VOLUME 23 2003

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THE IOWA ORTHOPAEDIC JOURNAL

VOLUME 23, 2003

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INSTRUCTIONS TO AUTHORS

Any article relevant to orthopaedic surgery, orthopaedic science or the teaching of either will be considered by *The Iowa Orthopaedic Journal* for publication. Articles will be enthusiastically received from alumni, visitors to the department, members of the Iowa Orthopaedic Society, residents and friends of The University of Iowa Department of Ortho-paedics. The journal is published annually in May or June. The deadline for receipt of articles for the 2004 journal is February 10, 2004.

Published articles and their illustrations become the property of the journal. *The Iowa Orthopaedic Journal* is oeer reviewed and referenced in the *Index Medicus*. Articles previously published will not be accepted unless the content has been significantly changed.

When submitting an article, it is essential that the following items be included:

1. The **original manuscript complete with illustrations**. The corresponding author must be clearly identified with address and telephone number. Manuscripts of accepted articles will not be returned.
2. A **bibliography** arranged in alphabetical order and double-spaced. Only those works referenced in the text should be included
3. **Legends** for all illustrations submitted, listed in order and double-spaced.
4. **Illustrations**
   a. One set of 5x7 inch, black-and-white, glossy prints of all photographs.
   b. *Original* drawings or charts.
   c. Color illustrations will not be used unless, in the opinion of the journal, they convey information not available in a black and white. If color is desired, please send sets of both color and black-and-white prints.
   d. Please follow the format in previous editions of *The Iowa Orthopaedic Journal* or *The Journal of Bone and Joint Surgery*.
5. **Electronic copies** of all items one through four above (photographs may be exempted from this requirement). These may be sent via floppy disk, Zip disk or email (to diana-johannes@uiowa.edu).

Preparation of manuscripts: Manuscripts must be typewritten and double-spaced using wide margins. Write out numbers under 10 except percentages, degrees, or numbers expressed as decimals. Direct quotations should include the exact page number on which they appeared in the book or article. All measurements should be given in SI metric units. In reporting results of surgery, only in rare instances can cases with fewer than two years' follow-up be accepted.

Preparation of illustrations: On the back of each illustration, write the figure number, author's name and indicate the top. Write the author's name on the back of each illustration. Send prints unmounted; paste or glue will damage them. Drawings, charts, and lettering on prints should be done in black; use white backgrounds. Put dates or initials in legends, not on prints. Make lettering large enough to be read when drawings are reduced in size. When submitting an illustration that has appeared elsewhere, give full information about previous publication and credit to be given, and state whether or not permission to reproduce it has been obtained.

Additional copies of these instructions may be obtained at www.medicine.uiowa.edu/ortho/ioj.htm.

Printed on acid-free paper effective with Volume XV, 1995.

EDITORS' NOTE

It is our honor and privilege to present the twenty-third edition of the *Iowa Orthopaedic Journal*. It has been an enjoyable and educational experience acting as editors this year, and we feel our work has been quite rewarding. We hope you find this edition thought-provoking and applicable to your practice. The *Journal*'s previous editors have certainly set a very high standard, and we hope to have maintained this standard.

In keeping with the tradition of the *Iowa Orthopaedic Journal,* we have provided the reader with general review manuscripts, articles in both clinical and basic science, case reports and reports of historical and philosophical interest. While our Orthopaedic department is well represented in the *Journal* (with submissions from residents, faculty, alumni and others with direct ties to the University of Iowa), clinicians and scientists worldwide submit articles for publication, and we encourage that. This can only improve the quality of the *Iowa Orthopaedic Journal* and reflect positively on our reputation.

Another of the rich traditions of the *Iowa Orthopaedic Journal* is the dedication of each edition. This year's edition is dedicated to Dr. Stan James (University of Iowa B.A. in 1953, M.D. in 1962, Iowa Orthopaedic Resident from 1964 to 1967). As a medical student and resident, Dr. James developed an interest in the basic science of ligamentous healing while working with Professor Charles Tipton and Dr. Ignacio Ponseti. While a resident here, Dr. James received intensive training in hand surgery with Dr. Adrian Flatt, and he subsequently began his practice as a hand surgeon in Eugene, Oregon, with Dr. Donald Slocum. Dr. James received an academic appointment at the University of Oregon in the early 1970's and began several studies in sports medicine. Over the years, his interests led him to alter his practice from hand surgery to sports medicine.

We would like to extend our special appreciation to faculty advisors, Dr. Jose Morcuende and Dr. Joseph Buckwalter, who provided guidance in the review of articles and practical advice on completing a quality edition of the *Journal*. In addition, several faculty provided analysis and suggestions for articles. Diana Johannes, our secretarial assistant, spent considerable hours working on this edition with us and behind the scenes, in addition to providing countless resources in producing this year's *Journal*. We would also like to gratefully acknowledge the generous contributions of our corporate sponsors who provide the financial backing for the *Journal.*

Finally, we encourage and look forward to your feedback regarding this year's *Journal*. We hope to continually improve the *Iowa Orthopaedic Journal* and make it a usable and important resource for your continuing education.

Mark Hagy, M.D.
Christopher Sliva, M.D.

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BONFIGLIO EDUCATIONAL ENDOWMENT FUND

In honor of Dr. Michael Bonfiglio's distinguished career, the University of Iowa Orthopaedic Department initiated a campaign for the Bonfiglio Orthopaedic Education Endowment in 1994. This serves as permanent recognition of Dr. Bonfiglio's commitment to the department and provides a variety of educational materials and activities for the fellows, residents and students. The new department Education Center was dedicated to Dr. Bonfiglio in September 1995 at the Iowa Orthopaedic Alumni Meeting. It includes a collection of microscopic slides and imaging studies, computers, educational computer software and literature-search capabilities, audiovisual equipment and educational programs.

The goal is to raise enough funds so that the Bonfiglio Endowment will support the Center's educational endeavors. In this way, the Center will enhance training opportunities for medical students, orthopaedic residents and fellows, clinicians and allied health care personnel for years to come.

Gifts and pledges to the Endowment should be directed to the Bonfiglio Educational Endowment Fund and qualify as charitable contributions.

Address:

Bonfiglio Educational Endowment Fund
University of Iowa Health Care
University of Iowa Hospitals and Clinics
Department of Orthopaedic Surgery, JPP
200 Hawkins Drive
Iowa City, IA 52242-1088

The Michael Bonfiglio Award for Student Research in Orthopaedic Surgery

The Iowa Orthopaedic Society Medical Student Research Award for Musculoskeletal Research

The University of Iowa Department of Orthopaedic Surgery, along with the Iowa Orthopaedic Society, sponsors two research awards involving medical students.

The first, the Michael Bonfiglio Award, originated in 1988 and was named in honor of Mike, who had an avid interest in students, teaching and research. The award is given annually at a medical convocation. It consists of a plaque and a stipend to be used for the purchase of an orthopaedic text. It is awarded to a senior medical student in the UI Carver College of Medicine who has done outstanding orthopaedic research during his or her tenure as a medical student. The student often has an advisor in the Orthopaedic Department; however, the student must have played a major role in the design, implementation and analysis of the project. They must also be able to defend the manuscript in public forum. The research project may have been either clinical or basic science, and each study is judged on the basis of originality and scientific merit. The winner presents the work at the April meeting of the Iowa Orthopaedic Society. This year's award winner is Jesse Templeton.

The second award is the Medical Student Research Award for Musculoskeletal Research, for students in the UI Carver College of Medicine who provide a research project involving orthopaedic surgery during one of their first three years. The award consists of a $2000 stipend, $500 of which is designated as a direct award to the student and $1500 of which is designated to help defray continuing costs of the project and its publication. The student must provide an abstract and a progress report on the ongoing research. The aim of this award is to stimulate research in the field of orthopaedic surgery/musculoskeletal problems.

This year, the committee, consisting of members of the Iowa Orthopaedic Society (Drs. Sterling Laaveg and Douglas Cooper) as well as members of the Orthopaedic Surgery Department (Drs. Charles Saltzman and Charles Clark), took a bold stance and recommended that the award be given to four students, since the purpose of the award is to stimulate interest in musculoskeletal research. All four of the awarded projects were deemed to be worthy of our acknowledgment. Farid Moussavi-Haami, Egon Ozer, Andrea Buckwalter and Brian Ludwig will divide the $2000 stipend.

I personally believe that the Michael Bonfiglio Award and the Medical Student Research Award for Musculoskeletal Research are very prestigious, recognizing student research on the musculoskeletal system. These awards have indeed attained their goal of stimulating such research and have produced many fine projects over the years.

Charles R. Clark, M.D.
Michael Bonfiglio Professor of Orthopaedic Surgery

Department of Orthopaedics

Sergio Mendoza
2003-present

Jose Morcuende
2001-present

Annunziato Amendola
2001-present

Joseph Chen
2000-present

Todd McKinley
1999-present

R. Kumar Kadiyala
1998-present

Leon Grobler
1996-1999

Brian Adams
1993-present

Charles Saltzman
1991-present

John Callaghan
1990-present

David Tearse
1989-2000

Ernest Found
1987-present

Lawrence Marsh
1987-present

Curtis Steyers
1985-present

James Nepola
1984-present

Fred Dietz
1984-present

James Weinstein
1983-1996

Barbara Campbell
1982-1984

Charles Clark
1980-present

William Blair
1980-1997

William Pontarelli
1980-1984,
1999-present

Joseph Buckwalter
1979-present

Thomas Lehmann
1978-1987

Stuart Weinstein
1976-present

Arthur Steindler
1912-1949

Theodore Willis
1917-1918

Joseph Milgram
1926-1932

Ernest Freund
1932-1936

Thomas Waring
1932-1939

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1936-1939

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1946-present

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1946-1952

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1948-1956

Michael Bonfiglio
1950-1995

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1950-1978

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1956-1979

Reginald Cooper
1962-present

Howard Hogshead
1964-1965

Maurice Schnell
1964-1965

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1967-1970,
1998-present

Donald Kettelkamp
1968-1971

Gerald Laros
1968-1971

Richard Stauffer
1970-1972

John Albright
1971-present

Doug Mains
1972-1973

Bruce Sprague
1972-1979

Richard Brand
1974-2002

Mike Mickelson
1976-1981

The University of Iowa
Roy J. and Lucille A. Carver College of Medicine

2003 GRADUATING SENIOR RESIDENTS

Jay C. Albright, M.D.

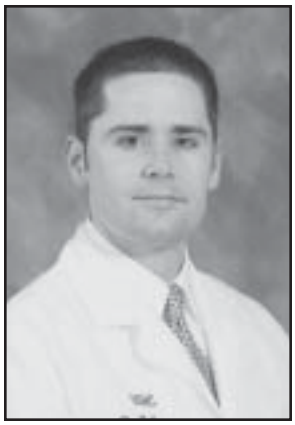
Jay was born in New Haven, Connecticut, but was raised in Iowa City, Iowa, the son of University of Iowa orthopaedic surgeon John P. Albright, M.D. Jay "committed a family felony" by attending the University of Michigan for his undergraduate studies. Thereafter, he attended The University of Iowa Medical School (before it was renamed).

Jay and wife Stacy, with son Jackson, will be going to San Diego, California after Jay completes his residency here. He will train in Pediatric Orthopaedics under Dennis Wenger, M.D., also an Iowa graduate. After the year in San Diego, Jay and his family will continue on to Auckland, New Zealand where he will train in Sports Medicine, and practice Pediatric Orthopaedics.

Jay adds, "I have had the incredible experience to train under my father, which has taught me a lot about myself and who I want to be. I hope that I have lived up to this example that has been set before me."

Karen E. Evensen, M.D.

Karen Evensen was born in the Upper Peninsula of Michigan to parents Harold and Liz Evensen. She was raised in Houghton, Michigan, along with two brothers, Hal and David. She earned her undergraduate degree in Biological Sciences at Michigan Technological University, then went on to medical school at The University of Michigan in Ann Arbor. Her hobbies during residency have included running (though less frequently than in the past), as well as spending time with her nearby nieces and nephew, Claire, Leah and Ethan. Next year she looks forward to heading out to sunny Los Angeles to enter a Musculoskeletal Tumor fellowship with Dr. Jeffrey Eckardt at The University of California, Los Angeles.



Jay D. Keener, M.D.

Jay was born in Detroit, Michigan, and later moved to Scott Depot, West Virginia. He earned a Bachelor of Science degree in Physical Therapy from West Virginia University in 1991. After a three-year career as a physical therapist, he entered medical school, receiving his M.D. degree from West Virginia University in 1998.

Jay and wife Tracy have a two-year-old son, Garrett. This summer, the Keeners will move to St. Louis where Jay will complete a fellowship in shoulder and elbow reconstruction at Washington University Hospitals under the direction of Dr. Ken Yamaguchi.

Joseph G. Khoury, M.D.

Joe was born and raised in Iowa City, Iowa. He received both his Bachelor of Arts and M.D. degrees at The University of Iowa. He met his wife, Carrie, while in college and they have been married five years. They have a one-and-one-half year old daughter, Anna. Joe enjoys movies, computer games and spending time with his family.

Joe plans a fellowship in pediatric orthopedics next year in Atlanta with Raymond Morrissey. After that fellowship, Joe and Carrie would like to live and work in the Midwest.

2003 GRADUATING SENIOR RESIDENTS (continued)

Stephen L. Knecht, M.D.

Steve was raised in Idaho Falls, Idaho, and attended high school there and in Salt Lake City, Utah, at Rowmark Ski Racing Academy. He studied chemistry at Oberlin College in Ohio, with a brief one-year "beach interlude" at U.C. Santa Barbara. Post-graduate career exploration took him to Los Angeles for a job in advertising, then back to Salt Lake City as a ski and mountain bike bum. Steve was accepted to New York Medical College in 1994, hung up the skis one more time, and convinced his parents to adopt his dog (which they can't live without now). His interest in orthopaedics was solidified during his rotation at the Hospital for Joint Diseases in New York City.

After residency, Steve will enter an Orthopaedic Trauma Fellowship in Auckland, New Zealand. After that, he plans to return home to the mountains of the western United States to join a general orthopaedics practice.

2002-2003 FELLOWS

Anthony H. Nguyen, M.D.

Anthony moved to the United States from Vietnam with his family in 1975. He grew up in northern California. He went to medical school at St. Louis University and completed an otolaryngology residency in Buffalo, New York. After being in private practice for one year as an Ear, Nose and Throat doctor, he wanted to broaden his surgical experience, and spent two years in New Orleans, Louisiana, to study plastic surgery. His exposure to and interest in hand surgery led him to Iowa for a one-year hand fellowship. He states, "The experience I've had here in Iowa has been amazing, such a relaxed atmosphere, so many superb surgeons." He will be joining a hand group in southern California after his fellowship in Iowa.

Soheil Najibi, M.D.

Soheil is originally from Persia and is fluent in Farsi, the language of that area. He immigrated to the United States in 1984 and grew up in northern California. He received his bachelor's degree from the University of California at Berkeley and went to Boston for his medical school training and residency. He received his M.D. and Ph.D. degrees from Boston University and completed his orthopaedic surgery residency at the same institution. Currently, he is the Sports Medicine Fellow in Orthopaedics at The University of Iowa. On completion of his fellowship here, he will begin a full-time academic orthopaedic faculty position at Henry Ford Hospital in Detroit.

NEW ORTHOPAEDIC FACULTY

Sergio Andres Mendoza Lattes, M.D.

Sergio Mendoza joined The University of Iowa Department of Orthopaedic Surgery faculty in January of 2003, as part of our Spine Service. Sergio is originally from Chile and has lived abroad for most of his life. Because of this he has attended eight different schools and is fluent in both Spanish and German. He received his medical degree at The Catholic University in Santiago, Chile, in 1984. He also completed an executive M.B.A. there, as well as his orthopaedic training. He has been in practice since 1996 and spent three years at a regional trauma institute where he did most of his spine specialty training. In 1999, he joined the academic ranks at The Catholic University in Santiago as a full-time orthopaedic faculty member.

He arrived in Iowa with his family in 2001, intent on increasing his knowledge in deformity surgery. His one-year plan became a long-term plan thanks to the "warmth and kindness" he and his family have found in the people of Iowa. "You have something very special here," he states.

Outside of his practice, his highest interest is in the four women in his life: his wife, Sandra Bruon Stocchero, and daughters Sofia, Consuelo and Constanza. Because he comes from mountain country, he is a passionate skier.

His main academic interests are non-fusion technology in degenerative lumbar spine disorders, the natural history of spinal stenosis, and minimally invasive access to the spine.

Department of Orthopaedics
2002-2003

First row (L to R): Sergio Mendoza, John Albright, Reginald Cooper, Joseph Buckwalter, Mark Hagy

Second row (L to R): Todd McKinley, Michael Huang, Joseph Khoury, Curtis Steyers, Kumar Kadiyala, Jose Morcuende, Michael Sander

Third row (L to R): Karen Evensen, Robert Kadoko, Soheil Najibi, Brian Adams, Jay Albright, Anthony Mollano, Kirk Clifford

Fourth row (L to R): John Callaghan, Lawrence Marsh, William Pontarelli, Stephen Knecht, Jay Keener, Phinit Phisitkul

Fifth row (L to R): Ernest Found, John Bell, Richard Johnston, Ned Amendola, Geoffrey Haft, Phillip Langer

2003-2004 SCHEDULE OF LECTURESHIPS AND CONFERENCES

(Larson Conference Room, 01090 JPP)

Carroll B. Larson Shrine Memorial Lecture
May 9-10, 2003

Reginald R. Cooper
Orthopaedic Leadership Lectures
Friday and Saturday, April 25-26, 2003
Richard Cruess, M.D., Professor of Surgery
McGill University, Montreal, Quebec, Canada

Sylvia Cruess, M.D., Assoc. Professor of Medicine
McGill University, Montreal, Quebec, Canada

2003 Senior Residents and Fellows Day
June 6-7, 2003
Michael A. Kelly, M.D.
Department of Orthopaedic Surgery
Insall-Scott-Kelly Institute
New York, New York

James A. Nunley II, M.D.
Professor and Chief
Department of Orthopaedics
Duke University Medical Center
Durham, North Carolina

Iowa Orthopaedic Alumni Meeting
October 16-18, 2003
Roy Sanders, M.D.
Michael Bonfiglio Visiting Professor
Tampa, Florida

Laura Tosi, M.D.
First Ruth Jackson Lecturer
Washington, D.C.

Hawkeye Sports Medicine Symposium
December 5-6, 2003
James Andrews, M.D.
Alabama Sports Medicine & Orthopaedic Center
1201 11th Avenue South, Suite 200
Birmingham, Alabama 35205

Carroll B. Larson Shrine Memorial Lecture
Spring 2004
(Speakers tba*)

2004 Senior Residents and Fellows Day
June 11-12, 2004

Seventh Biennial Johnston Lectureship
In Hip Reconstruction
Fall, 2004
(Speakers tba*)

* to be announced

STAN JAMES, M.D.
IOWA ORTHOPAEDIC JOURNAL DEDICATION

A native of Iowa City, Stan James graduated with a degree in physical education from the University of Iowa in 1953. He then spent four years serving his country in the Army as an aviator. Following his military obligation, he entered University of Iowa School of Medicine, graduating in 1962. He spent a year in Oregon for his internship and completed an orthopaedic surgery residency at the University of Iowa in 1967.

His interest in sports medicine began while a resident. He worked with the UI Exercise and Physiology Laboratory and noted researcher, Charles Tipton, who at the time was studying the effects of exercise on ligaments and ligament healing. These original animal studies contradicted medical practice at the time and ultimately led to fundamental changes in the treatment of ligamentous injuries. In addition, James worked with then Hawkeye team physician, "Shorty" Paul, caring for UI athletes.

Following residency, he joined a practice in Eugene, Oregon established by Iowa alumnus, Don Slocumb. He joined the group as a hand surgeon having been trained extensively by Dr. Adrian Flatt while a resident, in order to fill a void the practice had in upper extremity. As a hand surgeon, he began treating athletes from the University of Oregon and Oregon State University. This portion of his practice dramatically expanded over his career, leading him to author a number of papers most notably on the biomechanics of running and ACL reconstruction. Because of his expertise, a number of elite athletes in the NBA, NFL, track and tennis have sought his care. Through this excellence, he served as a pioneer in the field of sports medicine.

One of his more intriguing endeavors was as a consultant with Nike during the company's infancy. Nike co-founder Phil Knight and legendary Oregon track coach Bill Bowerman asked James to consult on the development of the company's footwear. James analyzed film of Oregon track athletes, evaluating running patterns and gait. These study sessions led to the development of prototypes, whose innovations in design led to the creation of the early line of Nike shoes.

In addition, James is also an accomplished cross-country skier. Holding two National Master Championships in 20k and 10k freestyle events. He also continues to run and was recently quoted in the Iowa Alumni Journal stating, "I began running before the running boom, and I used to run at night so that people would not see me and think I was crazy."

We would like to dedicate this journal to Dr. Stan James, an orthopaedist whose interest in research provoked fundamental change in patient care and whose devotion to orthopaedic surgery should serve as an example to us all.

CLINICAL BIOMECHANICS OF WEAR IN TOTAL HIP ARTHROPLASTY

John J. Callaghan, M.D.; Douglas R. Pedersen, Ph.D.; Richard C. Johnston, M.D.; Thomas D. Brown, Ph.D.

ABSTRACT

Complementary clinical and laboratory studies were performed to identify variables associated with polyethylene wear following total hip replacement, and to elucidate the mechanisms responsible for accelerated wear in the total hip arthroplasty construct.

Observational cohort studies were performed using a prospective clinical database of more than 4000 consecutive primary total hip arthroplasties performed by a single surgeon, to identify wear-related variables. These variables included head size, acetabular/femoral component impingement, and third body debris. Novel digital edge detection techniques were developed and employed to accurately measure wear, and to determine the relationships of head size and third body debris to acceleration of wear. A novel sliding-distance-coupled finite element model was formulated and employed to examine the mechanisms responsible for wear. The long-term cohort studies demonstrated smaller head sizes to be associated with less wear. Third body debris generated from cable fretting was associated with an increase in wear, osteolysis, and acetabular loosening, especially with larger head sizes. The sliding-distance-coupled finite element model replicated the wear rates occurring *in vitro* and *in vivo*, demonstrating the importance of sliding distance on polyethylene wear following total hip arthroplasty. It also demonstrated substantial increases in wear associated with femoral head scratching from third body debris. Further extension of the finite element formulation demonstrated the potential for acetabular component rim damage from impingement wear, and the enhanced potential for third body ingress to the bearing surface with larger head sizes. Edge detection wear measurement techniques demonstrated that early wear rates were predictive of long-term wear rates.

These complementary clinical and laboratory investigations have provided insight into 1) the significance of sliding distance and physiologic loci of motion as contributing factors in minimizing wear, 2) the deleterious effects of third body particulates in accelerating wear, 3) the potential for, and factors related to, impingement wear, and 4) the potential advantages and compromises related to the use of larger head sizes in the bearing surface construct.

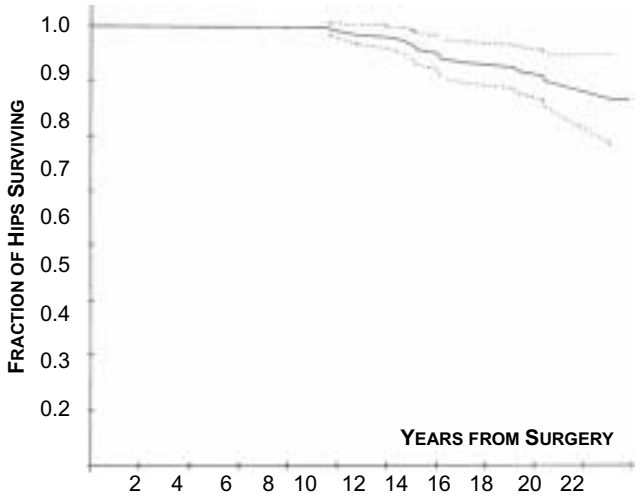


Figure 1. Survivorship curve (solid line) and 95% confidence intervals (dashed lines) for Charnley prostheses implanted by the senior author (RCJ) using first generation cementing techniques. The endpoint is aseptic loosening of the acetabular component, confirmed at revision. (Schulte et al., JBJS 1993).

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INTRODUCTION

Almost a decade ago, when the authors reported the first minimum twenty year follow-up of total hip replacement in North America[48], they confirmed the findings

TABLE 1
Results of wear in cohorts of hips with various acetabular-femoral head articulations

Follow-up Period Cohorts	Linear Wear Mean (mm/year)	Linear Wear Standard Deviation	Volumetric Wear Mean (mm^3/year)	Volumetric Wear Standard Deviation
5 Year				
22-mm machined	0.12	0.07	45.22	27.14
22-mm molded	0.11	0.12	40.91	46.97
28-mm molded	0.14	0.13	89.27	79.70
28-mm molded metal back	0.11	0.07	64.70	45.83
7–8 Year				
28-mm cementless metal back	0.11	0.06	65.78	39.01
10 Year				
22-mm machined	0.12	0.06	48.36	24.51
22-mm molded	0.08	0.06	32.71	24.59
28-mm molded	0.12	0.10	70.88	59.61
15 Year				
22-mm machined	0.11	0.07	41.20	25.79
22-mm molded	0.09	0.06	34.59	22.65
20–22 Year				
22-mm machined	0.10	0.07	40.69	26.24

(Pedersen et al., ASTM 1994; Callaghan et al., CORR 1995)

of others that osteolysis, acetabular loosening, and polyethylene wear were the major long term problems associated with the total hip arthroplasty procedure (Figure 1).

Although the conventional wisdom at the time was that head sizes in the range of 28 millimeters were associated with lower wear rates than either 22 or 32 millimeter heads,[28] the authors observed lower linear and volumetric rates of wear with 22 millimeter femoral heads[12] (Table 1). This initial observation, along with our reports[9,30] of osteolysis around secure cementless total hip arthroplasty devices helped redirect investigative efforts away from cement as the leading cause of failure in the total hip arthroplasty construct. Instead, attention moved toward investigation of clinical variables and biomechanical mechanisms associated with polyethylene wear. Since that time, a unique single-surgeon database with well-maintained serial radiographs over a thirty-year follow-up period, and laboratory efforts in experimental and computational biomechanics, enabled the authors to conduct an integrated series of complementary clinical and laboratory studies of total hip arthroplasty wear, aimed at identifying causative factors and elucidating underlying

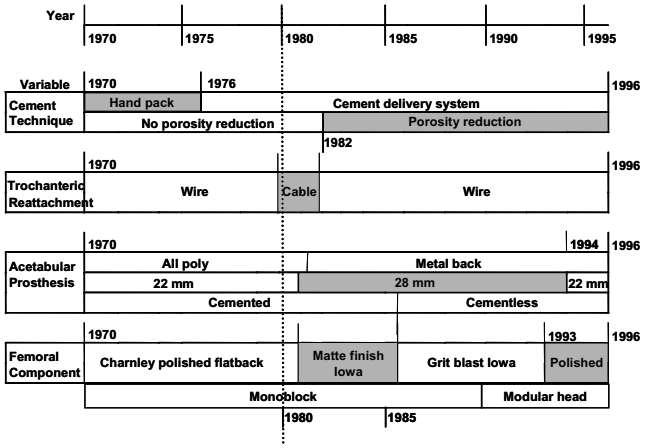

Figure 2. Changes made in prosthesis design or implantation technique over a 26-year period of 4164 hip replacements. Only ten changes were involved, and only two of those changes occurred simultaneously: 22⇒28 mm head size, and Charnley polished flatback⇒Iowa matte finish, in 1981.

mechanisms.[2,3,7,10,11,12,13,23,25,26,27,29,32,33,34,36,40,44,49,53,54,57]

The prospective single-surgeon database included more than 4000 consecutive primary total hip replacements. Only a few specific changes were made in either the design or technique, and these few changes

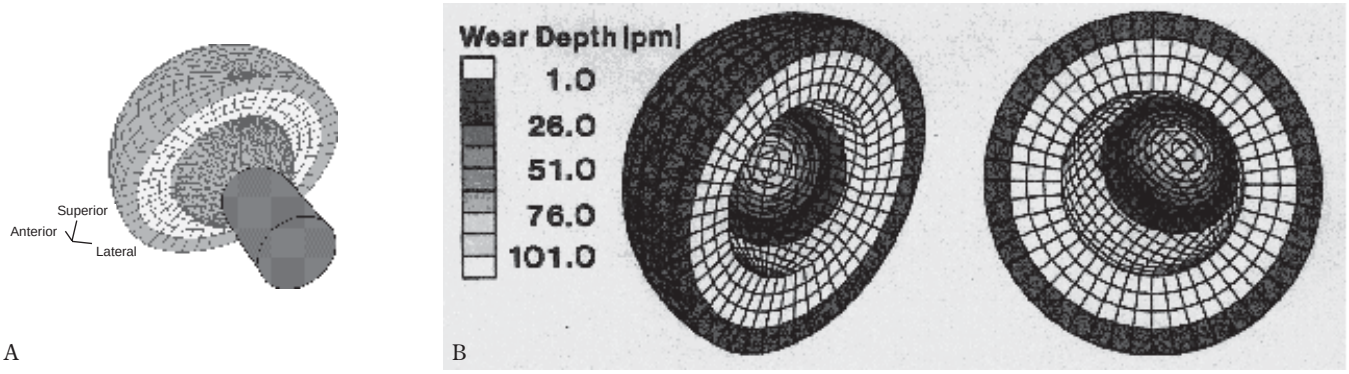

Figure 3. A three-dimensional finite element (FE) model of total hip reconstruction is used to calculate per-gait-cycle wear (in picometers, 10^{-12} meters) of the polyethylene liner of the acetabular component.

occurred at well-prescribed time points. This constituted unique material for performing observational cohort studies related to wear of polyethylene following total hip arthroplasty (Figure 2). Through these clinical studies, the authors identified a number of variables relevant to the understanding of wear mechanisms. This guided the development of laboratory models to elucidate underlying mechanisms of wear.

Over the last decade these authors have tested the following hypotheses:

Hypothesis 1: Across large clinical series, the relative long-term wear performance of contemporary design variants can be reliably predicted directly from their respective articulation dynamics (contact stress and sliding kinematics).

Hypothesis 2: Across large clinical series, long-term polyethylene wear and late loosening can be reliably predicted on the basis of early wear behavior.

Hypothesis 3: In otherwise similar constructs, third body particulate debris causes predictably accelerated polyethylene wear, leading to early radiographic and mechanical failure.

Hypothesis 4: The accelerated effects of third body wear associated with larger head sizes depend on debris access to the bearing surface, mediated by fluid convection.

Hypothesis 5: The polyethylene acetabular component rim damage observed at the time of component retrieval during revision hip surgery is associated with impingement of the femoral head, neck and collar on the acetabular polyethylene liner.

MATERIALS AND METHODS

Clinical Materials

Over a 26-year period the senior investigator (RCJ) performed more than 4000 primary total hip replacements, with very few and discrete changes in the designs and technique used (Figure 2). All radiographs and clinical data were prospectively accumulated, and were used to evaluate revision, radiographic loosening, osteolysis, and wear in a standard manner[4,20] over the last decade.[2,3,7,11,13,23,25,26,29,36,48,54,57] The follow-up studies involved comparisons between cohorts, aimed at identifying how specific changes in implant design or operative technique influenced long term outcome. This group of patients and their radiographs were used to determine variability in wear rates related to femoral head size, to predict late wear rates from early wear rates as determined by digital edge detection techniques, and to evaluate the effect on wear, loosening, and osteolysis of third body debris from cables used to reattach the greater trochanter.

Analytical Methods

To complement the long-term clinical data and to elucidate the mechanisms of wear, as well as to more reliably predict and measure wear, several conceptually new analytical paradigms were developed.

Sliding-distance-coupled finite element model

Three-dimensional nonlinear contact finite element analysis of total hip replacement was linked to interface sliding kinematics, enabling (for the first time) parametric computational study (Figure 3) of polyethylene wear rates and spatial wear distributions.[32,33,34] Hip resultant loads from an inverse Newtonian gait analysis model (validated *in vivo* with an instrumented implant) were used in the FE analysis to determine contact stress distributions on the polyethylene bearing surface (Figure 4) at each of 16 discrete instants of stance phase.[6,16,17,44] Incremental sliding distances of points on the femoral head (Figure 5) were computed from cor-

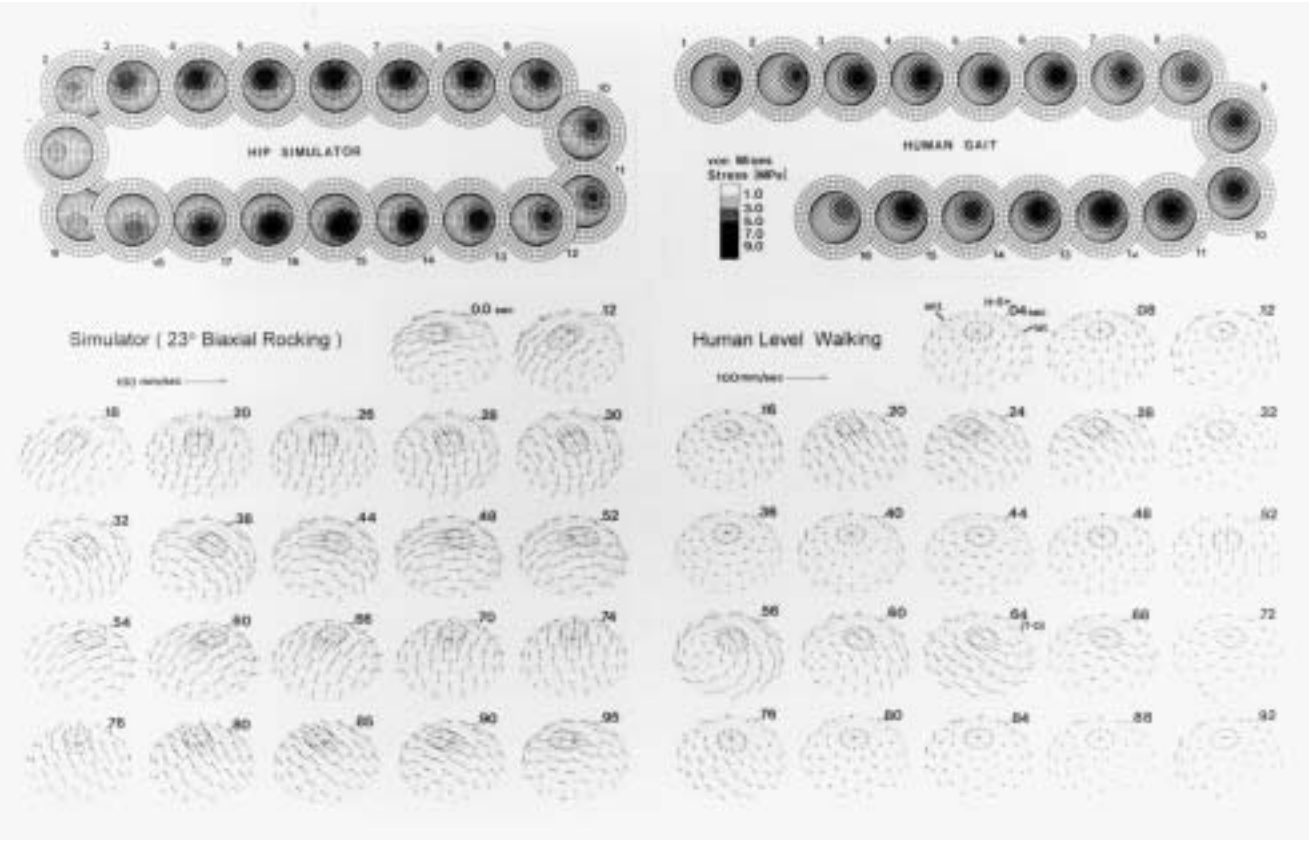

Figure 4. Time-wise variations of contact stress and sliding distance during the articulation cycle in THA. A finite element model (Figure 3a) is used to compute 3-dimensional polyethylene contact stress distributions (contour plots) at serial time points in the gait cycle. Corresponding distributions of bearing surface sliding velocity (vector plots) are determined from recordings of the three-dimensional joint motion patterns. These data are then input to a modified version of the Archard equation. Note that both the contact stress distribution history and the sliding distance history for conventional laboratory wear simulations (left panels) are markedly different from those for human locomotion (right panels), implying very different wear behavior.

responding flexion/extension kinematics.[44] Wear rates were determined by a custom-written computer program that implemented Archard's relationship,[1] coupling contact stress, sliding distance, and a tribologically-based wear coefficient. Later, adaptive remeshing capability was added to the FE model.[34] This feature was introduced to account for conformity changes accompanying progressive removal of polyethylene wear material from the bearing surface, thus allowing for extension of the postoperative wear simulations to the clinically more significant long term regime (as long as 20 years).

The algorithm's temporal convergence (i.e., the minimum frequency of remeshing updates needed to ensure a well-behaved solution) was investigated, with the finding that under most circumstances it was reasonable to make such updates at intervals of about $5x10^5$ loading cycles, corresponding to about 6 months of average *in vivo* service. To physically validate the model, a collaborative study was undertaken with colleagues experienced in laboratory wear measurement, to determine whether the finite element model could accurately predict wear occurring in a laboratory hip simulator.[33] The collaborative study design used two cohorts of otherwise identical hemispherical cups, one cohort having a 22-mm bearing surface diameter and the other having a 28-mm diameter. Both cup cohorts were subjected to 3 million cycles at 1 Hz in a biaxial rocking hip simulator, programmed with a Paul-type loading curve, articulating against similarly polished stainless steel balls with bovine serum lubrication. Volumetric wear was measured gravimetrically. Since the true wear coefficient prevailing in the experiments was not known *a priori*, iterative comparisons of computed versus experimentally measured volumetric wear for the 22-mm cups were performed to arrive at a specific wear coefficient value for which computation was brought into identical agreement with the physical measurements. Then, assuming that this same wear coefficient prevailed for the physical testing of the 28-mm cups (all tribological factors

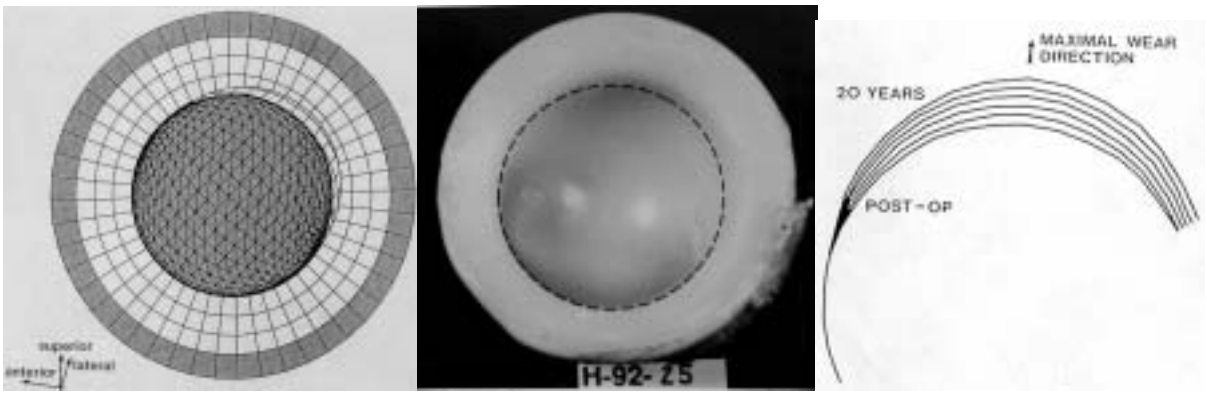

Figure 5. Long-term wear behavior was computed using the adaptively-meshed sliding-distance-coupled finite element model. Material removal at late times was based upon extrapolation of per-gait-cycle wear depth distributions (Figure 3b), but with the finite element mesh (and therefore the contact stress distributions) periodically updated to reflect material removal. Patterns of computed long-term wear (left) were consistent with material loss patterns on retrieval cups (middle), and in profile corresponded to unidirectional "test-tube" wear front advance (right).

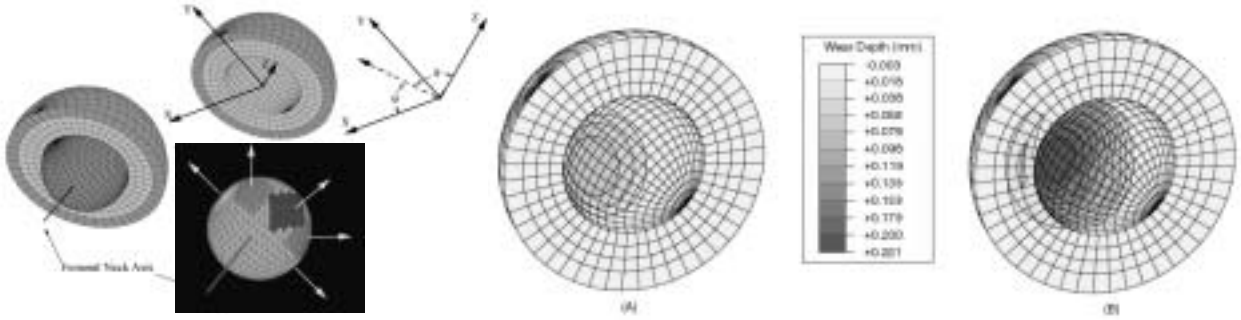

Figure 6. Site-specific wear coefficients are assigned to model local head roughening effects. A patch of Bezier surface facets, which define the femoral head (left panel), could be assigned an elevated wear coefficient value, associated with roughening. Effects of local roughening (wear coefficient = 1.065 X 10^{-7} mm2/N) on computed acetabular wear were simulated for 10^6 cycles of walking motion (B). Compared to the situation for an undamaged femoral head (A), a 2.13-fold increase in computed volumetric wear was induced. Note also that, for the roughened femoral head, the wear tract becomes less regular than the classic "test tube" pattern.

ostensibly being identical between the two cup cohorts), finite element trials were performed for the 28 mm case, and the volumetric wear results were compared with those obtained physically. Computed wear values agreed with measurements to within 4.1%, a discrepancy of less than one half of one standard deviation of the experimental measurements themselves.

These initial total hip arthroplasty wear simulations involved spatially uniform counterface roughness. These were (gait cycle) temporal-spatial integrations of the product of instantaneous local contact stress σ, times instantaneous local counterface sliding speed ϖ, times a spatially uniform wear coefficient κ, in hip-centered spherical coordinates. To study the effects of head roughening (from head scratches from 3rd body debris) in accelerating wear, algorithmic logic was developed to link head-based and cup-based coordinate systems, thus allowing identification of the head surface site apposing any given acetabular surface site at any given point in time. By means of an automated computational lookup table storing the wear coefficient for each of the large number of sectors (Bezier surface facets) making up the head surface, appropriate time- and site-specific wear coefficient variations were supplied for gait cycle Archard integrations, at each acetabular finite element node. Hence, a non-uniform κ, as in the case of regions of femoral head scratching created by 3rd body wear, could be incorporated at any site of the finite element model femoral head (Figure 6). Even with a relatively small roughened region (5% of head surface area) the resulting volumetric wear was substantially increased, and the direction of wear appreciably altered, relative to computed wear for otherwise similar non-roughened femoral heads.

In addition, the recognition of high levels of wear in acetabular components with rim damage retrieved at revision surgery, and a review of the long-term follow-up of dislocation[8] which demonstrated that 26% of dis-

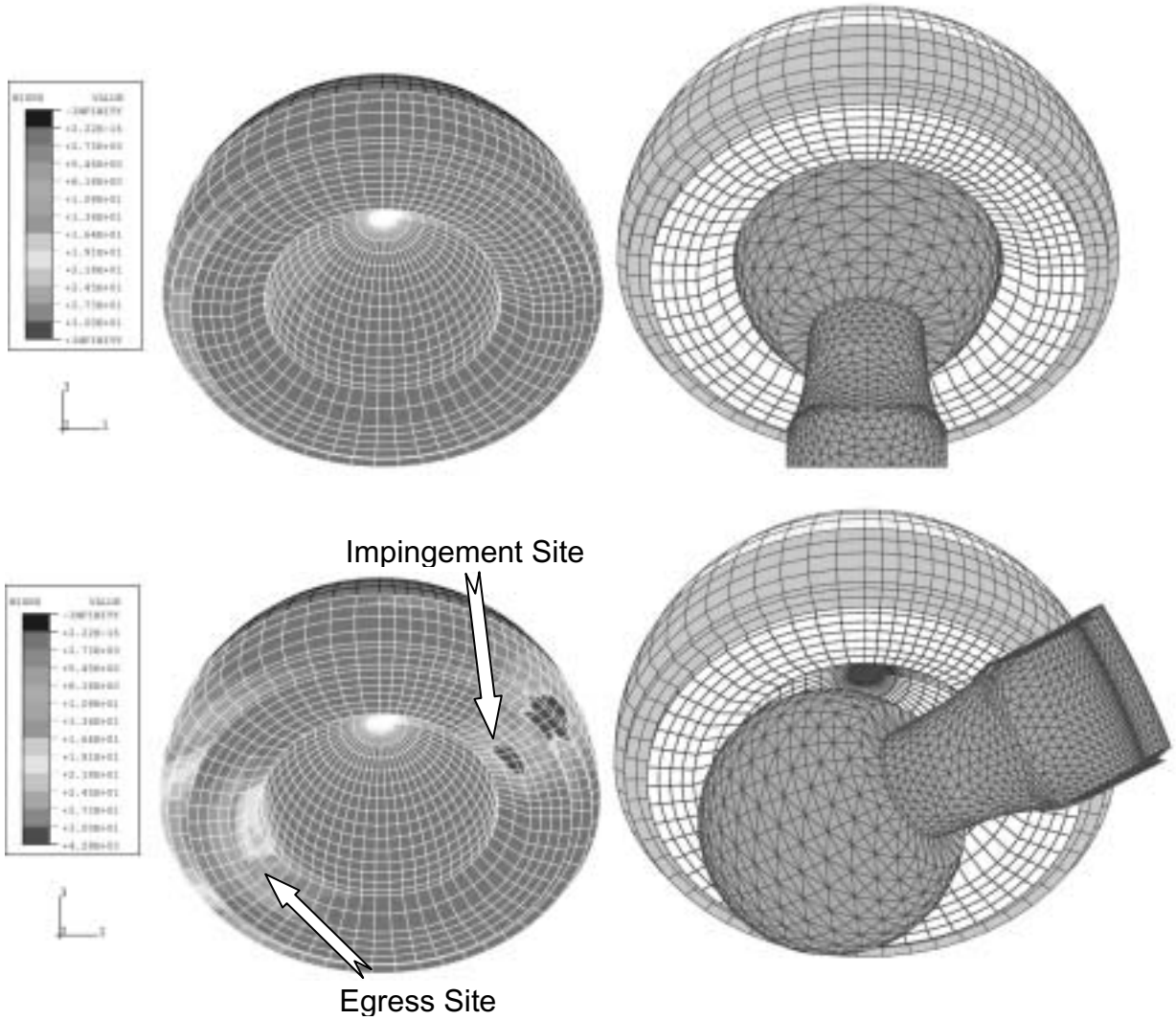

Figure 7. Finite element analysis of the kinetics of total hip impingement, subluxation and dislocation. The model was driven by triaxial motion sequences recorded from subjects undergoing dislocation-prone activities (e.g., leg crossing, rising from toilet seat). The joint loadings were inferred from a 47-muscle inverse dynamics model of the hip.[6] The model articulates normally until neck impingement on the cup. Computed local contact stresses at the impingement and (later) head egress sites are greatly elevated above those for normal articulation, and substantially exceed the yield stress of UHMWPE (lower left panel), even on the outer cup edge, which corresponds with the outer liner wear damage demonstrated in our dislocation studies[8] and in retrieval studies[24].

locations occur late, prompted the authors to use the sliding contact finite element model to study impingement, subluxation and dislocation (Figure 7).

The mechanistic link between rim damage and elevated bearing surface wear is that lever-out subluxation, accompanying impingement, draws debris-laden joint fluid into the opening created. This process was quantified by means of a finite element computational fluid dynamics (CFD) model. The results show that both volumetric fluid ingress and fluid velocities increase with head size (Figure 8), a finding consistent with the clinical observation[23] that larger head size components are preferentially vulnerable to accelerated wear in the presence of a third body challenge. Peak influx velocities for a 28-mm head are 1.29 times those for a 22-mm head, a ratio roughly in proportion to the respective head ratios (1.27), and expected intuitively: For a given distance of head center subluxation, the volumetric rate of fluid influx is proportional to the second power of head radius, whereas the available cross-sectional area available for influx peripherally is proportional to the first power of head radius.

Digital edge detection image analysis wear measurement techniques

Historically, the accuracy and precision of radio-

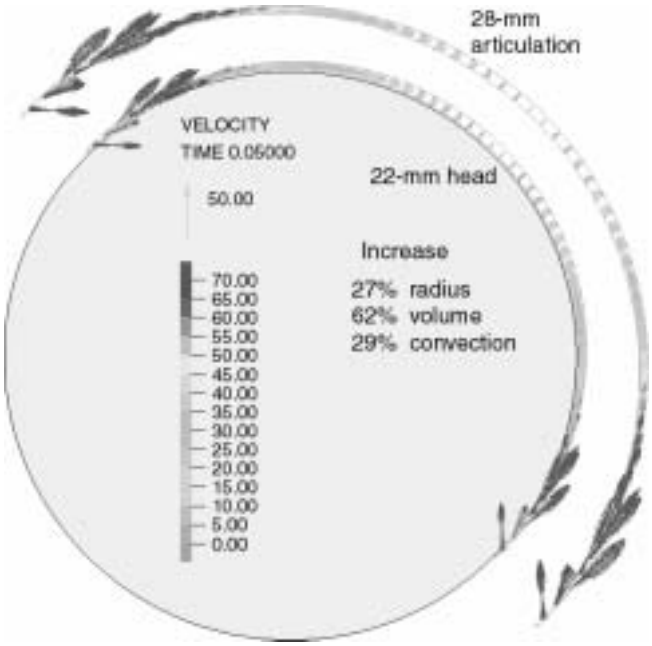

Figure 8. Computational fluid dynamics (CFD) models demonstrate the nonlinear increases in fluid ingress velocities, fluid ingress volume, and concomitant potential third-body debris convection around the subluxating components of increasing head-size total hip constructs.

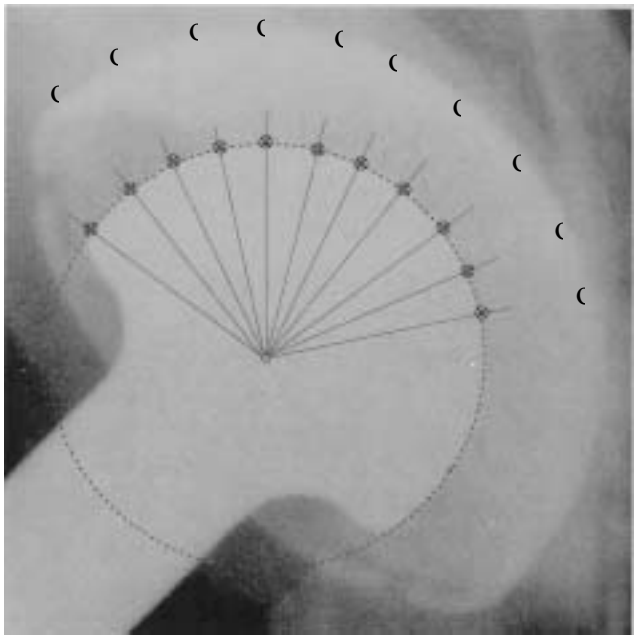

Figure 9. Application of digital edge detection to measure THA wear radiographically. Search rays are computationally generated at 0.5° increments (here, for clarity, rays are displayed only at 10° increments). The pixel grayscale gradient is calculated at each point along each ray. The points of maximal gradient (denoted by the "*f*" symbols for the femoral head and the "o" symbols for the cup backing) identify the respective component margins. Ellipses are least-squares best fit to these two sets of points, to determine the apparent penetration of the femoral head into the acetabular component. Doing this at follow-up, and subtracting the corresponding measurement postoperatively, allowed assessment of interval wear between those two time points.[51,52,53]

graphic measurements of penetration of the femoral head into the acetabular component had been compromised by reliance upon subjective–manual assessment of head penetration.[14,15,18,28,37,45,55,56] To reduce the degree of subjectivity entering into head penetration measurements, digital edge detection image analysis was introduced to more accurately determine the amount of head penetration.[51, 52] The edge detection technique allowed the first-ever automatic, fully objective penetration measurements, using ellipses best-fit to hundreds of component surface points that were identified computationally as sites of maximal local gray-scale gradient (Figures 9 and 10). The new penetration measurement technique was validated by measuring wear artificially produced by spherical-front milling of polyethylene liners in bench top series. Under such conditions, digital edge detection proved 6.4 times more accurate, and 7.1 times more reproducible, than manual measurements made with conventional circular templates.[53]

RESULTS

Wear measurements using a circular template technique were performed on consecutive series cohorts of patients with minimum five-year follow-up radiographs (210 hips). Significantly less wear was demonstrated for 22-millimeter head versus 28-millimeter head components[12] (Table 1). There was a strikingly wide range in the wear rates within the individual cohorts (0 to 0.8 mm per year). In addition, the wear rate distributions were strongly non-Gaussian, skewed by the small number of outlier patients with very high wear rates.[41] Digital edge detection techniques demonstrated lower wear rates with cementless acetabular components with 22-millimeter head sizes than with 28-millimeter head sizes (Figure 11).[43]

The sliding-distance-coupled finite element model was able to reproduce the wear patterns occurring during gait versus in biaxial rocking wear simulators (Figure 4). The model also accurately demonstrated the volumetric wear increase with increases in head size (Figure 12).[32, 34]

To evaluate the ability to determine long-term polyethylene wear from early femoral head penetration (at two years), edge detection techniques were utilized on 197 consecutive total hip replacements with 1,237 archived radiographs taken over a 10 year follow-up period (Figures 13 and 14). The digital edge detection measurements were analyzed using a novel random-coefficients statistical formulation, developed specifically

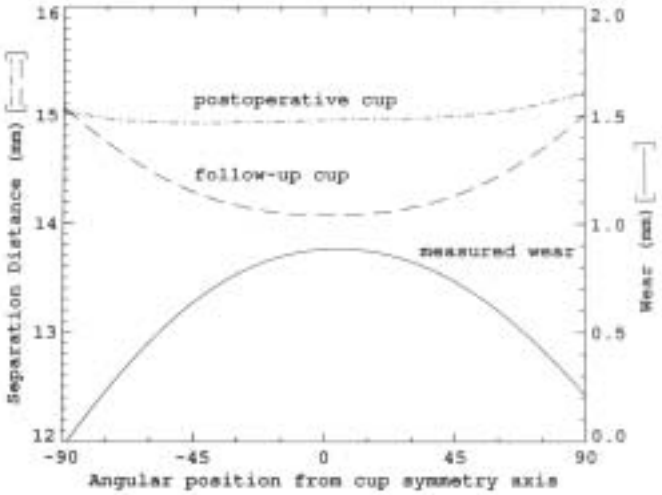

Figure 10. Distribution of the separation distances between ellipses on consecutive radiographs (left axis). Point-by-point subtraction of the distribution at the latest follow-up evaluation from the postoperative distribution represents polyethylene wear (right axis). This also allowed precise determination of the direction of maximum linear wear (i.e., peak of the measured wear curve).

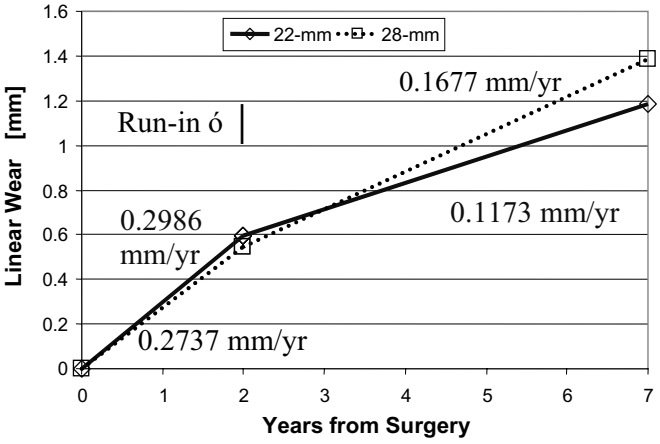

Figure 11. Group-average behavior for "bedding-in" (run-in) and long-term penetration into the polyethylene cups, by 22-mm and 28-mm femoral heads.

to address the distribution of wear rates observed across this population across time. A formal regression equation was reported, by means of which the ten-year wear depth could be quantitatively estimated from a given patient radiograph at the two-year follow-up visit. Series-wide, the correlation between predicted versus observed late wear depths was r = 0.683. Since many very early (less than 2 year) radiographs were available, the authors also used this database to make the first quantitative observations of the initial clinical "bedding-in" process, behavior which could be described mathematically in terms of a best-fit decaying exponential function. Finally, differences in wear direction were detected between hips, and also between subsequent time intervals for the same hip.[40]

Evidence for accelerated polyethylene wear due to third body debris was studied in 709 consecutive primary total hip arthroplasties performed over a five-year period overlapping a thirty-month interval of braided cable usage (Figure 15), all of which patients were followed for a minimum of ten years. The sequential switch from wire to braided cable coincidentally occurred during a period of transition from use of a Charnley femoral component with an all-polyethylene acetabular component, to an Iowa femoral component with an all-polyethylene acetabular component, and then to an Iowa femoral component with a metal-backed acetabular component. Thus, accelerated wear due to elevated third-body debris burden could be confirmed for three distinct implant constructs. Across the entire study population, (fretting prone) braided cable reattachment hips had significantly more acetabular wear ($p<.05$), osteolysis ($p<.0001$), and radiographically apparent loosening ($p<.001$) than did the wire reattachment group. The effect of cable debris on wear was less pronounced with 22-mm head articulations,[23] a finding consistent with the CFD finite element model.

Using the sliding distance-coupled finite element model to simulate femoral head roughening regions of various sizes, severities, and locations resulted in appreciable changes in the computed acetabular wear patterns, including shifts in vectorial wear direction, distortions of wear front sphericity, and changes in volumetric wear patterns (Figure 6). These findings reflect the variability in wear direction noted with edge detection radiographic measurements, and seen in retrieved acetabular components.[5,19,21,22,24,35,46,47,58,59] The fluid mechanics model demonstrated the fluid pressures associated with particle convection into the articulating surface of a larger head construct, a plausible explanation for the accelerated wear observed with 28-mm versus 22-mm head constructs in the cases with cable debris.

The computed local contact stresses (39 MPa) at the impingement and head egress sites in the impingement/dislocation model substantially exceeded the yield stresses of ultrahigh molecular weight polyethylene (Figure 7). Larger femoral heads with a constant head/neck ratio demonstrated a decrease in these impingement stresses, and in the corresponding head egress stresses. The dislocation FE model also demonstrated the gain in stability achievable with increase in head size: increases of approximately 0.59 N-m of peak resisting moment per millimeter of head diameter increase, up to 44 mm.

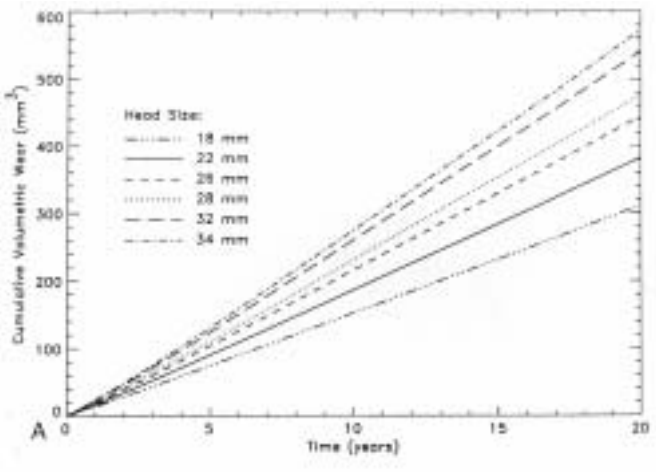

Figure 12. Effect of the head size on time-dependent accrual of volumetric wear, up to as many as 20 years. Volumetric wear increased in proportion to increases in head size.

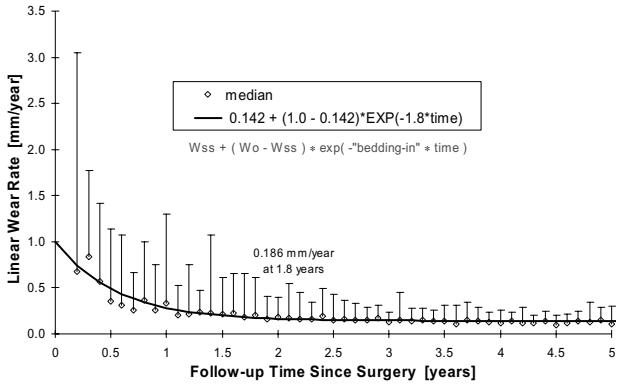

Figure 14. The initial clinical "bedding-in" process could be described in terms of a decaying exponential (best-fit), to quantify early-term femoral head behavior. The time point to achieve steady state wear (Wss), at which 95% of the initial transient concluded, was 1.8 years. (This figure has subsequently been rounded to 2 years to simplify description of the "bedding-in" period.[39])

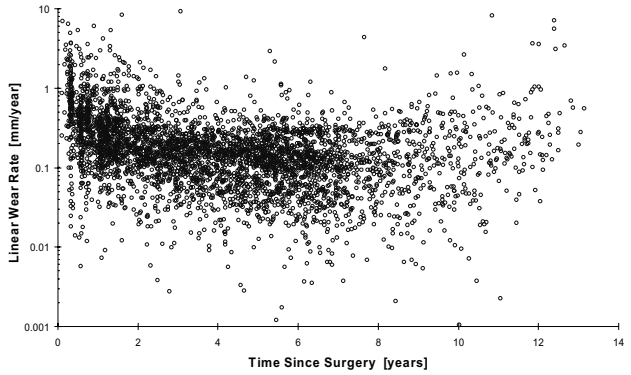

Figure 13. Temporal evaluation of radiographically apparent interval wear rates. Data for a single patient cohort are illustrated as a scattergram of individual interval linear-wear rate measurements (197 hips, 1237 archived radiographs taken over a 14-year follow-up period).[40]

DISCUSSION

This paper summarizes a ten-year period of clinical and laboratory investigation of wear associated with the long-term performance of the total hip arthroplasty construct. Using complementary clinical and laboratory studies (enabled by the unique 30 years clinical and radiographic database, and by novel laboratory computational and experimental techniques), the following questions were addressed:

1. Can long-term wear performance be reliably predicted directly from articulation kinetics (contact stress and sliding distance)?
2. Can long-term wear be reliably predicted on the basis of early wear behavior?
3. Do third body particulates cause accelerated polyethylene wear and early total hip arthroplasty construct failure?
4. What mechanisms are responsible for acetabular component polyethylene rim damage and wear?

This body of work included the first, or among the first, reports that, in the clinical situation, the finding that wear rates with conventional polyethylene are lowest with 22 millimeter femoral heads.[11,42,48] This was later corroborated with autopsy and revision retrieval studies.[24] The sliding-distance-coupled finite element model, initially developed in 1995,[31] documented for the first time that favorable articulation kinematics were the key reason for the lower wear rates observed with 22-millimeter heads, rather than head-size-dependent wear being attributable to Charnley's concept of low friction.[15] The digital edge detection techniques, introduced for the first time in 1994[51,52] opened the way to more accurately determine wear, and demonstrated that accurately measured early wear rates predicted long term wear. This body of work also included among the first, if not the first, quantitation of the bedding-in period associated with wear, and highlighted the need for advanced statistical means for analyzing wear, due to the non-Gaussian distribution of wear rates within cohorts.[38,39,40]

The unique opportunity to study an unfortunate group of hip replacement patients who encountered elevated third body debris due to fretting of trochanteric cable, enabled documentation, with minimum 10-year follow-up, of the accelerated rates of polyethylene wear, osteolysis and component loosening attributable to third body debris migration to the bearing surface.[23,27] Subsequent use of the finite element sliding-distance-coupled formulation to study femoral head surface roughness changes, consistent with third-body-gener-

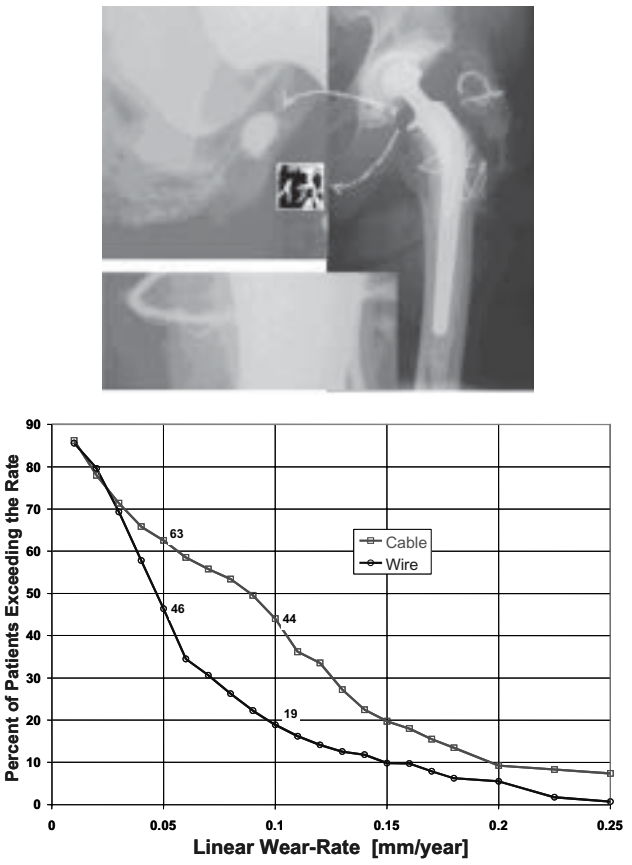

Figure 15. Effects of elevated third body debris burden on in vivo wear. Two patient cohorts were compared, both with the same implant: an Iowa femoral component articulating with a cemented 28 mm metal-backed Tibac (Zimmer) cup. One group ("Cable," 197 consecutive patients) had trochanteric fixation with 1.5 mm, 7-strand Co-Cr-W-Ni cable, subsequently found to be fretting prone (top) and its usage discontinued. The other group ("Wire," 157 consecutive patients) had trochanteric fixation with single-strand stainless steel wire. Series wide (bottom) there is a much larger fraction of problem "Cable" patients (10%) with linear wear rates exceeding the clinically problematic rate of 0.2 mm/year.

ated scratches, provided a plausible explanation for the extreme variability of wear vector direction demonstrated in autopsy acetabular component retrievals, and in serial digital edge detection wear measurement studies.[40,59] The computational fluid mechanics model demonstrated that pronounced fluid convection differences between smaller and larger femoral head articulations are a plausible explanation for the less deleterious effects on bearing surface wear for cable debris cases with 22-mm head articulations than with 28-mm head articulations.

Finally, the application of the finite element formulation to study component impingement and dislocation, the first of its kind, has helped explain the association between impingement-related acetabular component rim wear and dislocation following total hip arthroplasty.[49,50]

In the future, this same general approach of using complementary clinical and laboratory studies should prove useful to characterize and evaluate the wear mechanisms encountered with the new highly-crosslinked polyethylenes and hard bearing surfaces, and issues such as the benefits and compromises associated with the use of larger head sizes (i.e., 36 to 44 millimeters) in conjunction with these new bearing surfaces. In addition, continued work to elucidate the mechanisms by which third body debris enters the bearing surface of total hip replacement constructs should aid the evolution of component designs and surgical techniques to reduce this major additional risk to construct longevity.

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HOW DO TISSUES RESPOND AND ADAPT TO STRESSES AROUND A PROSTHESIS? A PRIMER ON FINITE ELEMENT STRESS ANALYSIS FOR ORTHOPAEDIC SURGEONS

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ABSTRACT

Joint implant design clearly affects long-term outcome. While many implant designs have been empirically-based, finite element analysis has the potential to identify beneficial and deleterious features prior to clinical trials. Finite element analysis is a powerful analytic tool allowing computation of the stress and strain distribution throughout an implant construct. Whether it is useful depends upon many assumptions and details of the model. Since ultimate failure is related to biological factors in addition to mechanical, and since the mechanical causes of failure are related to load history, rather than a few loading conditions, chief among them is whether the stresses or strains under limited loading conditions relate to outcome. Newer approaches can minimize this and the many other model limitations. If the surgeon is to critically and properly interpret the results in scientific articles and sales literature, he or she must have a fundamental understanding of finite element analysis. We outline here the major capabilities of finite element analysis, as well as the assumptions and limitations.

INTRODUCTION

The relationship between implant design and clinical outcome is unquestioned owing to excellent long-term results with some designs and poor results with others. Quite obviously, the outcome of total joint arthroplasty depends upon many patient-related and biological factors independent of the "mechanical environment" in the immediate vicinity of the implant. Equally obvious, not all mechanical phenomenon (e.g., wear) and their biological consequences (e.g., osteolysis) relate directly to that mechanical environment. However, with good evidence, most investigators believe outcome differences (and in particular, aseptic loosening) relate to long-term adaptation of tissues to the mechanical environment surrounding an implant.

What can we know about the mechanical environment, and what do we need to know? First, we can measure or estimate the time-varying load magnitudes and directions on an implant using a variety of approaches. Since experimental measurements and theoretical analyses of joint loads yield similar results, we have reason to believe current estimates are reasonably accurate.[4] Second, we can measure initial bone-implant motions (i.e., stability, mobility) using either *in vitro* or *in vivo* (roentgenstereophotogrammetry*) methods, and we can estimate late implant stability using the latter approach.[14,35,42] Third, we can readily estimate how the loads are distributed at the organ (whole bone), and small macroscopic levels, that is, estimate stresses and strains. The question is whether this is what we need to know: mechanistically it is not the load, motion, and stress or strain magnitudes at a given time or under given load conditions which relate to local matrix microdamage or cell and tissue remodeling causing loosening. Rather, local (i.e., microscopic, if not ultrastructural) *deformation histories* ultimately cause loosening. This latter point raises a question of what structural analysis can provide.

Finite element analysis (FEA), is a powerful computational tool for estimating stress and strain magnitudes and studying the mechanical interactions between bones and implants. While implant design certainly affects

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* Roentgenstereophotogrammetry, or RSA, is a technique to determine the relative positions of two rigid bodies using a minimum of three fixed locations in each body. In the case of implants, three or more tiny implanted tantalum beads in bone serve as those locations, while three fixed locations on the implant may be identified. Using simultaneously obtained biplanar radiographs and relatively simple trigonometric models, relative positions between the implant and bone can be obtained in each of two loading conditions, thus providing the relative motion between the two conditions.

stresses in bone, we do not yet know how bone stresses or strains relate to tissue adaptation. This chapter will explore the capabilities and limitations of current FEA in predicting tissue adaptation, and will suggest some new directions in an effort to improve the clinical applicability of this tool. Some issues are philosophical in nature, and while we do not focus on those issues, neither can we avoid them and adequately communicate the capabilities and limitations of FEA.

FINITE ELEMENT ANALYSIS (FEA)

Finite element analysis is merely a way to compute stress and strain fields throughout a structure: that is, how loads and deformations are distributed. Long-known relatively simple (partial differential) equations of static equilibrium readily accomplish this task for simple, solid structures (e.g., beams and rods), where an exact solution can be found which satisfies the equilibrium conditions. However, the equations for geometrically and/or materially complex or non-solid structures are intractable. FEA, introduced in the late 1940's, provided a means to estimate stress and strain fields within more complex structures. Simply stated, a complex structure is modeled as a grid or mesh of many small simple structures ("finite elements"), each with its own typical material and geometric properties, and each connected to its neighbors at discrete nodal locations to insure the coherent displacements and stresses which would occur in the actual structure when loaded. The solution in these cases is approximate, and satisfied only in a weak sense,[24] but becomes more accurate with increasing mesh refinement. Subsequently, stress and strain throughout a more complex structure can be computed since the states vary from element to element.

Despite the conceptual simplicity, the practical solution to adequately complex FEAs awaited the development of powerful digital computers. Two of the early FEAs of bone were reported only in 1972.[6,47] Modest in number of elements and other properties, the varying thickness of the two-dimensional section was modeled by varying the stiffness of the elements. (Ironically, the stress trajectories predicted with beam theory analysis more closely approximated trabecular architecture than with early FEA!) Within the next decade, many investigators applied the method to bones and prostheses,[26] although the accuracy and validity* of some of these early models was open to question in part owing to computing limitations and lack of approaches to accurately model interfaces (of differing materials or contacting surfaces) and properties of complex materials.

With modern computing power allowing high mesh resolution (many elements) and new approaches more accurately modeling interfaces and materials, FEA now affords stress analyses with greater biological and clinical implications. Because the approach offers substantial power to explore many aspects of implant design prior to actual construction, the surgeon interested in developing an implant or in understanding the structural implications of an implant should have at least a basic knowledge of the approach, including its capabilities and limitations.

Any FEA requires: 1.) a sufficiently-refined mesh reflecting the external shape and the geometry of any and all relevant structures; 2.) boundary conditions reflecting any and all relevant external constraints or loads surrounding the structure; 3.) material properties appropriately describing those within each element; 4.) proper modeling of interfaces of differing materials or non-connected structures.

These requirements are necessary, but not sufficient for confirming models. At a minimum, model confirmation requires: 1.) a convergence study demonstrating model results do not change if mesh resolution is increased. The greater number of elements, the greater the fidelity to shape and material characteristics of the modeled structure since the real structure may be viewed as one with an infinite number of elements. While the computed solution will change as one begins with few elements and increases mesh resolution, at a certain resolution the solution does not substantially change (i.e., it converges). Normally it is not necessary to further increase mesh resolution once one shows a convergent solution. 2.) a reasonable comparison of model results with independent observations (e.g., laboratory strain analysis, analytic solutions). Reports not demonstrating convergence and not containing compelling confirmation (e.g., laboratory strain analysis, ana-

* Oreskes, et al., appropriately distinguish "verification" from "validation."[43] "Verify" means to establish the truth of a proposition, or in our case a finite element model. "Validate" on the other hand, means to establish the soundness or legitimacy of a proposition; a valid proposition or model contains no detectable flaws and is internally consistent. Validity of a model is usually necessary, but not sufficient to insure the truth (in our case, the state of stress or strain in an actual biological structure). "Confirmation" implies agreement of model results with observational (e.g., strain gauge) data. In most cases, researchers mean "confirmation" when they use the term "validation." This distinction may appear semantic, but the terms have differing roots and meanings. Further, the distinction would be academic, except Oreskes, et al. effectively argue, "Verification and validation of numerical models of natural systems is impossible." This does not mean numerical models are not useful; quite the contrary, they are extremely useful, and without them, contemporary air and space flight would likely be impossible! (Realize, though, early planes and rockets were developed without such models!) Rather, the orthopaedic surgeon should recognize finite element models have inherent limitations, and should pay particular attention to model confirmation (the term to be used in this chapter).

lytic solution, natural observations, animal or experimental observations) should be viewed with great caution not only for absolute values, but even relative comparisons.

With current technology and appropriate confirmation, such analyses have the capability to provide reasonable estimates of stress and strain in bone* (as an organ) and implant materials. These models provide a powerful capability not readily achievable (and perhaps impossible to achieve) in any observational or experimental approach: the potential to vary only one parameter or feature and no other. In a clinical study, for example, one can use implants of two designs, but despite the best controls (e.g., matched patient groups, single surgeon, same approach, same post-operative regimen) the biological differences and other variables (e.g., biological activity of the tissues, patient activity levels) could obscure the effect of design variables despite a large study (see Frost[16] for a discussion of biological and mechanical interactions). (A very large study with radically different designs and very long-term follow-up might be able to ascertain differences with reasonable power, but such studies are usually impractical for a variety of reasons.). The same argument applies to the typically better controlled bench study (e.g., cadaveric study). In a FEA, however, all other variables are absolutely controlled, providing an unexcelled opportunity to examine relative differences between implants or a single implant with design differences. Such investigations vary a single parameter (e.g., stem thickness or stiffness) keeping others constant and are accordingly termed, "parametric" studies. These studies, in addition to precision of strain computation at the macroscopic level, is perhaps the most powerful capability of most current FEA implant studies.

Thus, FEA does afford the opportunity to identify high and low stress or strain regions in bone. As will be obvious from subsequent discussion, contemporary FEA computes stresses and strains over some averaged region, and not the extremes. Nonetheless, given sufficient mesh resolution, averages of small regions would give some indication if the stresses and strains exceeded the static or fatigue limits of bone strength, and how design changes may relieve or exacerbate these limits.

GENERAL ASSUMPTIONS AND LIMITATIONS OF FEA

Effective Continuum Assumption

FEA arises out of continuum mechanics, the science on which the very concepts of stress and strain are in fact based. "Continuum" implies a material is continuous or solid and its properties (e.g., elastic modulus, Poisson's ratio, yield criteria, volumetric density) are locally homogeneous (i.e., do not vary). Quite obviously, at an atomic level, no materials are "solid," although from a practical point of view they may be so considered at a macroscopic level. In any continuum model, a specific length scale of interest is usually implied by the dimensions of the model. Material properties at the length scale of a continuum model are typically just averages of the material properties at smaller length scales. Consequently, information pertaining to stresses and strains at small length scales is usually not provided by the continuum model. For example, in the design of an implant, for most purposes (but importantly, not all) cortical bone could be quite reasonably treated as a homogenous continuum, since the heterogeneities associated with osteons, vascular porosity, lacunae, and canaliculi exist on much small length scales. However, in the design of an implant in trabecular bone, it is more questionable to model the bone as a homogeneous continuum, since the length scale of the heterogeneities within the bone (length of individual trabecula and the associate pores) approaches that of the implant dimensions. Architectural features such as osteons effectively create "stress risers" or features which substantially influence local tissue stresses or strains. One should then ask: At what scale is the assumption of a homogeneous material valid? In an attempt "to establish the length scales over which the continuum assumptions is valid in cancellous bone," Harrigan et al., concluded, "Within three to five trabeculae of an (implant) interface a continuum model is suspect."[20] This means the average strain calculations do not apply at the trabecular (let alone cell) level, owing to variations from the continuum-assumed averages. We do not currently know the range of stresses or strains created by these stress risers, although the estimates range from 2-3 times (46),[51] to 10 times,[41] or even 300[23] times the continuum or average level. The biological (i.e., mechanistic), if not predictive importance of this point will become apparent later.

For the reasons stated above, the continuum assumption is most likely valid when modeling prosthesis implantation in the cortical bone of a cadaveric femur or when modeling the initial state in a living femur immediately after implantation. In fact, an FEA is often confirmed ("validated") using a strain-gauged** cadaveric

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* "Stress" as a concept does not likely apply at the cell or tissue level for two reasons: 1.) cells are complex structures with complex load distribution; 2.) deformation, rather than "stress" likely stimulates cells. When appropriate we will use the term "strain" since it is more closely related to deformation and likely cell response.

** Strain gauges record strains over length scales for which the continuum assumption applies.

experiment. Thus, despite the continuum assumption, FEA still has the potential to be powerful in predicting, although not mechanistically explaining, implant performance in a population. In such a situation, biological mechanism need not be incorporated into a model, although no model should contradict known biological mechanisms or clinical observation. However, most published FEA studies do not adequately address strain at the trabecular level, either because that architecture in the vicinity of the implant is not included, or when it is modeled, the variations in material properties at that level are simply not known.

Design Criteria

Designing "better" implants requires some "design criterion." That criterion usually minimizes or maximizes - i.e., "optimizes"—stress or strain, either globally or locally. That is, one identifies a design which reduces or increases some feature which will have beneficial effects. Given a design criterion, one parametrically varies a given design feature to determine which design best achieves that criterion. A typical criterion might be to minimize peak strain in bone cement. A parametric study is conducted, examining a single design change (say, stem thickness), then searching all cement elements for strain with each modeled thickness. One chooses that thickness which best achieves the criterion.

Design Features Not Directly Related to Stress and Strain

Many important or critical aspects of design, such as choice of implant material and surface finish are not directly addressed by FEA. While the differing material properties of an implant material are considered, FEA does not account for material differences affecting the system in biological ways not related to strain. For example, some implant materials (e.g., titanium) are believed to promote or tolerate bone ongrowth better than others (e.g., stainless steel). Surface finish at the microscopic level relates to cell responsiveness. An FEA does not consider such design features, when in fact they could be critical.

Design Features Related to Stress and Strain

Minimizing strain in an implant material (ceramic, metal, high density polyethylene, polymethylmethacrylate) is relatively straightforward, because the continuum assumption is typically reasonable, and because we have a good notion of the relationship between stress or strain and material failure. Thus, if the questions relate to failure of an implant material, including wear, FEA provides a powerful approach. On the other hand, if the questions relate to tissue adaptation, the FEA approach is not straightforward since we do not know the relationship between stress or strain and bone adaptation. In the case of an implant, one might empirically reduce bone strain in high strain regions, and perhaps increase strain in low strain regions if one presumed the low level would lead to reduced bone mass (density).

BONE ADAPTATION TO THE DEFORMATION HISTORY

The relationship between the mechanical environment and connective tissue adaptation has been known or suspected for centuries. Galileo recognized the generally similar shapes of animals of widely differing sizes, yet each adapted in particular size and shape to the size and anatomy of the animal. The observation of regularity of trabecular architecture (from individual to individual in the same bone) during the 19th Century suggested description by mathematics (since mathematics can theoretically describe all regularities). As early as 1851, Wyman suggested, "the cancelli in all those bones that assist in sustaining the weight of the body, or in locomotion, are arranged in definite directions, the directions being those of the reception and transmission of force."[56] The notion that trabecular architecture could be described by the principle tensile and compressive stresses which would occur in a solid, homogeneous structure of the same shape in the 1860's[39] led to the postulate that these stresses governed bone remodeling.[46,55] Following Wolff's classic monograph, "Das Gesetz der Transformation der Knochen," published in 1892, the general relationship between the mechanical environment and tissue adaptation has been generally termed, "Wolff's Law."[55]

Wolff specifically noted:

> Es ist demnach unter dem Gesetze der Transformation der Knochen dasjenige Gesetz zu verstehen, nach welchem im Gefolge primärer Abänderungen der Form Inanspruchnahme, oder auch bloß der Inanspruchnahme der Knochen, bestimmte, nach mathematischen Regeln eintetrende Umwandlungen der innerer Architectur und ebenso bestimmte, denselben mathematischen Regeln folgenden secondäre Umwandlungen der äusseren Form der betreffenden Knochen sich vollziehen.
>
> (It is therefore under the laws of transformation [remodeling] of bone, the one law to understand, after which in the wake of the primary changes of utilized form, or certainly just the use, after mathematical rules set in transformation of the inner architecture and just as certainly, following the same mathematical rules the secondary transformation which is carried out on the outer form of the bone in question.)

Or, more compactly:

> The law of bone remodeling is that mathematical law according to which observed alterations in the internal architecture and external form of bone occur as a consequence of the change in shape and/or stressing of bone.

Quite obviously, Wolff and subsequent investigators presumed something about stress and/or strain "governed*" bone adaptation. Two problems immediately arise: First is Wolff's implicit continuum assumption. Stress and strain concepts arise from continuum mechanics, in which solid, continuous materials are assumed at the level of interest. Bone and interfacial tissues are not solid, continuous materials, however. In fact, discontinuities observed at the trabecular macroscopic (not to mention those at the microscopic and ultrastructural) level undoubtedly result in substantial variations of "average" or continuum level stress or strain predictions**. Second is Wolff's implicit concept of "governing laws." While Wolff's understanding and/or philosophy of the meaning of mathematics in Nature are not known, he surely implied something inherent in mathematical "laws" "governed" bone remodeling in particular, and Nature in general. Historically, this notion reflects a school of thought believing mathematics inherent in Nature, and mathematical quantities "governed" or "controlled" natural processes. An opposing school of thought believes mathematics merely a man-made artifice, which despite its immense power, could be replaced by some alternative construct (e.g., Pythagorian versus Reimannian geometry) and in the best of circumstances merely described or predicted natural process. Newton reflected this latter school when in formulating the "laws" of gravity, he commented, "Hypotheses non fingo": "I propose no explanation." Thus, he recognized his "law" merely described Nature, not explained it.

This latter point is critical in interpreting modern formulations of tissue adaptation: some contemporary investigators apparently ignore the distinction between mathematics as a way to describe or predict, and mathematics as a way to explain. In the former case, empirically derived mathematical formulations may accurately predict tissue adaptation without consideration of mechanism, while in the latter, mechanism would be essential. Since the mechanisms of tissue adaptation are poorly understood, known "steps" in cascades of events are complex, and many steps undoubtedly remain to be identified, it makes little sense to artificially introduce mechanism into mathematics which at best describes and predicts. On the other hand, it makes equally little sense to introduce mathematical formulations contradictory to known mechanisms. Further, in contrast to the mechanics where most "mathematical laws" accurately predict at macroscopic levels, in biology, such laws only approximate outcome for a population (of individuals, tissues, cells). Thus, physical scientists and engineers need an understanding of biological constraints, while biologists and physicians need an understanding of computational constraints. Given this understanding, FEA offers the potential to describe or predict implant performance for a population.

CAN FEA PREDICT BONE ADAPTATION?

It is fair to state no current approach to numerically predicting bone adaptation to implants has been correlated with clinical outcome. Thus far, FEA cannot tell us whether a given implant will loosen owing to mechanically-driven bone adaptation. This is one of its greatest limitations. Why is this the case. First, continuum level average stresses or strains do not likely directly related to implant failure. Second, tissues "temporally process" deformation histories and this process is key to adaptation; the typical FEA uses static loads and does not account for either deformation histories or temporal processing. Third, the individual cells responsible for adaptation do not respond to stress or strain per se, although they might respond to some related quantity.

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* "Govern" in this paper will be used in a broad sense to mean describe or predict, and will not be used in any mechanistic sense implying something actually controls tissue adaptation.

** As noted earlier, "stress" and "strain" arise from continuum mechanics of materials with attendant assumptions. At the organ level, bones, tendons, or ligaments may behave as materials of a given geometry. However, at the cell level, they are not materials, but rather complex structures. A definition of traditional engineering strain requires establishing of reference frames and two distinct points between which strain is ascertained. It would be difficult to know which two points on a cell were most biologically relevant, even if one could ascertain changes in distance between them under load. Thus, while cells surely deform under load, it seems specious to speak of "cell strain." "Deformation" seems a more appropriate term for cells, since deformation of the cell membrane, cytoskeleton, or other structures (e.g., stretch-activated ion channels) are mechanistically believed to be involved in cell responsiveness.

Continuum Level Averages Do Not Likely Directly Relate to Implant Failure

Why are the continuum averages computed by FEA perhaps less important than the ranges of stresses and strains* on the microscale? The reasons are several fold: 1.) Very high levels of local strains may lead to fatigue fracture of dead or even living bone (microfractures) within weeks or months following prosthetic implantation. 2.) Very high levels of local strains may lead to bone resorption of living bone. 3.) Very low levels of local strains may lead to bone resorption of living bone (i.e., generalized disuse atrophy and localized "stress shielding"). In each of these cases, support lost at one small point, leads to load transfer elsewhere (presuming the loading is the same), perhaps overloading and fracturing or resorbing adjacent areas.

Several observations and one argument support these reasons: 1.) The presence of micromotion (albeit sometimes only in the range of 10-50 micrometers) between all implants and bone, both initially and subsequently. The presence of motion itself likely engenders levels of stresses and strains not computed by the typical FEA (unless it were a contact model). 2.) The roentgenstereophotogrammetry (RSA) documented initial settling of virtually all implants (again, in a small range for clinically successful large joint implants). The presence of near universal small but detectable initial settling suggests overloading of some initially supportive bone with fracture. However, given the long-term endurance of most implants, we must presume implants reach some point of stability where interfacial stresses at both global and local levels are tolerated by bone. The RSA evidence of continued settling eventually leading to clinically significant implant loosening suggests a point of stability is never reached in some implants. That is, local loss of support in one region causes overloading in previously appropriately loaded regions, then the new region fails, and so on. 3.) The higher levels of stresses and strains (i.e., above the continuum level computations) likely exceeds the breaking strain of bone. Various authors suggest the static breaking strain in the range of 14,000-35,000 microstrain[2,12] while the ultimate fatigue strain in laboratory cortical specimens is in the range of 4000-8000 microstrain**.[22] Thus, presuming the range of continuum-predicted strains in trabecular bone are 10 times the average, it is likely some trabeculae fail around implants some time shortly after implantation. This argument is consistent with nearly universal RSA-observed settling.

Temporal Processing of Deformation History Is Key to Tissue Adaptation

Realizing the state of strain around implants is not constant, some investigators have utilized iterative FEA, in which one assumes extreme regions of strain will change their properties as a result of the biological responses.[34,53] In such models regions of low strain energy density become less stiff, which increases the energy density level, and regions of high strain energy density become stiffer, which decreases the energy density level†. These approaches assume the tissue is attempting to reach some more ideal range of strain energy density. If new moduli are assigned to these regions, a new FEA solution may be obtained with a differing distribution of strain. The re-assignment of properties is continued until the solution converges (i.e., all elements of bone experience some specified range of strain energy density). These sorts of models can account for the clinically observed remodeling (changes in radiographic density) which occurs around implants.[13,18,36,49,52]

Models accurately predicting bone density necessarily require adaptation rules. Current investigators select some strain (or strain-related) magnitude, presuming tissues and/or cells differentiate the various forms of stress or strain††. In fact, we do not know which will most accurately predict bone response, and in fact we do not even know whether individual cells indeed differentiate say, compressive versus tensile strain. Thus, the predictive model presumes the response of cell population in a large tissue region is associated with a given stress or strain quantity, but one should not confuse individual cell responsiveness with population responsiveness. The magnitudes of some calculations of stress or strain (e.g., longitudinal tensile or compressive stresses) depend upon some more or less arbitrary reference frame, and it seems quite obvious these sorts of parameters may be irrelevant, since cells do not know

* Continuum estimates of peak loading range from 1000-3000 microstrain, and if the local strains are in the range of greater than ten times those levels, they may well be in the range of the breaking strain of bone, which is only 1.4-4.1 times the peak physiological levels.[2] The survival of the implant may therefore depend upon the volume of interfacial bone experiencing mechanically or biologically high levels of strain.

** This range is astonishingly low in strain magnitude, and as suggested by Biewener (2) would result in low safety factors. It is likely, however, living bone has adaptational mechanisms not present in dead bone.

† The ranges of acceptable stress or strain for bone homeostasis are not known, but Frost postulated such a range or "window".[15]

†† "Stress" and "strain" may be mathematically defined in many ways (e.g., principal compressive and tensile strains versus strain energy density).

the orientation of such frames. Other stress/strain parameters (e.g., strain energy density) do not depend upon a reference frame, and therefore seem more appropriate as candidate predictors. In attempting to identify particular aspects of loads (i.e., stress/strain parameters) relating to tissue adaptation, recent investigators assume tissues "seek" (i.e., remodel to achieve) some identifiable and "optimal" state of stress or strain (e.g., "attractor state"), and further implicitly or explicitly postulate the attractor state arises from peak loads,[9,27] "averaged" strains,[15,16] or "values which cause fatigue microdamage".[11,17]

Understanding the relationship between tissue adaptation and loading must involve some "cumulative effects of loading," but not the time-averaged sort suggested by Cowin.[11] Carter[9] and Whalen and Carter[54] formulated a remodeling rule based upon summing a number of discrete peak loads during some given number of occurrences of similar activities, assuming the entire loading history influenced bone maintenance and/or remodeling. One recent study, however, suggests stress/strain magnitude alone does not predict bone adaptation theoretically[7] or experimentally,[40] while another suggests magnitude and cycle number, regardless of how the two are weighted, also do not predict bone adaptation.[1]

Cowin noted, "The precise aspect of the strain history sensed by bone tissue is an open question."[11] The recognition that strain history, rather than merely some strain quantity at a given time, initiates remodeling arises from experimental evidence suggesting tissues account for temporal aspects of the stimulus; for example, bones respond very differently to static loads over time than similar magnitude dynamic loads.[10,32] Further, Gross et al. demonstrated the strain distribution in bone at the instant of peak strains differs from the distribution of strain at other times; therefore if submaximal strains initiate bone remodeling, the new bone distribution will differ substantially from that predicted from the location of peak strains.[19] Perhaps more importantly, some regions of bone habitually experience low strains while others experience high strains (at both continuum and cell levels), yet maintain spatial concordance.[3]

To date, however, these remodeling rules ignore additional features inherent in any mechanical signal: duty cycle (i.e., distribution of events over some time frame termed "dose-fractionation"[30,31] or "partitioning"[45]), the interlinked frequency and strain rate,[37,38] signal duration, and wave form. The biological ramifications of these features are likely interdependent in ways currently unknown. And although their importance is unquestionable, they are virtually ignored by current investigators using FEA to study bone adaptation.

An abundance of *in vivo* and *in vitro* studies suggest cells and tissues in fact "ignore" the majority of mechanical signal content, "selecting" and responding only to certain features.[5] For example, rather than exhibiting a dose response to cycle number, one observes a trigger response in many systems. These sorts of observations led to the hypothesis that tissues "temporally process" mechanical signals, and do so in distinct ways: 1.) They respond in a trigger-like manner after a relatively few events or cycles of loading; 2.) They respond only to some window of strain magnitude; 3.) They exhibit a refractory period after a response; 4.) They have a memory for previous stimuli. These characteristics do not necessarily mirror distinct cellular phenomena but rather reflect typical features of experiments.

FEA, even those which iteratively predict changes in bone density, do not account for temporal processing features of tissue adaptation. Rather, they assume the changes occur primarily, if not exclusively as a result of some stress or strain magnitude. This is not to say they could not incorporate such features. Based on his own work and that of others, Turner recently noted three "fundamental rules" of bone adaptation: "(1) It is driven by dynamic, rather than static loading. (2) Only a short duration of mechanical loading is necessary to initiate an adaptive response. (3) Bone cells accommodate to a customary mechanical loading environment, making them less responsive to routine loading signals."[50] These and other sorts of arguments can be combined with iterative FEA magnitude predictions at a phenomenological level without considering mechanism.

The question is whether FEA needs to incorporate such adaptation rules to successfully predict loosening. As earlier noted, a predictive model need not incorporate "mechanistic rules" to be successful, although a mechanistic model obviously would. However, there are times when incorporation of basic mechanisms into predictive models is useful either for enhancing predictive or for heuristic reasons. Thus, the question is not answered at this time.

Individual Cells Do Not Respond to Stress or Strain

Whether or not FEA can accurately predict bone adaptation, the question arises as to whether individual cells respond to stress or strain. Abundant experimental evidence suggests many connective tissue cells respond to various alterations in the mechanical environment at both physiological and supraphysiological (at the continuum computed) levels of strain. Almost any

sort of mechanical stimulus (hydrostatic pressure, stretching on a substrate, poking) elicits cell responses. While identical cell culture systems have not been systematically explored using differing stimuli, available evidence suggests it may not matter how the stimulus is applied. In turn, this suggests cell deformation of any sort may be the stimulus rather than a specific sort of stress or strain.

A number of experimental observations indicate osteoblast-like cells in culture respond to fluid flow.[21,28,29,44,48] Since bone has pores at various scales and contains water, deformation will cause fluid flow, leading to the hypothesis that fluid flow, rather than or in addition to deformation causes mechanically-driven adaptation. These hypotheses could be explored using poroelastic FEA formulations which simulate fluid flow. (It should be added that under normal loading conditions, poroelastic strain solutions of bone will not differ much from elastic solutions, so the advantage of poroelasticity is to explore hypotheses, not to create more accurate strain predictions.)

Other arguments lead to the same conclusion. Connective tissue cells are not rigid structures and are not rigidly attached to their relatively stiff matrix. Thus, even though we might infer a pure stress state (say compression), one cannot suppose cells loosely connected to a matrix in lacunae at all sorts of orientations to the load experience pure "compression." What they likely experience is deformation, to which they unquestionably respond.

FEA could theoretically model cells in lacunae at differing orientations if one knew the geometry of the structure (cells are indeed complex structures), the material properties of each element in that structure (including all the proteins connecting the cell to the matrix) and the structure and properties of the surrounding matrix. In this case, one could ascertain how an individual cell deforms under some pure load. However, the material and structural property information required for such a model is not available.

For the time being, we must accept that we can construct finite element models which compute only average stresses and strains for a large region of cells, matrices, and discontinuities. Thus, any true (biologically) mechanistic model is impossible (40).[43] That is not to say, however, some predictive model which considered only average stresses or strains might not accurately predict tissue adaptation to an implant.

In fact, a number of FEA studies incorporating some stress/strain magnitude and/or aspect of time do correlate with bone density, one aspect of tissue adaptation.[25,33,49,52,53] Interestingly, different loading conditions produced similar bone density distributions in one such model.[13] Bone density and in particular stress shielding, however, has not been shown to correlate with long term outcome.[8] That is not to say some other sort of FEA might not accurately predict mechanically-caused long-term aseptic loosening.

CONCLUSIONS

We are unaware of anyone who has used FEA to accurately predict clinical outcome. The primary value of FEA in exploring design changes has thus far been heuristic; that is, parametric exploration of design changes in the same model provides substantial insight regarding the mechanical interactions between bones and implants. Quite clearly, we should avoid implant designs associated with regions of very high and very low stresses and strains. But while we have a good idea of the static and fatigue behavior of bone and thus fracture, we typically do not compute the range of trabecular stresses and strains leading to fractures, and we do not know what mechanical environment will stimulate beneficial or detrimental bone adaptation over time.

An incomplete understanding of the biological mechanisms of bone adaptation does not preclude, however, an FEA model which might prove predictive of implant loosening. We suspect, however, the most promising approaches will require incorporation of two features: 1.) hierarchical modeling; 2.) iterative, adaptive modeling. Hierarchical modeling simply means the tissues are coherently modeled at multiple levels from micro- to macroscopic. Thus, the investigator will more reasonably predict the range of local strains at the level of interest: those quantities which lead to trabecular fracture or relate to cell responses and tissue adaptation. Iterative modeling recognizes the initial effect of the implant on the immediate mechanical environment does not remain the same for very long. Within days or weeks of implantation, a portion of the interface will change as a result of injury-repair reactions, and when that interface changes, the stress distribution and small implant position changes. (RSA studies confirm such position changes actually occur, and even predict outcome.) Iterative modeling can account for the changes since the local stresses/strains are known through the hierarchical modeling. Further, the introduction of these approaches does not preclude identification of empirical remodeling rules predicting bone adaptation and loosening. These currently available refinements in the use of FEA should enhance their value in implant design.

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THE BERNESE PERIACETABULAR OSTEOTOMY: REVIEW OF REPORTED OUTCOMES AND THE EARLY EXPERIENCE AT THE UNIVERSITY OF IOWA

Todd O. McKinley, MD

ABSTRACT

The Bernese Periacetabular Osteotomy (PAO) is an operation designed to relieve hip pain and improve function in patients with symptomatic dysplastic hips. The short-term results of the Bernese PAO indicate that there is a learning curve but most patients with dysplasia and mild arthritis have excellent pain relief. Outcomes and complications are the primary focus of discussion. Operative data and complications from the first 36 patients undergoing the Bernese PAO at the University of Iowa have been comparable to those reported in the literature.

INTRODUCTION

Development dysplasia of the hip (DDH) is a heterogeneous disorder that can lead to disabling hip pain and degenerative arthritis. The main pathoanatomic component is osteoarticular deficiency of the acetabulum with deficient lateral and anterior coverage of the femoral head. This acetabular deficiency substantially alters force transmission across the hip, subjecting the acetabulum and femoral head to increased and abnormally directed contact stresses[1]. Chronic abnormal stresses eventually can lead to subluxation and end-stage arthritis of the hip.

Patients with DDH who develop hip pain and subluxation are at nearly uniform risk to progress to end-stage arthritis. Unfortunately, many of these patients are young adults. Total hip arthroplasty is an outstanding option for elderly patients with disabling hip arthritis, but it is a much less desirable option in young patients. Ganz developed the Bernese periacetabular osteotomy (PAO), an operation designed to improve femoral head coverage by reorienting the shallow acetabulum, to relieve hip pain and slow the progression of arthritis in patients with symptomatic DDH who were young[4]. In this review, early and medium-term outcomes on Ganz's original patients and results from several other centers are reviewed. Finally, early operative and radiographic results as well as complications from the first 36 cases performed at the University of Iowa are presented.

REVIEW OF CLINICAL LITERATURE

The operation was originally described by Ganz in 1988 and often bears his name[4]. Ganz et al. reported on the initial 75 patients treated with the Bernese PAO. The authors cited several advantages compared to other innominate osteotomies. They emphasized that the Bernese PAO could achieve greater corrections which was especially advantageous in severely dysplastic hips. Secondly, they emphasized that the structural continuity of the pelvic ring is not disrupted, eliminating necessary postoperative immobilization or traction. Finally, they felt the Bernese osteotomy was technically easier than several of the described spherical osteotomies. However, patient follow-up was too short to report on any outcomes.

The authors emphasized that the operation has a learning curve, with substantial decreases in operative time and blood loss occurring throughout the series. They also noted clinically substantial complications occurred in the first 18 cases including several intraarticular osteotomies, several malpositioned fragments, a transient femoral nerve palsy, several cases of resubluxation of the femoral head, and four cases of heterotopic bone formation. They concluded that there is a significant learning curve but the operation had excellent potential to help young patients with symptomatic acetabular deficiency.

Trousdale et al. reported on 42 patients from Ganz's original series who had a diagnosis of DDH at an average of 4 years (2-8 years) after surgery[9]. Overall, Harris hip scores improved from 63 to 86 following surgery. The authors noted that the amount of preoperative arthrosis correlated with postoperative outcome. Patients with mild to moderate arthrosis (Tonnis Grades 0 - 2) had substantial improvements in their hip scores compared to patients with Grade 3 osteoarthrosis which showed little benefit. Five of the 6 patients who progressed to total hip arthroplasty had Grade 3 osteoarthrosis prior to their PAO. The authors reported

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TABLE 1

Magnitude of Correction

	Lateral Center Edge Angle (Degrees)	Anterior Center Edge Angle (Degrees)	Acetabular Index (Degrees)	Femoral Head Medialization (millimeters)
Siebenrock	28	22	20	6
Matta	28	28	22	6
Crockarell	22	44	N/A	N/A
Trumble	23	25	17	5
McKinley	24	26	19	4

Table 1. Magnitude of correction of the Lateral Center Edge Angle, Anterior Center Edge Angle, Acetabular Index, and Femoral Head Medialization for four previously reported series and the current series at the University of Iowa. Values represent differences between preoperative and postoperative values.

no major neurovascular complications. One patient required resection of heterotopic bone and one required neurolysis of the lateral femoral cutaneous nerve. Overall, the authors demonstrated that the Bernese PAO provided dramatic pain relief and improved function in selected patients with DDH who had minimal to mild pre-existing arthritic changes in their hips. They cautioned that the osteotomy was not a predictable operation for patients with advanced arthritic changes.

Recently, several other centers have reported short-term results on patients treated with the Bernese PAO[2,5,7,10]. Matta et al. reported on 66 hips treated with the Bernese PAO in 58 patients with an average follow-up of 4 years[5]. They had a similar experience in that patients with minimal to moderate preoperative arthritic changes (Grade 0 - 2) had 78% to 85% good or excellent results, and patients with Grade 3 arthritic changes had 33% good or excellent results. Three of the 5 patients who eventually required total hip arthroplasty had grade 3 osteoarthritic changes. Overall, the authors' results supported the Bernese PAO as an excellent option for symptomatic young patients with mild to moderate arthritic changes resulting from DDH. Crockarell et al. reviewed 21 patients treated with the Bernese PAO with an average follow-up of 3.2 years[2]. Overall, Harris Hip scores improved from 68 preoperatively to 86 postoperatively. Trumble et al. reported results on 124 Bernese PAO's performed on 115 patients with an average follow-up of 4.3 years[10]. Overall, Harris Hip scores improved from 65 preoperatively to 89 postoperatively. The authors' noted that preoperative arthrosis correlated with outcome. Patients with grades 0, 1, and 2 arthritic changes achieved Harris Hip scores of 89 – 92. Patients with grade 3 changes had an average Harris Hip score of 77. Murphy et al. reported on 135 Bernese PAO's performed on 130 patients with an average follow-up of 3.9 years[7]. Five hips, all with grade 3 arthrosis preoperatively, progressed to total hip arthroplasty. The authors did not specify outcomes for the remaining 130 hips.

Davey and Santore specifically reviewed complications occurring in the senior author's first 35 cases compared to his second 35 cases[3]. They categorized complications as major, moderate, and trivial. Major complications included 2 bleeding complications, reflex sympathetic dystrophy in 2 patients, one sciatic nerve palsy, one pulmonary embolus, and one ileum nonunion. Most notably, major complications decreased from 17% in the first group to 2.9% in the second group. Of note, none of the major complications had any substantial long-term sequela. Moderate complications included 3 ischial fractures, 2 superficial wound problems, 1 posterior column fracture, and 3 peroneal neuropraxias, none of which had any sequela. Matta et al. reported no major complications[5]. One patient underwent excision of symptomatic heterotopic ossification. Crockarell reported two intraarticular osteotomies in 21 patients that affected their ability to correct the dysplasia and two peroneal nerve palsies[2]. In Trumble's series, 3 patients had major arterial thromboses (2 femoral and 1 iliac) that required emergent intervention[10]. All of these occurred in patients treated through an ilioinguinal ap-

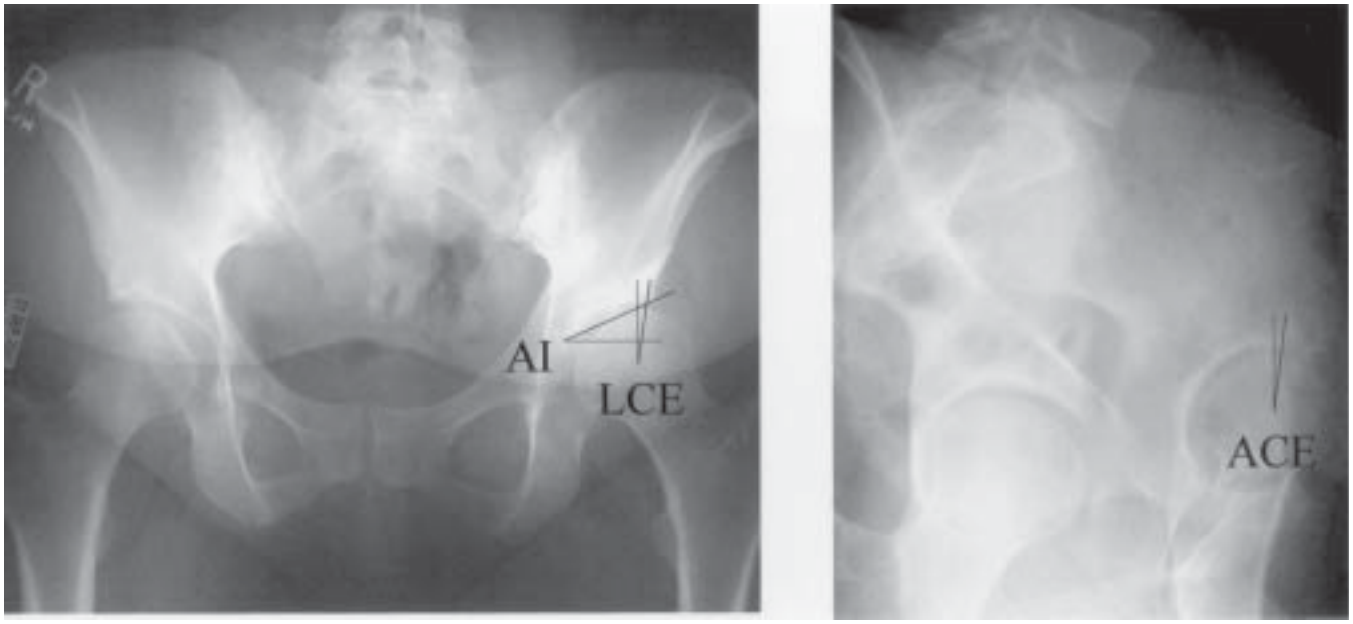

Figure 1A

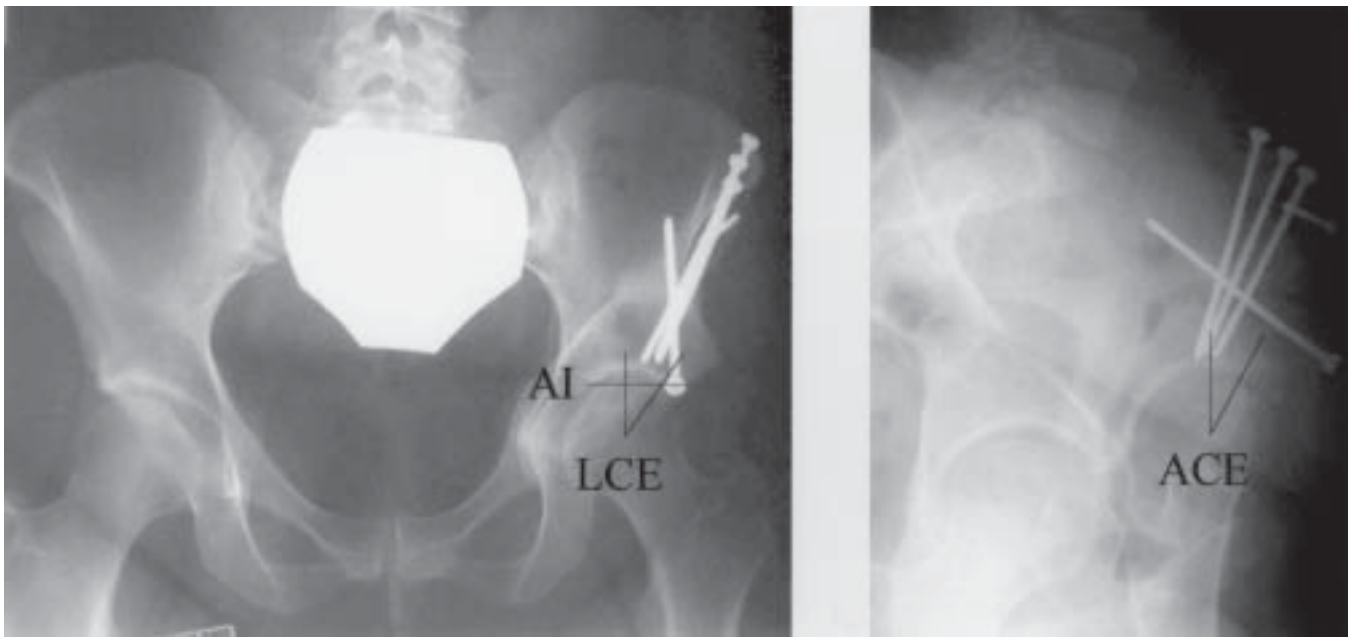

Figure 1B

Figure 1. Preoperative and postoperative AP and false profile x-rays of a 34 year old woman with left hip pain and dysplasia. Preoperative lateral center edge angle (LCE) was 7 degrees, anterior center edge angle (ACE) was 5 degrees, and acetabular index (AI) was 28 degrees (Figure 1A). Postoperative x-rays demonstrate an LCE of 36 degrees, an ACE of 30 degrees, and an AI of 0 degrees (Figure 1B).

proach and the authors subsequently have abandoned this approach. They reported 2 deep infections requiring multiple debridements and they also had to evacuate 5 postoperative hematomas.

In summary, short-term outcomes from multiple centers have remarkably consistent results. Patients with minimal to moderate arthritic changes usually achieve substantial pain relief and improved function. Conversely, patients with advanced radiographic arthritic changes had much less predictable success. There is a substantial learning curve in performing the operation. Major complications tended to occur early in all authors'

series and this was shown to be statistically significant by Davey and Santore[3]. Infection rates are uniformly low. Vascular problems in patients operated through an anterior approach are minimal. The most clinically significant complications have been intraarticular osteotomies. Finally, surgical corrections were markedly consistent (Table 1). The operation allows substantial three-dimensional improvement of femoral head coverage.

Siebenrock et al. were the first to report longer-term results on 71 of the original 75 patients operated on by Ganz[8]. Fifty-eight of 71 (82%) hips had been preserved over a mean follow-up period of 11.3 years. Hip reconstruction was performed at a mean of 6.1 years after the PAO in the 13 patients who did not preserve their hips. Fifty-two of the 71 hips (73%) were graded good or excellent by the Harris hip score and the D'Aubigne hip score. Eighty percent of preserved hips had grade 0 or 1 osteoarthritis. Again the authors found that advanced radiographic preoperative arthritis was a strong predictor of poor outcome.

UNIVERSITY OF IOWA EXPERIENCE

From October 2000 through February 2003, 36 Bernese PAO's have been performed on 30 patients at the University of Iowa by a single surgeon. This report summarizes preoperative and postoperative radiographic corrections, quantifies perioperative data, and details complications of these initial consecutive 36 operations. Patients include 24 women and 7 men with an average age of 33 years (+/- 7.9 years; range 15 – 47 years). The typical patient is a young woman with an unexplained, insidious history of progressive buttock or groin pain that has lasted approximately 6 months to several years. The pain is often mistaken for low back pain and a substantial percentage of patients have been treated for mechanical back pain.

Typical physical findings include fairly normal range of motion but substantial exacerbation of pain with flexion, adduction, and internal rotation. Standard radiographic examination begins with a standing AP xray of the pelvis to determine lateral femoral head coverage (lateral center edge angle), determine the slope of the roof of the acetabulum (acetabular index), and judge pre-existing arthritic changes (Figure 1). A false profile view (standing lateral of the affected hip which is rotated approximately 20 degrees anterior to the unaffected hip) is obtained to determine anterior coverage of the femoral head (anterior center edge angle) (Figure 1). An obturator oblique view is obtained to judge posterior coverage of the femoral head. An AP of the pelvis with the affected hip in maximum abduction and internal rotation combined with a false profile view in flexion allow assessment of how the hip joint will appear after correction.

Normal hips have a lateral center edge angle (LCE) and anterior center edge angle (ACE) greater than 25 degrees. The acetabular index (AI) is 8 degrees or less showing a horizontal roof of the acetabulum. In dysplastic hips, radiographs show deficient coverage of the lateral and anterior femoral head due to a shallow acetabulum (Figure 1A). The degree of lateral deficiency compared to anterior deficiency of the acetabulum is highly variable; therefore three-dimensional CT scans are obtained on all patients to aid in planning intraoperartive correction. In the 36 hips in this series, the mean preoperative lateral center edge angle was 8 degrees, anterior center edge angle was 5 degrees, acetabular index was 20 degrees, and Shenton's line was broken in 10 hips.

Surgery is performed through a modified anterior approach[5]. The acetabulum is mobilized through a series of cuts maintaining the continuity of the pelvic ring[4]. Meticulous care is taken to optimize the final position of the acetabulum. The goal is a femoral head that is medialized and well centered under a horizontal roof of the acetabulum. This is judged on AP and false-profile views taken with fluoroscopy and a hard-copy intraoperative AP pelvis radiograph. Care is taken to avoid uncovering to posterior aspect of the femoral head by overcorrecting anterior coverage or retroverting the acetabulum. This is checked with obturator oblique fluoroscopic view and the AP pelvis x-ray. Once the fragment is positioned, it is secured with four screws (Figure 1B). In the 36 hips in this series, the mean postoperative lateral center edge angle was 32 degrees, anterior center edge angle was 31 degrees, acetabular index was 1 degree (Figure 1B).

Average operative time was 217 minutes and blood loss averaged 956 ml for all 36 patients. Average operative time for the first 18 patients was 237 minutes (+/- 40 minutes; range 195 – 348 minutes) compared to 197 minutes (+/- 36 minutes; range 144 – 287 minutes) for the second 17 patients ($p = 0.003$) (Figure 2). Average blood loss for the first 18 patients was 1147 ml (+/- 681 ml; range 600 – 2800 ml) compared to 766 ml (+/- 353 ml; range 300 – 1500 ml) for the second 17 patients ($p = 0.04$) (Figure 3). Patients are allowed touch-down weightbearing for the first eight postoperative weeks and then progress as tolerated. The vast majority of patients are full weightbearing by three months after surgery and have returned to laboring occupations by six months. Patients with sedentary occupations are usually back to work within two months.

There have been six major complications in the whole series. These include four poor corrections, one pero-

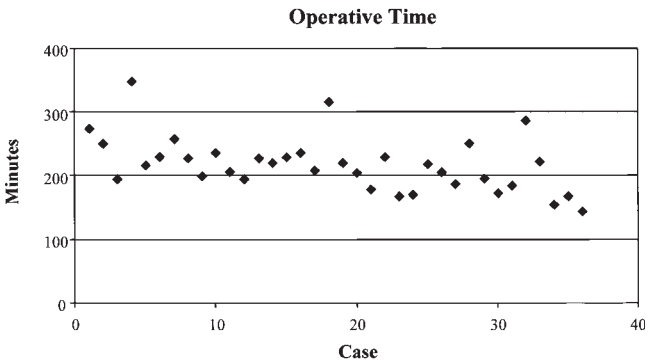

Figure 2. Operative time for 36 patients undergoing the Bernese PAO at the University of Iowa.

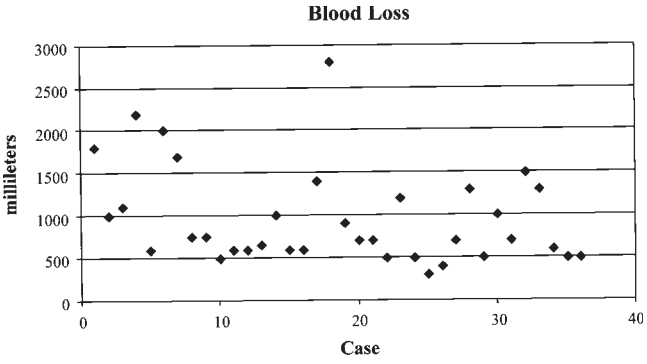

Figure 3. Estimated blood loss for 36 patients undergoing the Bernese PAO at the University of Iowa.

neal nerve palsy, and one case of post-operative pulmonary edema. Minor complications include seven cases of superficial wound problems and one case of asymptomatic heterotopic bone.

Two cases with poor corrections occurring early in the series (cases 1 and 4) were overcorrected with excessive anterior coverage. Both patients have had substantial clinical improvement but have not had complete resolution of pain. Two cases later in the series (cases 28 and 32) were undercorrected. One of the cases of undercorrection involved a 47 year old woman with severe dysplasia and preoperative hip subluxation. Radiographs taken two months after surgery showed her hip had resubluxated, however her acetabular fragment appeared well healed and unchanged in position. Her acetabulum was undercorrected, and her substantial femoral valgus deformity had been ignored. She is currently scheduled for a varus proximal femoral osteotomy. The second case of undercorrection (case 32) was a difficult case in a 41 year old male with moderate to advanced arthritic changes. This patient had a prolonged operation and suffered postoperative pulmonary edema requiring four days of mechanical ventilation. Review of preoperative abduction with internal rotation radiographs demonstrated poor improvement of joint space. In retrospect, the patient was a poor candidate for acetabular realignment.

There has been one postoperative peroneal nerve palsy (case 8). This patient had a proximal femoral osteotomy concurrent with her PAO. The patient has subsequently recovered some ankle and great toe dorsiflexion but still requires a shoe insert for persistent footdrop. Most patients have experienced transient numbness in the distribution of the lateral femoral cutaneous nerve, which typically resolves. No patients have had any ongoing complaints due to dysfunction of the lateral femoral cutaneous nerve.

There have been no intraarticular osteotomies or inadvertent fractures during surgery. There has been a substantial incidence (7 of 36 cases) of superficial wound dehiscence at the apex of the curved incision in the region of the ASIS. This problem has occurred only in obese patients. One patient did not respond to local wound care and required an operative debridement and repeat wound closure 6 weeks postoperatively. She has subsequently healed with no sequela. The other 6 patients responded to wet-to-dry dressing changes and a 10 day course of cephalexin. Obese patients now have their skin closed with monofilament, interrupted sutures, avoiding subcuticular techniques.

Only 13 patients have at least two years follow-up making it too early to report outcomes from this series. However, the majority of patients have had complete to nearly complete pain relief. Radiographic corrections are detailed in Table 1. Values compare well with other reports in the literature demonstrating the substantial correction of acetabular dysplasia which can be achieved with the Bernese PAO.

DISCUSSION

This consecutive series clearly demonstrates the significant learning curve associated with the Bernese PAO. Surgical complications have occurred throughout the entire case series, including two poor corrections later in the series (cases 28 and 32). The poor surgical corrections are likely the most significant complications in this series of patients. Fortunately, other serious complications including vascular injuries, neurologic injuries, and deep infections are rare in the current series and in other reports. Operative time and blood loss showed a significant improvement in the second 18 cases and figures 2 and 3 suggest they have not plateaued.

Outcomes from the current series are unknown. However, the majority of patients have enjoyed dramatic pain relief and have substantially improved their daily function. Outcomes from other authors reporting early results (mean postoperative periods of 3.2 – 4.3 years) have been consistently good, especially in patients who meet ideal indications. Siebenrock et al. have shown that the consistent early success reported by several institutions held up for greater than ten years, casting optimism for longer-term success of the Bernese PAO.

The literature strongly suggests that young patients with dysplastic hips and minimal to mild arthritic changes are ideal candidates for this operation. Patients with moderate arthritic changes have also consistently fared well. The operation becomes less predictable with advanced preoperative arthritic changes. However, the literature demonstrates that even a substantial percentage of patients with advanced arthritic changes (grade 2 and 3) have good short-term and medium term outcomes. The difficult patient becomes increasingly clear from the literature: a young patient with dysplasia with advanced arthritic changes. The PAO is less predictable but arthroplasty in a 20 to 40 year old active patient is undesirable. Murphy and Deshmukh have shown that patients with advanced arthrosis that improved their joint space with functional radiographs predictably improved with the PAO, even with advanced arthrosis. Functional radiographs included comparing anterior/posterior views with the hip in neutral position compared to the hip in maximum abduction and internal rotation. They also looked at false-profile radiographs with the hip extended compared to the hip flexed. Improvements in joint space on both views correlated with good outcomes in patients with advanced preoperative arthritic changes.

As with any major elective intervention, careful consideration of the ratio of benefits and risks dictate proper indications. Davey and Santore demonstrated complication rates significantly decrease with experience. More importantly they found that the major complications encountered by their patients left no long-term sequela. In general, patients undergoing the Bernese PAO are young and healthy, allowing them to tolerate and recover from predictable perioperative complications. While only one-half the size of Davey and Santore's series, the current series had similar findings. Complications with this operation occur but patients tend to overcome them and enjoy substantial benefits from acetabular realignment.

In conclusion, the Bernese PAO remains a technically demanding operation that has potential to profoundly improve patient function and prolong the useful life of dysplastic hips. The ideas conceived, executed, and taught by Ganz have now achieved reproducible short-term success, and in one report, continued longer-term success. While complications should be expected, they decrease with experience and usually cause minimal long-term sequela. Fortunately, patient outcomes have justified expected complications.

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THE EFFECT OF FIXATION TECHNIQUE ON GRAFT POSITION IN ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION

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ABSTRACT

The purpose of this paper was to determine the extent to which the technique used in ACL reconstruction and fixation influences graft placement.

This is a Comparative Radiographic cohort study.

Precise graft placement is one of the most crucial components of a successful anterior cruciate ligament (ACL) reconstruction. Two commonly used techniques of ACL reconstruction are arthroscopic bone–patellar tendon–bone (B-PT-B) autograft with interference screw fixation and semitendinosus and gracilis (ST + G) tendon autograft with endobutton femoral and multiple staple belt buckle tibial fixation. Using radiographic measurements of bone tunnel position following ACL reconstruction this study quantified the extent to which these techniques influenced graft placement.

Femoral and tibial tunnel position in ACL reconstruction was determined using the post-operative radiographs of 40 male patients who had undergone ACL reconstruction (20 B-PT-B and 20 ST+ G). The primary outcome measure was location of bone tunnel position following ACL reconstruction. Measurements were based on the guidelines of Amis et al.

Femoral tunnel position of B-PT-B grafts was an average of 9.36% more anterior in the sagittal plane than with ST + G grafts. The mean position of B-PT-B grafts was at 31.11% (SD= 5.45%). The mean position of the ST + G grafts was 21.76% (SD= 6.62%). This difference between the two was found to be significant (p<.001).

As demonstrated by this study, placement and orientation may vary to accommodate technique and fixation. Clinical outcomes measured were similar in both groups.

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INTRODUCTION

Reported success rates for current techniques of ACL reconstruction in terms of functional stability, relief of symptoms and return to activity range from 75% to 90%.[1,2,3,4] Reports of unsatisfactory long-term results range from 5% to 52%.[1,2,3,4,5] Graft failures with recurrent functional instability occur in 0.7% to 8% of ACL reconstructions.[1,2,6]

Precise graft placement is felt to be crucial in a successful ACL reconstruction.[7] In fact, the most common surgical cause of graft failure is non–anatomic bone tunnel placement.[1,8] Palmer emphasized the importance of graft placement over sixty years ago.[9] Since that time many different graft placements have been advocated and their merits debated and rejected.[10,11] If a femoral tunnel is placed too anteriorly, the graft will tighten in flexion and become slack in extension.[1,10,12]. A tibial tunnel placed too far anteriorly will cause graft impingement on the roof of the intercondylar notch.[1,11,13,14,15,16,17] A tibial tunnel placed too far posteriorly will be impinged on the posterior cruciate ligament (PCL).[1,11] Medial or lateral malpositioning of the tibial tunnel may result in impingement of the graft by the femoral condyles.[1,11,18]

Yoshiya et. al. has described the optimal orientation of bone tunnels when bending and strain on the graft throughout the range of motion of the knee is minimal.[19] The consequences of non-anatomical positioning of an ACL graft are abnormal length and tension changes within the graft throughout range of motion that may lead to unacceptable laxity.[1,4,10,12,20] Theoretically, placing the graft at isometric points would solve this problem. However, there are no such points on the tibia or femur that create isometry in the normally functioning knee.[10] Therefore, the current goal, is to position the graft anatomically in order to resist anterior translation of the tibia on the femur during the weight-bearing phase of gait. This is best accomplished by placing the femoral tunnel as posteriorly as possible, while the tibial location is anatomical.[1,4,10,21,22,23]

At the study centre, two techniques are commonly used for ACL reconstruction: B-PT-B reconstruction with interference screws fixation and doubled ST+ G reconstruction with Endobutton™ femoral fixation and tibial fixation with multiple low profile staples in a belt buckle arrangement. Endoscopic interference screw fixation requires 1mm to 2mm of cortical bone surrounding the tunnel to avoid cortical breakout[4,21,26] that may predispose to anterior femoral tunnel placement, while EndobuttonTM fixation allows posterior breakout at the notch entrance to the femoral tunnel. The purpose of this paper was to determine if the technique utilized affects graft position.

MATERIALS AND METHODS

All male patients undergoing primary ACL reconstruction by one surgeon at the center from 1994 to 1997 were identified through the patient database. All male patients 18 years of age or greater who had undergone isolated primary ACL reconstruction with autogenous B-PT-B or ST + G grafts at least 12 months previously were included in this study. Exclusion criteria included other techniques, additional procedures (i.e. meniscal allograft or posterolateral reconstruction), revision surgery and bilateral injury. Twenty patients who had undergone B-PT-B reconstruction and a matched cohort of 20 with ST + G reconstruction and who met the study criteria were randomly chosen. All patients in both groups returned for clinical and radiographic evaluation. An independent examiner carried out standard follow-up evaluations and operative findings and hospital charts were reviewed.

Primary outcome measures were radiographic measurements of bone tunnel position following ACL reconstruction. Graft position was assumed to be at the center of these bone tunnels on anteroposterior (AP) and lateral radiographs. Measurements were based on the guidelines of Amis et al.[27] True AP and lateral x-rays were taken at follow-up (Figure 1). The use of fluoroscopy helped eliminate rotation and allowed digital enhancement of the images. Clinical outcome measures included the International Knee Documentation Committee (IKDC) Assessment, The Mohtadi Quality of Life Assessment in Anterior Cruciate Deficiency[28] and KT 1000TM Arthrometer measurements of anterior tibial translation.[29]

The position of the femoral tunnel was measured on the lateral films as a percentage of the distance along Blumensaat's line extended to the outline of the femur (Figure 2). The over-the- top position was designated as 0%. The point at which this line crossed the anterior cortex of the lateral femoral condyle was designated as 100%. The position of the tibial tunnel was measured in the sagittal (lateral) plane as a percentage of the distance from anterior to posterior of a line drawn tangential to the medial tibial plateau. The positions of both the femoral and tibial tunnels were measured in the coronal (AP) plane as a percentage of the distance along the tibial plateau from medial to lateral (Figure 2).

Orientation of the tibial tunnel was measured relative to the posterior shaft of the tibia in the sagittal and the lateral shaft of the tibia in the coronal planes in all subjects. The orientation of the femoral tunnel was mea-

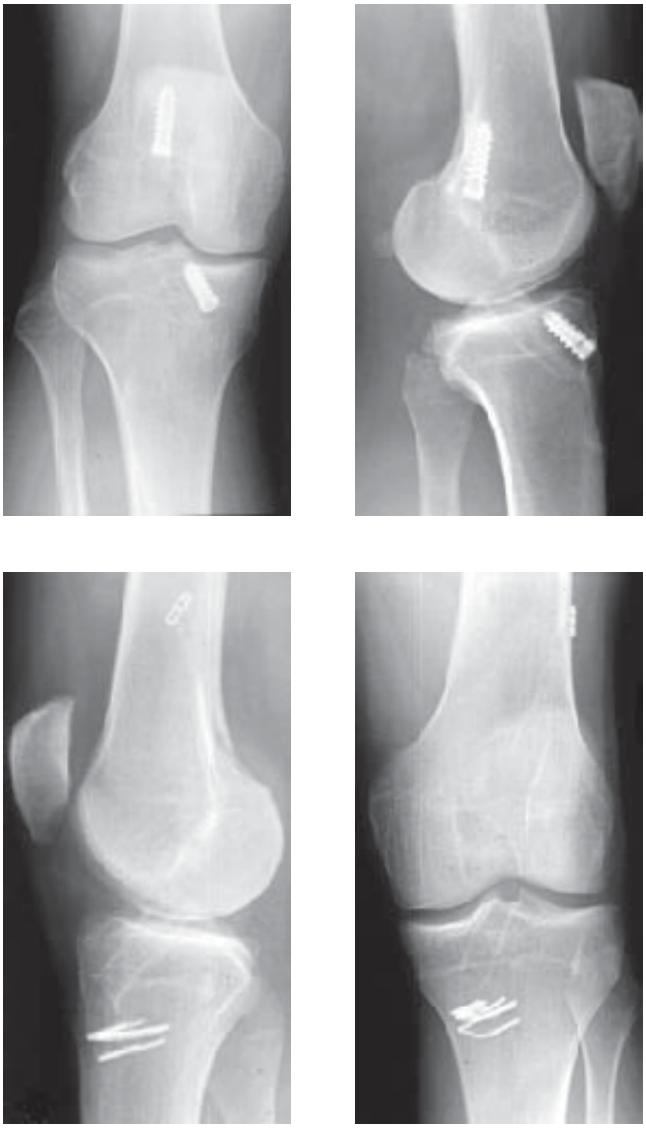

Figure 1: Radiographs obtained of postoperative Bone-Patellar Tendon-Bone and Hamstring ACL reconstructions. Rotation has been eliminated by the use of fluroscopy. (A: AP-BPTB; B: Lateral-BPTB; C: AP-ST/G; D: Lateral-ST/G).

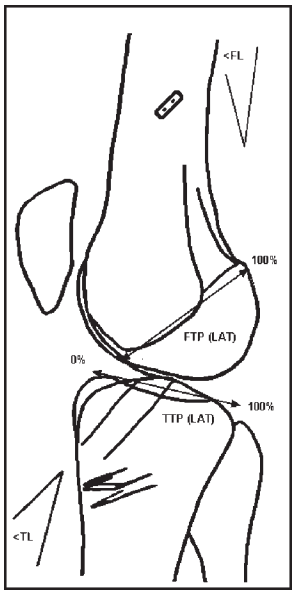

Figure 2: Measurements made on lateral radiographs (<FL = Angle of the femoral tunnel in the sagittal plane (lateral view); <TL = Angle of the tibial tunnel in the sagittal plane (lateral view); FTP(LAT) = Position of the femoral tunnel in the sagittal plane (lateral view); TTP(LAT) = Position of the tibial tunnel in the sagittal plane (lateral view).)

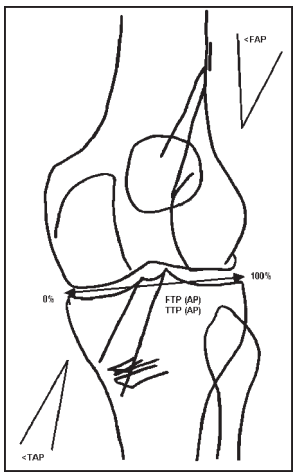

Figure 3: Measurements made an Anteroposterior radiographs (<FAP = Angle of the femoral tunnel in the coronal plane (AP view); <TAP = Angle of the tibial tunnel in the coronal plane (AP view); FTP(AP) = Position of the femoral tunnel in the coronal plane (AP view); TTP(AP) = Position of the tibial tunnel in the coronal plane (AP view).)

sured relative to the posterior shaft of the femur in the sagittal plane. The orientation of the femoral tunnel in the coronal plane was measured relative to the tibial plateau. This measurement differs from Amis et al. since sufficient length of the femoral shaft is not consistently present on AP radiographs of the knee for accurate measurement. A more detailed description of the measurements used and illustrated in Figures 2 and 3 can be found in Figure 4.

RESULTS

Average time to follow–up was 27.5 months (range: 12 to 54 months). Demographic information and follow-up times were similar in each group. The results of radiographic measurements are shown in Table 1. In the sagittal plane, the femoral tunnel in B-PT-B reconstruction was an average of 9.36% more anterior than in ST + G reconstruction. The mean position of B-PT-B grafts was 31.11% (SD=5.45%) and of the ST + G grafts was 21.76% (SD=6.62%). This difference is statistically significant (p<.001). Between groups no difference was noted in femoral tunnel position in the AP plane or in the tibial tunnel position in either AP or lateral planes. There was a significant difference (p=0.011) between

FIGURE 4

Description of measurements made on radiographs.

A - Lateral Radiographs

FTP(LAT): Position of the femoral tunnel in the sagittal plane (lateral view). Measurements are based on a line drawn along the sclerotic representation of the roof of the intercondylar notch (Blumensaat's Line) extended from the posterior cortex to the anterior cortex of the lateral femoral condyle. The point at which the line crosses the posterior cortex is designated as 0%. The point at which the line crosses the anterior cortex is designated as 100%. Measurements to the center of the femoral tunnel are reported as a percentage of the way along this line.

TTP(LAT): Position of the tibial tunnel in the sagittal plane. Measurements are based on a line drawn tangent to the medial tibial plateau. The point at which the plateau drops off anteriorly is designated as 0%. The point at which the plateau drops off posteriorly is designated as 100%. Tunnel position is reported as a percentage along the reference line.

<FL: Angle of the femoral tunnel in the sagittal plane (lateral view). The tunnel angle is measured with reference to the posterior shaft of the femur.

<TL: Angle of the tibial tunnel in the sagittal plane. The tunnel angle is measured with reference to the posterior shaft of the tibia.

B - Anteroposterior Radiographs

FTP(AP): Position of the femoral tunnel in the coronal plane (AP view). Measurements are reported as a percentage of the distance from medial to lateral along the tibial plateau.

TTP(AP): Position of the tibial tunnel in the coronal plane (AP view). Measurements are reported as a percentage of the distance from medial to lateral along the tibial plateau.

<TAP: Angle of the tibial tunnel in the coronal plane. The tunnel angle is measured with reference to the lateral shaft of the tibia.

<FAP: Angle of the femoral tunnel in the coronal plane. The tunnel angle is measured with reference to the tibial plateau. We discarded this measurement as this angle is not consistently evident on AP radiographs.

TABLE 1

VARIABLE	MEAN	STANDARD DEVIATION	DIFFERENCE BETWEEN GROUPS	STATISTICAL SIGNIFICANCE
FTP(LAT) – BPTB **FTP(LAT) – HAM**	31.11% 21.76%	5.45% 6.62%	9.36%	**P<0.001**
TTP(LAT) – BPTB **TTP(LAT) – HAM**	33.75% 37.45%	9.31% 11.25%	3.69%	P=0.257
FTP(AP) – BPTB **FTP(AP) – HAM**	55.27% 55.41%	8.31% 5.80%	0.13%	P=0.953
TTP(AP) – BPTB **TTP(AP) – HAM**	43.22% 42.49%	4.73% 4.76%	0.73%	P=0.620
<FL – BPTB **<FL – HAM**	23.6° 24.0°	9.4° 7.6°	0.4°	P=0.865
<TL – BPTB **<TL – HAM**	39.3° 34.8°	8.6° 6.1°	4.5°	P=0.063
<TAP – BPTB **<TAP – HAM**	13.4° 19.9°	9.1° 5.9°	6.5°	**P=0.011**
<FAP – BPTB / HAM	X	X	X	X

Table 1: Results of radiographic measurements. (Note: X denotes unobtainable value). (<FL=Angle of the femoral tunnel in the sagittal plane (lateral view); <TL=Angle of the tibial tunnel in the sagittal plane (lateral view); FTP(LAT)=Position of the femoral tunnel in the sagittal plane (lateral view); TTP(LAT)=Position of the tibial tunnel in the sagittal plane (lateral view); <FAP=Angle of the femoral tunnel in the coronal plane (AP view); <TAP=Angle of the tibial tunnel in the coronal plane (AP view); FTP(AP)=Position of the femoral tunnel in the coronal plane (AP view); TTP(AP)=Position of the tibial tunnel in the coronal plane (AP view); BPTB=Bone Patellar Tendon-Bone Group; HAM=Hamstring Tendon Group.)

TABLE 2

TEST FORCE	MEAN SIDE TO SIDE DIFFERENCE	STANDARD DEVIATION	DIFFERENCE BETWEEN GROUPS	STATISTICAL SIGNIFICANCE
20N – BPTB **20N – HAM**	1.67mm 1.06mm	1.15mm 1.45mm	0.61mm	P=0.239
30N – BPTB **30N – HAM**	2.05mm 1.53mm	1.29mm 1.34mm	0.52mm	P=0.304
MAX –MAN – BPTB	2.13mm 2.76mm	1.22mm 1.25mm	0.63mm	P=0.191

Table 2: Results of KT-1000 arthrometer measurements. (BPTB=Bone Patellar Tendon-Bone Group; HAM=Hamstring Tendon Group; MAX-MAN=Maximum Manual Translation.)

groups in tibial tunnel angle in the coronal plane. In the B-PT-B group the tibial tunnel was 6.5˚ more vertical the than in the ST + G group. The mean tibial tunnel angle was 3.4˚ (SD=9.1˚) relative to the shaft of the tibia in the B-PT-B group and 19.9˚ (SD=5.9˚) in the ST + G group. There were no other differences in tunnel orientation as measured radiographically.

No clinical differences were found between groups in KT-1000™ measures (Table 2), or Quality of Life (Table 3). IKDC scores in the B-PT-B were 29% A, 54% B, 17% C and 0% D compared to 6% A, 59% B, 35% C and 0% D in the ST + G group.

DISCUSSION

Several studies have measured graft position. This study compared graft position between two common techniques. Our results suggest that the B-PT-B technique with endoscopic femoral interference screw fixation may result in a more anterior and therefore less anatomical graft position. While this may be a factor in graft failure, tunnel position in both study groups was acceptable based on published recommendations and our clinical outcome was not significantly different.

TABLE 3

VARIABLE	MEAN	STANDARD DEVIATION	DIFFERENCE BETWEEN GROUPS	STASTISTICAL SIGNIFICANCE
S & P C – BPTB **S & P C – HAM**	84.69 75.50	9.54 20.10	9.19	P=0.106
W – R C – BPTB **W – RC – HAM**	83.80 73.33	17.49 20.55	10.47	P=0.118
R A S P / C – BPTB **R A S P / C – HAM**	67.15 60.73	24.68 23.41	6.42	P=0.421
L S – BPTB **L S – HAM**	79.27 76.14	18.37 18.58	3.13	P=0.610
S & E – BPTB **S & E – HAM**	72.98 64.75	22.43 22.79	8.23	P=0.268
TOTAL QOL – BPTB **TOTAL QOL – HAM**	**73.70** **67.90**	**18.41** **17.98**	**5.80**	**P=0.34**

Table 3: Results of Mohtadi Quality of Life Following Anterior Cruciate Ligament Reconstruction Questionnaire. (S & PC=symptoms and physical complaints; W-RC=work-related concerns; RASP/C=recreational activities and sport participation or competition; LS=lifestyle; S&E=social and emotional).

Previous work by Good and colleagues compared the tibial and femoral tunnel positions in cadaver knees after drilling one group of tunnels free hand and another group using a drill guide.[30] In this study, the femoral tunnel positions were further anterior in both the freehand and drill guide groups than in either of our two groups {Good: cadaver group: 34%; freehand: 40%; drill guide: 33%}. In fact Good et al. measured the femoral tunnels from anterior to posterior and we have adjusted the values here to correspond to our convention. Also, in this study grafts were more posterior than the cadaver grafts in the Good study. The tibial tunnels in both of our groups were comparable to all three of Good's groups {cadaver: 32%; freehand: 33%; drill guide: 33%}.

Lintner et al. showed that the femoral tunnel was placed more parallel to the shaft of the femur when the tunnels were drilled with endoscopic technique than when the tunnels were drilled from the outside in.[31]

Khalfayan et al. attempted to correlate clinical results with graft position.[32] They reported a positive correlation between clinical outcomes and posterior graft position. However, they divided their data into two groups: one with the femoral tunnel position greater than 40% anteriorly and one with the femoral tunnel less than 40% anteriorly. They also used the same method as Good et al to report their measurements. Khalfayan and colleagues were also able to calculate the interobserver and intraobserver variability for these radiographic measurements and found them to be acceptable. They concluded that the tunnel position should be at least 60% posteriorly along Blumensaat's line. In our study, grafts in both groups, were at least 60% posterior along Blumentsaat's line.

Yoshiya et al. determined the optimum orientation of bone tunnels in ACL reconstruction based on minimizing bending and strain on the graft.[19] They found that the tibial tunnel should be oriented 20˚ anteriorly in the sagittal plane and 30˚ medially in the coronal plane. In our study, the ST + G grafts were significantly closer to this ideal in the coronal plane than the B-PT-B grafts. (Hamstring: 19.9˚; B-PT-B: 13.4˚. p=0.011) The clinical outcome in both groups is similar.

It is generally accepted that the most anatomic placement of the ACL graft is posterior along femoral notch. However, most published reports suggest that there exists a range of acceptable graft placement, explaining the similar outcome in both groups in this study.

One limitation to this study is that tunnel sizes may vary from one subject to the next. It is common surgical practice to measure the diameter of the graft after harvesting and drill the tunnels accordingly. Therefore, tunnel diameters can vary from 7mm to 10mm. Tunnels may then be chamfered to allow a better graft fit. In our technique, the measurements were made to the center of each tunnel. The impact of this method on our measurement is not known, but should be minimal since the centre of the tunnel was used.

There is evidence that bone tunnels may change in shape and diameter following ACL reconstruction.[33,34] Current studies show that this is less apparent with autografts than with allograft tissue. Pilot data at our centre show that there may be a significant change in

the size and shape of the tunnels with time in our autograft procedures. This may affect the reliability of our measurement techniques. However, in this matched cohort study, the results and group are comparable. The next logical step is to investigate the exact nature of tunnel widening following ACL reconstruction. Prospective studies to evaluate this are currently under way at our centre.

In theory, graft placement should be similar regardless of fixation technique or type of graft. However, in practice, graft placement and orientation may, as demonstrated in this study, vary to accommodate technique and fixation. With the endoscopic interference screw fixation for the B-PT-B graft, cortical breakout needs to be prevented. Therefore, the tibial tunnel is slightly more vertically orientated and the femoral tunnel is slightly more anterior. With Endobutton™ fixation cortical breakout can occur without consequences and the femoral tunnel can be slightly more posterior. Clinical outcomes were similar because graft placement was within the ideal range in both groups.

In general the most common reported cause of graft failure is graft placement that is too anterior causing impingement on the roof of the notch. In the single incision, BTB ACL reconstruction, there is a tendency to remain somewhat more anterior to avoid posterior breakout when compared to the Endobutton™ hamstring technique.

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EARLY POSTOPERATIVE MORTALITY FOLLOWING TOTAL HIP ARTHROPLASTY IN A COMMUNITY SETTING: A SINGLE SURGEON EXPERIENCE

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ABSTRACT

The senior author performed 4967 total hip arthroplasties, 4164 primary and 803 revision, between 1970 and 1996 in a community setting. All charts were evaluated for postoperative death including in-house, 30, 60, and 90 days following the procedure. 1.0 per cent of patients (42) died following a primary procedure and 0.87 per cent (7) died following a revision procedure within 3 months of surgery. The number of in-house deaths were 26 (0.52 per cent), including 21 in the primary group (0.5 per cent) and 5 in the revision group (0.62 per cent). In both the primary and revision situation and independent of co-morbidities, age greater than 70 years was significantly associated with increased risk for postoperative mortality ($p<0.0001$), with 0.44% mortality in patients aged 70 years or younger (0.51 per cent primary and 0.00 per cent revision), and 1.45 per cent mortality in patients greater than 70 years of age (1.38 per cent primary and 1.86 per cent revision). This experience of a single surgeon with a high volume community practice performing both primary and revision total hip arthroplasty, documents a low but significant incidence of postoperative death in the first 3 months following total hip arthroplasty (0.98 per cent).

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INTRODUCTION

Death during or shortly following the total hip arthroplasty procedure has been well-documented. However, there are few series of large numbers of patients where the incidence and etiology have been studied.

The purpose for this study was to retrospectively examine the causes for postoperative mortality in the in-house period and first 30, 60, and 90 days following total hip arthroplasty performed by a single surgeon in a community setting with a high volume practice over a 26 year period. Age at surgery, primary diagnosis, anesthesia method and comorbid conditions were evaluated in primary and revision cases, including those with hip fractures or malignancy. The information from this single surgeon's experience of 4967 total hip arthroplasties over 26 years with few changes in the procedure should provide a benchmark for comparison of mortality rates associated with total hip arthroplasty performed with other anesthetics, other surgical techniques, other postoperative management regiments and in similar (i.e., community) or varied (i.e., tertiary care) settings.

MATERIALS AND METHODS

Between 1970 and 1996, the senior one of us (R.C.J.) performed 4967 total hip arthroplasties on 3865 patients, 4164 primary and 803 revision cases. There were no changes made in surgical technique during this time. Anesthesia method consisted of a combination general anesthesia with spinal anesthesia employed for improved muscle relaxation during the procedure. Routine postoperative care included in-house dextran administration and aspirin, anti-embolic stockings, and multi-vitamins for 2 months postop. Patients with revision surgery also received oral antibiotic prophylaxis postoperatively for 6 months. Earlier mobilization postoperatively was begun in 1982. Primary diagnoses included primary osteoarthritis, post-traumatic arthritis, rheumatoid arthritis, congenital hip dysplasia, osteonecrosis, nonunion of the femoral neck, Legg Perthes, acetabular fracture with secondary osteoarthritis, slipped capital femoral epiphysis with secondary osteoarthritis, and malignancy. Utilizing a retrospective review of these records, those who died while in-house and within 30, 60, and 90 days of total hip arthroplasty

were analyzed according to age, weight, year of surgery, primary diagnosis, primary versus revision, comorbid conditions and anesthesia method. All patients had a preoperative medical evaluation by an internist which was recorded in the office chart. Autopsies were performed in 20% of cases, with the remainder of the causes of death determined by clinical diagnosis and death certificates. In addition, all patients, who have now been extensively databased, were reviewed to see if the date of death (of deceased patients) occurred within 3 months of their operations. Fisher's exact test was used to compare primary and revision cases with regard to age, year of death and comorbid conditions.

RESULTS

49 patients (0.98 per cent) died within 90 days of total hip arthroplasty, including 42 primary (1.0 per cent) and 7 revision (0.87 per cent). The average age at the time of surgery in the primary cases was 70 years, compared to the senior surgeon's overall age in the primary situation of 63 years. In revision cases, the average age at the time of surgery was 79 years, compared to an average age of 61 years in the overall revision population. There was a statistical difference in age > 70 years in both primary and revision procedures ($p<0.0001$). There was no difference in death rate in primary vs. revision cases ($p=0.825$).

Classification of the 4164 primary cases by primary diagnosis included 3211 primary osteoarthritis, 273 post-traumatic including pathologic fractures, 339 nonunion, 179 congenital hip dysplasia, 178 rheumatoid arthritis, 83 osteonecrosis, 28 Legg Perthes, 27 acetabular fracture with secondary osteoarthritis, 26 slipped capital femoral epiphysis with secondary osteoarthritis, and 159 other cases including cases of malignancy.

Classification of deaths by diagnosis for primary procedures included 26 in the osteoarthritis group (0.81 per cent), 7 post-traumatic (2.6 per cent), 2 acetabular fractures with secondary osteoarthritis (7.4 per cent), 1 rheumatoid arthritis (0.56 per cent), and 6 other (3.8 per cent). There was no statistical difference between primary diagnoses ($p=0.0016$). Classification of deaths by reason for revision included 3 for aseptic loosening (1.0 per cent), 1 for septic loosening (0.1 per cent), 1 for recurrent dislocation (0.1 per cent), and 2 for femoral fracture (0.2 per cent).

Cause of death within 90 days postoperatively in primary cases included 12 myocardial infarction (0.28 per cent), 12 pulmonary embolism (0.28 per cent), 5 pneumonia (0.12 per cent), 4 congestive heart failure (0.09 per cent), 2 sepsis secondary to other infection (0.05 per cent), 1 respiratory arrest (0.02 per cent), 1 arrhythmia (0.02 per cent), 1 renal failure (0.02 per cent), 1 lung carcinoma (0.02 per cent), 1 suicide (0.02 per cent), and 2 multifactorial (0.05 per cent). Cause of death within 90 days postop in revision cases included 3 myocardial infarction (0.37 per cent), 1 pulmonary embolism (0.12 per cent), 1 pneumonia (0.12 per cent), 1 acute respiratory distress syndrome (0.12 per cent), and 1 hemorrhage (0.12 per cent) (Table I-V).

The number of in-house deaths were 26 (0.52 per cent), including 21 in the primary group (0.50 per cent) and 5 in the revision group (0.62 per cent). Most common causes for in-house deaths in the primary cases were 7 myocardial infarction (33 per cent), 8 pulmonary embolism (38 per cent), and 2 congestive heart failure (9 per cent), and in revision cases were 3 myocardial infarction (60 per cent), 1 pulmonary embolism (20 per cent), and 1 acute respiratory distress syndrome (20 per cent). There were no intraoperative deaths and cement was used in all cases. Average length of hospital stay was 14 days.

There were 27 deaths (0.54 per cent) noted within 30 days following surgery including in-house deaths, 22 in the primary group (0.52 per cent) and 5 in the revision group (0.62 per cent). Most common causes for 30-day deaths in the primary cases were myocardial infarction 7 (32 per cent), pulmonary embolism 9 (41 per cent), and congestive heart failure 2 (9 per cent), and in revision cases were 3 myocardial infarction (60 per cent), 1 pulmonary embolism (20 per cent), and 1 acute respiratory distress syndrome (20 per cent).

From 31-60 days, there were 15 deaths (0.30 per cent), including 14 in the primary group (0.34 per cent) and 1 in the revision group (0.12 per cent). Most common causes for death in the primary situation were pneumonia 5 (36 per cent), myocardial infarction 3 (21 per cent), and pulmonary embolism 2 (15 per cent). Most common cause of death in the revision situation from 31-60 days was pneumonia 1 (100 per cent).

There were 7 deaths that occurred 61-90 days following surgery (0.14 per cent), 6 in the primary group (0.14 per cent) and 1 in the revision group (0.12 per cent). Most common causes for death in these primary cases included myocardial infarction 2 (33 per cent), pulmonary embolism 1 (16 per cent), and in the revision group was a single pulmonary embolism (100 per cent)

Myocardial infarction was the most common cause of death overall (30.6 per cent), with fatal myocardial infarctions occurring in 12 within the primary group (0.28 per cent) and 3 in the revision group (0.37 per cent). Cardiac related complications (myocardial infarction, congestive heart failure and arrhythmia) accounted for 40 per cent (17 of 42) of primary deaths. In the primary situation, 8 fatal pulmonary emboli were noted

during the first 15 years of the senior surgeon's practice, whereas 3 were documented in the last 11 years. In addition, 34 of the 49 deaths (1.15 per cent mortality) occurred in the first 15 years, versus 15 deaths in the last 11 years (0.74 per cent mortality). The demographics of the postoperative deaths by time following surgery are recorded in Tables 1-4.

DISCUSSION

Postoperative mortality following total hip arthroplasty has been documented in many hip arthroplasty series.[1-10] Fatal pulmonary emboli has been the focus of most studies.[1,3,10] However, the literature is lacking in the study of postoperative mortality in a single surgeon's or multiple surgeons' practice involving a high volume of cases over an extended period of time.[2] The purpose of the present study was to evaluate the early mortality following total hip arthroplasty in a community setting performed by a high volume surgeon.

The incidence of mortality within 90 days after total hip replacement was 0.98 per cent in the present study. A significant correlate to the mortality rate was age greater than 70 years at the time of surgery, making the risk of mortality 0.44 per cent in those 70 years old or less, and 1.45 per cent risk of mortality in those greater than age 70. Death rates were similar for primary and revision cases. The lesser incidence of mortality following revision total hip replacement may represent the health of that population deciding to have the procedure.

Myocardial infarction, the most common cause of death (30.6 per cent) and cardiac related complications accounted for 40 per cent (17 of 42) of deaths following primary arthroplasties. Previous myocardial infarction, diabetes mellitus, and peripheral vascular disease in the patient histories did not correlate with increased incidence of fatal myocardial infarction following total hip replacement. Fatal pulmonary emboli decreased by a factor of 5 within the last 11 years of practice, likely related to earlier postoperative mobilization.

Dearborn & Harris[2] studied 2736 total hip arthroplasty procedures, and reported a 90-day mortality of 0.2 in per cent primary and 0.7 per cent in revision cases. These values are lower than the 1.0 per cent and 0.87 per cent, respectively, observed in this study. However, the average age of their patients was 59 years, well below the 70 year average of the present study. This difference may be related to the difference in practices between a community and tertiary care setting. Parvizi et al[4] reported a .29% 30 day mortality rate in the Mayo Clinic review once again a review in a tertiary center.

Sharrock[6] studied 15559 total hip arthroplasty and total knee arthroplasty cases, and noted a 3-fold decrease in in-house mortality associated with a change to epidural anesthesia with postoperative epidural analgesia (0.10 per cent from 0.36 per cent). Patients with fractures and metastatic cancer diagnoses were excluded.

Whittle[9] studied 6385 total hip arthroplasty cases in 1992 for 30 day postoperative mortality, and found mortality to be 0.34 per cent in patients aged 66-69 years and 3.75 per cent in patients aged 80 years or more, with the average mortality of 0.95 per cent. Patients with fractures were excluded from the analysis. Our study corroborates the increase in mortality with increasing age. Because the patient population undergoing elective or emergent total hip arthroplasty tends to be late middle age to elderly, the mortality rate in the general population not undergoing surgery must also be factored. Soderman[7] studied mortality following total hip arthroplasty in comparison with the general population, and found that the mortality for primary and revision arthroplasty patients was lower than an age- and sex-matched population.

Numerous studies have noted a significant increase in mortality following total hip arthroplasty in patients whose primary diagnoses included fractures and malignancy.[5] The present study did not find a significant difference in mortality between primary diagnoses, likely due to the much greater number of cases done for primary osteoarthritis versus post traumatic arthritis or malignancy conditions.

This study of a high volume community practice should provide a benchmark for comparison of mortality rates following total hip arthroplasty in other community settings as well as tertiary care settings. It should also provide a historical control for cases where newer anesthetic techniques and postoperative regiments are used.

TABLE 1
Total In-House Postoperative Deaths:

Primary Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Primary Osteoarthritis	74	M	12	Pulmonary Embolism
2	Primary Osteoarthritis	72	F	3	Congenital Heart Failure
3	Primary Osteoarthritis	75	F	0	Myocardial Infarction
4	Primary Osteoarthritis	75	M	2	Myocardial Infarction
5	Primary Osteoarthritis	80	M	18	Pulmonary Embolism
6	Primary Osteoarthritis	77	M	2	Myocardial Infarction
7	Primary Osteoarthritis	77	F	15	Pulmonary Embolism
8	Post Traumatic	88	F	5	Myocardial Infarction
9	Other	87	F	4	Myocardial Infarction
10	Primary Osteoarthritis	58	M	12	Pulmonary Embolism
11*	Primary Osteoarthritis	51	F	28	Pulmonary Embolism
12	Malignancy	76	M	16	Arrhythmia
13	Primary Osteoarthritis	80	F	7	Respiratory Arrest
14	Primary Osteoarthritis	81	M	15	Pulmonary Embolism
15*	Primary Osteoarthritis	73	M	14	Pulmonary Embolism
16	Primary Osteoarthritis	78	M	3	Myocardial Infarction
17	Primary Osteoarthritis	66	M	14	Pulmonary Embolism
18	Primary Osteoarthritis	80	F	27	Congenital Heart Failure
19**	Primary Osteoarthritis	85	M	21	Cardiac Failure
20	Post Traumatic	71	F	18	Myocardial Infarction
21	Post Traumatic	88	F	20	Sepsis

Revision Cases

Patient	Secondary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Aseptic Loosening	81	M	5	Adult Respiratory Distress Syndrome
2	Post Traumatic	87	M	1	Hemorrhage
3	Aseptic Loosening	85	F	9	Myocardial Infarction
4	Aseptic Loosening	73	M	0	Myocardial Infarction
5	Aseptic Loosening	77	M	2	Myocardial Infarction

**Patient underwent bilateral procedures within 30 day period.*
***Patient underwent abdominal surgery postop within 30 day period.*

TABLE 2
Total 30 Day Postoperative Deaths Including In-House Deaths:

Primary Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Bone Mets	56	F	20	Pulmonary Embolism
2	Primary Osteoarthritis	74	M	12	Pulmonary Embolism
3	Primary Osteoarthritis	72	F	3	Congenital Heart Failure
4	Primary Osteoarthritis	75	F	0	Myocardial Infarction
5	Primary Osteoarthritis	75	M	2	Myocardial Infarction
6	Primary Osteoarthritis	80	M	18	Pulmonary Embolism
7	Primary Osteoarthritis	77	F	15	Pulmonary Embolism
8	Primary Osteoarthritis	77	M	2	Myocardial Infarction
9	Post Traumatic	88	F	5	Myocardial Infarction
10	Other	87	F	4	Myocardial Infarction
11	Secondary Osteoarthritis	58	M	12	Pulmonary Embolism
12*	Secondary Osteoarthritis	51	F	28	Pulmonary Embolism
13	Malignancy	76	M	16	Arrhythmia
14	Primary Osteoarthritis	80	F	7	Respiratory Arrest
15	Primary Osteoarthritis	81	M	15	Pulmonary Embolism
16*	Primary Osteoarthritis	73	M	14	Pulmonary Embolism
17	Primary Osteoarthritis	78	M	3	Myocardial Infarction
18	Primary Osteoarthritis	66	M	14	Pulmonary Embolism
19	Primary Osteoarthritis	80	F	27	Congenital Heart Failure
20**	Primary Osteoarthritis	85	M	21	Cardiac Failure
21	Post Traumatic	71	F	18	Myocardial Infarction
22	Post Traumatic	88	F	20	Sepsis

Revision Cases

Patient	Secondary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Aseptic Loosening	81	M	5	Adult Respiratory Distress Syndrome
2	Post Traumatic	87	M	1	Hemorrhage
3	Aseptic Loosening	85	F	9	Myocardial Infarction
4	Aseptic Loosening	73	M	0	Myocardial Infarction
5	Aseptic Loosening	77	M	2	Myocardial Infarction

**Patient underwent bilateral procedures within 30 day period.*
***Patient underwent abdominal surgery postop within 30 day period.*

TABLE 3
Total 31-60 Day Postoperative Deaths:

Primary Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Bone Mets	61	F	56	Lung Cancer
2	Post Traumatic	72	M	35	Pneumonia
3	Post Traumatic	63	M	34	Pulmonary Embolism
4	Post Traumatic	78	F	39	Pneumonia
5	Primary Osteoarthritis	73	M	35	Pneumonia
6*	Primary Osteoarthritis	80	M	39	Sepsis
7	Primary Osteoarthritis	65	M	39	Myocardial Infarction
8	Primary Osteoarthritis	68	M	58	Suicide
9	Primary Osteoarthritis	78	M	37	Multifactorial
10	Primary Osteoarthritis	80	M	33	Myocardial Infarction
11	Primary Osteoarthritis	78	F	38	Myocardial Infarction
12	Primary Osteoarthritis	79	F	40	Pneumonia
13	Primary Osteoarthritis	76	M	51	Pulmonary Embolism
14*	Rheumatoid Arthritis	70	F	51	Pneumonia

Revision Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Dislocation	80	M	52	Pneumonia

**Patient underwent bilateral procedures within 30 day period.*

TABLE 4
Total 61-90 Day Postoperative Deaths:

Primary Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Post Traumatic	70	M	71	Renal Failure
2	Primary Osteoarthritis	74	M	83	Myocardial Infarction
3	Bone Mets	79	M	74	Myocardial Infarction
4	Primary Osteoarthritis	87	M	77	Multifactorial
5	Primary Osteoarthritis	78	M	74	Pulmonary Embolism
6	Septic Hip	94	F	73	Congenital Heart Failure

Revision Cases

Patient	Primary Diagnosis	Age at Surgery	Sex	Death on Postop Day	Cause of Death
1	Aseptic Loosening	73	M	74	Pulmonary Embolism

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CLOSED LOCKED INTRAMEDULLARY NAILING OF FEMORAL SHAFT FRACTURES IN THE ELDERLY

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ABSTRACT

A review was performed of all patients over the age of 60 years who were treated with a locked intramedullary nail for a femoral shaft fracture. There were 15 patients with 16 femoral shaft fractures. Four patients died perioperatively. Of the surviving 11 patients with 12 fractures, union occurred in 100 percent. Knee range of motion was greater than 100 degrees in 11 of the 12 knees. Nine of the 11 patients returned to their preoperative level of ambulation.

Intramedullary nailing of femoral shaft fractures in patients over the age of 60 years is an effective method of treatment. The mortality rate in elderly patients who sustain this injury is comparable to that seen after a femoral neck fracture in this age group.

INTRODUCTION

Intramedullary nailing has developed into a common method of treatment for femoral shaft fractures. Although a number of large studies have reported on the results of intramedullary nailing of femoral shaft fractures, few reports have concentrated on this form of treatment in the elderly population. Femoral shaft fractures are most commonly the result of high energy trauma in the young adult population, and therefore, little is known of the expected functional outcome after a femoral shaft fracture in the elderly. As our population ages, the incidence of femoral shaft fractures in older patients is likely to increase. Knowledge of the results of treatment in this age group is therefore important to the orthopaedic surgeon.

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Most of the literature on femur shaft fracture is predominantly in young patients. Most of the literature on elderly patients with femur fractures involve proximal femur of distal femur fractures and not femoral shaft fractures. Yet it is clear that femur shaft fractures do occur in the elderly and their outcome may very well be different from young patients with femur shaft fractures and different from elderly patients with proximal or distal femur fractures.

In order to better understand the problems associated with femoral shaft fractures in the elderly and to analyze their treatment with intramedullary nailing, we reviewed the results of all patients over the age of 60 years treated in this manner at the University of New Mexico Hospital for six consecutive years.

MATERIALS AND METHODS

A review of the medical records and radiographs of all patients over the age of 60 years who had a femoral shaft fracture treated with intramedullary nailing for six consecutive years was performed. Patients were excluded from the study if they had pathological fractures or a fracture about a prosthesis or implant.

Particular attention was given to survival rate, healing rate, the amount of knee motion, and the level of ambulation which was attained.

There were 15 patients with 16 femoral shaft fractures. This represented approximately 5% of the femoral shaft fractures treated with intramedullary nails during this same time period. The minimum length of follow up was six months (range six months to five years).

There were eight females and eight males. The average patient age was 72 years (range 62 years to 98 years). The mechanism of injury was a fall in seven, a motor vehicle accident in four, a pedestrian vs. motor vehicle in three, and a crush injury in one.

The fracture location was in the proximal shaft in eight, the middle shaft in seven, and the distal shaft in one. The amount of comminution was graded by the Winquist classification. Grade I comminution was present in six, grade II in two, grade III in six and grade IV in two fractures. The Singh index was used to give a determination of relative osteopenia. Grade VI bone trabeculae was present in zero, grade V in two, grade IV

in five, grade III in five, grade II in three, and grade I in zero patients.

The injury severity score (ISS) was determined for all patients. The ISS was less than 10 in eight patients, between ten and 20 in six patients, and greater than 20 in one patient.

TECHNIQUE

Closed reamed antegrade intramedullary nails were placed in all of the patients. The surgery was performed between zero and nine days from the time of injury.

General anesthesia was performed on all patients. A tibial skeletal traction pin was utilized, and the patient was then positioned on the fracture table. The supine position was used in 11 patients and the lateral position in four.

The skin incision began at the tip of the greater trochanter and was extended proximally. An awl was used to enter the intramedullary canal at the piriformis fossa, and reaming was performed over a guide rod which had been placed across the fracture site under fluoroscopic control. No fracture required an open reduction.

The diameter of the nail was 12 mm in one, 13 mm in three, 14 mm in eight, 15 mm in four. All patients had locking nails placed.

RESULTS

Four patients died in the perioperative period. One death occurred one month after the date of injury in a 72-year-old male with an injury severity score of 45 who had sustained multiple trauma, including a closed head injury after being struck by a car. One death occurred from cardiac failure in an 84-year-old woman three days after surgery. She had sustained a fracture from a fall and had a history of diabetes mellitus and coronary artery disease. One death occurred 11 days after surgery in a 68-year-old woman who had been struck by a car and who had an injury severity of 18. The cause of death is presumed to be from pulmonary embolism. The fourth death occurred from cardiac failure two weeks after surgery in a 98-year-old female who had sustained a fracture from a fall. This patient was a nonambulatory nursing home patient with a history of dementia and congestive heart failure.

Of the surviving eleven patients with twelve fractures, union occurred in 100 percent. No patient required reoperation to obtain union. There were no failures of the fixation, and fracture alignment was maintained in each of these patients.

The range of motion of the knee was 100 degrees or more in eleven of the twelve knees. One patient had only 90 degrees of knee motion present at three years follow up.

Before injury, all of the eleven surviving patients were independent community ambulators. After healing, nine patients returned to this level of ambulation, one patient required a cane, and one patient became nonambulatory secondary to a below knee amputation resulting from an open tibia fracture.

DISCUSSION

Femoral shaft fractures are usually the result of high velocity trauma and are more common in the younger population[6]. As our population ages, the incidence of femoral shaft fractures in the elderly is likely to increase.

There are few reports about this injury in elderly patients. In studies performed prior to the popularization of intramedullary nails, both Dencker and Hubbard were unable to show any benefit of operative fixation over traction in the treatment of femoral shaft fractures in the elderly[7, 8]. More recently, Moran et al reviewed the results of 24 patients who were over the age of 60 treated with an intramedullary nail[12]. They found this method of treatment to be effective in managing femoral shaft fractures, yet there was a 54 percent perioperative complication rate.

Local and systemic problems may compromise the results of fracture treatment in the elderly leading to increased morbidity and mortality. This has been well documented in the treatment of femoral neck fractures[2, 3, 4,5,9,11,16]. In addition, advanced age has a negative influence on the survival of trauma victims with similar injury severity scores[1, 13].

In our series, there was a 27 percent (4/15) mortality rate. Moran, et al reported a 17 percent mortality rate in their series of 24 elderly patients with femoral shaft fractures[12]. Most of the patients in that series had sustained their fracture from a simple fall. This rate of mortality is compatible with the rate reported in the elderly population who sustain a femoral neck fracture from a fall, but it is much higher than the rate reported in the younger patients with a femoral shaft fracture. In a prospective study by Bone et al on the results of operative stabilization of femoral fractures in patients less than 65 years old, only three deaths occurred in 83 patients who had an injury severity score of greater than 18, and no deaths occurred in 95 patients who had an injury severity score of less than 18[3]. Only one of the four patients who died in our series had a high injury severity score.

After femoral shaft fractures, (Jenkins et al and Brumback et al) reported loss of fracture fixation in elderly patients with osteopenia treated with intramedullary nails[4, 5, 10, 14]. While this remains a concern, in our study no patient experienced a failure of fixation or loss of reduction in spite of the number of individuals with

comminuted fractures and osteopenic bone. The use of large diameter interlocking nails is felt to have been beneficial in this respect. Four of the fractures in our series required a 15 mm diameter nail due to a relatively wide intramedullary canal, and previous studies have reported that elderly patients frequently benefit from a larger diameter nail.

The rate of union and the return of knee motion were similar in our series of elderly patients as has been reported in younger patients. In addition, the majority of our patients were able to return to their preoperative ambulatory status.

Few patients over the age of 60 with a femoral shaft fracture have been treated by methods other than intramedullary nailing at our institution. We therefore cannot comment on how this form of treatment compares with others. Based on our review, we feel that intramedullary nailing is an effective treatment for femoral shaft fractures in the elderly. It offers many of the advantages seen in the younger population. We caution, however, that the perioperative mortality rate may be similar to that seen after a femoral neck fracture in this age group[7, 8,9,15]. We are currently reviewing comparative efficacy of retrograde versus antegrade femoral nailing in the elderly population. With current trend toward smaller nails many sets do not have large diameter nails available. We are currently reviewing data to determine optimal diameter and the incidence of problems placing regular sized (12 mm) nails in patients with large (>16 mm) medullary canals.

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HEAT GENERATION DURING ULNAR OSTEOTOMY WITH MICROSAGITTAL SAW BLADES

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ABSTRACT

Ulnar shortening osteotomy is a surgical treatment option for patients with symptomatic ulnar positive variance for a variety of reasons. Delayed healing and nonunion of the osteotomized sites have been reported and present problematic complications of this procedure. Studies have shown nonunion rate with transverse cuts ranging from 8-15%. The goal is to achieve parallel cuts, thus maximizing the contacting bony surface area for a better union rate. The senior surgeon attempted using a custom thick blade to insure parallel cuts. The concern is whether the heat generated during such a cut would contribute to non-union. It is our hypothesis that complications with ulnar shortening osteotomy using a thick blade are secondary to excess heat generation. When generated heat surpasses the threshold temperature of bone tissue, the organic matrix is irreversibly damaged and necrosis of the bony ends may occur. The present study measured the heat generation during ulnar osteotomy using different blade thicknesses. Thirty-five fresh turkey femurs, having similar size and cortical thickness of the human ulna, were used. Loading was done at three different speeds of 0.66, 1.0, and 1.5 mm/second corresponding respectively to 30, 20, and 10 seconds for the complete cut. A general linear statistical model was fitted relating temperature rise to three predictive factors: blade thickness, sensor distance, and initial bone temperature. There was a statistically significant relationship between temperature rise and all three predictor variables at the 99% confidence level. There was no statistically significant relationship between temperature rise and the number of cuts with the same blade up to 10 times. Compared with the single microsagital saw blade, the temperature rise for the double thickness blade was 14% higher and for the triple thickness blade was 23% higher. The temperature rise was inversely related to the speed of the cut. The temperature rise for the bone cut in 30 seconds was 1.5 times higher than the temperature rise when the bone was cut in 10 seconds. Complications with ulnar shortening osteotomy may be secondary to excess heat generation. A new thick saw blade design and the use of proper internal/external irrigation may overcome the problem.

INTRODUCTION

Patients with a relatively longer ulna than radius at the wrist (positive ulnar variance) are susceptible to a complex of degenerative changes due to the abnormally high load supported by the ulnar head[16]. Positive ulnar variance can be congenital, a result of radial shortening from a malunited distal radial fracture or premature physeal closure, or dynamically produced from repetitive grip pronation[3,9,17,20]. Ulnar shortening is a surgical procedure to correct length discrepancy between the ulna and radius. Osteotomy at the diaphysis and removing a section of the ulna has been shown to produce good results. Often patients return to pain-free function at previous work levels[2,4,6,10,11,13,19].

Problems with delayed healing or nonunion after osteotomy have been reported in the literature and complicate the shortening procedure. Oblique osteotomy was developed when inadequate compression of bone ends was implicated in nonunion cases. The placement of a lag screw across the osteotomy site provides a secondary means of compression in addition to the inter-

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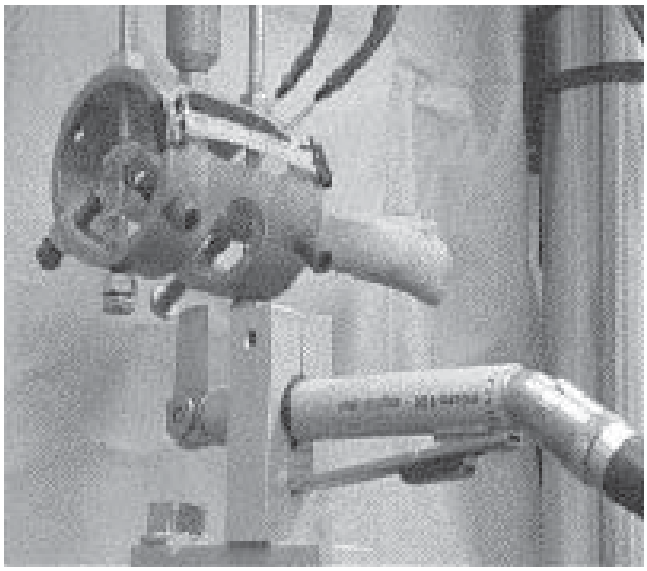

Figure 1. This picture shows the positioning of the bone and the saw blade in the testing machine. The bone specimen was held in a custom designed chuck, which was attached to the moving load frame of MTS. The oscillating saw was fixed to the floor of the MTS load frame and kept stationary. The three thermocouple probes are also shown in the picture.

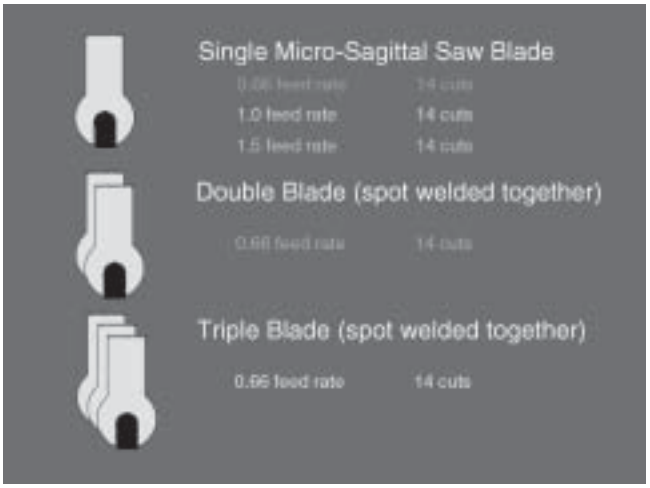

Figure 2. This figure indicates the number of cuts and the associated speed of the cut for single, double, and triple blades used in the study. Double and triple blades are simply two and three single blades spot-welded together, respectively.

nal fixation plate. Nonunion rates in oblique osteotomy patients have ranged from 0-4%[5] compared to 8-15% in patients with transverse osteotomy where placement of a lag screw is impossible. Though compression appears to be important in adequate healing, it does not address all of the challenges inherent in osteotomy surgery that increase the risk of complications.

Whether a surgeon is able to make parallel cuts during the osteotomy impacts how the bone ends will oppose each other. Even the most skilled surgeons may not be able to make these cuts perfectly using a freehand technique. The resulting surfaces may not match when they are fixed, leading to less bony contact. This may result in delayed healing or a nonunion requiring a bone graft. An unpublished trial by the senior surgeon investigator attempted to ensure parallel cuts by using one cut with a specially designed thick blade. The technique has been proposed by Labosky and Waggy[12] who found the single cut to produce a more predictable amount of shortening than the technique of using two parallel cuts. Surprisingly, the results of the aforementioned trial were not encouraging, showing high incidence of delayed and non-union of the ulna following osteotomy and fixation.

We hypothesize that the complications associated with ulnar shortening osteotomy using the thick blade are secondary to excess heat generation. When the heat generated during osteotomy surpasses a threshold temperature, bone tissue is damaged, potentially resulting in malunion or nonunion postoperatively.

Thermal injury in bone is described as irreversible damage to the organic matrix from exposure to an elevated temperature. Early studies described the damage in terms of temperatures in which key proteins were denatured. Matthews and Hirsch found bone alkaline phosphatase was inactivated in vitro at 56˚C[15]. Bonfield and Li attributed mechanical alteration of bone after heating to 50˚C to the reorientation of collagen molecules[1].

More recent research has focused not only on maximum tolerable temperatures but also on duration of exposure at an elevated temperature. Lundskog demonstrated an exponential increase in inactivity of diaphorase in the osteocyte, dependent both on the exposure temperature and the duration of heat exposure; necrosis was initiated at 50˚C[14]. Rouiller and Majno described necrosis in the long bones of rabbits when exposed to temperatures of 55˚C for one minute[18]. Most recently, Eriksson's microscopic studies of living bone tissue have shown bone to be more sensitive to heat than previously thought. He demonstrated that a temp of 47˚C maintained for one minute severely impaired bone regeneration[7,8].

Our study seeks to quantify the temperature increase in cortical bone while cutting with microsagittal saw blades of varying thickness. We hypothesize that thicker blades will generate temperature increase above baseline ($\Delta T \approx H$ 10˚C) that would begin to cause damage to the bone matrix if occurring in vivo.

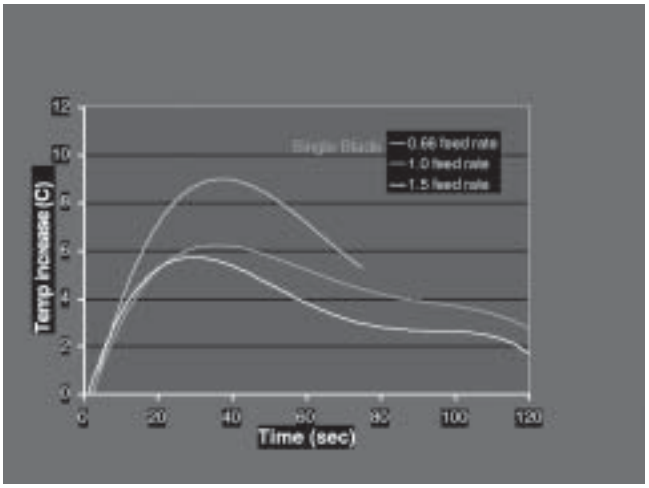

Figure 3. This figure shows the temperature rise as a function of time for three different speeds of 0.66, 1.0, and 1.5 mm/second, corresponding respectively to 30, 20, and 10 seconds for the complete cut.

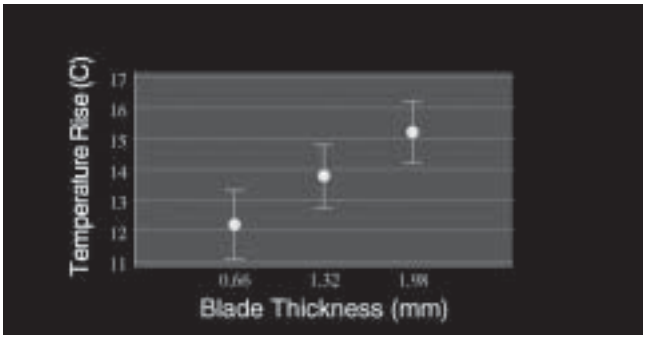

Figure 4. This figure shows the overall average temperature rise and its standard variation as a function of blade thickness.

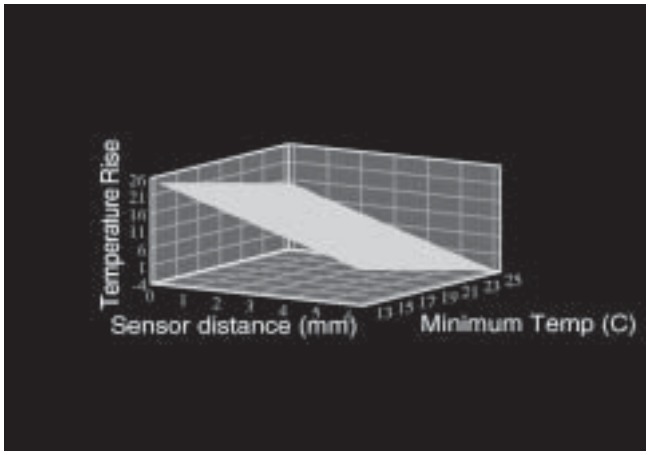

Figure 5. This figure represents a three-dimensional configuration of temperature rise as a function of both sensor distances from the cut and the initial bone temperature.

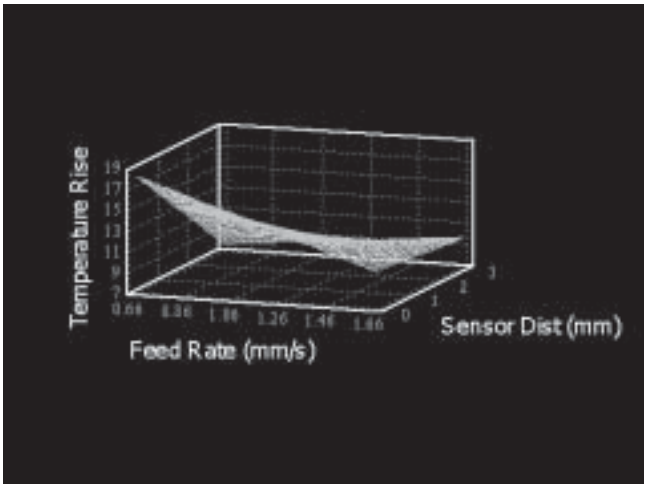

Figure 6. This figure represents a three-dimensional configuration of temperature rise as a function of both the speed of cut and the sensor distance from the cut.

METHODS

Thirty-five fresh turkey femurs, having similar size and thickness of the human ulna, were obtained from a local butcher, stripped of flesh, and frozen until needed. Hours before a testing session the bones were removed and allowed to warm to room temperature. Two cuts were made on each individual bone. Serial bone cross-section revealed an area of uniform thickness on the posterior aspect of the femurs. This area of 3-4 mm thickness was where we embedded the temperature sensors during cutting. Holes for temperature probes were drilled in the bone cortex. The holes were placed as close as possible to the intended line of cut of the saw blade (0.5-2mm). A thermoconductive paste was applied to the holes to rapidly transfer heat generated to the probes.

The bone specimen was held in a custom designed chuck mounted to an 85.8 Bionix System MTS moving load frame. The chuck and bone were lowered onto a Microaire microsagittal 2250 hand piece which inserted the blade, driven by compressed nitrogen at 95 pound per square inch (psi) (Figure 1). The blades were used in the single, double, or triple configuration (Figure 2). Double and triple blades are simply two and three single blades spot-welded together respectively. The hand piece was anchored to the floor of the MTS with a custom built vise (Figure 1). The bone was cut with the single width blade at three different feed rates, 0.66, 1.0, and 1.5 mm/second, corresponding, respectively to 30, 20, and 10 seconds for a complete cut. The double and triple width blades were evaluated only at the 0.66 mm/sec feed rate. Fourteen cuts were made for each combination of blade type and feed rate.

Temperature measurements were made through three Exacon type T-N0605 thermocouple probes mated with a computer workstation. The probes were held in

the bone cortex by a length of surgical tubing stretched to provide some forward pressure on the probes (Figure 1). The computer interface measured temperature input from the probes versus time. Recording began before cutting commenced and continued for 1-1.5 min after cut was complete to allow bone to cool back to room temperature. After each cut, calipers were used to measure the distance of each probe from the line of cut. Major and minor diameters of the femur were recorded at the cut site, as well as the number of cuts that were made with each blade to gauge any effect of blade wear on temperature.

A general linear statistical model was fitted relating temperature rise to three predictive factors: blade thickness, sensor distance from the cut, and initial bone temperature. Other variables included, number of cuts per blade, and speed of the cut. A p-value less than 0.05 was considered as statistically significant.

RESULTS

Figure 3 shows the temperature rise as a function of time for three different speeds of 0.66, 1.0, and 1.5 mm/second corresponding, respectively, to 30, 20, and 10 second for the complete cut. Figure 4 shows the average temperature rise and its standard variation as a function of blade thickness. Figure 5 represents a three-dimensional configuration of temperature rise as a function of both sensor distance from the cut and the initial bone temperature. Figure 6 represents a three-dimensional configuration of temperature rise as a function of both the speed of cut and the sensor distance from the cut. There was a statistically significant relationship between temperature rise and all three predictor variables at the 99% confidence level. Compared with the single micro-sagital saw blade, the temperature rise for the double thickness blade was 14% higher and for the triple thickness blade was 23% higher. The temperature rise was inversely related to the speed of the cut. The temperature rise for the bone cut in 30 seconds was 1.5 times higher than the temperature rise when the bone was cut in 10 seconds. There was no statistically significant relationship between temperature rise and the number of cuts with the same blade up to ten times.

DISCUSSION

For a variety of reasons, ulnar shortening osteotomy is a surgical treatment option for patients with symptomatic ulnar positive variance. The ulnar osteotomy is technically challenging using either free-hand techniques or guided systems to ensure parallel cuts. Oblique osteotomy fixed with lag screws, although more technically challenging, can improve clinical outcome. The placement of a lag screw across the osteotomy site provides a secondary means of compression in addition to the internal fixation plate. Nonunion rates in oblique osteotomy patients have ranged from 0-4%[5] compared to 8-15% in patients with transverse osteotomy where placement of a lag screw is impossible. For the case of transverse osteotomy, the goal is to achieve parallel cuts, thus maximizing the contacting bony surface area. Union rates should increase with greater bony contact.

Using thick saw blades for the osteotomy greatly reduces the technical challenge of the procedure as parallel cuts are assured each time with a free-hand technique. Unfortunately, delayed union and nonunion are a common complication with this procedure. Thermal injury to bone may occur during the osteotomy, thus increasing the risk for poor healing. Our study found the temperature rise for the double thickness blade was 14% higher than the single, and 23% higher for the triple thickness blade. We also found the speed of the cut to be a significant factor. The higher the speed, the lower the temperature rise.

Ulnar shortening osteotomy presents many challenges technically, yet provides great benefit clinically for many patients. This is a useful surgical option, and developing ways to minimize the complications of delayed union and nonunion would make it an even more appealing one as well. Limitations of the current study included the following: lack of in vivo study, initial bone temperature did not match body temperature, and the effect of irrigation on heat removal was not studied.

CONCLUSION

Complications with ulnar shortening osteotomy may be secondary to excess heat generation. A new thick saw blade design and use of proper internal/external irrigation my overcome the problem.

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INTEROBSERVER ERROR IN INTERPRETATION OF THE RADIOGRAPHS FOR DEGENERATION OF THE LUMBAR SPINE

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ABSTRACT

We examined 114 segments in 23 patients' lumbar spine plain radiographs affected by disc degeneration. Two consultant orthopaedic surgeons, two consultant radiologists, and one spine nurse practitioner made independent observations on the radiographs. MRI scan films of the corresponding 114 segments were used as a gold standard. Kappa coefficients were used to evaluate the interobserver error, and the error between the independent observers and the MRI scanning reports. The systematic differences between the observers for the diagnosis of the disc degeneration at each segment level was recorded.

There was significant interobserver error between the independent observers. The pairwise interobserver agreement ranged from fair to substantial on the plain radiograph observations [Weighted kappa coefficient, mean: 0.517 (CI=0.388-0.646)]. The pairwise interobserver agreement between the independent observers and the MRI scan ranged from fair to moderate [Weighted kappa coefficient, mean: 0.388 (CI=0.259-0.518)].

There is significant error in interpretation of the plain radiographs for the diagnosis of lumbar disc degeneration. MRI may be more accurate in the diagnosis of lumbar disc degeneration.

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INTRODUCTION

Degenerate disc disease affecting the lumbar vertebra shows up as morphological changes on plain radiograph as decreased disc height, osteophytes, Schmorl's nodes, vertebral end-plate sclerosis, and vacuum sign. These are usually late changes of a degenerate disc. Early changes of disc dehydration are not seen on a plain radiograph.

A MRI scan is the most accurate imaging modality to demonstrate gross intervertebral disc morphology[1,2]. Dehydration and change in the proteoglycan content associated with early degeneration manifests as loss of signal intensity with MRI[3,4]. Decreased disc signal intensity on MRI are correlated to histological and macroscopic degenerative changes[3,4,5]. It is difficult to separate pathologic degenerative processes from age-related changes[1,6]. Sether et al[5] showed that T2-weighted signal intensities do not decrease significantly with age if the disc is not pathologically degenerate. Their results suggested that age influences signal intensity less than pathologic degenerative process. Thus, changes in the disc signal intensity may correlate with the degree of degenerative change.

A radiologist's opinion of the lumbar spine radiograph may vary with that of the clinician due to the disadvantage of not seeing the patient. Interobserver studies have been done to quantify the level of disagreement between the radiologists on several variables[7,8].

The aim of our study was to detect the disagreement between a General Orthopaedic Surgeon, a Spine Surgeon, a Spine Nurse Practitioner and Radiologists in the diagnosis of degenerative disc disease on plain radiographs of the lumbar spine. Unlike other studies[7,8] we considered only one pathology, i.e. disc degeneration, to quantify the interobserver error. Also MRI scanning of the lumbar spine was used as a gold standard against which the plain radiograph interpretation was measured and disagreement quantified.

METHODS

Twenty-three consecutive patients with degenerative lumbar disc disease who had an MRI scan were selected for this study. There were 14 men and 9 women of average age 43.4 yrs (range:24-58). Plain AP and Lateral radiographs of the lumbar spine were examined independently by Consultant Orthopaedic Spine Surgeon (A), Consultant Orthopaedic Surgeon (B), Consultant Spine Radiologist (C), Consultant Radiologist (D), and Spine Nurse Practitioner (E). All the participants were blinded to the patient identification parameters.

They looked for the degenerative signs of osteophytes, subchondral vertebral end-plate sclerosis, Schmorl's nodes, disc space height and vacuum phenomena (Figure 1). 116 segments were studied in the 23 radiographs as one patient had lumbarization of the S1 vertebra. Visual appraisal is an accurate way to record the disc height[9]. Disc degeneration at each segment was classified as none, mild, moderate and severe. The following was the criteria for disc degeneration on plain radiographs[2,10-12]:

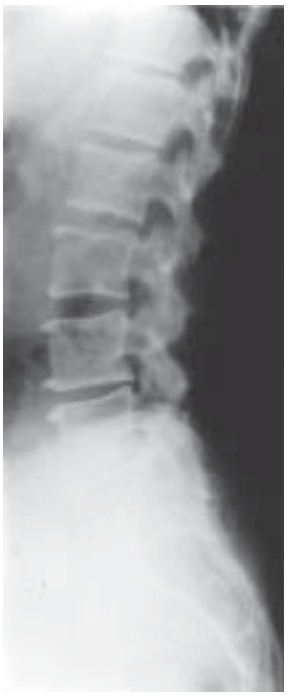

Figure 1. Changes of disc degeneration on plain radiograph.

Mild—Minimal loss of disc height; early osteophyte formation.

Moderate—Loss of disc height > 25%, but < 50%; osteophyte formation; mild end-plate sclerosis.

Severe—>50% loss of disc height; significant osteophytosis; obvious end-plate sclerosis; vacuum sign.

None—None of the above changes.

116 lumbar disc levels were examined on the MRI scan by an independent Consultant Radiologist. The visual classification system used for the disc signal intensity was bright (high-signal) appearance for normal, gray (intermediate-signal) appearance for early degenerative change, and dark (low-signal) appearance for well established degenerative change[2] (Figure 2).

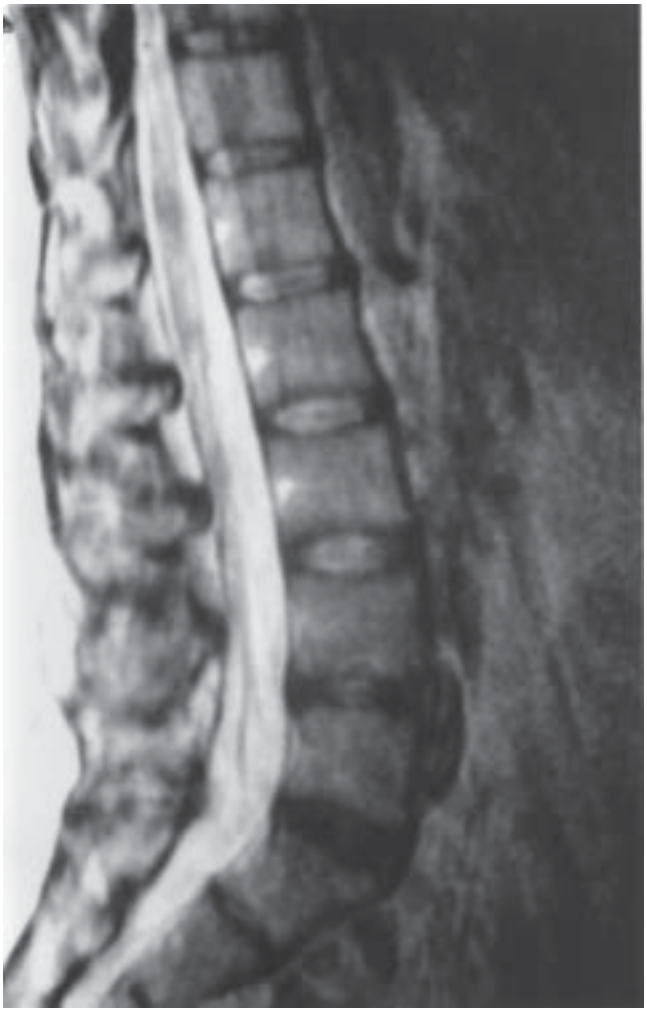

Figure 2. MRI disc signal changes from the lumbar spine.

Statistical Tests

We used the Landis and Koch's[12] interpretation of kappa: <0.00 = poor agreement, 0.00-0.20 = slight agreement, 0.21-0.40 = fair agreement, 0.41-0.60 = moderate agreement, 0.61-0.80 = substantial agreement, and 0.81-1.00 = almost perfect agreement. Weighted kappa was used rather than simple kappa and the disagreements were weighted by degree. Systematic differences in the ratings, i.e. one observer repeatedly reporting a greater or lesser number of abnormal findings compared with the other observer, were examined by the Bowker's test[14].

TABLE 1
Pairwise interobserver agreement between the 5 independent observers (doctor vs doctor)
(Weighted kappa in parentheses)

Slight (0.00-0.20)	Fair (0.21-0.40)	Moderate (0.41-0.60)	Substantial 0.61-0.80)	Almost Perfect (0.81-1.00)
	B&C (0.386)	A&B (0.436)	A&C (0.671)	
	B&E (0.351)	A&D (0.546)	A&E (0.622)	
		B&D (0.440)	C&E (0. 673)	
		C&D (0.521)		
		D&E (0.526)		

Weighted kappa coefficient WTKAP, Mean: 0.517 (CI=0.388-0.646)

TABLE 2
Pairwise interobserver agreement between the independent observers and MRI scan (a gold standard) (doctor vs MRI) (Weighted kappa in parentheses)

Slight (0.00-0.20)	Fair (0.21-0.40)	Moderate (0.41-0.60)	Substantial (0.61-0.80)	Almost Perfect (0.81-1.00)
	B (0.245)	A (0.432)		
	D (0.358)	C (0.468)		
		E (0.438)		

Weighted kappa coefficient WTKAP, Mean : 0.388 (CI= 0.259-0.518).

RESULTS

The interobserver variation for all the five independent observers is summarised in Tables 1 and 2. These tables show the pairwise interobserver agreement. The range of agreement is from slight (almost no agreement to almost perfect agreement).

The five independent observers are
A—Consultant Orthopaedic Spine Surgeon
B—Consultant General Orthopaedic Surgeon
C—Consultant Spine Radiologist
D—Consultant General Radiologist
E—Spine Nurse Practitioner

The interobserver agreement was assessed at each segment from L1-S1 for all the 23 patients. There were significant differences at each segment level when it was compared to the MRI scan. The number of observations on plain radiographs classed as none were 318; mild were 162; moderate were 79; and severe were 29. Overall interobserver agreement was calculated on 114 segments (2 segments were omitted because of < 5 observer evaluations). The overall weighted kappa coefficient, comparing the plain radiographs (four doctors and one nurse practitioner) with the MRI scanning report at each segment level was 0.245 (95% CI= lower: 0.193, and upper: 0.298). This shows that there was a fair agreement between the independent observers and the MRI scanning report.

Tables 3 and 4 show the results of the Bowker's test of symmetry.

DISCUSSION

Lumbar spondylosis and degenerative disc disease were assessed on plain radiographs of the lumbar spine by two Orthopaedic Surgeons, a Spine Nurse Practitioner, and two Radiologists. Many previous studies have shown poor agreement between observers in the interpretation of the plain radiographs of the lumbar spine and the sacroiliac joints[7,15-18]. An interpretation of radiographs depends on the observers, patients and the disease. There can be variation in the observation due to the heterogeneity in the population, diagnostic strategy and preference of the observers, and the importance, presentation and frequency of the abnormality[8]. This has implications in diagnosis and treatment.

TABLE 3
Systematic differences in the ratings between doctors using Bowker's test of symmetry

Test of symmetry		
Significant asymmetry	Sensitivity/Specificity	Insignificant asymmetry
A&B (p=0.005)	B less sensitive	A&C (p=0.239)
A&D (p=0.005)	D less sensitive	B&D (p=0.661)
A&E (p=0.038)	A less sensitive	C&E (p=0.374)
B&C (p=0.001)	B less sensitive	
B&E (p=0.001)	B less sensitive	
C&D (p=0.001)	D less sensitive	
D&E (p=0.001)	D less sensitive	

TABLE 4
Systematic differences in the ratings between doctors and the MRI scan using Bowker's test of symmetry

Test of Symmetry		
Significant asymmetry	Sensitivity /Specificity	Insignificant asymmetry
A (p=0.005)	less sensitive	None
B (p=0.001)	less sensitive	None
C (p=0.001)	less specific	None
D (p=0.001)	less sensitive	None
E (p=0.001)	less specific	None

We found variation in agreement between the observers in the interpretation of the plain radiographs at each segment level (L1- S1) for the diagnosis of degenerative disc disease. There was significant variation between the Consultant General Orthopaedic Surgeon (B), and the Consultant Spine Radiologist (C) and the Spine Nurse Practitioner (E) (Table 1). There was substantial agreement between the Consultant Spine Surgeon (A), Consultant Spine Radiologist (C), and the Spine Nurse Practitioner (E). Also the same group of observers (A, C, and E) had moderate agreement with the MRI scan which was used as a gold standard. Consultant General Orthopaedic Surgeon (B) and Consultant General Radiologist (D) had significant variation with the MRI scan. This suggests that the observers who specialize in the lower back problem of degenerative disc disease are more likely to diagnose this condition than the general orthopaedic and general radiologists.

Although the spine specialists had moderate agreement with the MRI scan in diagnosing lumbar disc disease, it falls far short of the MRI scan result. Also the overall weighted kappa coefficient comparing the observations of the plain radiographs by the 5 doctors with the MRI scan was 0.245 (mean). Thus there is only fair agreement between the doctors and the MRI scan in diagnosing lumbar disc disease.

As was noted by Espeland et al[8] when there was disagreement between the observers, one observer often diagnosed significantly more or less abnormality than the other colleague (Table 3). Consultant General Orthopaedic Surgeon (B) and Consultant General Radiologist (D) are less sensitive in diagnosing disc degeneration compared to the other observers. These two doctors and the Consultant Spine Surgeon (A) were more likely to give false negative report (less sensitive) than the MRI scans (Table 4). The Consultant Spine

Radiologist (C) and the Spine Nurse Practitioner (E) were more likely to give false positive report (less specific) than the MRI scan in the diagnosis of lumbar disc degeneration (Table 4).The systematic differences between the observers took a consistent direction at each lumbar segment, indicating that the observers had different thresholds for abnormal rating for disc degeneration. Such diagnostic threshold for ambiguous objects may depend on the observer's 'response bias', i.e. tendency to prefer one or another response category, independent of the characteristics of the object[19]. Thus the response bias and the different thresholds for actually reporting the 'minor' or clinically insignificant findings that were observed, may be the factors that have contributed to the systematic differences found in our study. There was significant systematic difference between all the observers and the MRI scan, indicating that the scan is more accurate in diagnosing disc disease in the lumbar spine. It is difficult to identify specific reasons for different diagnostic thresholds between the observers, although eliminating this could improve the diagnostic capability of the doctors[8,20].

In conclusion, we found in our study that there is wide variation in diagnosing lumbar disc disease between the Orthopaedic Surgeons and the Radiologists at our institution. There is also systematic difference in the interpretation between all observers. These variations and differences are particularly significant on comparing it to the MRI scan. The General Orthopaedic Surgeon, the General Radiologist, and the Spine Surgeon were diagnosing less degenerate discs. The Spine Radiologist, and the Spine Nurse Practitioner were diagnosing more degenerate discs. Due to this amount of inaccuracy, it is risky to comment on degenerate disc disease on a plain radiograph alone. Therefore MRI scan should only be used to comment on the disc diseases of the low back, as plain radiographs are unreliable.

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RATIONALE FOR TREATMENT OF HIP ABDUCTOR PAIN SYNDROME

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ABSTRACT

Patients with lower back or buttock pain that radiates into the posterior or lateral leg are often referred to physical therapy with a diagnosis of sciatica. Often the physical exam does not reveal neurologic findings indicative of radiculopathy. Instead, there is hip abductor muscle pain and weakness. This syndrome involves muscle imbalances that result in overuse strain of the gluteus medius and gluteus minimus muscles, myofascial trigger points, and trochanteric bursitis. This paper describes hip abductor pain syndrome and provides a rationale for the diagnosis and treatment.

INTRODUCTION

Low back pain is widely recognized as a common reason for health care visits. However, frequently the main complaint is not back pain per se, but "hip" or buttock pain, often extending to the lateral thigh or calf. Patients with these complaints are commonly referred to physical therapy with a diagnosis of "sciatica," which connotes nerve root irritation or pressure causing pain in the distribution of the sciatic nerve.[16] However, when the physical exam for this type of presentation reveals no neurologic deficits or neural tension signs, perhaps the more appropriate terminology would be "pseudosciatica." This paper provides a rationale and guide for explaining diagnostic conclusions, as well as symptom control for patients with this type of presentation. Rationale and treatment recommendations are based on physical exam and basic neuromuscular principles relating to problems with muscle length, strength, and motor recruitment. These patients typically complain of predominant hip and buttock pain, which worsens with prolonged standing or sitting, long bouts of walking, and with lying on the involved side. Physical exam for suspected spine problems usually focuses on lumbar spine mechanical findings, neurologic findings, and the absence or presence of tension signs. It is important to be thorough in this examination as radiculopathy involves a much different diagnostic and treatment process than referred pain. Patient history as well as the physical exam can often help to rule out serious pathology (infection, tumor, neurologic deficit, etc). However, when the neurologic exam is negative, further examination is required. There is often palpation tenderness in the gluteal muscles lateral to the posterior superior iliac spine (PSIS) and/or out toward the trochanter. In fact, studies indicate that as many as 20% of patients seen in a spine specialty clinic actually have trochanteric bursitis.[21] Manual muscle testing usually reveals hip abduction using tensor fascia lata (TFL) is normal. However, testing hip abduction with extension using the posterior gluteus medius (PGM) is very weak, testing no more than 3/5. It may be painful as well.

RATIONALE

Muscle performance is a critical aspect of movement that can influence tissue stress.[14] A commonly observed muscle imbalance present in those with posterior pelvic pain involves abnormal length, strength and motor recruitment of the PGM and TFL. The posterior portion of the gluteus medius muscle performs hip abduction with extension as well as some lateral rotation.[11] Often, a motor pattern fault involving habitual preference of the tensor fascia lata (TFL) in performing hip abduction movements can result in posterior gluteus medius disuse weakness and atrophy.[19,23] Specifically, this muscle imbalance presents as a lengthened/weakened PGM and a shortened/strong TFL, with preferred recruitment of TFL over PGM for hip abduction. For many, this imbalance may exist without any symptoms. However, when the PGM is overloaded/overused a strain may occur. This can be due to several factors involving overuse. Injury may occur by either high magnitude stress over a brief time, low magnitude stress over a long time, or moderate stress repeated many times.[14] For instance, weight gain greatly increases the demand on the PGM during gait and standing, which

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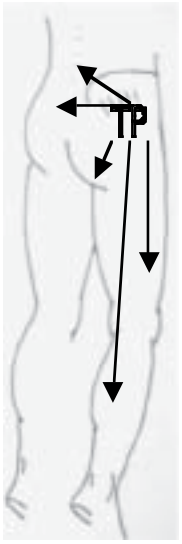

Figure 1. Trigger points (TP) with pain referral patterns (arrows) into the low back, sacrum, buttock, and leg that mimic S1 and L5 radiculopathy.

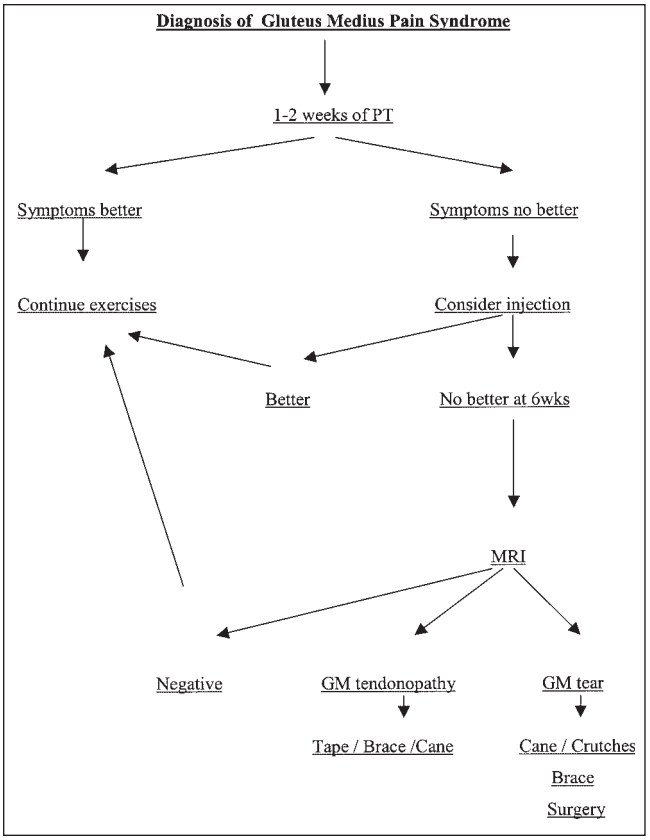

Figure 2. Algorithm for treatment.

may explain the prevalence of this problem in pregnant women.[6,17] In fact, one study indicated posterior pelvic pain to be four times more prevalent than lumbar pain in pregnant women.[17] Postural habits such as standing on one leg with the pelvis dipped to the other side or side lying with the top leg flexed and adducted over the other leg can result in adaptive lengthening of the PGM.[19] A Trendelenburg gait pattern results in both adaptive lengthening of the PGM and trochanteric bursal irritation.

Muscle strain injury is painful and is usually the predominant symptom. Persistent muscle guarding due to injury may cause relative ischemia due to muscle tension.[1,2] The "vicious cycle" of pain, increased muscle tension, decreased capillary fill, lactic acid build up, and pain is often self-perpetuating. This is thought to be the mechanism for myofascial "trigger points" and pain referral patterns.[22] The pain referral patterns for the gluteal muscles are well mapped out by Travell and others (Figure 1).[7,9,10]

Sometimes the bursal irritation due to the laxity caused by the PGM length and strength problems is the most prominent. Palpation in the bursal area is exquisitely painful. Weakness of PGM resulting in a Trendelenburg sign may also contribute to lumbar pain due to abnormal segmental movement of the lumbar spine if the pelvis is not stable during gait or standing. The lumbar facets can become irritable due to this abnormal movement repeated thousands of times a day.[13] There is more wear and tear to the intervertebral discs as well.

In summary, the physical exam for this specific entity involves manual muscle testing for the PGM vs the TFL[11], palpation of the trochanteric bursae and gluteal muscles, and checking for a Trendelenburg sign.[18] The Trendelenburg sign is the most sensitive physical exam finding for PGM tendonitis or tear.[3] An AP film of the pelvis may be useful to rule out hip dysplasia, DJD, stress fracture, calcific bursitis, or osteonecrosis.

TREATMENT

We propose a treatment algorithm based on the above understanding of this pathology (Figure 2). Treatment for muscular pain is with the use of cold packs for 20 minutes several times a day. The cold pack should reach out to the trochanter. Other soft tissue modalities may also be beneficial, such as ultrasound or iontophoresis. Strengthening exercises are directed specifically to the PGM to decrease the influence of the TFL. The exercises need to be graduated according to the level of strength assessed in the physical exam. Strengthening is done in the shortened position to facilitate normal length. They are done at least twice daily for 4 weeks. It takes at least 4 weeks to actually add sarcomeres to atrophied muscle.[15]

Recommendations are given for altering activities of daily living to decrease stretching of the muscle. Specifically, sleeping with pillows between the knees when

in side lying, not sitting with the legs crossed, standing with weight evenly distributed to both sides and the pelvis level. Recheck visits are at 1 or 2 weeks to assess improvement and to progress exercises as necessary. At 2 weeks, 20% of muscle strength change is attributed to contractile capacity and 80% to neural training.[15] In the 2-4 week time frame, bursal injection or trigger point injections may be considered to facilitate the exercise program. If the muscle strain is severe or too painful, the use of a cane is suggested.[19] Taping of the area to lessen forces during weight bearing could also be used.[14] If, at 4-6 weeks, there is no improvement in strength assessment, an MRI would be suggested. This would be to determine tendonopathy vs. tendon rupture.[12] A recent study reported the MRI findings of 24 people with this syndrome; nine demonstrated gluteus medius tendonitis, five had a tear, six had both evidence of tear and tendonitis, 3 were normal, and one showed avascular necrosis.[3] If a tendon rupture is found, treatment could involve surgical repair[4,8] and/or bracing in 0˚ of hip flexion and slight abduction with a hip-spica brace. Tendonitis would continue to be treated in a conservative manner.

SUMMARY

Evidence indicates that an important factor in conservative management involves providing accurate information to patients regarding an explanation of their problems and recommendations on methods of symptom control as well as limiting recurrence. Failure to receive an explanation of the problem results in patient dissatisfaction, increased use of diagnostic tests, and patients seeking treatment from other professionals.[5]

Often, a reasonable explanation of the probable causes of their pain and expected recovery will relieve anxiety and improve compliance with a home exercise program. The rationale for treatment of this syndrome involves mostly myofascial strain related to muscle imbalance that often mimics a lumbar radiculopathy. When the physical exam is negative for radiculopathy, further physical exam may reveal this syndrome. Recognition of this syndrome may well reduce the number of lumbar MRI's ordered and speed appropriate treatment because a more accurate diagnosis was achieved. We plan further studies involving gait patterns, pain during pregnancy, and acute care and chronic pain prevalence as well as specific interventions as described above.

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EVIDENCE-BASED MEDICINE IN ORTHOPAEDIC SURGERY—A WAY TO THE FUTURE

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"We are in this business to serve people, and that means not only maintaining and restoring their health, but doing it without violating the budget they are willing to spend . . . The unifying principle is that when we run into a tough choice, we should try to resolve it by reference to the people we serve: what is important to them and how they weight the different consequences." David Eddy, MD, PhD.[1]

Evidence-based medicine (EBM) is a buzzword, part of a health care movement that claims to improve medical decision making, thus improving patient care[1,2,3]. The rationale for EBM is that medical literature is similar to legal evidence that is used to prove (or disprove) a belief [4]. The scientific framework of evidence-based medicine is 1) systematic reviews based on clinical trials and 2) validated outcome measurements[5] and 3) evidence is then used to guide clinical practice. How EBM will affect orthopaedic practice depends on the quality of evidence (numbers 1 and 2 above) provided by clinical research and the willingness of orthopaedists to adopt the "best evidence" into their delivery of care (number 3).

The goal of evidence-based clinical information is to provide scientific information to orthopaedists that translates into quality patient care while mindful of costs, ethics and safety. As many in the academic orthopaedic community have pointed out, there is a history of resistance to performing well-designed clinical trials of orthopaedic procedures[6,7,8,9]. Past neglect now leaves us with few published orthopaedic clinical trials, and the consequences are that orthopaedics lags behind many fields in the "raw material" for evidence based medicine. The next few years will be crucial for the production and application of orthopaedic evidence and momentum is gathering in our publications to provide more of that is needed as evidence.

For example, there are no clinical trials with modern outcome measures compare operative versus non-operative treatment of large joint arthritis. This seems non-sensical to most orthopaedists. The principles of good orthopaedic practice are 1) first try non-operative treatment and 2) perform surgery when non-operative treatment fails. With this paradigm, there can be no valid comparison of operative and non-operative treatments because they are applied to mutually exclusive groups (though paradoxically the same patients!). What is needed in order to provide evidence is clinical trials that prove the efficacy of a surgical procedure versus other common treatments. And, in some instances there should be comparison of operative and non-operative care. That is how evidence will be assembled that answers patients' questions and provides the highest quality of orthopaedic care.

With the information explosion, patients have access to enormous amounts of information regarding diagnoses and treatments. I argue that the burden shifted to orthopaedic surgeons to provide evidence that they deliver the best available care to patients with musculoskeletal disorders. The case may be made that *orthopaedic surgeons should insist on evidence-based guidelines* for the highest quality, most cost-effective care for musculoskeletal patients[4,11]. The answer to the question of best orthopaedic treatment is to prove by patient-generated measurements that demonstrate how well orthopaedic surgery has improved the quality of life. In the case of fracture care, evidence may show that one treatment reduces impairment rather than provide an improvement in life quality.

Insurers and government payers currently monitor monetary cost, safety and durability (and sometimes the patient's return to work). In essence, they are holding orthopaedists fiscally accountable for professional behavior[1,5]. What orthopaedic surgeons should add to financial accountability of patient care is *patient satisfaction*. Thus the case for orthopaedic EBM is going "one better" than the insurance industry or government accountability.

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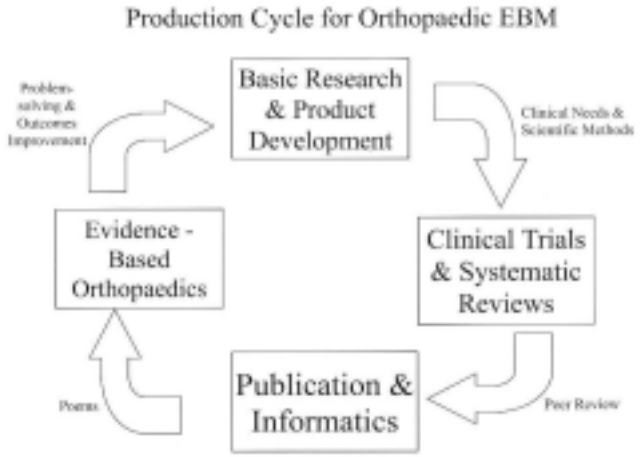

Figure 1. Production cycle for evidence-based orthopaedics

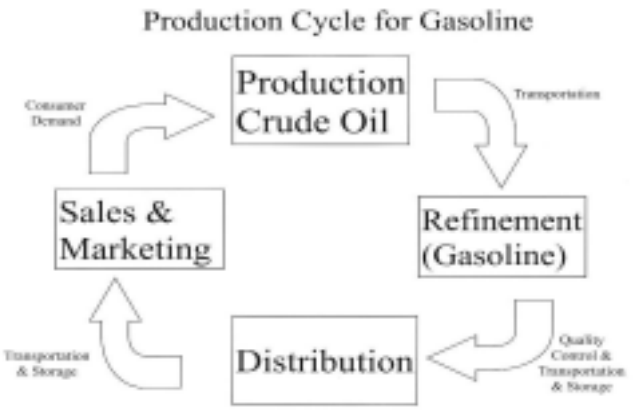

Figure 2. Production cycle for gasoline.

If patients prove that a hip replacement is better than medication and a cane, it is possible to justify expense and physical risks of surgery and the possible need for expensive revision surgery. If patients do not prove that a hip replacement is better than pills and a cane, then there is no need for further justificatio—it's a dead issue, no surgery. Once replacement arthroplasty for hip arthritis is justified, the same process justifies the application of improvements (changes) in technology. Thus if ceramic surface components are to prove more effective than the conventional metal-polyethylene interface, there must be patient oriented evidence to support that claim.

In a manufacturing analogy, EBM delivers information that is a high quality, cost-effective product to orthopaedic surgeon consumers. Basic research and product development produces information useful to clinical research that in-turn produce information that is packaged and transported as manuscripts and presentations at clinical conferences. Manuscripts that provide patient oriented evidence that matters (POEMs) are consumed by the orthopaedic surgeon and put into practice[12]. As defined, POEM is "patient-oriented evidence with which we can evaluate the efficacy of interventions in terms of results that patients care about and that we as clinicians want for our patients"[13].

Feedback from practicing orthopaedists will generate the next round of basic research and product development in order to solve problems that arise from the first cycle of production (Figure 1). This is similar to the figure (Figure 2) illustrating production and marketing of gasoline from crude oil: raw material, refining, distribution, sales & marketing giving rise to the demand of more product. As a practical matter, EBM is the application (sales & marketing) of POEMs that are derived from valid clinical trials of orthopaedic procedures as published in the appropriate peer-reviewed journal (or presented at a peer-reviewed meeting). Clinical trials are the refinery that takes basic research or applied technology and generates useful information. The peer-reviewed journals (e.g., JBJS American- Evidence Based Orthopaedics), peer-reviewed Internet bulletin boards (ScHARR- www.shef.ac.uk/~scharr/ir/netting.html) serve as the distribution network for the results of clinical trials and systematic reviews (Cochrane Database of Systemic Reviews, EBM Best Evidence).

Several recent clinical trials in the English language orthopedic literature illustrate a trend toward providing the type of evidence needed to keep orthopaedics in the mainstream of health care[3,14,15,16,17,18]. As this trend becomes more expansive and inclusive of musculoskeletal disorders, orthopaedic surgeons will more readily implement improvements and advancements in surgical treatment. This empowerment comes from the power of the people- patients want safe and effective treatments, at reasonable cost. Clinical trials and comparison studies are researches done on humans for the good of other people. When evidence from clinical trials pass the filter of scientific scrutiny and become POEM, it will benefit people when orthopaedists change their professional behavior by implementing the POEM.

Another example, if a relatively safe oral antibiotic given for a few days is as effective in reducing bone infections as a week of combination intravenous antimicrobials for open long bone fractures, this is worthy of implementation in practice. On the other hand, if ceramic hip prostheses are proven to produce far less

TABLE 1
Characteristics of a Clinical Trial

Posing a good question	What patients are available for study? What treatments should be compared? What is the comparison of interest? What outcome shall be measured? Who will benefit from the results?
Deciding if the question is answerable/feasible	Can enough patients be enrolled? Statistical plan – likelihood of reaching significance? Affordable/fundable? Adequate surgeons with expertise? Are the surgeons able to provide different treatments without technical bias? Means of randomization – many cases are subverted by investigators' bias.
Ethical	Patients are free to decline or withdraw without coercion. Controls must be ethically decided (non-treatment is rarely ethical). Independent quality control/monitoring. Will the study give the best answer that will benefit patients? Financial incentives for patient enrollment, investigator? Full disclosure to insurers that won't pay for services as "part of a study"? Surgeons must have equipoise – i.e. no prejudice about treatment efficacy.

particulate debris than conventional total hip, it still remains to be proven that painful loosening is diminished when using a ceramic device. What makes sense inferentially needs to be proven clinically, that is the reason for evidence in orthopaedic practice. Evidence based orthopaedics does not replace common sense and clinical judgment; it should reinforce good orthopaedic practice. And, orthopaedic surgeons still need to apply personal experience, a sense of risk, and a cost-benefit ratio to the individual patient. Because there is a guideline that suggests a treatment, does not mean that *all* patients get the *same* treatment.

The techniques and devices that are being developed for use in the future should prove their safety and efficacy in the court of best evidence. Similarly, evidence for pharmaceuticals commonly prescribed by orthopaedic surgeons should be held to the same standard as surgical technology. What evidence supports oral chondroitin sulfate/glucosamine compared with NSAIDs for painful osteoarthritis of the knee? Is there evidence for prescribing Cox-2 inhibitors versus less expensive anti-inflammatory medication?

As a profession, orthopaedic surgeons will learn how orthopaedic practice questions can be answered by valid clinical trials. Trials are expensive and take considerable time to produce results—research design; planning and execution are crucial to success. Though masking is difficult, there are several elements of randomized clinical trials that are relevant to orthopaedic surgery. Starting with a good question that can be answered by a clinical trial, the research team needs to design an ethical study that can capture enough patients to answer the question (Table 1).

Randomized clinical trials work much better for highly prevalent conditions than for rare events. It is easier to design a clinical trial for patients with low back pain and disc herniation than to determine the best surgery for adults with chondrosarcoma of the proximal humerus. Not all orthopaedic questions are answerable by clinical trials (e.g. risk factors in postoperative joint sepsis- this is a disease oriented measure) and other forms of patient oriented evidence are sometimes needed. Prospective clinical studies measuring outcomes that matter provide better evidence for decision making than disease oriented evidence[12,13].

These points are summarized, from the viewpoint of a clinical investigator by Cummings' acronym FINER: Feasible, Interesting to the investigator, Novel, Ethical and Relevant[19]. From the vantagepoint of the orthopaedic surgeon as consumer of evidence-based medicine, there is no authoritative orthopaedic resource that evaluates surgical clinical trials for accuracy and validity. But, efforts underway by the *American Journal of Bone and Joint Surgery* and orthopaedic subspecialty journals will soon produce a body of literature that will educate orthopaedic practitioners how to change with the times as good evidence is written.

This is not to say that orthopaedists have ignored good evidence in the past or refused to keep-up with improvements in patient care. What is new about EBM is the concept that orthopaedists change their practice more swiftly when compelling evidence is presented. The problem has been the lack of evidence and that is now being corrected. Orthopaedic surgeons who tried new things in the past have been "burned". With good reason, these surgeons are likely to resist moving quickly to implement a new procedure or technique. With the presence of one or two valid POEMs, evidence based orthopaedics decreases the likelihood of making bad practice decisions about using new technologies and procedures. Conversely, if a surgeon bases their decision to try a new technique based solely on safety and efficacy information (disease oriented evidence), there is greater probability that unreported problems or patient dissatisfaction will occur postoperatively. The occasional need to rely on entirely on disease oriented evidence is understandable if no POEMs exist.

As mentioned by Wright & Swiontkowski[3,20,21], the number of published clinical trials in orthopaedic journals has dramatically increased in the past decade. The nature of randomized clinical trials is they require multiple centers and several surgeons and a large infrastructure, both personnel and electronic informatics. There are many logistical problems that include finding surgeons who are competent to provide different surgical treatments without bias, truly random allocation of patients to the treatment groups, enrolling adequate numbers of patients and keeping the enrollees participating for the duration of the study. How the principal investigator and the study designers eliminate or reduce bias is critical to the clinical trial. If there are serious (fatal) flaws in design or implementation, serious issues of bias or credibility, serious problems with safety, the result will not be accepted by the practicing orthopaedic surgeon—even if published and presented.

Statistical correctness[6,7], akin to political correctness, is another issue that needs to be addressed in gaining the confidence of orthopaedists who do not have the training or inclination to understand the nuances of statistics and statistical terms. Evidence should be clearly recognized by reputable experts in the field and by local practitioners, without having an extensive knowledge of statistics. When there is no patient oriented evidence and orthopaedic practice is based upon disease oriented evidence, an orthopaedic research team should take up the challenge and design a clinical trial that measures treatment outcome in an attempt to make POEM. The creation and application of POEM is where a sea change in attitude is needed within the field of orthopaedic surgery.

As an aside, changing physician's behavior is not an easy task. A recent report by team of French epidemiologists indicated that when orthopaedists had access to a computer-based clinical decision support system, compliance increased with a validated deep vein thrombosis prophylaxis regimen[22]. And, when the computer system was removed from the hospital, surgeons reverted to their previous practice of haphazard DVT prophylaxis and the clinical outcome reflected the change in the surgeon's prescription. This type of study highlights the difficulty in changing surgeon's behavior, even when there clearly is benefit to do so.

Randomized clinical trials and evidence-based medicine for orthopaedic surgeons are not an oxymoron! Randomized clinical trials that answer orthopaedic questions, if given the chance, will provide the basis for much of the future practice of orthopaedic surgery. Orthopaedic literature *is* providing more and more evidence. The readers may not be completely familiar with the language of the investigator (e.g., statistics) but the conclusions (benefit to patients) should be clearly understandable—outcomes that are meaningful to patients. And, published orthopaedic POEM should be put into practice by orthopaedic surgeons for the benefit of their patients and to keep this process moving forward.

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OSTEOCHONDRAL REPAIR OF PRIMATE KNEE FEMORAL AND PATELLAR ARTICULAR SURFACES: IMPLICATIONS FOR PREVENTING POST-TRAUMATIC OSTEOARTHRITIS

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ABSTRACT

The risk of post-traumatic osteoarthritis following an intra-articular fracture is determined to large extent by the success or failure of osteochondral repair. To measure the efficacy of osteochondral repair in a primate and determine if osteochondral repair differs in the patella (PA) and the medial femoral condyle (FC) and if passive motion treatment affects osteochondral repair, we created 3.2 mm diameter 4.0 mm deep osteochondral defects of the articular surfaces of the PA and FC in both knees of twelve skeletally mature cynomolgus monkeys. Defects were treated with intermittent passive motion (IPM) or cast-immobilization (CI) for two weeks, followed by six weeks of *ad libitum* cage activity. We measured restoration of the articular surface, and the volume, composition, type II collagen concentration and *in situ* material properties of the repair tissue. The osteochondral repair response restored a mean of 56% of the FC and 34% of the PA articular surfaces and filled a mean of 68% of the chondral and 92% of the osseous defect volumes respectively. FC defect repair produced higher concentrations of hyaline cartilage (FC 83% vs. PA 52% in chondral defects and FC 26% vs. PA 14% in osseous defects) and type II collagen (FC 84% vs. PA 71% in chondral defects and FC 37% vs. PA 9% in osseous defects) than PA repair. IPM did not increase the volume of chondral or osseous repair tissue in PA or FC defects. In both PA and FC defects, IPM stimulated slightly greater expression of type II collagen in chondral repair tissue (IPM 81% vs. CI 74%); and, produced a higher concentration of hyaline repair tissue (IPM 62% vs. CI 42%), but IPM produced poorer restoration of PA articular surfaces (IPM 23% vs. CI 45%). Normal articular cartilage was stiffer, and had a larger Poisson's ratio and less permeability than repair cartilage. Overall CI treated repair tissue was stiffer and less permeable than IPM treated repair tissue. The stiffness, Poisson's ratio and permeability of femoral condyle cast immobilized (FC CI) treated repair tissue most closely approached the normal values. The differences in osteochondral repair between FC and PA articular surfaces suggest that the mechanical environment strongly influences the quality of articular surface repair. Decreasing the risk of post-traumatic osteoarthritis following intra-articular fractures will depend on finding methods of promoting the osteochondral repair response including modifying the intra-articular biological and mechanical environments.

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INTRODUCTION

Intra-articular osteochondral repair occurs under unique complex biological and mechanical conditions[4,6,20]. Disruption of subchondral and metaphyseal bone creates conditions similar to bone fractures, but the bone repair response extends into the chondral portion of the defect. At the same time the chondral and osseous portions of the defect are exposed to the synovial environment including the synovial fluid and the cellular and vascular reactions in the synovial membrane[4]. Joint loading and motion causes poorly understood mechanical stresses in the chondral and bony portions of the defect that do not occur with any other type of injury.

The success or failure of acute osteochondral repair is critical in determining the outcome of intra-articular

fractures, a clinical problem faced by tens of thousands of patients and surgeons each year[20]. Yet, basic research and clinical experience give only a vague outline of the osteochondral repair response and the factors that influence this response. Immediately following an acute articular surface fracture, the osseus portion of the defect and a part of the chondral defect fill with a fibrin clot. Over a period of approximately six weeks the fibrin clot is replaced with repair tissue that contains cells with the appearance of chondrocytes in the chondral part of the defect and osteoblasts in the osseous part of the defect[2,3,5,6,8,9]. In some instances the repair tissue remodels and functions well as a joint surface for decades, but in others the chondral portion of the repair tissue progressively degenerates, a process that frequently leads to the clinical syndrome of joint pain, deformity and dysfunction recognized as post-traumatic osteoarthritis. The mechanical and biological events that influence the formation and remodeling or degeneration of osteochondral repair tissue have received little attention.

Current treatment of intra-articular fractures consists of attempting to restore the alignment and position of the fragments of the fractured articular surface and stabilizing these fragments in the reduced position. Even when surgeons oppose the fracture fragments and restore the alignment and contours of the articular surface, the injured joint frequently undergoes progressive degeneration. The reasons for joint degeneration following anatomic restoration of a fractured articular surface remain poorly understood[20], but to a large degree this phenomenon is related to failure of the osteochondral repair response to restore a normal articular surface. Some intra-articular fractures cause such severe osteochondral damage that despite optimal treatment, large gaps or defects are left in the articular surface. In these gaps, the restoration of an articular surface depends on production of substantial quantities of chondral and osseous repair tissue and the remodeling of the repair tissue to form a functional articular surface. For these reasons better understanding of osteochondral healing is critical for the advancement of treatment of intra-articular fractures and decreasing the risk of post-traumatic osteoarthritis.

The limited ability of the natural osteochondral repair response to restore an articular surface has been recognized for more than 250 years[15,17,22]. Continuous passive motion treatment of one mm diameter osteochondral defects in rabbit knees has been shown to facilitate chondral repair[24], and more recently, multiple investigators have reported improved healing of acute experimental articular surface defects with growth factors, cell transplants and artificial matrices[2,3,5,6,10,11,13,14,16,18,19,21]. However these studies have not examined osteochondral healing in primates, potential differences in osteochondral healing in regions of the joint subjected to different patterns of mechanical forces, and the effects of passive motion on repair of different regions of the joint. The purpose of this study was to answer the following questions. How successfully does the natural osteochondral repair response restore an articular surface in skeletally mature primates? Does repair of patellar and femoral condylar osteochondral defects differ? Do cast and intermittent passive motion treatment of acute osteochondral defects produce different results?

MATERIALS AND METHODS

Experimental Design

We created identical cartilage defects in the central weight-bearing articular surface of the medial femoral condyle and the central articular surface of the patella in both knee joints of twelve skeletally mature cynomolgus male monkeys. All surgery was performed aseptically under inhalation anesthesia at the Ohio State Department of Veterinary Clinical Sciences. A 3.2 mm drill bit and a drill sleeve with an adjustable stop, which ensured that the depth of the hole would be 4 mm, were used to create uniform defects. Animals were given postoperative analgesia. For two weeks postoperatively, the animals were maintained in a seated position in padded restraint chairs so that one limb could be immobilized in a long leg cast in 15 degrees of flexion at the knee and the contralateral limb was subjected to IPM (one cycle/40 seconds) for 16 hours a day, seven days a week. The animals had free arm movement but were controlled from the waist down and the neck up. The casted limb was suspended from the chair with the hip flexed to 90°. The foot of the IPM limb was secured in a cloth boot that was attached to a pedal on a fly wheel. The pedal's height was adjusted to each individual animal so that the limb would go through a full range of motion in the seated position but the limb would not be over-extended or over-flexed. A bicycling motion was thus produced. The motor on the fly wheel was set at a speed that would take the limb through a full range of motion every 40 seconds. The animals were monitored constantly and their skin checked daily for problems associated with the restraint chairs. At the end of one week in the chairs, the monkeys were placed in their cages for 12 hours to allow for chair maintenance and examination of each animal. At the end of two weeks the cast was removed and the animals were allowed cage activity *ad libitum* for six weeks, before sacrifice. None of the animals developed complications of the surgery or treatment of the knee joints. The eight knees

from four randomly selected animals were used for studies of the material properties of the repair tissue and the sixteen knees from the remaining eight animals were used for histologic and biochemical studies. The knees of two normal cynomolgus monkeys were harvested for use as normal controls. Knee joints were harvested at the Ohio State University, frozen and shipped to the University of Iowa for analysis.

Articular Surface Restoration

At the University of Iowa, the articular surfaces of all 48 defects were photographed the images enlarged 40 times with maximum resolution and printed. At this magnification it was possible to identify regions of the repair tissue surface that had the same smooth white appearance as the surrounding articular cartilage and regions that had an irregular appearance. A transparent grid with 100 equally spaced points was superimposed on the images of the repair tissue surface and the number of points falling on repair tissue with the appearance of normal articular cartilage and the total number of points falling on the defect recorded. For each experimental group the percent of repair tissue with a normal appearance was determined. Differences among groups were evaluated using t-tests.

Repair Tissue Volume and Composition

To evaluate the chondral and osseus repair tissue volume and composition, 32 defect sites from eight animals were briefly decalcified and divided along the sagittal plane into two equal parts. One half of each defect was randomly selected to measure the type II collagen concentration in the chondral and osseous parts of the defect. A dissecting microscope was used to separate the chondral and osseous repair tissue in the specimens used to measure the type II collagen concentration. The osseous repair tissue was decalcified and the type II collagen concentration measured in the chondral and osseous repair tissue as previously described[12,23]. The other half of each defect was decalcified, fixed and embedded in paraffin. Beginning with the center of the defect, four full thickness sections of the defect and surrounding tissue were collected by taking the first, fifth, tenth and fifteenth section. After staining with Safranin O, images of the sections were projected with a superimposed grid with 200 equally spaced points. The total area of repair tissue relative to the total area of the original defect was determined by point counting for each section as was the area of hyaline cartilage and the area of fibrocartilage or fibrous tissue in the chondral part of the defect and the area of bone matrix, bone marrow, hyaline cartilage, fibrocartilage or fibrous tissue in the osseous part of the defect. The total areas of chondral and bone repair tissue and each repair tissue type were then calculated for each defect.

Repair Tissue Material Properties

The eight knee joints randomly selected for biomechanical testing were shipped frozen from the University of Iowa to Columbia University[1]. The joints were stored at -20˚C and underwent two freeze-thaw cycles before testing. Sixteen repair sites in the patella and the medial femoral condyle were identified. Two of the 16 defects were not tested, because they were devoid of repair cartilage. Normal, cynomolgus monkey knee joints were used as controls for the experimental specimens. Three mechanical properties of repair tissue (aggregate modulus, Poisson's ratio, and permeability), were measured as previously described[1]. After mechanical testing, the repair sites were sectioned and the thickness of the chondral repair tissue measured with a digital scale using a stereomicroscope[1].

RESULTS

Articular Surface Restoration

All 48 defect sites could be identified by differences in the appearance of the chondral repair tissue and the surrounding articular cartilage (Figures 1 and 2). The degree of restoration of the articular surface varied among animals (Table 1). The extent of filling of the articular surface defect with chondral repair tissue varied from more than 90% to less than 10% (Figures 1 and 2). The extent of articular surface restoration did not differ between femoral defects treated with CI or IPM, or between femoral and patellar defects treated with CI, but IPM treated patellar defects had significantly less restoration of the articular surface (Table 1 and Figure 3).

Repair Tissue Volume and Composition

The extent and quality of the repair response varied among animals (Figure 4), but certain characteristics of the repair response occurred consistently. Osseous repair was substantially more effective than chondral repair. The repair response filled 68% of the total volume of the chondral defects and 92% of the volume of the osseous defects (Figure 5). Repair of femoral defects produced significantly more hyaline repair tissue and contained higher concentrations of type II collagen than repair of patellar defects (Table 2 and Figure 6). Intermittent passive motion treatment did not increase the volume of osseous or chondral repair tissue in either patellar or femoral defects. Passive motion treatment did increase the type II collagen concentration in

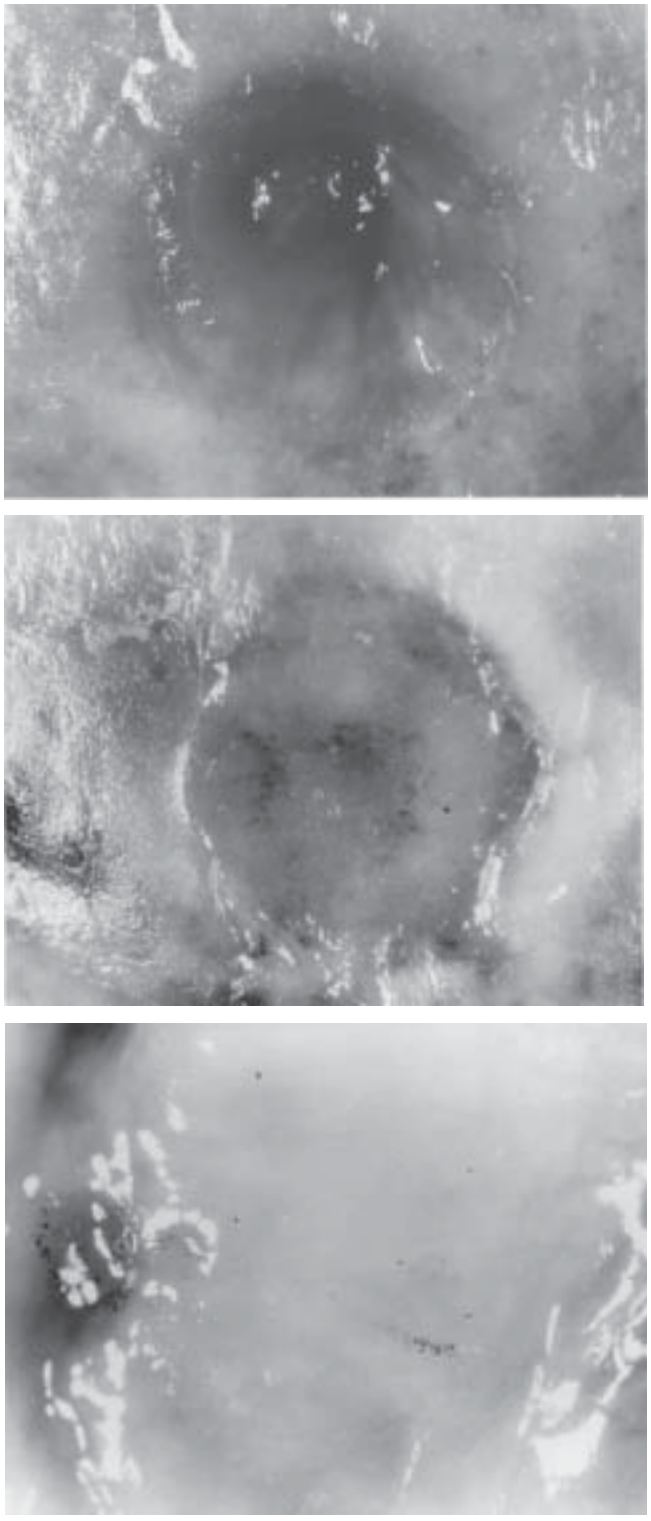

Figure 1. Photographs of the surface of femoral condylar defects eight weeks after creation of the defects showing the variability in the extent of chondral repair tissue among defects. Top: The poorest repair of a femoral condylar defect. Middle: Moderate chondral repair tissue. Bottom: The specimen with the most complete repair of the defect. The outlines of the defect are difficult to identify.

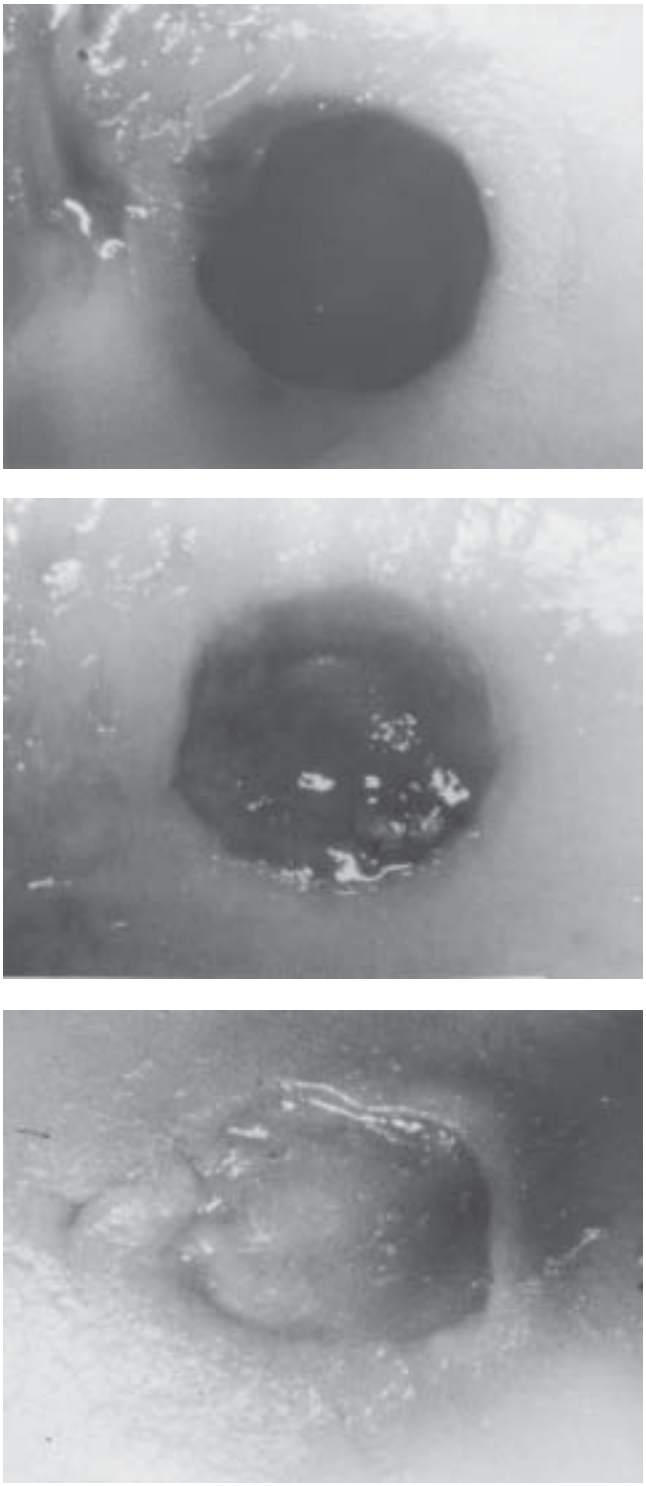

Figure 2. Photographs of the surface of patellar defects eight weeks after creation of the defects showing the variability in the extent of chondral repair tissue among defects. Top: The poorest repair of a patellar defect. Middle: Some chondral repair tissue is visible. Bottom: The specimen with the most complete repair of the defect.

TABLE 1
Articular Surface Restoration
(% of 3.2 mm diameter defect)

Treatment	Number of Animals	Location: Femoral Condyle	Location: Patella
IPM	12	53±29	23±17
Cast	12	58±22	45±26

Bars connect means that are not significantly different (p<0.01)

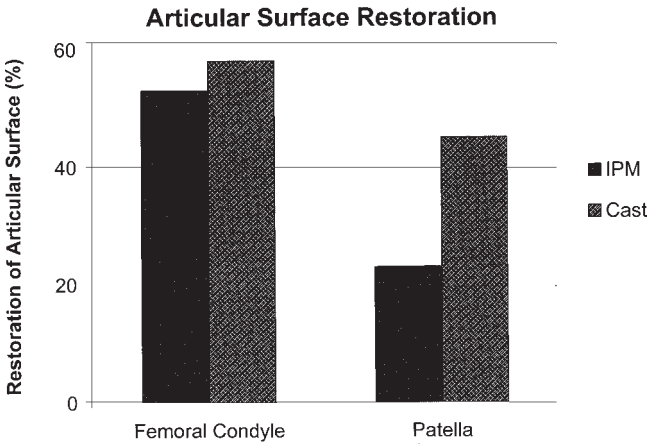

Figure 3. Histogram showing that the osteochondral repair response restored more of the articular surface in femoral defects than in patellar defects and that patellar defects treated by cast immobilization had better restoration of the articular surface than patellar defects treated by IPM.

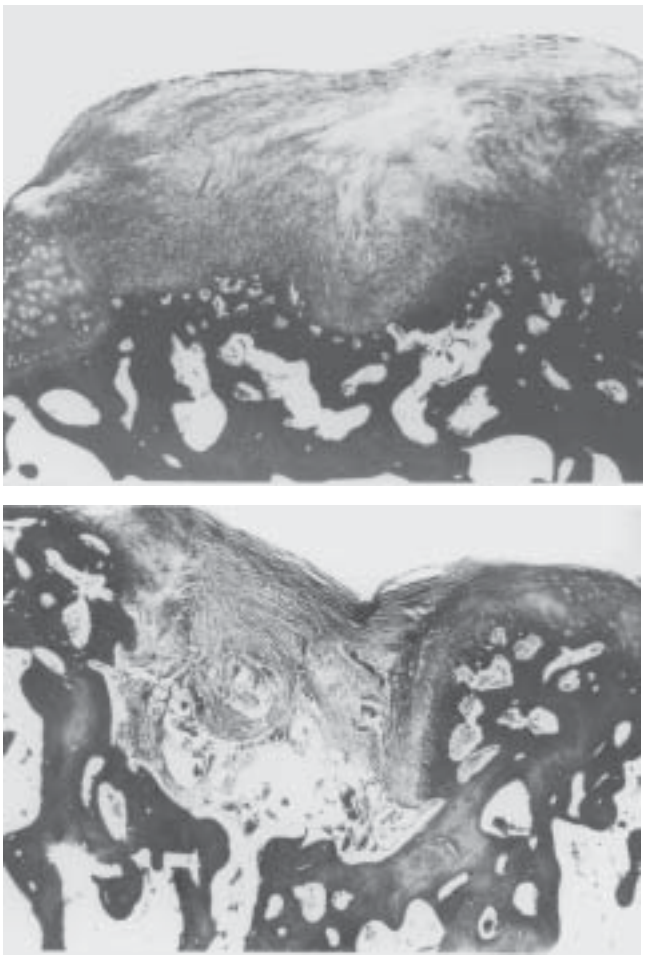

Figure 4. Photomicrographs showing the variability in osteochondral repair among animals. Top: The osseous portion of the defect is filled with new bone and chondral repair tissue projects above the normal articular surface. Bottom: The osseous portion of the defect contains fibrovascular tissue as well as bone and the chondral portion of the defect contains primarily fibrous tissue.

femoral and patellar chondral defects. In patellar defects, passive motion treatment produced a higher concentration of hyaline repair tissue.

Repair Tissue Material Properties

The material properties of repair tissue differed from those of normal articular cartilage (Figure 7)[1]. The normal articular surface (Table 3) was four times stiffer than the osteochondral repair tissue ($p<0.0001$). Similarly, the Poisson's ratio of the repair tissue was about half that of the normal articular surface ($p<0.01$) and the repair tissue was more permeable ($p<0.01$).

Passive motion and cast treated osteochondral repair tissue differed in material properties. The repair tissue had a higher compressive modulus ($p<0.10$) in joints treated with casts (HA=0.244±0.138 MPa) than with passive motion (HA=0.148±0.053 MPa). The aggregate modulus of patellar repair tissue was significantly higher in the cast treated group ($p<0.05$). Similarly, repair tissue in the femoral condyle was more permeable in the passive motion treated defects ($p<0.10$).

Chondral Repair Tissue Thickness

At eight weeks following creation of the osteochondral defects the mean thickness of the chondral repair tissue was 2.16 mm, compared with 0.72 mm for normal femoral articular cartilage and 0.71 mm for the cartilage adjacent to the defects (Table 3). The original

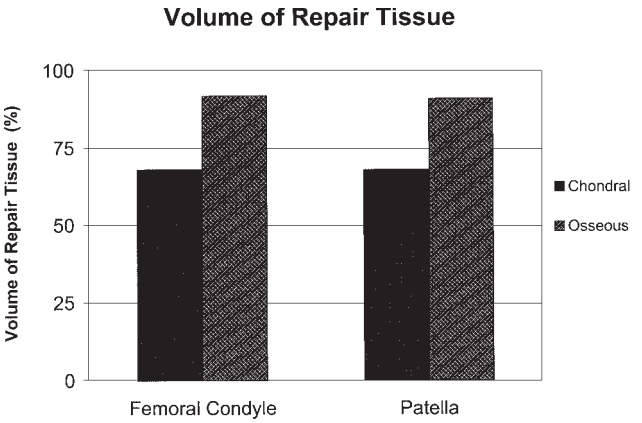

Figure 5. Histogram showing that repair tissue filled more than 90% of the osseous defect volume but only about 68% of the chondral defect volume.

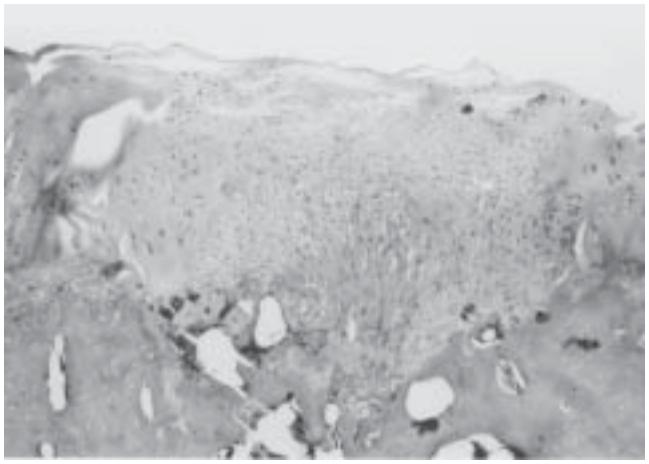

Figure 6. Photomicrograph showing chondral repair tissue with a hyaline appearance.

TABLE 2
Volume and Composition of Repair Tissue

Treatment and Location	Number of Animals	Cartilage Repair Tissue				Bone Repair Tissue					
		Total Volume (%)	Hyaline (%)	Fibrous (%)	Type II Collagen (%)	Total Volume (%)	Bone (%)	Marrow (%)	Hyaline (%)	Fibrous (%)	Type II Collagen (%)
IPM Patella	8	69±15	64±29	34±25	75±27	93±19	49±18	25±16	13±8	11±8	10±10
Cast Patella	8	67±22	42±37	57±39	67±20	89±17	48±17	26±19	15±16	9±9	8±5
IPM Femoral Condyle	8	65±23	83±23	17±23	87±19	92±7	40±12	24±8	26±13	9±8	35±32
Cast Femoral Condyle	8	71±19	83±19	15±20	81±19	93±17	38±16	23±19	25±16	9±9	38±33

Bars connect means that are not significantly different ($p<0.01$)

depth of the osteochondral defects was 4 mm, a 0.72 mm deep chondral defect and a 3.18 osseous defect. Thus, by eight weeks, bone had not filled the entire original bone defect and the chondral repair tissue was slightly more than three times the thickness of normal articular cartilage.

DISCUSSION

The results of this study confirm that the repair response to an acute osteochondral articular surface injury in a primate fails to restore a normal articular surface by eight weeks. It is possible that with more time the quality of the chondral repair tissue would have improved and the volume would have increased, but given previous studies of articular cartilage repair[2,5,9,12] it seems more likely that the chondral repair tissue would degenerate with time. Cynomolgus monkey normal articular cartilage was about four times stiffer than osteochondral repair tissue. The Poisson's ratio of the normal articular surface, which is indicative of the tissue's apparent compressibility, was twice that of the repair cartilage, and the permeability was greater. Because of its inferior material properties, normal joint use would subject the repair tissue to elevated strain fields causing progressive degeneration of the repair tissue.

TABLE 3
Material Properties of Repair Tissue

Treatment and Location	Number of Animals	Aggregate Modulus H_A (MPa)	Poisson's Ratio vs	Permeability $kx10^{15}$ (m^4/N-s)	Cartilage Thickness at Test Site (mm)	Cartilage Thickness Adjacent to Test Site (mm)
Normal Femoral Condyle	6	0.82±0.18	0.24±0.06	2.44±1.13	0.72±0.09	
IPM Patella	4	0.12±0.04	0.09±0.10	3.17±1.06	2.05±0.81	0.66±0.09
Cast Patella	4	0.22±0.06	0.10±0.01	3.38±0.48	2.18±0.73	0.87±0.57
IPM Femoral Condyle	3	0.17±0.06	0.11±0.03	3.86±0.55	2.22±0.56	0.71±0.14
Cast Femoral Condyle	3	0.27±0.22	0.16±0.11	2.40±1.45	2.19±0.58	0.60±0.21

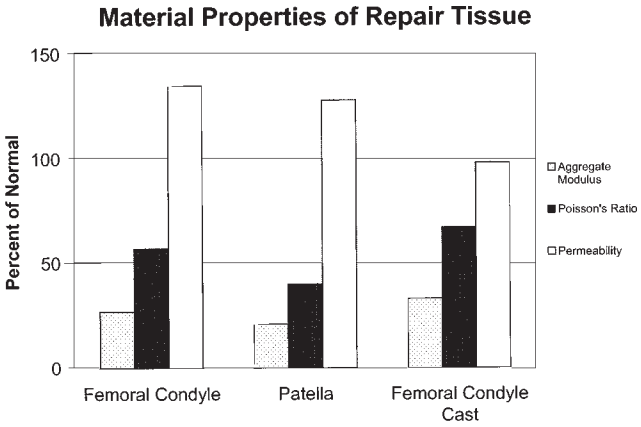

Figure 7. Histogram showing that the osteochondral repair response produced tissue that most closely approached the properties of a normal articular surface in femoral condylar defects treated by cast immobilization.

The difference in the repair tissue in the chondral and osseous portions of the defects eight weeks following injury was striking. Initially the chondral and osseus portions of the osteochondral defect fill with the same clot and then the same initial repair tissue[5,7,9]. Although hyaline cartilage and synthesis of type II collagen occur in both the chondral and osseous portions of the defect the concentration of hyaline cartilage and type II collagen was greater in the chondral portion. Bone matrix and marrow appeared only in the osseus part of the defect. The explanation for the differentiation of the tissue in the chondral part of the defect toward hyaline cartilage and the repair tissue in the osseus part of the defect toward bone remains unknown. Possible explanations include differences in mechanical environment, and diffusion of biologically active molecules from the synovial fluid, the bone or the cartilage.

An equally interesting result is the increased concentration of hyaline repair tissue and type II collagen in the femoral chondral and osseous defects as compared with the patellar defects. The initial repair tissue in both locations presumably is the same, yet by eight weeks the patellar chondral defects have more fibrous tissue and the patellar osseous defects have more bone matrix than the corresponding femoral defects. Since the defects were made in the same joint at the same time it seems unlikely that the maturing repair tissue would be exposed to important differences in biologically active molecules, cells, free radicals or oxygen tension. The only apparent difference between the two locations is the pattern of mechanical loading with joint use, an observation that suggests that different locations in the same joint and different joints may differ in osteochondral repair depending on the patterns of loading of the repair tissue.

The optimal mechanical environment for osteochondral and chondral repair has not been defined. In this study, two weeks of IPM did not stimulate more effec-

tive osteochondral repair than two weeks of cast immobilization. These conclusions do not discount possible beneficial effects that continuous passive motion treatments may have on articular cartilage healing in smaller defects or in other species.

This study shows that the important limitation of the natural acute osteochondral repair response in a primate is the failure to produce a sufficient volume and quality of chondral repair tissue. However, it also shows that the acute osteochondral repair response produces chondral repair tissue that more closely approximates articular cartilage than bone or dense fibrous tissue and that temporary cast treatment of femoral condylar defects followed by active movement produces the best chondral repair. These observations indicate that osteochondral repair can be improved and the risk of post-traumatic osteoarthritis following intra-articular fractures decreased by better understanding of the biological and mechanical conditions that govern acute osteochondral repair.

ACKNOWLEDGMENTS

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TENDON CREEP IS POTENTIATED BY NKISK AND RELAXIN WHICH PRODUCE COLLAGEN FIBER SLIDING

Mark L Wood, MD; William N Luthin, BS; Gayle E Lester, PhD; Laurence E Dahners, MD

ABSTRACT

The pentapeptide NKISK has been reported to inhibit the binding of decorin, a proteoglycan on the surface of collagen fibrils, to fibronectin, a tissue adhesion molecule. Relaxin has been shown to be effective in relaxing ligaments and other connective tissues. Through collagen staining studies, we have previously demonstrated that collagen fiber sliding is important during changes in ligament length. Because of our interest in fibril-fibril binding as it relates to changes in length of tendon or ligament, we investigated the potential of NKISK, relaxin, or both in combination to potentiate creep. We suspended stained rat tail tendons in test solutions under a constant stress and observed length changes and subsequent collagen fiber sliding. Both NKISK and relaxin potentiated rat tail tendon creep with changes in length most likely occurring as a result of collagen fiber sliding as evidenced by photomicrography.

INTRODUCTION

It is accepted that tendon and ligament have the ability to change lengths[2, 5,9,10,19]; however, little is known about the mechanism responsible for these phenomena. Our laboratory has focused on the hypothesis that sliding of collagen fibers past one another plays an important role during these length changes. We have shown in previous in vivo experiments that ligament length changes occur by the movement of collagen fibers past one another during growth and contracture[20] and that the length changes occur diffusely throughout the entire ligament or tendon[2,5,9,10,19]. The "sliding fibril hypothesis" requires that tapered, discontinuous fibrils[7,14,16,17] have a reversible mechanism of fiber to fiber bonding, which releases to allow such diffuse changes in length. A better understanding of collagen interaction during length changes is important because very little is currently known about the development or the mechanism of wound healing in tendon, ligament and other collagen-rich tissues.

Previous work in our laboratory has examined the impact of modifying interfibrillar bonding by immersing stressed tendons in different solutions (varying in pH, calcium ion concentration, osmolarity and concentrations of acetylcholine and epinephrine) all without success. We have, however, found and reported increased strain in stressed tendons when immersed in gentamycin and polylysine, both of which are small polycations with similar charge configurations[11].

Schmidt, et al showed that a pentapeptide, NKISK, competitively inhibits binding of fibronectin to decorin by mimicking the sequence in the decorin molecule, which is presumed to be the fibronectin binding site[12]. Decorin is a small proteoglycan which "decorates" the surface of collagen fibrils and fibronectin frequently functions as an adhesion molecule. Hedbom and Heinegard have proposed that one possible function of decorin is to connect neighboring collagen fibrils[6]. Unpublished work from our laboratory has shown that stressed tendons immersed in NKISK or in relaxin (a hormone effective in relaxing ligaments and other connective tissues)[1,8,13,18] stretch significantly more than control tendons and we have published that free, intact fibrils can be isolated after exposure of ligament to gentamycin or NKISK[2,4].

This study focuses on the effect of NKISK and/or relaxin as well as the possible additive effect of the two agents (in combination) on interfibrillar bonding and on subsequent collagen fiber sliding in the stressed rat tail tendon model. By applying a covalently bonding, fluorescent collagen dye, we were able to label collagen fibers and observe subsequent movement during tendon strain.

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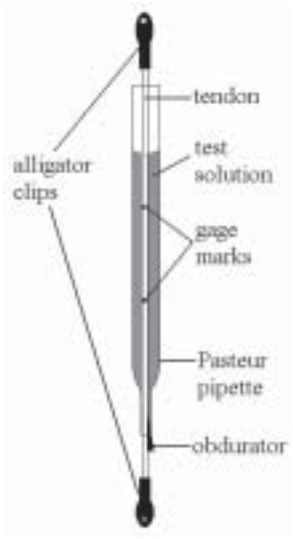

Figure 1. Diagram of the rat tail tendon suspension/loading apparatus.

METHODS

NKISK was synthesized for this experiment by the University of North Carolina Peptide Synthesis Facility and porcine relaxin was provided by Dr. Michael Fields at the University of Florida, Gainesville. The test solutions were 1 m*M* NKISK and 46 units/ml porcine relaxin (3,000 units/mg) in commercially prepared phosphate buffered saline (PBS - Invitrogen Corp, Grand Island, NY - 136m*M* NaCl, 3m*M* KCl, 1 m*M* KH_2PO_4, and 6 m*M* Na_2HPO_4), pH 7.4, to which 0.03% NaN_3 had been added. These concentrations were chosen after they were found to be appropriate in previously performed pilot studies. PBS with 0.03% NaN_3 was the control solution.

After Institutional Review Board approval, rat tail tendons were harvested from sacrificed, fresh frozen, 500 g Sprague Dawley rat cadavers under a dissecting microscope. Each experiment was done with the tendons from a single rat tail, and from the same segment of that tail (2 tails). The tails were thawed for 15-30 minutes then cut through a vertebral disc near the base of the tail and again 6 cm distally. Micro forceps were used to pull tendons out of the distal end of the tail under a dissecting microscope, obtaining similar (medium) sized tendons. The tendons were marked twice, at a gauge length of 15 mm near the center of the tendon segment using a 25 gauge hypodermic needle dipped in India ink. The tendons were pulled individually into glass tubes made from Pasteur pipettes, cut just below the tapered section and again 2 cm above the taper, so that the ends of the tendon protruded from both ends of the pipette segment tubes. The bottom end of the tube was plugged with silicone vacuum grease and a small obdurator, then the tube was filled with the test solution. During the experiment, small volumes of evaporation (approximately 1–2% of the weight of the entire apparatus) were replaced.

Alligator clips grasped the ends of the tendons and the tendon/tube apparatus was suspended from the upper clip (Figure 1). This assembly was weighed at the end of the experiment to determine the load applied to the tendon. There were 4 models (control, NKISK, relaxin, and NKISK/ relaxin mixture) each consisting of 10 tendons (a total of 40 tendons). The distance between the India ink gage marks was blindly measured (twice, then calculating the average) using a 1.5X magnifier and a micro caliper accurate to 0.02 mm at the beginning of the suspension period and again after 3, 6, 12, 27, 36 and 48 hours. The experiment was repeated using the above procedure in a second rat tail with the same 4 models, each consisting of 4 tendons (a total of 16 tendons).

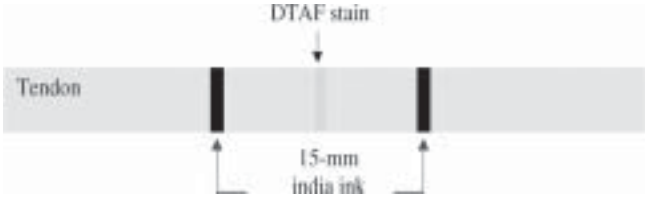

Figure 2. A diagram representing a rat tail tendon and the initial India ink marks with a dichlorotriazinyl fluorescein (DTAF) stain applied perpendicular to the collagen fibers.

In the 2nd experiment, the tendons were stained (perpendicular to the collagen fibers) between the ink marks, with an extremely thin line (Figure 2) using a 10-0 nylon suture dipped in the fluorescent collagen dye dichlorotriazinyl fluorescein (DTAF—5 mg/ml in fresh 0.2 M sodium bicarbonate)[3]. A picture, demonstrating the DTAF stain width, was taken with a fluorescence microscope at 16X magnification. Utilizing photomicrography for measurements allowed standardization between individual specimens as well as blinded measurements.

Each tendon was then suspended for 27 hours at which time a final picture was taken using the fluorescence microscope at 16X magnification. The initial and final fluorescent marking widths were blindly measured (twice, then calculating the average) on the 16X photomicrographs using the caliper (accurate to 0.02 mm) and the percent increase in width (defined as the increase in width normalized to the initial width, expressed as a percent) of the mark was calculated. When the edges of the marks were not sharp, the most uniform area was measured from "tip to tip" in both the initial and final photomicrographs. In both experiments, the India ink mark measurements were used to calculate the percent creep (defined as the increase in length normalized to the initial length, expressed as a percent) at each of the time points. At the end of the experiment stress was calculated[15]. The portion of tendon between the India ink marks was isolated using a scalpel under the dissection microscope and then air dried for three days. Each segment was weighed for 0.00 mg accuracy. For the 15 mm segment, multiplying the weight by the constant 0.133 (this depends on the tendon being 57%

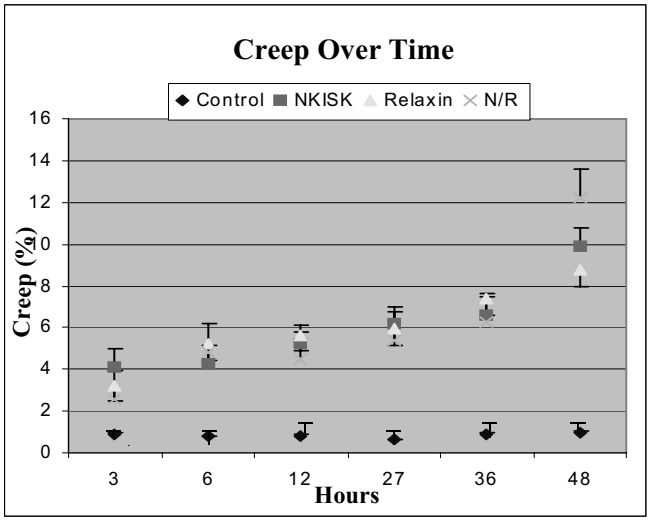

Figure 3. Percent creep over time for rat tail tendons exposed to NKISK, relaxin, and NKISK/relaxin mixture models versus control (PBS). Hash marks represent standard error and all experimental values are significant versus control ($p<0.05$).

water, 43% dry material and having a density of 1.16 gm/cc) converted that weight to cross sectional area in mm^2. Stress is measured in Megapascals (Mpa = N/mm^2), so conversion of the grams of the load to N (9.8N=1 Kg) was done by multiplying by 0.0098.

Stress was calculated by dividing this load by the cross sectional area. Differences between models versus control were evaluated for statistical significance using a Bonferroni modified Student's t-test.

RESULTS

The first experiment consists of 40 tendons (10 per model) without photomicrographs and the second experiment 16 tendons (4 per model) with photomicrographs. In the first experiment, the mean tendon stress in the control, NKISK, relaxin, and NKISK/relaxin mixture models were 0.662 +/- 0.116 (SE) MPa, 0.383 +/- 0.079 (SE) MPa, 0.589 +/- 0.102 (SE) MPa, and 0.481 +/- 0.118 (SE) MPa, respectively. In the first experiment, breakage occurred in 1 of 10 tendons in the control model at 27 hours, 2 of 10 in the NKISK model at 27 hours and 36 hours, 2 of 10 in the relaxin model both at 48 hours, and 0 of 10 in the mixture model. No breakage occurred in the second experiment at 27 hours.

Despite the higher stress in the control group of the first experiment, NKISK and relaxin treated tendons demonstrated significantly more creep than the controls from the same tail in both experiments. Figure 3 displays the induced creep in experimental models compared to that of the control model in the first experiment with all experimental values being statistically significant ($p<0.05$) from the 3 hour time point on.

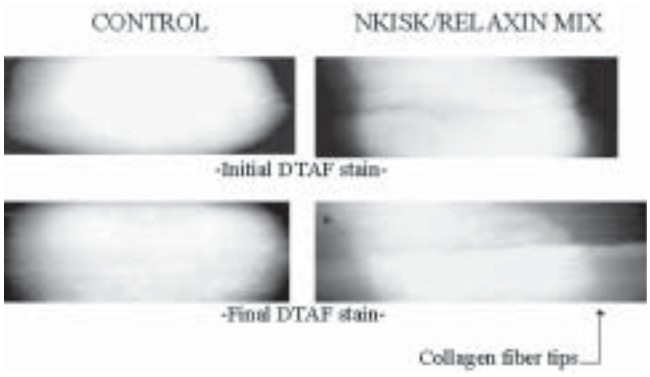

Figure 4. Representative initial (top row) and final (bottom row) photomicrographs of a DTAF mark in the control (left column) and NKISK/relaxin mixture (right column) groups. The final photomicrograph in the control model shows that the stained edges remain relatively sharp while in the NKISK/relaxin model, collagen fiber tips can be seen protruding from either side of the mark as would be expected with collagen fiber sliding.

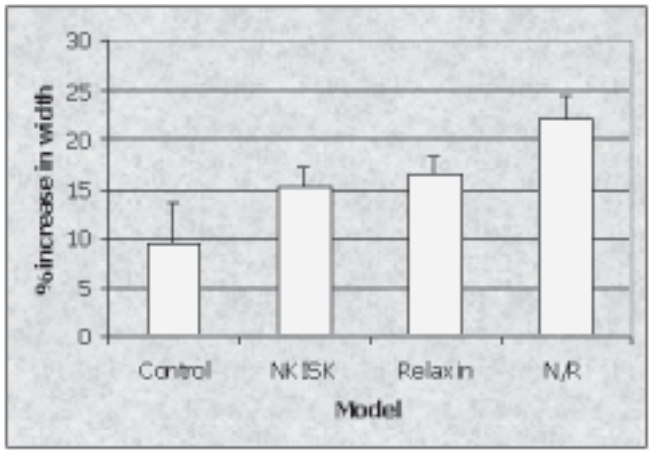

Figure 5. Percent increase in DTAF marking widths in the NKISK, relaxin, and mixture models all showed percent increases greater than in the control, but only the mixture model versus control was statistically significant ($p<0.05$). The hash marks represent the standard error.

Figure 4 shows representative initial and final photomicrographs of a DTAF mark in the control and NKISK/relaxin mixture groups. The final photomicrograph in the control model shows that the stained edges remain sharp while in the NKISK/relaxin model, it appears "fuzzy" and collagen fibers seem to be protruding from either side of the mark as would be expected with collagen fiber sliding.

Figure 5 shows that the DTAF marking widths in the NKISK, relaxin, and mixture models all showed percent increases greater than in the control though only the mixture model versus control was statistically significant.

DISCUSSION

NKISK is known to inhibit decorin-fibronectin binding[12] and postulated to interfere with interfibrillar bonding. Relaxin is known to allow stretching of collagenous tissues[1,8,13,18], but the mechanism is unknown. In this study, tendons exposed to NKISK and relaxin consistently stretched significantly more than the control tendons at each time interval. We feel that NKISK and relaxin potentiated tendon creep and that the changes in length are secondary to collagen fiber sliding as evidenced by DTAF photomicrography. As hypothesized, the fluorescence photomicrographs demonstrated stained collagen protruding from either side of the original mark as one would expect if fibers slid past one another and away from their initial location during changes in length. Actual fibril sliding could not be observed in this study with DTAF as collagen fibril diameters are 50-200nm and individual fibrils could not be resolved with light microscopy. We were able to observe translational collagen movement and it is possible that it may be at the fiber level rather than the fibril level.

DTAF selectively labels collagen in collagen-rich tissues such as tendon. By utilizing gel filtration experiments, Davison and Galbavy have shown that approximately 97% of applied DTAF dye will bind to collagen. The remaining small percentage binds to proteoglycan, glycoprotein and other stromal components. It is unlikely that the observed broadening of the bands is due to diffusion of the dye because of the covalent bonding of the DTAF to collagen. Likewise, it is unlikely that NKISK or relaxin affect the DTAF—collagen covalent bonding. Experiments have shown that collagen-rich cornea stained with concentric rings of DTAF in the growing rabbit show expansion of the stained ring, not diffusion from the ring, which would have narrowed the inner diameter[3].

The DTAF marking widths in the NKISK, relaxin, and mixture models all showed percent increases greater than in the control. Increased marking widths, in the absence of sliding, would require permanent fibril stretching, a phenomenon which is incompatible with the crosslinked structure of collagen fibrils. Collagen molecules wound into triple helices are cross-linked to one another in a staggered array to form fibrils, which by virtue of the fact that all connections are molecular, could be considered to be large “super molecules”. While perhaps not absolutely rigid, the tendons should at least be elastic and once unloaded for photomicrography, should return to their original lengths. The photomicrographs document collagen fibers protruding after unloading, thus stretching is unlikely to account for the observed changes.

Although this technique is limited to measuring movement only on the surface of the tendon, it seems unlikely that the collagen interactions are different in the depths of the tendon. NKISK and relaxin are small polypeptides and should penetrate the depths of the tendon quite readily. Interactions in the depths of tendon are likely similar to the surface because collagen composition is uniform throughout the tendon and it would be very unlikely to observe the increases in tendon length if the depth of the tendon’s length remained constant while the surface elongated.

All values were statistically significant in the first experiment, however, not in the second. The smaller number of tendons used (16 versus 40) in the second experiment was a result of the technically challenging and time consuming procedure involved in staining and photographing DTAF stained tendons. In the second experiment, there was statistical significance ($p<0.05$) only in the NKISK/relaxin model versus control for the percent increase in marking widths. In the NKISK and relaxin test solutions, the mean percent increase in marking widths were greater than the controls, however, the small “n”, or the large standard error, or both, made these differences not statistically significant.

We postulated that if NKISK and relaxin worked by different mechanisms they would have an additive effect when combined. No such additive effect was observed. Bundle ruptures were not likely the source of tendon elongation as this would have been indicated by sliding of large bundles in the DTAF photomicrographs.

There are now reports of three agents that interfere with fibril association (NKISK, relaxin and gentamycin) and to date, we are not aware of any reports that document these agents have anything in common. NKISK is known to inhibit decorin-fibronectin binding and postulated to interfere with interfibrillar bonding[12]. Relaxin is known to allow stretching of collagenous tissues, but the mechanism is unknown. Some authors propose that relaxin removes cross-linking of fibrils allowing them to slip and re-orient, which may be stimulated by increased collagenase expression and downregulation of collagen secretion[13,18]. Gentamycin is a small polycation and its size and charge configuration are very similar to the KKK tripeptide, which also allows increased tendon lengthening[11]. We hypothesize that it may also bind to the decorin-fibronectin binding site.

Though this study provides some understanding of vertebrate fibril sliding, much work remains to be done. Future work should include further investigations to elucidate the mechanisms by which collagen fibrils are presumably bound as well as the mechanism of collagen sliding. In this study, decorin-fibronectin bind-

ing appears to play an important role in the bonding of fibrils to one another to prevent constant sliding. These results provide further support for the "sliding fibril hypothesis" for length changes in collagenous tissues.

ACKNOWLEDGMENTS

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INTERNET USE BY PATIENTS IN ORTHOPAEDIC SURGERY

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ABSTRACT

This study evaluated Internet use among orthopaedic patients in a private practice general orthopaedic setting. Of 201 participants, 45% had used the Internet either personally or thru a surrogate to search for information about their orthopaedic condition. Utilization of the Internet was significantly higher than that reported for a community orthopaedic practice surveyed in 1998, suggesting that utilization by orthopaedic patient populations mirrors the increasing societal use for health information.

Most users in this study employed multiple search strategies, including using search engines and sites recommended by others. The majority of users found medical information on Internet sites to be useful and accurate. The number one choice for reconciling conflicting information was to ask a physician or a nurse. Most users and non-users reported that they would recommend the Internet to others as a source for medical information. We recommend several strategies for orthopaedic surgeons to stay abreast of these changes and to utilize Internet Patient education resources to their own advantage.

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Study conducted at OSMC, Elkhart, Indiana, and the University of Michigan Medical School, Department of Orthopaedic Surgery

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INTRODUCTION

We previously reported on Internet use by outpatient orthopaedic patients in a community and in an academic tertiary care setting based on data collected in 1998.[1,2] Internet use in the United States has grown exponentially over the past five years, and access to the Internet has increased yearly. In this study, we surveyed a population of patients in a community outpatient orthopaedic practice in 2002 to see whether there were changes in Internet utilization.

METHODS

After obtaining Institutional Review Board (IRB) approval, we invited patients visiting an outpatient community orthopaedic practice (OSMC, Elkhart, Indiana) during a one week period in July 2002 to participate. We used a questionnaire that had previously been found to be reliable in another outpatient orthopaedic sample.[2] Patients were recruited by a medical student investigator (JK) at time of check-in for their appointment in the outpatient office, and surveys were filled out on-site. As many patients as possible were invited to participate in the study. Informed consent was obtained and patients were surveyed regarding their Internet use and their attitudes about Internet-retrieved health information. Specifically, the questionnaire was designed to assess study group demographics, access to and usage of the Internet, and opinions about the utility of the Internet in obtaining medical information. As in previous studies, we assessed not only Internet use by respondents, but also Internet use by persons other than the respondent for the respondents' use (designated "surrogate" users). We also surveyed patients regarding their methods of identifying information to review, and asked how they planned to utilize the information. All survey responses were sorted and prepared for analysis using the SPSS (Chicago, Illinois, USA) statistical package.

RESULTS

During a one week period, 568 patients were seen in this clinic. Of these eligible patients, 210 were invited to participate in the study; 201 returned the questionnaire. Of the 201 respondents, 108 (54%) were female,

93 (46%) were male. The mean age of participants was 46 years (range 12-86 years), and the mean time between initial orthopaedic diagnosis and questionnaire completion was 1.8 years (range 0-46 years). Of the 178 patients responding to the question regarding prior surgery, 93 (52%) reported having had surgery for their orthopaedic conditions. One hundred fifty-one respondents (75%) reported that they had access to the internet at work or home. Of the 50 who reported no Internet access, 3 (6%) reported that they nevertheless personally searched the Internet for information on their condition, and 3 (6%) had a surrogate search for them. Of the 151 participants with Internet access, 59 (39%) reported that they personally searched the Internet for orthopaedic information; 45 (30%) had a surrogate search for them, and 69 (46%) reported that they had used the Internet either personally or through a surrogate to find information about their condition (further designated as "Internet users").

Internet users were slightly younger on average than non-internet users (42.7 yrs vs. 47.4 yrs), but the difference was not statistically significant. There was no significant difference in percentage of women among users vs. non-users. There was no significant difference is surgery status between the two groups.

Of the 68 subjects who reported how they found their sites, 55 (81%) reported that they used a search engine to find information about their orthopaedic condition. Forty-two percent used sites recommended by others, and 47% found sites through other means. The most frequently used search engine was Google (24%), followed by Yahoo (21%), AOL (17%) and Netscape (8%). Not everyone who reported using a search engine listed which one they used, but of those who did, many listed two or three different engines.

Of the 64 who responded to the question, 50 (78%) reported that sites they visited helped them better understand their medical condition. Of the 66 who responded to the question, 27 (41%) reported that sites they visited raised questions to discuss with their physicians. Of the 62 who responded to the question, 49 (79%) reported that *all or most* of the information on the sites they visited was accurate, and the remaining 13 (21%) reported that *some* of the information was accurate. Of the 62 who responded, 42 (68%) reported that they usually can tell if the information on a site is accurate, 16 (26%) reported that they can sometimes tell, and 4 (7%) reported that they can rarely tell. Seventy-five percent of those who responded would ask a physician or nurse to help them reconcile conflicting information they found on a site; 16% would compare information on different sites to reconcile conflicts; 12% would ask support group members; and 4% used other strategies. Many who responded gave multiple responses.

Of the non-Internet users with valid responses, 77% said that they would recommend the Internet to others as a source of medical information. Of the internet users with valid responses, 96% said that they would recommend the Internet to others as a source of medical information. Of the non-internet users with valid responses, 46% would use Internet access available in the clinic; of the internet users with valid responses, 74% would use Internet access available in the clinic

DISCUSSION

We identified a higher use of the Internet among community outpatient orthopaedic patients in this study (46%) than in our previous report (20%) from data collected only four years earlier, confirming that Internet utilization for medical information among orthopaedic patients mirrors the substantial increases in use of the Internet for health information seen in the general population.

Not only are more orthopaedic patients using the Internet, but they are having questions about the material they have researched which they plan to raise with their caregivers—75% in this study. Unfortunately, in this study as in others, patients are mainly finding material by using commercial search engines, a strategy which has proved to be inefficient of their time and often finds incomplete or inaccurate web sites.[3]

Patients may not be the best judges of the quality or accuracy of material they retrieve. A study by Consumer Web Watch looked at how Internet health experts rated sites compared to "consumers".[5] Not surprisingly, health experts highly rate such factors as the posting institution, presence or absence of solicitation or advertising, and the source of the material as the most important. Consumers, on the other hand, rated "design look" as the most important criteria in determining the credibility of a site.

What is the best strategy for addressing this growing trend among patients? Orthopaedic surgeons can a) familiarize themselves with available web material, b) recommend sites to patients, and c) participate within the AAOS and specialty societies to create high quality web-based instruction.

While the thought of reviewing all patient education material on the Web is daunting, it is also unnecessary. Several sources of high quality patient education are available, including the American Academy of Orthopaedic Surgeons (www.aaos.org), and the University of Iowa Virtual Hospital (www.vh.org). Reviews of Web

based patient education information can be found in the AAOS Bulletin[4], or at Orthopaedic Web Links (http://www.orthopaedicweblinks.com). Taking only a few minutes to review material in one's area will pay off.

By recommending sites to patients, the surgeon can ensure they are receiving quality materials to review, patients can be saved many hours fruitless searching and finding material which may or may not apply to them, and the surgeon can, to some extent, preempt patient explorations of material of questionable quality. Expedient ways of making these recommendations include a) making a handout of sites, especially focusing on specific documents or portions of web sites for a specialty practice, or b) placing links on one's own web site (which American Academy of Orthopaedic surgeons members can create for free at the AAOS web site).

Finally, as we strive to keep up with this trend in patient education, we should promote and contribute as much as possible to the Web based patient education efforts of our professional societies. If we, rather than the commercial sector, wish to be our patient's main educators, staying on the leading edge of the information wave is crucial.

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THE EFFECT OF THE INTERNET IN THE TREATMENT OF CONGENITAL IDIOPATHIC CLUBFOOT

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ABSTRACT

Parents have traditionally relied on health care professionals for advice and treatment for most orthopaedic conditions, including clubfoot. However, the unprecedented access to health care information offered by the Internet is changing how parents gather information and make treatment choices. This study was designed to evaluate the effect of the Internet in the treatment of clubfoot. We performed a retrospective review of the clinical census, statistics of the Virtual Hospital web pages on clubfoot, web sites, and web based support-groups related to clubfoot from 1995 to 2002. There were 157 patients that came to our clinic for treatment, a dramatic increase compared to previous years. There were a total of 790,084 hits to the Virtual Hospital web pages, with information requested from all states and 72 countries. Interestingly, the referrals also changed with patients coming from 30 states and 8 different countries, compared to previous years when the majority came from our own state. In addition, 75% of the patients were self-referred, many of them while on treatment at outside institutions. There were 160 web sites providing information on clubfoot and 5 large support groups. There were approximately 30,000 messages (average 1000 messages/month) posted into the support clubfoot websites. After visiting Internet support groups, 125 parents transferred or initiated the care of their children to a doctor practicing the Ponseti method. In conclusion, the Internet provides a mean for parents to obtain disease-specific information in a timely manner. Parents used the support-groups to find and proffer information, share experiences and opinions, and provide encouragement. This sharing of information is affecting how parents make their treatment choices and also has the potential to induce unexpected changes in clinical orthopaedic practice.

INTRODUCTION

The digitalization of information, the ability to network, and the rapid electronic interchange of information on a worldwide basis are recognized hallmarks of today's society. The Internet, with 130,000,000 users and growing at a rate of 2% per month, is a global, independent but cooperative network system that allows remote access to data and permits its quick access in unprecedented volume.

The world of medicine has not escaped the impact of the Internet, and health-related sites are among the most frequently accessed information resources. A recent survey indicated that 70,000,000 US adults had accessed the Internet to obtain health or medical information in 2001[18]. At least 100,000 health and medical sites are on the World Wide Web, and are maintained by entities ranging from academic medical centers and professional organizations to individuals. Thousands of other online self-help and support groups, bulletin boards, and mailing lists also are available. However, how this unprecedented access to health care information is affecting the way parents make decisions on treatment options, and how those decisions could impact clinical practice are not well understood[3-6,13,16,17]. Interestingly, in 1998, a web page with information on the treatment of clubfoot as described by Ponseti was posted in the Virtual Hospital of the University of Iowa. Since then, we have witnessed a dramatic change in our referral patterns. This study was designed to evaluate the effect of the Internet in clubfoot clinical practice at our institution and how web-based information and supports groups influence parent choices for the treatment of clubfoot.

MATERIALS AND METHODS

We performed a retrospective reviewed of the clinical census at our institution for the diagnosis of clubfoot from January 1991 to December 2001. Demographic data was evaluated with respect to living address, referral entity (self-referral or physician referral), age of the patient at presentation, previous treat-

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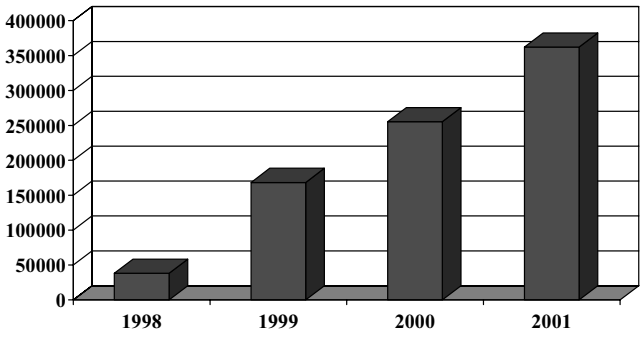

Figure 1. Number of hits at the Virtual Hospital clubfoot web page. Note the increase over the short period of time, to a total of 790,084.

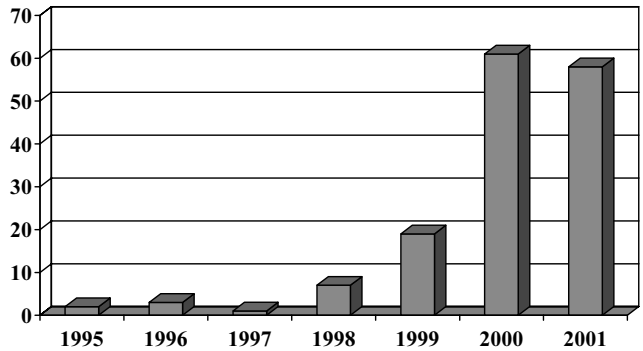

Figure 2. Number of patients seen in clinic from 1995 to 2001. Note the dramatic increase since 1998 when the Virtual Hospital clubfoot web pages were posted.

ment and type of treatment at the outside institution. We also performed a review of the statistics of the Virtual Hospital web pages for the treatment of clubfoot. Total number of hits, number of pages visited, and total visits were recorded. In addition, location of the request was also recorded.

We also performed a review of the public web sites providing information on clubfoot, as well as parent-support groups for clubfoot from October 1998 to December 2001. From the 5 most active sites, total number of members, new members per month, and total number of messages posted were recorded. In addition, evaluation of the content of the messages was performed, specifically information on decision making by the parents about treatment choices.

RESULTS

From the initial posting of the web pages on clubfoot in the Virtual Hospital in October 1998 through December 2001, there were 790,084 hits, with an average of 27,334 hits/month (Figure 1). Information was requested from all states and 72 countries. These included countries such as India, Singapore, Spain, Egypt, Australia, etc.

When evaluating patient referrals to the clinic, we observed there was an increase in the number of patients per year that paralleled the hits to the Virtual Hospital clubfoot web pages (Figures 1 and 2). In the years prior to 1998, there were an average of 5 patients seen in clinic by the senior author (IVP). This number increased to about 60 patients per year by 2001 (for a total of 157 patients). Interestingly, 75% of patients attending the clinic were self-referrals, with 71% having had treatment or currently on treatment at an outside institution.

Age at presentation demonstrated a significant difference between pre and post Internet web page posting. Patients in the early 90's were seen usually in their first month of life. After 1998, the average age at presentation was 3 months, with a range from newborn to 22 months of age. Eighteen per cent of the patients were older that 6 months at presentation (data not shown).

With regard to the parents-support groups, there were over 160 web sites providing information on clubfoot and 5 large support groups. There are sites and support groups for General Information, Surgery, Ponseti method, French Physiotherapy method, as well as parental sites in the UK, Australia, France, Germany and other countries. In the past few years, the fastest growing sites and topics have been related to conservative methods such as the Ponseti method (Figure 3). We have observed an increased number of people using group sites to obtain information and advice, with the growth of 40 new members per month.

There were approximately 30,000 messages (average 1,000 messages/month) posted on these websites and the messages are publicly available (Figure 4). Compared to the other sites, the Ponseti site had the greatest increase in number of messages. Importantly, after visiting the support groups and talking with active members in the groups, 125 parents brought their children to our institution, or to another institution where there are professionals practicing the Ponseti method. Interestingly, only 3 of these children required extensive corrective surgery after they were treated by the Ponseti method.

DISCUSSION

The results of this study, using clubfoot as a model, demonstrate that the Internet can have a profound effect in clinical practice patterns, and in the patient physician relationship. Since the introduction of information about clubfoot on the Virtual Hospital by the senior author (IVP), we have seen a dramatic increase in the

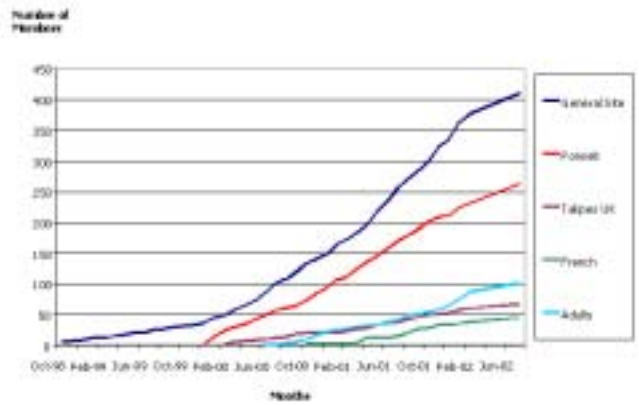

Figure 3. Total number of members at Internet sites. Note the exponential increase in the number in the general web sites and in the Ponseti method.

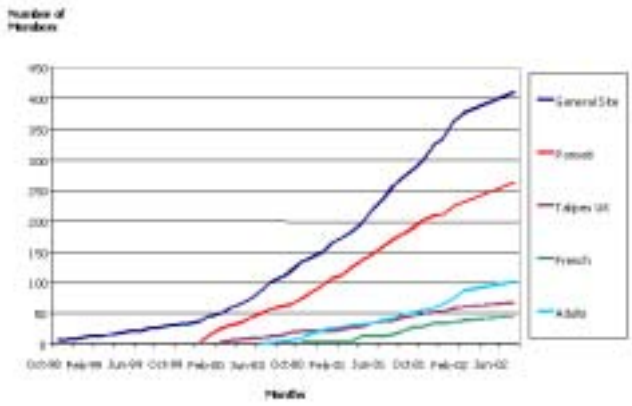

Figure 4. Number of messages posted at the different web sites. Note the correlation between the number of messages in the general site and the Ponseti web site (four months weighted average).

number of patients attending our clinic, with the majority of them coming from out of state and a significant number having been treated at an outside institution. In addition, general information and parent support groups for clubfoot have grown almost exponentially. Importantly, many parents decided to change treating physicians after consulting and sharing information on the web.

Increasing use of the Internet by consumers in general is being reflected in greater reliance on this medium for health information and health care. In part, the use of the Internet to find answers to health-related questions has been linked to several issues affecting existing practice patterns. Many people are using the Internet due to the belief that today's doctor-patient relationship lacks attention to detail and the personal touch that was present in the past. Patients also desire more involvement in and control over the management of their own health. Furthermore, there are alternatives to traditional providers and methods of treatment, and patients can not only find them on the Internet, but also retrieve information that is stated objectively and many times, non-technically. Finally, the Internet also offers a means to get a "second opinion" without the hassles of a referral and can provide a way to communicate with others patients or families in similar situations or with similar problems[3-6,13,16,17].

In the case of clubfoot, several of these issues seemed to be responsible for the changes observed in our practice. Until recently, non-surgical methods of correction have demonstrated a low success rate, with up to 90% of the patients requiring extensive corrective surgery. However, clubfoot surgery is frequently associated with persistent stiffness of the foot, and may lead to many complications (up to 25% of cases) and the need for secondary procedures (up to 47% of cases). Acceptance of these poor results, however, has been the "norm" because clubfoot has been viewed as a surgical deformity, very difficult to correct[1,2,7,9,10,11,12,14,15, 19,20,23-28].

Given that the results of surgical treatment can be so discouraging, parents looked for alternative treatments. Traditionally, parents would have relied on health care professionals for advice and treatment. However, the development of the Internet and the explosion of health care information allowed them to obtain disease-specific information in a timely manner. In addition, parents created and actively used Internet support-groups on clubfoot to find and proffer information, share experiences and opinions, and provide encouragement to other parents.

From information available through the Internet, parents became aware of the Ponseti method, and of the controversy that existed over the need or not for extensive surgical treatment for the correction of the deformity. Interestingly, clubfoot is a very "visual" deformity, i.e., it is very easy for the parents to see and assess the results of any treatment. The fact that the Ponseti method allows full correction of the deformity in a very short period of time and without the need for extensive surgery is of critical importance[8,21,22]. Parents realized the benefit of this treatment modality and share their experience with others. As a result, the number of messages on the Ponseti method at the different clubfoot web sites and parents support groups increased exponentially. This also resulted on many parents transferring the care of their child to a physician with knowledge on the Ponseti method (75 % of our referral population and 125 parents in the support groups).

In conclusion, the Internet provides a mean for parents to obtain disease-specific information in a timely manner. Parents use the support-groups to find and proffer information, share experiences and opinions, and provide encouragement. This sharing of information is affecting how parents make their treatment choices and also has the potential to induce unexpected changes in clinical practice. The implications of these effects deserve further investigation.

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OF HEDGEHOGS AND HEREDITARY BONE TUMORS: RE-EXAMINATION OF THE PATHOGENESIS OF OSTEOCHONDROMAS

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ABSTRACT

The osteochondroma is a common, benign, primary tumor of bone. A mechanism for its pathogenesis has not been identified, but loss of function of EXT genes is implicated in sporadic and hereditary multiple osteochondromas. Recent advances in the understanding of other molecular signaling pathways in the physis cast doubt on the latest pathogenetic theories. These advances are reviewed and used as the basis for a revised theory for pathogenesis: A clone of proliferating chondrocytes without functional EXT1 (or EXT2) expression fails to produce heparan sulfate; lack of heparan sulfate at the cell surface disrupts fibroblast growth factor signaling and Indian hedgehog diffusion, leading to focal overproliferation and adjacent bone collar deficiency, respectively; together these effects are proposed to contribute to osteochondroma pathogenesis.

INTRODUCTION

Osteochondromas, or exostoses, are cartilage-capped excrescences of bone that develop during physeal growth. Although their incidence may be underestimated given the fact that many sporadic osteochondromas cause no symptoms, they are nonetheless considered the most common of benign primary bone tumors[55].

Hereditary multiple exostoses (HME), alternatively called diaphyseal aclasis or osteochondromatosis, is a highly penetrant, autosomal dominant trait characterized by slightly stunted growth of long bones and multiple osteochondromas[30]. These osteochondromas are indistinguishable morphologically from the solitary cases (see Figure 1). HME has an incidence of about 1

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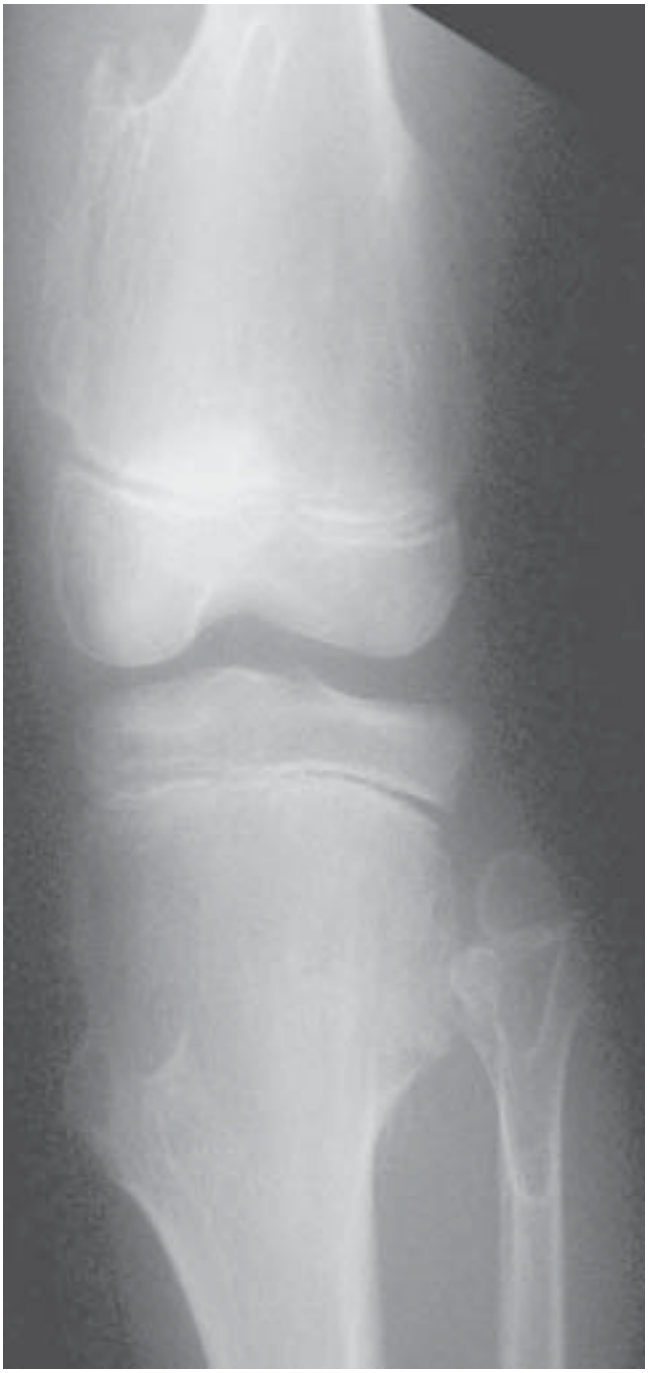

Figure 1. Anteroposterior radiograph of the knee of a skeletally immature patient with hereditary multiple exostosis. Note the multiple osteochondromas on the femur, tibia, and fibula.

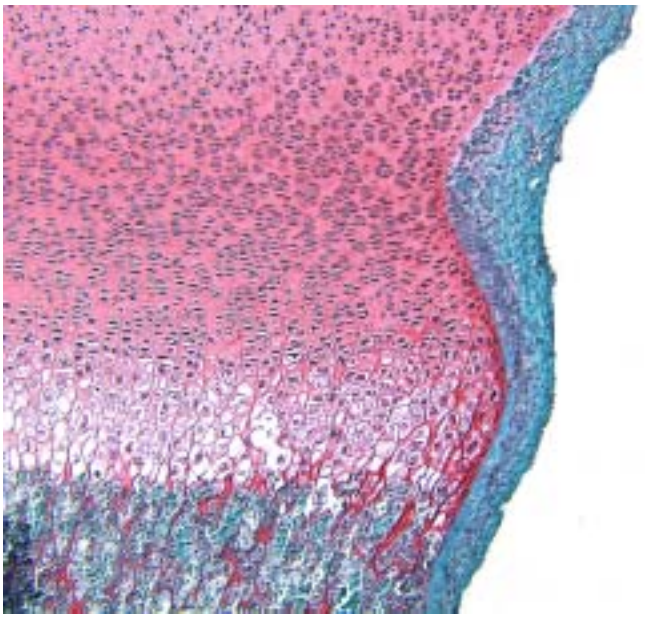

Figure 2. Photomicrograph of the physis of a rat stained with saffranin-o (A). The eosinophilic areas represent the proteoglycan-rich cartilaginous matrix. The basophilic regions are relatively concentrated with collagenous matrix. This enlarged view (B) shows greater detail in the ossification groove of ranvier (mostly closely approximating the physis) and the perichondrial ring of LaCroix (surrounding the groove).

in 50,000 live-births[55]. Many patients with HME require resection of an osteochondroma due to a mass effect or neurovascular impingement symptoms. Importantly, up to 3 percent of patients with HME will eventually develop a chondrosarcoma in the cartilaginous cap of the lesion[21,55,69,72]. Improved understanding of the pathogenesis of osteochondromas has implications not only for patients with HME, but for the more general understanding of mechanisms of neoplastic transformation and possible subsequent malignant degeneration. In this article, we will critically review the current theories of osteochondroma pathogenesis as well as how they came about; we will also propose an alternative theory, derived from the latest advances in physeal molecular signaling.

LITERATURE REVIEW

Through the twentieth century, varied etiological theories for osteochondromas have been derived from histological observation. Müller suspected osteochondromas to arise from erroneous differentiation of cells in the periosteum[42]. Others have hypothesized a migration of physeal chondroctyes into the metaphyseal periosteum[49]. Still others have supposed the perichondrial groove of Ranvier (see Figures 2 and 3) to be the source of osteochondromas[46,58]. The only animal model of osteochondroma formation involved irradiation of the

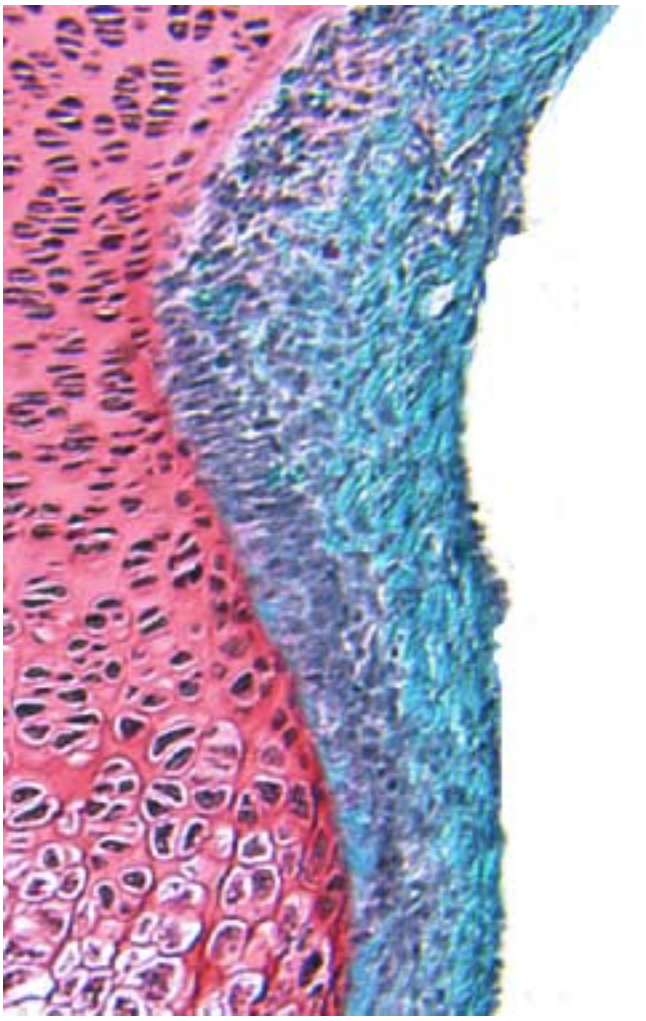

Figure 3. Schematic representation of the physis during development. The zones of the physis are labeled as well as some details of the periphyseal anatomy.

groove of Ranvier in rabbits[15]. Nevertheless, the cells of origin and pathogenesis of osteochondromas remain unclear.

Genetic Analysis of Hereditary Multiple Exostosis

Over the last 2 decades, we have witnessed a dramatic advance in molecular biology and genetics. These have permitted a glimpse into the molecular players underlying these lesions. Genetic linkage analysis has located three etiological genes for HME—EXT1 (8q24.1)[36], EXT2 (11p11-p12)[31,74], and EXT3 (19p)[29]. Interestingly, mutations in any of these genes demonstrate very similar clinical manifestations. The human EXT1 and EXT2 genes have been cloned[1,75], as have homologues in mice[11,34,61], *Caenorhabditis elegans*[11], and *Drosophila melanogaster*[4,25]. These EXT loci have defined a

new class of putative tumor suppressor genes, to which have been recently added three related genes, EXTL1[73], EXTL2[75,76], and EXTL3[54,66], which have also been cloned[24,26].

Because both sporadic osteochondromas and those associated with HME have been associated with loss of heterozygosity (or somatic loss of function of the wild-type allele) of one or more of the EXT loci, a neoplastic model of pathogenesis has been suggested[48]. The Knudson 'two-hit' theory of carcinogenesis[27], derived from familial retinoblastoma and displayed elegantly by Vogelstein and his colleagues in the genetics of colorectal cancer[67], has been applied to the osteochondroma. Both copies of the EXT1 gene have been observed to be microscopically deleted in osteochondromas of both sporadic and familial varieties[7,41]. The Knudson theory's application to osteochondroma pathogenesis has been strengthened by noted EXT gene losses and mutations in chondrosarcomas arising from osteochondromas[7,19,20,50].

These data are consistent with a neoplastic model of pathogenesis for osteochondromas; they provide the basis for the presumed tumor suppressor function of the EXT family of genes. However, while the classification and localization of genes in the EXT family continues, the biochemistry of EXT1 and EXT2 function presents questions as to how these genes can function as tumor suppressors.

Biochemical Studies on EXT1 and EXT2

Exostosin-1 and exostosin-2, the protein products of EXT1 and EXT2 are widely expressed type II transmembrane glycoproteins of 746 and 718 amino acids, respectively. Both localize to the endoplasmic reticulum and Golgi complex[38]. There, they together perform the N-acetylglucosamine (GlcNAc) and D-glucuronic acid (GlcA) transferase activities of a heteroligomeric heparan sulfate polymerase[56]. They function as enzymes, attaching sugar moieties to the surface of proteins that will ultimately be secreted into the extracellular matrix or integrated into the cell membrane. Loss of functional EXT1 or EXT2 in a chondrocyte alters its ability to attach heparan sulfate to the proteins intended for its cell surface and its immediate extracellular milieu1[7,33,39,40]. No other functions of EXT genes have yet been confirmed.

Possible mechanisms for tumorigenesis remain open for consideration, even if EXT genes exhibit cellular function limited to enzymatic catalysis of heparan sulfate proteoglycan (HSPG) synthesis alone. Insight was initially gained through study of the *Drosophila* homologue of EXT1, *tout-velu*. *Tout-velu*, like the EXT genes, is also a functional polymerase for HSPGs. It has been shown to be necessary for diffusion and long range signaling of hedgehog (Hh), a potent developmental patterning factor[4]. The hypothesis has thus been offered that EXT gene dysfunction results in failed long-distance signaling of human homologues of Hh. The known human homologues of Hh include Sonic hedgehog, desert hedgehog, and indian hedgehog (Ihh)[47]. Ihh specifically has been shown to be involved in signalling in the growing physis. Lending confidence to the extrapolation from the relationship between Hh and *tout-velu*-dependent HSPG synthesis, Ihh diffusion is indeed HSPG dependent[18]. Further, EXT1 null mice fail to gastrulate properly, consistent with dysfunctional diffusion of Hh proteins known to be critical for spatial patterning[32,33].

Ihh Signaling Pathway

The explanatory theories most recently presented in the literature focus on disruption of Ihh signaling due to loss of EXT-dependent HSPG synthesis[4,17]. However, these theories have not expanded attention beyond the disruption of a single function of Ihh in the growth plate. The Ihh function in focus is a negative feedback loop whereby chondrocytes that have begun to hypertrophy express Ihh, which diffuses long-range to induce expression of parathyroid hormone related protein (PTHrP) in the reserve zone and periarticular chondrocytes. PTHrP diffuses back to the yet proliferating chondrocytes to prevent their initiation of hypertrophy (See Figure 4). By this feedback loop, Ihh expression after cell cycle exit postpones the same cell cycle exit in the proliferating chondrocytes located just one layer closer to the epiphysis, allowing more rounds of proliferation.

The current theories of osteochondroma pathogenesis hypothesize that focal loss of this Ihh/PTHrP negative feedback loop for a clone of EXT null chondrocytes causes formation of an osteochondroma. However, the most recent knowledge regarding molecular signaling in the physis is incompatible with this hypothesis for two reasons.

First, osteochondromas represent an increased—although misdirected—focal linear growth of bone. Quite in contrast, a reduction in the PTHrP feedback loop results in focally decreased linear physeal growth. Without PTHrP signaling, chondrocyte differentiation, hypertrophy, and apoptosis occur after fewer rounds of proliferation, permitting the ossification front to focally advance into the physis[9,10,28]. The supposition that accelerated chondrocyte differentiation and early ossification could create a local excess of bone, simply fails to fit the data gathered from experiments in which PTHrP signaling to clones of cells was specifically disrupted[9,10]. If any effect were achieved through disrup-

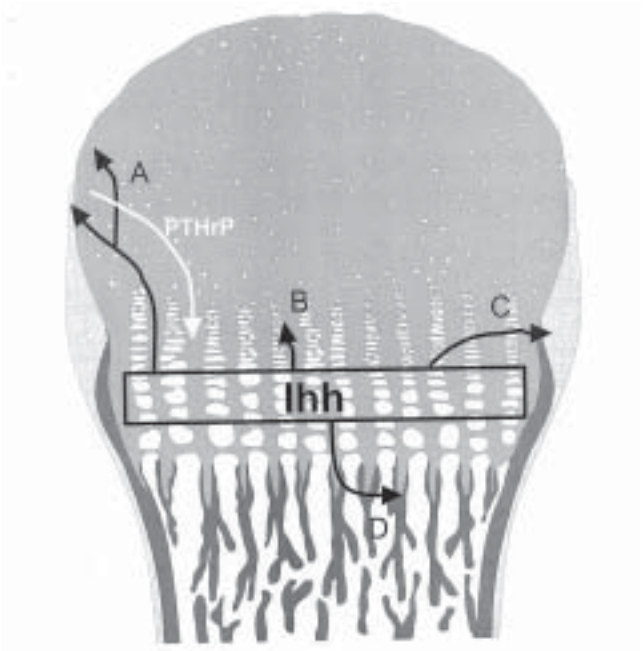

Figure 4. Schematic representation of the signaling pathways of indian hedgehog (Ihh) in the physis, including: (A) long-range signaling to periarticular cells which then secrete parathyroid hormone related protein (PTHrP) to return and complete a negative feedback loop, (B) mitogenic signaling to the proliferating chondrocytes, (C) signaling to the perichondium to induce osteoblastic differentiation and bone collar formation, and (D) signaling to the primary spongiosa to induce osteoblastic differentiation and endochondral ossification.

tion of this pathway, it would likely be the opposite of an osteochondroma.

Second, focal results of any kind would not be expected from interruption of the first of a two-step, long-range feedback loop. Poor diffusion of Ihh through an EXT-null clone of cells might result in poor return signaling of PTHrP, but the reduced long-range signal return would not be directed specifically toward the EXT-null cells. Instead the reduction in PTHrP signaling would more likely be distributed across the entire physis, EXT-null clone cells and neighboring wildtype cells alike. A change in the focal growth of an EXT-null clone according to this pathway would depend more upon the altered diffusion/signaling of PTHrP than that of Ihh itself. No disruption of PTHrP signaling in the absence of HSPGs has been confirmed or refuted. Focal loss of Ihh expression may mimic the focally disrupted Ihh diffusion through EXT-null chondrocytes. This has been tested in Ihh null/wildtype chimeric mice. Physis-wide stunted growth was observed in these mice[10,28]; they did not form osteochondromas. It is possible that such a mechanism contributes to the phenotypic shortening of long bones in individuals with HME, but that it would induce osteochondromas seems unlikely.

Despite these problems with the recent thinking about osteochondroma etiology, the attention paid to disrupted Ihh function may not be misdirected. The theories simply need to be updated with the most recent information from the rapidly advancing knowledge of physeal physiology and signaling. In addition to the Ihh control of PTHrP secretion by periarticular cells[68], Ihh has direct signaling to the proliferating chondrocytes[35], where it is a powerful mitogen[10,23,62], and to the perichondrial and primary spongiosal mesenchymal stem cells[10,62], which it induces toward osteoblastic differentiation[43,59] (see Figure 4).

The effect of lost EXT-dependent HSPG synthesis on Ihh mitogenic signaling to proliferating chondrocytes depends on whether diffusion alone is disrupted or ligand-receptor interaction is as well. If only Ihh diffusion is blunted around EXT deficient cells, then it could be reasoned that proliferating chondrocytes without functional EXT "see" a higher concentration of Ihh. As it cannot pass by, Ihh might build up near EXT-null cells, more powerfully signaling them to proliferate. This could contribute to focal overproliferation of cells and the formation of an osteochondroma. However, if signaling is less efficient or impossible without HSPGs, as has been suggested in EXT knockout mice[33], then this disrupted Ihh function would result in reduced proliferation, detracting from, rather than contributing to osteochondroma pathogenesis.

The third known function of Ihh in the growth plate is its regulatory role in inducing the perichondrial and primary spongiosal mesenchymal stem cells to differentiate into osteoblasts[62]. Expression of Cbfa1, which is required for osteoblast differentiation, does not occur in the absence of Ihh signals diffusing from the prehypertrophic chondrocytes to these two populations of cells[22]. Mice that do not express functional Ihh form no endochondral bone[62]. Specifically, focal loss of Ihh expression at the periphery of the growth plate results in a focal defect in the bone collar, or the advancing lip of the cortical bone, that normally forms to surround the metaphyseal aspect of the physeal chondrocytes[10].

It can be reasoned that a peripherally located, EXT-null clone of proliferating chondrocytes, unable to synthesize HSPGs, will prevent the diffusion of Ihh to the perichondrial cells in the abutting region of the groove of Ranvier (see Figure 5B). A focal defect will therefore develop in the bone collar peripheral to the EXT-null chondrocytic clone as those perichondrial cells fail to differentiate into osteoblasts in the absence of Ihh signaling. Perhaps, the loss of this rigid structural constraint of the bone collar is tantamount to losing some

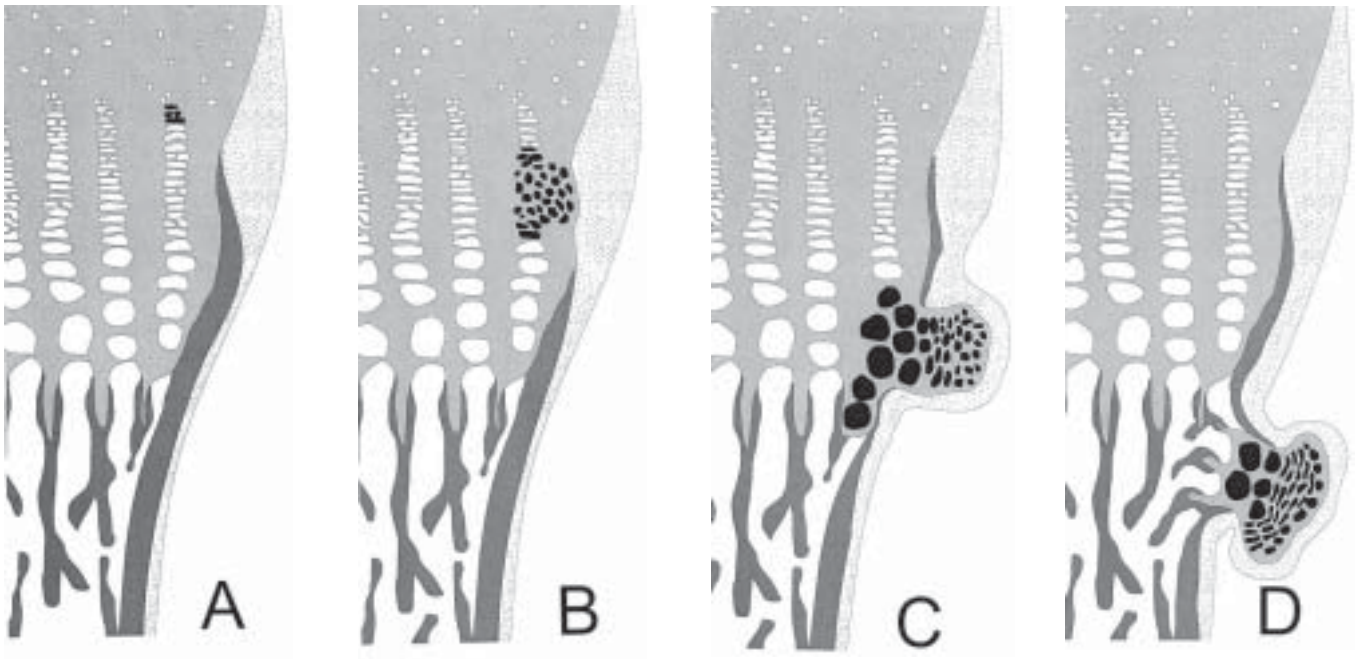

Figure 5. Proposed pathogenesis of an osteochondroma begins with proliferation of an EXT null clone of chondrocytes (shaded black) (A). As this clone over-proliferates due to hindered fibroblast growth factor signaling, it also impedes the diffusion of Indian hedgehog (Ihh) to the perichondrium, thus leaving a defect in bone collar formation (B). As the succeeding clones of chondrocytes progress into the proliferative zone behind the EXT null cells, the bone collar begins to form again epiphyseally to the now budding osteochondroma (C). As chondrocytes of the osteochondroma cap eventually exit the cell cycle and express Ihh, they induce immediately adjacent bone collar formation (although lagging due to lack of diffusion) around the osteochondroma stalk and eventually hypertrophy and permit endochondral ossification to form trabecular bone continuous with the rest of the primary spongiosa (D).

of the restraint to proliferate only longitudinally. When the bone collar is prevented from forming by reflection of the perichondrium overlying the growth plate, the chondrocytes beneath have been shown to proliferate in the peripheral direction[14]. As the EXT-null clone proliferates and "overflows" the defect in the bone collar, it maintains an outer lining of undifferentiated perichondrial cells. As the next generation of proliferating chondrocytes progresses toward hypertrophy, the bone collar is again formed around the next clone of normal chondrocytes, upstream to the outpouching osteochondroma (see Figure 5C).

The perichondrial cells lining the protruding EXT-null chondrocyte clone still express functional EXT. When Ihh signal diffuses to their location, their receptors will undoubtedly receive the signal, differentiate into osteoblasts, and appose a lip of cortical bone. However, because the outpouching cartilaginous cap of the forming osteochondroma does not permit Ihh diffusion, the only surrounding perichondrial cells that receive this Ihh signal are those immediately adjacent to the chondrocytes beginning hypertrophy, which express Ihh directly. In this way, the cartilaginous cap of an osteochondroma—although often compared to a normal physis directed peripherally—will never match the morphology of the normal physis. It will never form a surrounding, constraining bone collar. The surrounding lip of cortical bone that forms around the stalk of the osteochondroma will not catch up with the chondrocytic proliferation in the cap until cessation of growth at skeletal maturity (see Figure 5D). This effect may create the mushroom appearance of the typical, pedunculated osteochondroma.

While loss of Ihh function and the resultant effects on the bone collar may be critical to the formation of an osteochondroma, unless it is true that only Ihh diffusion and not signaling are disrupted by loss of HSPGs, the apparent overproliferation of the clone chondrocytes has not yet been explained by loss of EXT function. However, Ihh is not the sole regulator of proliferation in the physis. Some researchers have recently argued that the Ihh signaling pathways may indeed be downstream, at least in part, from signaling through the fibroblast growth factor receptor 3 (FGF-R3)[8,53].

FGF-R3 signaling Pathway

Many fibroblast growth factor (FGF) signaling pathways are highly mitogenic. However, FGF signaling to proliferating chondrocytes elicits an anti-proliferative,

pro-differentiation response by inducing FGF-R3 signal transduction[44,53]. This effect is the opposite of the effects from Ihh mitogenic signaling and the PTHrP feedback loop. FGF-R3 signaling may be upstream of Ihh expression, as FGF-R3 may control the initiation of hypertrophy, which begin Ihh expression. Ihh expression by the pre-hypertrophic cells [6] that have already exited the cell cycle may represent the beginning of negative feedback loop balanced against FGF-R3 initiation of hypertrophy[8,53].

FGF-R3 causes cell cycle exit by activating the transcription factor STAT1, which induces expression of p21[63]. After binding with cyclin E and cyclin-dependent kinase 2, p21 increases the concentration of Rb (retinoblastoma) relative to its phosphorylated counterpart, forcing cell cycle arrest at the G1-S checkpoint[2]. This effect is best illustrated by the autosomal dominant, gain of function mutations in FGF-R3 which generate the dwarfing chondrodysplasias: achondroplasia, hypochondroplasia, and thanatophoric dysplasia[5,45,51,52,57,64,65,70,71]. Alternatively, the opposite effect is seen in FGFR-3 null mice, which exhibit skeletal overgrowth[13,16].

Crucial to its relation to EXT deficiency, FGF-R3 signaling has been demonstrated to be exquisitely dependent on HSPGs[3,12]. Therefore, a clone of EXT-null chondrocytes is equivalent to a clone of FGF-R3 null chondrocytes with regard to FGF signaling; the chondrocytes will be freed from a powerful negative control on proliferation. This lost signaling increases the proliferation and postpones the differentiation of the EXT-null clone of chondrocytes, which, associated with the bone collar defect caused by blocked Ihh diffusion to the perichondrial groove of Ranvier, may begin the outpouching that ultimately yields an osteochondroma (see Figures 5A and 5B).

DISCUSSION

The plausibility of a theory for pathogenesis of an osteochondroma depends on its adherence to all available data regarding the implicated pathways involved and its ability to explain the character of the resulting lesion. Both Ihh diffusion and FGF-R3 signal transduction are demonstrably dependent on HSPGs present at the cell surface. Loss of EXT function results in HSPG synthesis deficiency. The proliferative results of lost FGF-R3 signaling and the focal absence of the bone collar from disrupted Ihh diffusion to the perichondrium have each been shown independently, as discussed above. While other signaling pathways are undoubtedly affected by loss of HSPGs in a clone of chondrocytes, the predictable effects on these two pathways form a theory for osteochondroma pathogenesis which fits all the available evidence.

In addition, this pathogenetic theory explains some of the characteristics of an osteochondroma. First, means for the creation of the resultant structural morphology of an osteochondroma are provided by the theory. The radiographic diagnosis of an osteochondroma depends upon demonstration of cortical and medullary continuity. In this theory, the osteochondroma cap forms from chondrocytes proliferating in the peripheral direction, but from the otherwise normal proliferative zone of the physis (see Figure 5D). Therefore the fronts of chondrocyte differentiation, hypertrophy, and apoptosis, and subsequent primary spongiosal ossification are in continuity with those in the rest of the physis. In addition, the focal distruption of the bone collar, which is then restored epiphyseally once the next wildtype clone of cells moves through the proliferative zone, provides a mechanism for the diagnostically important cortical continuity.

A final feature of the osteochondroma which any pathogenetic theory must address, is that of malignant degeneration into a surface chondrosarcoma. This was once thought to happen in up to 25 percent of patients with HME[37]. While that figure has more recently been reduced to 0.5 to 3%[21,55,69,72], the resulting surface chondrosarcomas do make up approximately one sixth of all the chondrosarcomas in humans[60]. While this theory for osteochondroma pathogenesis does not entirely explain malignant degeneration, it explains a scenario which is predisposed toward accumulation of genetic mutations; this would, at a certain frequency, lead to malignant degeneration.

In a classic parallel, familial adenomatous polyposis leads to colorectal cancer because a given population of cells accumulates somatic mutations while undergoing more than the usual number of cell cycles prior to terminal differentiation[67]. This theory of osteochondroma pathogenesis includes an extension of chondrocyte generations from a single clone due to disrupted FGF-R3 signaling. This necessarily increases risk for accumulating important malignant somatic mutations by genomic replication error alone. However, in contrast to the case of familial adenomatous polyposis, the osteochondroma cells are not intrinsically immortalized by the EXT mutations in this theory. Instead, they are freed from a potent extracellular restraint to cell cycling. Once the extracellular restraint of FGF-R3 signaling is removed, extended cell cycle iteration is permitted and with it, the increased stochastic likelihood of other proneoplastic mutations. Thus, the putative tumor suppressor function of EXT genes is not only indirect, but only enables oncogenesis in a specific milieu of extracellular signals.

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COMPLETE ARCUATE FORAMEN PRECLUDING C1 LATERAL MASS SCREW FIXATION IN A PATIENT WITH RHEUMATOID ARTHRITIS: CASE REPORT

Michael J. Huang, MD and John A. Glaser, MD

ABSTRACT

Case report of a complete arcuate foramen in a human atlas vertebra inhibiting the placement of lateral mass screw instrumentation at C1.

Our objective is to report the presentation of the case, the operative considerations, and the management for this anatomic variation.

The groove for the vertebral artery on the posterolateral surface of the atlas (C1) varies in size and depth from a slight impression to a clear sulcus. With anomalous ossification the sulcus can be bridged which results in a posterolateral tunnel within the posterior arch of the atlas. With increasing rates of screw fixation instrumentation that include the atlas, it is of paramount importance to know the location and course of the vertebral artery in relation to the planned route of instrumentation.

The patient underwent a posterolateral fusion from C1 to C4 using autogenous iliac crest bone graft. Internal fixation from C2 to C4 was obtained using lateral mass screw instrumentation. After the vertebral artery was identified passing through the posterior arch of C1, sublaminar wires were utilized for fixation from C1 to C2. The patient responded well to surgical intervention without complications.

Abnormal vertebral artery coursing through a posterolateral tunnel in the posterior arch of C1 has been described and its incidence has a range from 1.14% to 18%. When this variant is present, lateral mass screw fixation at C1 may be contraindicated. We recommend close scrutiny of preoperative radiographs to avoid the possibility of endangering the vertebral artery when this situation exists.

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PRECIS

This is a case report of a patient with rheumatoid arthritis and cervical instability requiring surgical intervention found to have a complete arcuate foramen within the posterior arch of C1 through which the vertebral artery coursed. The clinical presentation, operative considerations, and surgical management are outlined.

INTRODUCTION

The posterolateral margin of the atlas contains a sulcus or groove for the vertebral artery which can vary in size and depth.[1] This groove can be bridged by anomalous ossification and a posterior ponticulus (Latin for bridge). The opening in the posterior arch of the atlas is termed the arcuate foramen, through which pass the vertebral artery and first cervical nerve. This foramen has been known by many names, but most frequently by the eponym "Kimmerle's anomaly" since Kimmerle was an early describer of this structure.[2] Other terms appear in the anatomy literature to describe the same structure include: "foramen sagittale", "foramen atlantoideum", "foramen retroarticulare superior", "canalis vertebralis", "retrocondylar vertebral artery". In addition to its anatomic significance, the arcuate foramen has been postulated to play a role in clinically relevant entities such as migraines and vertebrobasilar artery stroke.[3,4] The incidence of the arcuate foramen range from 1.14% to 18% depending on the study.[1,4-8] Previous studies vary in study design (radiographic vs. cadaveric analysis), population studied, and grouping of the various types of arcuate foramen phenotypes.

If surgical management is performed, stabilization of the C1-C2 joint is typically accomplished through reduction and fusion of the atlantoaxial complex with internal fixation through a posterior approach. The type of internal fixation varies from wiring procedures such as the Brooks and Gallie techniques to Magerl's transarticular screw technique.[9-12] More recently, Harms and Melcher have published a posterior C1-C2 fusion using polyaxial screw fixation.[13] Biomechanically, screw fixation has been shown to be superior to posterior wiring.[14-19] In addition, fusion rates using screw fixation are also improved over wiring.[20-26] Although screw fixation has a biomechanical advantage and superior fusion rates compared to wire fixation, the technique is more de-

manding and carries greater risk of injuring the vertebral artery. When the vertebral artery courses above the posterior arch of C1, the placement of lateral mass screws is relatively safe[27]; however, the risk may increase significantly with any anomalous course of the vertebral artery. Indeed, the above-mentioned arcuate foramen would place the vertebral artery in the path of any C1 lateral mass screw.

This report describes a case of an anomalous vertebral artery coursing through the posterior arch of the atlas that precluded C1 lateral mass screw fixation in a patient with rheumatoid arthritis undergoing reduction and internal fixation of her cervical spine.

CASE

The patient is a 67-year-old female with rheumatoid arthritis who presented with complaints of neck pain with radicular symptoms into both upper extremities. Her pain was located posteriorly along the cervical spine with radiation up to the occiput and down into the shoulders bilaterally. She had been plagued by this pain for one year prior to presentation with significant worsening over the two previous months. She also complained of numbness in her hands in a glove-like distribution extending just proximal to the wrists bilaterally. She had difficulty buttoning buttons, holding coffee cups, and writing. Her pain and numbness persisted despite chiropractic treatment, physical therapy, and multiple narcotic medications. Her medical history included hypothyroidism and osteoporosis. Her surgical history included a right total hip arthroplasty in 1986, with a revision in 1997, a total abdominal hysterectomy and bilateral salpingoophrectomy in 1975, an L5-S1 decompression and fusion in 2000, appendectomy, and cholecystectomy. Physical examination revealed full range of motion of the neck with pain elicited on extension and lateral bending. She had a normal gait. There was 4 out of 5 strength in all muscle groups in the left upper extremity with numbness of both hands in a glove-like distribution extending just proximal to the wrist. Examination of the hands revealed mild metacarpo-phalangeal swelling diffusely and symmetrical ulnar drift of the digits bilaterally. Deep tendon reflexes in both upper and lower extremities were normal and symmetrical with no pathologic reflexes. Radiographs revealed diffuse cervical spine degeneration with notable C1-2, C2-3 anterolisthesis. The anterior atlanto-dens interval measured 8mm and the posterior atlanto-dens interval measured 15mm in flexion with near anatomic correction in extension. There were no signs of cranial settling. Further imaging with CT and MRI scans confirmed these findings.

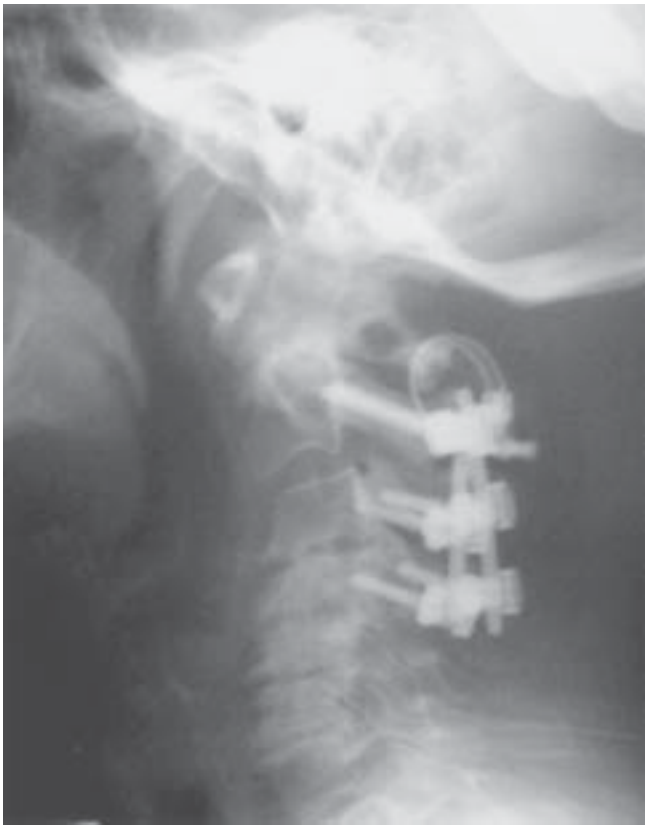

Figure 1. Postoperative lateral radiograph demonstrating a complete arcuate foramen in the posterior arch of C1 precluding facile screw placement.

There were many factors that prompted the decision to offer operative management to this patient. The major concern was quality of life. In her condition at presentation, the cervical instability caused intractable pain and was associated with progressive myelopathic symptoms involving her hands

The patient underwent Halo vest placement one day prior to surgery. Once the halo vest was placed, the patient's cervical spine was positioned so that the anterior atlanto-dens interval was less than four millimeters. Radiographs were taken to verify proper positioning. On the following day, the patient underwent C1 to C4 posterior arthrodesis utilizing autogenous iliac crest bone graft. After the patient was placed prone, the back of the halo vest was removed while leaving the front of the brace intact to maintain adequate alignment of the cervical spine. Segmental instrumentation was achieved using lateral mass screw fixation at the C2, C3, and C4 levels bilaterally. When the posterior arch of C1 was approached, the vertebral artery was found to enter the lateral aspect of C1 rather than coursing along its superior aspect. In order to avoid placing a C1 lateral mass

screw through the vertebral artery, lateral mass screw fixation was abandoned and substituted with sublaminar wire placement from C1 to C2 using the Brooks technique to achieve fixation at this level. The postoperative course was uneventful, and the patient was discharged home on postoperative day nine and managed with halo vest brace immobilization. Two months after surgery, the patient is doing well. She reports mild neck pain much improved compared to her preoperative status as well as mild hand pain bilaterally. She has noticed a significant improvement in the ability to use her hands for fine motor tasks in addition to a decrease in her hand numbness. Radiographs at this time show intact instrumentation and well-maintained correction of her cervical spine.

DISCUSSION

A review of the literature revealed multiple studies with differing results regarding incidence of the arcuate foramen. These results are further confounded by the differing types of studies, populations studied, and differing groupings of foramen types. Pyo and Lowman[8] found an incidence of 38 (12.67%) partial and complete foramen in 300 patients while Dugdale[7] found 47 complete and 37 partial foramen in 316 patients. Kendrick and Biggs[5] had a 15.8% combined incidence in 353 white children (ages 6-17) with a 5.49% and 9.15% incidence of complete and partial foramina, respectively. Stubbs[6] found a complete foramen in 13.5% and a partial foramen in 5.2% of the study population (n=1000). More recently however, a cadaveric study performed by Hasan et al.[1] on 350 dried macerated north Indian atlas vertebrae only reported a 3.42% incidence of complete foramen and a 1.14% incidence of a posterolateral tunnel. This study made a distinction between a complete posterior ponticulus and a more extensive posterolateral tunnel-like canal.

Screw fixation of C1 is evolving into newer techniques such as the one described recently by Harms and Melcher using C1-C2 polyaxial screws to achieve fixation.[13] This case illustrates the clinical relevance of a posterolateral tunnel in the posterior arch of C1 carrying the vertebral artery. When this abnormality is found, screw fixation through the lateral mass of C1 is not feasible and other modalities of internal fixation must be pursued. In our case, we found the abnormal vertebral artery entering the lateral aspect of C1 intra-operatively and opted to obtain C1-C2 fixation with sublaminar wires. However, in retrospect, the pre-operative lateral radiograph did demonstrate the posterolateral tunnel. Careful analysis could have prevented this near complication.

CONCLUSION

Abnormal vertebral artery coursing through a posterolateral tunnel in the posterior arch of C1 has been described and its incidence has a range from 1.14% to 18%. When this variant is present, lateral mass screw fixation at C1 may be contra-indicated. We recommend close scrutiny of pre-operative plain films to avoid the possibility of endangering the vertebral artery when this situation exists.

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ANEURYSMAL BONE CYST FOLLOWING TIBIAL FRACTURE: A CASE REPORT

Geoffrey F. Haft, M.D., and Joseph A. Buckwalter, M.D.

ABSTRACT

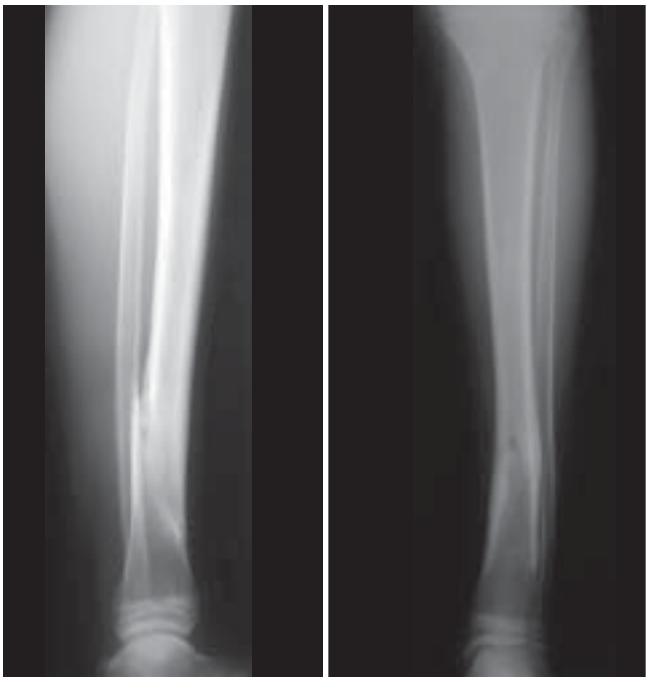
A 14 year-old boy developed a tibial aneurysmal bone cyst (ABC) following a closed tibia fracture. The tumor formed in a site remote from the fracture and was not radiographically apparent until one year following the traumatic event. Most ABC's present due to fracture or pain, but this lesion was discovered during routine follow up films of the tibial shaft fracture. This case lends support to the debated theory that ABC's are reactive bone lesions and provides a rare radiographic glimpse at the lesion's early rate of development.

INTRODUCTION

The aneurysmal bone cyst (ABC) is a benign solitary osteolytic and expansile bone tumor. It is extremely rare, occurring in 1.5 per one million persons per year[11]. It most commonly occurs in the metaphysis of long bones during the first two decades of life and usually presents due to pain, swelling, or fracture[2]. The etiology of the ABC is unknown but numerous authors have proposed that it forms in response to vascular disruption in the bone due to a preexisting primary tumor or a traumatic insult[1,3,4,12,13]. The early rate of growth of the ABC is also unknown. This case lends support to the theory that ABC's are reactive bone lesions and provides a rare glimpse at the early radiographic appearance of the lesion before a patient becomes symptomatic.

CASE REPORT

A 14 year-old healthy eighth grade boy suffered a closed left tibial shaft fracture at the junction of the middle and distal thirds in September of 2001 while playing football. There was no radiographic evidence of pathologic fracture. He was treated with closed reduction and long leg casting followed by functional bracing. The fracture healed uneventfully and by February of 2002, the fracture had healed radiographically (Figures 1a,b) and he had returned to all activities without any pain or limitations. During his routine final follow up visit in November of 2002, the fracture remained well healed, and he remained asymptomatic. However, the new x-rays revealed an eccentric osteolytic lesion in the proximal lateral tibial metaphysis (Figures 2a,b). The lesion is demonstrated well by CT scan (Figure 3). The patient was non-tender over this area.

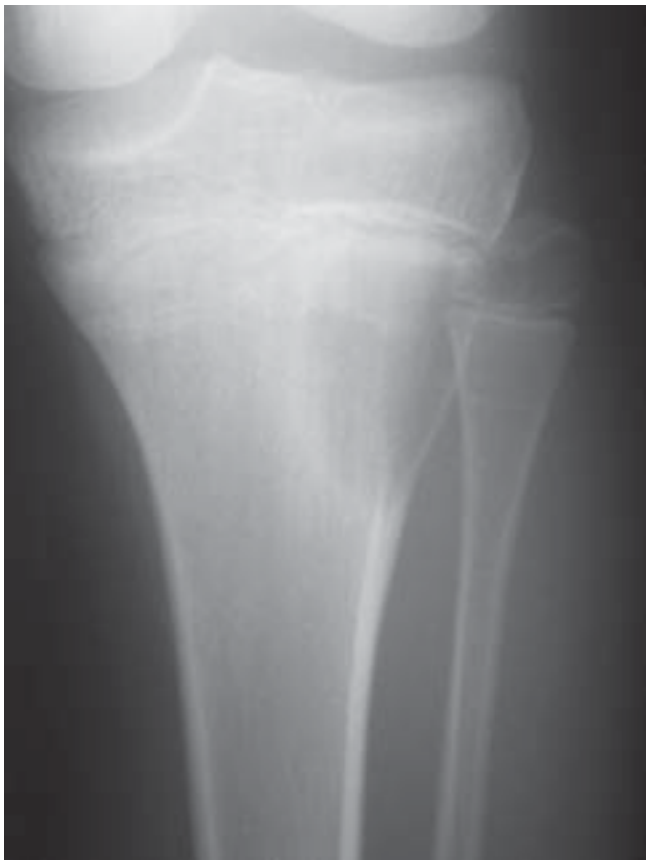


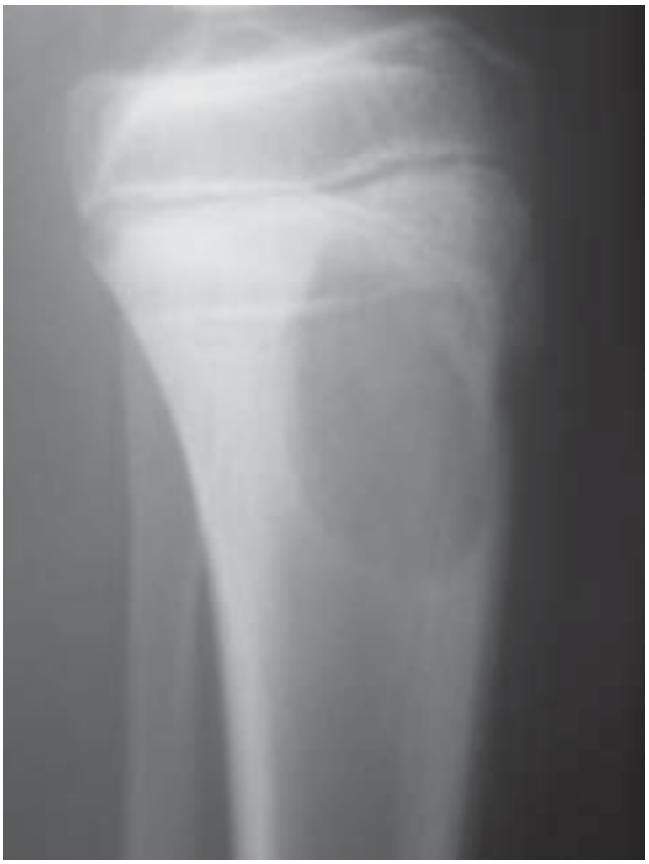
Figures 1a and 1b: AP and lateral x-rays of the tibia demonstrate a well healed distal tibial shaft fracture five months after injury. There is no sign of a lytic lesion in the proximal metaphysis.

After discussion with the family and patient, we performed an open biopsy. Frozen and permanent sections had all of the typical characteristics of an ABC. No evidence of a coexisting lesion was seen. Sections demonstrated multiloculated cyst-like walls without endothelial lining. Bands of cellular tissue separated fragments of bone and cartilage. This cellular tissue was composed of multinucleated giant cells and reactive fibroblasts showing moderate mitotic activity but no nuclear atypia. The lesion was thoroughly curettaged, soaked with ethanol, and then filled with a calcium sulfate paste. The patient recovered quickly and returned to all activities without restriction within six weeks of surgery.

DISCUSSION

Jaffe and Lichtenstein first described the ABC as a distinct pathologic lesion in 1942[9]. While initially thought the be an isolated primary tumor of bone, evidence





Figures 2a and 2b: AP and lateral x-rays one year after injury demonstrate an eccentric, geographic lytic lesion in the proximal lateral tibial metaphysis.

mounted over the ensuing three decades that suggested many ABC's are reactive lesions, caused by the presence of a preexisting bone tumor. Biesecker et al. observed 32% of their cases associated with other benign lesions of bone[1]. Mirra noted 40% of his cases with similar associations[13]. Others have reproduced these observations[3,4,5,12].

The pathogenesis of the ABC continues to be debated but many consider it to be a kind of arteriovenous malformation within the bone, likely caused by a previous or preexisting insult to the bone. This theory was supported by Biesecker et al.'s work in which they measured the intralesional pressure within three ABC's to be increased in the range of typically found in arteriovenous malformations[1]. The lead author wrote:

> Because ABC's are frequently accompanied by associated lesions and because these associated lesions are rarely accompanied by ABC's, it is probable that ABC's are secondary to the associated lesions of bone . . . Therefore, the inaugural event of the genesis of ABC's most likely is an antecedent, primary lesion of bone. The next step in the pathophysiologic development of an ABC is probably the production of an abnormal vascular component by the precursor lesion of bone . . . an arteriovenous fistula.

A few case reports in the last decade lend further support to Biesecker's theory. They have confirmed the presence of a posttraumatic ABC following an injury in which initial x-rays show no suggestion of a bone lesion. In one case, an ABC developed at the fracture site in the proximal tibia within six months of the injury[15]. In two cases, the lesions appeared on the proximal tibia following anterior cruciate ligament rupture and reconstruction[7,18]. In another case, the frontal bone of a soccer player developed an ABC, presumably after repetitive trauma from heading the ball[17]. In all of these cases, the tumor appeared at the local site of injury, suggesting that the injury led to the formation of the lesion.

In a new twist, the current case documents the appearance of an ABC at a site remote from the injury in the tibia. Given the rare nature of ABC's and the temporal relationship, it seems probable that this lesion was secondary to recent fracture. Again, this case provides support for the notion that the tumor is a reactive le-

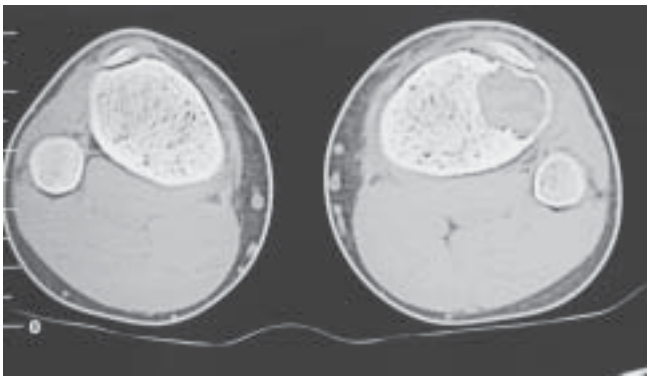


Figure 3: CT scan of the lesion one year after injury.

sion from an antecedent insult to the bone and its vascular supply.

This case also suggests a timeline for pathogenesis of the posttraumatic ABC. In this patient, the lesion developed sometime between six months and one year after the injury. In other case reports, the lesions were noted between three months and one year after injury. If the cells making up the lesion are indeed reparative tissues responding to the primary injury, it makes sense that the ABC would take several months to become mature and radiographically apparent.

While the circumstantial evidence above makes Biesecker's theory very compelling, it does not explain the etiology of the greater than 50% of ABC's with no apparent associated lesion. There have been a few case reports of a multiple ABC's within a single family suggesting a genetic cause or predisposition[8,10]. These cases may eventually prove to be the key to the puzzle. A genetic predisposition could explain why most of these apparent arteriovenous malformations of bone arise denovo, while other ABC's seem to require a major inciting insult to the bone. As with many tumors, modern genetics may eventually confirm or debunk our etiologic theories that have historically been based on strong reason but limited scientific evidence.

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AN ASSESSMENT OF THE BIOLOGICAL FIXATION OF A RETRIEVED MAYO FEMORAL COMPONENT

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ABSTRACT

Implant analysis was conducted on a retrieved Mayo femoral prosthesis that has a non-anatomic design with two distinct surfaces intended for osseous fixation. The prosthesis itself has a circumferential grit-blasted region interposed with the porous surfaces and involving the entire proximal stem. In addition, there are pads of mesh porous coating on the anterior, posterior, and medial surfaces proximally. A single, radiographically stable stem retrieved after 18 months secondary to recurrent dislocations was examined for osseous fixation by measurement of trabecular bone into the porous-coated and onto the grit-blasted surfaces of the stem. Results showed 17% ± 7% bone ingrowth into the porous coated areas and 20% ± 16% osseointegration onto the grit-blasted surfaces. These results are similar to percentages of ingrowth seen with more conventional, anatomic design prostheses.

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INTRODUCTION

The Mayo® Conservative (Zimmer, Warsaw, IN) femoral prosthesis for total hip replacement surgery is a marked departure from most cementless femoral components in design, shape, and methods for achieving bony or biological fixation (Figure 1). This femoral stem design has a non-anatomic shape with a distinctive profile described as a "double-wedged taper" that makes it unique.[15,16] Initial stability of this stem results from its unique geometric shape designed for three-point contact in the proximal femur.[12] The design provides proximal fixation of the implant without compromising bone distal to the lesser trochanter and, because of its higher neck-cut, more bone is preserved than in more conventional designs. Bone conservation is considered particularly important for younger patients undergoing primary total hip arthroplasty because of the likelihood of the need for future revision surgery.

Figure 1. Photograph of a Mayo® Conservative femoral component (Permission of Zimmer Corporation, Warsaw, IN)

For long-term fixation, the stem has a proximal circumferential region designed to enhance osseous fixation. This region differs from what is found on most femoral stems because a mesh porous coating is integrated with a grit-blasted region in attempt to maximize osseous fixation. The porous coated region consists of titanium fiber-mesh pads on the anterior, posterior and medial surfaces with a pore size averaging 80 microns (range, 4-250 microns). Additionally all proximal surfaces not treated with porous coating had been roughened or "corrundemized" by circumferential grit blasting with a twenty-four grit aluminum oxide. The grit-blasting provides a roughened surface (Ra 3-5μm) that is intended to enhance osseointegration or the direct contact of bone to the prosthetic surface.[11,14] In a series of 159 hips with a mean age of 50.8 years and an average six years follow-up,

the mechanical failure rate was 1.8% for this stem.[16] Thus, this implant has short-term results comparable to more conventional cemented and uncemented femoral components with regard to mechanical fixation.

Long-term mechanical success of cementless femoral components has been equated with bony fixation. Therefore, the majority of prosthetic designs have historically had an anatomic design to achieve stability by promoting bone ingrowth through maximizing prosthetic contact with endostial bone. Cementless femoral components with non-anatomic designs have met with varied success.[12] Regardless of stem design, clinical follow-up and implant retrieval analysis studies have suggested that differences between long-term clinical success and failure are due to the amount of biological fixation *in vivo*.[1,7-9,13]

The purpose of this case study was to measure the percentage of mature trabecular bone into the porous coated (ingrowth) and onto the grit-blasted surfaces (osseointegration) in a well-fixed stem of this non-anatomic design obtained at revision for dislocation after 18 months *in situ*.

CASE REPORT

The patient is a 44-year-old Caucasian female with a 10-year history of idiopathic osteonecrosis (Ficat IV) involving both femoral heads and culminating in bilateral total hip arthroplasties. Ten years prior to her left (index) total hip arthroplasty, the patient sustained a diaphyseal fracture of the left femur for which she had a first generation intramedullary nailing. As the femur healed it shortened and the distal segment rotated externally, and the rod was removed three years later because of pain where the rod had become prominent secondary to femoral shortening.

The left total hip replacement was done five years after the intramedullary rod's removal and two years after primary total hip arthroplasty on the right. The implants used were a Trilogy® acetabular component (Zimmer, Warsaw, IN) and a Mayo® Conservative femoral stem (Zimmer, Warsaw, IN) with a 22-millimeter diameter femoral head and long neck. A direct-lateral approach to the hip was used. The patient had an uneventful postoperative course, participating in a progressive weight-bearing regimen over six weeks. Shortly after beginning full weight bearing she experienced the first of eight dislocations, which continued over the next 12 months. Prior to each of the dislocations, the patient reported an episode of hip flexion beyond 90 degrees. All dislocations were closed reduced easily with hip flexion and longitudinal traction, under light sedation. The frequent dislocations were unsuccessfully treated with prolonged recumbence and bracing.

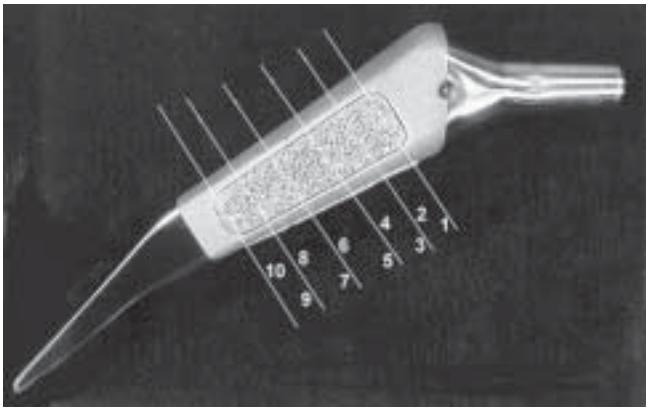

Figure 2. Photograph depicts the location of the cuts across the femoral stem providing 10 distinct levels or sections for microscopic examination of bone ingrowth and osseointegration.

At the time of implantation, the stem was unintentionally positioned in slight varus from the desired orientation. In addition, due to the prior femoral fracture and malunion, her femur was noted to have diminished anteversion. At 18 months after the index procedure, the patient had no hip pain, and, although the stem appeared radiographically stable without obvious mechanical impingement, it was felt that revision surgery was necessary to alleviate the problem of recurrent dislocations. During the revision operation it appeared that the prosthesis was appropriately placed and centralized in the proximal femur. The Mayo femoral stem was removed easily with osteotomes and revised without difficulty to an SROM® (DePuy - Johnson & Johnson, Warsaw, IN) in order to increase the femoral anteversion and to prevent future dislocation.

METHODS

At retrieval the stem was placed in 10% aqueous formalin and then fixed in 70 per cent ethanol, dehydrated in ascending grades of ethanol, infiltrated, and embedded in methylmethacrylate.[18] Once the specimen was polymerized the entire area intended for osseous fixation was serially cut perpendicular to the axis of the stem into three-millimeter thick sections using a custom, water cooled, high-speed, cut-off saw[4] (Figure 2). The sections were numbered serially from one to 10, proximal to distal, with sides A (cranial) and B (caudal) designated. These sections were ground and polished to an optical finish using a variable-speed grinding wheel (Buehler Incorporated, Lake Bluff, IL) and then sputter-coated (Hummer VI-A; Anatech Limited, Alexandria, VA) with a conductive layer of gold for approximately one minute to facilitate backscattered electron (BSE) imaging. The sections were then examined in a scanning electron microscope (SEM) (JSM 6100; JEOL Incorporated, Peabody, MA) using a BSE detec-

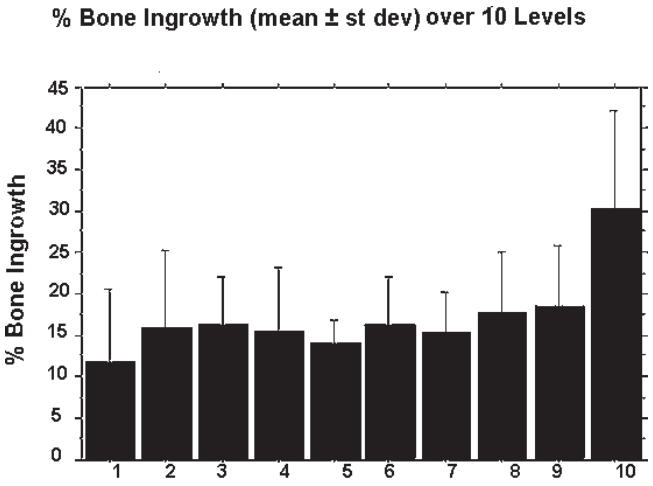

Figure 3. This bar chart demonstrates the percentage of bone ingrowth into the porous coating at each of the 10 levels.

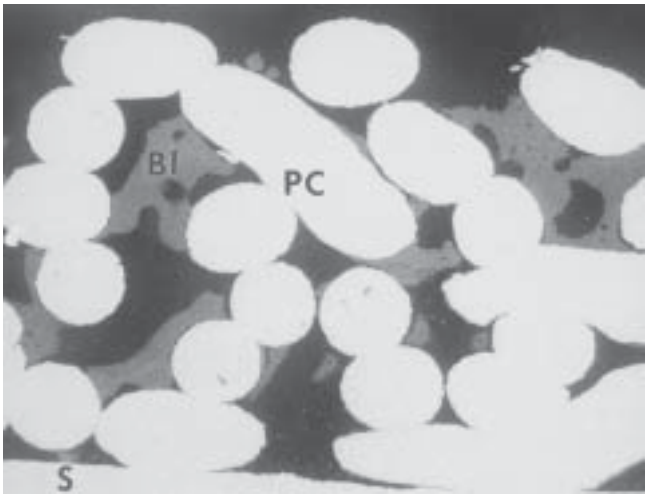

Figure 4. Backscattered electron photomicrograph X50 magnification of bone ingrowth (BI) into the porous coating (PC) at level 9, substrate (S).

tor (Tetra, Oxford Instruments Ltd., Buckinghamshire, UK). The entire porous coated interface and grit-blasted surfaces were imaged using a Kalman frame averaging technique to increase the signal to noise ratio using a computer-controlled image capture and retrieval system (ISIS 300 series, Oxford Instruments, Ltd., Buckinghamshire, UK). The images were analyzed over ten levels using a semi-automated image analysis system (IMQUANT, Oxford Instruments Ltd., Buckinghamshire, UK). The operating conditions of the SEM were: 15 millimeters working distance, 20 kilovolts accelerating voltage, 70 micron aperture setting, and 2.5 nanoampere probe current. Digital BSE images were recorded at varying magnifications.[2,3,5,6,19]

The percentage of bone ingrowth was determined by calculation of the actual amount of bone volume divided by the total volume of available pore space over all the sections of the femoral stem. Similarly, the percentage of bone that had osseointegrated with the grit blasted surface was measured and reported as a percentage of the total available bone contact along the entire grit blasted surface. Standard deviations of percentage calculations are reported.

RESULTS

The average percentage ingrowth was calculated as 17% (± 7 %) from the 10 sections or levels involving the stem's entire porous coated surface (Figure 3). Nine of the 10 levels ranged between 12% and 19% while the tenth and most distal level measured 30% (±12%). The percent of bone ingrowth was calculated for each of the three porous coated regions or pads on the femoral stem (anterior, posterior, and medial). The anterior pad measured 17% (± 8%) ingrowth, posterior 16% (±9%) and medial 18% (±9%). The electron microscopic evaluation of the sections showed that the bone that traversed into the porous coating was mature trabecular bone with osteocytes and lacunae (Figure 4).

The percent osseointegration along the grit-blasted region of the entire surface available for osseointegration for the 10 levels or sections averaged 20% (±16%) (Figure 5). Osseointegration was greatest at levels 7 and 8 and least at the most proximal and most distal levels. Backscattered electron imaging analysis suggested that the osseous layer in the porous coated regions consisted of mature cancellous bone (Figure 6).

DISCUSSION

The data from this report indicates that the Mayo® Conservative femoral stem design has the ability to achieve bone ingrowth (17± 7%) and osseointegration (20 ± 16 %). The amount of bone ingrowth measured in the fiber-mesh pads on the anterior, posterior and medial surfaces of this single femoral stem was roughly similar to the amount of bone ingrowth observed in previously reported autopsy implant retrieval studies. Engh et. al.[10] measured 30% bone ingrowth in three proximally coated and 37% in five extensively coated well-functioning AML (Depuy-Johnson and Johnson, Warsaw, IN) femoral prostheses. On the acetabular side, Pidhorz et al.[17] measured 12.1 ± 8.2% ingrowth in 10 of 11 Harris-Galante uncemented acetabular components, and Bloebaum et al.[5] measured 12 ± 6% bone ingrowth in seven anatomical porous replacement acetabular components (APR, Sulzer Orthopedics, Austin TX). In ad-

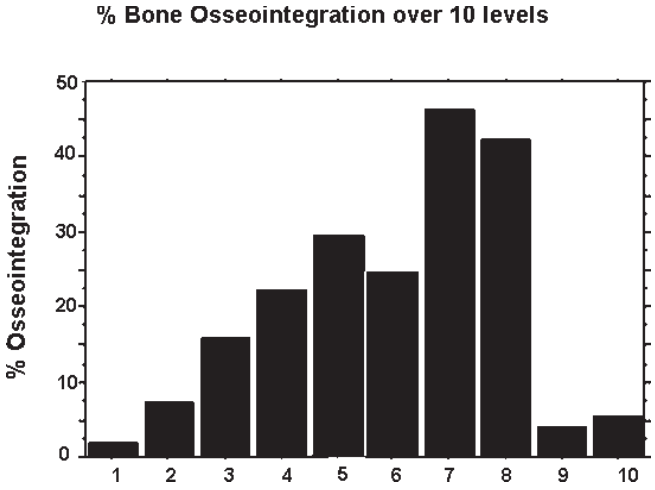

Figure 5. This bar chart demonstrates the percentage of osseointegration within the grit-blasted surface for each of the 10 levels examined.

Figure 6. Backscattered electron photomicrograph at X50 magnification of bone osseointegrated (O) with the grit blasted surface of the implant at level 5.

dition, similar percentages of bony ingrowth were measured with the methods employed here in other well functioning (total knee) porous coated prosthetic devices retrieved at autopsy. [2,3]

The percentage of bone osseointegrated with the grit blasted surface (20 ± 16 %) is similar to the percentage of ingrowth found with this stem and appears to provide adequate bony fixation. Variation in the amount of host bone present along the length of the implant may account for the large standard deviation observed in the osseointegration data. The highest percentage of osseointegration was attained distally (Figure 5) and coincides with the areas where the femoral stem was in greatest contact with the endosteal cortex. The data in Figure 5 suggest that the stem appeared to have progressive distal attachment except for levels nine and ten. The exception in levels nine and ten may be related to the increased distance between the cortex and the implant due to the relative varus positioning of the implant.

The most recent follow-up series of the Mayo® prosthesis implanted by a single surgeon includes 159 hips with an average 6.2 year follow-up (range, 2 to 13 years), and 98% of the implants were considered a clinical success.[16] However, the Mayo stem in this clinical series in that the stem in that study had a highly polished surface with only the fiber-mesh pads for bony ingrowth fixation. The grit-blasted surface was added as a second generation design, leaving a roughed titanium surface (Ra 3-5μm) similar to the clinically successful stem (CLS, Protek-Sulzer, Berne, Switzerland), which relies on osseointegration solely as its method for fixation.[17,20]

Though markedly different is its design, shape and methods for bony fixation, the Mayo stem appears to have similar percentages or ingrowth as other conventional cementless stem designs. Additionally, the percentages of ingrowth and osseointegration were similar (Figures 3 and 5), leading to the conclusion that grit blasting as a surface treatment to enhance osseointegration provides a similar skeletal response to porous coating and implies the possibility of a similar clinical success as the prostheses with mesh porous coating alone. The limitation of this study is that it is a single case study in which the implant-bone interface had been compromised due to the extraction of the femoral stem. Although the stem was clinically and radiographically stable, it had been placed in a slight varus position within a femur with diminished anteversion. Regardless, adequate bone ingrowth and osseointegration was achieved in the area available for biological fixation in this implant design. The long-term clinical durability of the stem has yet to be confirmed, nor has long-term stability been established through postmortem retrieval studies. One question raised by this study is whether both methods for bony fixation are needed for clinical success of this stem design. The results of ingrowth and osseointegration were similar in this stem retrieved at 18 months post-implantation. Thus, one could postulate that either grit-blasting or porous coating alone is sufficient to ensure long-term clinical success. On the other hand, the combination may provide greater stability over a longer time period. Future studies, preferably postmortem retrieval analyses, are required to better understand the relationship between the amount of bone osseointegration, stem design, and the amount at host bone at various anatomical regions along the endosteum of the cortical bone.

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THE VIENNA HERITAGE OF IOWA ORTHOPAEDICS

Joseph A. Buckwalter

ABSTRACT

Strong traditions of basic research, clinical innovation, teaching and integrating science and evaluation of outcomes into clinical practice have characterized University of Iowa orthopaedics for ninety years. These traditions were brought to Iowa City from Vienna when Iowa City was a town of fewer than 10,000 people in a sparcely populated rural state. In the last third of the 19th century, surgeons at the University of Vienna, led by Theodore Billroth (1829-1894), helped transform the practice of surgery. They developed new more effective procedures, analyzed the results of their operations, promoted the emergence and growth of surgical specialties and sought understanding of tissue structure, physiology and pathophysiology. Their efforts made Vienna one of the world's most respected centers for operative treatment, basic and clinical research and surgical education. Two individuals who followed Billroth, Eduard Albert (1841-1900) and Adolf Lorenz (1854-1946) focussed their research and clinical practice on orthopaedics. Their successes in the study and treatment of musculoskeletal disorders led one of their students, Arthur Steindler (1878-1959), a 1902 graduate of the Vienna Medical School, to pursue a career in orthopaedics. Following medical school, he worked in Lorenz's orthopaedic clinic until 1907 when he joined John Ridlon (1852-1936) at the Chicago Home for Crippled Children. In 1910, Steindler became Professor of Orthopaedics at the Drake Medical School in Des Moines, Iowa, and, in 1913, John G. Bowman, the President of the University of Iowa, recruited him to establish an orthopaedic clinical and academic program in Iowa City. For the next third of a century he guided the development of the University of Iowa Orthopaedics Department, helped establish the fields of orthopaedic biomechanics and kinesiology and tirelessly stressed the importance of physiology, pathology and assessment of the outcomes of operations. From the legacy of Billroth, Albert and Lorenz, Arthur Steindler created an internationally recognized center for orthopaedic care, research and teaching in Iowa City.

In my conception of scientific work, history and research are so indivisibly linked that I cannot even conceive of one without the other.—Theodore Billroth[26]

INTRODUCTION

For most of the past twenty-five centuries, surgeons who attempted to correct skeletal deformities, improve mobility of patients suffering from stiff dysfunctional joints and treat injuries of the limbs and spine, did so with little or no understanding of structure and function of the musculoskeletal tissues or the pathophysiology of diseases and injuries of the musculoskeletal system[8]. Even in the nineteenth and early twentieth centuries, most orthopaedists took little or no interest in seeking new knowledge that would improve understanding of diseases and injuries or in critically evaluating the results of their operations and manipulations. Although exercise, bracing and gentle manipulations were part of orthopaedic practice in the 1800s, many of the non-operative treatments consisted of applying brute force that corrected deformities or mobilized stiff joints by crushing and rupturing tissues[8,11]; an approach summarized by the motto of a group of European orthopaedists in the early 1800s, "That which cannot be bent must be broken[26]." Operative treatments were bloody, often ill-conceived and frequently complicated by loss of limbs and death[8,11]. Patients who did not exsanguinate during surgery commonly died later of surgical infections. Although most 19th century surgeons had little interest in collecting data on the results of their operations, they knew that opening a joint or exposing a fracture had a much greater risk of leading to an infection than opening the abdomen, bladder or skull. When open reduction and internal fixation of a closed patellar fracture or removal of a loose body from a joint had mortality rates that approached fifty percent, few

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surgeons were eager to perform the procedures, and patients suffering from these disorders, who understood the risk, avoided surgeons if at all possible. Understandably, orthopaedic surgery, the most physical of the healing arts, did not enjoy even the level of respect accorded to other medical specialties.

In the early and mid 1800s, to gain recognition and attract patients, surgeons needed considerable technical skill and unshakable self confidence, if not arrogance and theatrical talent. More than a few possessed these qualities including Robert Liston of London and Samuel Gross of Philadelphia.

ROBERT LISTON (1794-1847)

Before the development of inhalation anesthesia in the 1840s, no patient could tolerate meticulous dissection: speed was the measure of a surgeon's skill—few operations short or long produced good results, but shorter operations caused less pain[8]. Robert Liston, Professor of clinical surgery at London's University College, earned a well deserved reputation as one of England's greatest surgeons for his masterful knowledge of anatomy, willingness to attempt the most difficult and dangerous procedures and displays of dexterity, physical strength, speed and dramatic talent in the operating theater[8]. His operations and lectures attracted students and physicians from throughout the United Kingdom, Europe and America and his papers on surgical procedures were widely read and quoted[29-31]. Liston routinely performed leg amputations in less than three minutes, performances marked by flashing steel and spurting blood[18,38,54]. In his efforts to perform above knee amputations and hip disarticulations with speed and panache, Liston occasionally injured observers who stood too close to the operating table and amputated more than he had originally planned. On at least one occasion, he simultaneously performed an accidental orchiectomy and an intentional above knee amputation. Another of his leg amputations led to a 300% mortality. A distinguished spectator died after he was slashed during the operation, the patient died of sepsis several days later and Liston's assistant died of sepsis as a result of losing several of his fingers during the operation[18].

SAMUEL GROSS (1805-1884)

The rapid adoption of inhalation anesthesia in the late 1840s decreased the need for surgeons to operate with blazing speed, but it almost certainly increased the frequency and number of surgical complications including death[8]. Inhalation anesthesia made surgeons more willing to operate and patients more willing to have an operation. Yet, surgeons did not know how to prevent infections, they had difficulty controlling bleeding during operations and they had no way to restore blood volume in patients who had suffered massive hemorrhage.

Figure 1. The Gross Clinic, Thomas Eakins' 1875 painting showing Samuel Gross, age 70, debriding the femur of a patient with osteomyelitis.

Samuel Gross (Figure 1), the pre-eminent North American surgeon of the mid and late 1800s[19-22], known for his expertise in treating disorders of the extremities, listed the qualities of a good surgeon, " . . . a firm and steady hand, a keen eye, and the most unflinching courage, which can disregard alike the sight of blood and the cries of the patient[22]." He was among the surgeons who believed that a chance to cut is a chance to cure, and no surgeon should pass up a chance to cure. He stressed that " . . . as long as the various tissues of the body are subjected to disease and accident, so long will they require removal by the knife[22]." Gross felt that surgeons had been unnecessarily reluctant to resect diseased or injured joints, but that his encouragement and their "intelligence, zeal and skill[22]" would soon increase the use of the procedure and extend it to every joint in the body.

Gross knew that the complications of joint operations included "excessive suppuration, pyemia, and erysi-

pelas[22]," conditions that he treated with bleeding, leeches, purgatives and blistering[22]. Despite these treatments, pyemia proved " . . . fatal in nine out of ten cases[22]." He regarded these complications as part of the risk of surgical treatment and disparaged Joseph Lister's 1867 report[8,28] that anti-sepsis cut the mortality following elective surgery from forty-five percent to fifteen percent. Nearly ten years after Lister's publication, Gross commented, "Little, if any faith, is placed by any enlightened or experienced surgeon on this side of the Atlantic in the so-called carbolic acid treatment of Professor Lister," and in 1882 he added " . . . demonstration of living, disease-producing germs is wanting[39]." Gross was certain that the increased skill and enthusiasm of surgeons for resecting tissues damaged by disease or accident would insure that surgery would no longer be " . . . regarded merely as a kind of handicraft, fit to be exercised only by men of inferior attainment, ability and skill[22]."

In 1875, Thomas Eakens painted Samuel Gross at age 70 debriding the femur of a patient with osteomyelitis (Figure 1). Gross and his assistants wear their street clothes, splattered with blood and pus, and incise and retract the wound with their unwashed bare hands. The anesthetist sedates the patient with open-drop ether anesthesia. Lighting is poor and unsterile surgical instruments lie scattered where the surgeons can easily grab and discard them. The artist sits in the first row of spectators observing the procedure, making a drawing or taking notes. Other observers stand in an open entry way behind the anesthetist. A woman, possibly the sister, mother or wife of the patient, covers her face and recoils from the dissection and Gross's bloody hand.

Figure 2. Theodore Billroth

THEODORE BILLROTH (1829-1894)

The transformation of surgery from a dangerous, dramatic and often ineffective handicraft into a safe effective method of treatment did not result not from increased zeal and technical skill of surgeons, but from a growing appreciation of the importance of scientific investigation and evaluation of the outcomes of surgical practice. No institution did more to bring about this change than the University of Vienna where the faculty stressed the importance of the sciences as a basis for surgical practice[26]. They contributed to progress in almost every currently recognized surgical specialty and taught physicians from throughout the world, including approximately 10,000 Americans who studied at the University of Vienna between 1870 and 1914[33].

In 1867, recognizing the limitations of surgical practice, the medical faculty of the University of Vienna, demanded that the University appoint a professor of surgery "of whom the greatest promotion of science may be expected, a man who is not only famous in the field of practical surgery, but also in the areas of physiological and pathological research who has demonstrated a special genius as a teacher, a surgeon and a writer, who is still in the prime of his life, from whom it may be expected that he will represent the most modern trends in surgery in relation to physiology and pathological anatomy and who is able to establish a surgical school in Vienna which will bring fame to the University and the greatest benefit to the country[26]." The University of Vienna found a man who exceeded all of these expectations: Theodore Billroth (Figure 2), the first child of Johanna and Karl Theodor Billroth, the pastor of a Lutheran church in Rugen, Germany's largest island in the Baltic Sea.

Billroth's medical education in Gottigen, Berlin and Zurich had focussed on science and surgery[33]. He performed animal experimentation as well as microscopic examination of normal and diseased tissues and was recognized for his work in experimental physiology and

pathology. Billroth enjoyed the study of pathology, but he chose to become a surgeon because "Observation at the sickbed is so much more interesting than microscopy[26]." In 1853, he started working as an assistant to the prominent German surgeon Bernhard Langenbeck (1810-1887)[26]. Although he did not limit himself to orthopaedics, Langenbeck treated patients with osteomyelitis and clubfoot deformities. He performed subcutaneous osteotomies to correct skeletal deformities caused by rickets and ankylosis of the hip and knee and developed the idea of stabilizing femoral neck fractures by driving a nail through the greater trochanter[27]. While working with Langenbeck, Billroth rapidly gained recognition for his talent as a surgeon and investigator and in 1860 was named to the position of Professor of Surgery in Zurich. In 1863 he published his most well known work, *Die allgemeine chirurgische Pathologie und Therapie* (English Edition: General Surgical Pathology and Theraputics. 1871)[4], a book that illustrated the relationships between symptoms and abnormalities in tissues and demonstrated the importance of understanding pathophysiology for surgical practice.

Although he devoted much of his time to science and surgery, Billroth enjoyed history and music[33]. In his study on the history of treatments for gun shot wounds he commented, "Only the man who is familiar with the art and science of the past is competent to aid in its progress in the future[33]." He had initially intended to pursue a career in music and demonstrated talent as a pianist, but his mother convinced him to enter medical school. To some extent his passion for music was fulfilled through his relationship with Johannes Brahms. The two men met in 1865, and began a close friendship that continued until Billroth's death[2]. Brahms arranged to perform many of his works in Billroth's home and dedicated two of his string quartets to Billroth.

Like Joseph Lister, Billroth recognized that as long as more than 40% of patients suffered wound infections or sepsis following technically successful operations, surgeons should only operate to save lives or relieve unbearable pain. Under these conditions, elective operations to improve limb function or correct deformity were difficult to justify and progress in orthopaedic surgical practice was impossible. In 1855 Billroth began a series of investigations directed toward finding the cause of post operative infections. In 1864, the year before Lister first used carbolic acid in a patient with an open fracture to kill the "unseen vital particles" that caused putrification and blood poisoning[8,11,28], Billroth proposed that substances formed in wounds caused fevers and sepsis[26].

When Billroth joined the University of Vienna medical faculty in 1867, he continued his efforts to find the cause of wound infections. In the same year, Lister reported the efficacy of his carbolic acid antiseptic method in a consecutive series of patients[8,28], an event that stimulated Billroth to start new investigations of the role of microorganisms in wound infections and methods of antisepsis. He concluded that Lister's method needed further development, but he started using it in 1878[26]. Because of complications caused by carbolic acid Billroth switched to using iodoform as an antiseptic in the 1880s and in 1891 began the practice of asepsis for surgery.

Samuel Gross watched Billroth perform an operation in 1868 and described him as, "fearless and bold, almost to the point of rashness[33]." Given Gross's evaluation, it is reasonable to assume that Billroth did not lack confidence when he was operating, but he also carefully planned each operation and performed and tested new operations on animals to evaluate their efficacy and refine his techniques[26]. Among his most well-recognized achievements were successful esophageal resection in 1871, laryngectomy in 1873 and gastrectomy, the Billroth I, in 1881[26].

Billroth also had an interest in the pathophysiology and treatment of musculoskeletal diseases and injuries. He studied and treated club foot deformities, developmental dislocations of the hip, sarcomas of the extremities, fractures, malunions, pseudarthroses, bone and joint infections, joint injuries, joint dislocations, joint ankylosis and arthritis deformans (osteoarthritis)[1,4]. His description of the abnormalities of the acetabulum in patients with developmental dysplasia of the hip show that he understood the problem, ". . . not only is the head of the bone out of the socket, but the socket is irregularly formed—too shallow; later in life, in adults, it is greatly compressed and filled with fat." In Billroth's experience, treatment of developmental dislocations of the hip in older individuals was difficult if not impossible[4]. He commented, ". . . when you read in orthopedic pamphlets of the frequent cure of congenital luxations, you may be sure that in most cases there have been errors of diagnosis, or there is intentional deception[4]."

Throughout his career in Vienna, Billroth stressed the importance of the natural sciences in medical education[5], and devoted time to making sure that younger surgeons understood that acquisition of technical surgical skills must be combined with scientific investigations and understanding of physiology and pathology. He also taught that statistical comparisons of the outcomes of different operations should be used to determine which operations were most effective[26].

In 1890, Adelbert Seligmann painted Theodore Billroth at age 61 operating in the Allgemeines

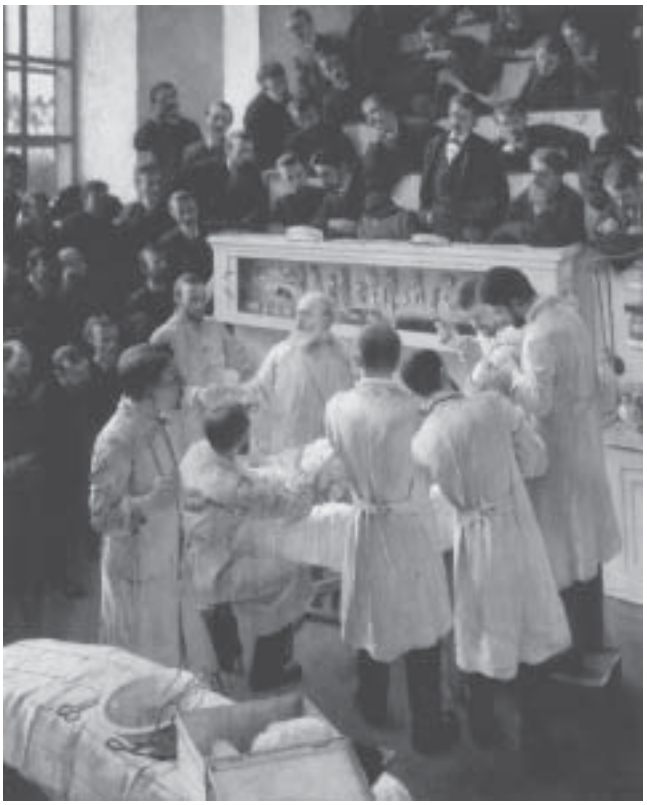

Figure 3. Adelbert Seligmann's 1890 painting showing Theodore Billroth, age 61, operating in the Allgemeines Krankenhous (General Hospital of the University of Vienna).

Krankenhous (General Hospital of the University of Vienna) (Figure 3). Although Seligmann's portrayal of Billroth as a commanding figure surrounded by assistants and spectators resembles Eakins depiction of Gross (Figure 1), there are differences. In Seligmann's painting surgeons and the anesthetist wear frocks resembling modern surgical gowns, the lighting from the windows is excellent and no one covers their face in horror. Billroth's expression and posture suggests that, at least on this day, he approached the performance of an operation with more thought and less drama than Gross, and the scene more closely resembles a current operating room than a theater.

Billroth was a skillful teacher and surgeon, and a dominating leader, a surgical Geheimrat[33]. He was also an unreservedly committed German nationalist and a man of his time, family and culture[1,33]. The decisiveness and confidence that made him a great surgeon may have contributed to his willingness to voice his opinions concerning the importance of establishing and maintaining order in medical education and of the fitness of different students for the study of medicine. In his book, "The Medical Sciences in the German Universities: A Study in the History of Civilization[5]" he expressed concern that " . . . there are many studying at Vienna who are not of the elect and who are trying to force themselves into the medical profession." He was concerned that accommodating these students would "reduce the German method of university instruction to the level to which the lay surgeons were formerly restricted, expressly for the sake of these elements that are piling up in Vienna, or to organize separate schools in which to transform stupid, ignorant, starving students into stupid, ignorant and starving physicians[5]."

Billroth argued that to avoid the degradation of German university medical education the academic hierarchy must be maintained and only the most desirable students should be selected for medical education. He believed that the rules of academic conduct and advancement should be enforced by the state, "no faculty can exist without a definite code for both students and professors" and "it is the duty of the state to see to that such a code exists[5]." Commenting on the aspirations of some individuals for a career in medicine he said, "No profession except, perhaps, the clergy, is so often exploited by uneducated families who aim to climb into the cultured classes on the shoulders of the younger generation, as is the medical profession. For the Jews a medical career offers comparatively fewer difficulties than any other[5]." He also expressed concern over the composition of the medical student body in Vienna, "Among the non-Germans studying in Vienna a lack of money is often coupled with the lack of talent and of decent home training . . ." and "I have repeatedly pointed out that the undesirable elements are not German, but Galacian and Hungarian Jewish elements[5]." Other members of the University of Vienna faculty in the late 1800s and early 1900s may not have been as willing as Billroth to record their views of the Germanic academic hierarchy and the desirability of Jewish students, but it is likely that many of them would have agreed with him. In this environment, students who were "not of the elect" faced multiple barriers and frustrations if they attempted to pursue a career in medicine.

EDUARD ALBERT (1841-1900)

While Billroth achieved widespread recognition for developing methods of resecting the stomach, esophagus and larynx, a younger Viennese surgeon, Eduard Albert (Figure 4) focused his practice and research on joint diseases and skeletal deformities[26,27,32,36]. Albert, a watchmaker's son from Senftenberg, Bohemia, graduated from the Vienna Medical School in 1867, the year that Billroth was named professor of surgery. Following graduation Albert became a disciple of Billroth's

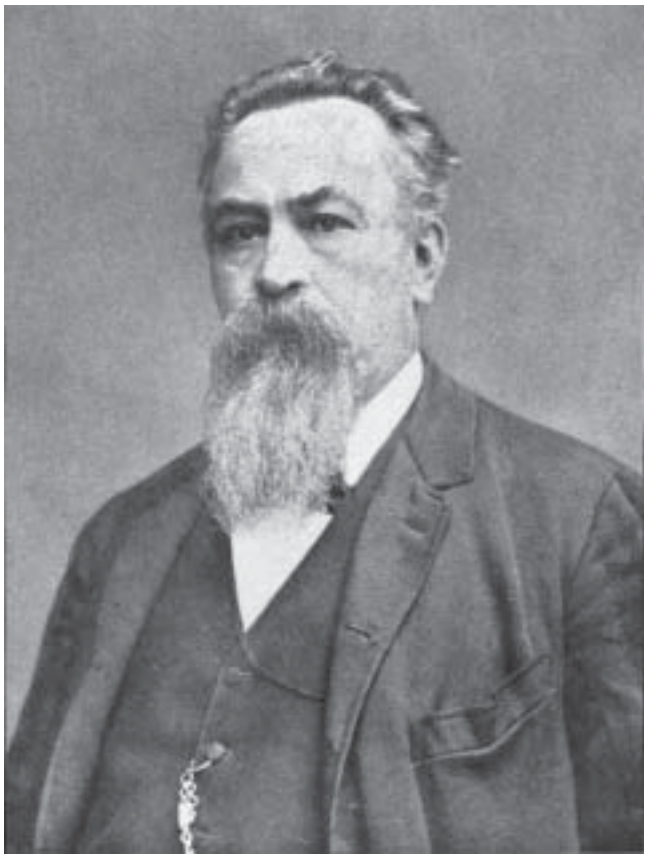

Figure 4. Eduard Albert

arch rival, a prominent aristocrat and surgeon, Johann Heinrich Baron Dumreicher von Osterreicher (1815-1880). Dumreicher, a talented surgeon and excellent teacher, had a strong interest in surgery of the extremities. Unlike Billroth, he believed that surgery should be based on established facts and that introduction of scientific questions did nothing but create confusion. In addition, he had feuded with Billroth's teacher, Langenbeck, so the hostility between Billroth and Dumreicher was intense and long standing. Perhaps for this reason, as well as the desire to advance the career of one of his own disciples, Vincenz Czerny, Billroth bitterly opposed the appointment of Albert as head of the First Department of Surgery at the University of Vienna. Billroth considered it his greatest defeat when Albert was given the position in 1881.

Had Billroth known Albert better he might have considered him a worthy choice for the position of Head of Surgery. Albert showed that he recognized the importance of Billroth's emphasis on scientific investigation, not only through his own work, but in an address praising Billroth's academic contributions in 1892[26]. Albert published the first textbook advocating use of antisepsis to prevent post-operative infections in 1880 and during his time as Head of Surgery he proved to be an enthusiastic investigator who performed basic scientific investigations to advance surgical practice. His clinical experience with joint diseases combined with an interest in mathematics and a unique ability to visualize structures and their relationships in three dimensions led him to study joint mechanics, human movement and the mechanical changes in the spine associated with scoliosis. Albert investigated the structure of the synovial membrane and bone autografts[3,27]. He injected fluid into joints to study the resistance of the capsule and surrounding structures, and conducted a series of studies on the mechanics of the knee joint, the ankle joint, the shoulder girdle and the hip joint[26]. He was especially interested in the hip and described the function of the hip capsule during joint motion, in particular the movement and torsion of the capsular fibers. His studies of the pathology and therapy of coxitis and his manuscripts and lectures describing surgery of the extremities increased interest in operative treatment of joint diseases.

At the time Albert was conducting his studies of joint structure, function, mechanics, and diseases, other surgeons were treating joint diseases, primarily tuberculosis, by joint resection, often to the detriment of their patients. Albert argued for non-operative treatment of joint diseases, except for patients with the most severe joint deformities, instability or pain, or limb paralysis[26]. He proved that these patients could be helped by joint fusion, a procedure that he named arthrodesis[3], and showed that arthrodesis could eliminate bracing and improve function of otherwise useless extremities[3,26,36]. He was best known for performing the first successful shoulder arthrodesis in 1881[27].

ADOLPH LORENZ (1854-1946)

Albert's favorite student, Adolf Lorenz (Figure 5), became one of the most influential orthopaedists of the 20th century[3,27,32,36]. Lorenz, the son of an Austrian innkeeper and harness-maker, moved to Vienna in 1878 to study medicine[32]. To help pay for his medical studies he worked as an assistant in the anatomy department and acquired an encyclopedic knowledge of anatomy and considerable skill in dissection. Following graduation from medical school he sought employment in a surgical clinic. His background in anatomy gave him an advantage over other applicants and he was hired by Dumriecher to work as an assistant surgeon. Within a year, Dumbriecher died and Eduard Albert succeeded him. Lorenz commented later that the only teachers at the University of Vienna who made a lasting impression on him were Billroth and Albert[32].

Figure 5. Adolf Lorenz. Lorenz autographed this portrait in 1908.

Lorenz's ambition, energy and intellect led Albert to appoint Lorenz as a lecturer in surgery at the University in 1884[26]. Lorenz showed considerable talent as a surgeon, and decided that he wanted to become one of the great Viennese surgeons, like Billroth and Albert. However, an unexpected event forced him to temporarily choose another direction. Albert insisted that all surgeons in his department use Lister's carbolic acid antiseptic techniques. Lorenz developed severe eczema when his hands were exposed to carbolic acid and was unable to continue operating in the presence of carbolic acid. Albert advised him, "if you can't get along with wet surgery, why not try dry surgery[26,32]"; dry surgery referred to manipulative and casting treatment of diseased or injured limbs and the spine. Albert took this advice and established the "University Outpatient Department for Orthopaedic Surgery." He became interested in the "bloodless" treatment of children's musculoskeletal disorders including clubfoot deformities, bone and joint tuberculosis, paralytic deformities, congenital dislocation of the hip and scoliosis[26]. His ability to correct a skeletal deformity and maintain the reduction with a plaster dressing led Viennese surgeons and students to call him the "Gipsdozent" or plaster docent, a title that gave him great satisfaction[32].

When the enthusiasm for carbolic acid antisepsis waned and other less toxic antiseptics became accepted, Lorenz started operating again using alcohol as an antiseptic; but, it was his experience with non-operative treatment of skeletal deformities that led to his most well known contributions to orthopaedics. Demonstrating that dangerous operations were unnecessary gave him great pleasure[32]. He showed that his treatment of joint tuberculosis with prolonged plaster immobilization produced better results than traction or joint resection, and he found that plaster bed treatment of spinal tuberculosis improved the general health of the patients and made surgery unnecessary in most cases[32]. He was correct that prolonged immobilization of an entire limb produced better results than joint resection for many patients with joint tuberculosis, but he apparently did not appreciate that this treatment damaged normal joints.

The best recognized of Lorenz's contributions to non-operative treatment was his refinement and promotion of modelling redressment (molding reduction) of growing tissues and structures to correct deformity[36]. Lorenz had learned during his studies with Albert to consider and analyze the effects of mechanical forces on form and function of the musculoskeletal system. He considered it absurd to treat congenital or acquired deformities or loss of motion in such delicate and balanced structures as joints with "redressment force" (brute force), an approach that followed the teaching "if it can't be bent, then it must be broken[26]." Surgeons using the redressement force approach applied whatever force was necessary to correct a deformity, such as a clubfoot, and then fixed the limb in the reduced position with a cast or a similar appliance. As he described it, Lorenz's modelling redressment consisted of hundreds of successive applications of measured and moderate force until the corrected limb showed no tendency to recoil into the deformed position[32]. In his studies of clubfoot, he found that his gentle successive manipulations could overcome even resistant deformities if the surgeon was patient. He declared, "that no clubfoot, either in children or adults, of what ever origin could resist the modelling redressment" and that all bone operations on club feet were superfluous and mutilating[32].

In his view, his greatest success was molding reduction treatment of developmental dislocation of the hip. He found that with traction and manipulation he could stretch the contracted soft tissues and lower the head of the femur from its displaced position to the level of the acetabulum[3,32]. Then, holding the femoral head in

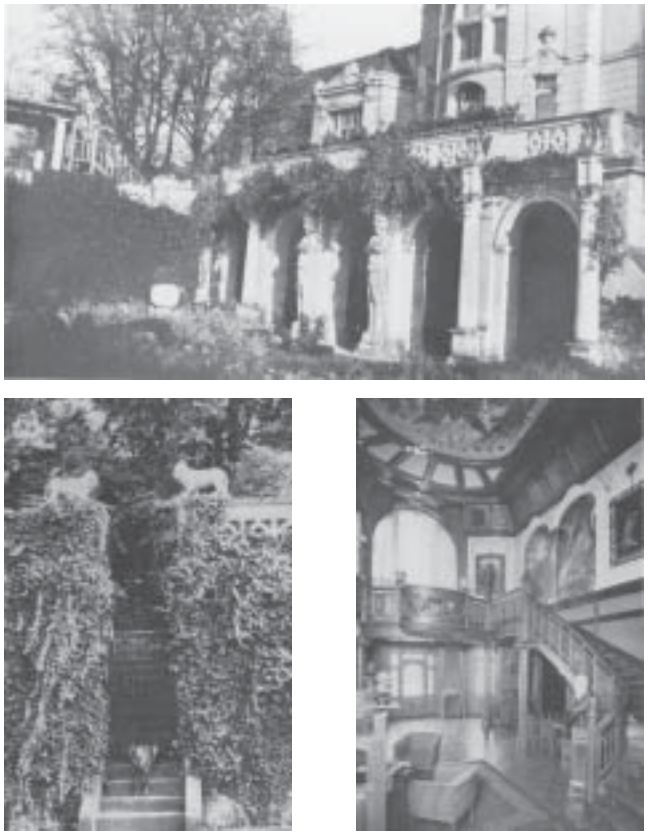

Figure 6. Lorenz Hall. A. Exterior. B. Lion Gate entrance. C. Entry hall

the reduced position with a plaster dressing caused remodeling of the soft tissues and the flattened acetabulum. He realized that the presence of the femoral head influenced the growth and development of the acetabulum and stressed that it was necessary to achieve reduction of the hip at an early stage to give the defective upper rim of the acetabulum "unhindered expression to its intrinsically normal growth tendencies[26]" thereby deepening the acetabulum and permanently stabilizing the hip in a reduced position. Lorenz's work with molding reduction of hip dysplasias led to a nomination for the Nobel Prize. He reported that he fell one vote short of receiving the award[26,32].

As Lorenz's fame and clinical practice increased, an increasing number of surgeons from Europe and United States came to Vienna to learn his techniques[32]. His success convinced him that he needed an appropriate house, " . . . like a small castle on the slopes of Wienerwald, overlooking the broad valley of the Danube, the Tullnerfeld, and rising out of a nice garden with beautiful old trees[32]." He selected a site with a commanding view of the Danube and designed an imposing structure, Lorenz Hall, with impressive terraces and gardens (Figure 6). Above the entrance he had a

Figure 7. Adolf Lorenz in Chicago in1902. A large audience watches him perform a procedure.

sentence from Horace cut in stone: "Lucro appone quem fors dierum cumque dabit." (Consider as gain whatever chance may bring)[32].

In 1902 and 1903, Lorenz traveled extensively in the United States[32,36]. During an extended stay in Chicago he performed a series of consultations and operations and reportedly collaborated with the prominent American Orthopaedist and Professor of Orthopaedic Surgery at Northwestern University John Ridlon (1852-1936)[34,36,49]. Figure 7 shows Lorenz performing a procedure, possibly a closed reduction of the hip, before a large audience in Chicago[32]. Ridlon subsequently challenged Lorenz's claim that the Lorenz method of closed reduction of the hip produced perfect reductions in fifty percent of treated hips and good results in eighty percent[49]. In 1904, at the New York Academy of Medicine, Ridlon stated that anatomic reductions using the Lorenz method were rare and did not exceed ten percent. Ridlon also reported that the method promoted by Lorenz had been developed previously by Agostino Paci (1845-1902)[3,35,36] and that the extreme force used by Lorenz gave deplorable results[49].

World War I devastated Vienna and made Lorenz's investments worthless. Partially because of his difficult financial situation he continued to practice well into his 70s. Although he was generally complimentary concerning the United States, he was "stricken from the roles" of the American Orthopaedic Association, apparently during World War I[32]. When he asked to be reinstated in 1924, his request was refused. The reasons for these decisions are unknown. He completed his autobiography at age 80[32], and noted, "I always enjoy the thought that excellent California wine will take the place of ice-water upon my table," and "Moderate drinking, moderate eating, and moderate smoking will help you reach an average age of 80." He died at age 92.

Figure 8. Steindler Family in Vienna. Arthur (1878-1959) stands in the back in the center of the photograph. His younger brother Julius (1890-1970) is seated in front. His sisters Olga (1879-1933) and Irma (1876-1935) are to his right and his parents Leopold (1849-1906) and Caroline (Goldberg) (1851-1936) to his left.

ARTHUR STEINDLER (1878-1959)

Arthur Steindler, the son of Leopold Steindler (1849-1906) and Caroline (Goldberg) Steindler (1851-1936), was born in Graslitz, a town near Prague in the Austria-Hungarian province of Bohemia[9,10,12,37]. Shortly thereafter his family moved to Vienna (Figure 8). His father was a lawyer who valued rigorous classical education. In Arthur's early years he studied literature, language, philosophy and music. He eventually mastered five languages which enabled him to follow the world orthopaedic and scientific literature, translate manuscripts for others, write for foreign publications, and teach students from other countries. As a young man he enjoyed music and expressed interest in becoming a professional musician. However, his father strongly recommended that he choose a career in medicine. As a result, Steindler entered medical school at the University of Vienna in 1896 and graduated in 1902. Although Billroth had been dead for two years when Steindler started medical school, many of his former students were still present: Albert was 55, a prime age for a department head, teacher, surgeon and scientist, and Lorenz was only 42.

Albert was regarded as one of the greatest teachers at the University of Vienna. Students never missed his lectures and he had an exceptional ability to explain complex clinical problems and make them interesting[32]. Steindler was among the students who found Albert's work exciting and he decided to work and study in University Outpatient Department for Orthopaedic Surgery directed by Lorenz following graduation from medical school[13]. Attitudes like those expressed by Billroth were common in Vienna in the early 1900s, and Jewish physicians, including Steindler, found that they had little chance for advancement[13]. After five years in Lorenz's clinic, Steindler left Vienna to join John Ridlon at the Chicago Home for Crippled Children in 1907. He later stated that he left Vienna because, "It was plain what was going to happen in Europe. There was nothing but privilege and preference. The working man had no chance. And nothing but aggression could come out of the politics of the government[17]".

It seems probable that Steindler became aware of Ridlon's work as a result of Lorenz's previous stay in Chicago and the subsequent controversy stimulated by Ridlon over the Lorenz method of closed reduction of the hip. Ridlon received his medical and orthopaedic education in New York, but he also studied with Hugh Owen Thomas (1834-1891), a leading British Orthopaedist. During his years in Chicago, Ridlon advocated and practiced Thomas's methods of treating deformities with bracing and manipulation rather than surgery. In addition to learning the American and Liverpool approaches to orthopaedic practice in Chicago, Arthur Steindler met Louise Junk, a young woman from a small town in Iowa[53], and one of Ridlon's surgical nurses[49].

In 1909 or 1910, John Ridlon was offered the position of Professor of Orthopaedics at the Drake Medical School in Des Moines, Iowa. Ridlon was well established in Chicago and had no interest in moving to Des Moines. In the short time Steindler had been in Chicago he had impressed Ridlon with his ambition, knowledge and clinical skill, and Ridlon suggested that Drake offer the position to his younger associate. He encouraged Steindler to take the opportunity to establish his own program. Drake seemed like a good choice. Des Moines, the state capital and largest city in the state, had good prospects for future growth, and Drake was a respected private University. Furthermore, there were no orthopaedists in the state. After moving to Des Moines in 1910, Steindler built a large clinical practice at Iowa Methodist and Lutheran hospitals[17].

While Arthur Steindler was starting his orthopaedic program in Des Moines, a former high school principal, Abraham Flexner[14], was reviewing medical education in the United States and Canada. Between January of 1909 and April of 1910, Flexner visited all of the medical schools in the United States and Canada, in one 90 day period he visited 69 medical schools[7]. By June of 1910, Flexner had submitted and published a blistering critique revealing the dismal quality of North American medical education and exposing multiple instances of fraud[15,39].

When Flexner visited Iowa in April of 1909 he found four medical schools: two in Des Moines, Drake University College of Medicine with 106 students, and the Still College of Osteopathy with 115 students, and two in Iowa City, the State University of Iowa College of Medicine with 267 students and the State University of Iowa College of Homeopathic Medicine with 42 students[15]. Des Moines had a population of 89,113 and Iowa City had a population of 9,007. Flexner concluded that none of the schools in Iowa met minimal standards for medical education and that the state had between two and three times as many doctors as it needed. He regarded the Des Moines osteopathic school as a disgrace to the state that should be summarily suppressed. The Drake Medical School and the Iowa City Homeopathic School he considered "well intentioned but feeble institutions that only a large outlay could convert into acceptable and efficient schools[15]." His advice to Drake was, "It would be the part of wisdom to retire from a contest to which the institution is clearly unequal[15]." He was not much kinder to the State University in Iowa City. His criticisms included a hospital and a patient population that were too small and a weak resident faculty: the Professor of Surgery for the State University of Iowa Medical School lived in Sioux City and the Dean who was also the Professor of Gynecology lived in Dubuque.

Drake University decided to take Flexner's advice and retire from the contest. Soon after this decision, physicians in Des Moines began a campaign to move the state's medical school to their city, arguing that that Des Moines could provide a better clinical experience for the medical students than Iowa City[17]. Flexner supported the argument that medical schools should be located in a large cities, " . . . where the problem of procuring clinical material, at once abundant and various, practically solves itself[15]." However, he also noted that the difficulties in establishing quality medical schools in what he referred to as "out-of-the-way locations" were not insuperable. "At relatively greater expense, it is still possible to develop a medical school in such an environment[15]." He identified two out-of-the-way locations where "The faculty of medicine in these schools may even turn the defect of the situation to good account: for, freed from distraction, the medical schools at Iowa City and Ann Arbor may the more readily cultivate clinical science[15]."

John G. Bowman, the ninth president of the University of Iowa from 1911 to 1914, apparently understood Flexner's hint and did not intend to allow the medical school to close or to leave Iowa City. He recognized the urgent need to recruit faculty with clinical expertise who could also integrate science into their teaching and practice. He could not have found an individual who met these criteria better than Arthur Steindler. Steindler could have continued his practice in Des Moines, but he had a strong desire to establish an academic program. On October 6, 1913, President Bowman wrote to Dr. Steindler to tell him that he had been appointed Instructor in Orthopaedic Surgery at the University of Iowa with compensation at the rate of $800 for the academic year[6]. Bowman noted that, " . . . this appointment is temporary pending the judgment of the permanent surgeon who will shortly be elected . . . the probability seems to me, however, that you will remain with us not only for the present year but for a longer time[6]."

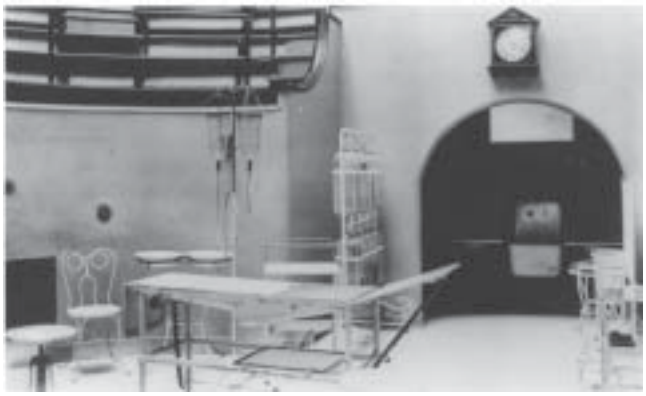

Figure 9. Operating theater in the University Hospital built on the east side of the Iowa River 1898.

Although Iowa City and Vienna did not have much in common, the operating theater Steindler used during his first seven years in Iowa City closely resembled the one used by Theodore Billroth (Figure 9). On March 1, 1914, confident that he would be spending a long time in Iowa, Arthur Steindler married Louise Junk (1882-1963) and in the same year he became an American citizen. In 1915, he was appointed to the Chair of Professor of Orthopaedic Surgery at the State University of Iowa[24].

By 1916, Steindler had established a large clinical practice in Iowa City and had helped convince the University and the State Legislature to construct a hospital for crippled children and pass legislation that supported the care of these children in Iowa City[9,10]. The completion of Children's Hospital in 1920 (Figure 10) gave Steindler and the University an exceptional facility that included inpatient areas, outpatient clinics, operating rooms (Figure 11), a brace shop, a large gymnasium, a hydrotherapy unit and laboratories. Many of the children sent to Steindler for treatment lived in the hospital for months while they received medical, surgical and physical treatment[9,10]. After 1920 Steindler's orthopaedic program functioned as an autonomous academic and clinical unit and was referred to as the Department of Orthopaedic Surgery[24].

Figure 10. Aerial view of Children's Hospital in the late 1920s. The building was located on a bluff overlooking the Iowa River. The river is visible in the upper right corner of the photograph. Other than the entrance under the cupola, the hospital consisted of a single story with a lower level that was partially underground. All the inpatient wards were on the first floor and every ward had direct access to the outside. In the lower left of the photograph, several patients in their beds who have been moved out on to a patio can be seen. The operating rooms were located on the same floor at the back of the hospital and the brace shop, gymnasium, laboratories and hydrotherapy unit were located on the lower level. The U-shaped roof projecting into the central court yard, covers a ramp connecting the first and lower floors. This ramp made it possible to transport patients from the first floor to the lower level.

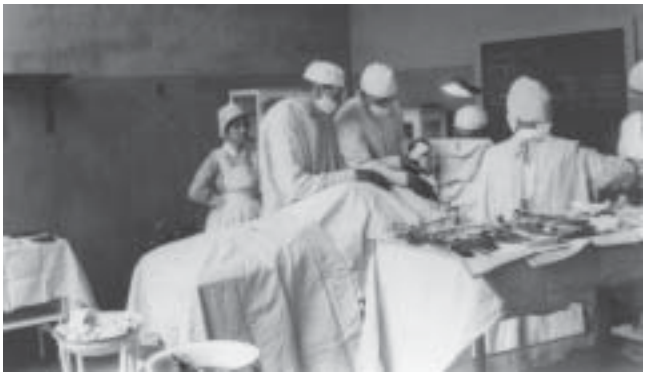

Figure 11. Operating room in Children's Hospital in the 1920s. The operating schedule on the blackboard lists a shoulder arthrodesis, a procedure first described by Eduard Albert. There are no seats for observers, the surgeons wear gowns, gloves and masks, a cheerful nurse observes the operation and a small single electric lamp illuminates the surgical field.

The rapid growth of Steindler's clinical program and his role in promoting legislation that brought patients to Iowa City from all parts of the state were critical for the development of the University of Iowa Hospitals and the College of Medicine. In 1925, Abraham Flexner noted that, "Iowa is now completing an adequate series of clinics[16]" and in a review of the growth and development of the health sciences at the University of Iowa the Iowa City Press-Citizen noted on October 6, 2002, "It was Arthur Steindler who opened the way for this modern institution [University of Iowa Health Care] by securing a steady flow of patients in the rural midwest[23]." His research and the students he attracted earned the College of Medicine national and international respect. On May 10, 1927, Walter Jessup, the fourteenth president of the University of Iowa from 1916-1934, wrote to Dr. Steindler formally appointing Steindler Head of the Department of Orthopaedic Surgery and granting him $3,500 for his research[25].

Through his teaching, speaking and writing Steindler encouraged basic research and incorporation of the basic sciences into orthopaedic education. In his 1933 presidential address to the American Orthopaedic Association, he emphasized the value of basic research in orthopaedics pointing out the need for study of anatomy, pathology, physiology and biochemistry[48]. In addition to encouraging study of the sciences, Steindler immersed himself in the investigation of human mechanics[43]. He felt that the mechanics of the musculoskeletal system represented a new field with great potential application and observed that "biomechanics is a powerful and indispensable ally of the orthopaedic clinician[48]." When discussing operative or non-operative treatment of musculoskeletal diseases and injuries he incorporated his knowledge of the biomechanics and biology of the musculoskeletal system. His book "Orthopaedic Operations[46]" begins with a chapter titled "The Biology of Functional Restoration" and he states that, "A knowledge of the possibilities and limitation of tissue regeneration, and tissue adaptation is essential equipment for every orthopaedic surgeon; his judgment depends on it." In Steindler's view, orthopaedic progress would occur not through technical perfection of operations, but through advances in the basic sciences.

In his study of orthopaedic problems and their treatment, Steindler pointed out the importance of understanding the natural history of diseases and critically reviewing the results of treatment[10], and some authors credit him with being the first to report on the long-term outcomes of all of his operations[40]. In his publications he presented and critically analyzed his operative experience, even when the results were unsatisfactory. In his book, *Orthopaedic Operations: Indications, Technique and End Results*, he discussed the indications and surgical techniques for each operation[46]. He defined four principles that should be used to evaluate operative procedures[46]: 1) Is the operation rational from the physiological and mechanical point of view? 2) Does the experimental evidence corroborate the expectations of regeneration and repair placed on the operation? 3) Is the operative technique in keeping with our experimental or empirical observations? and 4) Are reliable statistics available to justify the procedure in light of definite end results?

Figure 12. The Steindler's home in Iowa City on the bluff overlooking the Iowa River at the intersection of Grand Avenue and Riverside Drive. A. The appearance of the house from Grand Avenue, the street leading from the Iowa River to the Field House. B. Path and entrance stairway leading to the house from Riverside Drive. C. Terraced gardens. D. Garden paths.

In 1920, Steindler selected a bluff overlooking the Iowa River at the intersection of Riverside Drive and Grand Avenue as the site for a new home (Figure 12). The construction of the house and the terracing of the surrounding land was completed in 1922[13]. Mrs. Steindler planned the landscaping and creation of gardens including stone paths, pools and bridges[13]. Their home was large and located in what at the time was an attractive and pleasant location (now the site of the University of Iowa College of Law) over looking the Iowa River and with a view of the Old Capital. Above the entrance to his home, Steindler had a sentence from Horace inscribed: "Ille terrarum mihi praeter omnis anguls ridet" (The corner of the earth that smiles above all others)[13], recognition that he had found a place that offered him the opportunities he had sought when he left Vienna.

Figure 13. Arthur Steindler in the departmental library.

Figure 14. Steindler and his staff and residents in 1945. Seated (left to right) Ponseti, Thornton, Steindler, Greteman, Kaplan, Leveton. Standing (Left to Right) Salguero, Silensky, Diamond, Hicks, Le Noir, Pugi-Guri, Peidrahita, Nelson, Samaniego.

In the 1930s, Steindler monitored political events in Europe closely, and became increasingly concerned about the future of Germany and Austria and the lives of family and friends in Vienna. He started traveling to Austria regularly so that he could bring people to Iowa City where they could start a new life, as he had done. He continued this practice until the political changes in Germany and Austria preceding World War II made it impossible for him to visit Vienna[13]. Many of these individuals, including Hans Ehrenhaft, one of the most important contributors to the development of cardiothoracic surgery and Steindler's nephew, lived in the Steindler home until they became established in the United States[13].

THE VIENNA INFLUENCE

It is not difficult to see the Vienna influence in Arthur Steindler's career or in the department he founded in Iowa City. It is apparent in his research and clinical practice, teaching and in the traditions he established for Iowa orthopaedics. Eduard Albert's interest in the mechanics of joint movement and spinal deformity may have helped stimulate Steindler's studies of biomechanics and kinesiology. Like Albert and Lorenz, Steindler devoted much of his clinical practice to the care of children with developmental and paralytic deformities. Correcting deformities and stabilizing joints by arthrodeses, similar to Albert's operations, in patients crippled by polio and other neuromuscular disorders were an important part of Steindler's clinical work throughout his career[44-46,52]. Like his teachers, he also investigated the mechanics of scoliosis and devised a non-operative treatment for spinal curvature, compensation-derotation[41,42,50,51].

Steindler learned from Albert and Lorenz, but he did not accept everything they taught and practiced. He agreed with Ridlon's criticisms of Lorenz's treatment of developmental dislocation of the hip and noted that Lorenz's claims of the success of this treatment were exorbitant[49]. In his discussion of Lorenz's manipulative treatment of clubfoot he makes another rare comment concerning Lorenz, "It is well to recall at this point that the earlier surgeons (Lorenz) went much further with their conservative methods than we do today. They applied a great deal more force, and their attempts of redressement were limited by circulatory embarrassment only. In other words, anything that the circulation could stand in the way of manipulation or mechanical correction was accepted. It is in this sense that Lorenz speaks of modeling redressement. Today we find our ideas of what constitutes the tolerance to manipulation radically changed. We no longer "manipulate" as much as the circulation will stand. Our own attitude is that 90

percent of clubfeet in children can be successfully treated by conservative methods[47]." Steindler and Lorenz clearly had different views of what constituted modelling redressment and the outcomes of this treatment.

In Iowa City, Arthur Steindler found the opportunity he was seeking when he left Vienna in 1907, but he also created opportunities for others. He helped establish the University of Iowa as a major health science center. He became an international leader in orthopaedics and created an orthopaedic department based on the values and practices that made the University of Vienna one of the most important institutions in the history of orthopaedic surgery. Throughout his career, he emphasized the fundamental principle of the Vienna surgical tradition: excellence and innovation in the techniques of surgery combined with clinical and basic research, critical evaluation of the outcomes of operations and teaching the next generations of surgeons. Although he committed himself to his profession, students and patients, he enjoyed his time with family and friends; and, through the department he founded he enriched the lives of those who followed him.

ACKNOWLEDGMENTS

Arthur Steindler's nephews, Martin Steindler and Hans Ehrenhaft, provided invaluable information and insight into their uncle's life and aspirations. Webster Gelman, Dr. Steindler's colleague and friend, helped me to understand Dr. Steindler's humanity, sense of humor and values.

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IOWA AND EUGENE, OREGON, ORTHOPAEDICS

Joseph A. Buckwalter

ABSTRACT

Over the last 50 years, the commitment of orthopaedic surgeons to basic and clinical research and evaluation of treatment outcomes has made possible remarkable improvements in the care of people with injuries and diseases of the limbs and spine. A group of Oregon orthopaedic surgeons has had an important role in these advances, especially in the orthopaedic specialties of sports medicine and hip reconstruction. Since Don Slocum (Iowa Orthopaedic Resident, 1934-1937), started practice in Eugene, Oregon, in 1939, three orthopaedic surgeons, Denny Collis, Craig Mohler and Paul Watson, who received their orthopaedic residency education at the University of Iowa, and three orthopaedic surgeons, Stan James, Tom Wuest and Dan Fitzpatrick, who received their undergraduate, medical school and orthopaedic residency education at the University of Iowa, have joined the group Dr. Slocum founded. These individuals, and their partners, established and have maintained a successful growing practice that serves the people of the Willamette valley, but in addition, they have made important contributions to the advancement of orthopaedics.

INTRODUCTION

University of Iowa Orthopaedics and Orthopedic Healthcare Northwest, in Eugene and Springfield, Oregon, have long enjoyed a strong connection. For more than half a century, both groups have recognized the importance of expertise in orthopaedic specialties and a commitment to improving patient care through research and critical evaluation of outcomes. The connection has grown stronger since Don Slocum (Iowa Orthopaedic Resident, about 1934-1937), established his practice in Eugene, Oregon, in 1939. Since then, six orthopaedic surgeons, Stan James, Denny Collis, Craig Mohler, Tom Wuest, Paul Watson and Dan Fitzpatrick, who received their medical education, or orthopaedic residency education, or both, from The University of Iowa have joined Orthopaedic Healthcare Northwest. Ken Singer, another member of Orthopedic Healthcare Northwest, was born and raised in Sac City Iowa; and, Arthur Steindler[3] (1878-1959), the founder of University of Iowa Orthopaedics, treated Ken for a knee injury.

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DON SLOCUM

Don Slocum spent two years as an orthopaedic resident at The University of Iowa with Arthur Steindler in the late 1930s and then another two years with Willis Campbell at the Campbell Clinic in Memphis, Tennessee. In his last year at the Campbell Clinic, Don decided to establish a practice in Eugene, Oregon, a small university town in Oregon's Willamette Valley, partially because his wife, Margann, was from the area. Willis Campbell wanted Don to stay at the Campbell Clinic and advised him that if he went to Eugene, no one would ever hear of him again. Willis Campbell's leadership of the Campbell Clinic confirms his ability to direct an orthopaedic program, but, examination of the major contributions to Orthopaedics in the 20th Century, shows that he underestimated Don Slocum.

When Don opened his office in Eugene in 1939, the city had a population of about 30,000 people clustered along the banks of the Willamette River. During World War II, Don left Eugene to serve in the military for six years. He spent some of his service time at Walter Reed Medical Center in Washington, D.C., where he wrote *An Atlas of Amputations*[43]. Don asked Arthur Steindler review the book for him, particularly the chapter on gait; Dr. Steindler immediately recognized the importance and value of the book. As Steindler predicted, Slocum's *An Atlas of Amputations* became the definitive reference of its time and continues to rank among the texts that advanced orthopaedic practice. Following World War II, Don returned to Eugene and recruited a group of talented orthopaedic surgeons, including Howard Molter, Don Moore, Bob Larson and Jim Degge to join his practice.

Initially Don practiced general orthopaedics, but he became interested in the care of athletes when his son

Figure 1. Don Slocum.

Tom suffered a knee injury while playing high school football in 1958. An exploratory operation on Tom's knee revealed an impacted tibial plateau fracture. This experience led Don to study the limited medical literature concerning the treatment of athletic injuries. The paucity of basic and clinical research on sports injuries and improving physical performance convinced him that many athletes failed to return to their pre-injury level of physical activity because surgeons lacked expertise in the treatment of sports related injuries: an observation that made him focus more of his study and practice on athletic injuries. His clinical skill attracted the attention of Oregon coaches and athletes and within a short time he was providing all of the orthopaedic care for intercollegiate athletes at the University of Oregon and Oregon State.

Stimulated by challenges of trying to help athletes regain their ability to compete following injuries, Don began investigating the mechanisms of sports injuries and analyzing human movement to restore or improve physical performance[44-46]. Bill Bowerman, the internationally recognized Oregon track coach, shared Don's interest in studying human performance and in particular running gait[46]. Based on films of runners they concluded that a more upright posture could increase speed as compared with accepted approach of leaning the trunk forward. The upright running posture became a trade mark of the Oregon runners coached by Bowerman; and, in 1960, Otis Davis, an Oregon runner using the upright style, won the Olympic gold medal for the 400 meters. Don Slocum was among the first to investigate the relationships between symptoms of joint dysfunction and ligamentous laxity detectable by physical examination, and his studies of the roles of knee ligaments in stabilizing the joint while allowing normal motion were critical in advancing the diagnosis and treatment of knee injuries[8,34,47-52].

Along with several other orthopaedists, including Don O'Donahue, Jack Hughston, Jim Nicholas and Bob Larson, Don Slocum helped establish the American Orthopaedic Society for Sports Medicine. As a result of his clinical expertise, research and contributions to national professional organizations, he is recognized as one of the founders of the specialty of sports medicine. He was also a founding member of the Hand Society, making him one of the few individuals to have a role in establishing two orthopaedic specialties.

STAN JAMES

In the early 1960s, Charles Tipton, an accomplished Professor of Exercise Science at the University of Iowa, began studying the effects of exercise on ligaments and ligament healing[2,36,55-60]. At that time, orthopaedists believed that ligament tears should be treated with at least six weeks of rigid immobilization[7]. Based on the results of his work on other tissues, Professor Tipton suspected that the orthopaedic surgeons were wrong. To test the hypothesis that early physical activity promoted ligament healing, Professor Tipton needed someone who could skillfully transect dog medial collateral ligaments, repair the ligaments and apply a cast. Stan James (UI 1953 B.A., 1962 M.D., 1967 Orthopaedic Resident) volunteered to help with the project and spent two years performing the animal surgery. To compare the effects of immobilization with activity he applied casts to half of the limbs and maintained the casts for six weeks. Professor Tipton tested the mechanical properties of the ligaments treated with immobilization and with activity; Stan, with help from Dr. Ponseti, and Victor and Angela Pedrini (scientists studying connective tissue biochemistry with Dr. Ponseti), analyzed the collagen content of the ligaments. They found that activity increased intact and repaired ligament strength, collagen concentration and collagen fibril diameter[57], an observation that contradicted established concepts of ligament healing and called into question accepted clinical practice. Professor Tipton must have been impressed with Stan's work; he subsequently worked with two other orthopaedists, Gerald Laros and Reginald Cooper[4] on a study of the effect of limb immobilization on ligament insertions[36].

During his residency, Stan learned of Don Slocum's practice in Eugene and Don's interest in sports medicine. He visited Eugene in 1965 and two weeks later received a contract, a document that he quickly signed and returned. Because of a need for a hand surgeon between Portland and San Francisco, Don contacted Adrian Flatt, the founder of hand surgery at the University of Iowa[6], and asked him to provide Stan with further intensive training in hand surgery. Dr. Flatt taught many generations of talented residents at the University of Iowa, but he remembers Stan as an exceptionally capable and mature surgeon[26].

Stan arrived in Eugene in the summer of 1967, and started practice as a hand surgeon. Although Don Slocum encouraged Stan to develop a hand surgery practice, he was intrigued by Stan's work on ligament healing and he included Stan in his studies of athletes[46]. When Don learned that his friend Jack Hughston had

been appointed the program chairman of a sports medicine conference sponsored by the American Academy of Orthopaedic Surgeons, Don advised Jack to include Stan's work on ligaments in the conference. Jack responded that such a "highfalutin" paper might be of interest. After Stan presented his findings, Marcus Stuart, an orthopaedist from the Campbell Clinic commented politely: "Young man, that was a very erudite paper[29]." The new orthopaedic specialty of sports medicine was not ready to accept that an observation based on basic research, including biomechanical testing and collagen chemistry, should change entrenched beliefs and practices, and, for the next few years, presentations and discussions of ligament reconstruction and treatment of ligament tears continued to focus on the appropriate angle and duration of knee immobilization. Only after another decade had passed did the concepts that prolonged immobilization of a joint causes tissue damage and that early controlled activity promotes healing become widely accepted[7].

Shortly after starting practice, Stan received an academic appointment in the Department of Exercise and Movement Science at the University of Oregon and began a series of clinical studies in sports medicine. Although he continued to practice hand surgery, his experience with University of Oregon athletes increased his interest in sports medicine, in particular, sports related injuries of the knee and injuries in runners. Don Slocum supported Stan's interest in sports medicine and they worked together on studies of knee ligaments[33-35,47,50-52]. Don also introduced Stan to Bill Bowerman. The relationship with Coach Bowerman allowed Stan to conduct a series of investigations of running gait and injuries in runners that helped form the foundation of improved treatment of track athletes[1,30-32,46,54]. In the late 1970s, Stan reoriented his practice toward knee surgery so that he could spend more time working with athletes and investigating sports injuries. In the 1980s, his studies of the patellofemoral joint led Orthopaedists to appreciate the complexity of the clinical disorders of this articulation[9,10]. Stan's expertise rapidly earned him an international reputation and led many of the world's greatest runners to select him as their physician. He also worked with the founders of the Nike Company, including Phil Knight, to develop a research program and improve the design of running shoes. Stan attributes his life-long interest in research, development of orthopaedic specialization and academic orthopaedics to his education at the University of Iowa, and to the encouragement and support of Don Slocum[29]. Stan remembers Don as one of the most stimulating people he has encountered in his career and their clinical and academic collaborations have had a substantial impact as demonstrated by their multiple often cited publications[27,33-35,37,46,47,50-53].

DENNY COLLIS

In 1970, Denny Collis (UI 1970 Orthopaedic Resident) joined the group and rapidly established a large and successful practice dedicated to the treatment of patients with hip arthritis. During his residency, Denny worked with Dr. Ponseti[41] to define the natural history of idiopathic scoliosis. Their 1969 publication, *Long Term Follow-up of Patients with Scoliosis not Treated Surgically*[22], stands as one of the most important in clarifying the natural history of spinal curvatures and as an example of the critical importance of long term follow up of patients with musculoskeletal disorders. In Eugene,

Figure 2. Jody Buckwalter and Denny Collis in front of the Oregon Duck's Football Facility in Eugene, Oregon (2002).

Denny continued traditions established by Don Slocum and Stan James in producing important clinical research[11-17,19-21,23-25,28,39,42]. He established a computer database for the evaluation of the results of treatment of hip disease in 1970[18], well in advance of similar efforts in large institutions and has maintained this data base since then. Denny has authored or coauthored numerous papers analyzing the outcomes of hip surgery[11-17,19-21,23-25,28,39,42] including work with Dick Johnston and John Callaghan[19,39]. Denny's many contributions to hip surgery were recognized by his peers when he was elected president of the Hip Society for 1996-97. Denny's selfless activities and commitment to research that advanced orthopaedic practice are exemplified by his service on the board of directors of the Orthopaedic Research and Education Foundation (OREF) for eight years, including his service as chairman of the board from 1996-99 and his generous support of this foundation. In addition, to honor Dr. Ponseti and ensure continuing excellence in pediatric orthopaedics at the Uni-

versity of Iowa, Denny served as co-chairman of the campaign to endow the Ignacio Ponseti Chair of Orthopaedics at the University of Iowa, a position now held by Stuart Weinstein[5].

CRAIG MOHLER

In 1989, while performing a knee operation with John Albright, Craig Mohler (UI 1991 Orthopaedic Resident) heard Dr. Albright ask for a "Slocum"retractor. Shortly thereafter, Dr. Ponseti mentioned the scholarly contributions of Stan James and Denny Collis. In the next year, while relaxing in Dr. Cooper's[4] prosthetic clinic with Don Shurr (an expert on orthotics and prosthetics who has educated generations of orthopaedic residents), Craig, like many residents before and after him, listened with rapt attention to stories of Don's legendary career as a Hawkeye football player. One of those stories includes the reconstruction of Don's knee performed by Stan James when Don was a freshman, an operation that, despite Stan's great skill, only could restore Don to his former level of speed and agility. John Callaghan further increased Craig's interest in Orthopedic Healthcare Northwest when he described an elite group of orthopaedists in Eugene who divided their time between practicing the highest quality of orthopaedics, clinical research and exercising. A meeting with Denny Collis at the 1991 American Academy of Orthopaedic Surgeons and two visits to Eugene convinced Craig that the career opportunity in Eugene was exceptional. In 1992, Craig joined the group after finishing a fellowship in joint reconstruction at Rush Medical Center in Chicago. Denny Collis welcomed and encouraged Craig's interest in joint replacement and clinical research[20,21,39,40].

TOM WUEST

In 1991, Dr. Ponseti told Denny Collis that another Iowa Resident, Tom Wuest (UI 1981 B.S., 1987 M.D., 1992 Orthopaedic Resident), would be a great addition to the Eugene group. An interview convinced the group that Dr. Ponseti was correct. Following his residency, Tom and his wife Megan moved to Stoke-on-Trent, UK, where Tom worked with Professor John Templeton at the North Staffordshire Royal Infirmary. From Stoke-on-Trent the Wuest's moved to Eugene; a community that he reports has the most attractive feature of Iowa City, a strong University in a relatively small town. About half of Tom's practice is general orthopaedics except for joint reconstruction and spine surgery, the other half is devoted to musculoskeletal trauma. His specific interests include pelvic, acetabular, calcaneal, pilon and plateau fractures[38,61]. He serves as Secretary/Treasurer, Finance Committee Chairman and recruiting coordinator for the group. Megan works as a family therapist with the county juvenile detention center and their children Veronika and Samuel attend a French Immersion school. Tom and Megan note that their neighborhood in Eugene resembles Manville Heights in Iowa City, without tornados, snow, sleet and freezing rain. Tom enjoys the academic orientation, professionalism, clinical skill and camaraderie of the Orthopaedic Healthcare Northwest group, and he appreciates the "Iowa Influence"—critical thinking, technical skill, long-term follow-up of patients, and emphasis on non-operative as well as operative treatment.

PAUL WATSON

When Paul Watson (UI 1999 Orthopaedic Resident) looked for a practice opportunity, he identified only one private practice group that valued academic work as well has clinical expertise. He found that the partners in Orthopedic Healthcare Northwest enjoyed working together and helped new members to develop their own interests. A long tradition of excellence maintained by people who work well together and support each other has been important to Paul in selecting places for his education and work. This explains why he chose McGill University for Medical School, the University of Iowa for his residency and Orthopaedic Healthcare Northwest for his work. His decision to attend Harvard as an undergraduate is the only deviation from this pattern. Since Paul joined Orthopaedic Healthcare Northwest, his experience has been everything he expected and more. Although he has a special interest in shoulder surgery, he enjoys every aspect of orthopaedic practice including joint replacement and hand, foot, ankle, tumor and pediatric surgery. Paul has been able to arrange his schedule so that he has free time to spend with his family: he works four days a week and has dinner with his family almost every night, and Eugene has proven to be an ideal place to raise his family.

Figure 3. Paul Watson and Dan Fitzpatrick at the Wuest's home in Eugene, Oregon (2002)

DAN FITZPATRICK

Last year, Dan Fitzpatrick (UI 1991 B.S.E, 1993 M.S., 1997 M.D., 2002 Orthopaedic Resident) joined the group. During his residency, Dan took advantage of his background in engineering and the facilities and expertise of the University of Iowa Orthopaedic Biomechanics Laboratory to conduct an investigation of ankle joint articular surface incongruities that won an American Orthopaedic Association resident research award. As he was considering his career direction after residency, Dan was attracted by the rich clinical and academic traditions of the Orthopaedic Healthcare Northwest group. During their first visit to Oregon, Dan and his wife Denise immediately recognized that Eugene offered great professional and personal opportunities and a warm and welcoming social life. Dan now finds himself very busy and enjoys being part of the great orthopaedic tradition in Eugene and Springfield. He is planning to pursue several research projects.

CONCLUSION

Since Don Slocum moved to Oregon in 1939, the population of Eugene has grown to more than 140,000, the group he founded has thrived and become a model of a private practice that has made sustained and substantive academic contributions, and the Iowa influence on orthopaedics in the Willamette Valley has become more pronounced. The members of the Orthopedic Healthcare Northwest group with an Iowa connection feel that their strong clinical practice, support for research and success in conducting clinical research stem from the principles and values established by Don Slocum and further developed by Stan James and Denny Collis. The group represents not only the highest standards of clinical practice, but demonstrates that a group of committed orthopaedic surgeons can make important contributions to advancing the specialty through development of specialized care, evaluation of the outcomes of their treatments and research.

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THE SUSPICIOUS DEMISE OF AMY ROBSART

Sachin K. Patel, M.D., and Richard Jacobs, M.D.

Does there not lurk within the heart of every orthopedist interest in the unusual? With childhood days, there were always Sherlock Holmes, the Shadow, the Green Hornet, Charlie Chan and Nero Wolfe. The current spate of forensic offerings notwithstanding, we were all fairly sophisticated many years ago.

That being the situation, let us introduce a classic case for your perusal and deductions. The year was 1560. It seems that Sir Robert Dudley, soon to be Earl of Leicester, was infatuated with the good Queen Bess and she with him. Rumors abounded that there was early and probably frequent consummation. A certain "Mother Dowe of Brentwood" questioned whether Sir Robert had, on one occasion, given Elizabeth the gift of a petticoat or of a child! Mother Dowe had big legal troubles, but finally escaped with her neck intact. Others, for the same offense, had their ears cut off.[3]

The fly in the ointment, so to speak, was Sir Robert's marital status. His twenty-eight year old wife Amy Robsart, was the prime heiress of the wealthy Sir John Robsart. She was said to be ravishingly beautiful. However, there was the question of her health—it was rumored that she suffered from what we now know as advanced carcinoma of the breast and that Sir Robert and Elizabeth had plans for the future after her anticipated sad demise.[4]

All of this changed on September 8th, 1560. The beauteous Amy was found dead of a broken neck. Sprawled at the foot of a flight of stairs at her mansion, Cumnor Place, the hood of her robe still covered her lifeless head. Her relatives cried "Murder!" and the matter was put to a closed coroner's jury. Their verdict was never formally disclosed, but obviously death was attributed to accidental causes. This was as well for Sir Robert, because even in that era trial by ordeal was often practiced. Could he have withstood having his hands held in a fire or other dreadful tortures and still proclaimed his innocence? That might have been what he would have faced were he formally charged! Patricia Cornwell specifically referred to this case in her recent book.[1]

***Amy Robsart,* William Frederick Yeames, c.1877, Tate Gallery**

Now is the time to cogitate, to ruminate or however it is that you usually reach a conclusion about serious matters. Was it murder? Many loyal Englishmen

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thought so, despite the jury verdict. Rumors persisted, and years later on the London stage, an actor uttered the lines "The surest way to chain a woman's tongue is to break her neck! A politician did it . . . "

The scandal was such that it put an end to the affair between Sir Robert and Elizabeth. The politically canny daughter of Henry VIII and Anne Boleyn was not about ready to risk her reign by further dalliance, however sweet it might be. I am assured by a prominent hand surgeon of English descent with strong ties to the Iowa Orthopedic program "She was pushed!" He also shared the further information that Anne Boleyn had reduplication of the distal phalanx of the little finger on each hand—she was never caught short-handed.

Another possibility is metastatic carcinoma to the cervical spine. Difficult to prove when they didn't even have the concepts of Virchow and microscopic pathology in that era! We all remember Mike Bonfiglio and our study of the work of Batson in explanation of spinal spread of carcinoma of the breast! Without radiographic evidence and even in the absence of trauma, she could have suffered "a broken neck."[2]

What we now know as tuberculosis was endemic in that era. Did she have tuberculosis of the spine at a higher level and suffer quadriplegia instead of the more common (Sir Percivall) Pott's paraplegia?

Did she have a berry aneurysm and a fatal stroke?

Did she have a lethal arrhythmia?

Did she have the Guillain-Barre syndrome or some acute transverse myelopathy?

Did she have cervical spondylosis?

Did she have Gorham's vanishing bone disease?

Did she have extradural lymphoma deposits?[5]

We think not, for two main reasons. First, because we treasure the memories of scoundrels past. They give color to the lifestream without any threat. This is what is called history! Second and even more germane, Adrian told us "she was pushed!" We have, after all, the utmost trust in him!

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