al.: Prolyl 3-hydroxylase 1 deficiency causes a recessive metabolic bone disorder resembling lethal/severe osteogenesis imperfecta. Nat Genet, 39(3): 359-65, 2007.

8. Chapman, J., and Bransford, R.: Geriatric spine fractures: an emerging healthcare crisis. J Trauma, 62(6 Suppl): S61-2, 2007.

9. Chapman, J. R.; Agel, J.; Jurkovich, G. J.; and Bellabarba, C.: Thoracolumbar flexion-distraction injuries: associated morbidity and neurological outcomes. Spine, 33(6): 648-57, 2008.

10. Chebli, C.; Huber, P.; Watling, J.; Bertelsen, A.; Bicknell, R. T.; and Matsen, F., 3rd: Factors affecting fixation of the glenoid component of a reverse total shoulder prothesis. J Shoulder Elbow Surg, 17(2): 323-7, 2008.

11. Clinton, J.; Franta, A. K.; Lenters, T. R.; Mounce, D.; and Matsen, F. A., 3rd: Nonprosthetic glenoid arthroplasty with humeral hemiarthroplasty and total shoulder arthroplasty yield similar self-assessed outcomes in the management of comparable patients with glenohumeral arthritis. J Shoulder Elbow Surg, 16(5): 534-8, 2007.

12. Cunningham, M. R.; Warme, W. J.; Schaad, D. C.; Wolf, F. M.; and Leopold, S. S.: Industry-funded positive studies not associated with better design or larger size. Clin Orthop Relat Res, 457: 235-41, 2007.

13. Do, K. D.; Bellabarba, C.; and Bhananker, S. M.: Exertional rhabdomyolysis in a bodybuilder following overexertion: a possible link to creatine overconsumption. Clin J Sport Med, 17(1): 78-9, 2007.

14. Donescu, O. S.; Battie, M. C.; Kaprio, J.; Levalahti, E.; Risteli, J.; Eyre, D.; and Videman, T.: Genetic and constitutional influences on bone turnover markers: a study of male twin pairs. Calcif Tissue Int, 80(2): 81-8, 2007.

15. Dunbar, R. P., Jr.: Treatment of infection after fracture fixation. Opinion: retain stable implant and suppress infection until union. J Orthop Trauma, 21(7): 503-5, 2007.

16. Dunbar, R. P.; Taitsman, L. A.; Sangeorzan, B. J.; and Hansen, S. T., Jr.: Technique tip: use of "pie crusting" of the dorsal skin in severe foot injury. Foot Ankle Int, 28(7): 851-3, 2007.

17. Eary, J. F., and Conrad, E. U.: PET imaging: update on sarcomas. Oncology (Williston Park), 21(2): 249-52, 2007.

18. Elgafy, H., and Bellabarba, C.: Three-column ligamentous extension injury of the thoracic spine: a case report and review of the literature. Spine, 32(25): E785-8, 2007.

19. Elias, F. N.; Yuen, T. J.; Olson, S. L.; Sangeorzan, B. J.; and Ledoux, W. R.: Correction of clawed hallux deformity: comparison of the Jones procedure and FHL transfer in a cadaver model. Foot Ankle Int, 28(3): 369-76, 2007.

20. Eyre, D. R.; Weis, M. A.; and Wu, J. J.: Advances in collagen cross-link analysis. Methods, 45(1): 65-74, 2008.

21. Fernandes, R. J.; Harkey, M. A.; Weis, M.; Askew, J. W.; and Eyre, D. R.: The post-translational phenotype of collagen synthesized by SAOS-2 osteosarcoma cells. Bone, 40(5): 1343-51, 2007.

22. Fernandes, R. J.; Weis, M.; Scott, M. A.; Seegmiller, R. E.; and Eyre, D. R.: Collagen XI chain misassembly in cartilage of the chondrodysplasia (cho) mouse. Matrix Biol, 26(8): 597-603, 2007.

23. Franta, A. K.; Lenters, T. R.; Mounce, D.; Neradilek, B.; and Matsen, F. A., 3rd: The complex characteristics of 282 unsatisfactory shoulder arthroplasties. J Shoulder Elbow Surg, 16(5): 555-62, 2007.

24. Gardner, M. J.; Kendoff, D.; Ostermeier, S.; Citak, M.; Hufner, T.; Krettek, C.; and Nork, S. E.: Sacroiliac joint compression using an anterior pelvic compressor: a mechanical study in synthetic bone. J Orthop Trauma, 21(7): 435-41, 2007.

25. Gardner, M. J., and Nork, S. E.: Stabilization of unstable pelvic fractures with supraacetabular compression external fixation. J Orthop Trauma, 21(4): 269-73, 2007.

26. Goradia, D.; Linnau, K. F.; Cohen, W. A.; Mirza, S.; Hallam, D. K.; and Blackmore, C. C.: Correlation of MR imaging findings with intraoperative findings after cervical spine trauma. AJNR Am J Neuroradiol, 28(2): 209-15, 2007.

27. Grassbaugh, J. A., and Mosca, V. S.: Congenital ingrown toenail of the hallux. J Pediatr Orthop, 27(8): 886-9, 2007.

28. Greisberg, J., and Sangeorzan, B.: Hindfoot arthrodesis. J Am Acad Orthop Surg, 15(1): 65-71, 2007.

29. Han, M.; Yang, X.; Lee, J.; Allan, C. H.; and Muneoka, K.: Development and regeneration of the neonatal digit tip in mice. Dev Biol, 315(1): 125-35, 2008.

30. Hanel, D. P., and Chin, S. H.: Wrist level and proximal-upper extremity replantation. Hand Clin, 23(1): 13-21, 2007.

31. Hanel, D. P.; Pfaeffle, H. J.; and Ayalla, A.: Management of posttraumatic metadiaphyseal radioulnar synostosis. Hand Clin, 23(2): 227-34, vi-vii, 2007.

32. Heike, C. L.; Avellino, A. M.; Mirza, S. K.; Kifle, Y.; Perkins, J.; Sze, R.; Egbert, M.; and Hing, A. V.: Sleep disturbances in 22q11.2 deletion syndrome: a case with obstructive and central sleep apnea. Cleft Palate Craniofac J, 44(3): 340-6, 2007.

33. Hjorten, R. et al.: Type XXVII collagen at the transition of cartilage to bone during skeletogenesis. Bone, 41(4): 535-42, 2007.

34. Howard, J. L.; Agel, J.; Barei, D. P.; Benirschke, S. K.; and Nork, S. E.: A prospective study evaluating incision placement and wound healing for tibial plafond fractures. J Orthop Trauma, 22(5): 299-305, 2008.

35. Hu, H. M.; Zielinska-Kwiatkowska, A.; Munro, K.; Wilcox, J.; Wu, D. Y.; Yang, L.; and Chansky, H. A.: EWS/FLI1 suppresses retinoblastoma protein function and senescence in Ewing's sarcoma cells. J Orthop Res, 2008.

36. Hunter, D. J. et al.: Cartilage markers and their association with cartilage loss on magnetic resonance imaging in knee osteoarthritis: the Boston Osteoarthritis Knee Study. Arthritis Res Ther, 9(5): R108, 2007.

37. King, J.; Stamper, D. L.; Schaad, D. C.; and Leopold, S. S.: Minimally invasive total knee arthroplasty compared with traditional total knee arthroplasty. Assessment of the learning curve and the postoperative recuperative period. J Bone Joint Surg Am, 89(7): 1497-503, 2007.

38. Knox, J.; Ambrose, H.; McCallister, W.; and Trumble, T.: Percutaneous pins versus volar plates for unstable distal radius fractures: a biomechanic study using a cadaver model. J Hand Surg [Am], 32(6): 813-7, 2007.

39. Lee, M.; Chebli, C.; Mounce, D.; Bertelsen, A.; Richardson, M.; and Matsen, F., 3rd: Intramedullary reaming for press-fit fixation of a humeral component removes cortical bone asymmetrically. J Shoulder Elbow Surg, 17(1): 150-5, 2008.

40. Lenters, T. R.; Davies, J.; and Matsen, F. A., 3rd: The types and severity of complications associated with interscalene brachial plexus block anesthesia: local and national evidence. J Shoulder Elbow Surg, 16(4): 379-87, 2007.

41. Lenters, T. R.; Franta, A. K.; Wolf, F. M.; Leopold, S. S.; and Matsen, F. A., 3rd: Arthroscopic compared with open repairs for recurrent anterior shoulder instability. A systematic review and meta-analysis of the literature. J Bone Joint Surg Am, 89(2): 244-54, 2007.

42. Linnau, K. F.; Blackmore, C. C.; Kaufman, R.; Nguyen, T. N.; Routt, M. L., Jr.; Stambaugh, L. E., 3rd; Jurkovich, G. J.; and Mock, C. N.: Do initial radiographs agree with crash site mechanism of injury in pelvic ring disruptions? A pilot study. J Orthop Trauma, 21(6): 375-80, 2007.

43. Luria, S.; Waitayawinyu, T.; Nemechek, N.; Huber, P.; Tencer, A. F.; and Trumble, T. E.: Biomechanic analysis of trapeziectomy, ligament reconstruction with tendon interposition, and tie-in trapezium implant arthroplasty for thumb carpometacarpal arthritis: a cadaver study. J Hand Surg [Am], 32(5): 697-706, 2007.

44. Lynch, J. R.; Cunningham, M. R.; Warme, W. J.; Schaad, D. C.; Wolf, F. M.; and Leopold, S. S.: Commercially funded and United States-based research is more likely to be published; good-quality studies with negative outcomes are not. J Bone Joint Surg Am, 89(5): 1010-8, 2007.

45. Lynch, J. R.; Franta, A. K.; Montgomery, W. H., Jr.; Lenters, T. R.; Mounce, D.; and Matsen, F. A., 3rd: Self-assessed outcome at two to four years after shoulder hemiarthroplasty with concentric glenoid reaming. J Bone Joint Surg Am, 89(6): 1284-92, 2007.

46. Lynch, J. R.; Taitsman, L. A.; Barei, D. P.; and Nork, S. E.: Femoral nonunion: risk factors and treatment options. J Am Acad Orthop Surg, 16(2): 88-97, 2008.

47. Lynch, J. R.; Waitayawinyu, T.; Hanel, D. P.; and Trumble, T. E.: Medial collateral ligament injury in the overhand-throwing athlete. J Hand Surg [Am], 33(3): 430-7, 2008.

48. MacLennan, A. J.; Nemechek, N. M.; Waitayawinyu, T.; and Trumble, T. E.: Diagnosis and anatomic reconstruction of extensor carpi ulnaris subluxation. J Hand Surg [Am], 33(1): 59-64, 2008.

49. Manner, P. A.: Universal health care and the single-payer system. Orthopedics, 30(8): 601, 604, 2007.

50. Martin, B. I.; Deyo, R. A.; Mirza, S. K.; Turner, J. A.; Comstock, B. A.; Hollingworth, W.; and Sullivan, S. D.: Expenditures and health status among adults with back and neck problems. Jama, 299(6): 656-64, 2008.

51. Martin, B. I.; Mirza, S. K.; Comstock, B. A.; Gray, D. T.; Kreuter, W.; and Deyo, R. A.: Are lumbar spine reoperation rates falling with greater use of fusion surgery and new surgical technology? Spine, 32(19): 2119-26, 2007.

52. Martin, B. I.; Mirza, S. K.; Comstock, B. A.; Gray, D. T.; Kreuter, W.; and Deyo, R. A.: Reoperation rates following lumbar spine surgery and the influence of spinal fusion procedures. Spine, 32(3): 382-7, 2007.

53. Matsen, F. A., 3rd: Clinical practice. Rotator-cuff failure. N Engl J Med, 358(20): 2138-47, 2008.

54. Matsen, F. A., 3rd; Bicknell, R. T.; and Lippitt, S. B.: Shoulder arthroplasty: the socket perspective. J Shoulder Elbow Surg, 16(5 Suppl): S241-7, 2007.

55. Matsen, F. A., 3rd; Chebli, C. M.; and Lippitt, S. B.: Principles for the evaluation and management of shoulder instability. Instr Course Lect, 56: 23-34, 2007.

56. Matsen, F. A., 3rd; Clinton, J.; Lynch, J.; Bertelsen, A.; and Richardson, M. L.: Glenoid component failure in total shoulder arthroplasty. J Bone Joint Surg Am, 90(4): 885-96, 2008.

57. Matsen Iii, F. A.; Boileau, P.; Walch, G.; Gerber, C.; and Bicknell, R. T.: The reverse total shoulder arthroplasty. Instr Course Lect, 57: 167-74, 2008.

58. Miller, B.; Dodds, S. D.; deMars, A.; Zagoreas, N.; Waitayawinyu, T.; and Trumble, T. E.: Flexor tendon repairs: the impact of fiberwire on grasping and locking core sutures. J Hand Surg [Am], 32(5): 591-6, 2007.

59. Mirza, S. K.: Either surgery or nonoperative treatment led to improvement in intervertebral disc herniation. J Bone Joint Surg Am, 89(5): 1139, 2007.

60. Mirza, S. K., and Deyo, R. A.: Systematic review of randomized trials comparing lumbar fusion surgery to nonoperative care for treatment of chronic back pain. Spine, 32(7): 816-23, 2007.

61. Molnar, R. B., and Routt, M. L., Jr.: Open reduction of intracapsular hip fractures using a modified Smith-Petersen surgical exposure. J Orthop Trauma, 21(7): 490-4, 2007.

62. Morgan, T. M.; Pitts, T. E.; Gross, T. S.; Poliachik, S. L.; Vessella, R. L.; and Corey, E.: RAD001 (Everolimus) inhibits growth of prostate cancer in the bone and the inhibitory effects are increased by combination with docetaxel and zoledronic acid. Prostate, 68(8): 861-71, 2008.

63. Oakley, B. B.; Francis, C. A.; Roberts, K. J.; Fuchsman, C. A.; Srinivasan, S.; and Staley, J. T.: Analysis of nitrite reductase (nirK and nirS) genes and cultivation reveal depauperate community of denitrifying bacteria in the Black Sea suboxic zone. Environ Microbiol, 9(1): 118-30, 2007.

64. Pfaeffle, H. J.; Waitayawinyu, T.; and Trumble, T. E.: Ulnar nerve laceration and repair. Hand Clin, 23(3): 291-9, v, 2007.

65. Poliachik, S. L.; Threet, D.; Srinivasan, S.; and Gross, T. S.: 32 wk old C3H/HeJ mice actively respond to mechanical loading. Bone, 42(4): 653-9, 2008.

66. Rubin, J. et al.: Caveolin-1 knockout mice have increased bone size and stiffness. J Bone Miner Res, 22(9): 1408-18, 2007.

67. Schiff, M. A.; Tencer, A. F.; and Mack, C. D.: Risk factors for pelvic fractures in lateral impact motor vehicle crashes. Accid Anal Prev, 40(1): 387-91, 2008.

68. Schildhauer, T. A.; Bellabarba, C.; Selznick, H. S.; McRoberts, D.; Vedder, N. B.; and Chapman, J. R.: Unstable pediatric sacral fracture with bone loss caused by a high-energy gunshot injury. J Trauma, 63(4): E95-9, 2007.

69. Schoots, I. G.; Simons, M. P.; Nork, S. E.; Chapman, J. R.; and Henley, M. B.: Antegrade locked nailing of open humeral shaft fractures. Orthopedics, 30(1): 49-54, 2007.

70. Srinivasan, S.; Ausk, B. J.; Poliachik, S. L.; Warner, S. E.; Richardson, T. S.; and Gross, T. S.: Rest-inserted loading rapidly amplifies the response of bone to small increases in strain and load cycles. J Appl Physiol, 102(5): 1945-52, 2007.

71. Standaert, C. J.; Manner, P. A.; and Herring, S. A.: Expert opinion and controversies in musculoskeletal and sports medicine: femoroacetabular impingement. Arch Phys Med Rehabil, 89(5): 890-3, 2008.

72. Tencer, A. F.; Kaufman, R.; Huber, P.; Mock, C.; and Rout, M. L.: Reducing primary and secondary impact loads on the pelvis during side impact. Traffic Inj Prev, 8(1): 101-6, 2007.

73. Waitayawinyu, T.; McCallister, W. V.; Nemechek, N. M.; and Trumble, T. E.: Scaphoid nonunion. J Am Acad Orthop Surg, 15(5): 308-20, 2007.

74. Waitayawinyu, T.; Parisi, D. M.; Miller, B.; Luria, S.; Morton, H. J.; Chin, S. H.; and Trumble, T. E.: A comparison of polyglycolic acid versus type 1 collagen bioabsorbable nerve conduits in a rat model: an alternative to autografting. J Hand Surg [Am], 32(10): 1521-9, 2007.

75. Waitayawinyu, T.; Pfaeffle, H. J.; McCallister, W. V.; Nemechek, N. M.; and Trumble, T. E.: Management of scaphoid nonunions. Orthop Clin North Am, 38(2): 237-49, vii, 2007.

76. Waitayawinyu, T.; Robertson, C.; Chin, S. H.; Schlenker, J. D.; Pettrone, S.; and Trumble, T. E.: The detailed anatomy of the 1,2 intercompartmental supraretinacular artery for vascularized bone grafting of scaphoid nonunions. J Hand Surg [Am], 33(2): 168-74, 2008.

77. Williams, S. L.; Bachison, C.; Michelson, J. D.; and Manner, P. A.: Component position in 2-incision minimally invasive total hip arthroplasty compared to standard total hip arthroplasty. J Arthroplasty, 23(2): 197-202, 2008.

78. Woodhouse, E.; Schmale, G. A.; Simonian, P.; Tencer, A.; Huber, P.; and Seidel, K.: Reproductive hormone effects on strength of the rat anterior cruciate ligament. Knee Surg Sports Traumatol Arthrosc, 15(4): 453-60, 2007.

79. Yang, L.; Clinton, J. M.; Blackburn, M. L.; Zhang, Q.; Zou, J.; Zielinska-Kwiatkowska, A.; Tang, B. L.; and Chansky, H. A.: Rab23 regulates differentiation of ATDC5 chondroprogenitor cells. J Biol Chem, 283(16): 10649-57, 2008.

Contributors to Departmental Research and Education

April 2007 Through March 2008

We express our appreciation to all who have contributed to the ongoing discoveries by 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. We owe a special thanks to the University of Washington Resident Alumni who have made significant contributions to help further the education of our current residents. We have tried to include in this list all who contributed; if anyone was overlooked, please be sure to let us know!

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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. If you have any questions, please contact our Chair, Rick Matsen (matsen@u.washington.edu), or our Administrator, Ken Karbowski (kkarb@u.washington.edu).

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