VOLUME 16
1996

The Iowa Orthopaedic Journal

Published by the Residents and Faculty
of the Department of Orthopaedics
The University of Iowa

THE IOWA ORTHOPAEDIC JOURNAL

1996 • Volume 16

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 Orthopaedics. The journal will be published annually in May or June. The deadline for receipt of articles for the 1997 journal is March 1, 1997.

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

When you send an article it is essential that the following items be submitted:

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,** alphabetical and double-spaced, or references made in text only. Refer to bibliographies in *The Journal of Bone and Joint Surgery* and follow the style exactly.
3. **Legends** for all illustrations submitted, listed in order and typed double-spaced.
4. **Illustrations**
   a. *Black-and-white glossy prints* of photographs. Give *magnification* of photomicrographs.
   b. *Original* drawings or charts.
   c. Color illustrations cannot be used unless in the opinion of the journal they convey information not available in a black-and-white print. If color is desired, please send both color and black-and-white prints.

Preparation of manuscript: Manuscripts must be typewritten, double-spaced with wide margins. Write out numbers under 10 except percentages, degrees, or numbers expressed in decimals. A direct quotation should include the exact page number on which it 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: Number all figures and indicate *top* plainly. 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 usually 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.

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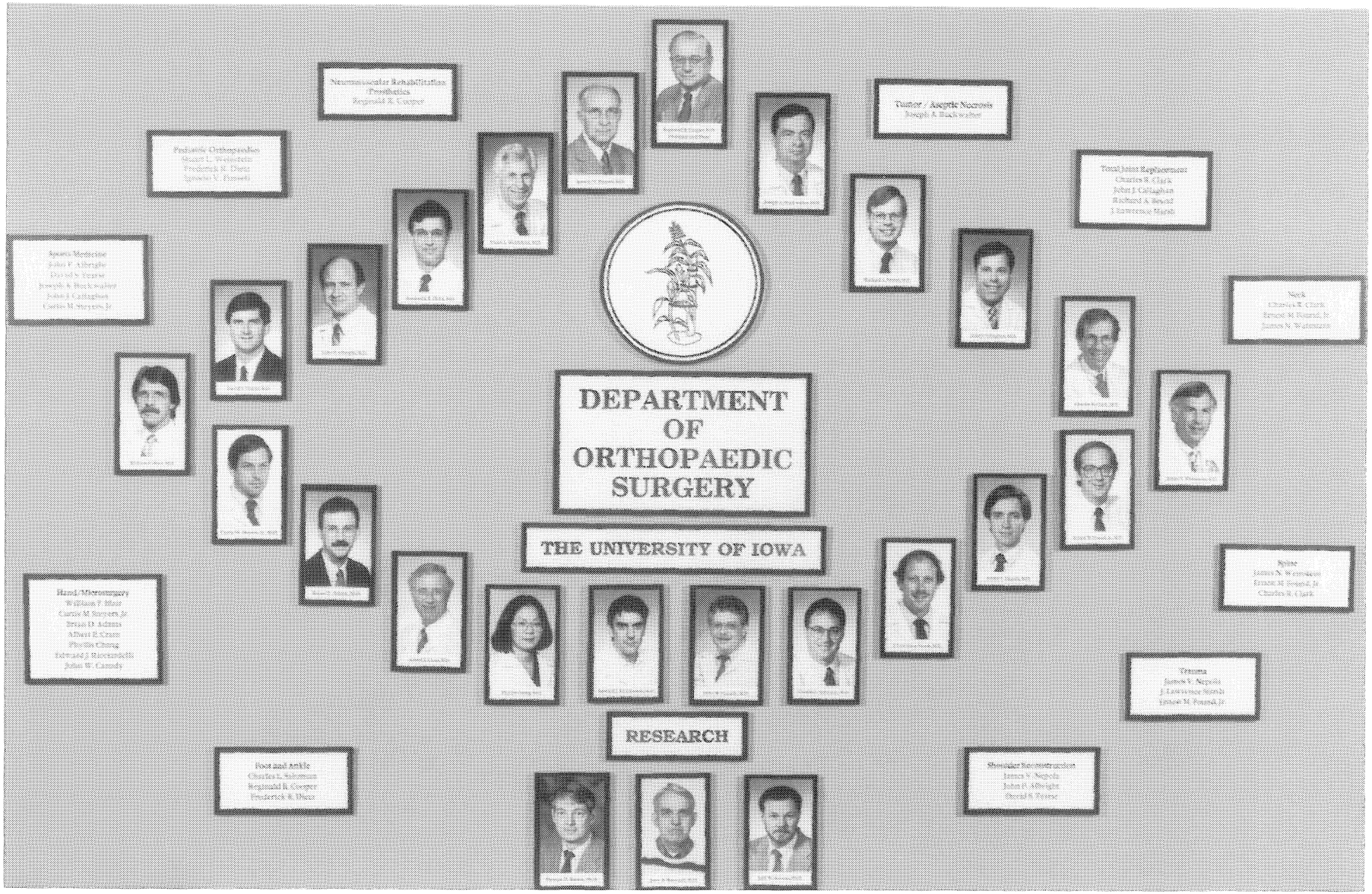
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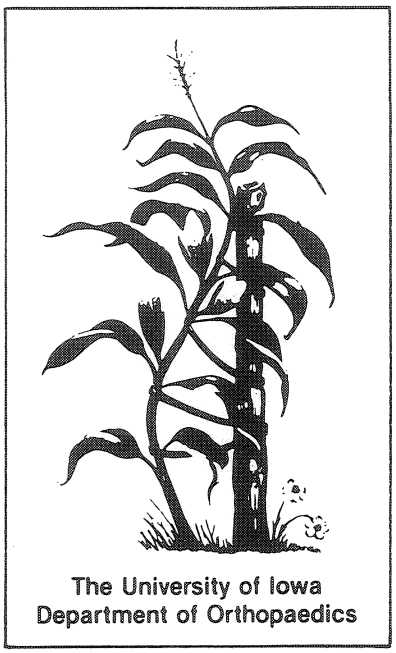
EDITORS' NOTE



The primary purpose of this journal is education, and those who participate in its production undoubtedly learn the most. We have considered it a privilege to produce the *Iowa Orthopaedic Journal* and have gained valuable experience in doing so. However, we hope that each reader is able to learn something new from the journal and benefit from reading it.

As the demands on physicians' time for clinical and administrative duties increase, the time available for research and education will decrease. Therefore, we greatly appreciate the efforts put forth by the authors in this journal. We were particularly happy with the strong contribution from The University of Iowa Orthopaedic staff, residents, and medical students.

This journal specially recognizes Dr. Richard Johnston for his contribution to the field of orthopaedic surgery. From his work, an amazing amount has been learned about hip disease and the results of hip arthroplasty. To his credit, four of the senior residents have chosen to study his patients for their research projects.

The editors of the *Iowa Orthopaedic Journal* would like to thank our faculty advisers Drs. Reginald Cooper, Joseph Buckwalter, and Charles Clark. We also thank Laura Cole for her dedication and excellent secretarial assistance. Diane Thomas and The University of Iowa Printing Department also deserve special recognition for their assistance. The journal is published under the administrative guidance of Paul Etre and Kay Redlinger-Phillips.

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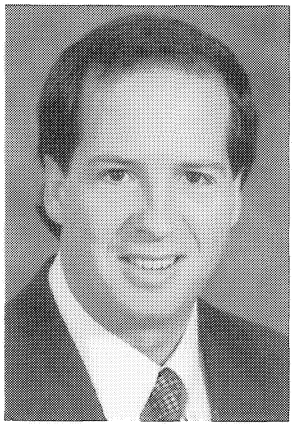
1996 GRADUATING SENIOR RESIDENTS

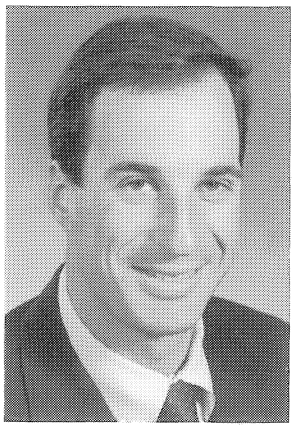
Kristy L. Weber, M.D.
Kristy grew up with her parents and younger brother in St. Louis, Missouri. She received a B.S. in Animal Science at the University of Missouri-Columbia with plans to become a veterinarian. Changing career plans late in college, she decided to attend the Johns Hopkins School of Medicine and obtained her M.D. degree. After completing her orthopaedic residency, Kristy will pursue a two year research and clinical fellowship in musculoskeletal tumor surgery at the Mayo Clinic in Rochester, Minnesota. Her interests include backpacking, skiing and barbecuing.

Margaret J. Fehrle, M.D.
Margaret was born in Des Moines, Iowa, and raised in Columbia, Missouri. She graduated from Drake University in 1984 with a B.A. degree in Biology. She attended the University of Iowa Medical School and received her M.D. in 1991. Next year Margaret plans to move with her son, Jonathan, to Marshalltown, Iowa, where she will practice general orthopaedics with her husband, Doug.

Steven M. Madey, M.D.
Steve was born in New York City and raised in nearby Long Island. He received a B.A. in Biology from Hofstra University and his M.D. from Columbia University College of P & S. He did his Internship at Presbyterian Hospital in the City of New York. He completed a basic science research fellowship at the University of Iowa before beginning his residency in orthopaedic surgery. Next year Steve, with his wife Kim and son, Clayton, will be in Iowa for a fellowship in Hand Surgery.



Mark G. Creighton, M.D.
Mark was born in Ft. Benning, Georgia, but calls Jamesport, Long Island, New York home. He attended the University of Notre Dame, and graduated from Stanford University with a B.S. in Biological Sciences. After receiving his medical degree from the Mt. Sinai School of Medicine in New York City, he completed an orthopaedic research fellowship at the University of Texas at San Antonio. Mark plans to complete a sports medicine fellowship next year with Dr. Robert Hunter in Denver and Aspen, Colorado.



Thomas M. Fyda, M.D.
Tom was born and raised in Youngstown, Ohio. He attended Dartmouth College in Hanover, New Hampshire where he received an A.B. in mathematics in 1987. After college he went to Duke University School of Medicine where he obtained his M.D. in 1991. Next year Tom and his wife Kristin will be moving to Salt Lake City, Utah where he will be doing a sports medicine fellowship.

DEPARTMENT OF ORTHOPAEDIC SURGERY
LECTURESHIPS AND CONFERENCES

TITLE AND GUEST SPEAKERS

Carroll B. Larson Shrine Memorial Lecture
February 3-4, 1995
William Cole, MBBS, MSC, Ph.D., FRCAS, FRCSC
University of Toronto
Toronto, Canada

Senior Residents' and Fellows' Day
May 26-27, 1995
Richard D. Coutts, M.D.
University of California
San Diego, CA

Lawrence D. Dorr, M.D.
University of Southern California School of Medicine
Los Angeles, CA

Michael Bonfiglio Iowa Orthopaedic Alumni Meeting
September 21-23, 1995
Robert H. Cofield, M.D.
Mayo Clinic
Rochester, MN

Edward McCarthy, M.D.
Johns Hopkins Hospital
Baltimore, M.D.

Russell Warren, M.D.
Hospital for Special Surgery
New York, NY

James Urbaniak, M.D.
Duke University
Durham, NC

TITLE AND GUEST SPEAKERS

Sports Medicine Symposium
December 1-2, 1995
Joseph S. Torg, M.D.
Joe Torg Center for Sports
Medicine and Athletic Trauma
Hahnemann University Hospital
Philadelphia, PA

Carroll B. Larson Shrine Memorial Lecture
May 2-4, 1996
Dennis R. Wenger, M.D.
University of California
San Diego, CA

Senior Residents' and Fellows' Day
May 23-25, 1996
John Kenwright, M.D.
Oxford University
Oxford, England

Bernard F. Morrey, M.D.
Mayo Clinic
Rochester, MN

Johnston Lectureship in Hip Reconstruction
October 11-13, 1996
Andrew A. McBeath, M.D.
University of Wisconsin
Madison, WI

Eduardo Agustin Salvati, M.D.
Hospital for Special Surgery
New York, NY

Sports Medicine Symposium
December 6-7, 1996

IN APPRECIATION OF SANDY BREDMAN

The editors of the 1996 *Iowa Orthopaedic Journal* recognize Sandy Bredman for 35 years of service and contribution to the Department of Orthopaedics.

In 1961, the Department of Orthopaedics was located in the Steindler Building. Drs. Larsen, Ponseti, Bonfiglio, and Flatt constituted the faculty. Sandy arrived in Iowa City with her husband, and started working in the department, typing operative notes and discharge summaries. Since her desk was in the orthopaedic clinic waiting room, she also took patients to exam rooms, made appointments, and attended to insurance and billing questions.

In 1962 Dr. Cooper joined the Department of Orthopaedics. Since his arrival, Sandy has acted as his assistant. She has helped Dr. Cooper with all of his endeavors, including Department Chairman, Director of the Residency Program, and chairmanships and memberships of numerous national and international orthopaedic organizations, societies, and committees.

As she has done for many years, Sandy continues to devote a great amount of time and energy to the organization and operation of the residency program. She receives and maintains about 650 applications each year, circulates complete applications to the staff, arranges interviews, and keeps the Department in compliance with the rules and regulations of the National Residency Matching Program. Sandy develops resident rotation and vacation schedules and serves as a most valuable day to day resource for residents.

Sandy has continuously provided outstanding service to the Department of Orthopaedics. In 1987 she was recognized with a University Staff Council Staff Excellence Award. In 1992 she was honored with the State Board of Regents Staff Achievement Award.

Sandy's organizational skills and numerous memos have helped keep residents in line with administrative duties, and most importantly, in good graces with the orthopaedic faculty. We are most grateful for her loyal and dedicated service.

Jay Jansen, M.D.
Laura Prokuski, M.D.

IN MEMORY AND HONOR OF DR. MICHAEL BONFIGLIO

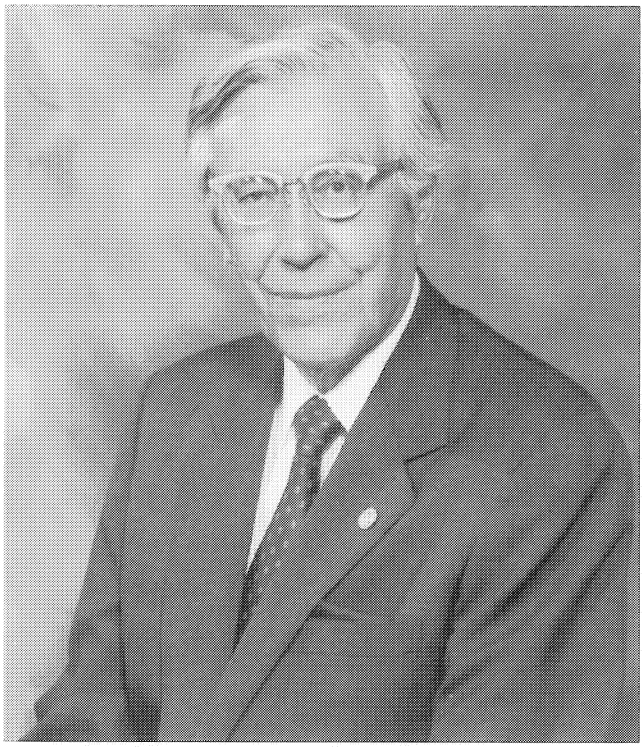
This past year the orthopaedic community lost an outstanding teacher, clinician, and investigator with the death of Dr. Michael Bonfiglio on June 13, 1995. He died after a prolonged struggle with multiple myeloma.

Michael Bonfiglio—affectionately known to his friends as "Dr. B."—was born in Milwaukee, Wisconsin, on April 3, 1917. His parents had immigrated from Sicily to the United States because education in this country was free. Neither of his parents was formally educated—his father worked as a laborer on a coal barge and his mother, as a homemaker—but they believed that education was extremely important for their children. His father died when Michael was nine years old, and his mother died when he was thirteen. He and his younger sister were initially cared for by his three older brothers, but he learned self-reliance at an early age.

He finished high school at the age of sixteen but had to work for three years to earn money for college. When he was nineteen, he entered Columbia University in New York City, where he majored in premedicine.

While in medical school at the University of Chicago, he spent a summer as an extern in the orthopaedic department at the Home for Destitute and Crippled Children. There, he reported directly to Dr. Howard Hatcher, the Chairman of Orthopaedics, who was in charge of the country home. He did his internship at the University of Chicago and initially planned to pursue a career in pathology, pediatrics, or surgery. After his first month of internship, the poliomyelitis epidemic struck Chicago, and all of the hospital beds in the city were filled. Dr. Hatcher opened a floor at his facility for these patients. It was he who invited Dr. Bonfiglio to enter orthopaedics.

Dr. Bonfiglio began his residency in Chicago; he worked there for one and a half years before entering the military service at Madigan General Hospital in Fort Lewis, Washington. There, he acquired much experience in the treatment of osteomyelitis. In April 1950, after twenty-six months in the service and following completion of his residency, Dr. Bonfiglio joined the Department of Orthopaedics at the University of Iowa in Iowa City. He came at the encouragement of Dr. Ignacio Ponseti, who had heard him present a paper on eosinophilic granuloma to the Chicago Orthopaedic Society. Dr. Bonfiglio's early projects included ongoing research on the immunology of bone transplantation and completion of a paper on the effects of drilling and bone-grafting of the necrotic femoral head in dogs; the paper was published in *Surgery, Gynecology and Obstetrics* in 1954. This work represented an important early contribution to the understanding of necrosis of the femoral head. Dr. Bonfiglio also contributed twenty-eight articles and book reviews to *The Journal of Bone and Joint Surgery* and wrote at least twelve papers on the histopathology and treatment of necrosis of the femoral head. (This work is cited in almost every article and text on the topic).



Dr. Michael Bonfiglio

Dr. Bonfiglio had a lifelong commitment to education. Throughout his career, he emphasized the importance of in-depth education and rigorous thinking. His enthusiasm for basic musculoskeletal pathophysiology allowed him to build a premiere learning and research center containing more than 11,000 full-size lantern slides, more than 27,000 microscopic slides, and hundreds of radiographic slides. He served as chairman of the Pathology Committee of The American Academy of Orthopaedic Surgeons. During his extended membership on the American Board of Orthopaedic Surgery, he was Chairman of the Examination Committee and was known for his support of resident education. Much of the material for the Board's oral examinations throughout the years came from Dr. Bonfiglio's University of Iowa files. In 1983, Dr. Bonfiglio delivered The American Orthopaedic Association's prestigious Shands lecture, in which he said: "We have obligation to continue the tradition of scholarly pursuits which nutured us by inspiring and encouraging the interest of our students and residents to study problems in depth." He was President of the Orthopaedic Research Society in

1965 and an Associate Editor of *The Journal of Bone and Joint Surgery* from 1968 to 1984.

Dr. Bonfiglio's Saturday-morning pathology conferences terrorized generations of residents but made them better orthopaedic surgeons. In addition to his clinical, academic, and research interests, he continued to coordinate the medical student teaching program for the department and to lecture to third-year medical students every two weeks concerning the interpretation of bone radiographs and musculoskeletal lesions. In recognition of Dr. Bonfiglio's dedication to teaching, the Michael Bonfiglio Research Award is presented annually to the fourth-year medical student who has written the best research essay.

Dr. Bonfiglio remained dedicated to orthopaedics despite physical adversity, including hereditary thrombocytopenia, a splenectomy, and a stroke. He eventually recovered from the visual impairment and paralysis of the left lower extremity caused by the stroke and was left with only a partial palsy of the left upper extremity. He missed only a short period of work and was soon back to clinical practice and teaching.

For more than forty-five years, Michael Bonfiglio gave of himself unselfishly. All of his teaching flowed from his insistence that the care of the patient must always be the highest priority in our professional lives. Mike did not merely articulate high ideals as ethereal concepts; he made those ideals the very essence of his being and literally lived them each day, with vigor and enthusiasm. Few came into contact with him without being irrevocably altered. We will always remember his genuine kindness and sincerity, his unpretentious nature, and his willingness to tackle tough issues. He had boundless energy and an appreciation of humor. He was a person of deep conviction who could be uncompromising in his demands for excellence in himself and others, and he had faith in the ultimate triumph of good. To many of us, he was the ideal role model. He is greatly missed but we remain forever grateful for his productive life and for having been blessed with the privilege of knowing and working with him.

J.A.B., C.R.C., J.J.C., and R.R.C.

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. B.'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 only the earnings from 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
Department of Orthopaedic Surgery, JPP
University of Iowa Hospitals and Clinics
200 Hawkins Dr.
Iowa City, IA 52242

RICHARD C. JOHNSTON, M.D.
EVALUATOR AND PRACTITIONER OF HIP SURGERY

John J. Callaghan, M.D.

Richard Charles "Dick" Johnston was born on September 10, 1933 in Onawa, Iowa (population 2000-3000). His father, Charles, born in Clarinda, Iowa, was one of 13 children. Charles played football and obtained his degree from Buena Vista College prior to taking a job as a science teacher and coach. Dick's mother, Susan, one of five children, was born in Galva, Illinois. She obtained her teaching certificate after Dick left home, and received a Bachelor's degree from Drake University in her late fifties.

Dick relates that his father lived the way he talked. Honesty was higher than godliness. It was a requirement that was stressed by both parents while he was growing up. They also stressed the importance of learning, critical evaluation, and the need to ask questions. Through his parents, Dick learned these traits that have helped him become the success he is today.

Dick was an only child. When he was four years old, his family moved to Kirkman, Iowa (population 250) where his father became the school superintendent. The family moved two more times (in 1941 to Correctionville, Iowa and in 1948 to Bloomfield, Iowa) so that Charles could take better school superintendent jobs.

In Bloomfield, with his father serving as the school superintendent, Dick was required to be "well behaved" during his high school years. He participated in football and basketball (Figure 2) and performed well in school, finishing third in his class.

After high school Dick went on to college. For as long as he can remember he was interested in medicine. Good students in "small town Iowa" became either doctors or engineers. Although Dick was fond of math, he never pursued an engineering career. Instead, he entered the

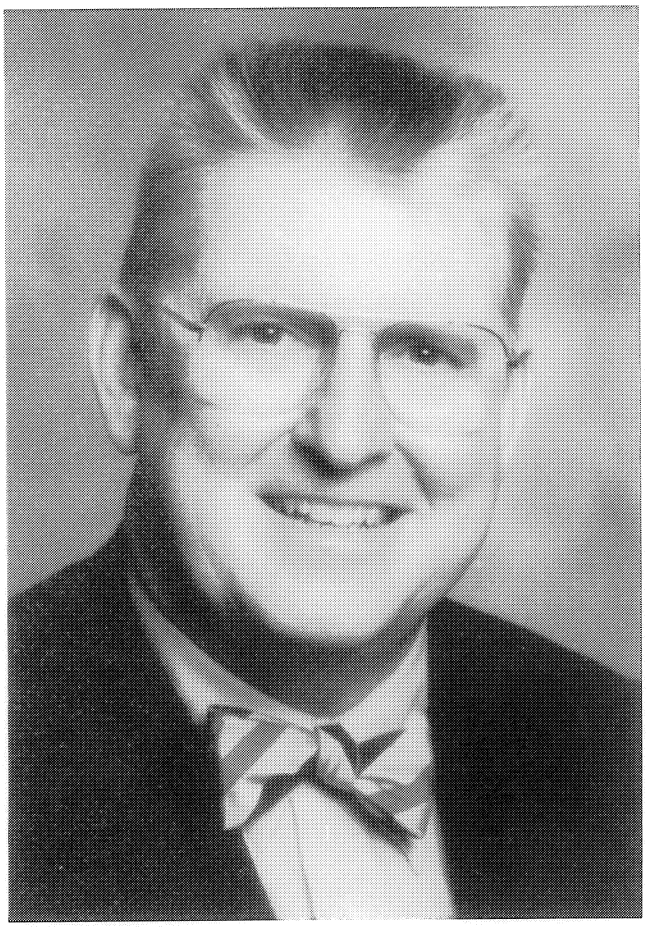


Figure 1. Richard C. Johnston, M.D., 1994

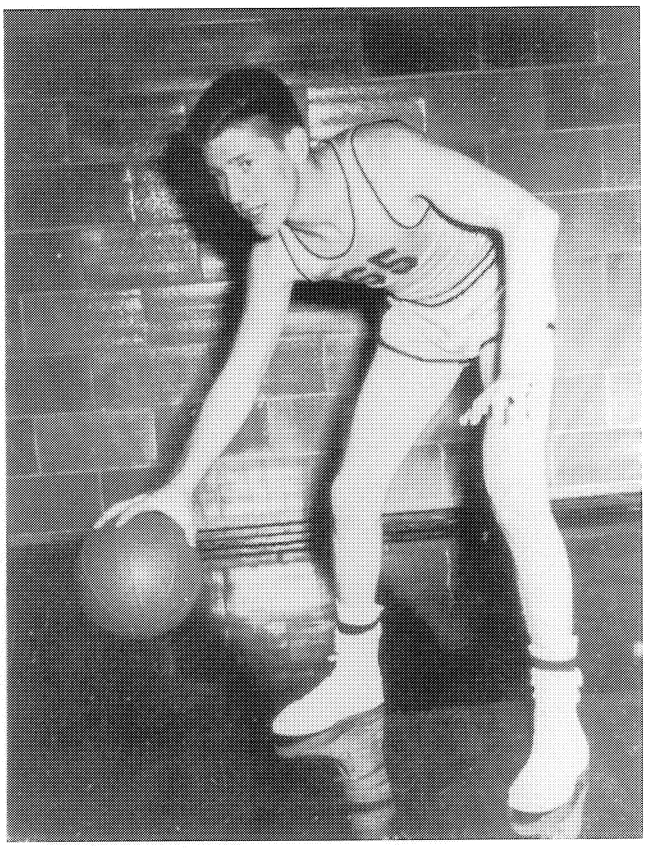


Figure 2. Dr. Johnston as a Bloomfield High School basketball player.

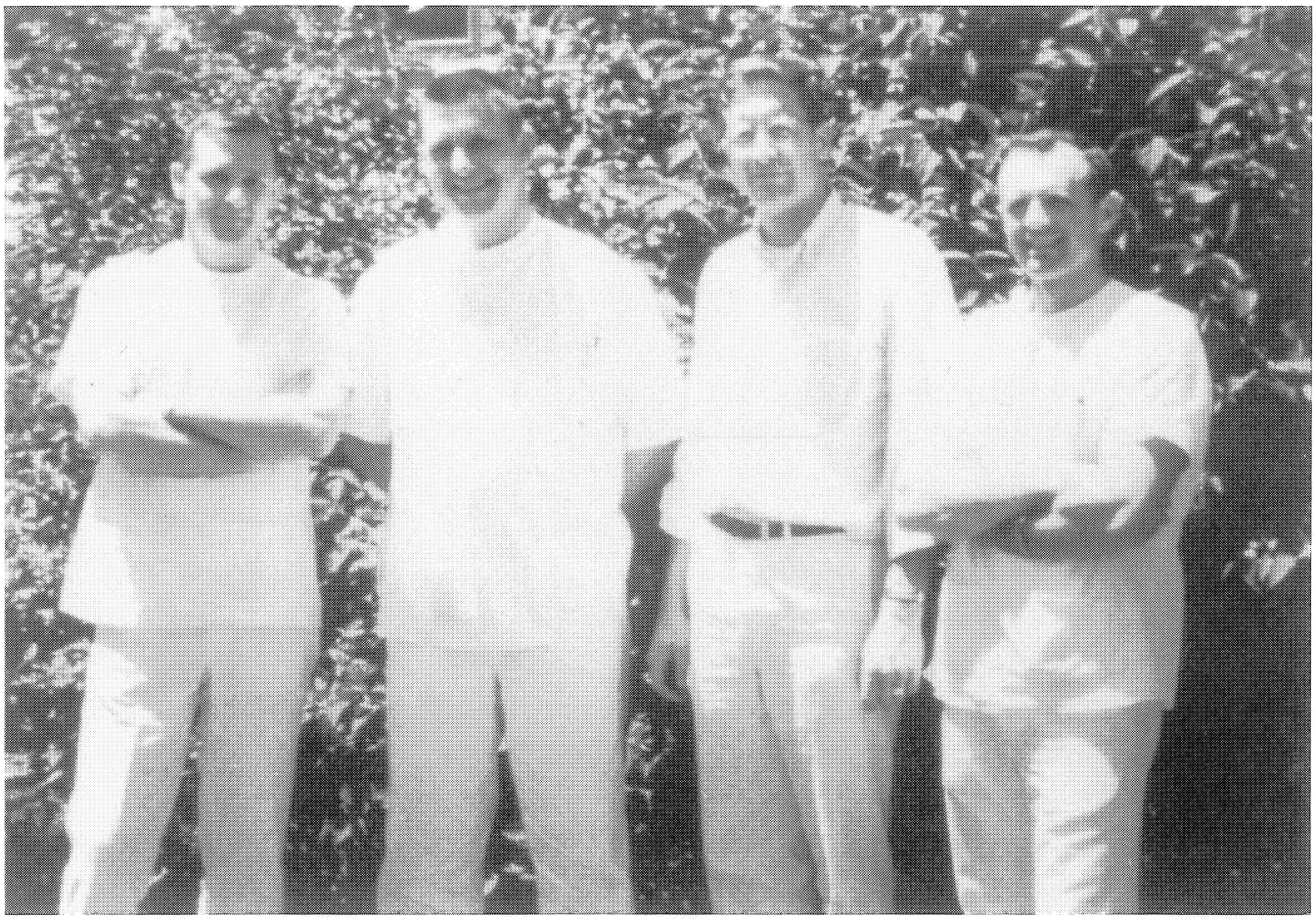


Figure 3. Dr. Johnston with his residency classmates. From left to right, Bob Porter, Dick, Ray Fenner, and Stan James

University of Iowa as a pre-medical student in 1951. He entered medical school in 1954 before obtaining an undergraduate degree.

Medical school proved to be traumatic in those days as the entire grade was based on the final exam. Despite this, many of Dick's teachers, including Drs. Theilen, Clifton, January, Ziffren, Flocks, and Bonfiglio, left a great impression on him. Interestingly, at this time Dick did not know his future mentor Dr. Carroll Larson.

During medical school, Dick had three children (Mike, Lynn, and Doug; his youngest daughter, Hannah was born later) and lived in the old Quonset huts. To finance his medical education, he joined the Air Force and was put on active duty during his final two years of school.

Upon graduating from medical school, Dick entered the Air Force to fulfill his military obligation. From 1958-1959 he performed a rotating internship at Madigan Hospital in Tacoma, Washington. After that, he was stationed at Luke Air Force Base in Arizona for four years. To avoid the outpatient dispensary, he did on the job training in obstetrics and gynecology, and in two years performed nearly 1,000 deliveries. In his final two years in the service, Dick did internal medicine, general surgery, and the dreaded dispensary. During this time, he served as the deputy hospital commander.

After completing his military commitment in 1963, Dick moved to Fairmont, Minnesota and joined four other doctors in family practice. After three months in practice, he decided to pursue an orthopaedic surgery residency. When Dr. Joe Fellows (Dick's future partner) was drafted into the service, Dr. Carroll Larson was able to offer him a spot at the University of Iowa. He started residency in 1964 with Drs. Stan James, Bob Porter, and Ray Fenner (Figure 3). For his military service, he was credited with one year of residency.

After completing projects in foot biomechanics and in hand surgery with Dr. Flatt, Dick was leaning toward hand surgery. However, the need for three dimensional thinking in hip surgery stimulated his interest. From 1966-1967, what was supposed to be his last year in residency, Dick was brought on staff in a fellowship position in order to concentrate on the study of hip surgery (Figure 4).

During this fellowship year, Dick scrubbed on all hip reconstructive procedures and performed 40 to 50 cup

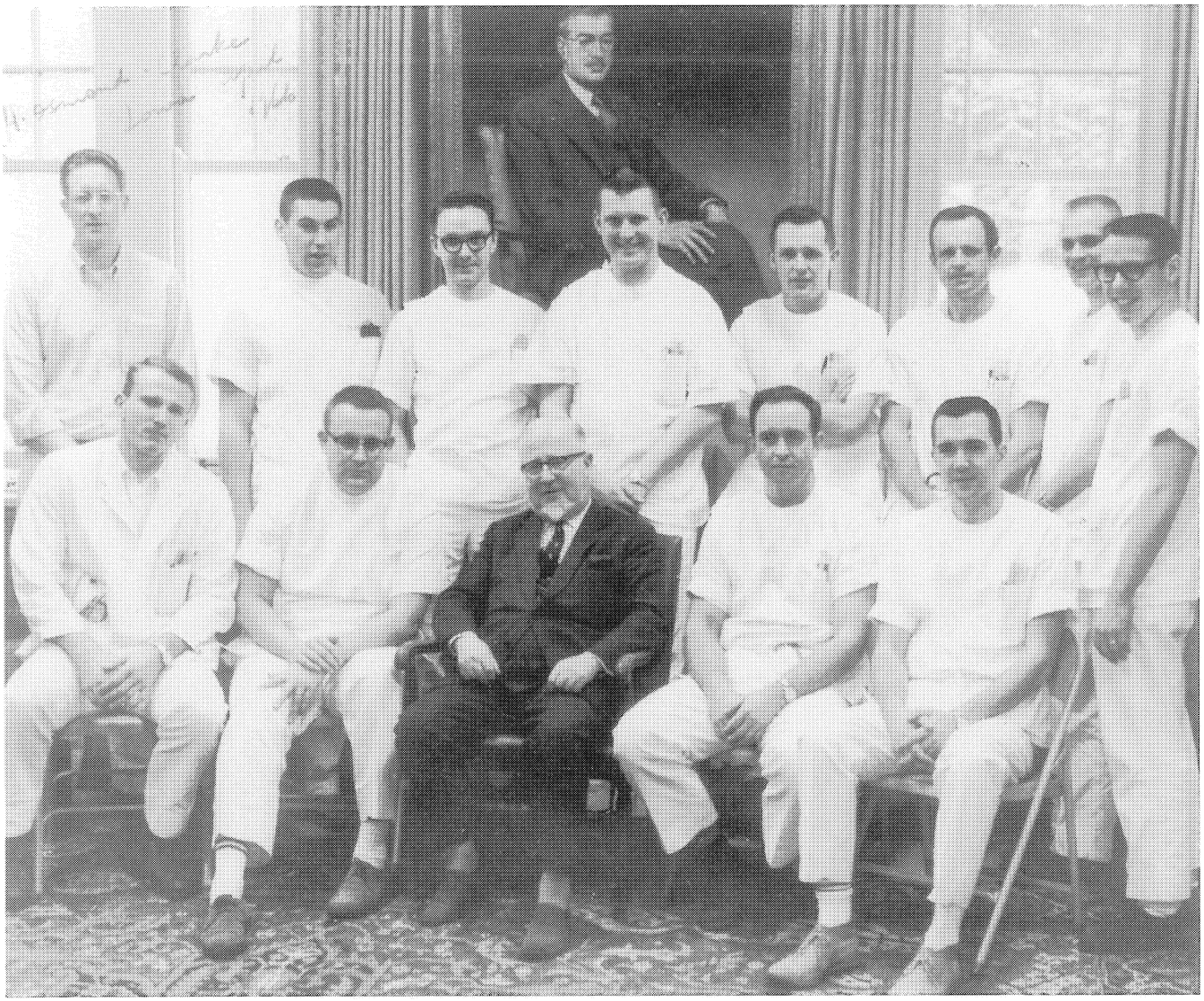


Figure 4. Picture of residents with Sir Osmond Clarke in 1966. In top row from left to right, Ray Fenner, Bob Johnson, Bill Zayler, Richard Johnston, Bob Porter, Stan James, Ron Bunton, Bill Dougherty. Seated from left to right, Andy McBeath, Steve Haug, Sir Osmond Clarke, Ed Volke, Gerry Becker

arthroplasties. He performed follow-up on Dr. Larson's cup arthroplasties[1] and wrote the article on the mechanics of cup arthroplasty[2]. He also helped develop the electrogoniometer and began to formulate ideas to study hip function for the Orthopaedic Research and Educational Foundation grant as well as the National Institutes of Health (NIH) grant.

During the same year, he visited many of Dr. Larson's friends in the hip surgery community. Dr. Larson wanted him to separate out the "wheat from the chaff." He spent three months in Boston with Otto Aufranc and his fellow Rod Turner. Dr. Aufranc was described by Dick as "poetry in surgery" the way he handled tissues and performed cup arthroplasty. While in Boston, he met William Harris, beginning a professional relationship and friendship that has flourished over the years.

He also visited Emmit Lunceford in Columbia, South Carolina to study the results of Austin Moore femoral prosthesis and McBride cups. He reviewed Bill Howe's osteotomies in Rochester, New York, McCarroll's open reductions for CDH in St. Louis, Missouri, and Ted Fox's hip fusions in Chicago, Illinois.

When Dick returned to Iowa, Dr. Larson was leaving for a year to work with the Shriner's Organization, so Dick "filled in the holes" in the department. In addition to performing hip surgery and research projects on oxygen consumption, the electrogoniometer, and the force plate, he took over the teaching of anatomy to residents from Dr. Flatt. Although he could not embarrass the residents

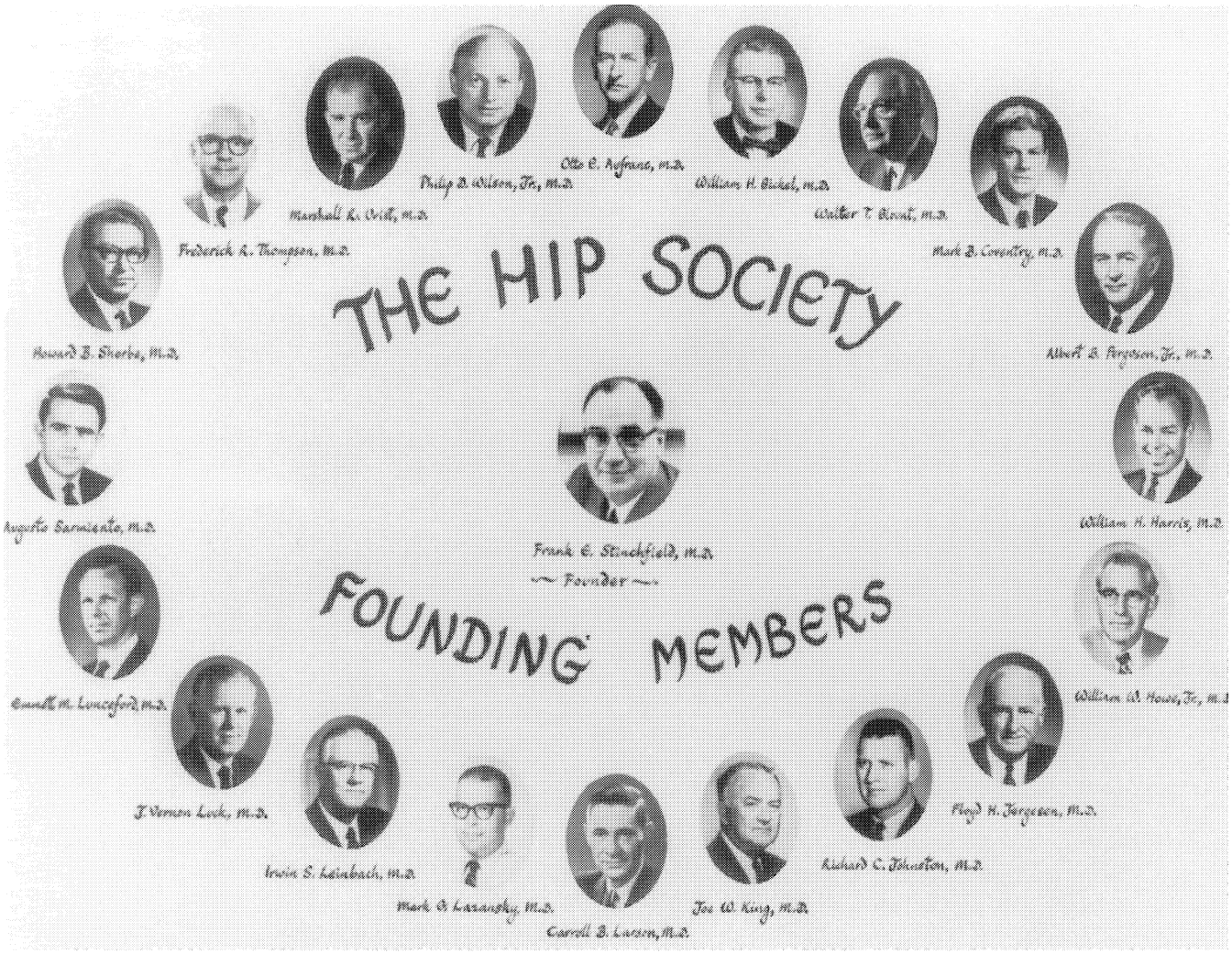


Figure 5. Founding members of the Hip Society. From top going clockwise, Otto Aufranc, William Bickel, Walter Blount, Mark Coventry, Albert Ferguson, William Harris, William Howe, Jr., Floyd Jergensen, Richard Johnston, Joe King, Carroll Larson, Mark Lazansky, Irwin Leinbach, J. Vernon Luck, Emmet M. Lunceford, Augusto Sarmiento, Howard Shorbe, Frederick R. Thompson, Marshal Urist, and Phillip D. Wilson, Jr. In the center is the Founder of the Hip Society, Frank Stinchfield.

who didn't do their homework quite as well as Dr. Flatt, Dick enjoyed the experience and learned anatomy thoroughly.

When Dr. Larson returned in the summer of 1968, he decided that Dick should travel to Europe to learn more about total hip arthroplasty. Before Dr. Larson would allow him to go, however, he made Dick perform the operation on dogs at the University of Iowa.

It was also in 1968 that Dr. Stinchfield founded the Hip Society. Dr. Larson made it clear that he would join only if Dick was invited. So, at the age of 35, Dick became the youngest founding member (Figure 5).

Through all of his studies, Dick became convinced of the need to have objective measurements of evaluation. He spent the next decade in pursuit of this goal and remains committed to it today.

Between August and October of 1969, Dick toured the major hip surgery centers in Europe. Joe Mulier from Belgium, who had trained under Dr. Larson, was Dick's gracious host in Europe. He traveled with Dick to see Drs. Maquet, Powells, Postel, Kerboul and Mueller. His interpretive skills were greatly appreciated by Dick. Dick also visited Drs. Hirsch and Riedell. After the continental European tour, Dick visited Drs. Graham Apley, John Paul, and Mike Freeman in England. In addition, he visited John Charnley at Wrightington for two weeks. He found Dr. Charnley to be a clear thinking, logical person with courage to persevere after his failure with teflon. He appreciated Dr. Charnley's wish for surgeons to watch him operate before performing the procedure so that the procedure wouldn't get a bad reputation.

On his return to Iowa in October 1969, Dick performed his first total hip arthroplasty (A McKee Farrar) on an 89

year old man. When he presented the case at conference, Dr. Bonfiglio shook his head and said, "I guess we are going to have a lot of femoral heads to look at." He continued to perform total hip arthroplasty and to perform biomechanical research related to the hip. During the years 1967 to 1970 he developed the lower extremity biomechanics laboratory. The study using the electrogoniometer to measure the hip motion required for activities of daily living was the first biomechanics paper to be presented at the Orthopaedic Research Society Meeting in 1969. Dick was blessed with energetic engineering students during this time, including Don Bartel, Dwight Davie and Ed Chao, all who have gone on to outstanding biomechanical engineering careers. Jim Andrews from the Engineering Department also provided valuable input.

In July of 1970, Dick left the University of Iowa to join his friend from residency, Ron Bunton, and John Kelley in private practice in Des Moines, Iowa. The group has expanded to 13 members over the years and has provided exemplary general and subspecialty orthopaedic care to central Iowans (Figure 6). Although he left his teachers, they all had an enduring effect on his practice of orthopaedics and hip surgery. From Dr. Bonfiglio, he obtained an understanding of injury and repair, and of cellular response. From Dr. Ponseti, he learned thoughtfulness and kindness to patients and tissues. From Dr. Flatt, he learned the importance of anatomy and surgical dissection as well as the ability to "size people up." Most importantly, he learned from Carroll Larson, his mentor. Dr. Larson was "a role model at approaching people objectively in an unemotional realistic manner but with dignity." Dr. Larson was concerned not only with how good a hip joint was but also how it affected life. He was interested in comparing how hip disease affected life preoperatively and projecting how it could improve the patient postoperatively. He then projected the risk-benefit ratio of this intervention. He was a thoughtful investigator.

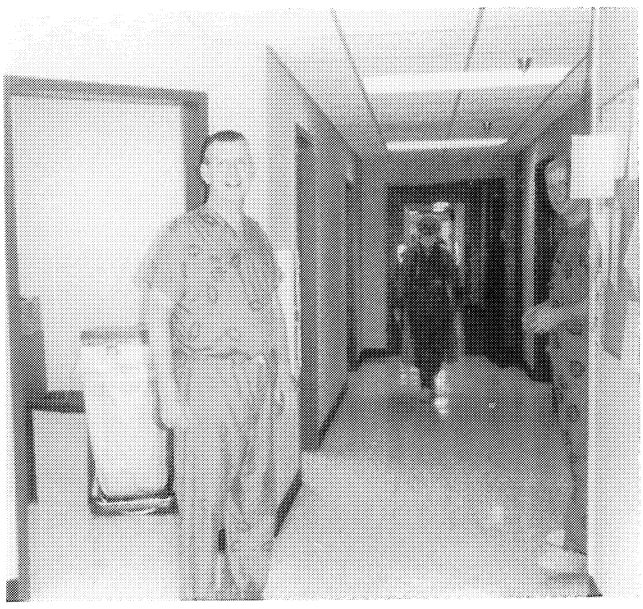


Figure 6. Dr. Johnston on rounds with his partner, Pat Sullivan at Methodist Hospital in Des Moines.

Although Dick left the University in 1970, his career in Des Moines has been no less academic. He remained extremely active in hip biomechanics research in the department. He commuted to Iowa City From Des Moines every Friday during the 1970's and early 1980's. For much of the 1970's, he was the principal investigator on the NIH Hip grant.

Shortly after Richard Brand joined the orthopaedic surgery staff in 1974, he sold Dick on the concept that a good engineer could work with orthopaedic surgeons. Dr. Johnston gives full credit to Richard Brand for hiring Roy Crowninshield in 1975 for this job. Roy became an endeared colleague and friend over the next eight years. (He left in 1983 to pursue a career in industry with Zimmer.) In collaboration with Richard Brand and Roy Crowninshield, much of our present understanding of hip joint forces and cemented prosthetic hip design were developed. Classic manuscripts including "Reconstruction of the Hip: A Mathematical Approach to Determine Optimum Geometric Relationships,"[3] "An Analysis of Femoral Component Stem Design in Total Hip Arthroplasty,"[4] "An Analysis of Collar Function and the Use of Titanium in Femoral Prostheses,"[5] and "The Effect of Femoral Stem Cross-Sectional Geometry on Cement Stresses in Total Hip Reconstruction"[6] were published during that time period. A Kappa Delta and a Hip Society Award came from these research efforts. In addition, the Iowa Total Hip was developed. Dr. Johnston has designated all royalties from this prosthesis to be used by the University of Iowa Orthopaedics Biomechanics Laboratory. These funds presently total over one million dollars.

During the 1980's, Dick continued to personally evaluate his Charnley total hip replacement results with regular patient follow-up and began to evaluate his results with the Iowa prosthesis. He did this while continuing to perform large volumes of hip and knee arthroplasty surgeries (He has performed over 5,000 total hip arthroplasties and 3,000 total knee arthroplasties).

Dick's thorough preoperative and follow-up evaluations have enabled the residents and myself to report the results of all aspects of his hip surgery. During these investigations, Dick has allowed full access to his records, a testament to his honesty and search for the truth. As Dr. Ponseti put it, "We have examined his practice inside and out like a thorough gastrointestinal examination." These studies include the 20 year Charnley follow-up,[7] the 16 to 22 year follow-up of patients under age 50,[8] as well as

Figure 7. Dick and Jan on a combine at Dick's son's farm outside of Des Moines.

many other aspects of revision and primary hip surgery using cemented and hybrid fixation.

Dick has had a profound effect on many of his students and colleagues. Every resident that takes the opportunity to watch Dick operate at Methodist Hospital in Des Moines leaves realizing they have witnessed an orthopaedic and surgical experience that cannot be matched. Whether he is doing a routine primary or difficult revision procedure, his surgical exposure is atraumatic and appears effortless. An example of his extraordinary surgical skills is his ability to dissect out the lateral femoral circumflex vessels with a beaver blade before ligating them. He does this in every case before performing a complete capsulectomy. The harmony and efficiency of his surgical team is something every surgeon dreams about. This is largely due to his assistant and wife, Jan. During my time as a resident, the older residents would all say, "You have to see Dick operate, and wait until you see how his nurse helps." I have always said that if Dick couldn't do my hip replacement, I would at least want Jan there to help. Today Dick spends much of his free time with Jan and continues to dabble in farming (Figures 7 and 8).

A number of the present residents have had the opportunity to do their senior research projects with Dick. Most would agree that the ability to quickly obtain lists of patients, all of their long term radiographs (which are kept at Dick's office), and to have the help of Dick's staff (including his nurse, Pat Kratz, and research assistant, Jason Olejniczak) in contacting patients is much appreciated. By talking to patients at follow-up and reading letters they have sent to Dr. Johnston (he keeps these in their charts), one really develops an understanding of the long term improvement in function and decrease in pain that Dick has provided by performing total hip arthroplasty on patients suffering from arthritis. At these reviews one also appreciates Dick's ability to relate to his patient's medical and social situation.

In the world of academic hip surgery, Dick has been a big brother or father figure to many of us. The list of Hip Society members shows a large Iowa contingent that includes Denny Collis, Andy McBeath, Bill Capello, Richard Brand, Stuart Weinstein, Roy Crowninshield, Tom Brown, Don Bartel, Ed Chao, and myself. After Hip Society dinners there have been many a late cocktail hour going over old Dr. Larson stories as well as other Iowa residency stories. On such occasions it is easy to appreciate Dick's warmth and personability as well as his keen sense of humor.

At this stage in his career, in many ways, Dick is just beginning to be fully appreciated for his skills. Although he was president of the Hip Society in 1981 at the age of 48, he has just recently served as the third President of the American Association of Arthritis Hip and Knee Surgeons. He is an active member of the International Hip Society which has only 20 United States members. Dick has remained extremely active in the development of outcome tools to properly assess total joint arthroplasty results. He is committed to helping define the functional improvement

Figure 8. Dr. Johnston and the operative team in action.

obtained from total joint arthroplasty in economic terms.

In 1992 the "Richard C. Johnston Adult Reconstruction Lectureship" was started. It is held every other year when there is not an Iowa Alumni Meeting. At each Lectureship we have a distinguished lecturer and a distinguished alumni lecturer. In 1992, the distinguished lecturer was Dr. William Harris and the distinguished alumni lecturer was Dr. Dennis Collis. In 1994 the distinguished lecturer was Dr. Jorge Galante and the distinguished alumni lecturer was Dr. William Capello. On October 11 and 12, 1996, we will have the third lectureship. Dr. Eduardo Salvati will be the distinguished lecturer and Dr. Andrew McBeath will be the distinguished alumni lecturer. Those of you who have had the opportunity to attend the Lectureship can attest to the influence that Dick Johnston and the legacy of Dr. Larson have had on the evaluation and understanding of patients with hip disease and on the operations devised to functionally improve those patients suffering from hip arthritis.

ACKNOWLEDGMENTS

A special thanks to Dennis Collis, Andrew McBeath, William Capello, William Harris, Roy Crowninshield, Richard Brand, and Ignacio Ponseti for their help in preparing this manuscript.

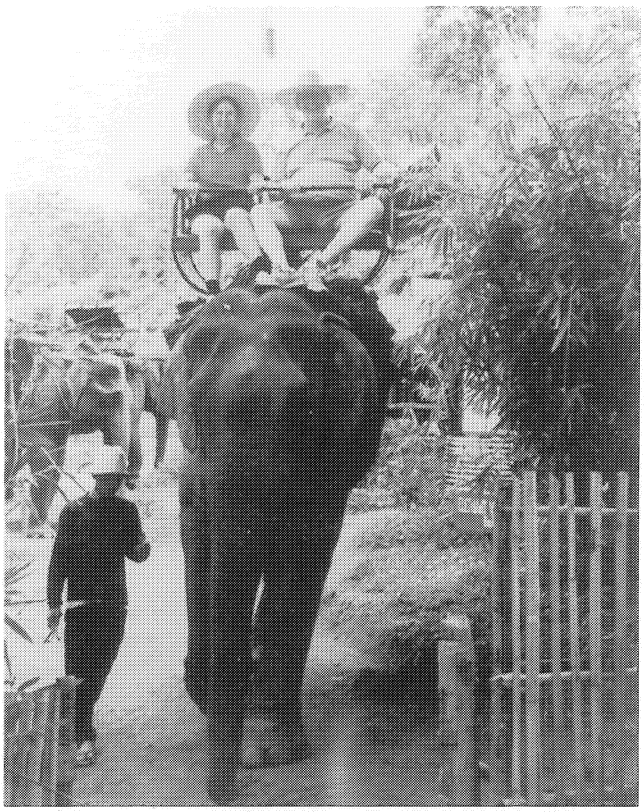


Figure 9. Dick and Jan on an elephant ride during a trip to Asia for the International Hip Society Annual Meeting.

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WORDS

Adrian E. Flatt, M.D., F.R.C.S.

ON BEING AN EDITOR

"WORDS KEEP GETTING IN THE WAY OF WHAT WE ARE TRYING TO SAY."—RONALD REAGAN

If the great communicator is in trouble so are we all. English is the richest language with the largest vocabulary on earth[1]. It behooves us to use it with skill and precision.

When Julius Caesar landed in Britain 2000 years ago, the English language did not exist[1]. It began as a rude tongue spoken by Germanic tribes who had invaded Britain in the 5th century. Later it was augmented by the Scandinavian words of the Vikings as they raped and pillaged their way through the 9th century. England was invaded—for the last time—in 1066 by the French. They too left many words in the English language[2].

This dialect, originally spoken in one small island, has become the most cosmopolitan and the most democratic of all languages[3]. It is unique among contemporary languages because it accepts new words and is more friendly to change than any other language[2].

Nearly 350 million people speak English as their primary language, over 400 million use it as a second language, and one out of every seven persons in this world has some knowledge of it[3]. It is the international language of aviation; China has more English broadcast programs than Canada; Russia has more than Australia; and, when Gandhi appealed for an end of violence after the assassination of his mother, he had to speak to his nation in English[1].

English is the richest language with the largest vocabulary on earth[4]. *The Oxford English Dictionary* lists over 500,000 words compared to German which has about 185,000 words and French which has less than 100,000, including "le hit parade."[1] It is brevity of use which is the strength of the English language. If one translates a paragraph written in English into French, the latter will need about one-third more of a page to transmit the same message.

English can express shades of meaning that aren't even possible in other languages. For example, one can distinguish between sky and heaven; the French, Italians and Spanish cannot. The gender of a noun in modern English is determined by meaning and does not require a masculine, feminine, or neutral article. In French, the moon, "la lune," is female, while the sun, "le soleil," is masculine. However, in German the moon is male and the sun is female. As Mark Twain put it, "In German, a young lady has no sex, but a turnip has."[1]

Modern English has two vocabularies. One is Germanic made up of native English words and words borrowed from other Germanic languages. The other comes mainly from Latin. Thus, there are two basic ways of saying anything in English. When using Latin derivatives, one increases the length of the words and the length of the sentences. Nobody objects to using technical terms derived from Latin or Greek if there is no suitable English equivalent. When there is a choice of words, the shorter is usually Germanic. "Most" is Germanic while "the majority of" is Latin.

Today about ninety percent of substantial scientific advances are reported in English. In practice there are two scientific English languages: British and North American. The divergence is considerable and it continues to grow, for there are substantial differences in both vocabulary and spelling[4]. George Bernard Shaw pointed out that "England and America are two countries separated by a common language."

Creative writing in English is fun. One can rummage around and come up with exactly the right word to express one's thought. If English is such a wonderfully expressive language, then why were most of the manuscripts I received so awful? The problems are well summed up by the accomplished novelist, Michael Crichton, who earned his M.D. degree at Harvard, but never practiced. He has chastised us in an article "Obfuscation in Medical Writing." His complaints are many: poor flow of ideas, verbiage, redundancy, repetition, wrong words, poor syntax, excessive abstraction, unnecessary complexity, excessive compression, and unnecessary qualification. He also complains that in medical writing the voices are passive, verbs are transitive, modifiers are abstract and qualifying clauses abound[5]. In summary, it is probably fair to say he thinks medical writing stinks. He makes a good point that the general tone is one of extreme timidity for going far beyond sensible caution. He typifies it by describing the eminent surgeon who strides purposefully into the operating room each day; but to read his papers you wonder how he finds the courage to get out of bed in the morning[6].

Michael Crichton is not alone in his complaints. Doctor Ingelfinger, that great former editor of the *New England Journal of Medicine,* grumbled about the massive infiltration of language by the words of an extravagant technology, authorship by committee, and the failure, even inability, of doctors to read. All these contribute to the

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poor manuscripts that editors receive[7]. It seems that the inferior quality of most medical writing is now accepted as a fact of life[8].

AN EDITOR'S TASK

Journals and editors survive by publishing good papers. A good paper is one that takes a definite structure, makes its point, and then shuts up. A good manuscript uses nouns and verbs and not adjectives and adverbs. The scientific structure is crisp and each individual section does what it is supposed to do and no more[9]. Despite Michael Crichton's view that the prognosis is hopeless, medical writing is being revived, at least in part, by the efforts of dedicated reviewers who select out the manuscript with potential, and of editors who distill the message by cutting away the verbiage and improving clarity[10]. When I submit a paper for publication, I certainly agree with Victor C. Strasburger who wrote, "I like to think of medical papers as a kind of present we authors give to the journal."[8] As an editor receiving these presents, I sometimes wondered why so many generous authors showered such illiterate and deadly dull gifts upon me.

Review and judgment by one's peers seems to be inherently, if not a perfect system, at least the best of many alternatives. It is the basis of our jury system, and is the method used by all authorative subscription journals. Most controlled circulation journals, or "throwaways," do not use the peer review system. When reviewers are allowed to remain anonymous but also know the provenance of the manuscript, there is the possibility of bigotry and prejudice. New or young authors may find difficulty in getting their work published and there could be delay in them publishing innovative work[11].

I firmly believe that reviewers should not know the provenance of the papers they receive. I carefully removed all identifying data in the manuscripts I sent for review. When non-blind refereeing practices are used, serious problems can occur. A study published in *The Behavioral and Brain Sciences* illustrated this problem[12]. Twelve previously published research articles by investigators from prestigious and highly productive American psychology departments were selected from twelve highly regarded and widely read American psychology journals which have 80% rejection rates but non-blind refereeing practices. These papers were formally resubmitted to the journals that originally published them, but with fictitious authors' names and institutions. All had been published within the last eighteen months to three years. Three articles were detected as resubmissions, therefore nine papers progressed to review. Eight out of the nine were rejected. Sixteen out of the eighteen referees (89%) recommended rejections and their editors concurred. In many cases, rejection was based on "serious methodological flaws."[12]

At least my authors were not subjected to such problems. I compared two reviewers' recommendations with my own and made the vital decision: accept, reject or maybe. Few were accepted or rejected outright. Most papers have some message worth publishing, but their authors seem to have a terrible problem defining the message. Authors offer journal editors their brain child, sometimes their first born, with pride and trepidation. I have always believed that the parents' style should be allowed to show through the printed word. Most contributions have a kernel of value buried amongst their verbiage. It is the editor, who by his letter, encourages the author to disinter the value and present it in better fashion.

Sometimes there is no hope and a rejection letter must be written. I hated to do this and always tried to point to some good idea, phrase, or comment. The letter must offer encouragement of the future but bury the rejected contribution. Rejections are sometimes couched in fierce and unforgiving terms. My former chief, Sir Reginald Watson-Jones, once wrote, "Dear Sir, Of course we cannot publish this; it is really quite incredible that you ever thought we might." A "Peanuts" cartoon had editor Snoopy typing a letter, "Dear Contributor: We have received your latest manuscript. Why did you send it to us? What did we ever do to hurt you?" The editor of *The British Medical Journal* recently published a letter he longed to send, "Your paper was good and original, but sadly the good bits were not original and the original bits were not good."[13]

Having done the interesting part—the research—it is always hard to start writing the paper. Some authors have a terrible time getting started. Charles Darwin wrote to his children, "Observing the intricate functions of nature is sheer pleasure, but writing is sheer pain." I believe that the editor's responsibility is to help the contributor report his work in as short but as distinctive a way as possible[14]. Therefore, I wrote a detailed and constructive letter to my authors. Unfortunately physicians usually are not used to having their authority questioned very often, if ever!![15] My letters were not always appreciated, and indeed, some authors got downright angry. Luckily our society is not yet so egalitarian as to have equated freedom to publish with the "right" to be published[14].

The relationship between the author and editor can be a touchy one and needs to be seen as a mutually beneficial joint undertaking. Authors do not seem to understand that the aim of scientific communications is to convey information succinctly, accurately, and unequivocally. Editorial surgery has to be done with great delicacy, but the task must be done. A good way to start was suggested by Dr. Whimster, "Cut away the obvious verbiage."[6] Then I looked at the grammar in a simple way. I checked all verbs

for their subjects, all pronouns for their nouns, and all adjectives and adjectival clauses to see what noun they were telling more about. Then all adverbs and adverbial clauses were checked for the verbs they modified. Finally, every dull, passive construction was examined to see if it could be made more active.

I tried to retain the author's way of presenting his views, but inevitably, I would have to enter changes. This led to an unevenness of style in the contents of any one issue, but I don't think that is all bad.

Some journals rewrite the papers they accept into a common mold; usually extremely dull reading. Doctor Ingelfinger described the *Journal of Bone and Joint Surgery* as "solid as a colonial empire in Victoria's day."[16] I have always thought of it as the old lady of Shattuck Street. The noted medical historian, Doctor Henry E. Sigerist, wrote of famous medical journals in which every article is edited or even entirely rewritten by a highly competent staff of elderly ladies following rigidly set standards. The result is that every article reads as if it has been written by the same person. He stressed that "No two individuals are alike and no matter how factual an article is, we should be allowed to get a grasp of the personality behind it."[17]

Such rewriting provokes the common criticism that editors meddle with an author's "style." Unfortunately, a distinctive style is an unlikely attribute of the average physician/writer, who has neither the time nor the requisite inherent talents needed to develop a personal writing style like Dr. Somerset Maughan, Conan Doyle, or Lewis Thomas. Certainly rewriting can be overdone. The aim is to produce simple, direct, and unambiguous sentences. However, I do agree with those who contend that too vigorous shortening reduces scientific papers to the literacy level of a laundry list.

Lawrence D. Grouse has, over the years, complied a laundry list of another kind[18]. He has identified a group of outlaw manuscripts which are clinical articles artfully crafted to confuse and mislead the reader. They are:

SHELL GAME Submits to two or more journals at the same time an original article, a clinical note, and a letter to editor.

CLONE Distributes widely and infects the entire literature with marginally dissimilar fragmentary studies.

ZOMBIE Never dies. "Revised version breathing heavily in the mail. Shaman author makes incessant phone calls."

CHAIN LETTER For each new version, two new authors added as first and second, final two dropped and others moved back two places, and one new table is added.

GAY DECEIVER Link paper with some large medical problem, i.e., AIDS. The media tends to cover these papers.

MAD BOMBER Attacks respected colleague.

ANKLE BITER Trusted collaborator tries to scoop his associates.

BAD WRITING

Probably the best way I can explain an editor's problem is to give some examples of the verbal pathology I faced in the manuscripts I accepted. Even the casual reader of medical books and journals gets a vague feeling that most of the writing is obscure and dull, and only occasionally is it attractive and informative. Lester S. King, a former editor of the *Journal of the American Medical Association*, believes the faults relate to the defects of modern American education, particularly the lack of education in language and humanities[19]. Unfortunately, 27 million adult Americans are illiterate, unable to read a newspaper advertisement or even a product label[2]. It was incredible to this editor how many of them seem to become successful surgeons, at least judging by the manuscripts I received!

The major faults of current scientific writing are:

1. The automatic use of the passive voice and impersonal style.
2. The padding of sentences whereby timid, inexperienced writers who think that their sentences do not have sufficient impact add a second and sometimes a third thought to the first before nailing a period in place.
3. The use of pompous words by naive authors who, trying to impress the readers, reach for the impressive word and achieve the exact opposite[20].

Editors abhor the deliberate avoidance of the words "I" or "me." Mark Twain, a wonderful wordsmith, pointed out that "only Presidents, editors, and people with tape worms have the right to use the editorial 'we'." Authors are responsible for their contributions. It does no good to hide behind the passive voice. Passively one would write, "The median nerve was cut by the surgeon." More courage, fewer words, and an active voice would have produced, "The surgeon cut the median nerve" or, more truthfully, "I cut the median nerve."

Pompous words are best avoided. Winston Churchill stressed that "Short words are best; old words, when short, are best of all." Even Julius Caesar advised that "an unusual word should be shunned as a ship would shun a reef." Consider this shipwreck of a sentence: "The patient underwent a left upper limb amputation performed by our surgical team." The passive voice is used, the style is impersonal and pompous, and a pejorative word is used (underwent = ? torture). What it means is "we cut off the patient's left arm." Short, sharp words and only seven instead of eleven.

Use short words in sentences which should be short. The longer the sentence, the more likely it is that the beginning can be amputated and the deadwood can be found in the middle. "Appears to be suggestive of the possibility that" has used seven unnecessary words to say "suggests."[21] Of course, authors need to present the facts, but there is no need to repeat what everyone already knows, nor to labor every point six times, repeat it in a table, illustrate it in a figure, show it in a histogram and then say it twice more in the summary[22]. Most factual writing gains clarity by being shortened. To shorten a passage, one of the best sites to examine is the beginning of paragraphs or sections. Authors frequently reveal their difficulty in getting started when they use roundabout phrases and clauses ending in "that": "Quite briefly, may I point out that . . .", It has been our observation that . . .," "It is obvious at a glance that . . . ."[23]

The use of polysyllabic abstract nouns, conceptualization, or prognostication is not impressive; it is boring[10]. Some advise substituting a short word for every long word, but that is foolish. The good writer selects the most appropriate word for his context. Problems arise when the physician encumbers a simple thought with a profusion of long words and chooses the more pretentious term for a non-medical word: "approximate" for "about," "termination" for "end," "ambulate" for "walk."

Even worse is the general spread of polysyllabilism. The "poor" have vanished, instead we have "the underprivileged" and "the disadvantaged." The "city dump" has become a "sanitary landfill facility." "Men at work" has become a polysyllabic jungle: "Our people resources in action making it easier for your people resources to do business in a dynamic ever-changing competitive environment." Government obscurantism is rampant. Short definite words are shunned. "Finish" is "finalize," "do" is "implement," and "now" is "at this point in time."

HOW TO CONSTRUCT A PAPER

Before you start, promise yourself whatever you write will be interesting to your readers. Lois de Bakey feels that many medical papers have the literary value of a weather report, conforming to a common mold in which different data are simply inserted[24]. It is true that there are certain commonly accepted patterns of writing, but their contents do not have to be dull.

Originality in scientific writing is hard to achieve. There are only a few ways to neatly express important thoughts and sometimes they are kidnapped. The Reverend Dean Inge defined originality as undetected plagiarism. Plagiarism generates a lot of indignant accusations and has been characterized by Grey Easterbrook as "the world's dumbest crime. If you are caught there is absolutely nothing you can say in our own defense."[25] Others differ. Anatole France's advice was, "When a thing has been said and said, we have no scruples, take it and copy it." In between is the mass of us writers who, despite a choice of over 500,000 words, may inadvertently repeat or paraphrase some apt saying. (This short essay may well be guilty.) Plagiarism is an occupational disease of those of us who write. However, there should be honor among thieves and sources should be acknowledged. In recently reviewing twelve years of literature for the revision of a book, I have been interested, and occasionally flattered* by seeing how much is copied from one author to another. I've always enjoyed the three quotations below but who had the idea first, goodness knows.

"A good composer does not imitate, he steals." Igor Stravinsky, 1882-1971.

"Immature poets imitate, mature poets steal." T.S. Elliott, 1888-1965.

"Immature artists imitate, mature artists steal." Lionel Trilling, 1905-1975.

There is no such thing as *the only way* to prepare an article. A good way to prepare a scientific paper is to pose Bradford Hill's five classic questions[26]. What did I do? How did I do it? Why did I do it? What did I find? What does it mean? The editor will certainly ask these questions. The two hardest parts to write are the title and the abstract. Each needs clarity, specificity, and brevity; neither can be written until the paper is complete.

THE ABSTRACT

An abstract is the most important part of a paper because, although it is written last, it is looked at first by virtually all readers. An abstract is a self-contained document which is the bridge between the title and the full article. The title should announce the topic and the abstract succinctly develop it[27]. An abstract should not be a mere recital of the contents full of expressions such as "is considered" and "is described." It should be a concentration of the essential qualities of the paper. There are five absolute requirements:[28]

*No pun intended

1. It must be understandable without reference to the paper.
2. It must set forth in specific terms all the major points of the paper.
3. It should be one paragraph of not more than 100 words.
4. It should preserve the form of the paper in miniature.
5. It must not contain material not covered in the paper.

An abstract that did not meet these criteria appeared in a recent throwaway journal. "We found that diagnostic arthroscopy of the shoulder is systematic and reproducible when attention is given to normal and variational shoulder anatomy and consistent technical detail."[29] It simply makes no sense. Economy of words is virtuous, but let's not follow the foolish virgins and run out of the oil of intelligibility.

I had to rework an abstract for a paper I accepted. The version the author sent read:

> "The distal radius is a common site for giant cell tumors. Treatment of these lesions may involve thorough curettage of the tumor and packing the cavity with polymethylmethacrylate cement. This is a report of a case of spontaneous rupture of the extensor pollicis longus tendon following such a procedure. It was treated successfully with tendon transfer of the extensor indicis proprius to the extensor pollicis longus. Proposed mechanisms for the pathophysiology of the rupture are discussed."

The original version used 76 words. By reworking it, the same message was given in 59 words:

> "This is a report of a case of rupture of the extensor pollicis longus tendon following treating a giant cell tumor of the distal radius by packing the cavity with polymethylmethacrylate cement. The lack of extension was treated successfully with tendon transfer of the extensor indicis proprius to the extensor pollicis longus. The pathophysiology of the rupture is discussed."

The final version used only forty words. Note that the first eight words have been eliminated. There is no need to say the obvious, that it is a case report.

> "An extensor pollicis longus tendon ruptured after a giant cell tumor of the distal radius was excised and the cavity packed with polymethylmethacrylate cement. Thumb extension was restored by transferring the extensor indicis proprius tendon to the extensor pollicis longus."

THE TITLE

An accurate title of a few words, but much substance requires considerable verbal ingenuity and discipline[30]. A title needs clarity, brevity, and specificity. There are few good titles around, and an awful lot of bad ones. Some are ambiguous: "Joint Study of Orthopaedic Problems." Some are grammatically incorrect: "Toxoplasmosis in Humans Derived from Cats." Some are simply illogical: "Survival after Drowning."[30] I had a weird experience once, with the Old Lady of Shattuck Street**. I submitted a paper titled "A Locking Little Finger," a short, informative, alliterative title. The paper was accepted, but the title was changed to "Intermittent Obstruction to Full Extension of the Metacarpophalangeal Joint of the Fifth Digit." Fourteen words instead of four! Eventually I won, but it took a lot of letter writing.

THE BODY OF THE PAPER

It is in the body of the paper where the author has a little license. Success depends on the choice of words and their grammatical arrangement. There are eight parts of speech: nouns, verbs, adjectives, adverbs, pronouns, prepositions, interjections, and conjunctions[20]. If the nouns and verbs are selected carefully, there is less need for adjectives and adverbs; "lurched" rather than "had an unsteady gait."[20] These individual components of a sentence all have their proper place and responsibility in communication. There are basic rules which should have been taught to you in school. When followed, they make reading a manuscript a delight. There is not much written delight around nowadays.

In fiction, written words are sacred; in medical writing, words cost money and journal space is scarce[8]. Thus, the choice of words is vital. A century ago Humpty Dumpty said, "When I use a word, it means what I choose it to mean, neither more or less."[31] However, for lesser mortals, words do have set meanings and part of the pleasure of writing is training oneself to select and use the exact word, not its first or second cousin[31]. The author John Irving believes that ninety percent of all writing consists of 1000 basic words[33]. Word lovers use words sparingly, choose them artfully, and arrange them dexterously to make the most of each[34].

I regard the following short paragraph as the epitome of good precise medical writing that cannot be misunderstood:

> "They say man has succeeded where the animals fail because of the clever use of the hand, yet when compared to the hands, the sphincter ani is far

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***The Journal of Bone and Joint Surgery* is published from Shattuck Street, Boston

> superior. If you place into your cupped hands a mixture of fluid, solid, and gas, and then through an opening at the bottom, try to let only the gas escape, you will fail. Yet the sphincter ani can do it. The sphincter apparently can differentiate between solid, fluid and gas. It apparently can tell whether its owner is alone or with someone, whether standing or up sitting down, whether its owner has his pants on or off. No other muscle in the body is such a protector of the dignity of man, yet so ready to come to his relief. A muscle like this is worth protecting."[35]

For another example, read Mark Twain, a writer for whom the English language was a playground.

THE USE AND ABUSE OF WORDS

To help in the choice of words, there are two essential tools, a dictionary and a thesaurus. I believe *Stedman's Medical Dictionary* has the right approach. "A dictionary, if it is to be a useful guide to a living language, must spell, pronounce and define words as they are used, not wistfully as they should have been."[36] The best known thesaurus was written by a physician, Roget, who obtained his M.D. from Edinburgh in 1798 and retired from practice at age 61 in 1840. By 1805, he had completed a classified catalogue of words which was the converse of an ordinary dictionary—not with the words arranged in alphabetical order, but according to the idea which they expressed[37]. Use this book. It will help you avoid dull repetitious writing, but don't get carried away; some of the words are somewhat exotic.

In English, nouns become verbs and verbs nouns in a way that is impossible in other languages,[1] but the use of nouns *and adjectives* as verbs is now a major epidemic: optimize, impact, interface, maximize, institutionalize, finalize, and prioritize[38].

LOGOMEGALY

When examining the physiology and pathology of the prose we use in our journals, one is struck by the great variety of the lesions[39]. Probably the most common is logomegaly, "a great swelling of words." Expansively described by the *British Medical Journal* as "A condition which threatens the body corporate with malignant possession growing upon our most sensitive faculty, our common speech, and thereby depositing dangerous secondaries in the common mind."[7]

One of the most common forms of logomegaly is the cliché. Clichés are highly infectious, once allowed into a piece of prose they breed like ravenous pathogens. Cliché infected writing doesn't just depress readers, it obstructs understanding[39]. "Cliché" is derived from the French verb "clicher," meaning "to cast from a mold" or to stereotype[40]. Theodore Bernstein has stressed that clichés are not all bad. When Archimedes' bath ran over, he was perhaps justified in sprinting into the street. But that does not mean every kid can rush out of his bath naked yelling "Eureka!" The distinction is between the coiner of a bright phrase and the mere echoer of that phrase. It is the echoing that turns the phrase into a cliché[41].

Clichés come in many forms. Aphorisms (life is short, science is long), Biblical quotations (physician, heal thyself), mythology (Herculean task), foreign phrases (in extremis), figures of speech (at death's door), paradoxes (operation success, patient died), epithets (mad scientist), pet phrases (major breakthrough), vogue expressions (multifactional etiology), slang (brittle diabetic), and empty inanities (all in all)[40].

There is also the *curdled cliché*; Samuel Goldwyn consciously created them. ". . . they're always biting the hand that lays the golden eggs." But Goldwynisms always seem contrived. Addicts usually confine their efforts to speech. The politician often unconsciously creates curdled clichés. They result from a single-barreled effort and a double-barreled deficiency[41]. Mayor Jane Byrne of Chicago started a whole subculture of "fruitworthy" phrases when she hoped a certain investigation would prove "fruitworthy." This linking of the two good words "fruitful" and "praiseworthy" produces an abomination.

One doesn't find too many curdled clichés in medical writing, but the regular variety can be culled from any manuscript. Examples I have plucked out are: "A valuable addition to our therapeutic armamentarium," "aggressive surgical attack," "as can readily be seen," "beyond the scope of this paper," "decision making process," "it does without saying," "uneventful recovery," "vast majority," "warrants further investigation," "state of the art," "renders inoperable," "surgical intervention," "multidisciplinary approach," "stimulating concepts," etc.[41] People tend to build clichés on other unnecessary nouns, as in "on a professional basis" instead of "professionally," or "over a 12-day period" instead of "for 12 days."[39]

EUPHEMISMS

Euphemisms are another form of logomegaly. They are longer and less precise than the words or terms they replace. A low bridge is "impaired vertical clearance," a desk is an "individual work station." A garbage collector is a "solid waste ecologist."[30] They are often used as a way of getting around saying something unpleasant. The phenomenon of death has become encrusted with euphemisms. I even got a manuscript in which some animals were not sacrificed, a pejorative word anyway, but were "euthanized." Similar genteel hedging sometimes represents academic cowardice, "It may be reasonable to

suggest that necrotic effects may possibly be due to involvement of some toxin-like substance." "Necrosis may be due to toxins."[10]

OXYMORONS

Oxymorons are another padding device used, one hopes, inadvertently by the author. Defined as a figure of speech by which a locution produces an effect by a seeming self-contradiction. It is better defined by Warren S. Blumenfeld as "Contradictory expressions that make absolute sense." In more common speech, there are endless examples: "routine emergency," "jumbo shrimp," "legal ethics," "scholar athlete," and "instant classic."

STACCATO WRITING

The contrast to logomegaly is such sparse staccato writing that it is almost unintelligible. Foreign manuscripts seem to think it a sin to use the inoffensive article "the," a habit that is increasingly apparent in native born manuscripts. Bernstein blames this on *Time* magazine or what he calls, "the school of Luce writing." Not only must no story begin with "The," but in addition, no sentence may begin with "The." "Cause of the disease was . . . ." Nor is the beginning of the sentence the only point attacked by this cancer. Nouns in the middle of a sentence now occasionally suffer the loss of their articles too, "Cause of disease was . . . ."[41]

MISUSE OF WORDS

Every editor has his pet peeves, and one of mine is the factual misuse of words. Doctor Philip Eibel has collected a number of examples[42]. "The patient was medicated four times daily." Medicate means to impregnate or charge, as in medicated soap. I doubt very much the patient's entire nature is radically changed four times a day. However, the soap is permanently changed. "The patient was symptomatic." How can he be? Symptomatic is characteristic or indicative of "The patient had marked pain." What was the pain marked with or by whom was it "marked"? "He had *severe* pain."

Ambulate is often wrongly used as a transitive verb. *The Oxford English Dictionary* gives only four examples of its use and then only as an intransitive verb. There are *no* transitive verb examples[42]. Anyway, it's a pompous word. Why not use "walk"?

Equally abhorrent is the use of five or six words to express something that can be conveyed by one word. "In view of the fact that," instead of "since." "In the opinion of the author," instead of "I believe." "In the majority of cases," for "usually." "Serves the function of being," "is."[21]

Another common error is unskillful positioning of the words. Position is crucial to conveying the message clearly[43]. The way words are arranged may mean the difference between "writing that only a mentor could praise, or writing that a mentor could *only* praise."[43] The misplaced modifier can cause ambiguity, misstatement, or inadvertent humor. "Being moribund, the Doctor could do nothing for the patient. The patient was referred to a psychiatrist with a severe emotional problem."[44] "The patient was carefully followed by a doctor and nurse."

"ABBREVS"

Even worse is the use of abbreviations. No one is allowed to create his own anencephalic lingo. Unfortunately, mental flaccidity, impatience and ignorance of proper English has led to such a proliferation of these abominations that hospital record rooms are forced to issue lists of "acceptable abbrevs"[44]; editors do not abide such lists.

GRAMMAR

Grammar is a set of principles by which language functions[45]. There is no universal grammar; like every language, English has its distinctive grammar[46]. It is one of great simplicity and flexibility. Since all living languages are continuously changing the way they operate, grammar must change accordingly[24]. Some people consider themselves, grammarian legislators, but they are not. The rules that they derive are like laws of nature, they are generalized formulas based on the way the language operates[24]. That well known author, spy (in World War I), and sometime physician, Somerset Maugham, commented that "Grammar is common speech formulated." Lois de Bakey believes that a piece of writing should not be judged on the basis of an occasional grammatical lapse. It is the overall grammatical integrity and style that should be used in evaluating it.

THE INFINITIVE - SPLIT

The infinitive seems to be in constant trouble, to split or not to split, that is the question. Eighteenth and 19th Century grammarians for one reason or another frowned on such splitting, and most grammar teachers have been frowning ever since. The natural position of a modifier is before the word is modifies. Thus, the natural position for an adverb modifying an infinitive should be just ahead of the infinitive and just after the "to."[41] I believe that to deliberately split an infinitive is no sin. The placement of adverbs also causes a lot of problems. Writers blinded by the split infinitive obsession seem determined not to split anything but hairs. More often than not the proper and natural place for an adverb is between the parts of a compound verb. Ambiguity can be caused. "Three plans for removing sutures gradually were proposed by the staff."[41] It is better to say, "Three plans for gradually removing sutures . . . ."

Another obsession perpetuated by teachers of English is "Thou Shalt Not End a Sentence with a Preposition." Fear of transgression constructs sentences that are incomprehensible on even the third and fourth reading. "People worth listening to" is better than "People to whom it is worthwhile to listen." E. B. White wrote a classic letter in which he said, "I want to tell how to end a sentence with five prepositions. A father of a little boy goes upstairs after supper to read to his son, but he brings the wrong book. The boy says, "What did you bring that book that I don't want to read *to out of up for?*"

PUNCTUATION

Policing all these words are punctuation marks which are the traffic signs and signals placed along the reader's road. They tell him when to slow down and when to stop; and sometimes they warn him of the nature of the road ahead. Traffic engineers do not always agree on what signs should be used or where they should be placed and neither do writers, editors or little old school teachers employed by journals. Much of punctuation is arbitrary[41].

THE GOOD PAPER

If you have enjoyed reading a scientific paper, go back and read it again. Ask yourself why it gave you pleasure. The chances are that you will find that correctly chosen words are arranged in each sentence so that their relationship to one another is clear and their message is readily understood[47]. Unfortunately, scientists, by and large, tend to be suspicious of lively attractive writing on their own subject[43]. However, those who publish do have the responsibility to present data in an interesting way[48].

Scientific writing is a craft, not an art. It does not take special inspiration, it takes practice, attention to detail, technique, and more practice. Sit down and do it regularly. It is a frustrating experience for the beginner[15]. W. Somerset Maugham wrote in his memoir *Summing Up* that in his writing he strove for simplicity and lucidity, and he was a physician; proof that we can write with distinction[20].

You cannot teach someone to write. You can teach them to write better. In fact, good teachers may guide and impel, but really educated people are frequently self-educated, having read, marked, learned and invariably digested many books. Many have actually become educated without filmstrips and thus learned to write well in English along the way[49]. Most people can learn to write well if they want to. Vigorous writing is concise. It uses as few words and as short words as possible. It uses short sentences, a help to quick understanding. Good writing is like a good watch, there should be no extra parts and every part should be doing some useful work[50].

Some papers have only a limited message—perhaps they make only one important point. If so, wrapping it like a present may obscure its recognition. A brief report makes much more impact. Winston Churchill said, "Pray let me have one sheet of paper . . . ."[22] He also wrote across a manuscript, "This paper, by its very length, defends itself from ever being read." A well-written paper is concise and direct. Limit sentences to single units of thought. It is the simple declarative sentence that communicates best[47].

When you have written a paper, do not regard it as your perfect newborn baby; many are born with congenital defects. Put it away in a drawer. Do not read it for two weeks and then reread it. It may appear wordy and windy and needing remedial surgery, often more ablative than reconstructive. Samuel Johnson warned that one should "Read over your composition and whenever you meet with a passage you think is particularly fine, strike it out." Quarrel with the need for every paragraph, every sentence, every word[17]. Physicians should write every word they intend for publications so that any third-year medical student can understand it[5]. The eighth grade teacher was right when she said, "It is not enough to write so you may be understood, you must write so you cannot be misunderstood." Remember Lord Chesterfield who wrote, "My dear son, pray excuse this long letter, but today I have no time to write a short one."

Practice the four Rs of revision, reduction, rearranging, and rewording. When you think your revision is crystal clear, put it away for another two weeks. The next time you read it you will find more areas to be pared and clarified. And so it goes, one should never be completely satisfied with a piece of writing. My first draft of this essay was written in 1987. This is the twelfth version. Such perseverance pales in comparison with that of an accomplished writer. Ernest Hemingway rewrote the end of *Farewell to Arms,* thirty times just to "get the words right."

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For those of you who are wordsmiths or delight in the nuances of the English language consider a subscription to *Verbatim,* a quarterly journal—an erudite but easy read. P.O. Box 78008, Indianapolis, IN 46278-008.

THE EFFECTS OF THE ILIZAROV DISTRACTION TECHNIQUE ON BONE AND MUSCLE IN A CANINE MODEL: A PRELIMINARY REPORT

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ABSTRACT

This study examined the functional and morphological changes experienced by bone and muscle during Ilizarov distraction osteogenesis. Although extensive research has been conducted in the area of regenerate bone formation, the effect of limb lengthening on the biomechanical properties of bone and muscle has not been thoroughly addressed. In this study, an Ilizarov external fixator was applied to one tibia of nine skeletally mature dogs, and distracted 3 cm over thirty days. The contralateral tibia served as a control. Histology and weekly radiographs assessed muscle morphology and bone growth. The contractile capabilities of the gastrocnemius muscles from the experimental and control limbs were measured prior to sacrifice, and the bending stiffness of the tibias of five dogs was determined. All dogs experienced loss of knee extension secondary to muscle contracture and/or stiffness about the joint. These dogs did not bear weight on the experimental limb. In one dog, spontaneous resolution of the muscle contracture allowed partial weight bearing during the last three weeks of consolidation. Despite 3 cm distraction, tibial lengthening ranged from 1.7 to 3 cm. Biomechanical testing revealed a significant reduction in the bending stiffness of the lengthened bones when compared with control values ($p<0.003$). The weight of the lengthened muscles was 35% less than control values, a finding consistent with the histology which showed mild muscle fiber degeneration in all dogs. The contractile capabilities of the lengthened muscles were reduced to 29 - 80% of control values ($p<0.005$). In contrast, the lengthened muscle from the weight bearing dog retained 85% of the weight and 104% of the maximum contractile force of the control muscle.

INTRODUCTION

Corrective limb lengthening dates to 1905 when Codivilla of Bologna, Italy, was credited with publishing the first study of limb lengthening in the English literature[12]. Methods initially employed included stretching under narcotics, constant traction, and surgical stretching followed by application of a plaster cast[6]. One of the more recent advances in limb lengthening techniques came from the observations of G.A. Ilizarov, who in 1951 stimulated bone to regenerate across a fracture site by applying a steady distraction force[8,9]. From this observation and further studies, Ilizarov formulated the "Law of Tension-Stress" regarding bone formation[13]. This principle, applied with the use of a fine wire external fixator, has been used in a variety of clinical settings to lengthen long bones or to replace segmental bone defects.

Since Ilizarov's formulation of the "Law of Tension-Stress," there has been extensive research in the area of regenerate bone formation. However, during distraction of bone, the associated soft tissue including skin, muscle, blood vessels and nerves, must lengthen accordingly. The physiologic effects of lengthening on these soft tissues are not well understood. Ilizarov postulated that distraction of soft tissue results in both a reorganization of collagen and other connective tissue elements, and neohistogenesis[13]. He believed that the involved tissue had the ability to regress into a fetal state, and subsequently undergo histologic and functional adaptive changes that recreate conditions of normal growth[9,13].

While distraction osteogenesis has been extremely useful in the correction of limb length discrepancies, angulation deformities, non-unions, fractures, etc., the procedure is not without complications. Paley, in his series of sixty limb lengthenings, has documented that complications are prevalent[13]. He found a 25% rate of minor complications including deformity and transient contractures, and a 15% rate of major complications including

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equinus contractures, transient reflex sympathetic dystrophy, and transient motor nerve palsies[13,14]. Complications develop when soft tissue does not respond by adjusting to the increase in bone length. In clinical practice, one of the most prevalent complications is muscle contracture; a painful condition that often necessitates a premature halt of distraction[14].

In this study, we have created a non-weight bearing animal model for limb lengthening that results in the development of muscle contracture about the distraction site. In recent years, investigators have postulated several possible causes of muscle contracture. Among these are ischemia, denervation/motor plate trauma, scar tissue formation, calcium influx secondary to rupture of the sarcoplasmic reticulum, and histochemical changes. It is hypothesized that soft tissue regeneration, notably capillary repair, occurs at a slower rate than the distraction osteogenesis. The capillary insufficiency that ensues induces an ischemic episode in the muscle, resulting in changes at the cellular level[5,11]. We hypothesized that this microvascular ischemia in the muscle produces pain which, in turn, is most likely responsible for the contractures observed during limb lengthening.

Although the biological properties of regenerate bone have been thoroughly investigated, the functional changes that accompany bone lengthening are less understood. Ilizarov and Aronson have independently described the morphological characteristics of regenerate bone by measuring bone density levels[2]. To complement, and perhaps support the aforementioned findings, we determined the functional strength of this new bone by measuring bending stiffness about the distraction site. Because the forces of distraction are also transmitted to the surrounding soft tissue, muscle may experience morphological changes that greatly affect its biomechanical properties. Investigators have reported that a variety of structural, morphological and functional changes occur when a muscle is immobilized under stretch[7,15]. These circumstances are potentially reproduced when an extremity undergoes lengthening by Ilizarov distraction. In this study, we analyzed both the mechanical properties of the lengthened bone and the structural and functional adaptability of muscle in response to limb lengthening by the Ilizarov distraction technique.

MATERIALS AND METHODS

Nine adult mongrel dogs were used in this study. Adult dogs were used in order to eliminate bone growth about the physeal growth plate as a variable. The dogs were housed and cared for at a university facility, with all surgery and recovery taking place at the vivarium. Additionally, two normal dogs underwent biomechanical testing of muscle to determine whether there exists any natural discrepancy between the right and left hind limbs.

Initially, each of the nine experimental dogs underwent surgical application of an external fixator modified to accommodate their prominent hamstrings. The proximal half ring was fixed to the tibia at the level of the fibular head using a 1.5 mm wire and a 4 mm half pin. The distal ring was fixed to the distal third of the tibia using two 1.5 mm wires. The threaded rods connecting the proximal and distal rings were removed and corticotomy was performed near the junction between the proximal and middle thirds of the tibia. Osteotomy was executed using drill holes and an osteotome with care being taken to maximize soft tissue preservation and vascular continuity within the medullary canal. Through a separate, lateral incision the fibula of the experimental limb was approached, and a partial fibulectomy was performed. The contralateral tibia served as a control.

The wounds were closed, the fixator was reassembled, and radiographs were taken to ensure proper reduction of the bone fragments (Figure 1). Next, a second half ring was connected to the distal ring using threaded rods. The calcaneus was fixed to this half ring with a 1.5 mm wire, immobilizing the ankle joint at 120 degrees of plantar

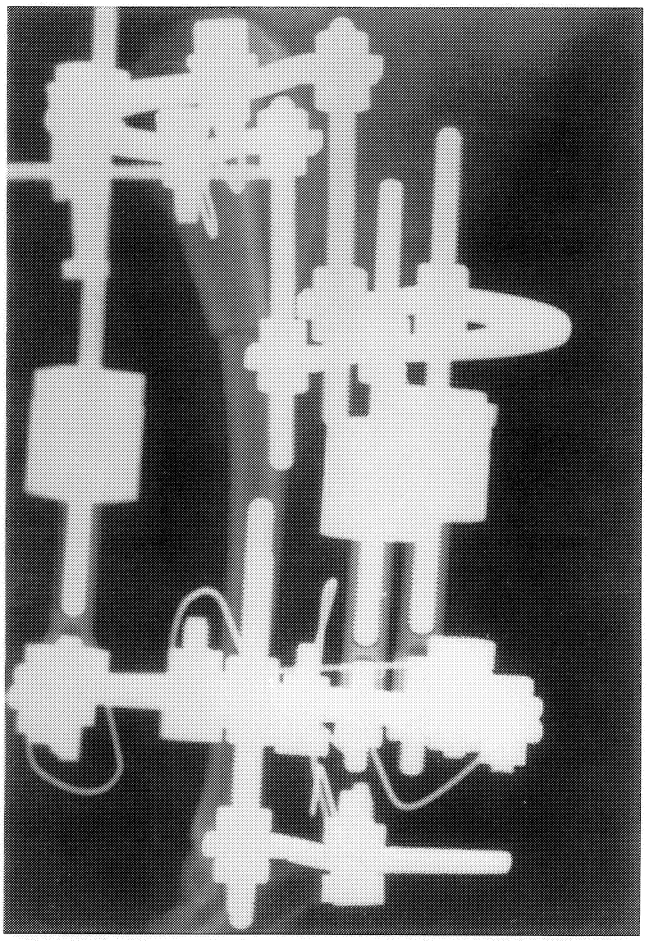

Figure 1. Post-operative lateral roentgenogram showing adequate reduction and good alignment of bone fragments.

flexion. Fixation of the ankle joint served to limit plantar flexion as a compensatory mechanism for bone and muscle lengthening. The dogs were returned to their runs and allowed one week of neutral fixation for soft tissue recovery.

On day 7, distraction was started at the rate of 1 mm/day in three increments (0.5, 0.25, and 0.25 mm) for 30 days.

Radiography was performed weekly on all dogs to assess the adequacy of bony alignment, and to document the progress of bone formation.

Blood levels of lactic acid, LDH, and CPK were measured daily from one dog during the distraction period, and randomly from a second dog. Also, lactic acid levels were measured from extracted muscle of five dogs at sacrifice.

Using a Stryker pressure monitor system, the compartmental pressure of both the experimental and control limbs was measured on a regular basis both during and after the distraction period.

Angiography was performed on six dogs postoperatively and prior to sacrifice. In the first two dogs a femoral approach was utilized, while in the last four dogs a carotid approach was selected. Abnormalities in vessel diameter or presence of vessel spasm were sought.

Following distraction, the dogs were sacrificed at various times: one dog immediately post-distraction, one dog at one week post-distraction, one dog at two weeks post-distraction, and six dogs at six weeks post-distraction. Prior to sacrifice, the Ilizarov external fixator was removed under general anesthesia. While anesthetized, the gastrocnemius muscle and associated neurovascular pedicle from the experimental and control limbs of seven dogs, and also from the right and left limbs of two normal dogs, were surgically exposed. The contractile force capabilities of these muscles were measured and compared using an Instron Model 1321 servohydraulic materials testing system. The femur and tibia were mounted to a frame via transfixion wires. The calcaneus was excised with the gastrocnemius tendon attached to it and clamped to the Instron actuator. In four dogs, the resting length of the control muscle was determined by loading the muscle with a 100 g tensile force; this same

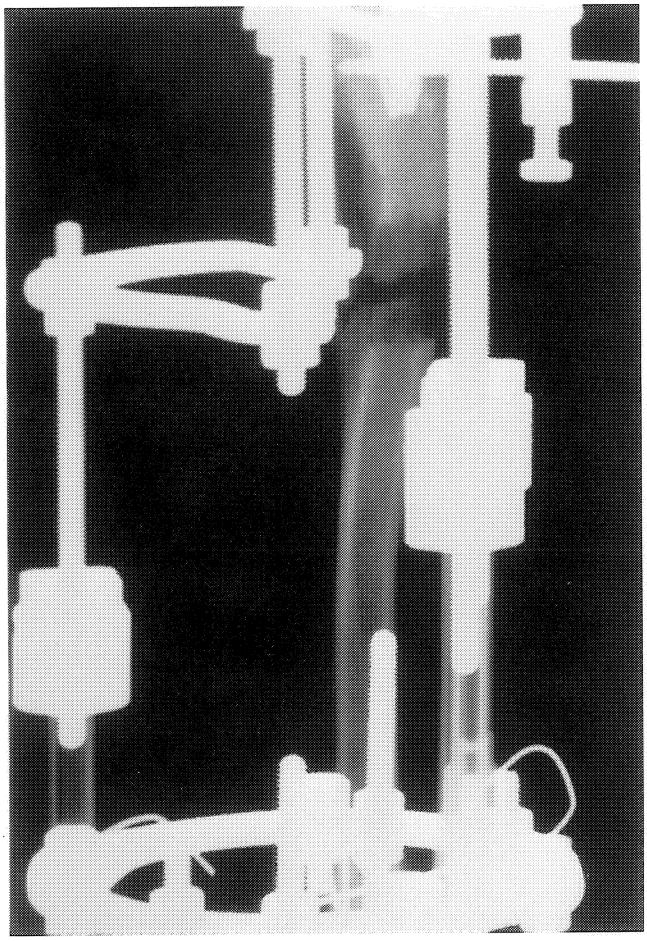

Figure 2. Lateral roentgenogram at one week post-distraction. Parallel columns of bone emanate from both corticotomy surfaces towards a central radiolucent plane.

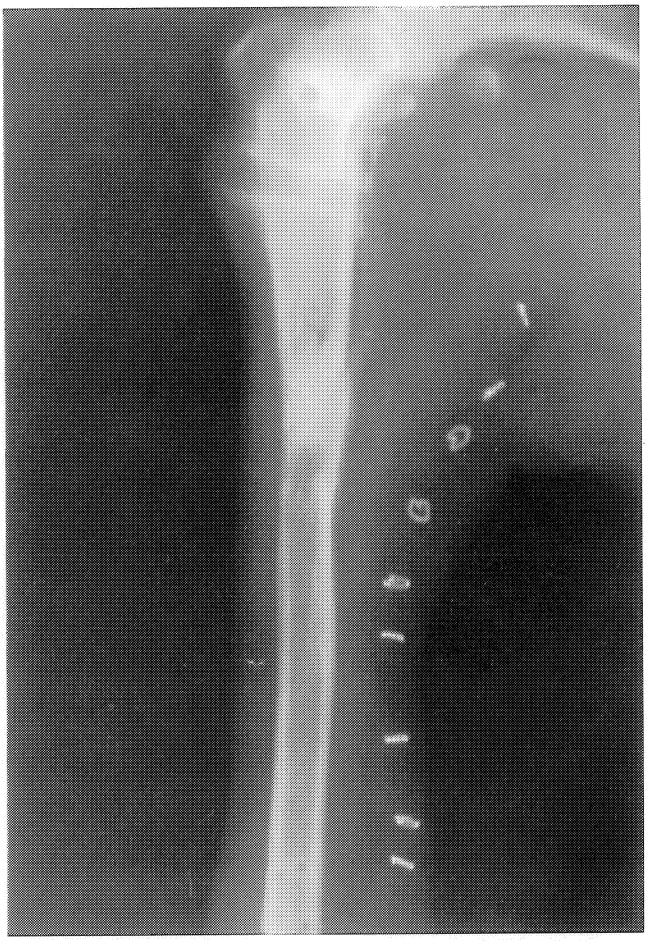

Figure 3. Lateral roentgenogram at six weeks post-distraction. The cortex is established and the distraction gap has completely solidified.

position was used as the resting length for the experimental muscle. In two dogs, the resting lengths were determined by loading each muscle with 100 g. In the remaining dog, the resting length of the control and experimental muscles was determined by fixing the ankle and knee joints at specific angles of plantar flexion and extension, respectively. In the normal dogs, the resting lengths of one dog were determined by loading each muscle with 100 g, while in the other dog, the resting lengths were determined by fixing the ankle and knee joints at specific angles as previously described. Starting 1 cm below the resting length, the muscle was stimulated at 2 mm increments via the sciatic nerve using a Grass S44 nerve stimulator (three times threshold voltage and 64 Hz).

After sacrifice, the gastrocnemius muscles from each animal were excised and weighed. Sections of tissue were harvested for histological analysis using a hematoxylin and eosin (H & E) stain. Samples from two dogs were analyzed with the von Kossa and Alizarin red stain to determine the presence of calcium deposition, and with a Sirrius red stain to determine the presence of increased fibrous tissue. Also, samples from the weight bearing dog were analyzed with a Masson trichrome stain to determine the presence of inflammatory cells.

The tibias of five dogs were harvested for biomechanical testing of the distraction site. Each tibia was tested by a cantilever bending test using an Instron materials testing system. The proximal end of each tibia was embedded to a point 1 cm from the osteotomy site or the equivalent point on the control tibia. A linear displacement of 2 mm at a rate of 1 mm/sec was sequentially applied to the anterior, posterior, medial, and lateral surfaces of the distal end of the tibia, approximately 6 cm distal to the potted area. The loading was repeated five times, and measurements were obtained during the fifth cycle. The stiffness of each tibia was calculated from the load and deflection recordings, and the stiffness of the experimental tibias was expressed as a percentage of the control tibias.

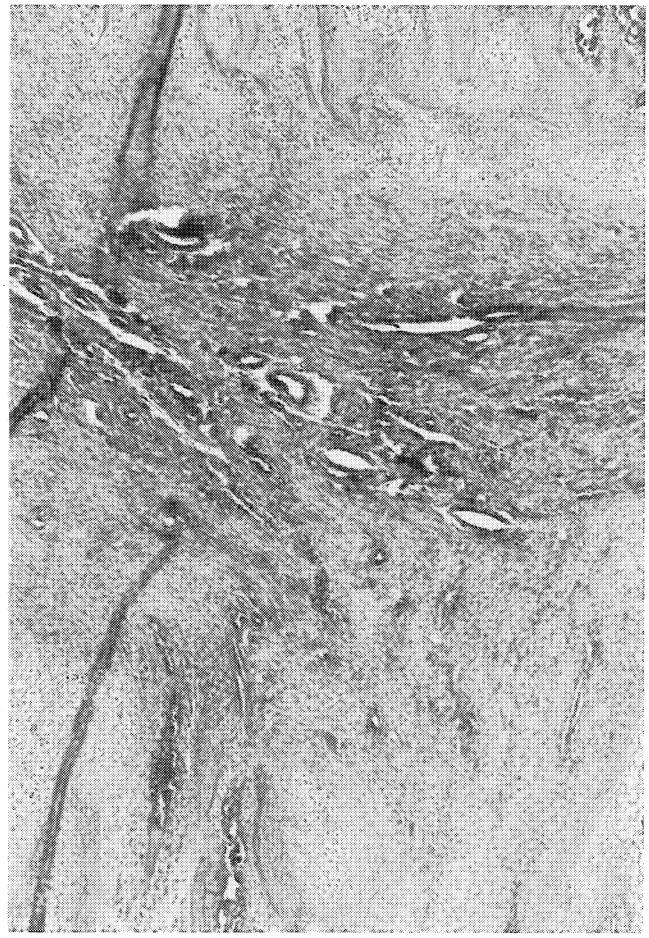

Figure 4. Distraction gap, day 44. A fibrous interzone consisting of collagen and small mesenchymal cells separates the parallel columns of bone. Note the islands of cartilage between the bony columns. (Hematoxylin and eosin; original magnification, x 40).

Figure 5. Distraction gap, day 79. Bone marrow from each end of the distraction gap is approaching each other in order to establish continuity. Note that the cortex is established. (Hematoxylin and eosin, original magnification, x 11.5).

After biomechanical testing, tibias from each dog were sectioned for analysis of the osteotomy site. Coronal sections of each bone were stained with H & E for histological examination.

Statistical analysis between the experimental and control limbs was performed using two-way Student's T tests. The confidence limit was predetermined at an α value of 0.05.

RESULTS

With fixation of the calcaneus, eight of the nine dogs did not bear weight on the experimental limb. One of the dogs was observed to bear weight on the experimental limb at three weeks post-distraction. All dogs experienced a loss of knee extension. Weekly radiographs revealed adequate bony alignment and complete bridging of the experimental gap by regenerate bone formation in all nine dogs. During the first week of distraction, the gap appeared entirely radiolucent. By day 14, columns of new bone began to emanate from both the proximal and distal surfaces of the osteotomy, parallel to the direction of distraction. During the remainder of the distraction and consolidation periods, the experimental gap continued to experience an increase in bone density and subsequent decrease in the size of the radiolucent growth zone such that by day 79 (six weeks post-distraction), the cortex was completely established and the distraction gap had solidified in all but one dog (Figures 2 and 3). In this one dog, a radiolucent area remained intact within the distraction gap.

Despite the fact that each fixator was distracted 3 cm over 30 days, the actual increase in tibial length ranged from 1.7 to 3 cm.

Histological examination of the bone revealed findings previously reported in the literature. By day 37 (immediately post-distraction), the experimental gap was filled with fibrous tissue, consisting of collagen and small mesenchymal cells, arranged parallel to the long axis of the tibia. Interspersed within this fibrous interzone were small islands of cartilage. At one week post-distraction, islands of cartilage were more prevalent, especially at the periphery of the fibrous interzone. Cones of bone were also present admixed with the cartilage (Figure 4). By day 51, the parallel columns of bone had increased in size. Between the bony columns, fibrovascular tissue remained.

Table 1. Biomechanical testing of tibia at sacrifice.

Dog	Stiffness of Experimental limb as a % of Control			
	Direction of Bending			
	AP	PA	ML	LM
1	22.9%	25.0%	34.0%	30.5%
2	38.0%	37.3%	49.5%	49.5%
3	39.8%	38.7%	60.0%	59.0%
4	46.9%	46.1%	39.8%	38.8%
5*	32.3%	30.0%	**69.7%**	**64.1%**
AVG (104)±SD	36.9±7.0%	36.8±5.9%	45.8±8.9%	44.5±9.8%
Significance	p<0.001	p<0.002	p<0.003	p<0.002

AP - Anteroposterior bending
PA - Posteroanterior bending
ML - Mediolateral bending
LM - Lateromedial bending
* dog #5 was weight bearing

The cantilever bending test measured the stiffness of the regenerate bone in four separate directions. There was a significant reduction in the bending stiffness of the experimental limb in all dogs.

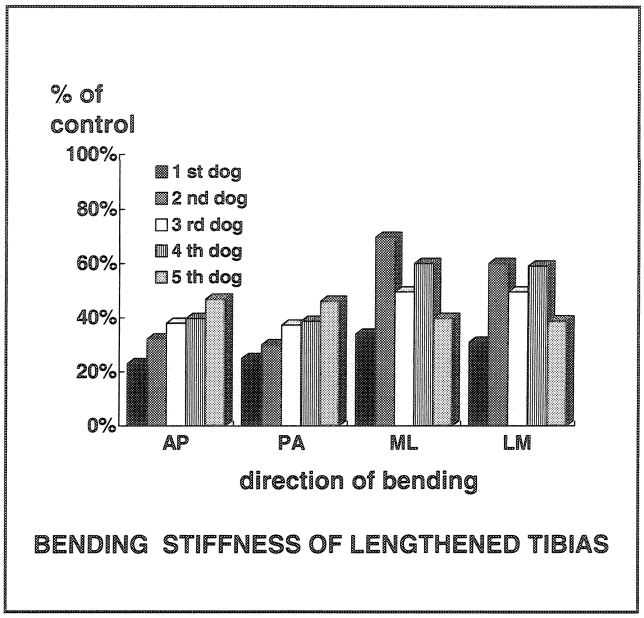

Figure 6. Bending stiffness of the lengthened tibia as a percentage of control, day 79. Note that there is no significant difference in bending stiffness between the non-weight bearing dogs and the dog which appeared to bear weight (Dog #5). AP - anteroposterior, PA - posteroanterior, ML - mediolateral, LM - lateromedial.

By six weeks post-distraction, the fibrous interzone was completely replaced by regenerate bone arranged parallel to the direction of elongation; no angulation was detected (Figure 5).

Using a cantilever bending test, the bending stiffness of the regenerate bone was measured in each of four perpendicular planes. In all dogs, the bending stiffness of the new bone was significantly less than the control. Values ranged from 23-69% of control with an average of 46.6 ± 10.4% (mean ± standard deviation) ($p<0.003$ in all planes) (Figure 6 and Table 1).

In the five non-weight bearing dogs sacrificed at six weeks post-distraction, the weight of the lengthened muscle ranged from 55-70% of control values, with a mean of 65 ± 5%. The cross-sectional area ranged from 57-69% of control values, with a mean of 65 ± 4%. In the weight bearing dog, the weight of the experimental muscle was 85% of the control, and the fiber cross-sectional area was 77% of the control.

The increase in muscle resting length in the non-weight bearing six week group ranged from 0.25 - 1.2 cm, while the increase in muscle length in the weight bearing dog was 1.2 cm. Greater than 95% of this increase in muscle length was seen in the belly of the muscle. Tendon length remained relatively constant. Values for the increase in muscle length as a percentage of the corresponding increase in bone length ranged from 12-48%, with a mean value of 25 ± 12% in the non-weight bearing dogs versus 57% in the weight bearing dog.

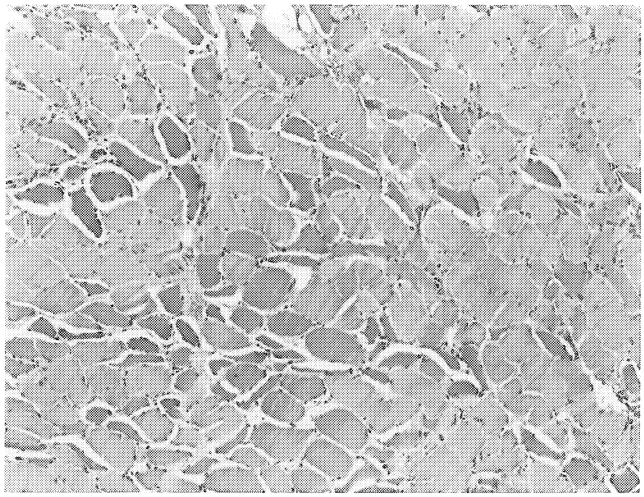

Figure 7. Cross-section of the muscle belly, day 37. Atrophic muscle fibers are present throughout the lengthened muscle. (Hematoxylin and eosin; original magnification, x 100).

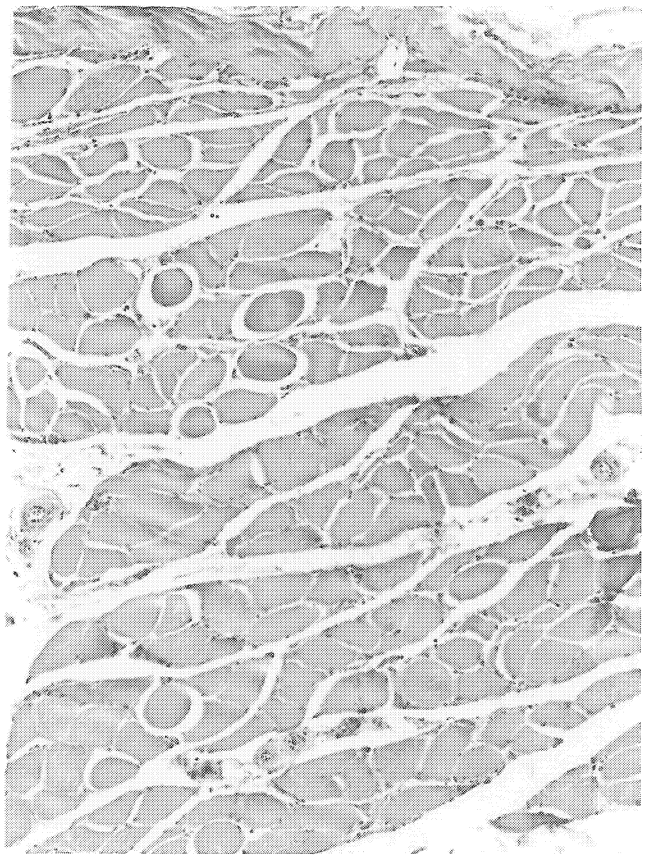

Figure 8. Cross-section of a muscle belly, day 79. Atrophic muscle fibers persist at six weeks post-distraction. (Hematoxylin and eosin; original magnification, x 100).

Although all dogs preferred flexion of the knee joint both during and after distraction, passive range of motion was abnormal in only five of the nine dogs. In these five dogs,

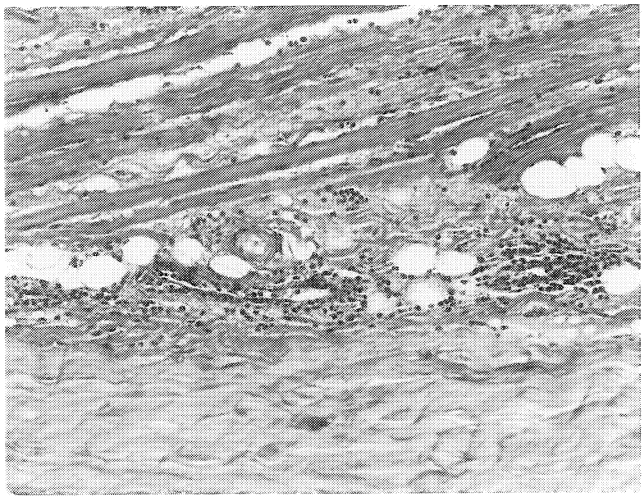

Figure 9. Muscle tendon junction, day 79. Note the extensive infiltration of inflammatory cells throughout the muscle tendon junction. (Masson-trichrome; original magnification, x 170).

knee extension was limited 15 - 25°. The weight bearing dog did not experience any loss of knee extension.

On histological examination, H & E stains of lengthened muscle showed mild muscle fiber degeneration including the presence of hypercontracted muscle fibers, nuclear clumping and localized atrophy (Figures 7 and 8). Von Kossa and Alizarin red stains did not reveal calcium deposition, and examination with the Sirrius red stain failed to show any evidence of increased fibrous tissue formation. In addition to the aforementioned findings, a Masson trichrome stain of the lengthened muscle in the weight bearing dog revealed inflammatory cells at the muscle tendon junction (Figure 9).

In the non-weight bearing group, the maximum contractile force produced by the gastrocnemius muscle of the experimental limb was consistently less than that produced by the control limb. Force values of the experimentally lengthened muscle as a percentage of the control ranged from 29 - 80%. Two dogs were not tested. There was a 35% reduction in the contractile force capabilities of the dog sacrificed immediately post-distraction, and a 28% reduction in the dog sacrificed two weeks post-distraction. Of the four dogs tested in the six week group, the average decrease in force capabilities was 51 ± 14% ($p<0.005$) (Figure 10). The length of the muscle at which maximum contractile force was produced was recorded in six of the eight dogs. The increase in length from L_o (resting length) was calculated as a percentage of the L_o, with the resultant value directly related to the distance the muscle has to stretch to reach its maximal force capabilities. In three of the six dogs (immediate, two week, and one six week post-distraction dog), the experimental muscle reached its point of maximum contractile force sooner than did the control muscle. Conversely, the experimental

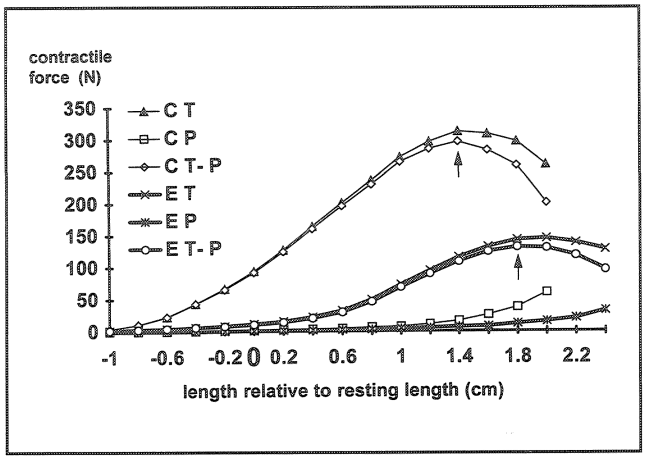

Figure 10. Sample Force vs. Length curve of a non-weight bearing dog, day 79. The x-ordinate represents the change from resting length at which passive and total tension were measured. The arrows indicate the point of maximum contractile force.
CT = total force of control muscle
CP = passive force of control muscle
CT-P = total - passive force of control muscle
ET = total force of experimental muscle
EP = passive force of experimental muscle
ET-P = total- passive force of control muscle

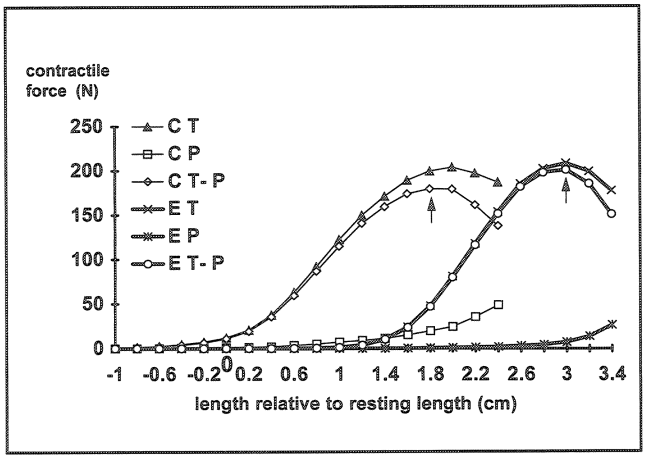

Figure 11. Force vs. Length curve of the weight bearing dog, day 79. Note that the maximum contractile force of the lengthened muscle exceeds that of the control muscle. Also, the point of maximum contractile force is reached at a distance further from the muscle's L_o than the control.

muscle from the remaining three dogs reached its point of maximum contractile force later than did the control muscle (range of 0.4 - 1.4 cm longer). For the control muscles, the average increase beyond the muscle's L_o at which maximum force was produced was 13.6 ± 0.8% with a range of 12.7 - 15%. For the lengthened muscles, the average was 9.6 ± 2.3% with a range of 6.6-13.6%.

In the dog which was observed to bear weight, the maximum contractile force of the lengthened muscle was 104% of the control side (Figure 11). The control muscle reached its point of maximum force 1.8 cm above L_o, a difference corresponding to 18.9% of L_o. The lengthened

muscle reached its point of maximum force 3.0 cm above L_o, a difference corresponding to 16.8% of L_o.

In the normal dogs, there was an 8 - 10% difference in the active force capabilities between the right and left hind limbs. There was also a 0.4 cm difference in the increase from L_o at which maximum force was produced between the right and left hind limbs. The normal muscle reached its point of maximum force at an average distance of 1.9 ± 0.3 cm above L_o, a distance corresponding to 17.9 ± 2.8%, of L_o.

Angiography was performed on six dogs. Analysis of the popliteal and distal femoral arteries revealed no significant morphological difference between the control and experimental limbs. Spasm of the distal femoral and popliteal arteries was apparent in the experimental limbs of the two dogs in which a femoral approach was utilized. Histological examination of these particular vessels was normal.

Both intramuscular and compartmental pressure studies and histochemical analysis failed to show any significant difference between experimental and control limbs.

DISCUSSION

In our study, eight of the nine dogs did not bear weight on the experimental limb, presumably due to fixation of the ankle joint and preferred flexion of the knee joint. However, despite this partial immobilization, weekly radiographs and post-mortem histological analysis revealed regenerate bone formation similar to that described in the previous literature[1,8,9,13]. The observation that osteogenesis occurs in a non-weight bearing model lends credence to Ilizarov's original observation that, under the law of tension-stress, tissue is stimulated to regress into a fetal state with cells in the distraction zone sharing features of embryonic, fetal, and neonatal tissue[8,13]. Paley echoes Ilizarov's observations, stating that the fibrous interzone, which consists of undifferentiated mesenchymal cells, acts as a pseudo-growth plate during limb lengthening[13].

Although the fixator was distracted 3.0 cm in all dogs, the actual increase in tibial length ranged from 1.7 - 3.0 cm. This finding, which has been observed clinically in some of our patients, may suggest premature bone consolidation and/or angulation of the lengthened limb. Causes of premature bone consolidation and angulation include inadequate rigidity of the external fixator and/or associated Kirschner-wires. Aronson has described the importance of pin rigidity in eliminating premature bridge formation within the distraction zone,[3] and Ilizarov has reported that fixator stability enhances the quality of regenerate bone formation[8]. In our study, radiographs confirmed that premature bone consolidation did not occur, as evidenced by the presence of a radiolucent plane between the columns of new bone that persisted well into the consolidation period (Figure 2). Also, angulation of bone or associated Kirschner-wires was negligible except in one dog which showed moderate to severe angulation. Thus, it is difficult to attribute this inadequacy of tibial lengthening to premature bone consolidation and/or angulation.

Previous studies have used bone density levels as a measure of the adequacy of regenerate bone formation by distraction osteogenesis[2,8]. In Ilizarov's original study, he reported that the osteogenic area attained 80% of cortical bone density by day 119[2]. In contrast, Aronson found that the regenerate bone reached a peak of 70% of cortical bone density by day 35, and then normalized to 40% (normal metaphyseal levels) by day 77[2]. While such measures aid in analyzing the quality of regenerate bone, functional studies, such as the cantilever bending test, are a direct measure of the strength of this new bone. In our study, the average strength of the regenerate bone in the non-weight bearing dogs was only 37% of control values in the AP and PA directions, and 45% of cortical values in the ML and LM directions. These findings suggest that, although radiological and histological appearances of regenerate bone resemble that of normal bone, the functional strength inherent to mature bone has not completely developed by day 79 in this model. In the weight bearing dog, the strength of the regenerate bone was not significantly different in the AP and PA directions, but was significantly stiffer in the ML and LM directions (Table 1). Although Ilizarov reports that bearing weight is a major stimulus for regenerate bone formation,[8] we are unable to confirm that it had such an effect in the weight bearing dog. However, we do propose that bearing weight earlier in the distraction and consolidation periods would have a positive effect on bone maturation. Consequently, it is difficult to assess whether the decrease in bending stiffness observed in regenerate bone is due to a lack of weight bearing, to the immature status of the bone, or to a combination of these factors.

One of the purposes of this study was to determine the etiology of muscle contracture observed during limb lengthening by the Ilizarov distraction technique. Initially, ischemia was considered both a likely and testable hypothesis for etiology of muscle contracture. However, our results do not indicate ischemia as a likely cause of muscle contracture in this model. Histological examination of the lengthened muscle failed to reveal calcium deposition or any abnormalities in local capillary microstructure. Angiography failed to show any evidence of vascular insufficiency such as narrowing or spasm of the blood vessels in the experimental limbs. Compartmental pressure studies did not reveal any significant difference between experimental and control limbs, thus eliminating compartment syndrome as an etiologic agent in ischemia-induced contracture. Also, Sirrius red stain failed to show evidence of increased fibrous tissue formation, and histochemical analysis was entirely normal. These results help rule out either scar

tissue formation or histochemical changes as other possible causes of muscle contracture.

At six weeks post-distraction, the increase in muscle length in the non-weight bearing dogs was only 25 ± 12% of the corresponding increase in bone length. This significant discrepancy is likely due to muscle contractures that occurred secondary to distraction and immobilization. These muscle contractures and resultant knee flexion deformity may act to inhibit bone lengthening during the distraction period and may, indirectly, initiate or potentiate premature bone consolidation and/or angulation of the lengthened limb. In the weight bearing dog, the muscle contractures resolved spontaneously, allowing some use of the experimental limb during the last three weeks of consolidation. In this dog, muscle elongation more closely parallels bone elongation (57% vs. 25%).

To date, the structural and functional response of muscle to limb lengthening is not completely understood. Adaptation of a muscle's length, weight, and fiber cross-sectional area to limb lengthening is important because it is these physical characteristics that determine a muscle's mechanical properties in vivo. In our model, there exists two opposing factors that together determine a muscle's biomechanical properties. First of all, immobilization inhibits protein synthesis and limits a muscle's ability to maintain the appropriate sarcomere number, thus limiting normal growth and decreasing a muscle's functional capabilities[10,17]. Baker and Matsumoto have reported that, regardless of muscle position, immobilization causes degenerative changes including myofibrillar disruption, swollen mitochondria, and focal necrosis[4]. In our study, histological analysis of the lengthened muscle revealed mild degenerative changes in all dogs (Figures 7 and 8). Additionally, the weight and cross-sectional area of the lengthened muscle in the non-weight bearing dogs was 35% less than control values. These histological changes and reduction in size are attributed to degeneration of muscle fibers secondary to immobilization—a phenomenon termed disuse atrophy. In opposition to the inhibitory effect of immobilization, muscle immobilization under stretch experiences the stimulatory effect of stretch-induced tension. Previous literature has reported that muscle first responds to elongation by stretching pre-existing sarcomeres[16]. Shortly thereafter, the muscle experiences neomyogenesis by both sarcomere addition and recruitment of satellite cells[8,15,16]. Goldspink has reported that muscle adapts to elongation by adding sarcomeres onto the ends of pre-existing myofibers[16]. This increase in sarcomere number causes a decrease in the length of individual sarcomeres such that filament overlap eventually returns to control values[16]. It is near these control values that filament overlap is considered optimal, thus maximizing the force capabilities of the lengthened muscle[16]. Despite the stimulatory effect that stretch-induced tension has on muscle, the atrophic changes secondary to immobilization produced a net loss in muscle weight and fiber cross-sectional area in our non-weight bearing model.

In the weight bearing dog, the experimental muscle retained 85% of the weight and 77% of the cross-sectional area of the control muscle. These values are significantly greater than those of the non-weight bearing dogs. This ability to better maintain muscle weight may be attributed to early mobilization and weight bearing of the experimental limb.

In the non-weight bearing dogs, there was a significant reduction in the active force capabilities of the lengthened muscle ($p<0.005$) (Figure 10). This observation indicates that, at least functionally, the contractile component of the lengthened muscle was not adapting adequately to elongation. The most plausible cause for this decrease in active force capabilities is atrophy of the muscle secondary to disuse. One particular finding that supports this observation was the fact that the dogs in the six week group experienced a 20% greater reduction in force capabilities than the immediate and two week post-distraction dogs. This is reflective of the increased period of immobilization experienced by the six week group. Furthermore, the dog which appeared to bear weight produced active force values that were approximately equal to control values, further emphasizing the importance of mobilization in maintaining the functional characteristics of muscle.

While atrophy secondary to disuse can be implicated in a muscle's inability to adapt functionally to elongation, the role that structural adaptability plays must also be assessed. If muscle does not adapt structurally to elongation, its mechanical properties will be altered[15]. The increase in length from L_o (resting length) at which maximum contractile force is produced was calculated as a percentage of the muscle's original resting length. The authors propose that this value represents the distance muscle fibers have to stretch in order to reach their maximal contractile capabilities. In four of the five dogs tested (including the weight bearing dog), the aforementioned value in the lengthened muscle closely resembled that of the control. While the exact values were not identical, they were similar to the discrepancy that existed between the right and left limbs of the two normal dogs tested. The observation indicates that the lengthened muscle in the experimental dogs is adapting structurally to elongation.

In summary, we found that despite a distraction of 3.0 cm, the actual increase in tibial length was less than 3.0 cm in all but one dog. Functional testing revealed that the regenerate bone was 42.6% as stiff as control bone. Thus, although radiological and histological appearance of the

regenerate bone approached normalcy, the biomechanical properties were not equivalent to those of mature bone. In the soft tissue, the lengthened muscle experiences a significantly smaller increase in length than does the bone. This discrepancy is thought to be secondary to muscle contracture and prolonged loss of extension at the knee. We found that muscle lengthened during the Ilizarov distraction technique in a non-weight bearing model retained its structural adaptability. However, despite adequate structural adaptability, the experimentally lengthened muscle was not able to adapt functionally, as evidenced by a significant decrease in the force capabilities of the muscle when compared to the control side. This observation was attributed to disuse atrophy secondary to immobilization. Consequently, the rate and frequency of distraction required for adequate regenerate bone formation, coupled with immobilization, was not optimal for the associated soft tissue, particularly skeletal muscle. In contrast, the lengthened muscle from the single weight bearing dog displayed adequate structural and functional adaptability to elongation. The muscle retained 85% of the weight and 104% of the maximum contractile force of the control muscle. Thus, the authors suggest that early mobilization and weight bearing may have a stimulatory effect on both the structural and functional adaptability of muscle to elongation during the Ilizarov distraction technique.

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RESULTS OF THE CEMENTLESS MALLORY-HEAD PRIMARY TOTAL HIP ARTHROPLASTY: A 5 TO 7 YEAR REVIEW

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ABSTRACT

We reviewed the results of 172 plasma-sprayed, titanium primary total hip arthroplasties inserted without cement and followed 5 to 7 years. Hips were replaced for a wide range of diagnoses and patient ages. No femoral components had been revised nor were any considered unstable. Clinical results have been excellent; 88% of hips had either no or a slight amount of pain and only 5% of patients had thigh pain when specifically asked. Radiographically, this femoral component achieved stability after an initial subsidence in 9% of cases. Extensive femoral bone resorption was rare, and distal cortical hypertrophy was commonly seen. Pelvic osteolysis occurred in 16 (9%) cases and was considered major in 10 of these. It was the cause of failure of 3 acetabular components. Femoral endosteal lysis was not observed.

We concluded that mechanical stability of the Mallory-Head titanium total hip prostheses is excellent. However, significant pelvic osteolysis had occurred commonly with this implant design, and will continue to pose major reconstructive problems in the future.

INTRODUCTION

Cementless fixation continues to evolve as a potential and superior option to cement fixation in total hip arthroplasty. Early cementless stems were troubled by component instability[21,22,26], thigh pain[2,19,26,30] and worrisome femoral bone resorption[14]. Osteolysis, once thought due solely to acrylic cement, seems to occur more frequently and have a more progressive course after cementless stem insertion[15,36]. However, the analysis of femoral lysis has often been with non-circumferential porous coated stems, known to predispose to lysis, and by authors who have a bias favoring cement fixation[1,15]. Cementless socket fixation has become generally well accepted and proven in the short-term, but the best mode of fixation is unknown as are the long-term results of these implants.

Currently, many cementless total hip arthroplasties are being implanted as potential improvements over earlier designs and cemented fixation, with little knowledge of their clinical efficacy. We present the results of a consecutive series of plasma sprayed, titanium Mallory-Head primary total hip arthroplasties inserted without cement and followed 5 to 7 years. These results should serve as a comparison to other implants and as a basis for improvement in future design.

MATERIALS AND METHODS

A total of 193 primary total hip arthroplasties, using the cementless Mallory-Head components, were performed at the University of Western Ontario Hospital between October 1987 and September 1989. Fifteen patients with 16 hips died prior to a 5 year review, and another 4 patients with 4 hips were lost to follow-up and could not be contacted. One total hip arthroplasty was resected due to deep sepsis. The remaining 172 total hip arthroplasties in 169 patients have had a 5 to 7 year clinical and radiographic review, with a mean follow-up of 5.8 years. Hips were replaced on the right side in 87 cases and the left in 85. There were 93 females and 79 males. The diagnosis leading to total hip arthroplasty was osteoarthritis in 140 (81%), rheumatoid arthritis in 13 (7%). The mean age at last follow-up was 63 years old (range 38 to 85 years).

All surgeries were performed under the supervision of two senior surgeons (RBB, CHR) using a modified direct lateral approach. Laminar airflow, body exhaust suits and peri-operative antibiotics were routinely used.

The acetabular component has a circumferential plasma-spray coating, 4 peripheral fins for fixation and multiple screw holes for supplemental fixation if necessary. All sockets were inserted line-to-line. The femoral component has a small nonfunctional collar, a proximal third plasma spray coating and a textured middle third. The stem is tapered or wedged 3° in both the sagittal and coronal planes. All femoral components were inserted

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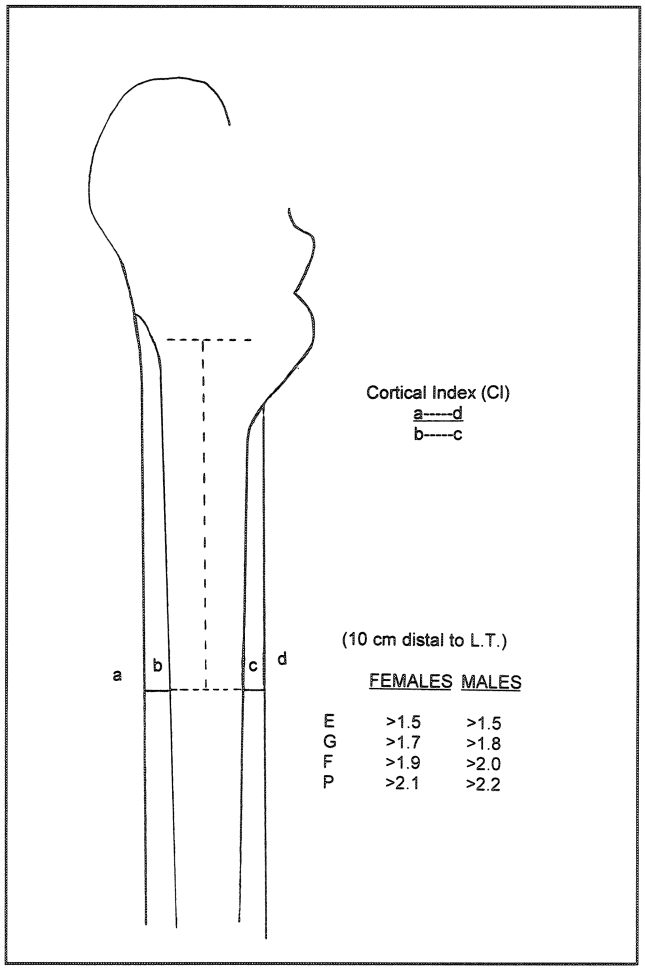


Figure 1. The cortical index was used as a measure of cortical bone quality, and arbitrarily divided into poor through excellent grades as shown. E = Excellent, G = Good, F = Fair, P = Poor

line-to-line after hand reaming and broaching, without milling or machining the femur. Ultra high molecular weight polyethylene, secured to the shell by a hexagonal locking mechanism, and 28 mm titanium femoral heads were used in all cases. The first 60 cases employed untreated titanium heads, and the remainder of the hips employed ion implanted titanium heads[23].

Patients were encouraged to walk partial weight bearing by the second postoperative day. Full weight bearing was instituted by 6 weeks, and patients were generally released to fully activity by 3 months after surgery. Patients were followed at 3, 6, and 12 months postoperatively and yearly thereafter. Clinical information was recorded prospectively in a computer data bank using the Harris hip rating[17] and the American Academy of Orthopaedic Surgeons Hip Society rating forms[20] at different times during the period of study. Only individual parameters (pain, limp, etc.) will be reported here[6]. Thigh pain was asked specifically at each visit, and graded 1 to 10 using a visual analogue scale.

Anterior posterior pelvis and cross-table lateral radiographs were taken pre and postoperatively, and at each follow-up visit. Radiographic analysis was carried out by two of us (BDM, NN) without knowledge of the clinical results. The preoperative Cortical Index, a measure of cortical bone quality, was taken as the ratio of cortical to medullary diameters 10 cm distal to the lesser trochanter (Figure 1)[11]. A modified Singh Trabecular Index was also used (Figure 2)[35]. Both indices were arbitrarily graded as shown in the figures, with the following distribution (Table 1).

Postoperative femoral stem alignment was referenced from the proximal medullary canal on the anteroposterior film, and considered neutral if it was 3° or less from being colinear (Figure 3). Collar-calcar contact was recorded as present or absent. Metaphyseal fit was considered good if the stem occupied 80% of the canal on the anteroposterior film and 70% on the lateral film at the level of the lesser trochanter. Isthmal fill was measured 3 cc proximal to the tip of the implant with similar criteria for good and poor[22] (Figures 4 A and B). Three-point cortical contact seen on

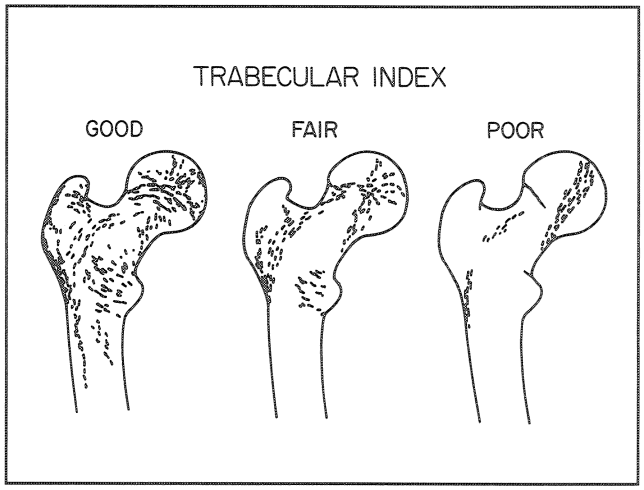


Figure 2. The trabecular index was arbitrarily divided into poor, fair, and good grades as shown.

Table 1. Distribution of cortical and trabecular indices, based on the grading systems shown in Fig 1 and 2.

	Cortical Index	Trabecular Index
Excellent	50%	
Good	27%	52%
Fair	15%	43%
Poor	7%	6%

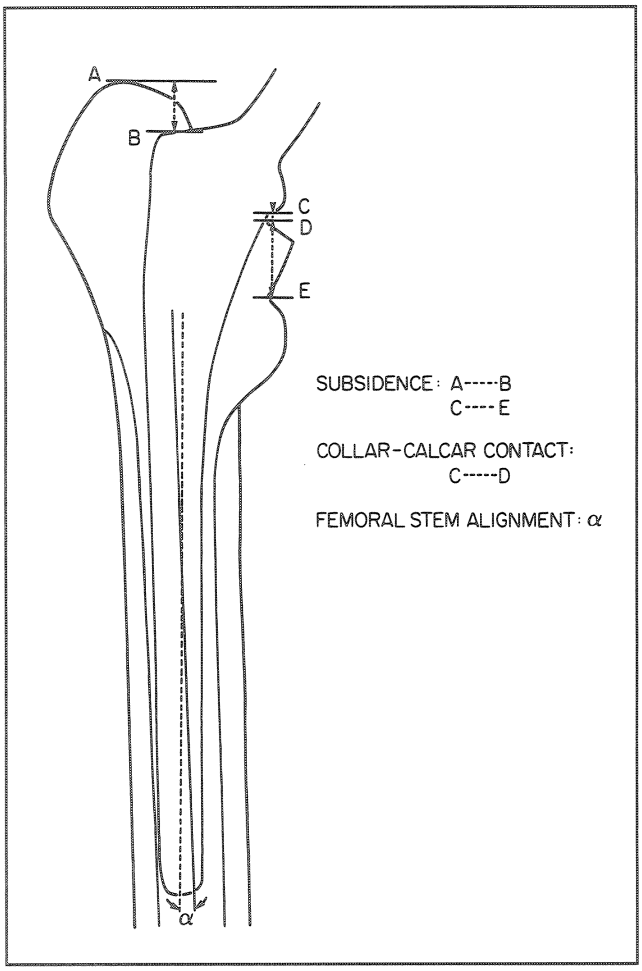


Figure 3. The method used to determine femoral stem alignment, the presence of collar-calcar contact and femoral component subsidence.

either the anteroposterior or lateral film, was also considered evidence of a good isthmal fill (Figure 5).

Acetabular inclination was referenced from the intertear drop line. Any initial gaps or lines behind the socket were recorded. No attempt was made to estimate either acetabular or femoral component version.

Serial radiographs were available for each of the 172 total hip arthroplasties. Radiolucent and radiodense lines, and bone remodeling changes were analyzed in 3 acetabular and 7 femoral zones on both the anteroposterior and lateral radiographs[9,16,20]. For simplicity in reporting, the findings were combined into just 3 acetabular and 7 femoral zones.

Femoral bone resorption was recorded as mottling if there was loss of trabecular density or corticocancelization, or as cortical thinning, if present. Cortical hypertrophy was defined as new bone of cortical density and was classified as either periosteal, endosteal, or both. Endosteal "spot welds" were defined as new bone bridging the endosteum and porous coating of the implant. A halo pedestal was defined as a thin sclerotic line extending around the tip of the smooth portion of the stem; and shelf pedestal as partial filling of the canal with bone under the tip of the stem.

Femoral component subsidence was measured using the greater trochanter and lateral border of the implant, and the femoral collar and lesser trochanter as references (Figure 3). A difference of 3 mm in both measures on serial films were considered significant and measurable.

We defined an unstable femoral stem by progressive subsidence and/or a circumferential radiolucent/radiodense line. We inspected for femoral endosteal lysis as defined by scalloping or a cystic lucency adjacent to the stem. We observed for any evidence of plasma-spray shedding or stem breakage.

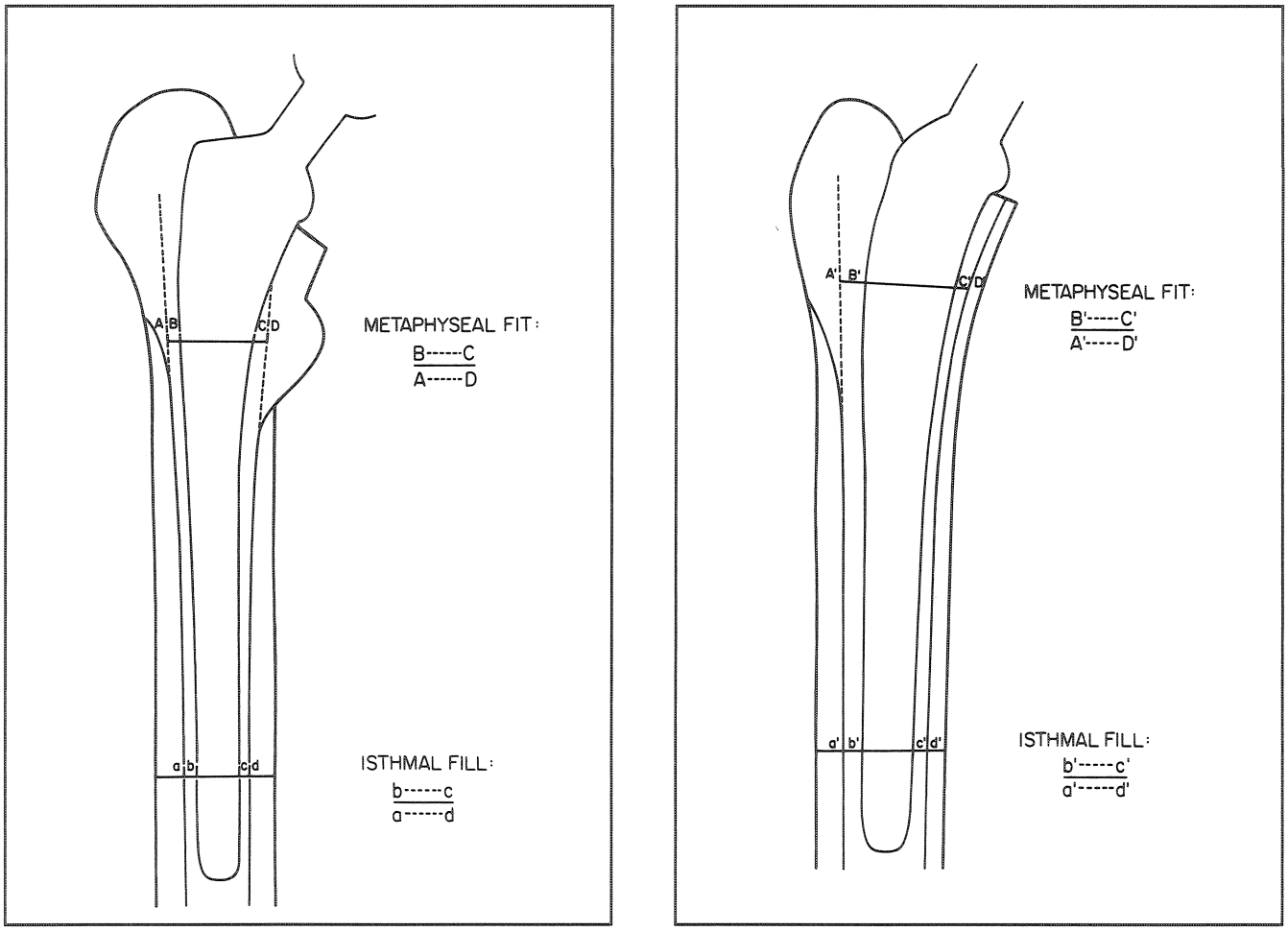
The adequacy of the anteroposterior pelvic radiograph to measure acetabular migration, and migration itself were measured using the technique of Massin et al.[27] (Figure 6). A difference of 3 mm or greater was considered significant and measurable. Acetabular component instability was defined by migration and/or a circumferential radiolucent/radiodense line. Pelvic osteolysis was defined as a new or expanding cystic lucency adjacent to the socket as seen on either the anteroposterior or lateral radiographs. Radiolucent lines, pre-existing cysts that did not enlarge and osteopenia were not considered osteolysis. We arbitrarily called osteolysis major if it compromised greater than one-third the bony support for the socket, a criteria set forth by Maloney et al. for considering surgical intervention[24]. Progression of the osteolysis on serial films were also recorded. Heterotopic ossification was graded according to the classification of Brooker et al.[4].

RESULTS

Postoperative femoral stem alignment was neutral in 94%, varus in 2% and valgus in 4%. Collar-calcar contact was present in 83% and absent in 17%. Metaphyseal fit was considered good in 91% and poor in 9%. Isthmal fill was considered good in 76% and poor in 24%. The mean acetabular inclination was 41°, with a range of 29° to 58°. A small gap or radiolucent line, thought to represent incomplete contact of the socket with bone, was seen behind the acetabular shell in 22 (13%) cases; always in Zone 1 and/or 2. In 20% of cases, the surgeon deemed it necessary to use supplemental acetabular screw fixation.

Instability/Revision

The clinical records and serial radiographs were reviewed for each of the 172 total hip arthroplasties presented here. At last follow-up, no femoral components had been revised, and none were considered unstable. Three acetabular components (1.7%) had been revised for com



Figures 4 a-b. The method used to judge metaphyseal fit and isthmal fill on the a) AP radiograph and b) lateral radiograph.

ponent instability. Three acetabular components (1.7%) had been revised for component instability. In two cases, there was initial mechanical stability, but osteolysis developed and the socket subsequently migrated into osteolytic cysts (Figure 7). At the time of revision, large osteolysis defects were discovered and bone grafted in both cases. There was severe polyethylene wear into the metal shell, and histologic examination revealed the typical macrophagic response to polyethylene and metal particulate debris. The third acetabular revision was for traumatic socket instability in a patient who did well initially, but developed pain after a severe water waverunner accident. Radiographs revealed initial component stability followed by acetabular migration and a femoral calcar fracture after his injury. In this case, there was no significant bone loss and no bone grafting was necessary. Another 3 sockets had migrated and were considered unstable, but have not been revised. In one case, the socket migrated into late osteolysis and the patient is symptomatic and considering revision. In another, vertical tilting occurred early, but the patient has remained asymptomatic 6 years since surgery. In the last case, the acetabular shell protruded medially into poor quality rheumatoid bone, but again the patient has remained asymptomatic to last follow-up (Figure 8). Thus, component instability has occurred in 6 (3.5%) of 172 acetabular reconstructions. In 4 of the 6 cases, initial mechanical stability was present, but late loosening developed because of osteolysis (3) and trauma (1). The demographic data for each of these six cases is shown in Table 2.

CLINICAL RESULTS

The clinical data recorded via the American Academy of Orthopaedic Surgeons Hip Society rating form was converted to the point scale of the Harris Hip rating for reporting purposes. The clinical scores included the last available score prior to revision for those patients who eventually came to acetabular revision. At last follow-up, the mean score for pain was 41 out of 44 points. Sixty percent of hips were pain-free, 28% had slight pain, 9% mild and 4% moderate pain. No patient had severe hip pain. The mean score for limp was 9 out of 11 points.

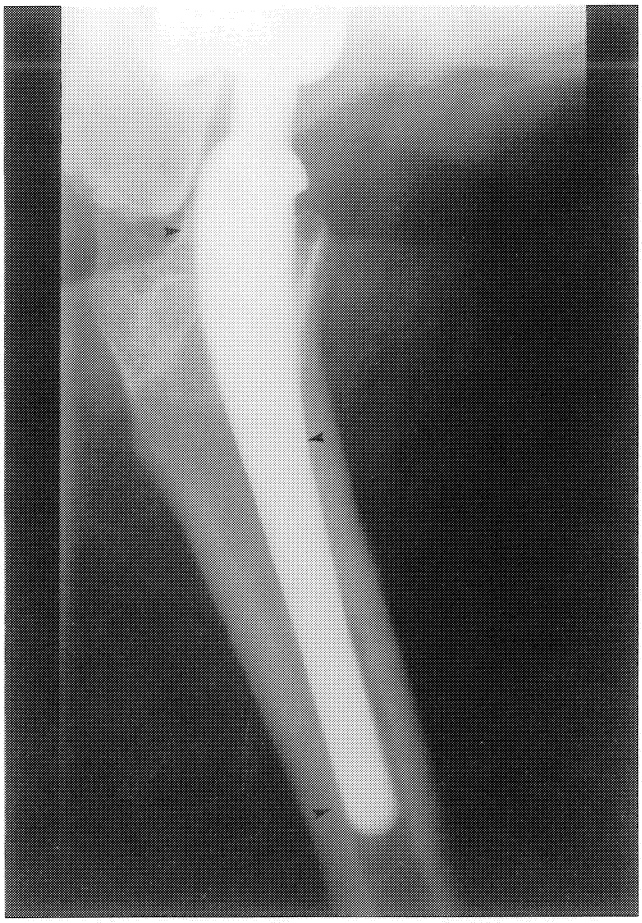
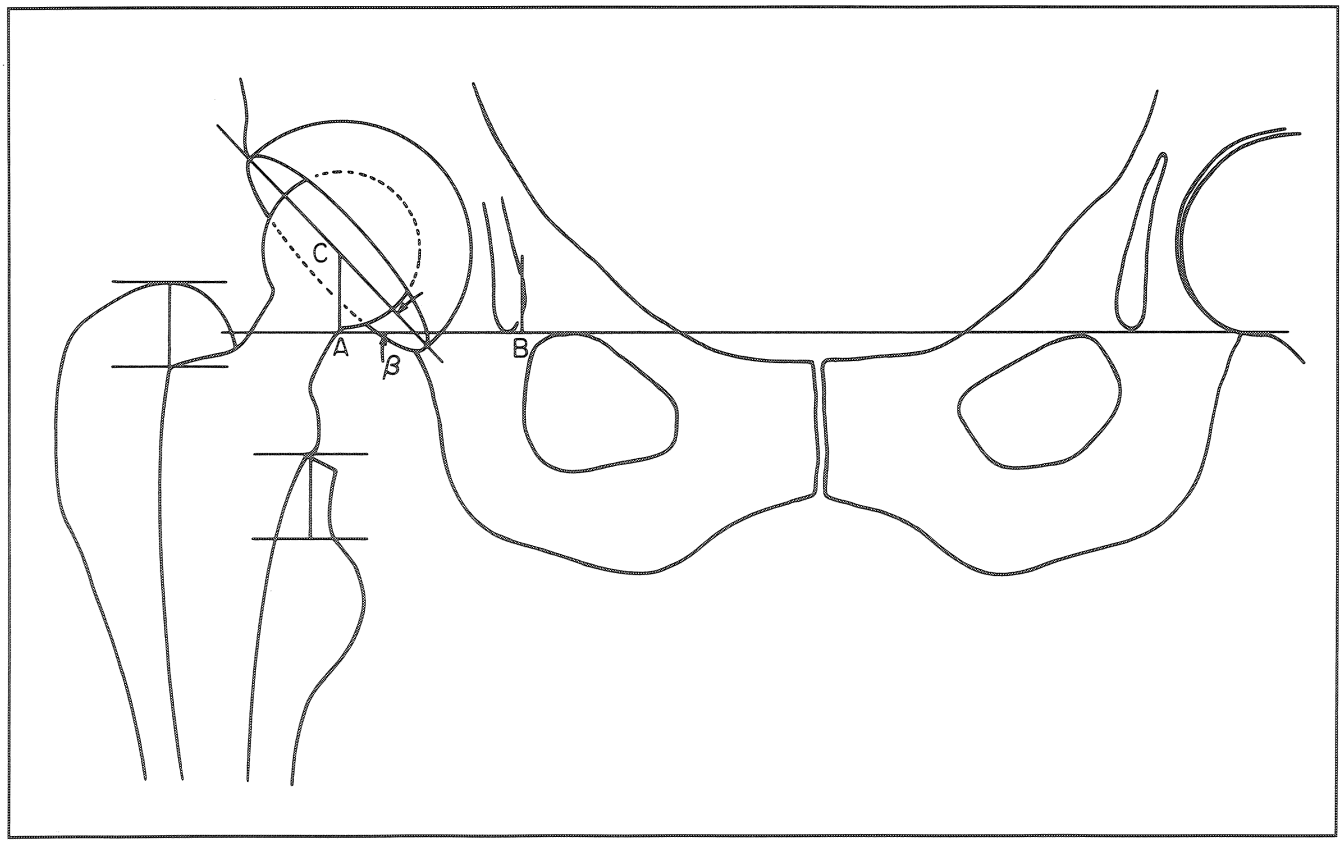


Figure 5. Lateral radiograph demonstrating three-point cortical contact.

BELOW:
Figure 6. Acetabular component inclination and migration were measured referencing the inter-teardrop line.



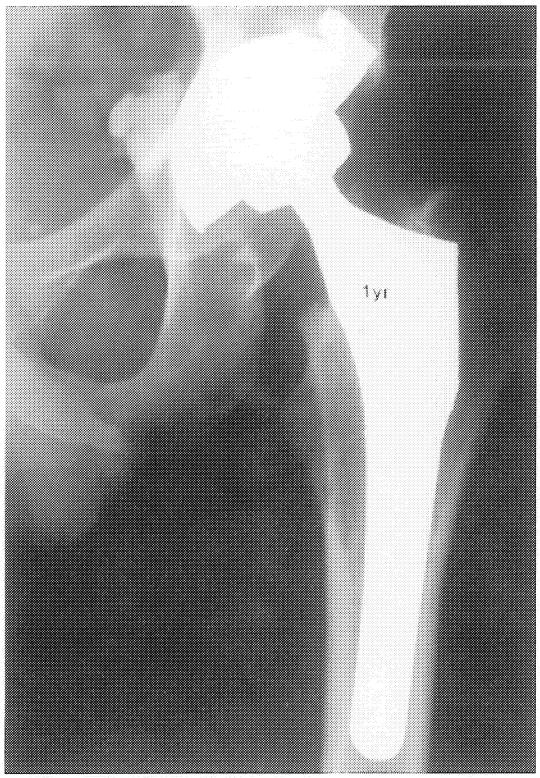


Figure 7a.

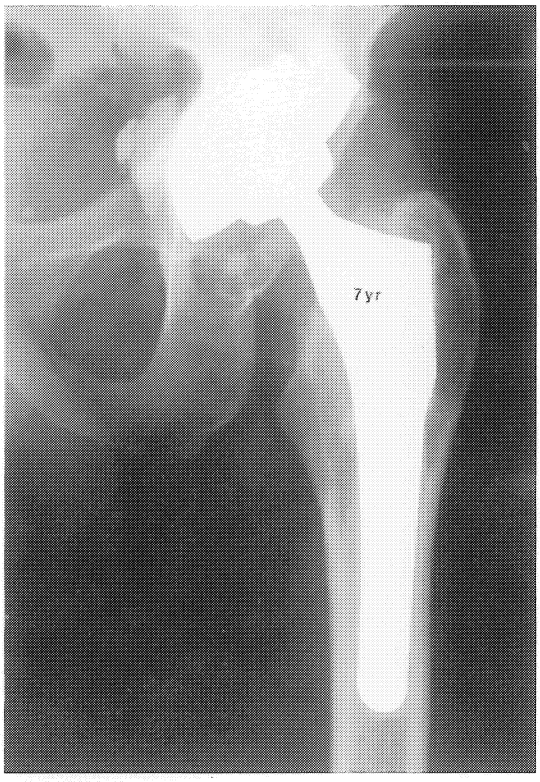


Figure 7b.

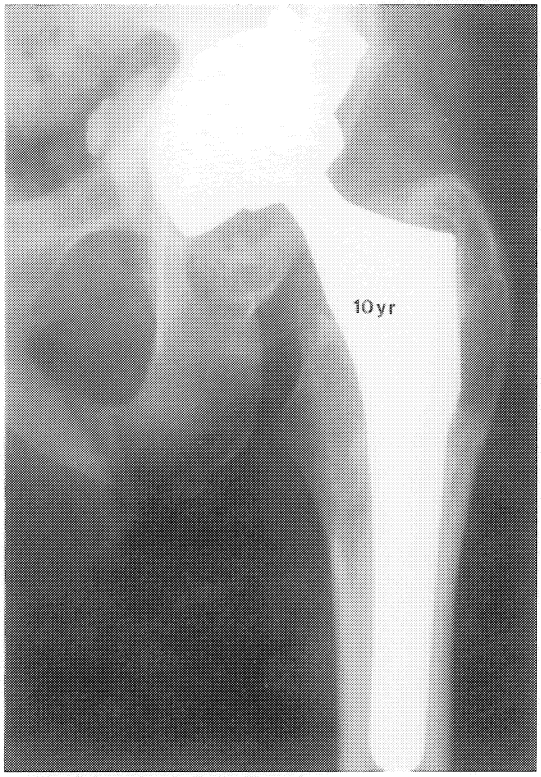
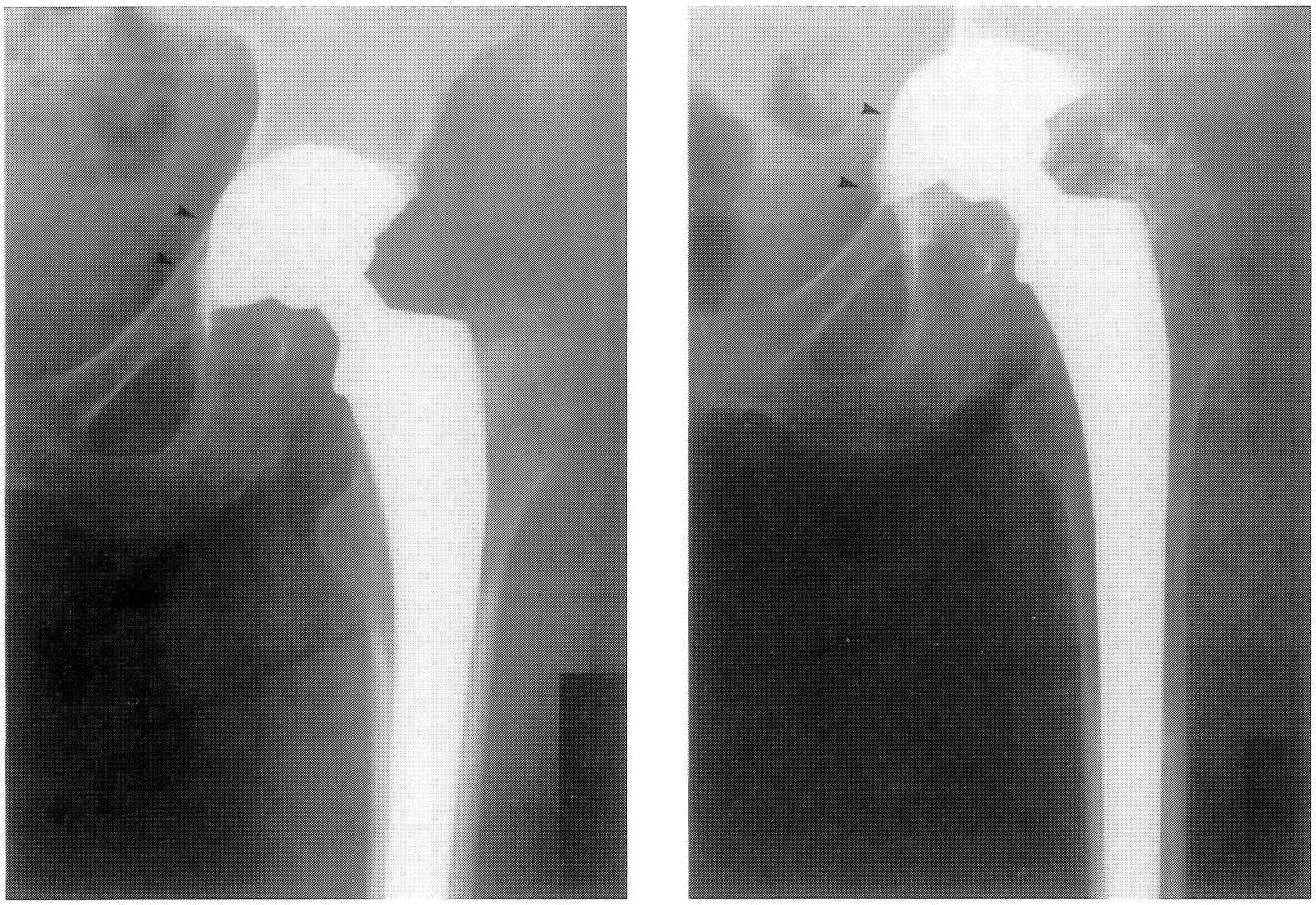


Figure 7c. Serial films demonstrating initial socket stability (7a) followed by osteolysis and eventual migration of the socket into the osteolytic defects.



Figures 8 a-b. a) Immediate postoperative and b) 5-year follow-up radiographs of a patient with rheumatoid arthritis (Case 6) who had early and progressive medial migration of the socket and a complete radiodense line. The migration has stabilized and the patient has remained asymptomatic throughout.

Fifty-six percent of patients had no limp. 26% had a slight limp (detected by examiner) 14% had a moderate limp (noted by patient) and 3% had a severe limp. The mean score for use of walking aids was 9.7 out of 11 points. Three-fourths of patients used nothing, 13% used a cane part time, 8% used a cane full-time, and 3% used more than a cane. Thigh pain was present in 8 (5%) cases. Pain was considered mild (VAS 1 to 4) in 4 (2.3%), moderate (VAS 5 to 7) in 3 (1.7%) and severe (VAS 8) in 1 patient.

Radiographic Results

Rounding or maturation of the femoral neck osteotomy occurred in 70% of femora. Radiographically visible femoral bone resorption was present in 58%, and absent in 42%. Resorption was limited to mottling of the very proximal femur (Zones 1 and 7) in 45%, and mottling combined with cortical thinning in another 13%. Mottling was most common in Zones 1 and 7 together (33%), and Zone 7 (18%). Cortical thinning occurred in Zones 2 and 6 in 5%, and was distal to Zones 2 and 6 in only 2.3% of femora. There were no cases of complete cortical resorption.

Endosteal "spot welds" were observed in only 9%. A single "spot weld" seen on only one side of the implant, occurred in another 9%. Distal femoral cortical hypertrophy occurred in 53%. It was both periosteal and endosteal in 32%, periosteal only in 12% and endosteal in 9%. Periosteal hypertrophy was most common in Zones 3 and 5 (21%), as was endosteal hypertrophy (32%). The hypertrophy ranged from a faint amount of new bone to a massive response that encased the stem and "ballooned-out" the cortex. Thin radiodense lines were extremely common around the distal smooth portion of the stem. A "halo" pedestal formed in 40%, a "shelf" pedestal occurred in 6% of cases (Figure 9).

Initial subsidence occurred with 16 (9%) of femoral stems. The sinkage measured between 3 and 5 mm in 13 cases and was greater than 5 mm in 3 cases. In every case, the subsidence ceased within 6 months. No femoral stem demonstrated progressive subsidence nor had a circumferential radiolucent/radiodense line. Therefore, no stem was considered unstable at last follow-up. No evidence of femoral endosteal lysis was seen in any case. No

Table 2. Demographic data for the 6 patients with acetabular component instability, including the proposed mechanism of failure (MOF), age at last follow-up, and the interval from surgery to the migration of the socket.

	ACETABULAR COMPONENT INSTABILITY					
	Pt. initials	Age	Interval	MOF	Symptoms	Outcome
1.	MB	58	3 yrs	Migration into lysis	Pain/failure	Revision
2.	DL	40	4 yrs	Migration into lysis	Pain/failure	Revision
3.	PH	41	3 yrs	Trauma	Pain	Revision
4.	KF	65	5 yrs	Migration into lysis	Pain	Observation
5.	JB	60	Initial	Vertical tilting sclerotic bone	Asymptomatic	Observation
6.	BB	61	Initial	Medial protrusion rheumatoid bone	Asymptomatic	Observation

porous coating delamination or shedding was observed and there was no stem breakage in this series.

On the acetabular side, the small gaps seen behind 22 (13%) of the sockets immediately after surgery generally filled in over time. New acetabular radiolucent/radiodense lines occurred in 9 cases; in 8 of these, the lines were neither circumferential nor progressive, and were not associated with migration. In one case, migration occurred with a circumferential radiodense line, and this socket was considered unstable (Case 6, Table 1).

One anteroposterior pelvic radiograph, taken at last follow-up, was malrotated and considered inadequate to determine migration. As previously discussed, 6 (3.5%) acetabular components had migrated and were considered unstable.

Pelvic osteolysis occurred around the socket in 16 (9%) cases. Ten osteolytic lesions were considered major and 6 were considered minor. Progression of lysis, as judged by enlargement of the cysts, occurred in 13 of the 16 cases (Figure 10). In three cases, the sockets had migrated into the osteolytic cysts. Two of these had failed and been revised (Cases 1 and 2, Table 1) and the other is symptomatic. In the other 13 cases, the osteolysis was asymptomatic.

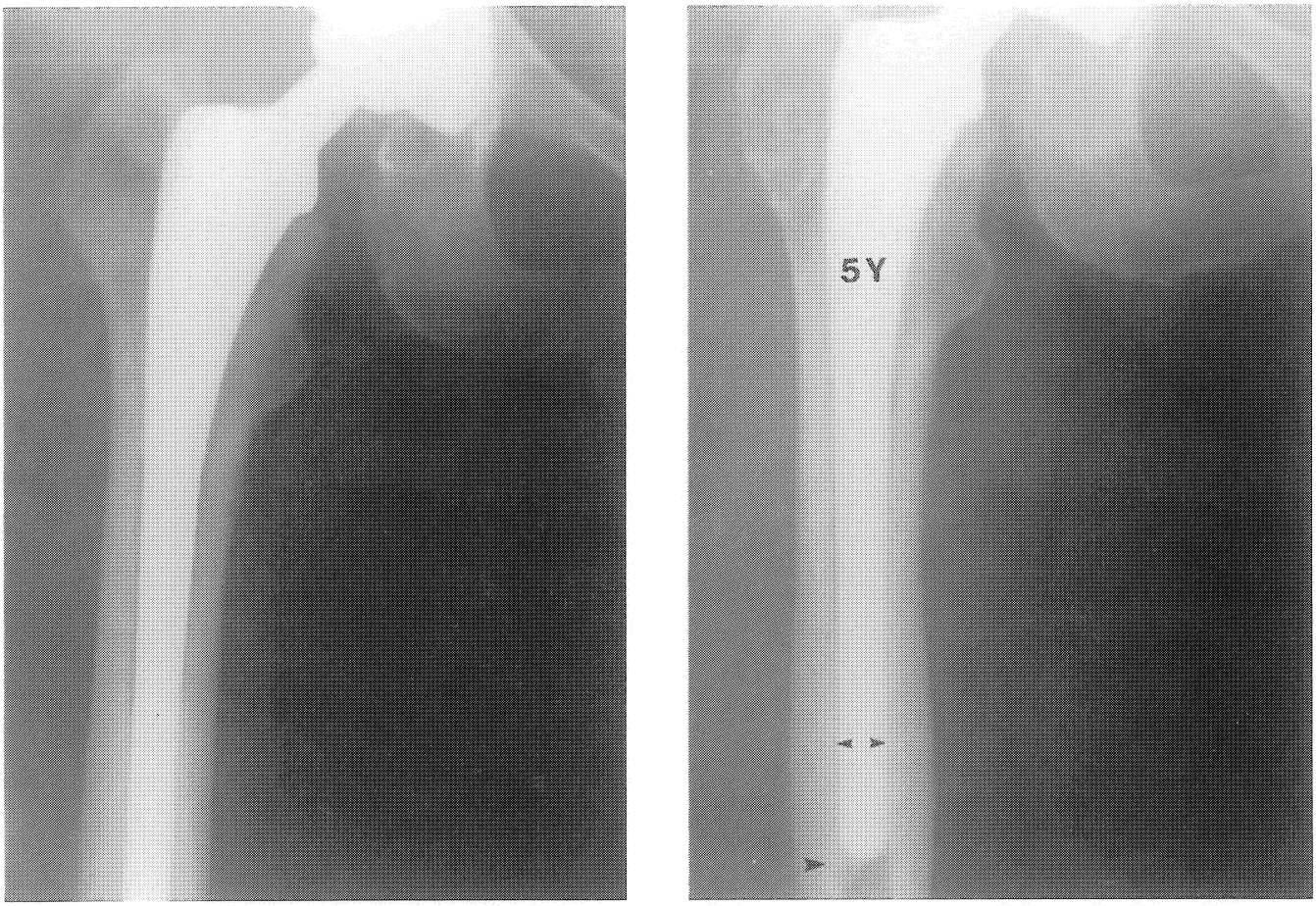
The osteolysis presented from 2 to 6 years (mean 3.8 years) after reconstruction and typically occurred next to an open screw hole in Zone 2. It was always associated with gross eccentricity of the femoral head in the acetabulum (Figure 11). The average age of patients with osteolysis was 58 years, an average of 5 years less than of the entire series. The outer diameter of the acetabular shell ranged from 48 to 60 mm (Mean 53 mm). Other demographic data of patients with osteolysis is presented in Table 3.

Heterotopic ossification occurred in 30% of cases; Grade I in 24%, Grade II in 4%, and Grade III in 2%. There were no cases of apparent ankylosis (Grade IV). Cerclage wiring was performed in four intra-operative longitudinal fractures of the calcar region, all without further event. No distal intraoperative fractures occurred, and no other complications related to the procedure were noted. No postoperative dislocations have occurred in this series.

DISCUSSION

The optimal design features of porous ingrowth cementless total hip prostheses are unknown. Early designs developed problems with thigh pain, lack of ingrowth and stem instability, and osteolysis. Specifically, the prevalence of thigh pain following insertion of the Porous Coated Anatomic (PCA) stem has ranged from 4 to 33%, with the need for revision due to thigh pain in some instances[2,5,19,22]. Stem instability has occurred in up to 11%, and may occur after a period of apparent stability and bone ingrowth[19,22]. Sintered bead shedding has occurred in up to 32% of femoral components[19], and osteolysis has been reported in as many as 33%[25].

The prevalence of stem instability of the Harris-Galante (HG) femoral component has been reported as high as 10% and may increase over time[22,26]. A significant problem with osteolysis has been discovered, and this lysis seems to occur earlier and progress more rapidly than with cemented stems[15,26,36].



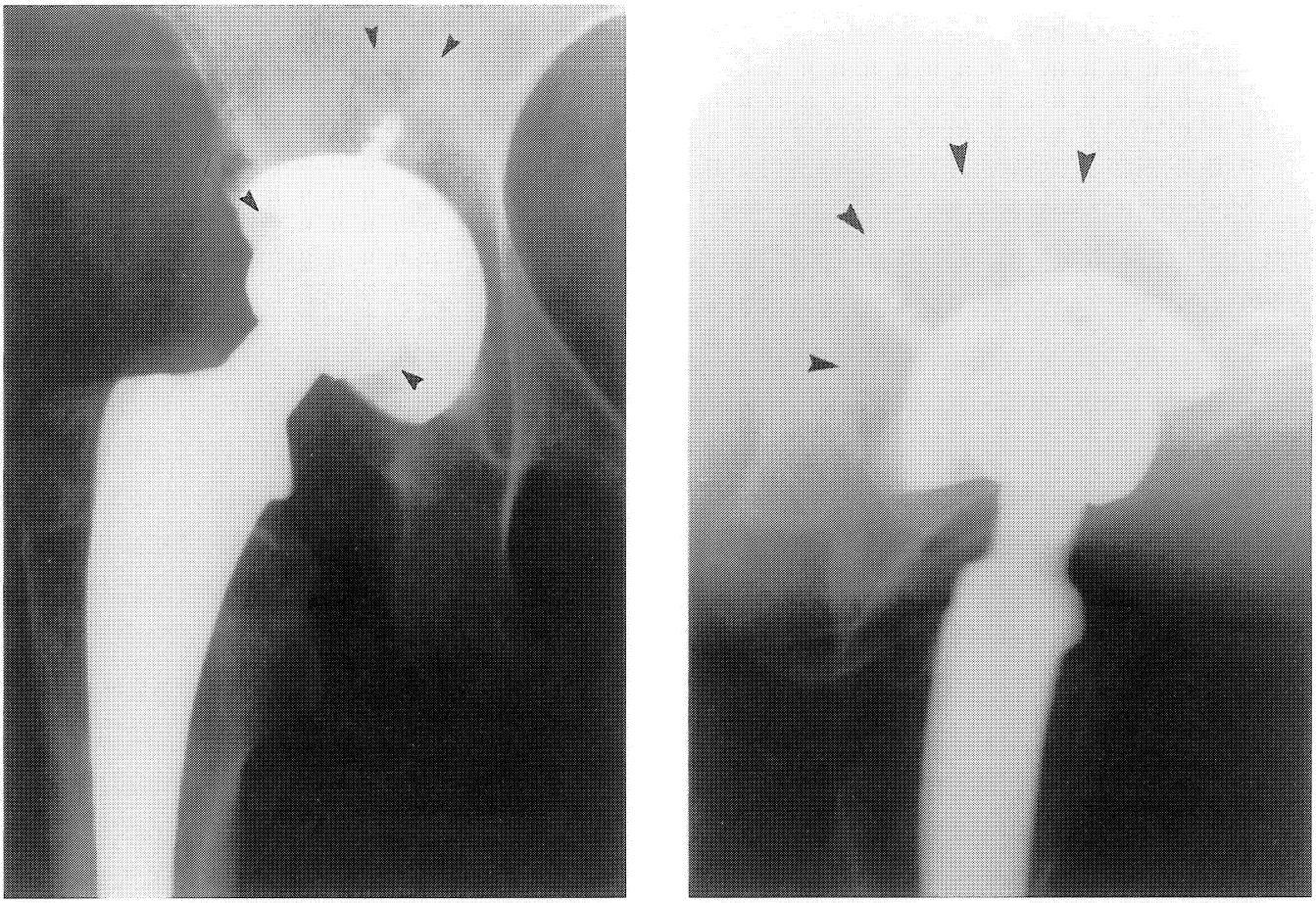
Figures 9 a-b. a) Immediate postoperative film and b) 5-year follow-up demonstrating limited proximal bone resorption, distal cortical hypertrophy (small arrow) and a small shelf pedestal (large arrow). This type and degree of proximal femoral bone remodeling was common following insertion of this stem.

On the other hand, Engh (1990) has reported excellent long-term clinical and radiographic results of the Anatomic Medullary Locking (AML) stem, with a revision rate of only 1% of those stems followed 2 to 12 years. However, others have failed to duplicate these results as of yet, and great concern exists regarding the often extensive resorptive bone remodeling seen after insertion of this femoral component[13,14].

Thus, the attempts at improving cementless hips have focused on the femoral component, where most of the shortcomings seem to have occurred. Currently, the trend towards a circumferential proximal porous coating for bone ingrowth that may form a seal to prevent egression of particulate debris, yet not cause distal femoral stress shielding and bone resorption. However, the optimal component substrate, the type of ingrowth surface and its method of application are controversial[7]. Most authors favor filling the femoral canal to enhance stability, but the best method (e.g. anatomic design, multiple sizes, modularity) of doing so is unknown. The desirability of a collar is debatable, as is the need for bone ingrowth enhancing factors such as hydroxyapatite.

Cementless acetabular fixation has proven reliable and durable in the intermediate-term, and has generally become well accepted[13,33]. However, it should be remembered that failure of cemented sockets rarely occurred before an 8 to 10 year period. Therefore, a much longer follow-up of cementless cups is necessary to conclude their superiority over acrylic fixation. In addition, the best mode of achieving fixation (e.g. over-sizing, screws, pegs, fins, etc) of cementless sockets is unknown[8].

Many different cementless total hip systems have been designed based on the tremendous number of variables available to manufacturers. Often these implants have little scientific basis and have no reports on their clinical efficacy. Our purpose is to report the pertinent results of one cementless total hip arthroplasty, with a short-term 5 year follow-up, in order to guide future efforts of improving cementless fixation. It is not our goal to either espouse or condemn the use of this particular implant or any other.



Figures 10 a-b. a) AP and b) lateral radiograph of the 5-year follow-up demonstrating a gross eccentricity of the femoral head within the socket and a large osteolytic defect. Note that the lesion is clearly larger as seen on the lateral view compared to the AP film.

Two striking findings became evident during this review. First, this femoral component performed extremely well clinically and radiographically, with minimal thigh pain, no revisions, no femoral osteolysis and limited femoral bone resorption. The second and most alarming finding was the tremendous prevalence and severity of pelvic osteolysis behind the cementless socket, at a minimum follow-up of only 5 years.

The long, straight and tapered design of this stem clearly plays a role in achieving stability within the femoral canal. A tight, rotational stable interference fit is usually obtained at surgery and three-point contact of the stem within the cortical bone is often seen postoperatively. Although 9% of femoral stems had early measurable subsidence, no stem had progressive sinkage beyond 6 months, and all appeared stable over serial films including last follow-up. Thus, it has been our experience that even when initial stability is not optimal after stem insertion, secondary stabilization or wedging may occur that is not detrimental to ultimate stability[5]. This has been observed in over 400 Mallory-Head femoral components followed for greater than 2 years at our institution, even in the face of poor bone quality such as in rheumatoid patients, as measured by the cortical and trabecular indices[28]. It is our opinion that stability of this component is easier to obtain than with anatomic stems which require extensive and intimate cortical contact, and non-tapered stems which appear to tolerate subsidence poorly. The failure rate of this Mallory-Head stem is clearly an improvement over other femoral components with a similar length of follow-up.

Very few patients actually complained of thigh pain after receiving this implant. When specifically asked, only 5% could recall any thigh pain and only 1 of the 172 hips was described as having severe and activity-related thigh pain. The potential causes of thigh pain and reasons for lack thereof, are beyond the scope of this paper, and are discussed elsewhere[30]. The avoidance of thigh pain has been a major improvement over the Porous Coated Anatomic stem in our experience[2].

The lack of femoral endosteal lysis in the face of significant pelvic lysis in 9% of patients might be attributable to two ore more factors. First, the stem has a circumferential plasma spray coating which apparently

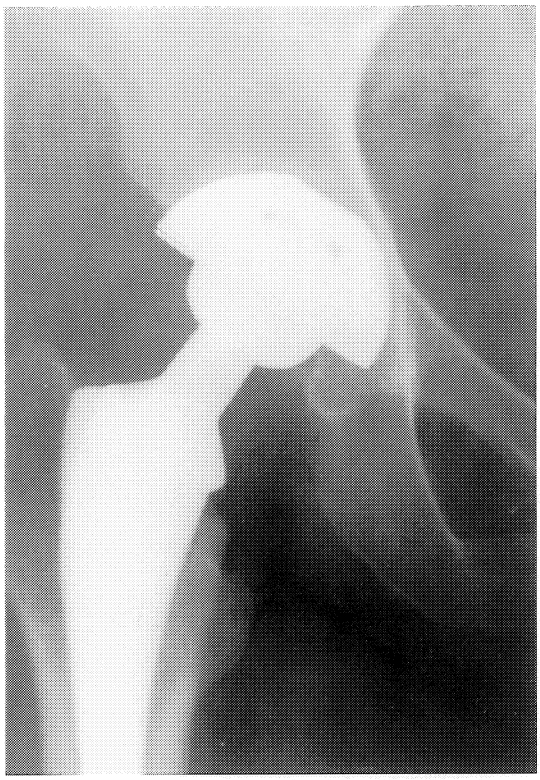


Figure 11a.

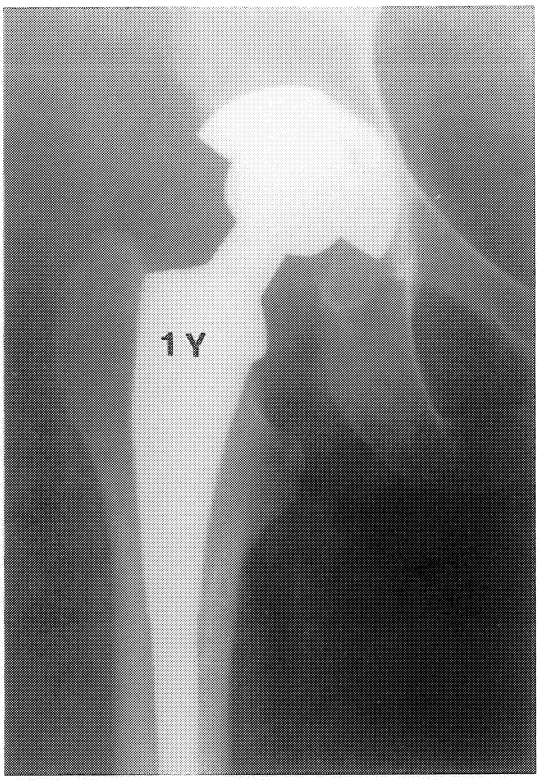


Figure 11b.

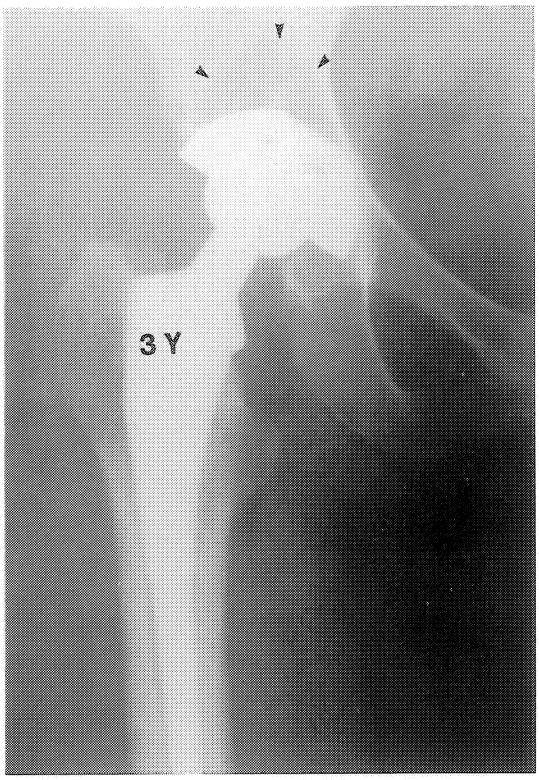


Figure 11c.

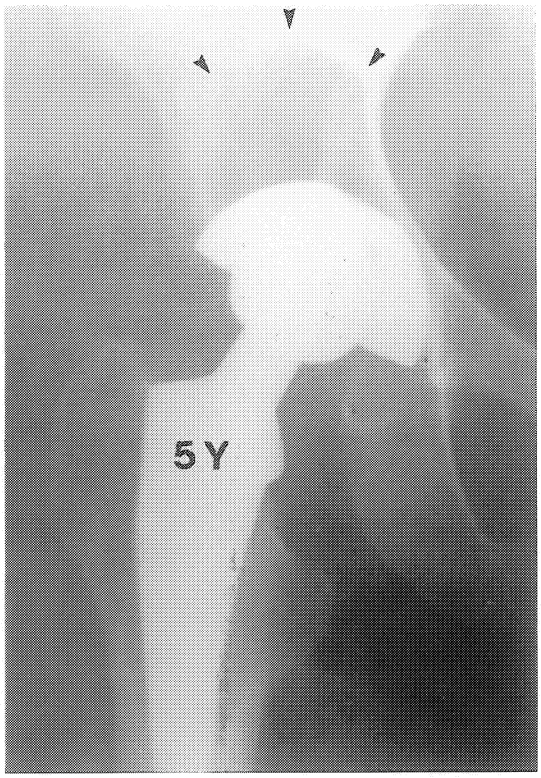


Figure 11d.

Figures 11a-d. Serial radiographs of osteolysis Case 16, where lysis occurred early and was more aggressive than other lesions. Note the tremendously expansile "balloon-line" nature of the lesion. Of note, this hip was inserted with an ion implanted femoral head.

forms a stable interface with bone, in effect, creating a seal against egression of polyethylene debris into the "effective joint space"[1,34]. Secondly, the manufacturer has reported a plasma surface to substrate pull out strength of over 6000 Psi, decreasing the likelihood of particle shedding and osteolysis due to metal debris. This lack of particle shedding and femoral lysis has been observed with the same implant in over 900 cases reported previously (Mallory 1992). We have not observed any endosteal lysis in over 400 of these stems followed from 2 to 7 years. To our knowledge, femoral endosteal lysis has not been reported as a complication of using this stem. Thus, the assumption that femoral lysis occurs more commonly and progresses more rapidly after cementless stem insertion appears to be based on studies of out-dated noncircumferential porous coated stems. In addition, no radiodense/radiolucent lines were seen around the plasma-spray coating of any stem in this series, and there was no radiographic evidence of plasma-coating shedding. Due to lack of retrieval and revision studies, we are unable to comment on the extent of bone ingrowth using this stem. The 3 hips that underwent acetabular revision each had solidly fixed femoral components that resisted every attempt to dislodging the stem from the femoral canal.

Finally, the observation that this femoral component rarely induced distal femoral bone resorption is not surprising, given that the titanium alloy has a modulus of elasticity one-half that of cobalt chrome. The stiffness of this stem more closely approximates that of the normal femur, allowing stress transfer to surrounding bone. This stress transfer, along with tapering of the stem, are also the likely explantations for the distal cortical hypertrophy seen in over one-half of femora in this series. Although cortical hypertrophy has been reported previously with other cementless stems, it seemed to occur more frequently and often in an exaggerated fashion following insert of this stem[28]. However, it appeared to be a benign radiographic finding without other clinical or radiographic indicators of stem instability.

Pelvic osteolysis following cementless socket insertion has emerged as major concern in total hip arthroplasty. Santavirta et al. reported aggressive granulomatosis lesions around the acetabular component in 5 patients following cementless hip insertion[31]. All patients had pain and required revision. Histology confirmed a histiocytic reaction thought to be due to the plastic debris from the acetabular liner. Maloney et al. reported 14 cases of severe pelvic osteolysis discovered from 53 to 84 months after cementless acetabular reconstruction[24]. Despite the lysis, patients were functioning well, and only one acetabular cup had migrated. Ten hips required reoperation with the findings of severe bone loss and polyethylene debris. The factors considered potentially responsible were thin polyethylene, large diameter femoral heads and open screw holes that acted as conduits for particulate debris. Schmalzaried et al. reported pelvic osteolysis in 19 cases of 113 hips reconstructed with a Chamfered-Cylinder acetabular component, and followed an average of 64 months[34]. They attributed the lysis to the invasion of inflammatory tissue at the soft periacetabular cancellous bone, which was not influenced by the lack of screw holes in the socket. In our series, pelvic osteolysis has already developed in 16 (9%) cases at a minimum of only 5 year follow-up. These often "balloon-like" lesions have occurred early (an average of only 3.8 years after reconstruction), and were noted to progress in size in 13 of the 16 cases. As testimony to the significance of the problem, 10 of these lesions were thought to occupy greater than one-third the circumference of the socket, and lysis had caused failure (migration) in 3 cases thus far.

Two potential sources of particulate debris causing this osteolysis are titanium metal from the implant and polyethylene from the liner. Titanium does have a lower fatigue strength than cobalt chrome, and is more adversely affected by abrasion and wear. The titanium has also been shown to incite a more exaggerated inflammatory response than cobalt chrome ions, potentially leading to more aggressive bone lysis[18]. On the other hand, polyethylene has been a consistent finding in essentially all pathologic specimens from osteolytic lesions. It is our opinion that the osteolysis seen after insertion of this cementless total hip replacement is due to polyethylene wear debris. This is based on the observation of gross femoral head eccentricity within the acetabular shell, especially in those patients demonstrating osteolysis. In addition, there has been a lack of component migration to correspond with the lysis, and no evidence for plasma spray metal debris. To date, only 2 of these hips with osteolysis have come to revision surgery. In both cases, there was wear-through of the polyethylene to the metal shell and metal and polyethylene were both contributory to the lytic process.

The potential causes of this rapid polyethylene wear and severe osteolysis are many. Titanium alloy Ti-6A1-4V femoral heads were used in each of the total hip arthroplasties reported here. The first 60 were made of pure titanium and the remainder were ion-implanted to improve the hardness of the femoral head. Although this alloy many have many favorable qualities, it has been shown to be a poor bearing surface, subject to excessive wear with the generation of metal and polyethylene debris. Ion implantation has been reported to improve the resistance to wear and fatigue properties of titanium, making it a more suitable bearing surface[24]. Others have questioned the effectiveness of ion implantation, proposing an actual increase in wear from the surface of treated implants over

Table 3. The demographic data for those patients with pelvic osteolysis where OD equals the outer diameter of the socket and interval equals the time from surgery to the first appearance of osteolysis on x-ray. The size of the lesion was considered major if it occupied greater than one-third the circumference of the metal shell.

PELVIC OSTEOLYSIS

Initials	Age	OD	Gender	Pelvic Interval	Osteolysis Site	Progression	Zone	Symptoms	Outcome
RW	65	54	M	6 yrs	major	yes	2	asympt.	following
JA	45	60	M	6 yrs	major	yes	2	asympt	following
KF(unstable cup, case 4)	65	54	M	4 yrs	major	yes	1,2	pain	following
MG	60	50	F	6 yrs	major	yes	2	asympt.	following
FZ	56	60	M	4 yrs	minor	yes	2	asympt.	following
AB	76	58	M	3 yrs	minor	no	2	asympt.	following
PT	62	56	M	4 yrs	major	yes	1,2	asympt.	following
RD	43	52	M	4 yrs	minor	yes	2	asympt.	following
MB (unstable cup, case 1)	58	52	M	2 yrs	major	yes	1,2	pain/failure	revised
WB	60	56	M	3 yrs	minor	no	3	asympt.	following
EG	80	54	F	5 yrs	minor	no	2	asympt.	following
NB	52	50	M	2 yrs	major	yes	1,2	asympt.	following
HO	67	52	M	3 yrs	minor	yes	2	asympt.	following
DL (unstable cup, case 2)	40	48	F	3 yrs	major	yes	1,2	pain/failure	revised
MO	75	50	F	4 yrs	minor	yes	2	asympt.	following
VS	40	48	F	3 yrs	major	yes	2	asympt.	following

the long-term[29]. In this series, ion implantation did not appear to affect the eventual prevalence of pelvic osteolysis, as 10 of the 16 hips with lysis were inserted with femoral heads that had been implanted. The remaining six cases were with heads that were untreated. It is our opinion that a titanium alloy, treated or untreated, should not be used as a bearing surface, and this has been our practice for the past 4 ½ years.

Another potential factor is the hexagonal locking mechanism used to secure the polyethylene to the socket in each of these hips. This noncongruent surface is susceptible to edge loading and volume pumping of particulate debris. Currently, this design has gone out of favor for more congruent shell-in-shell designs, which we currently use today. Multiple, unused screw holes present in the acetabular shells of each of these hips may act as a source and pathway for debris. The surgeon deemed it necessary to use screws in only 20% of cases, and we currently select solid acetabular shells in all cases, unless initial component instability is an issue. It should be emphasized that initial instability occurred in only 2 of 172 acetabular shells, both of which have remained asymptomatic. Osteolysis did develop in patients who were younger than the average age of the patients in the entire series by 5 years. The average socket diameter was only 53 mm in those patients with osteolysis. It may be that placing high demands on then polyethylene is the most important factor in the production of polyethylene wear debris and osteolysis. Currently, we are performing three-dimensional wear studies on total hip arthroplasties, both with and without osteolysis to identify the potential factors responsible for polyethylene wear, and to correlate this wear with osteolysis[10].

Clearly this magnitude of osteolysis will present tremendous reconstructive challenges in the future. This magnitude and severity of pelvic osteolysis has not been reported in a single series to date. However, we are certain that other centers are seeing similar problems with this and other cementless hip arthroplasties, and we may be seeing only the "tip of the iceberg" . Only when we can identify the sources, and correct the shortcomings involved in this osteolysis, can a long-term biological fixation be expected with this type of implant. Hopefully, the changes made by the manufacturer (e.g. eliminating titanium femoral heads, hexagonal liners) will help remedy this problem.

In conclusion, we have described the results of the cementless Mallory-Head total hip arthroplasty followed 5 to 7 years. The clinical results have generally been excellent. The rate of revision has been low to date, and involved only the acetabular side. Femoral component stability has been excellent without the concerns regarding resorptive bone remodeling or early osteolysis. Initial mechanical stability of the acetabular shell has also been excellent, but severe pelvic osteolysis has developed in 9% of patients at only a minimum 5 year follow-up, and will continue to be a major source of problems in the future.

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A SURGICAL PROTOCOL FOR COMBINED INTERNAL AND EXTERNAL FIXATION OF COMPLEX INTRAARTICULAR DISTAL RADIUS FRACTURES USING DORSAL AND VOLAR APPROACHES

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BACKGROUND

The majority of distal radius fractures are stable and amenable to closed treatment methods. Unstable fractures with intra-articular displacement require an alternative approach. This concept has become increasingly apparent since Knirk and Jupiter demonstrated that accurate restoration of the articular surface of the distal radius is the most critical factor in achieving a successful outcome[6]. Restoration of the articular surface can be accomplished with either internal or external fixation methods[1,3,5,7,8]. However, not all intra-articular fractures of the distal radius are amenable to treatment with one method alone. There is a subset of complex high energy fractures that appear to be best treated by a combination of internal and external fixation. This is presumably necessary because of increased fracture comminution and displacement, and the inability to achieve and maintain a satisfactory reduction with one method alone. In 1989, such a strategy was developed by the senior author. This strategy has been used prospectively with successful results[2]. The strategy features internal fixation performed through dorsal and volar approaches in addition to external fixation used for neutralization. The purpose of this paper is to describe in detail the technical features of the protocol using an illustrative case example. The strict methodical nature of the protocol is extremely helpful in reconstructing these complex, challenging injuries.

The primary indication for this strategy is the AO-C3 type injury, a severe intra-articular fracture with dorsal and volar metaphyseal comminution (Figure 1). The classification is based primarily on the appearance of the fracture on initial and post-reduction radiographs. Tomograms and computerized tomography, if obtained to better delineate the fracture pattern, can also assist with classification. The presence of associated carpal bone fractures, dislocations, or fracture-dislocations are not considered contraindications. Furthermore, the combined dorsal and volar approaches should not be used in those fractures where either approach alone could be successfully used; namely, B2, C1 and C2, or C3 type fractures where the dorsal or volar fragments are non or minimally displaced.

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SURGICAL TECHNIQUE

The procedure is performed under a general anesthetic. The injured upper extremity and contralateral iliac crest are prepped and draped. The extremity is exsanguinated

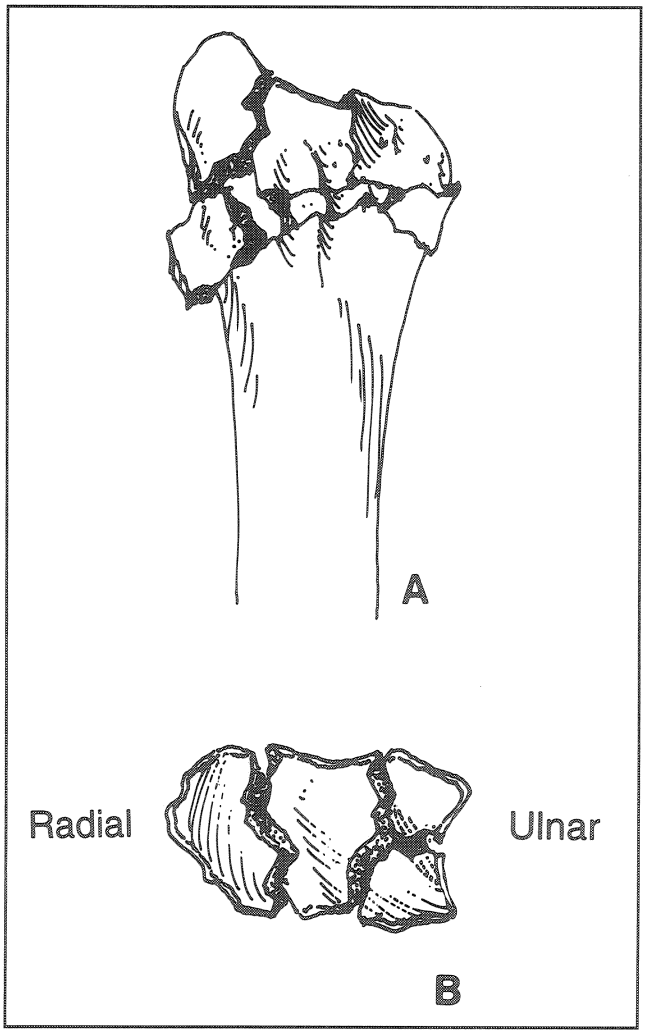

Figure 1. AO - C3 fracture of the distal radius in (a) coronal and (b) axial planes.

and the under tourniquet control, a dorsal longitudinal skin incision is made, centered over Lister's tubercle. The third dorsal wrist compartment is entered and the extensor pollicis longus (EPL) tendon is retracted radially. The distal radius is then exposed subperiosteally in a radial and ulnar direction. Care should be taken during subperiosteal dissection of the fourth compartment to avoid detaching the triangular fibrocartilage complex (TFCC). The proximal aspect of the radial styloid fragment must be completely exposed using subperiosteal elevation. Preliminary identification of the dorsal fracture pattern is performed after the fracture hematoma is evacuated. An attempt should be made to preserve any soft tissue attachments to all fragments. If free fragments are present, their position is noted and they are temporarily removed and preserved in lactated Ringer's solution. A moist sponge is then placed in the wound and the forearm is gently fully supinated.

The volar approach is performed through the flexor carpi radialis (FCR) sheath. The pronator quadratus is exposed and the distal radial corner is sharply released. The pronator is elevated subperiosteally and retracted proximally and ulnarly. All volar fragments are mobilized and the overall fracture pattern identified in relation to the dorsal fragments. A volar capsulotomy is not performed so the integrity of the extrinsic radiocarpal ligaments can be preserved.

The Dynafix distal radius external fixator (EBI Medical Systems, Parsippany, NJ) (Figure 2) is then applied to the second metacarpal and radial shaft using a limited open technique. The second metacarpal is initially exposed via a longitudinal incision beginning at the metacarpal base and extending distally three centimeters. The second metacarpal shaft is exposed bluntly. The hand held drill guide is positioned over the metacarpal at a 45° angle with respect to the plane of the palm and perpendicular to the metacarpal shaft. The drill sleeve is inserted and the proximal hole drilled first, while lifting the guide slightly away from the bone to observe the drill engaging the center of the metacarpal. The first 70/20 screw is placed by hand through both cortices using the T-handled wrench. The drill guide is placed over the first screw, the drill sleeve inserted, and a second, more distal hole is similarly drilled. The second screw is then placed. The fixator is tentatively positioned on the metacarpal screws in a preset shortened configuration. The anticipated location of the incision for placement of the proximal screws is marked through the proximal clamp. This should be planned so that the screws will be proximal to the fracture. A longitudinal incision is made in the dorsoradial forearm and the antebrachial fascia exposed bluntly. The superficial radial nerve is identified, mobilized and protected. The radial shaft is exposed between the wrist extensors and brachioradialis. The proximal 80/30 screws are placed in the radius in a similar manner. Screw lengths are assessed with fluoroscopy. Screws that have not penetrated the opposite cortex are advanced until one thread is beyond the far cortex. Because the bone screws are tapered, 'long' screws should not be backed out. The incisions are then closed with interrupted 5-0 nylon sutures. Any tension in the skin around the screws is released. The Dynafix frame is then applied placing the short bar distally and the C-frame dorsally (Figure 2). If the C-frame is not centered over the fracture, the site of the screw clamps on the bar may need adjusting. Reduction of the fracture is then performed.

Alignment of the hand relative to the forearm is fundamental to achieving a satisfactory reduction; it is accomplished using a specific reduction maneuver. Firm longitu-

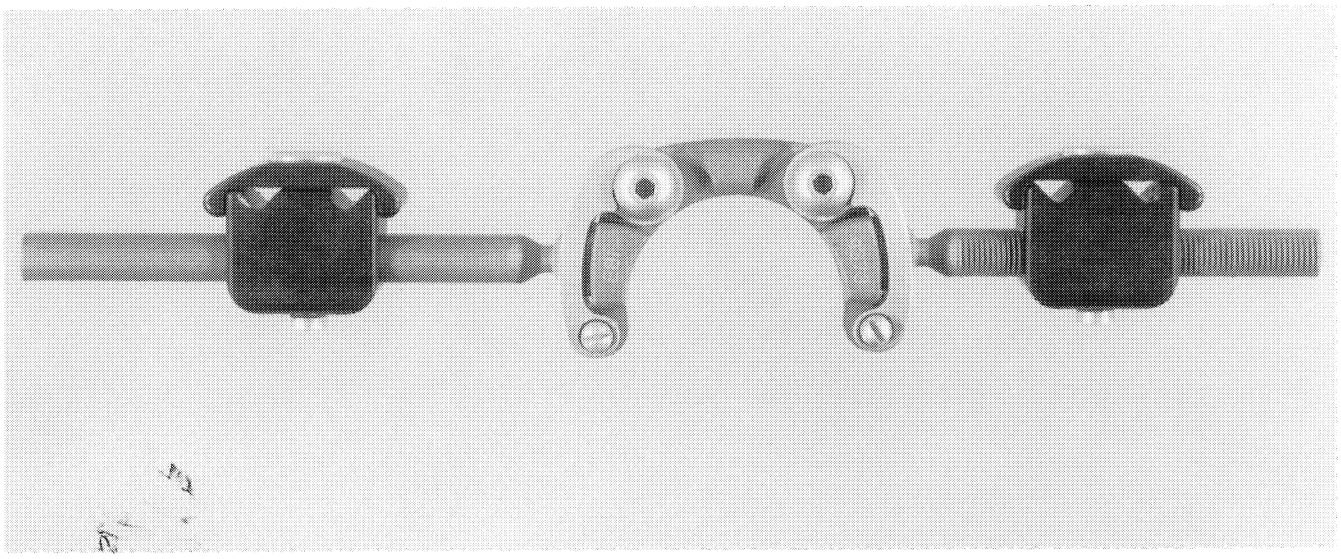

Figure 2. The Dynafix distal radius external fixator (EBI Medical Systems, Parsippany, NJ).

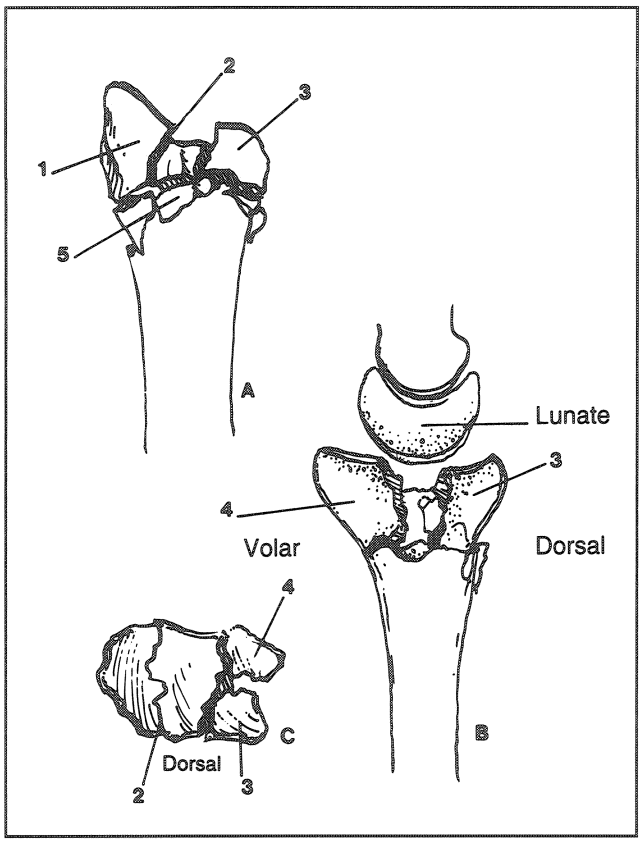

Figure 3. Schematic representation of an AO C type 3.2 distal radius fracture in the (a) coronal (b) sagittal and (c) axial planes. Note the (1) radial styloid fragment, (2) fracture line dividing the scaphoid fossa, (3) lunate fossa 'die punch' fragment, (4) volar lunate fossa fragment, and (5) dorsal metaphyseal comminution.

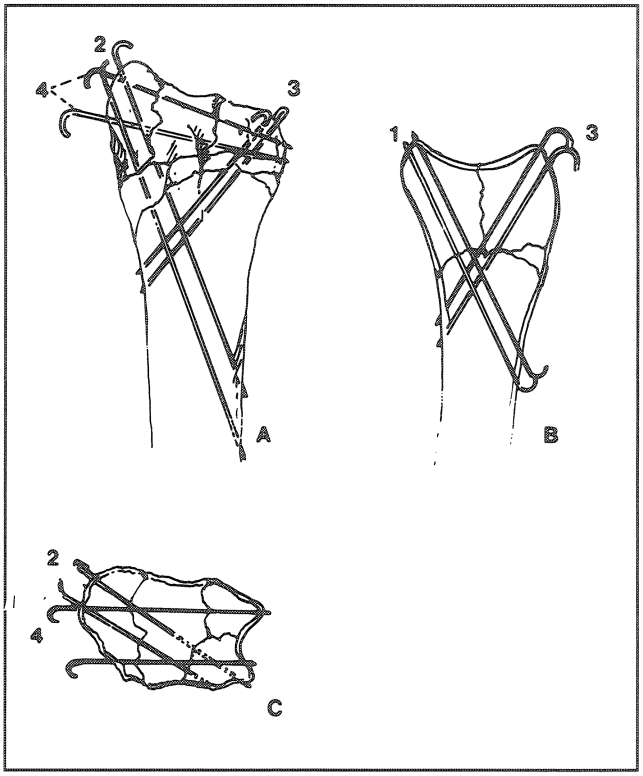

Figure 4. The fixation sequence of the AO C type 3.2 fracture shown in Figure 3. Reconstruction of the fracture is begun volarly in a proximal to distal direction (Figure 4B). The preferred sequence for Kirschner wire placement is numbered in order and demonstrated in the accompanying illustrations (Figures 4A and C).

dinal traction through the index and long fingers is applied with the elbow secured. The ulnar aspect of the wrist is supported and when proper alignment and rotation of the hand relative to the forearm is obtained, the fixator is locked into place. Posteroanterior (PA) and lateral fluoroscopic images of the wrist are obtained. Confirmation of optimum alignment of the hand relative to the forearm is critical. The axes of the radius, lunate, and capitate must be colinear in both views before proceeding to reconstruction of the fracture. The degree of restoration of radial length, inclination, and palmar tilt, as well as congruence of the radiocarpal and distal radioulnar joints should be assessed[4].

Fluoroscopic images should be scrutinized for any carpal injuries. If a carpal fracture or ligamentous injury is identified, distraction across the wrist is released, and the carpal injury is treated.

A dorsal transverse capsular incision is then made just distal to the dorsal radial lip of selected fragments to better visualize the distal radius articular surface. The capsular incision is extended longitudinally and distally along the axis of the lunate, thus creating an inverted T-shaped capsulotomy. The carpus is inspected for fracture and chondral or ligamentous injuries. Gentle distraction is then applied and the sequence of reduction, fixator tightening and radiographic assessment repeated.

Reconstruction of the fracture is performed under direct visualization through both incisions. Anatomic reduction is usually accomplished by reducing and fixing the fragments in a proximal to distal and volar to dorsal sequence. We prefer to use smooth Kirschner wires for fixation but neutralization plates or interfragmentary screws are appropriate for selected fracture patterns.

The volar fragments are reduced first and secured to the radial shaft with Kirschner wires drilled in a distal volar to proximal dorsal direction (Figure 4B). The wires are advanced through the dorsal skin and then backed out dorsally until the tips are left just penetrating the volar cortex. Wire placement into the area of dorsal metaphyseal comminution or the extensor tendons must be avoided. Reconstruction is then continued dorsally. The radial styloid fragment is reduced and secured to the shaft by two wires inserted percutaneously (Figures 4A and C). The articular surface is then reassembled and stabilized via dorsal to volar and/or radial to ulnar directed wires placed directly or percutaneously (Figures 4A and B). The wires

may stabilize the reduced fragments by engaging them directly or by acting in a buttress mode beneath, or adjacent to, the anatomically reduced fragments. Rarely, wires can be placed through the fragments and into the carpus to achieve fixation if necessary. The construct is then assessed on PA and lateral fluoroscopic images. The previously mentioned anatomic parameters, wire positions, and wire lengths are checked. The wires are cut and bent above the skin. Extensive metaphyseal bone loss is invariable, and cancellous bone grafting from the iliac crest is usually required. Adequate cancellous graft can usually be obtained using coring instrumentation. The dorsal capsulotomy is closed with nonabsorbable suture in an interrupted figure-of-eight fashion. The extensor retinaculum is closed beneath the EPL tendon. The pronator quadratus is sutured to the lateral margin of the radius with a nonabsorbable suture whenever possible. The tourniquet is deflated and hemostasis is obtained prior to skin closure. Drains are placed if necessary and a compressive dressing is applied.

POSTOPERATIVE TREATMENT AND REHABILITATION

The extremity is elevated, and aggressive active and passive digital range of motion and daily pin care are initiated on the first postoperative day. The skin sutures are removed at two weeks, at which time full active index finger range of motion should be verified. The goal is full active range of motion of all fingers within three weeks of the operation. In general, Kirschner wires used for distal radius fracture fixation are removed 6-8 weeks postoperatively. Wires used for carpal fractures or ligamentous injuries are removed 8-10 weeks postoperatively. If radiographs confirm consolidation of the fracture and graft at 10-12 weeks, the external fixator is removed and a volar forearm based thermoplastic splint is fashioned. The patient is instructed in a supervised progressive wrist range of motion and strengthening program.

COMMENTARY

The preliminary results of this treatment strategy for AO-C3 fractures of the distal radius have been reported by the senior author (WB)[2]. The co-author of this paper is gaining considerable experience with this protocol and has found the strict methodical nature of the protocol to be extremely helpful in reconstructing these complex, challenging injuries.

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KI-67 IMMUNOSTAINING AS A TOOL IN THE DIAGNOSIS OF CENTRAL CARTILAGE LESIONS

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INTRODUCTION

Intramedullary cartilage lesions of bone present diagnostic dilemmas which have challenged orthopaedic surgeons, radiologists, and pathologists for decades. One such dilemma, distinguishing enchondroma from central chondrosarcoma, represents one of the most difficult problems in bone pathology. Distinguishing these two lesions has traditionally been based on clinical, radiographic, and histologic criteria which are evident at a particular point in time. Using these criteria, high-grade chondrosarcoma is usually easily distinguished from enchondroma. Unlike patients with an enchondroma (Figure 1), patients with a high grade chondrosarcoma have pain, their radiographs show aggressive growth patterns (Figure 2), and their biopsies show cartilage with marked atypia. By contrast, low-grade chondrosarcoma may be very difficult to distinguish from enchondroma because of considerable overlap in clinical, radiographic and pathologic features. In fact, based on traditional diagnostic criteria, these lesions may be impossible to distinguish. As a result, pathologists may render such diagnoses as "borderline cartilage lesion" or "cartilage neoplasm of uncertain malignant potential." Unfortunately, these diagnoses are of little use to surgeons who must make therapeutic decisions.

The diagnostic dilemma posed by some intramedullary hyaline cartilage lesions may be solved by considering a dynamic parameter in addition to traditional static diagnostic criteria. Diagnosticians can ask, "Is the lesion growing?" This parameter, which considers what is happening to the patient, is often a more reliable indicator of prognosis than diagnostic labels based on traditional static features. If we define an enchondroma in an adult long bone as a non-growing cartilage lesion and a low-grade chondrosarcoma as a slow-growing lesion, evidence of growth has important therapeutic implications. For example, if a lesion is not growing, the patient may be followed. A growing cartilage lesion, however, requires surgical ablation.

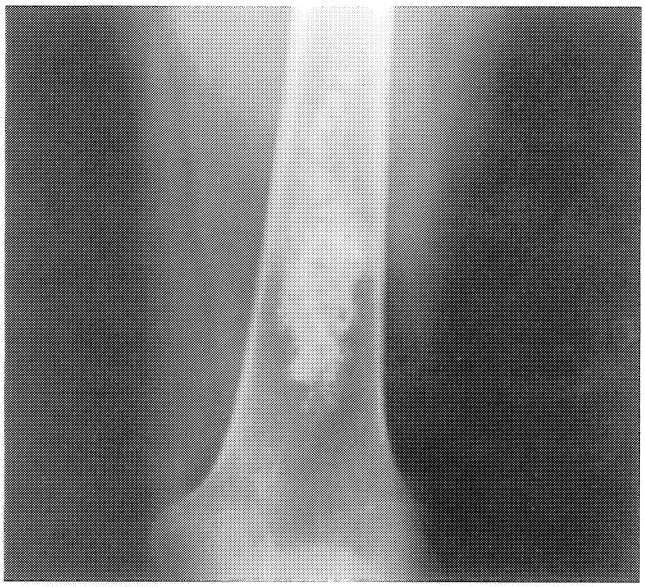

Figure 1. Typical enchondroma.

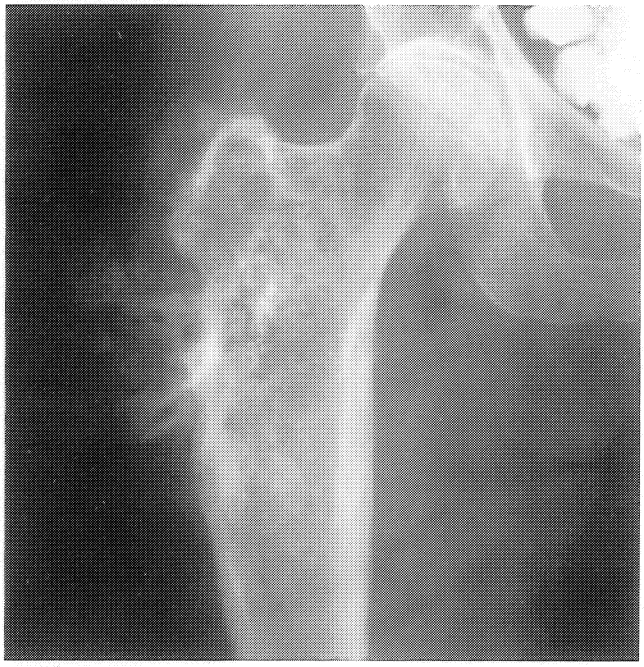

Figure 2. High-grade chondrosarcoma.

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Proliferative activity of cells is a reliable index of neoplastic growth. In most neoplasms, this activity can be evaluated by counting the number of mitotic figures. However, mitotic figures are extremely rare or even absent in low-grade chondrosarcomas. Therefore, the absence of mitotic figures in a cartilage lesion does not mean the lesion is stationary. Fortunately, dividing cells may be recognized even when mitotic figures are not visible. Cycling cells synthesize several nuclear proteins which are not present in resting cells. One of these proteins may be identified by its immunochemical reaction with the monoclonal antibody Ki-67. The presence of this protein indicates proliferating cells[2,21]. This staining reaction has been useful in studying the proliferative activity of several solid tumors including brain tumors and carcinomas of the colon and uterus. Studies show that the more Ki-67 positive cells, the more aggressive the neoplasm[3,4,10,11,16,18,20]. However, its use in bone tumors has been explored only to a limited degree[17,19]. We examined the usefulness of the Ki-67 immunostain in determining the growth potential of intramedullary cartilage lesions. We studied 36 lesions which, based on standard diagnostic criteria, had been classified as enchondroma, low-grade (grade 1) central chondrosarcoma, and high-grade (grades 2 and 3) central chondrosarcoma.

MATERIALS AND METHODS

Thirty-six central cartilage lesions from thirty-six adult patients were studied. Patients were seen at the Johns Hopkins Hospital between 1975 and 1995 or called from the personal consultation files of one of us (EFM). The cartilage lesions had been diagnosed as enchondroma, low-grade (grade 1) chondrosarcoma, or high-grade (grades 2 and 3) chondrosarcoma by a combination of clinical, radiographic, and histologic features. For this study, the clinical history, radiographs, and histologic slides were reviewed and the original diagnoses were confirmed. Then, from the original formalin-fixed paraffin blocks, 5 mm sections were stained with Ki-67 monoclonal antibody (Immunotech, Westrook, ME) using the immunoperoxide technique. The dilution was 1:150 and visualization was achieved with an avidin-biotin-horseradish peroxidase system. Diamino-benizidine was used as the chromagen and hemotoxylin was used as a counterstain. Two tissue blocks containing fetal cartilage were also studied by the same method and used as controls.

When available, sections from at least two tissue blocks from each case were studied. Nuclei from all the neoplastic chondrocytes were evaluated.

Ki-67 staining was scored on a three-tiered scale. A score of 0 indicated no chondrocyte nuclear staining; 1+ indicated staining of 1 to 10% of chondrocyte nuclei; and 2+ indicated staining in 11 to 20% of chondrocyte nuclei. No cartilage lesion had greater than 20% of the nuclei stain positive with Ki-67.

RESULTS

Of the 36 lesions studied, 16 were classified as enchondroma. These lesions occurred in locations other than the hands or feet. Four additional lesions classified as enchondromas of the hand were also studied. Six lesions were low-grade central chondrosarcomas and ten were high-grade central chondrosarcomas (Table 1).

The fetal cartilage controls contained Ki-67 positive nuclei in 20 to 35% of the chondrocytes. This was an expected finding given the rapid growth of fetal tissue. The chondrocyte nuclei in all 16 cases of non-small bone enchondromas were uniformly unstained. Almost all of these lesions had been treated by curettage. There were no recurrences in the fifteen patients on whom we had follow-up.

Unlike our enchondromas in the long bones, three of the four lesions diagnosed as enchondroma in the hand contained Ki-67 chondrocytes. These lesions had been treated by curettage; no patient had a recurrence.

Ki-67 positive chondrocytes were present in four of the six low-grade chondrosarcomas. Patients had been treated by segmental resection of the lesions. No patient had a recurrence.

The ten high-grade chondrosarcomas all contained Ki-67 positive chondrocytes. In five cases, the staining was 2+. Patients had been treated by resection or amputation, and follow-up was available on all but one case. Two patients died with lung metastases at four and eight years. Two more recent patients have lung metastases after two years; they are both still alive. Except for one patient who died of postoperative complications, the others are alive and disease free.

DISCUSSION

In 1943, Lichtenstein and Jaffe formulated histologic criteria for the diagnosis of chondrosarcoma. After reviewing 10 cases of "central" chondrosarcoma and 27 cases of enchondroma, they suggested that a cartilage tumor could no longer be regarded as benign "if, when viable noncalcifying areas are examined, it shows, even in scattered fields: (1) many cartilage cells with plump nuclei; (2) more than an occasional cell with two such nuclei; and especially (3) giant cartilage cells with large single or multiple nuclei or with clumps of chromatin."[8] These cytologic criteria have been used by many pathologists to distinguish enchondroma from chondrosarcoma. However, because many enchondromas are cellular and may contain some plump and binucleated chondrocytes, the distinction between low-grade chondrosarcoma and enchondroma is difficult to make.

TABLE 1

Ki Immunostaining of Cartilage Lesions

Patient	Diagnosis	Location	Immunostaining	Treatment	Follow-up
30M	Enchondroma	Pelvis	0	Curettage	4 yrs. No recurrence
48F	Enchondroma	Humerus	0	Curettage	6 yrs. No recurrence
34F	Enchondroma	Humerus	0	Curettage	2 yrs. No recurrence
39F	Enchondroma	Humerus	0	Curettage	5 yrs. No recurrence
42F	Enchondroma	Fibula	0	Curettage	5 yrs. No recurrence
52F	Enchondroma	Fibula	0	Resection	4 yrs. No recurrence
44M	Enchondroma	Femur	0	Curettage	7 yrs. No recurrence
45F	Enchondroma	Fibula	0	Resection	20 yrs.No recurrence
66M	Enchondroma	Rib	0	Resection	6 yrs. No recurrence
43F	Enchondroma	Humerus	0	Curettage	Lost to followup
44F	Enchondroma	Femur	0	Curettage	5 yrs.No recurrence
50F	Enchondroma	Humerus	0	Curettage	2 yrs.No recurrence
49F	Enchondroma	Humerus	0	Biopsy only	No growth
61F	Enchondroma	Rib	0	Resection	3 yrs.No recurrence
36F	Enchondroma	Tibia	0	Curettage	8 yrs.No recurrence
46F	Enchondroma	Tibia	0	Curettage	2 yrs.No recurrence
19F	Enchondroma, Hand	Metacarpal	1+	Curettage	12 yrs.No recurrence
47F	Enchondroma, Hand	Metacarpal	1+	Curettage	1 yr. No recurrence
32M	Enchondroma, Hand	Phalanx	2+	Currettage	3 yrs.No recurrence
50M	Enchondroma, Hand	Phalanx	0	Curettage	4 yrs.No recurrence
36M	Chondrosarcoma,Low-grade	Humerus	1+	Resection	6 yrs.No recurrence
19F	Chondrosarcoma,Low-grade	Rib	1+	Resection	10 yrs.No recurrence
20M	Chondrosarcoma,Low-grade	Fibula	0	Resection	2 yrs. No recurrence
60M	Chondrosarcoma,Low-grade	Ulna	1+	Resection	12 yrs.No recurrence
40M	Chondrosarcoma,Low-grade	Humerus	1+	Resection	1 yr. No recurrence
45F	Chondrosarcoma,Low-grade	Humerus	0	Resection	7 yrs.No recurrence
37F	Chondrosarcoma,High-grade	Humerus	2+	Resection	died 8 yrs., lung met.
83F	Chondrosarcoma,High-grade	Femur	2+	Amputation	died 4 yrs., lung met.
62M	Chondrosarcoma,High-grade	Femur	1+	Amputation	2 yrs., lung met.
43M	Chondrosarcoma,High-grade	Rib	1+	Resection	10 yrs.No recurrence
52M	Chondrosarcoma,High-grade	Scapula	2+	Resection	lost to follow-up
63M	Chondrosarcoma,High-grade	Fibula	1+	Resection	2 yrs. lung met.
35F	Chondrosarcoma,High-grade	Rib	2+	Resection	2 yrs.No recurrence
36F	Chondrosarcoma,High-grade	Femur	2+	Resection	3 yrs.No recurrence
52F	Chondrosarcoma,High-grade	Scapula	1+	Resection	3 yrs.No recurrence
64M	Chondrosarcoma,High-grade	Pelvis	1+	Resection	died post-op

1+ indicates staining of from 1 to 10% of nuclei

2+ indicates staining of from 11 to 20% of nuclei

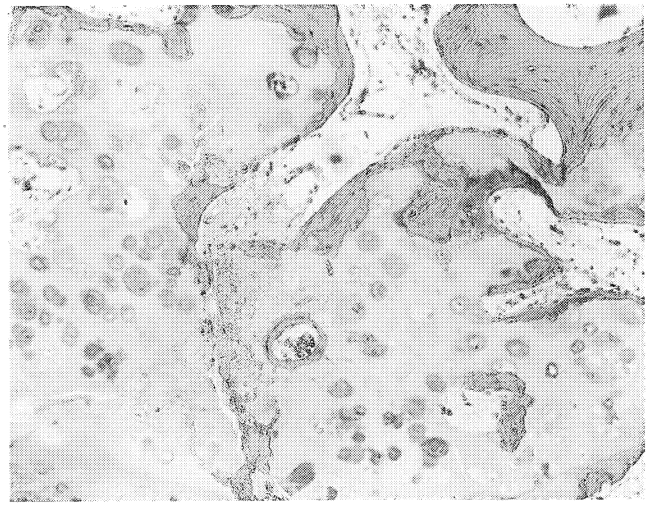

Figure 3. Enchondroma with lobules encased by endochondral bone.

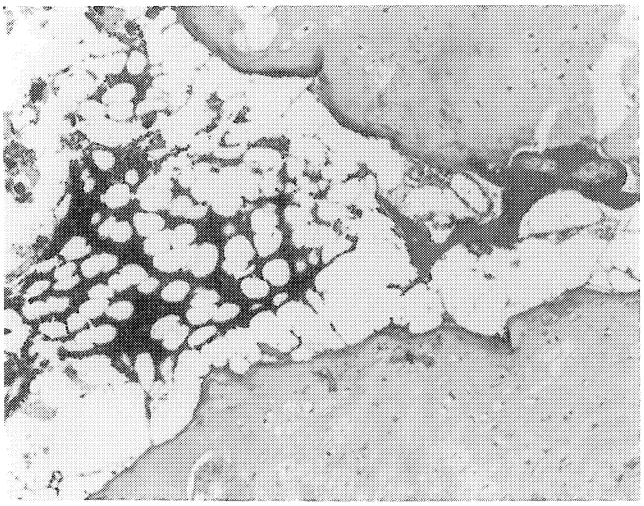

Figure 4. Enchondroma with lobules separated by bone marrow.

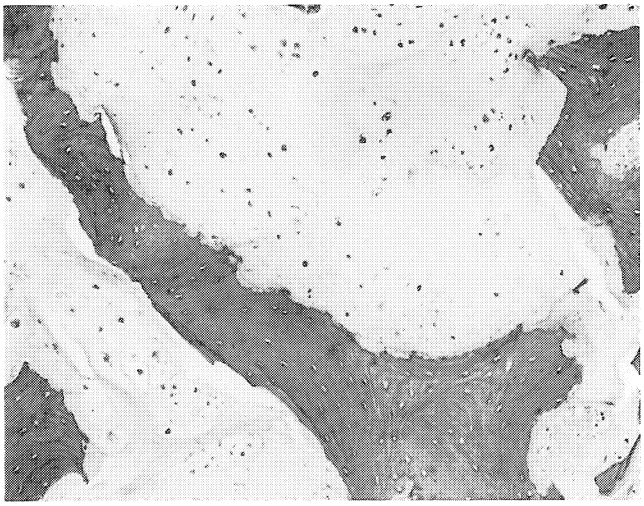

Figure 5. Chondrosarcoma with encasement of native trabecular bone by neoplastic cartilage.

Because of overlapping radiographic and histologic features, numerous studies have attempted to refine diagnostic criteria of central hyaline cartilage lesions[5,9,12-15]. One study is of particular note. In 1985, Mirra et al. studied 51 cases of central cartilage lesions of long bones[9]. They described several distinctive histologic patterns which, when present, are useful in distinguishing enchondroma from chondrosarcoma. For example, enchondromas typically show lobules of cartilage which are encased by endochondral bone (Figure 3) and/or separated by normal bone marrow (Figure 4). By contrast, chondrosarcomas show neoplastic cartilage surrounding native trabecular bone (Figure 5) and/or penetrating the cortex. In addition, lobules of chondrosarcoma are often separated by fibrous bands. The authors concluded that these histologic patterns are manifestations of cartilage growth activity.

In an effort to develop more objective criteria of predicting the behavior of cartilage neoplasms, researchers have correlated behavior with the DNA content of the chondrocytes using cytophotometry[7] or flow cytometry[1]. While these studies show that hyperploid chondrocyte nuclei are associated with aggressive behavior, they are of little use in distinguishing low-grade chondrosarcoma from enchondroma. Most low-grade chondrosarcomas are diploid and thus have the same DNA content as enchondromas.

Despite refined histologic and radiographic criteria, and despite DNA quantification, it still may be difficult to distinguish enchondromas from low-grade chondrosarcomas. We have found that framing all diagnostic parameters with the question, "Is the lesion growing?" has been useful in managing patients.

Growth of a cartilage lesion may be assessed in two ways: radiographic changes over time and the presence of pain. First, we feel that a patient with an equivocal cartilage lesion should be followed by monthly plain radiographs for six months and then at six month intervals for two years. If the lesion is growing, radiographic features will change and the diagnosis of low-grade chondrosarcoma can be established. The prognosis should not be worsened by a delay of this duration. In addition to follow-up radiographs, efforts should be made to find any previous radiographs of the affected bone. The problem of a worrisome or ambiguous lesion may be clarified if a radiograph, taken years earlier, shows exactly the same lesion.

In addition to radiographic changes over time, the presence of pain is a second important clue indicating growth of a cartilage lesion. However, the orthopedic surgeon must be certain that the pain is due to the neoplasm. Often, unrelated joint pain, particularly of the

knee or shoulder, will prompt radiographic studies which reveal incidental cartilage lesions. Relief of pain by resting the joint or analgesic injection indicates that the pain is not due to the cartilage lesion.

Another cause of pain associated with a non-growing cartilage lesion is a stress fracture. Large enchondromas, particularly in weight-bearing bones, may suffer this complication. A more aggressive neoplasm may be suspected, especially if an MRI study shows marrow and soft tissue edema. With awareness of this complication and patient observation, the non-growing nature of the cartilage lesion usually declares itself.

Approaching a cartilage lesion with the question, "Is it growing?" often helps surgeons avoid needless procedures, including biopsies which may be misinterpreted. Occasionally, however, tissue from a borderline cartilage lesion must be sampled. Our study indicates that Ki-67 immunostaining is a valuable adjunct to routine histologic analysis. Positive nuclear staining to any degree indicates neoplastic growth. Therefore, a central borderline cartilage lesion in a long bone of an adult should be regarded as a low-grade chondrosarcoma if it contains Ki-67 positive cells.

In our study, all lesions diagnosed as enchondroma by conventional methods showed no Ki-67 reactive cells. None of these lesions developed recurrence after curettage or growth after biopsy. By contrast, all ten high grade chondrosarcomas showed significant staining reaction. Four of six low-grade chondrosarcomas showed Ki-67 positive cells. Only one of these five lesions recurred. Two explanations are possible for the two low-grade chondrosarcomas which did not stain. First, they may have been enchondromas which were overdiagnosed using traditional methods. Alternatively, since 50% of central chondrosarcomas show areas of preexisting enchondroma[9], limited sampling may have failed to obtain proliferating tissue. Therefore, because of the possibility of sampling error in borderline lesions, a negative Ki-67 stain does not unequivocally indicate a stationary lesion. To minimize sampling error, all paraffin blocks containing neoplastic tissue should be studied. Although a negative reaction is inconclusive, a positive stain indicates neoplastic growth; the lesion should be regarded as a chondrosarcoma. Thus, Ki-67 staining may be useful in distinguishing low-grade chondrosarcoma from an enchondroma when traditional diagnostic criteria are ambiguous (Figure 6).

Unlike in the long or flat bones, Ki-67 positivity does not indicate chondrosarcoma in cartilage lesions of the hands or feet. In these locations, lesions which have been regarded as enchondromas are typically more cellular, contain more atypical chondrocytes, and often show destructive radiographic patterns. These features in a long bone cartilaginous lesion would be considered evidence of malignancy. Our findings of Ki-67 positive cells in three of four enchondromas of the hand parallel the aggressive histologic and radiographic features. Therefore, in the hands and feet, we can expand the definition of enchondroma to include some growing lesions because they are associated with little morbidity. For these lesions, less radical treatment, such as curettage, is effective. The distinction between enchondroma and chondrosarcoma in the hands and feet must, therefore, be based on special histologic features such as invasion of soft tissues, necrosis, and myxoid change. Although we did not study the enchondromas in patients with Ollier's disease, we suspect that these lesions are also Ki-67 positive. Similar to enchondromas of the hand, lesions in these patients are more cellular, show more chondrocyte atypia, and show active radiographic features.

Like cartilage lesions in the hands or feet, Ki-67 positivity in a childhood cartilage lesion does not indicate malignancy. In children, cartilage lesions are subject to growth stimulae similar to the epiphyseal plate. These cartilage lesions, although benign, grow until skeletal maturation. A Ki-67 stain would be positive.

Other studies of Ki-67 staining of cartilage lesions have been limited. In 1989, Vollmer et al. studied Ki-67 staining on frozen sections of a wide variety of bone tumors[19]. They studied three conventional chondrosarcomas and four enchondromas. From 2.8 to 6.8% of chondrosarcoma cells stained with Ki-67; the enchondromas contained from 2% to 2.5% positive cells. However, three of the enchondromas were in the feet, a location in which cartilage lesions behave like those in the hand.

In 1995, Scotlandi et al. studied Ki-67 staining in various bone tumors in 205 patients[17]. These investigators evaluated K-67 by indirect immunofluorescence performed on cytologic specimens. Although they did not study enchondromas, sixteen primary chondrosarcomas showed Ki-67 positivity in from 2.9 to 4.5% of cells.

In addition to Ki-67, other antibodies may be used to identify nuclear proteins expressed in cycling cells. One such antibody is PC10 which recognizes proliferating cell nuclear antigen (PCNA). This antibody was employed by Hasegawa et al. in 1995 to study 24 cartilage lesions[6]. Only one of six enchondromas showed positivity, and 11 of 13 chondrosarcomas showed staining. The two negative chondrosarcomas were low-grade neoplasms, a finding similar to ours.

The diagnosis of cartilage lesions will always be difficult. Approaching a lesion with the question, "Is it growing?" will eliminate unnecessary surgery. The Ki-67 immunostain helps to answer this question. Growing lesions, irrespective of the diagnostic label, must be surgically removed. Non-growing lesions may be observed.

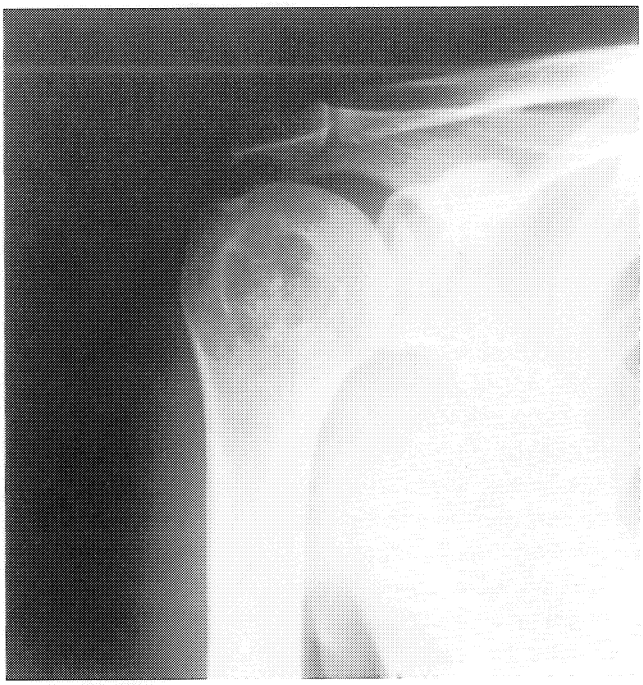

Figure 6A. Radiograph of borderline cartilage lesion.

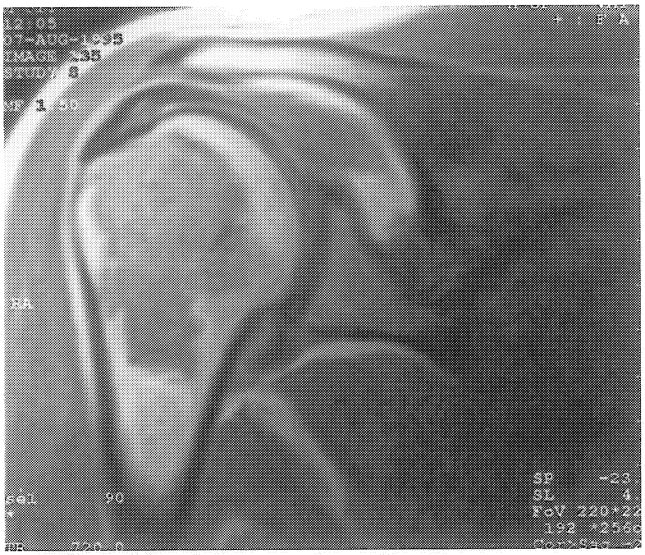

Figure 6B. MRI of the lesion.

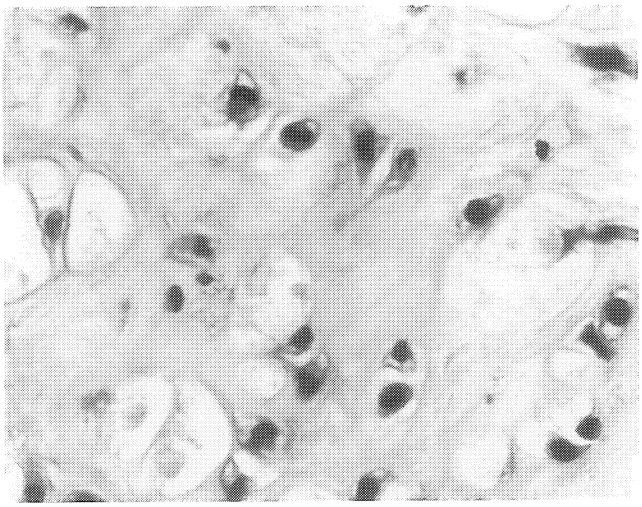

Figure 6C. Biopsy showing hyaline cartilage with mild chondrocyte atypia.

The histologic features of the biopsy based on H & E stains were equivocal. However, the Ki-67 stain was positive. The resected specimen showed a permeative growth pattern typical of chondrosarcoma.

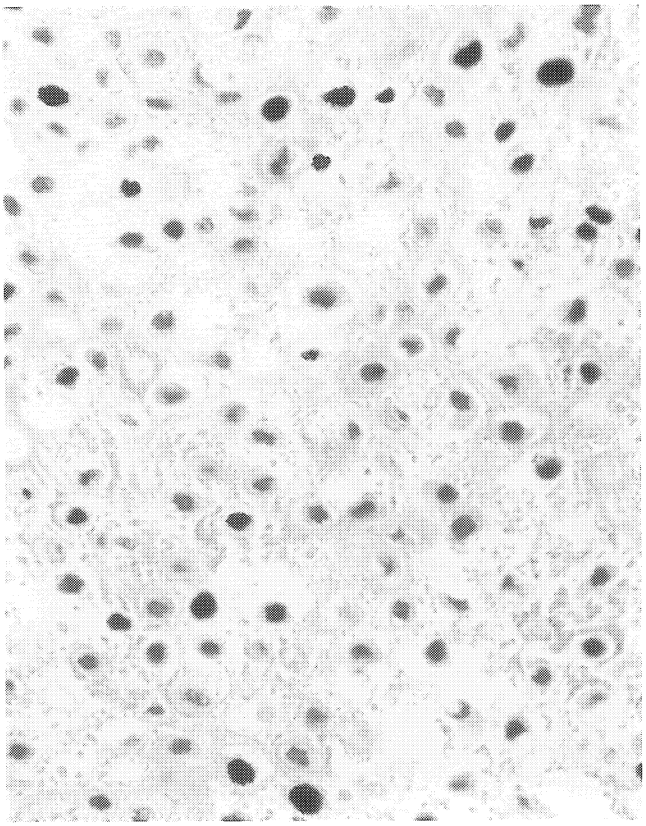

Figure 6D. Ki-67 stain showing positive chondrocyte nuclei. The dark nuclei are Ki-67 positive.

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PERIPHERAL NERVE EXTRACT EFFECTS ON MESENCHYMAL CELLS

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ABSTRACT

Several common congenital limb disorders are characterized by normal tissue differentiation but abnormal somatic growth. These include: idiopathic clubfoot, idiopathic leg length discrepancy, hemi-atrophy and hemi-hypertrophy. Both clinical and research studies have suggested that peripheral nerves may be important in regulating somatic growth of limb tissues.

To investigate the hypothesis that peripheral nerves convey trophic substances to mesenchymal tissues that are involved in the regulation of growth, we developed an *in vitro* assay to assess the effect of fractions of peripheral nerve on myoblast and chondroblast growth and differentiation in a mammalian (rat) system. Whole rat sciatic nerve extract was fractionated by ammonium sulfate precipitation and by affinity chromatography. Concavalin A chromatography resolved whole nerve extract into a glycoprotein and a non-glycoprotein fraction. Serial ammonium sulfate precipitation yielded three pellet fractions designated as 35%, 70%, and 100% pellets; corresponding to ammonium sulfate concentrations of 0 to 35%, 35 to 70%, and 70 to 100% saturation, respectively. Dialyzed solutions of these pellets as well as the fractions from Concavalin A chromatography were assayed for biological activity in micromass cultures of rat limb bud mesenchyme, which allowed assessment of both myoblast and chondroblast stimulation.

Stimulation of protein synthesis and myoblast proliferation (as measured by MF20 staining) occurred with both 70% and 100% ammonium sulfate fractions. Stimulation of chondroblasts (as measured by the number of alcian blue staining nodules) occurred with the 35% and 100% fractions. The glycoprotein fraction from the affinity chromatography stimulated protein synthesis and myoblast proliferation and inhibited chondroblast development. Stimulation of chondroblasts was seen with the non-glycoprotein fraction. No effect on protein synthesis, myoblast proliferation or chondroblast proliferation was found in cultures treated with rat transferrin (transferrin has been reported to stimulate myoblasts in avian culture systems).

INTRODUCTION

Congenital and developmental abnormalities of limbs are common. The etiology of most such anomalies is not known. Dramatic increases in knowledge of the determinants of limb tissue differentiation and pattern (axis) formation have occurred in recent years, often by utilizing techniques of molecular biology. Differentiation of limb tissues results from tissue induction involving both diffusable factors and cell-to-cell interactions[35]. Axis determination involves chemical gradients, growth factors, homeobox genes and their complex interactions[71].

We have been interested in the determinants of normal and abnormal limb growth and development after the occurrance of normal mesenchymal tissue differentiation in limbs. Several common congenital disorders of limbs are characterized by normal *differentiation* of mesenchymal tissues but abnormal *somatic growth*. Disorders with this characteristic include idiopathic clubfoot, idiopathic leg length discrepency, hemi-atrophy, and hemi-hypertrophy. Several lines of research and some clinical evidence suggest that peripheral nerves may be important in the control of somatic growth of limb tissues[2,5,9,17,18,21,66]. More than a half century ago, the differentiation of limb tissues was shown to be independent of innervation. Even these pioneering studies found a diminished somatic growth in denervated limbs[24,25,26,32,69]. We attempted to determine if peripheral nerves contain factors trophic for mesenchymal tissues. The ubiquitous presence of nerves in mesenchymal tissues including ligaments and bone as well as muscle suggest a possible role of peripheral nerves in the control of mesenchymal tissue growth and perhaps homeostasis after completion of growth.

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In order to explore this possibility, we developed an *in vitro* assay to assess the effects of peripheral nerve proteins on mesenchymal cell growth. Our *in vitro* assay allows the simultaneous evaluation of myoblast and chondroblast differentiation and growth in single cultures of limb-bud mesenchyme in a mammalian species (rat). Trophic factors are probably involved in the cascade of tissue interactions that are required for normal limb development and may be implicated in causing some human limb deformities. Prior work has demonstrated the ability of peripheral nerve and peripheral nerve extracts to enhance myoblast growth and differentiation. Disagreement exists as to the identity of the growth promoting factor. Some investigators believe it to be transferrin[4,57], while others dispute this contention[14,50]. We assessed the effect of peripheral nerve extracts and purified transferrin on both myoblasts and chondroblasts in vitro to explore the interaction of peripheral nerve and limb development.

MATERIALS AND METHODS

Summary

A crude peripheral nerve extract was prepared from rat sciatic nerves. This material was fractionated by ammonium sulphate precipitation and affinity chromatography. Fractions of nerve extract were assayed for biological activity using a modification of the microtiter micromass culture of limb-bud mesenchymal cells described by Paulsen and Solursh[58,70]. This system allows simultaneous growth of myoblasts and chondroblasts. The specific assays for assessing myogenesis and chondrogenesis employed were protein determination, counts of cells staining with MF20 (a muscle specific antibody to muscle myosin[3]), and counts of nodules staining with Alcian blue (a chondrocyte specific stain).

Peripheral nerve extract preparation

Frozen rat sciatic nerve (Pel-Freez Clinical Systems, Brown Deer, WI) was pulverized using mortor and pestle while immersed in liquid nitrogen. All subsequent operations were performed at 4 degrees centigrade or in an ice water bath. The pulverized tissue was suspended in ice-cold 10 mM tricine-NaOH buffer, pH 8.0, at approximately 40 grams wet tissue weight in 100 ml buffer. The tricine buffer contained the following protease inhibitors: 0.01 M EDTA, 0.1 M epsilon amino-n-caproic acid, and 0.005 M benzamidine hydrochloride. The suspension was homogenized in a Vertis 45 homogenizer (Virtis Co., Gardner, N.Y.) at medium speed for 4 to 5 minutes. The blade was cleaned and the suspension was homogenized for another 4 to 5 minutes. The homogenized suspension was then stirred with a magnetic stir bar for 4-5 hours at 4 degrees centigrade.

The suspension was centrifuged at 31,000 x g in a Beckman J21B centrifuge for 30 minutes. The lipid layer that floated on the top was removed with a spatula and the supernatant was decanted. The supernatant was further centrifuged at 105,000 x g in a Beckman ultracentrifuge for 2 hours. The final supernatant was filtered through a sterile 0.45 micron syringe filter (SLHV 0130S, Millipore Corp., Bedford, MA) and stored as such or as freeze dried powder at -70 degrees centigrade until used.

Ammonium sulfate precipitation

Freeze-dried nerve extract powder dissolved in 20 mM potassium phosphate buffer (pH 7) or thawed nerve extract (pH 7) was used as the starting material. To this solution finely ground ammonium sulfate (19.4 g/100ml starting volume) was added gradually (in circa 1 h) with stirring; the final concentration of ammonium sulfate was 35% saturation. The slurry was allowed to stand for 1 hour and then centrifuged in a Sorval RC-5B centrifuge (Du Pont Instruments, Wilmington, DE) at 26,000 x g for 20 minutes. The pellet was saved and the supernatant was treated with ammonium sulfate as described above in two successive steps giving 70% and 100% saturations of ammonium sulfate (by adding 21.8 g and 20.9 g ammonium sulfate per 100 ml starting volumes, respectively). The pellets obtained were designated as 35% pellet (precipitated between 0 and 35% saturation), 70% pellet, (precipitated between 35 and 70% saturation) and 100% pellet (precipitated between 70 and 100% saturation), and stored at -70 degrees centigrade.

Before screening for activity in tissue culture, the frozen pellets were dissolved in 20 mM potassium phosphate buffer (pH 7) and the respective solutions were dialyzed against sterile distilled water. In some instances the dialysates contained precipitates which were solubilized by adding sodium bicarbonate solution to a final concentration of up to 70 mM.

Affinity (Concavalin A-Sepharose) chromatography

A buffer designated Con A with pH 7 was used and contained the following: immidazole-HCL, 10 mM; NaCl, 150 mM; $MgCl_2 \cdot 6H_2O$, 1 mM; $MnCl_2 \cdot 4H_2O$, 1mM; NaN_3, 3 mM[40]. A 1.5 cm x 30 cm Econo-column (Bio-Rad Laboratory, Richmond, CA) was packed with Con A-Sepharose (Pharmacia Biotech, Piscataway, NJ) to a bed height of 8.1 cm giving a bed volume of 14.3 ml. The column was equilibrated with Con A buffer and onto this was loaded 64 ml of freshly thawed nerve extract (in 20 mM potassium phosphate buffer and containing 42 mg protein) that was diluted with 64 ml of Con A buffer. Then the bed was washed successively with 85 ml of Con A buffer and 200 ml of 50 mM D-mannopyranoside in Con A

buffer. The flow through the column was maintained at the 30 ml/h rate using a peristaltic pump, and fraction volumes of 2.75 ml were collected throughout the loading and washing periods. The elution of proteins from the column was monitored by measuring the A280 of the fractions using a Perkin Elmer Lamda 3A Spectrophotometer (Norwalk, CT).

Microtiter micromass cultures of rat limb-bud mesenchymal cells

Media. CMRL Media (GIBCO, Grand Island, NY) supplemented with 5% fetal calf serum (FCS) (GIBCO) and antibiotics was used for all mesenchymal cell cultures. Antibiotics added were 50 micrograms per ml of penicillin (Apthecon), 25 micrograms per ml of streptomycin sulfate (GIBCO), and 1.25 micrograms per ml of Fungizone (GIBCO).

The fractions of nerve extract were dialyzed for a minimum of 2 hours against CMRL at 4 degrees centigrade with at least one change of media. This material was then filtered through 0.22 micrometer syringe filters to obtain sterility.

Isolation of limb mesenchymal cells. Cell suspensions were prepared from the forelimbs of 13 to 14 day old rat embryos. The forelimbs were removed under a dissecting microscope in a petri dish containing tyrodes buffer. Cells were dissociated in trypsin-collagenase (0.1% trypsin and 0.1% collagenase [Worthington, Freehold, NJ] in calcium- and magnesium-free Puck's saline G containing 10% chicken serum) in a rotary shaker bath at 37 degrees centigrade for ten minutes. The forelimbs were then pipetted up and down with a Pasteur pitpette to obtain a cell suspension to which two to three ml of CMRL with FCS was added to inactivate the enzymes. The cells were harvested by centrifugation at 130 x g for 6 minutes, washed, and resuspended in CMRL with FCS before filtration through four layers of Nitex 20 nylon mesh.

Inoculation of the cultures. The density of harvested cells was determined using a hemacytometer and adjusted to $2x10^5$ cells per ml. Ten microliters of cell suspension were added to the center of each well in a 48 well cluster plate (Costar, Cambridge, MA). An additional 10 microliter drop of media without cells was placed on the edge of each well to maintain hydration during the attachment period. The tissue cultures were incubated for 2 hours at 37 degrees centigrade in a water-jacketed incubator providing a humidified atmosphere of 5% CO_2: 95% air. After the attachment period, 0.25 ml of media with various amounts of nerve extract or rat transferrin (Sigma, St. Louis, MO) was added to each well. Transferrin has been implicated as the growth stimulating factor supplied by peripheral nerves[4,47,57]; thus its inclusion. Control cultures fed with media not containing nerve extract or transferrin were also included in each experiment. Media was changed with the appropriate concentration of nerve extract every 24 hours until harvesting of cells.

Assays for biological activity

Protein assay. Cultures for protein assay were harvested on day 3. A modification of the method of Lowry was employed to determine values for total tissue protein per culture well. Unfixed cultures were washed several times with phophate buffered saline G. We added 0.3 ml of 10% TCA to the precipitate and scraped the wells with a rubber policeman. The recovered cells were placed in microcentrifuge tubes and rinsed with 0.3 ml of 10% trichloroacetic acid. The tissue was sonicated while being kept cold in an ice bath and then centrifuged at 1500 x g for 15 minutes. The supernatant was removed. The tissue was redisolved in 0.1M NaOH for protein determination. BCA Protein Assay Reagent (Pierce Biologicals) was added per manufacturer's instructions. After incubation at 37 degrees centigrade for 1/2 hour, the absorbance of this mixture at 562 nm was measured. Bovine albumin was used as the standard.

Myogenesis assay. Myogenesis was assessed immunohistochemically using MF-20, a monoclonal antibody directed against sarcomere myosin[3]. Cultures for myoblast analysis were harvested at day 2 or 3. MF-20 staining was performed as follows: cultures were washed in 0.02 M phosphate buffered saline (PBS); covered in 0.3% hydrogen peroxide in methanol to inactivate endogenous peroxidase; washed again in PBS (and after each subsequent step); covered with 5% normal rabbit serum in PBS for 10 minutes; incubated with hybridoma culture supernatant containing MF-20 for 45 minutes; incubated with rabbit-anti-mouse IgG for 45 minutes; incubated in 40 micrograms/ml mouse peroxidase-antiperoxidase in PBS containing 1% normal rabbit serum for 45 minutes; diamiobenzidine substrate was added and color development was monitored microscopically for 4 minutes before stopping the reaction by distilled water rinsing and covering with glycerine. All cells staining for MF-20 were counted using a Wild stereo dissecting microscope.

Chondrogenesis assay. Chondrogenesis was assessed by counting nodules staining for Alcian blue on day 5[34]. Alcian blue at pH 1 specifically binds and precipitates sulfated proteoglycans[11]. Culture wells were washed several times with saline G; washed with PBS; rinsed in 95%, then 70% ETOH for 5 minutes each; stained with 1% Alcian blue, pH 7.0 overnight; rinsed in 0.1 N HCl; and rinsed in double distilled water. Cartilage nodules, which stain with Alcian blue, were tabulated for each well.

Protein assay and gel electrophoresis of cell extract fractions

Protein concentrations were determined according to Bradford[6] using the Bio-Rad Laboratories dye reagent;

Table I

Ammonium Sulfate Fractionation

Ammonium Sulfate Fraction	Protein (mg) X ± S.D. (n)** p value	MF20 Stain (# of positive cells/well) X ± S.D. (n)** p value	Alcian Blue Stain (# of positive nodules/well) X ± S.D. (n)** p value
Control*	0.055±0.004 (3)	115 ± 11 (2) N.S.	17 ± 4 (4)
35% Precipitate			
0.005 mg/ml***	0.091 ± 0.015 (3) N.S.	111 ± 7 (3) N.S.	43 ± 3 (3) <0.001
0.001 mg/ml	0.083 ± 0.027 (3) N.S.	137 ± 23(3) N.S.	45 ± 1 (3) <0.001
70% Precipitate			
0.05 mg/ml	0.180 ± 0.055 (3) <0.05	177 ± 33 (3) N.S.	27 ± 3 (3) <0.02
0.01 mg/ml	0.157 ± 0.022 (3) <0.02	182 ± 24 (3) 0.05	24 ± 0 (1) N.S.
100% Precipitate			
0.05 mg/ml	0.145 ± 0.021 (3) <0.05	180 ± 30 (3) N.S.	57 ± 3 (3) <0.001
0.01 mg/ml	0.132 ± 0.007 (3) <0.05	157 ± 30 (3) N.S.	32 ± 3 (3) <0.001

* Control cultures were treated exactly as experimental cultures but without nerve extract
** n is the number of cultures available for evaluation.
Cultures contaminated by bacteria or fungus were excluded.
Most cultures were in triplicate for each concentration of nerve extract.
*** This is the concentration of nerve extract fractions present in the culture wells.

N.S. = not significant

Legend: Protein synthesis was 3-fold greater with both 70 and 100% fractions compared with control cultures. Myoblasts (MF20 staining cells) were also increased in number with these fractions over control cultures. Chondroblast nodules (Alcian blue staining tissue) were greater in number than control with all extract fractions, but were most increased with the 100% and 35% fractions.

bovine serum albumin was used as the standard. Sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) was carried out at 12.5% acrylamide concentration according to Laemmli[40].

RESULTS

Culture system

Myoblast differentiation was usually visually apparent by Day 2 after culture innoculation and was well established by Day 3. Between Day 3 and Day 5 varying amounts of chondroblast differentiation occurred, depending on specific culture conditions (e.g. the exact age of the embryos and the specific fraction of nerve extract added to the media).

Activity in ammonium sulfate fractions

Both 70% and 100% ammonium sulfate precipitation fractions caused a three fold increase in protein synthesis compared to control cultures (Table I). An increase in cells staining with MF20 was also noted with these fractions, although statistical significance was reached only with the lower concentration of the 70% fraction (Table I). Stimulation of alcian blue staining nodules (cartilage nodules) was observed with the 35% and 100% fractions and to a lesser extent with the 70% fraction (Figure 1). SDS-PAGE acrylamide gel electrophoretic patterns of these three fractions are shown in Figure 2. Note the complex character of these fractions and the presence of the same molecular weight bands in two or all three fractions.

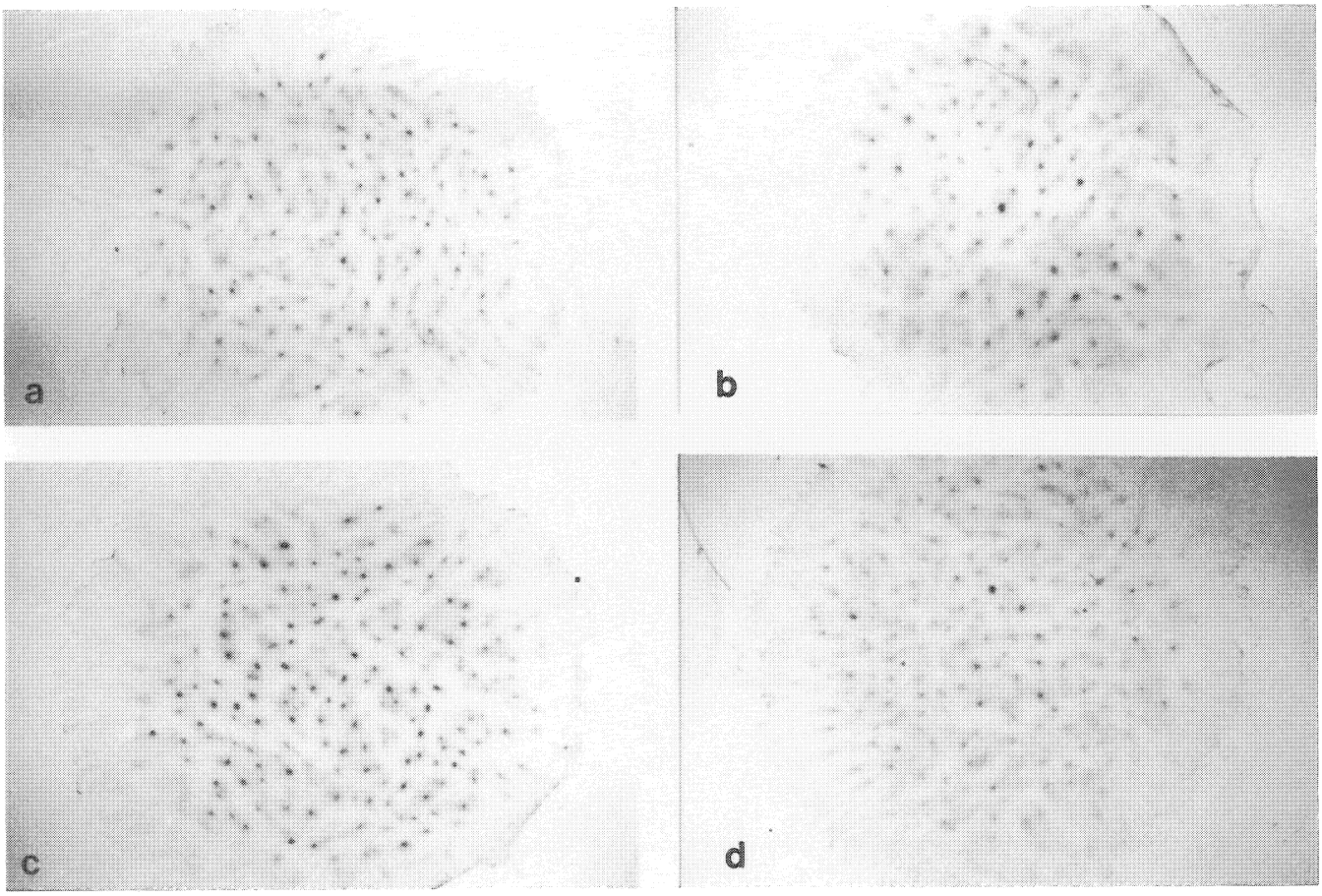

Figure 1. Day 5 tissue cultures in 48 well plates stained with alcian blue (magnification, 50x). The cartilage nodules stain dark black. a,b, and c are cultures treated with 0.005 mg/ml of 35%, 0.05 mg/ml of 70%, and 0.05 mg/ml of 100% ammonium sulfate precipitate fractions of whole nerve extract, respectively. d is a control culture with media alone. An increase in cartilage nodules above the control number is seen in all cultures, but is most pronounced in the presence of 35%(a) and 100%(c) ammonium sulfate precipitate fractions.

Activity in affinity chromatography fractions

We also attempted separation using Concanavalin A affinity chromatography[46]. A major portion of the cell extract protein did not bind to Con A Sepharose, but rather eluted as a broad peak during sample loading and in the early part of the Con A buffer wash that followed. Fractions from this peak were pooled and this crop was designated as the non-glycoprotein fraction. The wash with 50mM D-mannopyranoside yielded a minor protein peak and the corresponding crop was designated as the glycoprotein fraction. These crops were freeze dried. The dried powders were dissolved in water and the solutions were dialyzed against water. The dialyzed solutions were stored at -20 degrees centigrade until further use.

The results of two experiments with the glycoprotein and non-glycoprotein fractions are shown in Table II. The protein and MF20 cell stimulating properties resided in the glycoprotein fraction (Table II and Figure 3). Stimulation of alcian blue nodules was a property of the non-glycoprotein fraction. In fact, the glycoprotein fraction inhibited the formation of cartilage nodules.

Transferrin effects

Transferrin, in a wide range of concentrations, showed no stimulation of myoblasts or chondroblasts (Table III).

DISCUSSION

A trophic effect of peripheral nerve on limb growth has been suggested by several lines of animal research and by human clinical data. Denervation studies in immature animals have shown a decrease in limb growth whether performed before or after limb tissue differentiation[2,17,24,25,26,32,69]. A delay in maturation of denervated limbs has been shown in an amphibian model[17,18]. Limb regeneration experiments in amphibian and avian models have shown limb regeneration to be nerve dependent[19,41,68]. Limb regeneration experiments have shown that a threshold quantity of peripheral nerve is required for regeneration rather than a specific nerve type, e.g. motor or sensory[68]. This suggests a direct trophic effect rather than an indirect effect by muscle activity. Clinical experience with polio reveals a poor correlation between leg length discrepancy and muscle

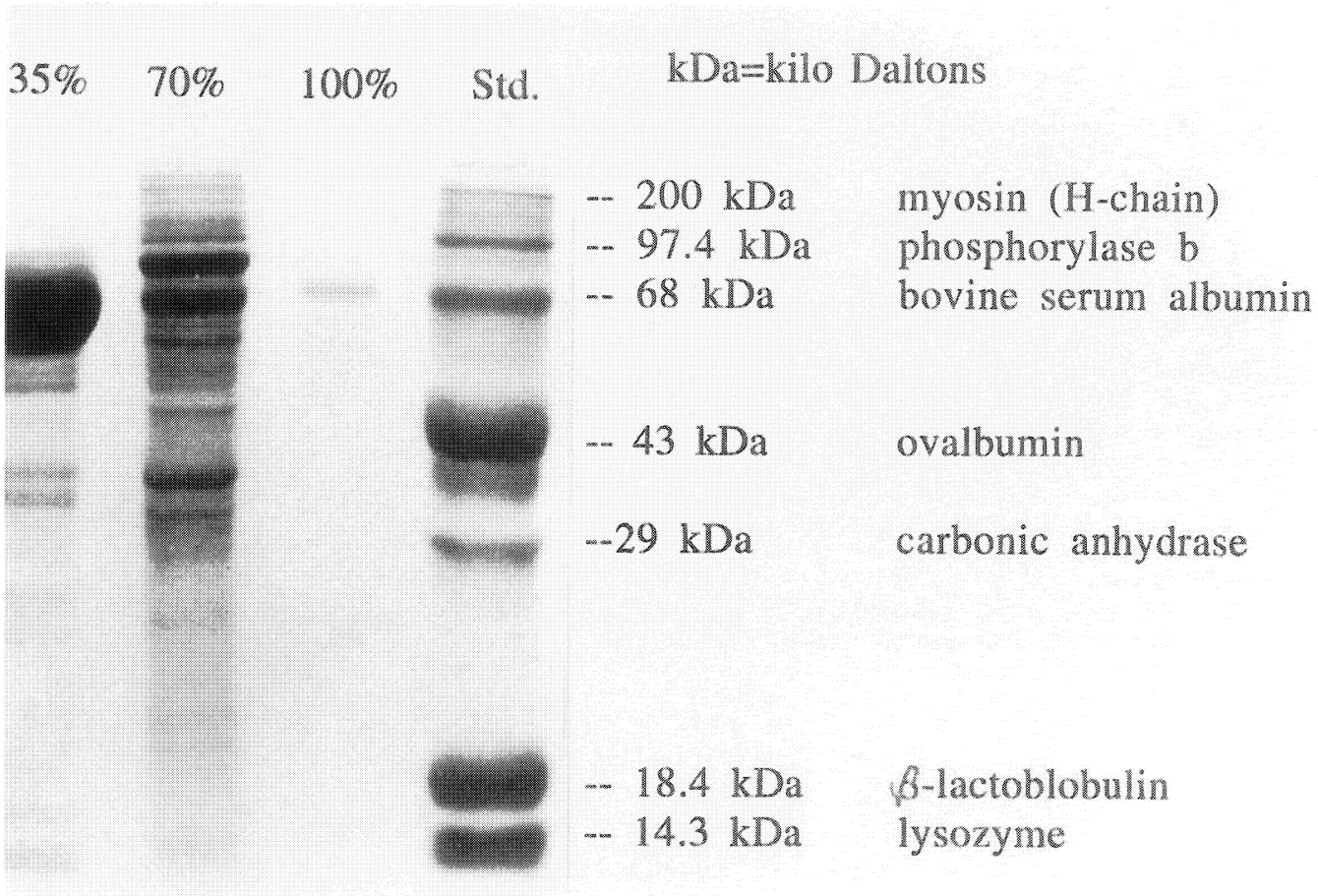

Figure 2. SDS-PAGE patterns of the ammonium sulfate precipitation fractions of whole nerve extract.

strength, again suggesting a direct trophic nerve effect[21,66]. Finally, congenital disruption of the neuroaxis at both upper and lower motor neuron levels is associated with limb length discrepancy in some patients as seen in meningomyelocele and cerebral palsy[5,9].

The mechanism of this nerve-dependent trophic effect on limb growth is unknown and not easily explained by known determinants of limb growth and development[20,24,25,69]. The complex process of limb induction and differentiation has been shown to be nerve-independent in animal models. The endocrine factors that influence limb growth (Vitamin D, thyroxine, androgens, growth hormone, and insulin-like growth factor) are also nerve-independent. Other systemic and autocrine growth factors that stimulate osteoblast growth and replication in vitro have been identified[7,8,49,65]. None are under neural control and whether these factors have a function in limb growth and development is uncertain.

A large body of experimental work has demonstrated a trophic influence of peripheral nerve on muscle development and function unrelated to electrical impulse conduction, acetylcholine release and muscle contraction[22,23]. Several types of experiments have demonstrated this trophic effect of nerve on muscle: 1) Interruption of axoplasmic flow using colchicine or vinblastine results in denervation changes in muscle without disturbing impulse conduction or acetylcholine release[1,31,72]; 2) A stump of nerve left attached to muscle organ cultures prevents changes associated with denervation such as partial membrane depolarization, extrajunctional sensitivity to acetylcholine, increased RNA synthesis, and increased insulin-sensitive amino acid uptake[16,60,67]. A longer attached stump prolongs these effects compared with shorter stumps[16,67]; 3) Spinal cord and peripheral nerve extracts maintained tetrodotoxin sensitivity, membrane action potentials and cholinesterase activity in muscle organ cultures[27,28,39,42]; 4) Crude extracts of brain, spinal cord and peripheral nerve have stimulated single cell myoblast cultures to grow, differentiate, replicate, synthesize proteins and acetylcholine receptors as well as to ameliorate the effects on muscle of denervation *in vivo*[12,13,15,29,34,43,44,45,47,51,52,53,54,55,56,59,61,62,63,64].

Table II

Concanavalin A Chromatography

Concanavalin A Fraction	Protein (mg) X ± S.D. (n)** p value	MF20 Stain (# of positive cells/well) X ± S.D. (n)** p value	Alcian Blue Stain (# of positive nodules/well) X ± S.D. (n)** p value
Control*	0.179 ± 0.071 (2)	6 ± 1 (2)	28 ± 6 (6)
Glycoprotein			
0.01 mg/ml***	0.401 ± 0.029 (3) <0.02	67 ± 3 (3) <0.001	19 ± 5 (3) N.S.
0.005 mg/ml	0.302 ± 0.067 (3) N.S.	60 ±30 (3) N.S.	21 ± 6 (3) N.S.
0.001 mg/ml	0.235 ± 0.054 (3) N.S.	38 ± 13 (3) <0.05	10 ± 2 (3) <0.02 (inhibition)
Non-Glycoprotein			
0.01 mg/ml	0.212 ± 0.03 (3) N.S.	0 N.S.	58 ± 10 (3) <0.02
0.005 mg/ml	0.105 ± 0.05 (3) N.S.	0 N.S.	45 ± 11 (3) N.S.
0.001 mg/ml	0.152 ± 0.131 (3) N.S.	0 N.S.	35 ± 9 (3) N.S.
Repeat Con A Fraction			
Control	0.163 ± 0.067 (3)	66 ± 11 (6)	25 ± 7 (6)
Glycoprotein			
0.01 mg/ml	0.257 ± 0.084 (3) N.S.	387 ± 12 (3) <0.02	6 ± 2 (3) <0.005 (inhibition)
0.005 mg/ml	0.294 ± 0.026 (3) 0.05	444 ± 67 (3) <0.005	4 ± 3 (3) <0.005 (inhibition)
0.0005 mg/ml	0.224 ± 0.092 (3) N.S.	267 ± 60 (3) N.S.	7 ± 7 (3) <0.01 (inhibition)
Non-Glycoprotein			
0.005 mg/ml	0.202 ± 0.021 (3) N.S.	84 ± 40 (3) N.S.	23 ± 6 (3) N.S.
0.0005 mg/ml	0.170 ± 0.065 (3) N.S.	86 ± 34 (3) N.S.	19 ± 2 (3) N.S.

* Control cultures were treated exactly as experimental cultures but without nerve extract

** n is the number of cultures available for evaluation.
Cultures contaminated by bacteria or fungus were excluded.
Most cultures were in triplicate for each concentration of nerve extract.

*** This is the concentration of nerve extract fractions present in the culture wells.

Legend: Protein and myoblast stimulation are evident with the glycoprotein fraction compared with controls. A lesser development of chondroblast nodules (Alcian blue staining) is found with this fraction. The non-glycoprotein fraction resulted in an inconsistent stimulation of chondroblast nodules compared with control cultures. MF20 cells were markedly decreased or absent. This might be due to an inhibition of myoblast cell differentiation or a rapid transition of the micro-mass cultures through the myoblast stage to the chondroblast stage with our harvesting times coming after the myoblast presence had peaked.

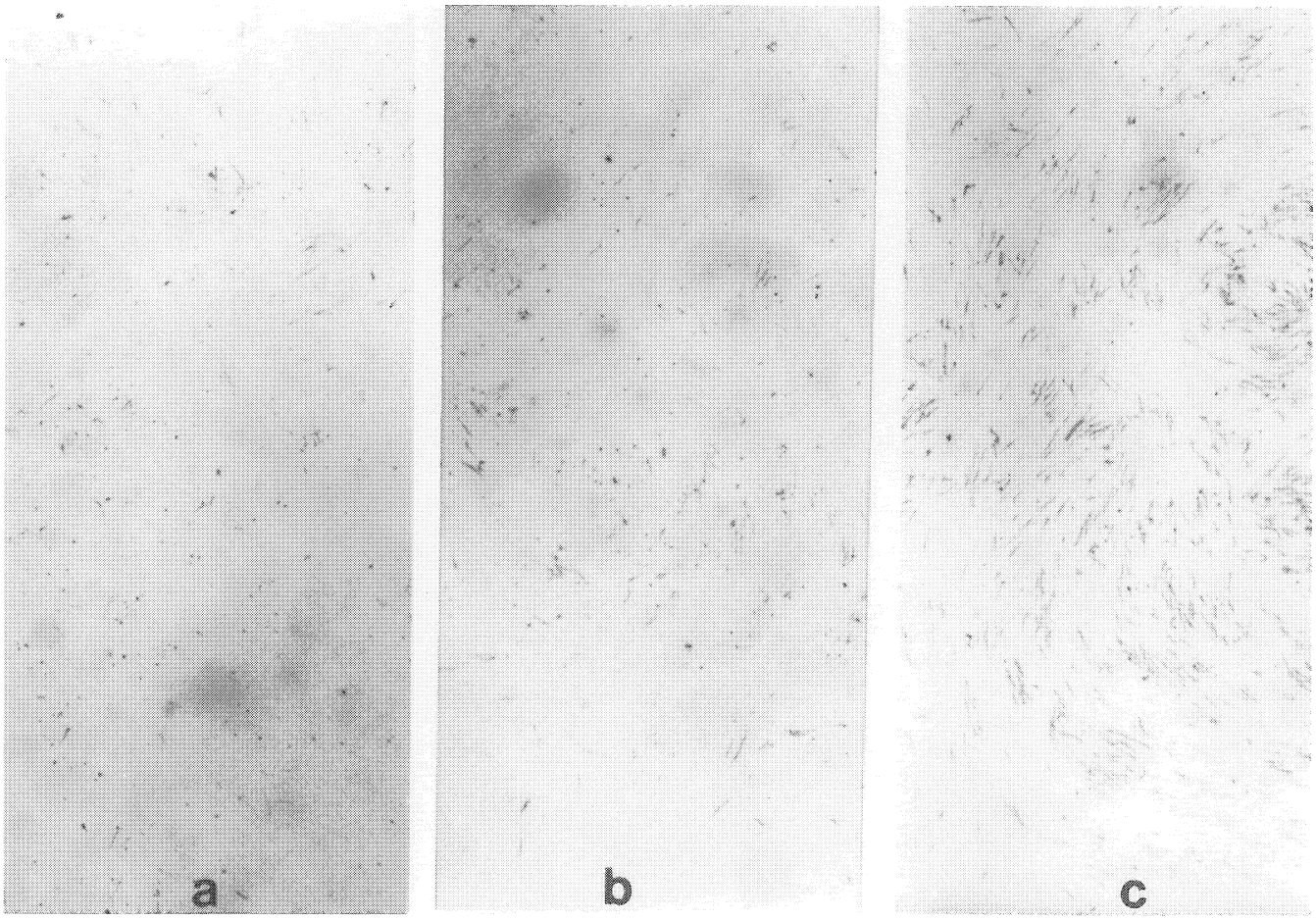

Figure 3. Photomicrographs of Day 3 cultures stained with MF20 to identify myoblasts. a, the control culture in media only; b, treated with 0.005 mg/ml of the non-glycoprotein fraction from Con A chromatography; c, myoblast stimulation in a culture treated with 0.005 mg/ml of the glycoprotein fraction from Con A chromatography.

Investigation into the specific factors responsible for the trophic effect of peripheral nerve on muscle seemed to halt after reports from two groups of investigators that the trophic factor was transferrin, rather than a novel growth factor[4,57]. Both groups reporting this finding employed chicken derived nerve growth factors and chicken muscle cells in their assays. Investigators using a mammalian system (rat) were unable to duplicate the trophic effects of peripheral nerve extracts on muscle with transferrin[14]. Furthermore, the major active protein (named sciatin or neurotransferrin) has been shown to be distinct from transferrin in its three-dimensional structure, although having many structural similarities even in chickens[50]. Finally, a difference between mammals and avians exists in their responses to growth factors. For example, chick motor neuron survival is unaffected by brain-derived neurotrophic factor, neurotrophin-3 and neurotrophin-5; whereas rat motorneuron survival is greatly enhanced by all of these growth factors[30].

Our results support the hypothesis that peripheral nerves supply trophic factors to mesenchymal tissues, unrelated to electrical impulse conduction. We found a stimulation of cell growth as evidenced by increased protein synthesis with the 70% and 100% ammonium sulfate fractions of whole nerve extract. Affinity fractionation of the crude nerve extract revealed that this effect resided in the glycoprotein portion. MF20 (myoblast) staining cells were increased by the presence of both the 70% and 100% ammonium sulfate fractions, although statistical significance was reached only in the 70% fraction. This property was also found in the glycoprotein fraction of nerve extract as resolved by affinity chromatography. Alcian Blue staining nodules (chondroblasts) were increased over controls in all fractions with the most modest effect offered by the 70% ammonium sulfate fraction. The glycoprotein fraction apppeared to inhibit cartilage nodule formation. These results demonstrate that more than one trophic substance for mesenchymal tissues is present in peripheral nerves. Whether a single trophic factor capable of stimulating more than one mesenchymal cell type is present in the 100% ammonium sulfate fraction was not investigated.

Table III

Transferrin	Protein (mg) X ± S.D. (n)** p value	MF20 Stain (# of positive cells/well) X ± S.D. (n)** p value	Alcian Blue Stain (# of positive nodules/well) X ± S.D. (n)** p value
Control*	0.602 ± 0.088 (6) N.S.	113 ± 28 (3) N.S.	160 ± 16 (3)
0.01 mg/ml***	0.583 ± 0.117 (3) N.S.	118 ± 9 (3) N.S.	131 ± 42 (3) N.S.
0.001 mg/ml	0.611 ± 0.033 (3) N.S.	99 ± 26 (3) N.S.	119 ± 15 (3) <0.02 (inhibition)
0.0001 mg/ml	0.513 ± 0.056 (3) N.S.	66 ± 10 (3) N.S.	88 ± 12 (3) <0.005 (inhibition)

* Control cultures were treated exactly as experimental cultures but without nerve extract

** n is the number of cultures available for evaluation.
Cultures contaminated by bacteria or fungus were excluded.
Most cultures were in triplicate for each concentration of nerve extract.

*** This is the concentration of nerve extract fractions present in the culture wells.

Legend: A wide range of transferrin concentrations failed to replicate the effect of nerve extracts in this rat system. Although avian culture systems will respond to transferrin as to nerve extract, [4,43,52] this appears not to be so in our mammalian culture systems.

Our results support the view that transferrin is not the trophic factor supplied by peripheral nerve to mesenchymal tissues. Transferrin had no stimulatory effect on myoblasts or chondroblasts in our culture system over a very wide range of concentrations. Furthermore, we have developed an assay system that allows simultaneous investigation of both myogenesis and chondrogenesis in a mammalian species.

Variation in the quantitative response of our culture system to the same fractions of peripheral nerve in repeated experiments can be explained in several ways. Slightly different maturity of the embryos used can significantly affect the ability of the cells to become myoblasts or chondroblasts under our short term culture conditions. Exposure to proteases in the enzymatic digestion can result in variable attachment and cell death with the slight differences present with new stock solutions or enzyme lots[11]. Also, preparation and purification of peripheral nerve is a laborious task and some variability and possibly degradation of protein components may have occurred.

The ubiquitous presence of nerves in mesenchymal tissues, including cortical bone, suggests the possibility that nerves interact with the mesenchymal tissues they supply to help regulate growth, development, and/or homeostasis after maturity[9,31]. Peripheral nerve is a complex organ and it is not surprising that trophic activity resides in more than one component of this structure. The specific activity found under our *in vitro* conditions does not speak directly to the influence of peripheral nerve on limb growth or mesenchymal tissue homeostasis after maturity. *In vivo,* peripheral nerves transport proteins in both antegrade and retrograde directions by use of cytoplasmic microtubles. Peripheral nerves also are able to concentrate organic molecules such as transferrin in their cytoplasm by uptake from surrounding tissues. These capabilities add to the number of potential candidates for trophic substances. Furthermore, minute quantities of known or novel growth factors might be responsible for trophic influences of nerve on mesenchymal tissues. Such factors would be difficult to identify by standard protein purification techniques starting with whole peripheral nerves.

Many peptide growth factors have been shown to affect mesenchymal cells and cartilage differentiation *in vivo.* For example, limb mesenchyme is stimulated by fibroblast growth factor-4 and inhibited by bone morphogenic protein-2[48]. Both of these peptides are produced in the apical ectodermal ridge. Transforming growth factor stim-

ulates cartilage differentiation from mesenchymal cells; whereas cartilage differentiation is inhibited by basic fibroblast growth factor, fibronectin, integrins, and heparin sulfate[36,38,75]. Our finding of a mitogenic effect with concomitant inhibition of cartilage differentiation in the glycoprotein fraction is noteworthy because glycoproteins have been shown to bind some growth factors and increase or inhibit their biological activity[73,74,75]. For example, the proteoglycan decorin binds transforming growth factor and inhibits its growth promoting effects. Conversely, heparin-like molecules bind basic fibroblast growth factor and increase its receptor binding and activity[73,74].

The purification protocols described here are simplistic, yet they provided preliminary insight into a complex phenomena. Thus, a rigorous program on the purification of candidate factors is a worthwhile undertaking and it could lead to the development of nucleic acid and antibody probes for *in situ* studies of the phenomena. Also, purification of trophic substances from peripheral nerve may enhance our knowledge of the determinants of normal and abnormal somatic growth of limbs. On the other hand, an approach consisting of molecular genetic linkage followed by positional cloning may be a more parsimonious approach to seeking the cause of limb growth disorders with a genetic component, such as idiopathic clubfoot.

ACKNOWLEDGMENTS

This work was funded in part by a grant from the Orthopaedic Research and Education Foundation. We thank Endang Purwantini for her critical comments and help in cell extract fractionation. We greatly regret that Dr. Solursh died before reviewing this manuscript.

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PEARLS AND PITFALLS OF DEFORMITY CORRECTION AND LIMB LENGTHENING VIA MONOLATERAL EXTERNAL FIXATION

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I. Introduction

Over the past eighty odd-years (Figure 1), there have been great advances in the use of external fixation for musculoskeletal problems. Currently, external fixation is widely used for fracture treatment. In addition, these devices are utilized in a highly specialized arm of orthopaedics that has developed for the treatment of limb length discrepancies and angular deformities. These clinical problems are usually complex, the treatment of which depends on a good understanding of the varied etiologies, pertinent biomechanics and the science of fracture healing. The patient benefits when these principles are applied in combination with extensive clinical experience. The purpose of this paper is to condense and combine the applied science of limb lengthening and angular correction with the clinical experience of the senior author (C.T.P.) using monolateral external fixation. Advantages of monolateral fixators include ease of application, decreased risk of neurovascular injury, minimal muscle transfixion, office removal and patient acceptance. Attention will be first directed to principles common to both lengthening and deformity correction; subsequently we will discuss issues peculiar to each problem. It is hoped that this manuscript will enable the reader to avoid some of the varied and frequent pitfalls that accompany treatment of these problems.

II. History

External fixation was first used to stabilize fractures in 1878 by German orthopaedist Jacob von Heine. However, it was the external fixator proposed by Albin Lambotte in 1902 that marks the beginning of this technique in the modern practice of orthopaedics[1]. This and other similar devices were originally used for the stabilization of long bone fractures.

Following the turn of the century, Codivilla carried out the first bone lengthening in 1905[2]. Putti later performed acute long bone lengthenings with traction following corticotomy in 1921[3,4]. He realized that distraction should be gradual to overcome the resistance of surrounding soft tissues. During the 1920 and 1930's, technical advances

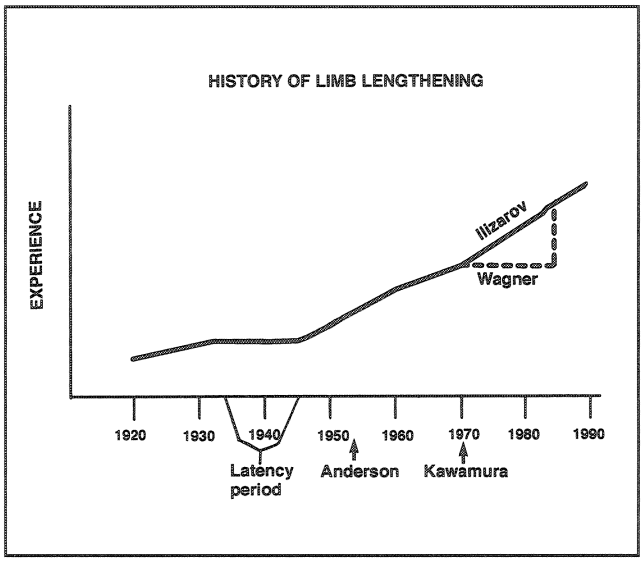

Figure 1 History of Limb Lengthening

A qualitative assessment of the knowledge and practice of limb lengthening throughout history. In the mid-1970's to the mid-1980's surgeons within the United States did not have access to the advances by Ilizarov. During this period, the Wagner method predominated.

were made to improve the mechanics and the rigidity of lengthening devices. These improvements utilized multiple pins (half pins and transfixing pins), multiple bars (single or in multiple planes), universal joints and compression and distraction capabilities. The use and study of external fixation waned between the few years before and after World War II. During this period, attention was directed towards shortening procedures (epiphyseal stapling, epiphysiodesis and bone resection) of the longer leg.

The next major focus on bone lengthening was by Anderson who reported on his experiences of limb lengthening in children with subcutaneous osteotomy and mechanical distraction[5]. Slightly later, Kawamura recommended distraction in three to six stages[6]. In addition to this work, circular ring fixators were being developed and used in Germany by Bier.

The next stage in lengthening in the United States involved the development and use of the Wagner method. Wagner developed a lightweight, versatile unilateral fixator which is strong enough to resist the tremendous forces generated with large lengthenings. Rapid distraction was followed by plating and bone grafting, alternating screw removal and eventual plate removal. Although

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successful, the technique and its multiple procedures were often associated with a high complication rate.

While the Wagner method became the procedure of choice in the United States; Ilizarov in the Soviet Union continued to build upon the work of Bier and developed gradual distraction with ring fixators. Eventually the difficulties of the former technique and the newly discovered successes of callotasis developed by Ilizarov and De Bastiani in Italy led to increased use of the latter principles.

III. Normal Alignment of the Lower Extremities

Lower extremity alignment can be described in terms of mechanical and anatomic axis (Figure 2). In the coronal plane, the mechanical axis is the line of force during bipedal stance that passes through the center of the hip and the center of the ankle. Although slightly variable, the mechanical axis generally passes through or is just medial to the center of the knee[13]. The anatomic and mechanical axes can also be drawn for each bone. It is important to realize that the axes in the femur are not the same; within the tibia the anatomic and mechanical axes are normally co-linear.

The femur and tibia can be individually characterized according to each mechanical and anatomic axis. As mentioned above, this is easily done for the tibia as both axes are the same. The proximal tibial articular surface is usually in 3 degrees of varus with the mechanical and anatomic axes (87 degrees), while the distal articular surface is perpendicular to both axes and parallel to the floor.

The femur is slightly more difficult to analyze. The proximal articular relationship can be described from an index line connecting the center of the hip to the tip of the greater trochanter. The normal femoral mechanical axis (hip to knee center) forms a 90 degree angle with the proximal index line. The femoral anatomic axis (mid-diaphyseal line) intersects the proximal index line at 81 to 85 degrees. The neck-shaft angle can also be used to characterize the proximal femur (130+/- 10 degrees). The anatomic and mechanical axes normally converge at the knee center at 5 to 9 degrees. The distal femoral articular surface is usually in 3 degrees of valgus (87 degrees) to the mechanical axis. Subsequently, the angle of the distal femoral articular surface and the anatomic axis is 78 to 82 degrees (Figure 2). As the previous values are averages derived from several large screening studies, their use should be limited to cases of bilateral deformity. Whenever possible, corresponding values for these angles should be taken and used for comparison from radiographs of the unaffected side.

A good understanding of these principles is paramount for deformity and length discrepancy correction. In general, realignment is performed to maintain a neutral mechanical axis - colinearity of the hip, knee and ankle centers. During routine callotasis, it is ideal to lengthen along the mechanical axis to avoid translation. The mechanical axis in the sagittal plane can also be characterized, however, this is rarely of much clinical importance as long as the normal anatomic alignment is restored.

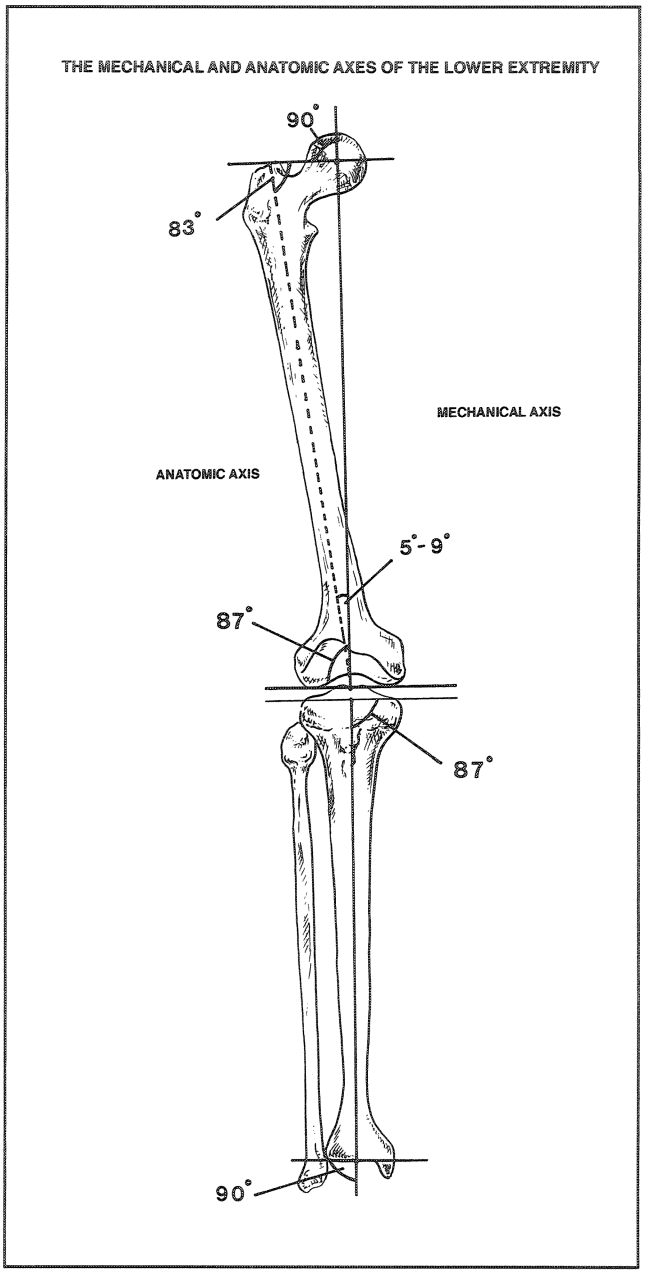

Figure 2 The Mechanical and Anatomic Axes of the Lower Extremity

The normal mechanical axis passes through the hip, knee and ankle centers. Deviation of the axis out of the middle of the knee is termed mechanical axis deviation. The proximal and distal articular surfaces form uniform angles with the mechanical axis of each bone. The anatomic axis of the tibia and femur intersect with valgus alignment of 5 to 9 degrees. Note that as opposed to the tibia, the femur mechanical axis and anatomic axis are not the same.

Finally, the orientation of the joint surface should be appreciated. In general, the knee and ankle joints should be parallel with the ground during gait to prevent excess shear force. Ideally both knee levels should be within two centimeters of each other during stance.

IV. Fixator Biomechanics

Although ideal fixator mechanics have not been described for each clinical situation, the optimal fixator should allow resistance to cantilever bending and torsion while allowing controlled axial loading. In both angular correction and limb lengthening, fixators that can be dynamized are used to increase callus maturation. Previous work has clearly shown that axial movement of tibial fractures treated with external fixation stimulates increased healing and fracture stiffness[22]. Recent work has demonstrated that early dynamization, or unlocking the fixator, causes progressive fracture gap closure and a subsequent increase in cyclic motion important for healing[23]. On the basis of this work, we currently unlock or dynamize the fixator and osteotomy site at six weeks.

Several mechanical principles must be kept in mind when using monolateral external fixation for deformity correction and limb lengthening. Importantly, the structural rigidity can be affected by its application. Increased pin diameter, placing the fixator closer to the bone, keeping the pin clusters close to the osteotomy site, spreading the distance between the pins in each fragment, and using more pins increases the structural stiffness[15]. As extremely rigid fixation may retard bone healing, it is desirable to use a relatively flexible fixator construct for fractures and osteotomies for angular correction. For limb lengthening procedures, a more rigid construct is necessary to avoid angulation during distraction. Once length has been achieved, callus maturation may be stimulated by removing one or two pins, moving the fixator away from the bone or dynamization of the device.

During limb lengthening, T-clamps should be avoided as they are substantially more flexible than pin placement in clusters parallel to the axis of lengthening. Three pins are placed in each pin cluster to increase stiffness. If a pin becomes infected it can be removed without completely destabilizing the construct, a common problem given the long duration needed for callus maturation. Fixators without ball joints are preferred for significant lengthenings as tensile forces will commonly lead to failure of these joints. Ball joint devices are occasionally used for angular corrections combined with shorter lengthenings of two to three centimeters. When used in this manner, movable joints should be cemented with polymethylmethacrylate. It is essential to check and tighten the ball joints every one to two weeks due to loosening from repetitive loads.

During deformity correction two pins are used in a linear construct proximally and distally. Fixators with ball joints are commonly used and cemented in larger patients or in angular corrections of the femur. Preliminary trials of fixators with racheted swivel clamps may obviate the use of the weaker ball joint.

V. Assessment

It is impossible to outline the pertinent issues for each cause of limb deformity; however, a detailed history is clearly indicated in these complicated cases. During the physical exam, the surgeon must clinically document functional (umbilicus to medial malleolus) and absolute (anterior superior illiac spine to medial malleolus) length discrepancies, hip-knee-ankle alignment, degrees of pro- or recurvatum and long bone malrotation. A relatively high rate of neurovascular complications during treatment necessitates a good preoperative evaluation. Joint range of motion and instability must be assessed as abnormalities may worsen, or fail to accommodate to changes in length or alignment. Preoperative joint contractures may require concomitant manipulation or release.

Several radiographic tools are needed. Scanograms, orthoroentgenograms and CT scans have been used with great accuracy to determine the degree of length discrepancy. Standing alignment radiographs are taken with the patella forward to include the hip, knee and ankle. Specialized tests such as tomograms, CT or MRI may be useful to study the "health" of the growing cartilage. Standard left PA wrist and hand films are obtained and compared with the Greulich-Pyle[9] atlas to obtain the skeletal age. In patients with a physiologic age that is less then 10 years, the above atlas may not be as reliable in quantifying bone age as in older patients. Greater accuracy is obtained by examining the phalangeal physeal pattern[25].

In immature patients, it is beneficial to assess maturity and growth potential. The Mosley straightline graph[10], derived from data collected at Boston Children's Hospital[11], has become a cornerstone in preoperative planning. This graph allows the surgeon to determine the final length at maturity. The graph is also designed to plan the appropriate point for epiphysiodesis in smaller discrepancies. In acquired or progressive limb length inequalities this technique requires several data points. At our center we have also used the Mosley graph with just one data point for cases of congenital shortening. Knowing that at time zero both extremities were at equal length, a line can be drawn from this point to the sole data point. This provides a rough estimate of length difference in nonprogressive deformities at maturity.

The White method is also useful for a quick assessment of growth remaining[12]. The distal femoral growth plate can predictably increase length by 3/8 of an inch (10 millimeters) per year until maturity. Similarly, the proximal tibia growth plate can be expected to contribute 1/4 of an inch (6 millimeters) per year until maturity. This allows the

physician to make a quick assessment of future discrepancy as these physes close in boys and girls at 16 and 14 years of age respectively.

VI. Osteotomy Techniques

The location of the osteotomy depends on several variables: fixator placement, level of deformity and adjacent soft tissue integrity. During distraction callotasis, the osteotomy is performed in metaphyseal bone whenever possible. At this level the regenerate is wider, and therefore more resistant to torsional and bending forces after device removal. As pin placement may limit this, the osteotomy is often placed as close as one centimeter from the nearest pin. Care is taken to avoid communicating the pin tracts and the osteotomy site. During angular correction it is optimal to perform the osteotomy at the same level as the deformity. This can be somewhat difficult and is outlined below.

Although many types of osteotomies have been described,[16] we generally utilize a transverse corticotomy through a very limited incision after fixator placement. Attention is directed to preserve the periosteum and to protect, as much as possible, the intramedullary blood supply of each fragment. For both femoral and tibial lengthenings the approach is anterior; in the femur we gently split the fibers of the rectus femoris. The periosteum is incised and elevated over the osteotomy site, the cortex is predrilled and an osteotome is used to cut the bone in the manner of De Bastiani. The key is to perform the osteotomy in a gentle low-energy manner to avoid an exaggerated inflammatory response. Once the osteotomy is complete and appropriate alignment insured, the site can be slightly compressed during the waiting period of distraction callotasis. This reduces the length and intensity of the inflammatory stage of fracture healing.

In tibial deformity correction, one centimeter of fibular diaphysis is removed; if lengthening is planned, the fibular resection is at least five centimeters proximal to the ankle. In addition, the anterior tibial compartment is released to prevent postoperative compartment syndrome[26]. The risk of this complication rises in cases of traumatic osteotomy or in cases of opening wedge osteotomies accompanied with large immediate angular correction. Although we do not uniformly release the fascia of the thigh, compartment syndromes have also been observed following osteotomy associated with postoperative epidural analgesia. It is our opinion that epidural analgesia increases the risk of compartment syndrome due to concomitant vasodilation. In addition, this technique may also mask the early clinical signs.

VII. Routine Pin Placement and Care

The key to patient tolerance and acceptance of external fixation begins with pin placement and care. Pins are placed without soft tissue tension, transfixing the least amount of muscle possible; guides are utilized to avoid skin and muscle necrosis. Pins in each clamp should be placed parallel to avoid pin torque and bone necrosis. Bone necrosis is minimized with sharp drills and the use of tapered conical pins. Experimental testing has demonstrated that these pins generate less heat and theoretically less necrosis during pin insertion[24]. In addition, conical pins are easy to remove in the office and seldom require any anesthetic or sedation.

Psychological and physical acceptance of the fixator begins in the operating room. After the wounds have been closed with absorbable suture, nonadherent dressings are applied to the pins and any osteotomy wounds. Sterile gauze is loosely applied to the pins so that the first dressing change is relatively atraumatic. The dressing is applied in such a way that the fixator can be seen by the patient and family in the immediate post-operative period. We believe this to be important in acceptance of the device.

Parenteral antibiotics (first generation cephalosporin) are given preoperatively and postoperatively for one or two days, followed by ten days of oral antibiotics. This may prevent significant pin tract infection prior to pin tract epithelialization and colonization with normal skin flora. In the acute postoperative period pin sites are cleaned daily with hydrogen peroxide; later pin sites are cleaned with soap and water. Patients are encouraged to shower and swim in chlorinated pools without covering the device.

CORRECTION OF ANGULAR DEFORMITY

I. Indications and Contraindications for Angular Correction

A variety of pathological conditions can lead to angulatory deformity that is mechanically and cosmetically unacceptable. Angulatory deformity may arise from metabolic conditions (nutritional and vitamin D resistant rickets, renal osteodystrophy), developmental conditions (tibia vara, hemihypertrophy, fibular hemimelia), growth arrest or stimulation (infection, proximal tibia fracture, physeal fractures), congenital dysplasia (metaphyseal chondrodysplasia, multiple epiphyseal dysplasia, dysplasia epiphyseal hemimelica, dwarfism, tibial pseudarthrosis) and neoplasia (fibrous dysplasia, osteochondromatosis, Paget's disease). The reader should recognize that a good proportion of angular deformities may be multiplanar with a malrotation component to the deformity. Very few absolute contraindications to angular correction exist. However, caution should be used in those patients with metabolic deformity

that are not medically well managed, as well as in younger patients with a high chance of recurrence.

Opening wedge osteotomies are generally preferred in children when the deformity is 25 degrees or less; however, the surgeon should be cognizant of neurovascular structures that may be compromised following realignment. In a valgus flexion deformity of the proximal tibia, correction with an opening wedge osteotomy may lead to a peroneal nerve palsy. In these cases we prefer a closing wedge osteotomy and a short lengthening to accommodate any resultant discrepancy.

II. Preoperative Planning

Accurate and thoughtful preoperative planning is the most important aspect of deformity correction[14]. Using long leg standing radiographs, the surgeon assesses coronal anatomic deformity as well as the mechanical axis and any deviation. It is rare for a deformity to be fully characterized on AP radiographs since all deformities have three dimensional components. For example, if angulation is 20 and 10 degrees on AP and lateral radiographs respectively, the actual magnitude is greater than 20 degrees and in a plane between the coronal and sagittal planes. This magnitude and direction can be calculated with trigonometric principles.

In cases such as fracture malalignment, the mechanical axis is disrupted and an *uncompensated* deformity is present. The primary deformity may be obvious, easily recognized and treated. However, a subtle deformity may coexist, and if present, it should also be detected and appropriately planned for. These smaller deformities may significantly alter the mechanical axis once the primary deformity is corrected. In cases with an intact mechanical axis and obvious anatomic deformity, it is very important to realize that *compensating* deformities are present. This is well demonstrated in metabolic conditions where the entire bone may be bowed. In other cases, a diaphyseal angulation may be accommodated by a compensating juxtaarticular deformity. When surgery is indicated in the later case, both deformities need to be corrected. Finally, it is not uncommon to have deformities in both the tibia and femur.

The first step in preoperative planning is assessing the entire lower extremity mechanical axis. If intact with obvious anatomic deformity (*compensating*) or if deviated out of the knee axis (*uncompensated*), the next step is identifying which bone is deformed and where. This is done by drawing the mechanical and anatomic alignment of each individual bone. A radiograph of the unaffected contralateral limb is used to obtain normal angulation for the differing axis. In some cases, significant intraarticular deformity (tibia vara) hinders accurate drawing of the joint surface. In this instance, one can extend the normal mechanical axis from the adjacent bone.

It is easiest to start with the tibia as both axes are co-linear. The proximal and distal axes are drawn, and if these values deviate greater than five degrees from the contralateral side (if normal) or from standard values (as described previously), deformity is present. Tibial malalignment is located by constructing normal axis from the

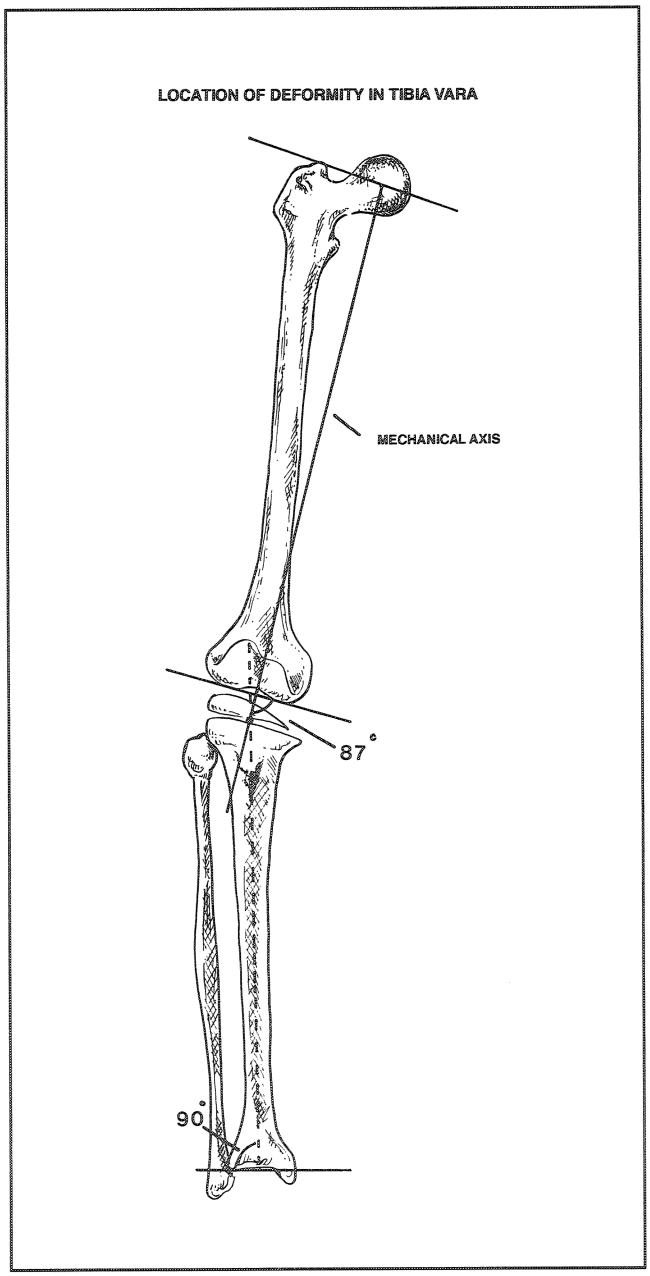

Figure 3 Location of Deformity in Tibia Vara

The deformity site is located by intersecting the normal distal axis (perpendicular drawn from the ankle articular surface) with the proximal mechanical axis, drawn by extending the femoral mechanical axis distally. The location of the deformity is at the intersection of these axes.

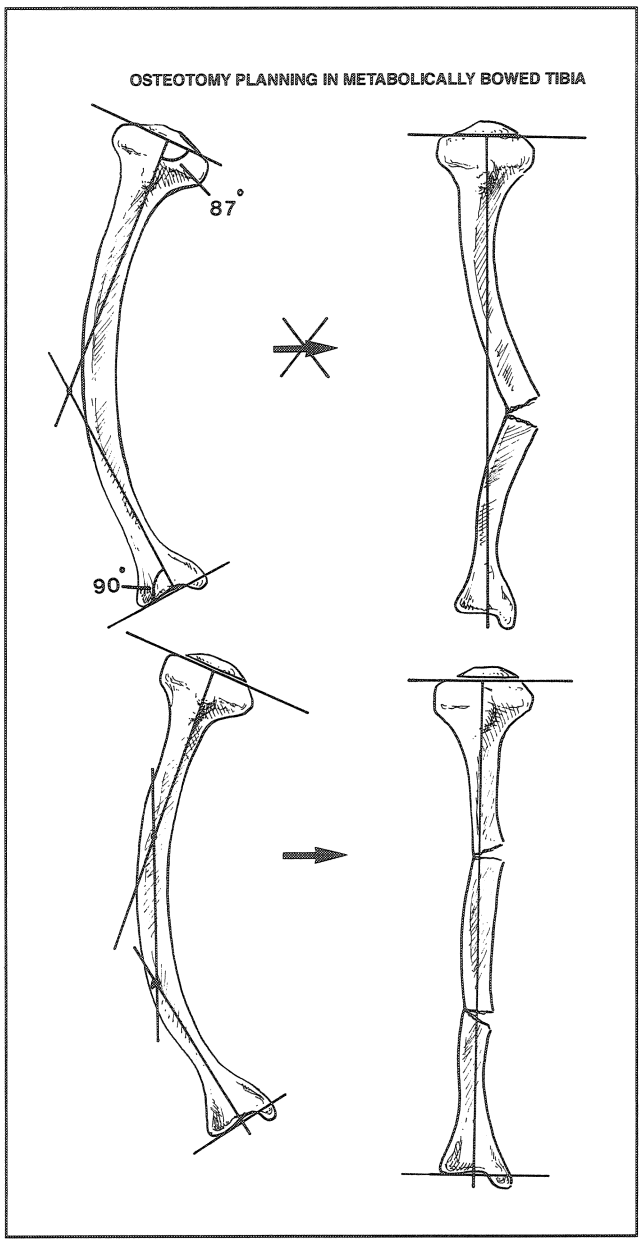

Figure 4 Osteotomy Planning in Metabolically Bowed Tibia

The intersection of the proximal and distal axes is not over bone. Osteotomy at this level could conceivably correct the mechanical axis with significant residual anatomic deformity. A bilevel osteotomy is planned by constructing a third mechanical/anatomic axis in the mid-diaphysis. The intersection of this line with the previously constructed axes locates two sites of deformity. Osteotomy at these levels results in restoration of the mechanical axis with acceptable anatomic deformity. This same technique can be used in bilevel deformity where the intervening segment is normal.

proximal and distal joints. The deformity is centered at the intersection of these two axes. In cases such as uncompensating tibia vara, the intersection of the distal shaft and a 3 degree varus line from the knee joint surface will locate the deformity, usually at the physis (Figure 3). If the intersection of constructed axis is not over bone or at the site of obvious deformity, the apex of angulation cannot be located to one region such as in metabolic bowing or bilevel deformity (Figure 4). Osteotomy at this level may correct the mechanical axis, however significant anatomic variability may result.

Two approaches can be used for locating and planning deformity correction in the femur. In one option, the

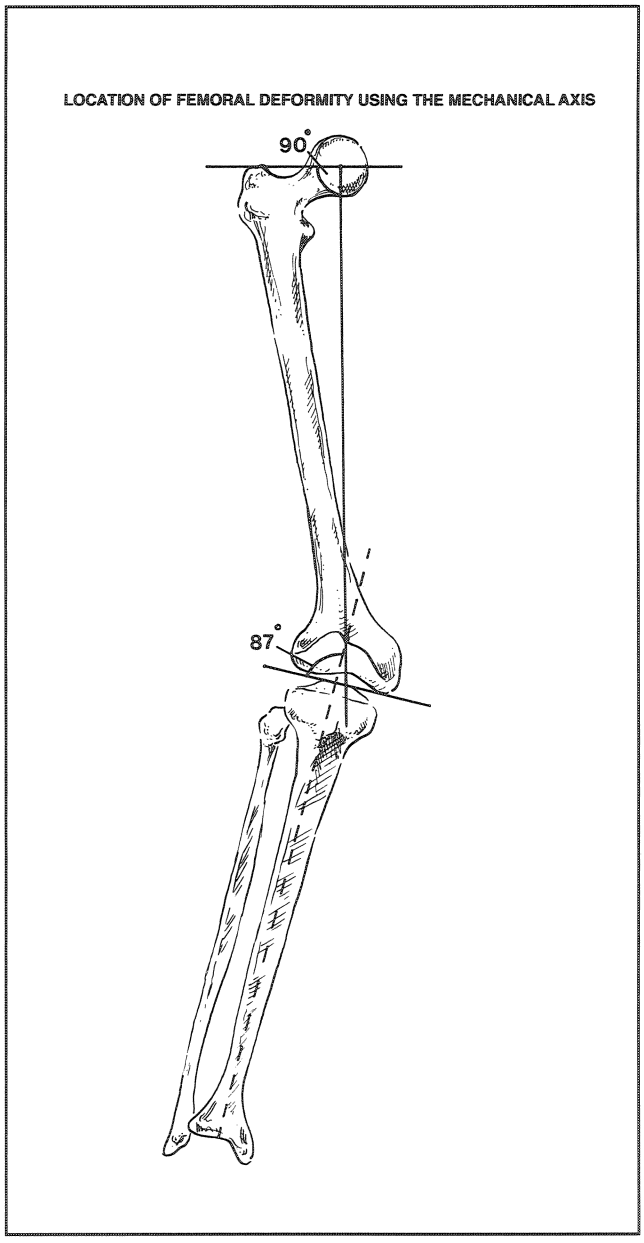

Figure 5 Location of Femoral Deformity using the Mechanical Axis

In this case of distal femoral valgus deformity following physeal injury, the site of deformity is located by constructing normal proximal and distal mechanical axis. The proximal mechanical axis is a perpendicular from the femoral index line connecting the center of the head and the tip of the greater trochanter. The distal mechanical axis can be drawn as an extension of the tibial mechanical axis, or an 87 degree angle from the distal articular surface.

normal mechanical axis of the distal and proximal femur is constructed, and the intersection of these lines locates the deformity and possible site of corrective osteotomy. This technique works best when the suspected deformity is in the proximal or distal metaphysis (Figure 5). The second technique requires construction of the normal proximal

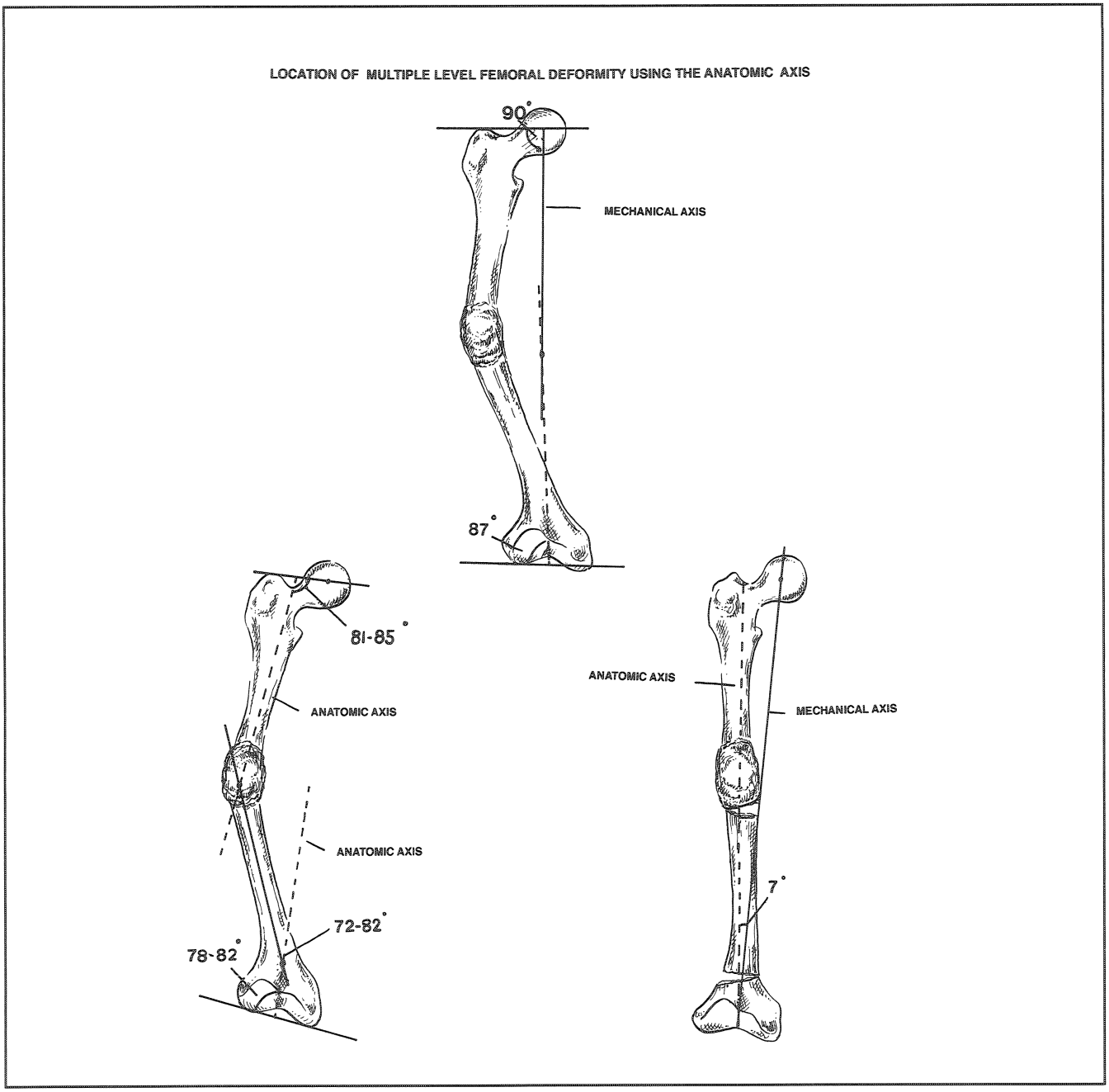

Figure 6 Location of Multiple Level Femoral Deformity using the Anatomic Axis

In this case of a malunited femoral shaft fracture, it appears that all of the deformity is present in the diaphysis. However, construction of the proximal and distal femoral mechanical axes demonstrates that the intersection is away from the obvious anatomic deformity. Two angulations must therefore exist; obvious diaphyseal deformity and subtle distal femoral valgus deformity. To determine both levels of osteotomy it would be necessary to draw the mechanical axis of the intervening segment—a difficult task. Planning multiple level correction is best handled with anatomic assessment. To plan the appropriate osteotomy levels, the anatomic axes of the proximal, distal and intervening segment are drawn. The proximal anatomic axis is reproduced from the contralateral side. If the opposite side is affected then a line can be drawn from the proximal femoral index line at 81 to 85 degrees. The intervening segment is drawn in the mid-diaphysis. The distal segment is drawn from values of the contralateral femur. If this is not normal, a 78 to 82 degree angle with the distal surface should approximate normal anatomic alignment. Osteotomies at these two sites should be followed by realignment of all three segments. Mechanical axis correction can then be assessed in the standard manner.

and distal anatomic axes. Intersection of these lines also locates the site of angulation. This method is appropriate in diaphyseal deformity or in bowed or multilevel deformity (Figure 6).

Ideally the corrective osteotomy should be made at the level of the anatomic deformity. We try to avoid placing the osteotomy in heavily scarred soft tissue or recent bone injury. If the osteotomy is not at the level of deformity the surgeon must shift the distal fragment to avoid production of a translational deformity.

Finally, in metabolic cases where the entire bone is bowed, the osteotomy can be made at any level. The major decision in these cases is how many osteotomies to make. In relatively minor bowing or short bones, only one level is required. In cases with larger deformity or longer bones, two or even three osteotomies may be needed. It is relatively easy to use paper cutouts to determine the appropriate number and still allow for fixator pin placement.

The bulk of planning is directed to the correction in the coronal plane, yet the surgeon must be cognizant of any sagittal or rotational abnormalities. Sagittal anatomic alignment can be easily corrected at the time of surgery with corrective angulation of the fragments. Rotational deformity is also easily corrected through the osteotomy site (See below.)

III. Technique of Deformity Correction

Fixator placement—The patient is placed supine on a radiolucent operating room table with both lower extremities prepped and draped. The hip joint must be accessible for radiographic alignment.

External fixator pins are placed under fluoroscopic control; if several key principles are observed, a normal mechanical axis can be expected after osteotomy and placement of the fixator body. When a fixator with ball joints and/or multiple ranges of motion is used, a certain degree of inaccuracy in pin placement can be tolerated without disrupting the eventual mechanical axis. Use of the following principles will allow accurate realignment with any type of monolateral fixator.

Under fluoroscopic guidance the limb is rotated until a true AP view of the proximal joint is obtained. By placing the joint in neutral rotation radiographically, the horizontal plane of the floor can be used as a reference for rotational alignment during pin placement. The most proximal pin of the proximal grouping is placed in the midline of the bone perpendicular to the mechanical axis (in the coronal plane) and parallel with the floor. After checking initial placement, the remaining pins are placed parallel to the first pin in the proximal clamp. It is critical that all pins within each cluster are placed in the midline of the bone in the sagittal plane. If the most proximal pin in the cluster is placed posterior and the most distal pin is placed anterior, posterior angulation of the osteotomy will result after fixator placement.

The limb is similarly rotated until a true AP view of the distal joint is obtained. The most distal pin of the distal grouping is placed in the midline of the bone in the sagittal plane, perpendicular to the mechanical axis in the coronal plane and parallel with the floor. The remaining distal pins are placed as above. The surgeon will note rotational deformity when the proximal and distal pins are not in the same plane. After osteotomy, the pins can be aligned to correct the rotational deformity. Additionally, any significant anterior or posterior angulation will be reduced if the realigned pins were placed in the midline of the bone in the sagittal plane.

Osteotomy and realignment—After osteotomy the fixator is applied. All pins should be parallel in the coronal, sagittal and transverse planes which should automatically realign the mechanical axis (Figure 7). If the pins were not perfectly placed, small corrections can be made when using fixators with ball joints or other degrees of freedom. If the osteotomy is not at the same level as the initial deformity, translation is performed to reduce any significant anatomic malalignment.

With the leg positioned so that the patella points forward; a bovie cord is stretched from the hip to ankle center under fluoroscopic guidance. An AP view is obtained of the hip, knee and ankle. If the mechanical axis has been restored the cord will be visualized in the center of the knee. If any question remains, a 36 inch film cassette is placed under the leg and a radiograph is taken to assess the mechanical axis. The final alignment is insured with a standing long leg radiograph at the first postoperative visit. If undetected residual deformity remains, the fixator is manipulated under sedation.

LIMB LENGTHENING VIA DISTRACTION CALLOTASIS

I. Indications, Goals and Contraindications for Limb Lengthening

In general, most authors recommend lower limb lengthening in those conditions that result in a final discrepancy of five centimeters or more. Limb discrepancies may result from congenital conditions (hemihypertrophy, femoral and tibial dysplasias, posterior medial bowing or congenital pseudarthrosis of the tibia), neoplasia (Ollier's disease, multiple hereditary exostosis, neurofibromatosis), growth arrest or stimulation (trauma, infection), acquired deformity (joint dislocation or contracture, fracture malunion, supra-pelvic obliquity), neuropathic conditions (poliomyelitis, cerebral palsy, myelodysplasia and sacral agenesis) and a variety of miscellaneous conditions (dwarfism, radiation therapy, slipped capital femoral epiphysis and Perthes disease).

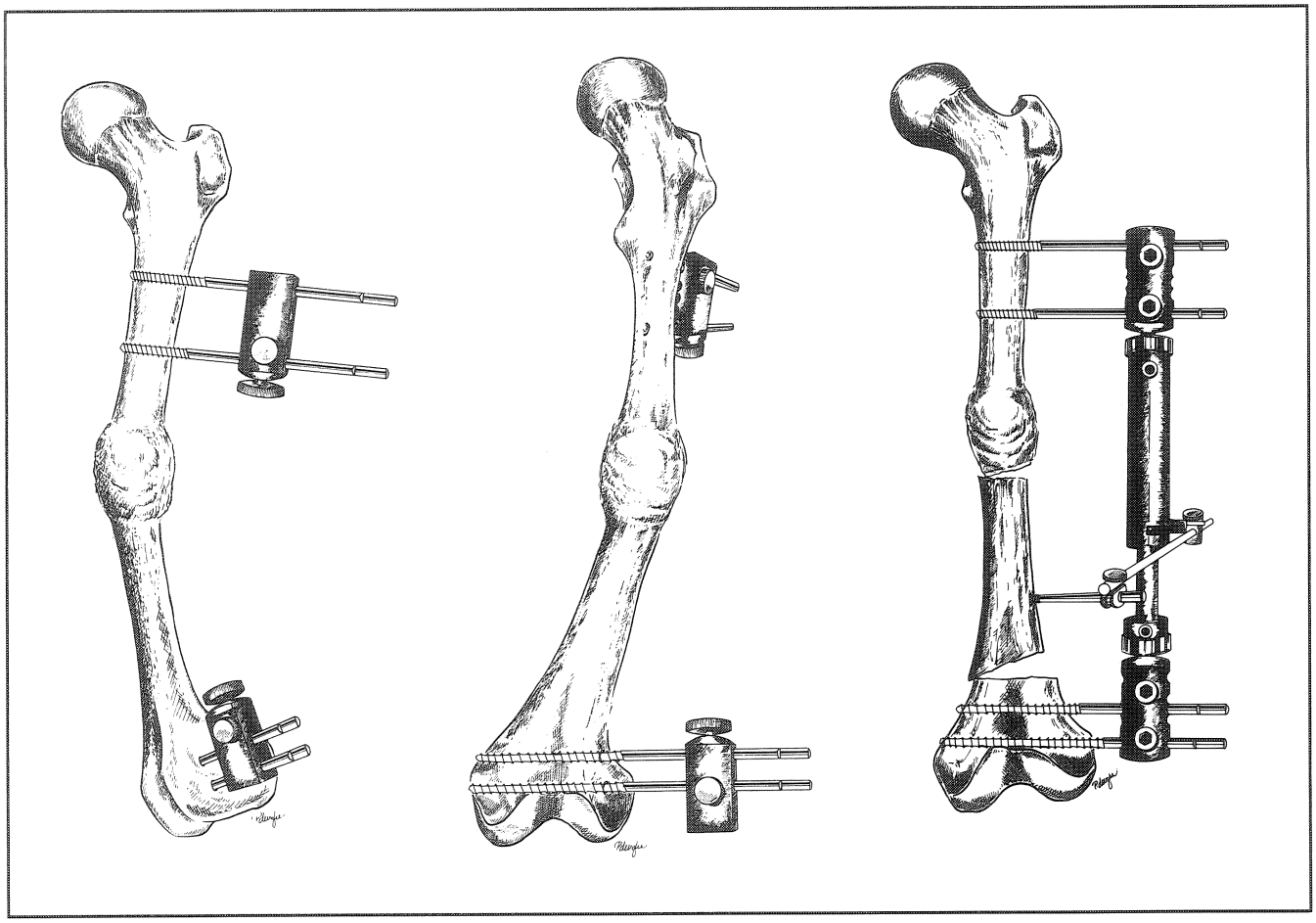

Figure 7 Axes Realignment after Osteotomies

In this case of multiple level femoral deformity, all three planes are corrected after accurate pin placement and osteotomy at the appropriate level. Both pin clusters are placed perpendicular to the mechanical axis in a true AP view of the proximal and distal bone ends. After osteotomy and parallel realignment of the pins, rotational and coronal alignment is restored. Sagittal alignment is insured by placing the pins in the middle of the bone in the sagittal plane.

The actual procedure of corticotomy and fixator placement is the easiest part of limb lengthening. Difficulty arises in the avoidance and management of problems during distraction. Both the physician and the patient must recognize what are reasonable goals for limb lengthening. The rate of problems begins to increase in lengthenings greater than 15 per cent of the original bone length. When lengthenings of greater than 20 per cent are required, staged lengthenings are recommended. If all is proceeding well, 20 per cent or more may be obtainable; however, patients should be advised to expect two procedures. Amputation and prosthetic fitting is usually recommended for congenital discrepancy greater than 15 centimeters. Usually one can expect easier lengthenings of femurs as opposed to tibias, and a harder treatment course in patients with congenital versus acquired limb length discrepancies.

Limb lengthening is relatively contraindicated in those patients who have significant sensory or motor deficits, unstable or painful joints at either end of the proposed bone, psychological problems or those patients whose discrepancy is better managed with amputation and prosthetic fitting. Ipsilateral tibia and femur lengthenings should be avoided due to higher incidences of knee complications. Bi-level lengthenings have been additionally abandoned as the surrounding muscle and neurovascular structures may be adversely affected by the rapid increase in length (2 millimeters per day).

II. Technique and Sequence of General Limb Lengthening

The techniques of limb lengthening are derived from the work of De Bastiani[7,8], and can be divided into subsequent stages.

Fixator Application—We currently use a monolateral slide lengthener applied to the lateral aspect of the femur or the medial side of the tibia. Under fluoroscopic control the proximal pin in the proximal clamp is placed in the

midline of the bone perpendicular to the mechanical axis of the bone to be lengthened. If rotational or angular correction is not planned, the lengthener is clamped to the proximal pin and used as a guide for the distal pin in the distal cluster. This can be quite helpful in assisting parallel pin placement; however, it is paramount to avoid the tendency to abduct or adduct the fixator as it directs distal pin placement. Deviation of more than a few degrees will not hinder fixator placement; however, after osteotomy the proximal and distal fragments will automatically angulate to the degree of pin malposition. Although initially inconsequential, this deviation combined with significant lengthening will lead to mechanical axis deviation. Once the initial proximal and distal pins are placed, the remaining two proximal and distal pins are placed for a total of six screws. After the fixator is positioned, the osteotomy is performed in the metaphyseal region if possible.

If angular or rotational correction is planned, the proximal and distal pin clusters are individually placed perpendicular to the mechanical axis in appropriate rotation and sagittal alignment (see Fixator Application in Deformity Correction). With femoral lengthenings, the knee is manipulated after fixator placement from full flexion to extension. This bluntly dissects the iliotibial band and lateral musculature around the pin clusters preventing early onset of joint contractures during lengthening. In addition, it is ideal to place pins as far away from joints as possible. These pins are usually more comfortable, less prone to loosen or lead to septic arthritis.

Lengthening—Distraction callotasis can be divided into several important treatment periods[18]. The "waiting period" from osteotomy to lengthening corresponds with the inflammatory stage of fracture healing. Although this duration is not exactly known, it is derived from clinical experience and varies between seven and fifteen days. In general, the waiting period is shorter in immature patients. This interval is increased in older patients, and in cases where the osteotomy is in the diaphysis or in previously damaged bone. In addition, this period is increased if lengthening is accompanied by acute angular correction.

Distraction is then begun at the rate of one millimeter per day in four 0.25 millimeter increments. Evidence suggests that distraction of 0.5 millimeters per day leads to premature consolidation, and distraction of greater than 1.5 millimeters per day exceeds the rate of osteogenesis[19,20]. Quarterly turns have proven optimal for bone formation. Gradual motorized distraction has not been shown to have any increased benefit. The treating surgeon explains the lengthening process to the patient and family with a spare fixator and wrench. The family is shown how the fixator slide is loosened, lengthened 0.25 millimeters and then re-locked. To avoid confusion, a directional arrow is painted on the fixator body with fingernail polish. As callotasis depends on patient education and compliance, a radiograph of the osteotomy site is obtained after 7 to 10 days to confirm lengthening at the osteotomy. The patient is examined and radiographs of the osteotomy gap are taken at regular intervals, characteristic callus formation is usually detected at four to five weeks. The rate of distraction can then be adjusted to compensate for abnormal bone production. In those cases where very little callus is being formed, the distraction can be slowed, halted or even reversed. When an overabundance of callus is present, the rate of distraction can be increased to five turns per day.

When the desired length has been achieved the fixator is locked and the callus undergoes maturation and ossification. During distraction bone forms via intramembranous ossification. This regenerate must consolidate and corticalize prior to fixator removal. This process is accelerated by increasing activity, full weight bearing and dynamization of the fixator. The fixator is dynamized by releasing the locking screw when radiographs show a smooth cortical surface. A dynamization collar prevents full collapse when there is any question about callus stability.

The fixator is removed when radiographs show good bone consolidation and well formed cortices on orthogonal radiographs. The pins are left in for a few days after the fixator body is removed. Should a fracture occur, the fixator can be reapplied without anesthesia. If no fracture occurs, patients can usually unscrew and remove tapered pins themselves without sedation.

In general, the total duration of external fixation is about five weeks for each centimeter of length gained, or about three to four more months after the desired length has been achieved. The total time to fixator removal may be longer in mature patients with large discrepancies. In these cases we have performed lengthenings over intramedullary rods. After the desired length is obtained, the fixator is removed and the nail is locked. Adequate intramedullary fixation allows for the early fixator removal during the long consolidation phase.

III. Special Conditions

During tibial lengthenings it is mandatory to osteotomize the fibula. One to two centimeters of fibular middiaphysis is removed using an oscillating saw. This is done at least five centimeters proximal to the ankle joint. In cases of fibular hemimelia and concurrent tibia shortening, the fibrocartilaginous fibular anlage is similarly sectioned. In cases of tibia lengthening longer than 4 centimeters or greater than 15 percent, it is important to stabilize the distal tibial-fibular syndesmosis. This is routinely done with a cortical screw, or with one of the distal external fixator pins passing through the tibia into the fibula. In addition, casting the lower leg and ankle or the use of

orthotics may prevent contracture or ankle subluxation during lengthenings in fibular hemimelia or congenital pseudarthrosis of the tibia

In cases of lengthening for proximal femoral focal dysplasia or congenitally short femur, it may be necessary to release the iliotibial band, lateral intramuscular septum, rectus femoris, hamstrings and adductors. In addition, these conditions are often associated with hip or knee instability that is secondary to dysplasia or congenital absence of the cruciate ligaments. It is critical to periodically examine and obtain radiographs of these joints for the presence of subluxation. Knee subluxation can be partially prevented by placing the distal pins close to the knee axis. This provides stabilization by tethering the iliotibial band. Any sign of knee subluxation requires cast application. Hip subluxation may be expected in cases of acetabular dysplasia or valgus proximal femur. Instability may be prevented by acetabular osteotomy or supplemental pin fixation to the pelvis.

IV. Problems and Complications during Limb Lengthening

Alterations in the planned treatment course during distraction callotasis may be divided into minor or major problems and complications. Minor problems are commonly encountered and are of no practical significance. Major problems require a change in the treatment plan while complications may result in an unsatisfactory outcome or require additional surgical procedures. The following is a list of conditions and options for management of these conditions.

Pin site problems—Pin site inflammation is very common and presents with mild erythema and serous drainage. It appears to be more common in juxtaarticular pins and in pins with substantial soft tissue from skin surface to periosteum. The incidence of actual pin site infection has been estimated to be only 2 percent in monolateral fixation and distraction callotasis[21]. Most of these cases require oral antibiotics and pin care with hydrogen peroxide or surgical soaps. Pin site osteomyelitis is a much less common yet more severe complication. In these rare instances, pin removal, pin site curettage and intravenous antibiotics is required. The pin is surgically replaced at a site that is clear of inflammation.

External fixation and specifically distraction callotasis will often lead to unacceptable scarring. Fascial defects from lengthening will often yield pitting and indentation of the soft tissues. Scar revision of the skin and subcutaneous tissues will frequently improve the cosmetic appearance. For maximal improvement in scars from lengthening, it is necessary to revise and close the underlying fascial defects.

Joint contractures—Joint stiffness is commonly seen during limb lengthening, and may occasionally compromise results. Distraction is halted when there is joint subluxation or extreme limitation of motion. In general a progressive hip contracture greater than 30 degree of flexion and 30 degrees of adduction or abduction, knee flexion contracture greater than 30 degrees or knee range of motion less than 30 degrees is an indication to stop distraction. Progressive ankle equinus may be prevented in expected lengthenings of 20 per cent or greater with orthotics or casting around the distal pin cluster. It is important to remember that stiffness is a result of myotendinous tightness and not a primary joint contracture. Joint manipulation will not address the primary pathology and may in fact damage the articular surface. At one month after device removal there should be improved range of motion, and gradual improvement should occur every two weeks. Tendon lengthenings are considered when there is no improvement in motion over a period of one month.

Premature callus consolidation—Premature consolidation of the callus occurs in approximately 7% of monolateral lengthenings.[21] This may be due to an incomplete osteotomy, failure to distract at an appropriate rate, or result from an unstable fixator construct. Increasing the rate of distraction to 1.25 millimeters per day may prevent consolidation if exuberant callus formation is discovered early. Open or closed torsional osteoclasis will be required if the regenerate prematurely consolidates.

Failure to form adequate regenerate—Poor callus formation or a distraction gap may result from excessive immediate osteotomy gap, shortened waiting period, rapid distraction (greater than one millimeter/day), larger distraction steps (greater then 0.5 millimeter/step), or poor bone metabolism. This may be reversed by actually compressing the distraction site at the same rate for 7 to 10 days followed by normal distraction.

Angulation or axial deviation—Angulation and deviation usually follows characteristic patterns. Prevention begins with stable construct application; axial deviation is rarely a problem when this is achieved. Femoral lengthenings may tend to drift into varus and posterior angulation while tibial osteotomies tend toward valgus and anterior angulation. This may be due to asymmetric callus consolidation opposite fixator placement. In addition, angulation may also be due in part to a predominance of muscle and other soft tissue that are opposite each fixator.

A shift or displacement of the lengthened portion may be due to other causes. Failure to lengthen along the mechanical axis, incomplete osteotomy or immediate osteotomy angulation due to pin pretensioning (see above) may result in early deviation. Fixator failure due to ball joints, pin loosening, soft bone (metabolic conditions) or other types of mechanical failure may account for deviation throughout the treatment course. Significant deviation can usually be managed with manipulation and translation

under anesthesia after the length has been obtained. In cases of extreme lengthenings and expected deviation, the surgeon can preset the tibia and femur in 5 degrees of varus or valgus by slightly converging the proximal and distal pin clusters.

Summary

In conclusion, monolateral external fixation can be effectively utilized in the management of limb length discrepancy and angular deformity. This manuscript outlines the pertinent theory, application and problems important in these cases. When faced with specific congenital conditions the surgeon is encouraged to reference relevant literature that is more focused than the current paper.

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IMAGING STUDIES FOR DIAGNOSING STRESS AND INSUFFICIENCY FRACTURES

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INTRODUCTION

Stress fractures are a common orthopaedic problem, not only in athletes and active individuals, but also in the elderly and patients with osteopenia due to metabolic bone diseases. Early detection of these injuries is important because continued activity in the presence of a stress fracture can lead to a complete, displaced fracture. However, it can be very difficult to demonstrate a stress fracture, and frequently, several different imaging modalities are needed to evaluate a patient with suspected stress fracture. Optimal and cost efficient use of imaging to diagnose stress fractures requires an understanding of the types of stress fractures, their common clinical presentations, and the advantages, limitations, and indications for each imaging modality.

Stress fractures develop most commonly in the metatarsals, tarsals, tibia, femur, pelvis and spine, but they can occur in almost every bone[4,6-9,11,13,14,17]. The initial reports of stress fractures described them in the normal bones of young adults. Most authors identified an association between stress fractures and repeated episodes of increased activity, such as with military training or participation in sports[7,8,12]. These reports led to the concept that stress fractures result from fatigue failure of normal bone matrix. Experimental work shows that repeated application of non-traumatic impulsive loads can cause stress fractures in normal bones[1]. In addition, multiple clinical reports have described stress fractures in individuals with rheumatoid arthritis, lupus erythematosis, osteoarthritis, pyrophosphate arthropathy, renal disease, osteoporosis and joint replacements, and in older patients without apparent musculoskeletal disease[2,5,10,13,15-17]. Most of these individuals have not had a history of increased activity, but many of them have decreased bone density. Some physicians refer to stress fractures that occur in these patients with decreased bone density as insufficiency fractures.

Most patients with nondisplaced stress fractures describe deep pain, often poorly localized, that may be exacerbated with loading of the bone or with selected activities. Few patients have night pain. Some individuals have tenderness with compression or direct palpation of the involved bone. In long bones, percussion or vibration applied to an asymptomatic region of the bone may cause pain at the fracture site. The clinical diagnosis of stress fracture can be difficult as poorly localized musculoskeletal pain can be a symptom of many disorders, including neoplasms, infection, joint diseases, and muscle or nerve disorders. Patients usually will note an increase or change in activity, but often do not remember this until directly questioned. Therefore, the history leading toward the diagnosis of stress fracture is often lacking or equivocal. Because the diagnosis is often difficult to make on clinical grounds, imaging studies are usually needed to evaluate patients with suspected stress fractures.

ROENTGENOGRAMS

Despite the fact that roentgenogram findings characteristic of stress fractures often lag the clinical onset of pain by two to four weeks[11] (Figure 1), they are the first radiologic study that should be ordered. Plain film changes that suggest stress fracture include linear areas of sclerosis within the bone, subtle periosteal reaction at the location of pain, incomplete lucent lines within the cortex at the site of pain, or focal osteopenia in the region of the fracture. Most stress fractures are oriented transversely to the bone, but occasionally longitudinal stress fractures are encountered (Figure 2).

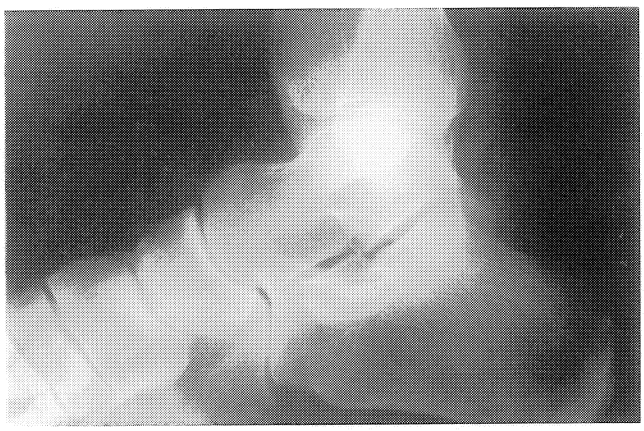

Figure 1. 50 year-old patient with progressive heel pain.
Figure 1A. Initial lateral radiograph of the heel is normal.

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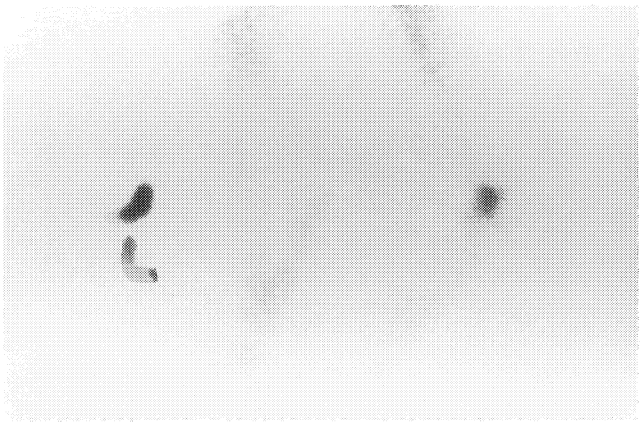


Figure 1B. Because of continued pain over 2.5 months, a bone scan was done which shows increased uptake in the region of the calcaneus.

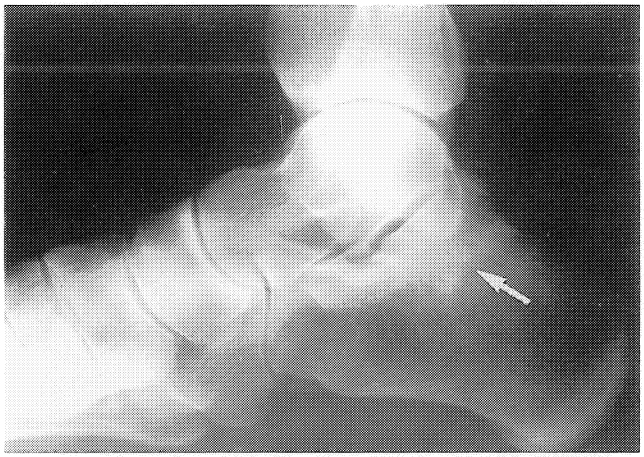


Figure 1C. Repeat radiograph obtained at time of the bone scan shows a calcaneal stress fracture (arrow).

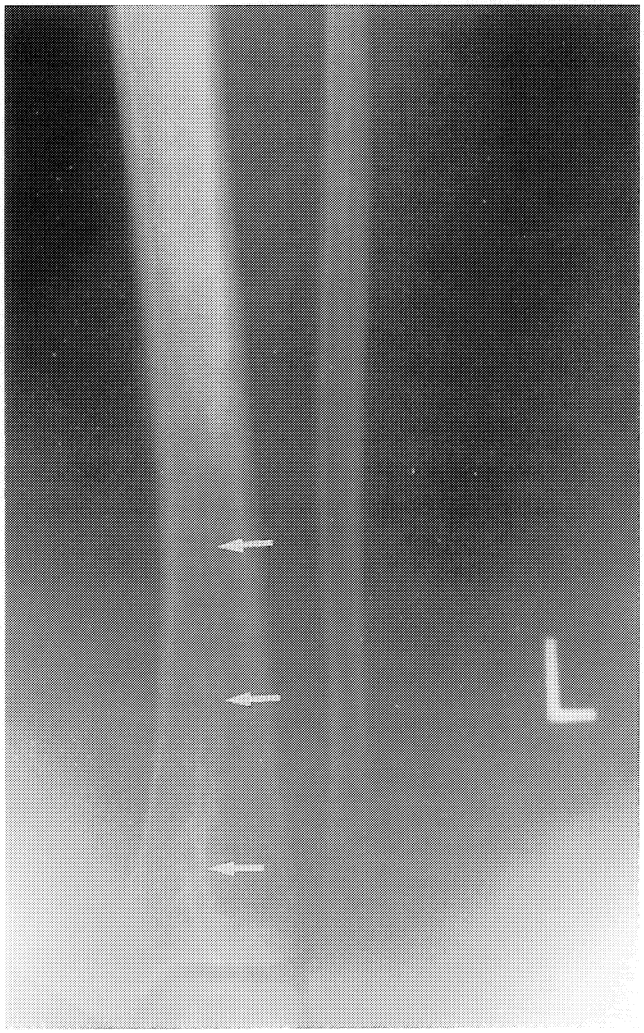


Figure 2. 59 year-old woman with increasing anterior left tibial pain following right total knee replacement.
Figure 2A. AP radiograph demonstrates longitudinal stress fracture (arrows).

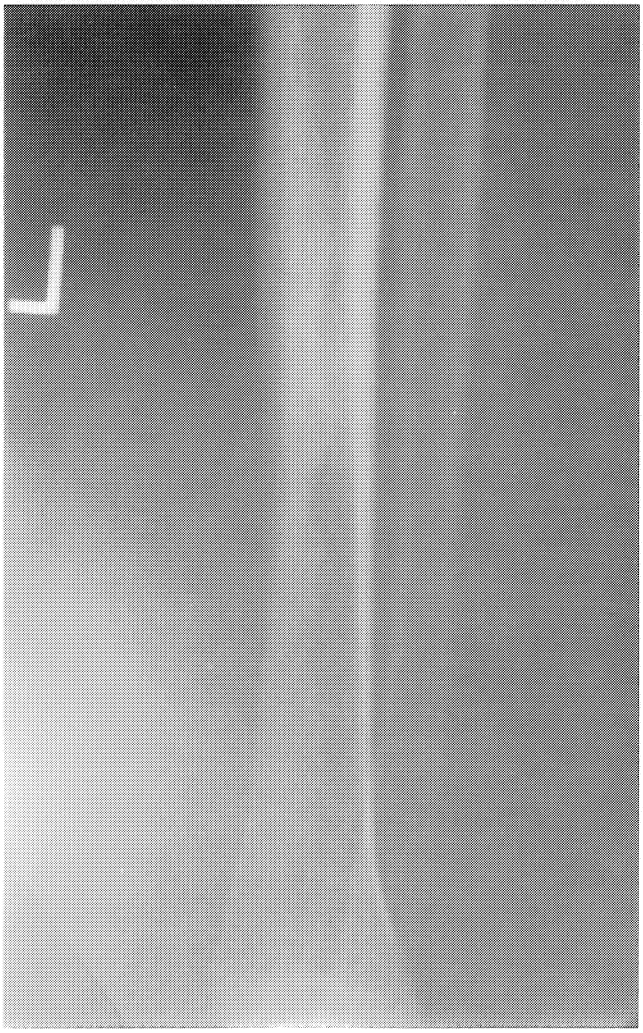


Figure 2B. Lateral view is less conspicuous, but cortical irregularity is present anteriorly.

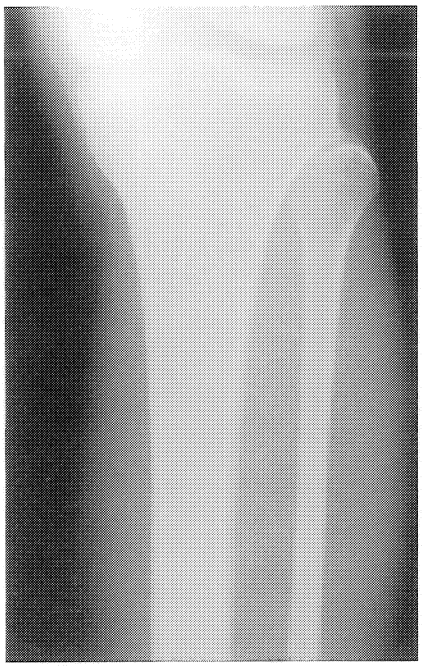


Figure 3. 22 year-old competitive runner with left proximal tibia pain.
Figure 3A. AP radiograph is normal.

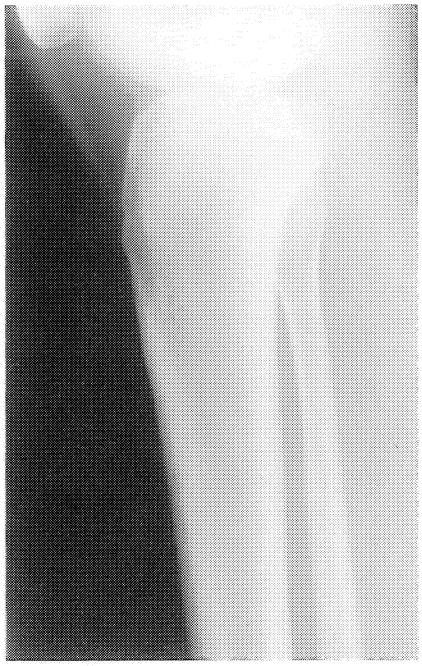


Figure 3B. Lateral radiograph is normal.

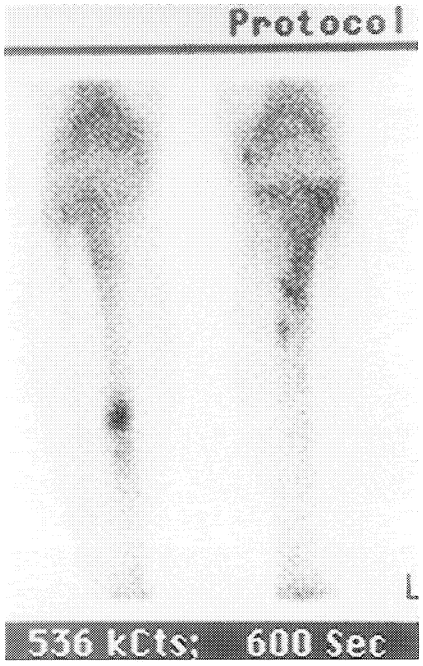
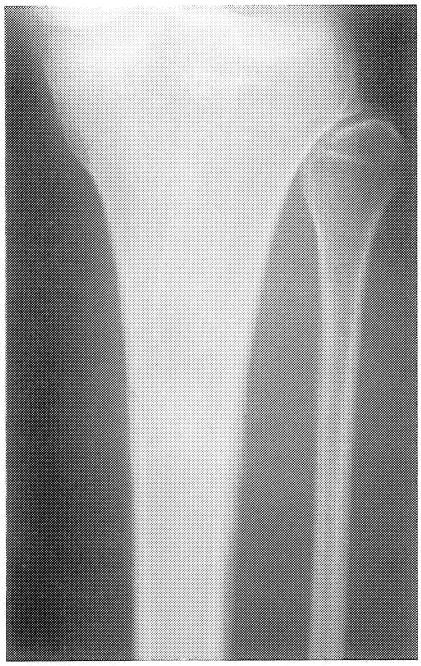


Figure 3C. Bone scan shows abnormal increased uptake in the proximal left tibia and the midshaft of the right tibia. Patient was more symptomatic on the left.



Figures 3D and E. Follow-up radiographs, obtained four weeks later, demonstrate typical transversely-oriented stress fracture.

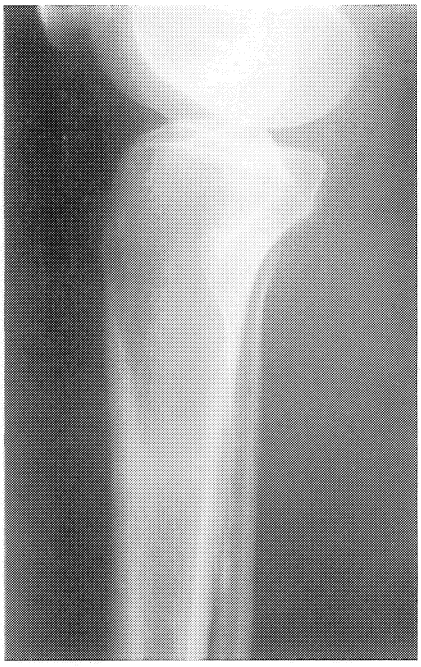


Figure 3E.

BONE SCINTIGRAPHY

When plain radiographs are negative and the suspicion for fracture is high, other imaging tests can be used to detect stress fractures (Figure 3). Bone scintigraphy, using Technetium^{99m} radiolabeled methylene diphosphonate (Tc^{99m} MDP), typically shows increased uptake of the tracer at the site of the fracture. Bone scans are much more sensitive than plain films for detecting stress fractures and can become positive as early as three days after onset of clinical symptoms[11]. One disadvantage of a positive bone scan is that it is not specific for fracture; it occurs with almost any injury or disease that increases osteoblastic activity, such as infection, neoplasm and bone infarction. Bone scans may show increased blood flow to the affected extremity, but the delayed static images are where the diagnosis is usually made. Single photon emission computed tomography (SPECT) can be used to improve the sensitivity and specificity for diagnosing stress fractures, especially of the axial skeleton. However, in most cases, planar images will be sufficient.

Stress fractures of the pars interarticularis can cause chronic back pain that clinically resembles the more common problem of activity related mechanical low back pain[8,14]. These fractures frequently occur in young, active people and despite the symptoms they may remain undetected. A fracture line in the pars interacticularis shown on plain radiographs establishes the diagnosis, but in some patients plain radiographs will be normal. In these individuals a bone scan with SPECT may show focal increased activity in the pars interarticularis, confirming the presence of a stress fracture[14].

COMPUTED TOMOGRAPHY

Computed tomography (CT) is more sensitive than plain radiography and can show fractures that are not apparent on plain radiographs (Figure 4). CT may also be used to evaluate healing when plain radiographs do not adequately demonstrate the fracture. One disadvantage of CT is that scanning is limited to the axial plane in many circumstances. Since most appendicular stress fractures are transversely-oriented, axial images are not optimal for fracture demonstration. Careful attention to patient positioning can minimize this limitation. Helical or spiral CT has the potential to minimize this limitation because thin, overlapping slices can be obtained and multiplanar reconstructions performed to demonstrate fractures not well seen on initial images. CT is particularly suited for demonstrating longitudinally oriented stress fractures, and for detecting fractures in the foot, especially of the tarsal navicular (Figure 5).

Uncommonly, stress fractures can run in a longitudinal orientation. This causes problems with diagnosis and delay of treatment. Most longitudinal stress fractures occur in the distal tibia, usually in the anterior cortex[6]. Plain films are often normal initially, and bone scanning will show an area of increased uptake along the anterior shaft of the tibia. MRI can be confusing, with abnormal marrow signal extending over a distance in the medullary cavity, unlike the usual appearance of transverse fractures. CT is characteristic, with a small break in the cortex seen on several consecutive images, associated with endosteal and periosteal reaction.

Figure 4. 82 year-old woman with ill-defined abdominal pain and suspicion of metastatic disease.

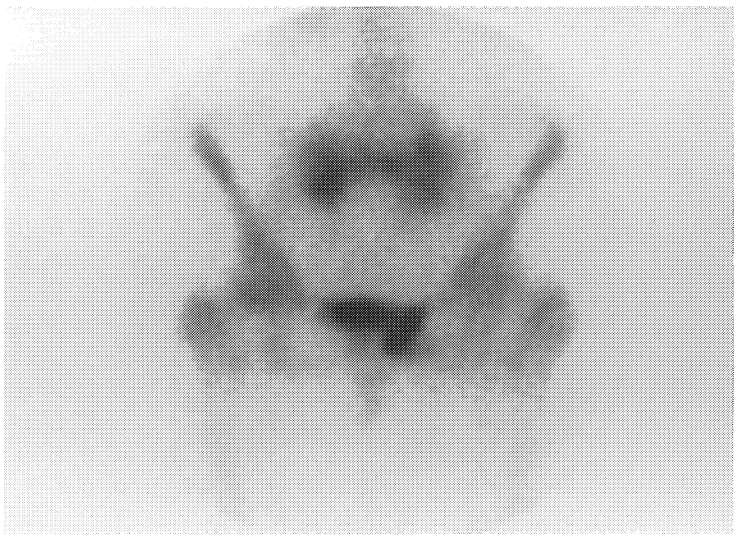


Figure 4A. Bone scan shows increased uptake in the sacral ala bilaterally. Note increased uptake in left superior pubic ramus.

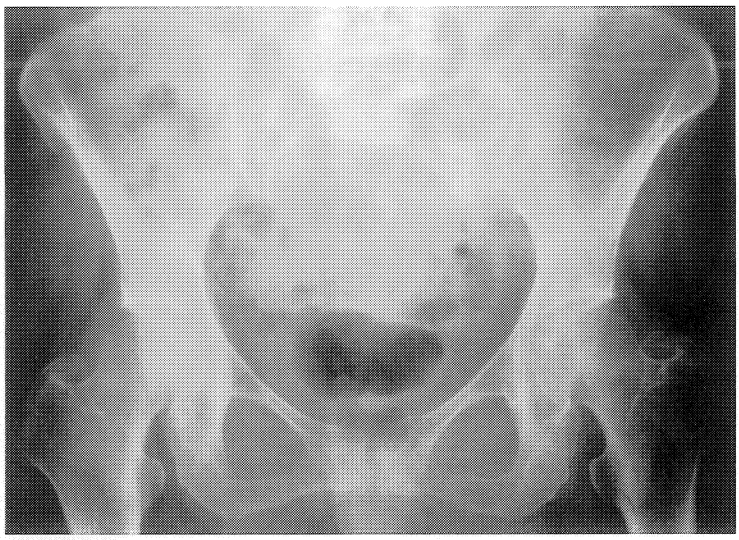


Figure 4B. Plain radiograph shows cortical stepoff at left superior pubic ramus, sacral ala are obscured by bowel gas.

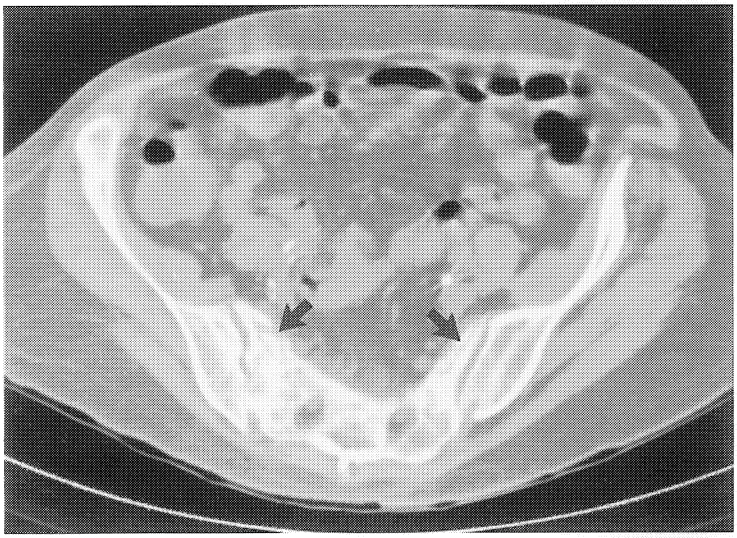


Figure 4C. CT image through the sacrum shows bilateral insufficiency fractures (arrows).

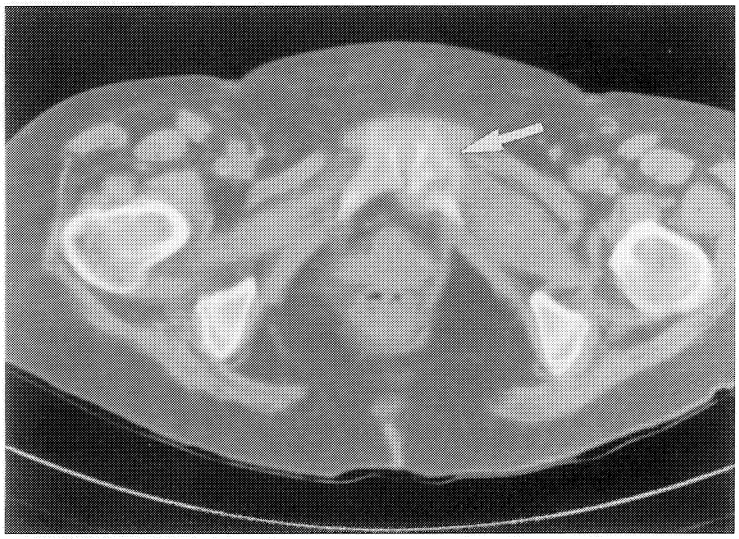


Figure 4D. CT image at level of the symphysis pubis demonstrates the left pubic ramus fracture (arrow).

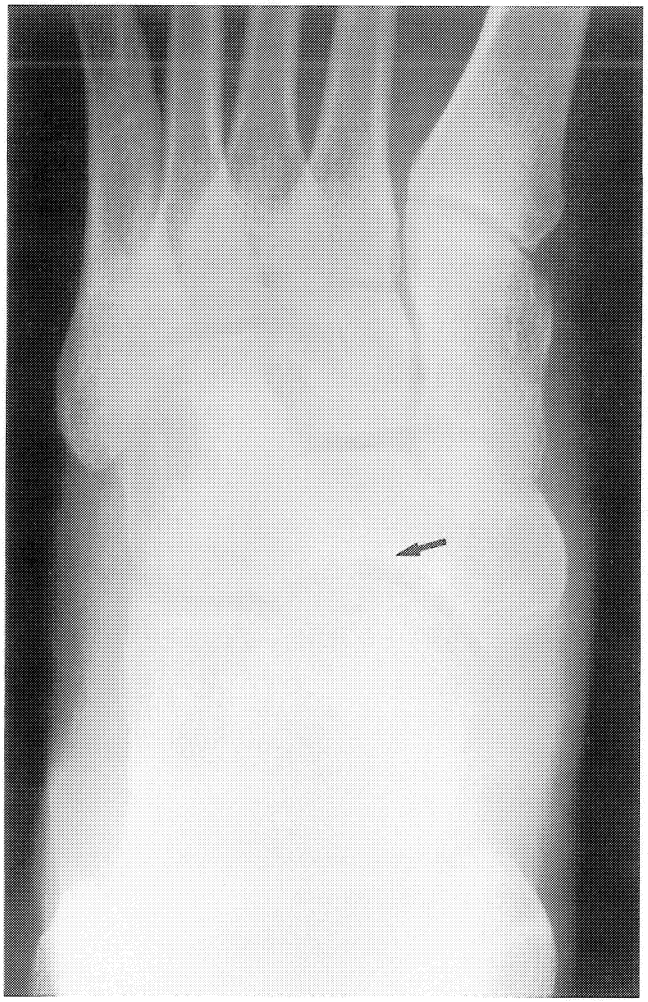


Figure 5A. AP radiograph demonstrates very subtle abnormality of the tarsal navicular with involvement of the talonavicular joint (arrow).

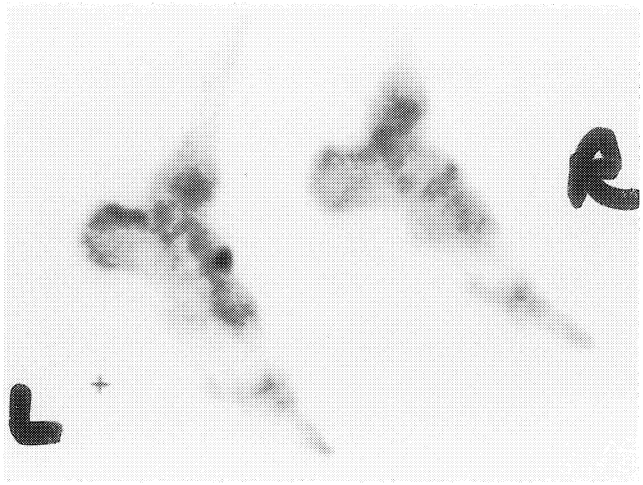


Figure 5C. Bone scan shows increased uptake at the left talonavicular joint.

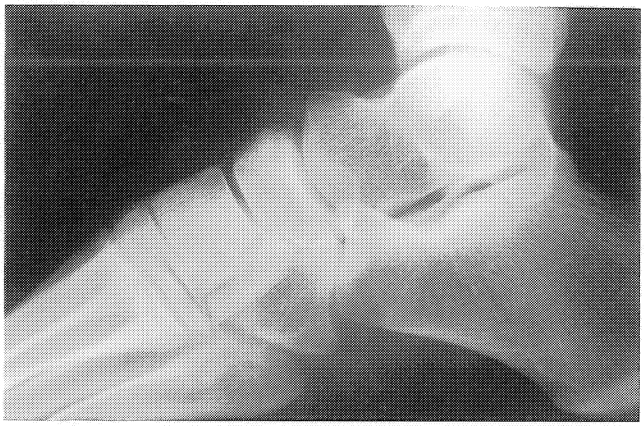


Figure 5B. Lateral radiograph is normal.

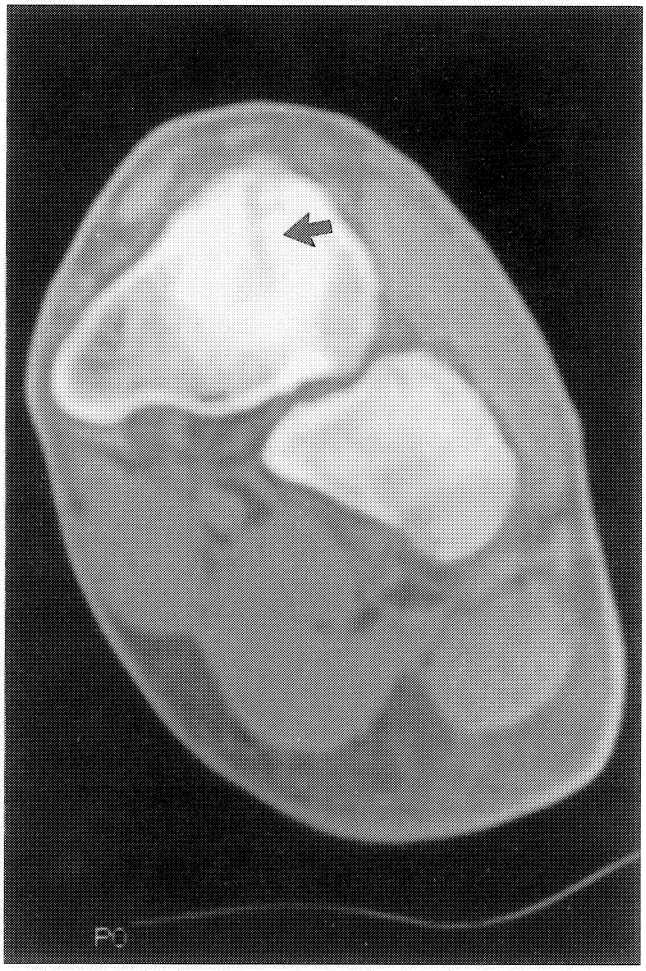


Figure 5D. CT scan with image parallel to the talonavicular joint demonstrates incomplete stress fracture of the navicular (arrow).

Figure 5. 21 year-old tennis player with ongoing pain in dorsum of midfoot.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) is sensitive for detecting abnormal edema of bone marrow which is usually present in fractures (Figure 6). MRI has been shown to be effective for detecting stress fractures of the tibia [3]. The characteristic pattern seen with MRI is a linear area of abnormal signal involving the cortex and medullary cavity. However, signal changes on MRI can be nonspecific and can be seen with neoplasm or infection. Although MRI is very sensitive for bone marrow edema, CT better depicts the osseous anatomy of stress fractures. MRI is more expensive than either CT or bone scans. MRI, with its superior soft tissue contrast, does have an advantage in that soft tissue abnormalities mimicking a stress fracture can be properly characterized.

Traditionally, T1- and T2-weighted spin echo sequences have been the mainstay of MRI. With these sequences, edema is seen as bright signal on the T2-weighted images. However, many centers are using a modified T2-weighted sequence, called fast spin echo (FSE). The advantages of this sequence are faster examinations and less motion artifact. The main disadvantage is that fat, which is low in signal on traditional T2-weighted images, remains high signal on FSE T2-weighted studies. Subtle areas of edema within the bone marrow can be masked by the high signal of marrow fat.

To correct for this limitation, one or two "edema sensitive" sets of images are frequently obtained to detect subtle edema in the bone marrow. These "edema sensitive" sequences are called inversion recovery or "fat-suppressed" T2-weighted images. Both types of images have technical advantages and disadvantages; both show edema as very conspicuous bright signal areas and we routinely include one of these sequences when searching for stress fractures.

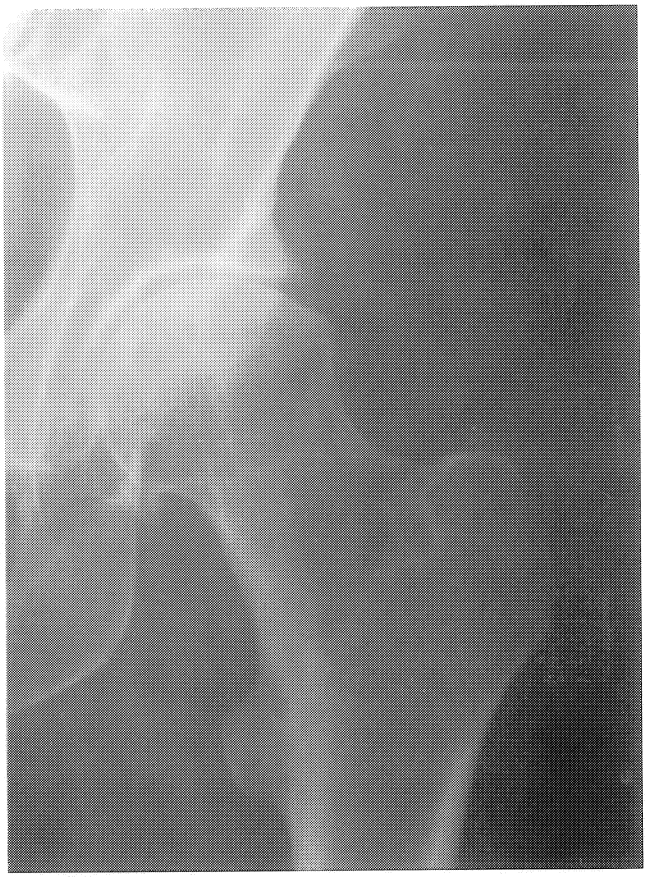


Figure 6. 70 year-old woman with increasing left hip pain and clinical suspicion of fracture.
Figure 6A. AP radiograph of left hip is normal.

Figure 6B. Because of strong clinical suspicion of fracture, an MRI was performed. The T1-weighted (TR = 450 msec, TE = 18 msec) coronal image shows a nondisplaced intertrochanteric fracture (arrows).

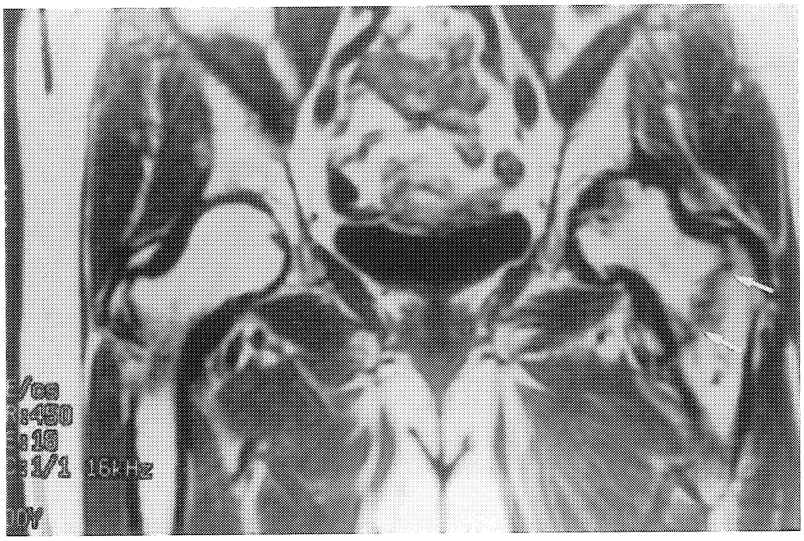


Table 1. Radiographic Evaluation of Stress and Insufficiency Fractures

Study	Findings	Advantages	Disadvantages
Plain Radiographs	-periosteal, endosteal or medullary new bone formation -fracture line	-inexpensive -can be diagnostic	-may be normal
Technetium^{99m} Bone Scan	-increased tracer localization	-sensitive (normal bone scan makes diagnosis of fracture extremely unlikely)	-non-specific
CT Scan	-cortical disruption -periosteal, endosteal or medullary new bone formation	-sensitive and specific for fracture	-limited ability to demonstrate soft tissues (may not dectect soft tissue or medullary lesions) -limited scanning planes
MRI	-marrow and soft tissue edema	-sensitive -demonstrates soft tissues well -images can be obtained in any plane	-non-specific -CT better for demonstrating changes in cortical bone

USE OF IMAGING STUDIES IN THE DIAGNOSIS OF STRESS FRACTURES

Patients with persistent localized skeletal pain should be evaluated for possible stress or insufficiency fractures. Increased pain with activity, along with localized bone tenderness, strongly suggests the presence of a stress or insufficiency fracture. Plain radiographs are indicated in patients with a suspected stress or insufficiency fracture. If these studies confirm the presence of a stress fracture, no further imaging investigations are needed.

If the plain radiographs are normal or nondiagnostic, the physician may choose to treat the patient for a presumed fracture and repeat the studies within six weeks or order further studies. For example, in an individual with a possible metatarsal or tibia fracture who is not participating in a physically demanding occupation or competitive athletics, treatment with decreased activity or a cast and repeat radiographs will often be appropriate. However, some stress fractures are at risk for displacement even with decreased activity. These fractures need prompt and accurate diagnosis. That is why a patient with a suspected stress or insufficiency fracture of the femoral neck should have other studies. For these patients, bone scans will often show increased uptake, but a limited MRI study will show evidence of the fracture more quickly and with more specificity which can facilitate the decision between operative and conservative management. Recognizing the need for early diagnosis of stress fractures of the femoral neck, The American College of Radiology (ACR) has recently recommended a limited MRI of both hips as the imaging test of choice for patients suspected of femoral neck fracture in whom plain films are negative.

In elderly patients with decreased bone density, detecting fractures of the pelvis and sacrum with plain radiographs can be extremely difficult. In these individuals, a CT scan is often the most direct and cost effective method of demonstrating the fracture. When scanning a patient for suspected sacral insufficiency fracture, tilting the gantry of the scanner will improve visualization of fracture lines. Tilting the gantry towards the feet will generate images perpendicular to the sacrum, yielding cross-sections which are optimal for detecting fractures. For images coronal to the sacrum, the gantry can be tilted toward the head. Alternatively, if spiral or helical CT scanning is available, a volumetric data set can be acquired which allows for reconstructed images in any plane.

Bone biopsy should be avoided in patients whose history, physical examination and imaging studies strongly support the diagnosis of stress or insufficiency fracture. Although a biopsy can confirm the presence of fracture callus, fracture callus may also be present in other conditions including neoplasms, infections, and metabolic bone diseases. Importantly, a biopsy may confuse the clinical picture and lead to incorrect diagnoses because fracture callus can be very difficult to differentiate from bone forming neoplasms, even by experienced pathologists. The defect from a biopsy may weaken the bone, putting the patient at risk for subsequent fracture displacement. When the diagnosis of stress or insufficiency fracture cannot be clearly established by imaging studies, evaluation by a musculoskeletal oncologist may be appropriate.

For some patients, observation and follow-up imaging studies will be sufficient, but in others a carefully planned biopsy may be indicated.

CONCLUSION

Stress fractures typically present as localized pain that develops without a history of a specific acute injury. The differential diagnosis includes primary or metastatic neoplasms, infections, musculoskeletal soft tissue injuries, nerve compression syndromes and joint diseases. Imaging studies can be helpful in distinguishing stress fractures from these other conditions. In cases where plain radiographs demonstrate changes consistent with a fracture, including a fracture line or fracture callus, no other studies are needed. In other cases, plain radiographs will be normal or non-diagnostic and the diagnosis may be delayed. In these instances, more sensitive tests, such as bone scans, computed tomography or magnetic resonance imaging will usually demonstrate abnormalities consistent with a fracture. Optimal use of these tests requires understanding of the specific advantages, indications and approach to the interpretation of each imaging modality

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TOWARD DETERMINING CONSTRUCT MECHANICAL OPTIMALITY FOR FIBULAR BONE GRAFTING IN FEMORAL HEAD OSTEONECROSIS

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Thomas D. Brown, Ph.D.†

INTRODUCTION

While most cases of femoral head osteonecrosis progress to collapse and require total hip arthroplasty, the disturbingly high rates of prosthesis loosening seen in this patient population[8] have prompted a continuing search for head-preserving surgical alternatives. Of the various procedures devised to slow or arrest collapse of the natural femoral head, vascularized fibular bone grafting has shown the most promise[6], especially in cases where structural failure of the head has already begun or is incipient[9]. In this procedure, a 12-15 cm vascularized fibular segment anastomosed to the proximal femoral retinacular vessels is inserted up into a core tract drilled superomedially through an entry site on the proximal lateral cortex. Mechanically, the graft functions as a cantilever beam, uptaking some of the load that would otherwise pass through (necrotic) cancellous bone in the weight-bearing tract of the femoral head.

To date, intra-operative placement of fibular grafts in osteonecrosis has relied upon intuitive judgement, the general objective being to site the graft tip near the top of the central region of the lesion. However, two recent finite element models of this situation[3,10] have independently demonstrated that the degree of structural protection actually afforded to the lesion depends sensitively upon the details of graft placement. While a few generic operative guidelines (e.g., the desirability of subchondral plate engagement) emerged from those models, the highly idiosyncratic nature of lesion involvement patterns makes it difficult to know what graft position is structurally optimal for a given patient. The general availability of MRI-based information about the three-dimensional extent of individual lesions, coupled with contemporary high speed computing capabilities, now makes it plausible to entertain the possibility of patient-specific finite element analysis as a surgical planning vehicle for structurally optimal fibular grafting in osteonecrosis. In order to be logistically practical, however, means are needed to move beyond the current prohibitively labor-intensive practice of trial-and-error finite element consideration of alternative graft positions.

We here report development of a new computational procedure whereby the process of determining the structurally optimal position of a vascularized fibular bone graft can be automated. For a demonstration case involving a typical segmental lesion, the algorithm gives a clinically reasonable result, in that its identified site of optimal graft placement is found to lie well within the range of placements empirically recognized as being appropriate in current surgical practice. Several computational factors governing robust algorithmic performance are systematically studied: graft repositioning step size, convergence criteria, solution stability, and solution uniqueness. Also, the algorithm's ability to coordinate simultaneous modifications of multiple graft placement parameters is demonstrated. The new optimization algorithm involves a substantially greater computational burden than that seen with conventional finite element modelling. Its avoidance of the need for manual intervention is attractive, however, when viewed against longstanding trends of increasing health care personnel costs and decreasing computational resource costs.

METHOD

In any optimization problem, the key first step is to formally define an objective mathematical function to be maximized or minimized, along with any physical constraints which must be satisfied either by the objective function itself or by the variable parameters upon which the objective function depends. In the case of fibular grafting for femoral head osteonecrosis, the general strategy is to determine the particular graft position and graft diameter which maximally offload mechanical stress from collapse-prone necrotic cancellous bone. Such a problem can be formulated as follows.

Let the geometric position of the graft axis entry point (graft root) on the lateral femoral cortex be designated as point $\mathbf{P}_1$, and let the position of the graft tip be designated as point $\mathbf{P}_2$ (Figure 1). The fibular graft is assumed to be straight and to have a diameter d. These three geometrical parameters ($\mathbf{P}_1$, $\mathbf{P}_2$, and d) constitute design variables, upon which a to-be-optimized objective function $\mathbf{f}(\mathbf{P}_1,\mathbf{P}_2,d)$ depends. Previous patient-specific finite element modelling in a series of osteonecrosis patients at the University of Iowa Hospitals and Clinics showed that the

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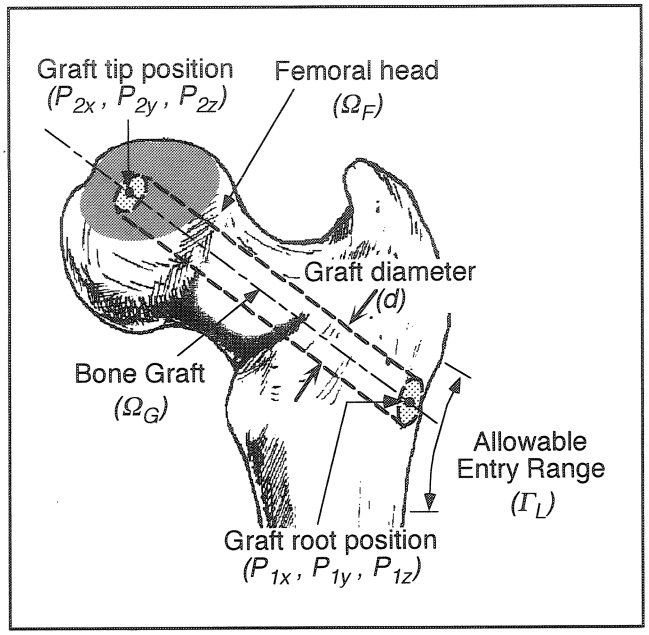

Figure 1. Optimization problem for graft positioning and sizing in femoral head osteonecrosis

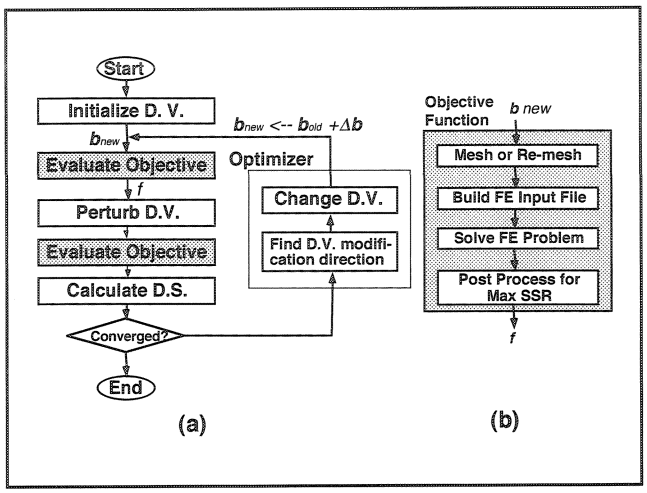

Figure 2. Flow chart of the computer program for optimization bone graft positioning. (a) Loop of function evaluation, sensitivity evaluation and change of design variables. (b) Objective function evaluation module.

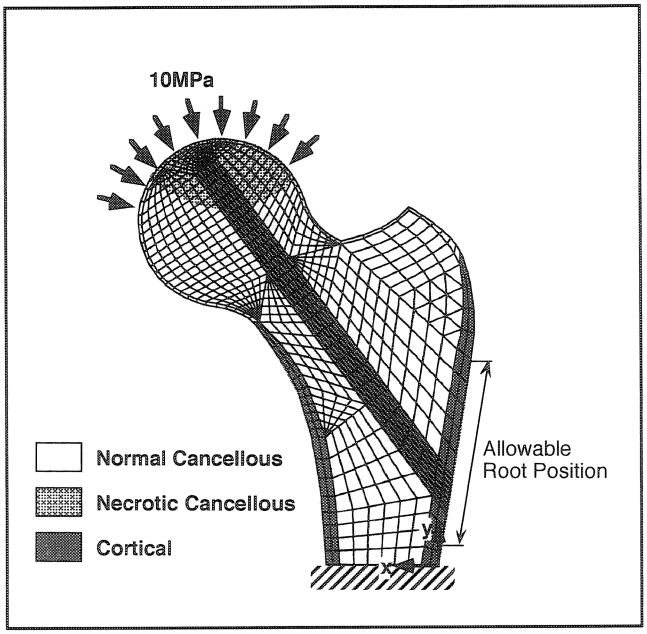

Figure 3. Finite element subdivision of 2-dimensional graft model of femoral head osteonecrosis

relative propensity for radiographically apparent collapse has a statistically significant rank order correlation with the peak local stress-to-strength ratio (SSR) computed in the respective individual lesions[2]. (In this context, SSR is formally defined as the local principal compressive stress divided by the local uniaxial compressive strength of bone tissue.) Thus, for a given patient, a fibular graft can be considered as being optimally positioned when it minimizes the peak local SSR occurring anywhere within that patient's necrotic lesion. The design variables therefore need to be systematically altered by the optimization algorithm, in such a manner as to satisfy the design objective: f ($\mathbb{P}_1$, $\mathbb{P}_2$, d) = Peak Local SSR $\rightarrow$ minimum. Points $\mathbb{P}_1$ and $\mathbb{P}_2$ are vectorial quantities whose full specification involves sets of component coordinates, e.g. (P_{1x}, P_{1y}, P_{1z}) and (P_{2x}, P_{2y}, P_{2z}), respectively. The graft diameter, by contrast, is a simple scalar variable.

One important physical limitation on the set of admissible design variables is that the graft tip should not penetrate the subchondral plate. Mathematically, this is represented by the constraint condition: $\Omega_G \in \Omega_F$, where Ω_G designates the physical domain of the graft, Ω_F designates the physical domain of the femur, and the symbol $\in$ denotes boolean inclusion. A second constraint condition is that the graft entry point $\mathbb{P}_1$, on the lateral cortex, should lie somewhere on that portion Γ_L of the periosteal surface (Figure 1) proximal to the insertion of the gluteus maximus, and distal to the ridge of the vastus lateralis, i.e., $\mathbb{P}_1 \in \Gamma_L$.

The algorithmic logic (Figure 2) for solving the optimization problem involves recursive use of the ABAQUS finite element program to calculate the stress distribution (and hence, the SSR distribution) throughout the proximal femur, for specific values of the design variables. Once the peak local SSR is calculated for a given (trial) graft configuration, it is necessary to determine whether or not a slight alteration of that graft configuration will lead to a reduction of peak local SSR, and if so, what particular alteration of graft configuration achieves that reduction most effectively. This step is achieved, at any given graft configuration, by calculating a sensitivity vector Ψ which expresses the rate of change of f with respect to changes of the individual design variables:

$\Psi = (\partial f/\partial P_{1x}, \partial f/\partial P_{1y}, \partial f/\partial P_{1z}, \partial f/\partial P_{2x}, \partial f/\partial P_{2y}, \partial f/\partial P_{2z}, \partial f/\partial d)$, where the operator ∂ designates partial differentiation. The respective partial derivative terms are evaluated numerically, by computing the change of the objective function accompanying small perturbations of each of the design variables. For example, $\partial f / \partial P_{1x} \approx (1 / \Delta x) [f (P_{1x+\Delta x}, P_{1y}, P_{1z}, P_{2x}, P_{2y}, P_{2z}, d) - f (P_{1x}, P_{1y}, P_{1z}, P_{2x}, P_{2y}, P_{2z}, d)]$, where Δx represents a small incremental change of the x-coordinate.

Evaluation of the full sensitivity vector Ψ requires a family of finite element solutions, one solution for each component direction of perturbation of each of the design variables. To obtain this family of solutions (each of which involves a slightly different graft configuration geometry), it is necessary to invoke automated re-zoning of the finite element mesh. This is implemented by characterizing the problem geometry hierarchically, based on a small group of regional patch entities whose vertices are linked to specific bony landmark points. These patches, in turn, are each evenly sub-divided into conventional finite elements, subject to the condition of maintaining continuity of element meshing on either side of the inter-patch borders.

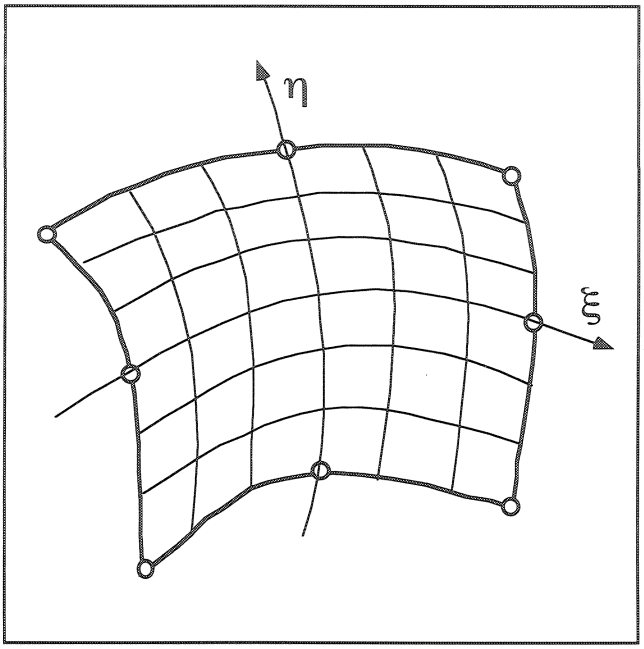

Figure 4. Local coordinate map of a patch.

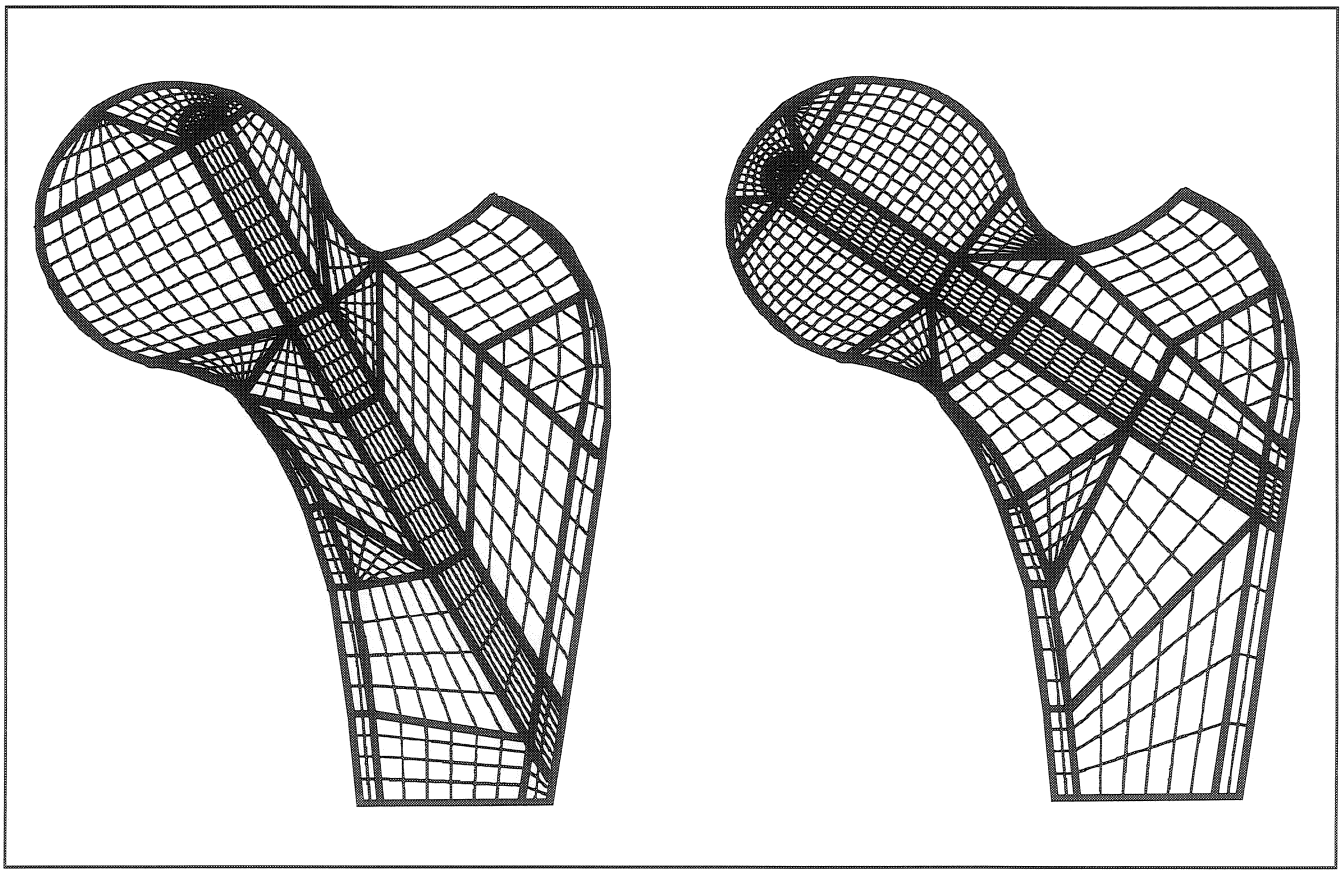

Figure 5. Automated finite element meshing used for graft position changes.

Having evaluated the sensitivity vector at a given graft position, a new graft position is determined by moving the existing graft a small distance, δ, in the direction that achieves the greatest reduction of the objective function f. At this new graft position, the sensitivity vector is again evaluated, and the direction of greatest further reduction of f is again determined. The process is iteratively repeated until there results a converged situation in which no possible perturbations of graft position achieve further appreciable reductions of the objective function. Mathematically, convergence is considered to exist when the relative change of the objective function accompanying design variable perturbation is negligibly small, i.e., $\Delta f / f < \epsilon$, where ϵ is an empirically determined convergence criterion. Overall control of the various steps of the optimization procedure (ABAQUS stress calculation, objective function evaluation, perturbation of the design variables, patch-based finite element mesh regeneration, and monitoring of solution convergence) is performed under the auspices of a UNIX shell script. Importantly, no manual intervention is necessary.

Application of the optimization technique is illustrated by a simplified two-dimensional (plane strain) representation of a proximal femur with a segmental infarction (Figure 3). The model includes three homogeneous material regions: normal cancellous bone, necrotic cancellous bone, and cortical bone (the latter including the fibular graft). These regions were assigned isotropic Young's moduli of 0.3 GPa, 0.1 GPa, and 20 GPa, respectively, and all were assigned Poisson's ratios of 0.3. Contact stress of 10 MPa is uniformly distributed across the articular surface of the femoral head, and the distal cross-sectional margin of the femur is considered to be rigidly fixed. Anchorage points for 30 regional patches (Figure 4) were specified at 34 bony landmark positions on the femur or the graft. As remeshing proceeded during the optimization problem (Figure 5), special logic was invoked to test whether or not each newly formed cancellous element lay inside or

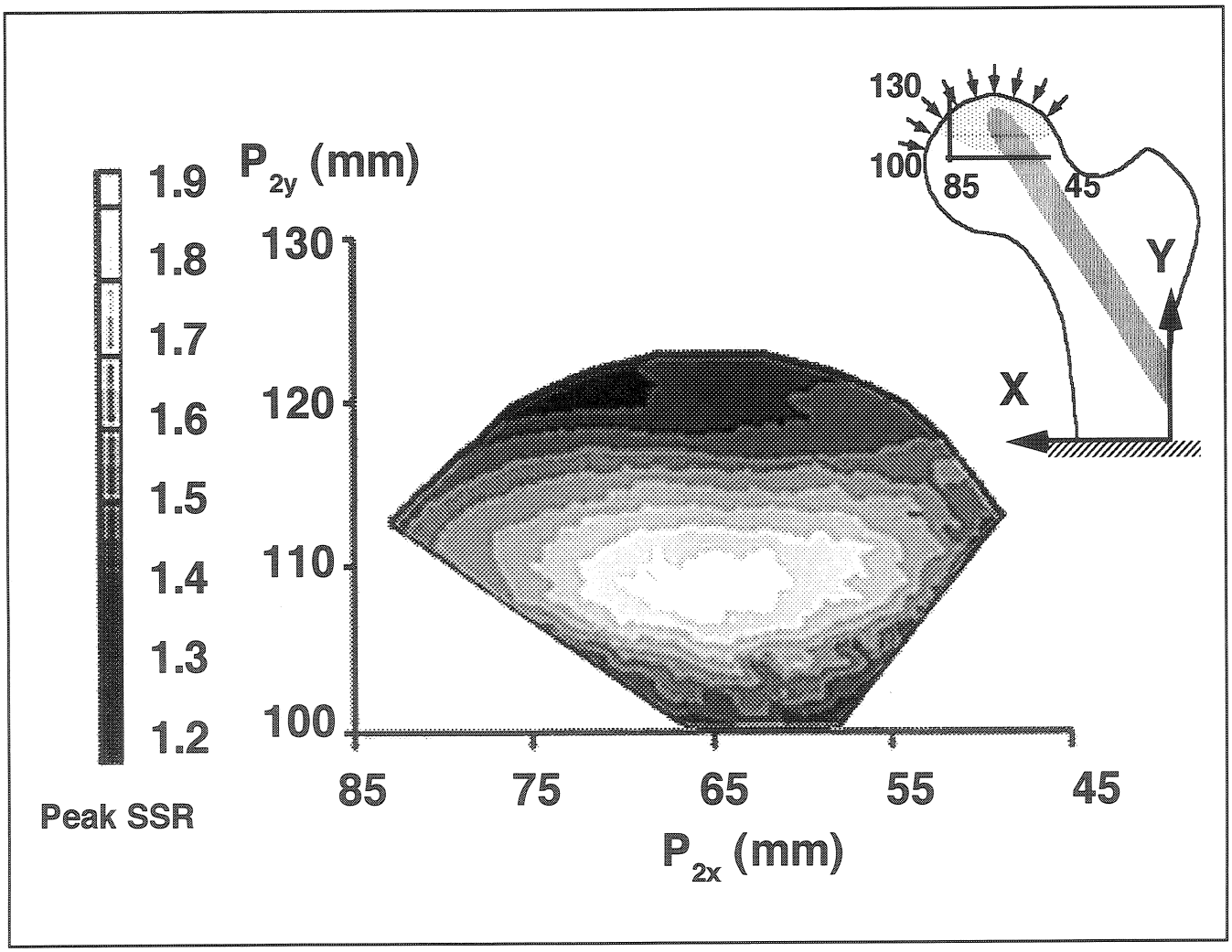

Figure 6. Peak SSR contours in terms of graft tip position P_{2x} and P_{2y}.

outside of the (unchanging) zone of necrosis, with Young's modulus values then being assigned appropriately.

RESULTS

As a prelude to exploring the behavior of the optimization algorithm in this problem, it is helpful first to spatially map the manner in which the objective function f depends on the respective design variables. Such background information is particularly useful for choosing an initial graft configuration from which the optimization can proceed expeditiously. To make a mapping of the objective function space, we first considered a rectilinear array of a large number of closely spaced (1 mm intervals) candidate locations for the graft tip $\mathbf{P}_2$, assuming that the graft axis entry point (P_{1x} = 0 mm, P_{1y} = 20 mm) and graft diameter (d = 8 mm) remain unchanged. The topography of the corresponding distribution of SSR values shows a single peak (bright region in Figure 6) occurring centrally in the lesion, decaying to lower values as one moves toward the periphery of the lesion or toward the subchondral plate. Since these regions of lower SSR are physically separated from one another, it follows that the problem possesses several local optima, and that the convergent solution finally obtained will depend upon the initial graft position chosen. Focusing on the most clinically significant of these local optima (i.e., the one near the subchondral plate), increasing the graft diameter involves lower peak SSR values (Figure 7). The site of optimal $\mathbf{P}_2$ varies only weakly as a function of graft diameter, although sensitivity to positioning precision is more pronounced for a large-diameter graft than for a small-diameter graft. If instead, the graft tip is held constant in a near optimum position ($\mathbf{P}_2x$ = 75 mm, $\mathbf{P}_2y$ = 120 mm) while the entry point $\mathbf{P}_1$ is altered, the data show a nearly monotonic improvement in SSR, for all graft diameters, as the entry point is moved proximally (Figure 8). Combined, these two parametric mappings demonstrate that the SSR maxima are much more sensitive to precision of the graft tip positioning than to precision of the entry point positioning.

Beginning from a graft tip position in the centro-medial region of the lesion and with the entry point held constant, the algorithm successfully finds a locally optimal solution near the subchondral plate (Figure 9). For this particular iterative process, (using a convergence criterion ϵ of 0.05 x 10^{-2} and an initial graft movement step δ of 2 mm), the peak SSR decays by 14% from its initial value. The optimal graft configuration is achieved after seven position adjustments, consisting first of four "large" steps superiorly, followed by two "large" steps medially, and finally one "small" step medio-distally. Summary data for alternative choices of the convergence criterion (Figure 10) indicate that the required number of iterations increases, and the

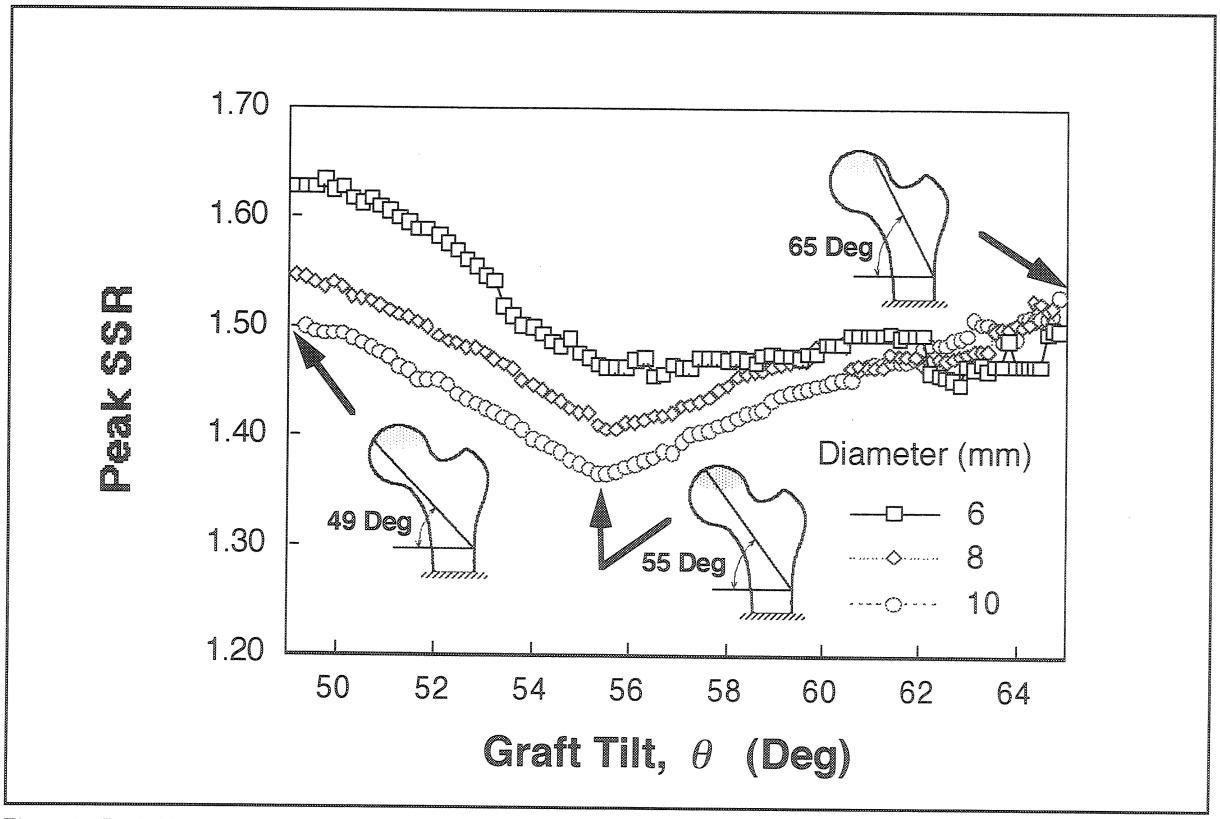

Figure 7. Peak SSR value versus graft tilt angle for a constant graft root position.

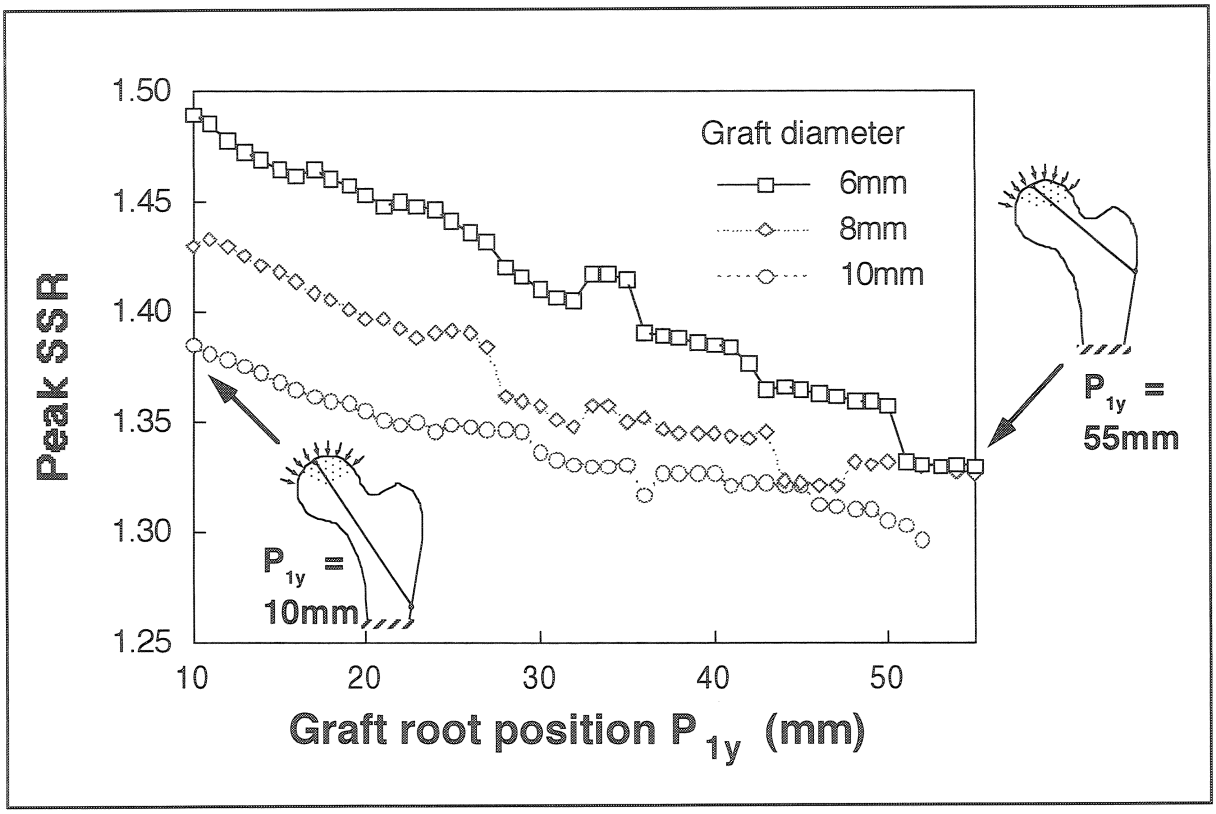

Figure 8. Peak SSR value versus graft root position (P_{1y}) for a constant graft root position.

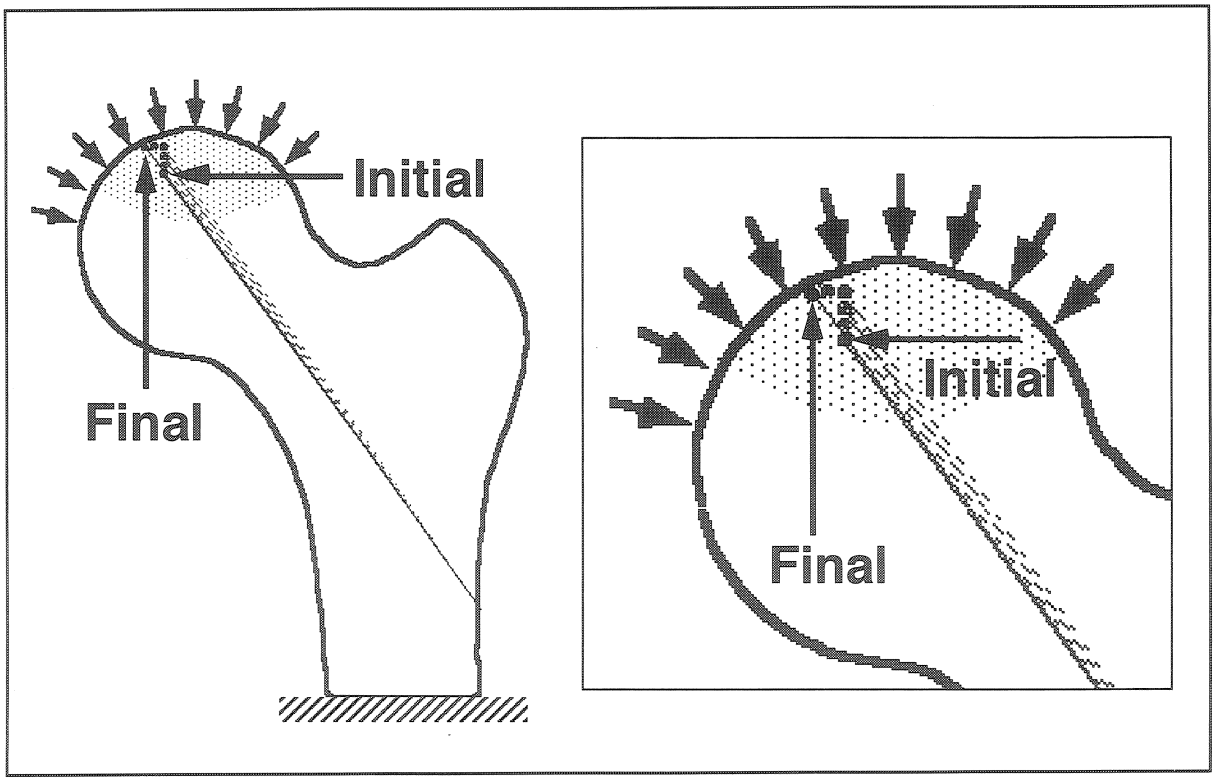

Figure 9. Graft repositioning history during movement to optimal graft tip position (graft diameter: 8mm)

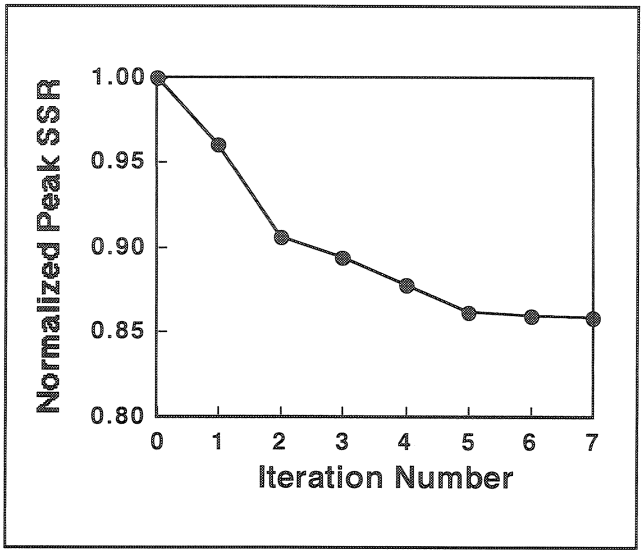

Figure 10. Peak SSR history during movement to optimal graft tip position (constant root position).

final value of peak SSR decreases, as ever more stringent convergence criteria are adopted. Since for practical purposes the final solutions for the design variables P_{2x} and P_{2y} are indistinguishable for $\epsilon = 0.05 \times 10^{-2}$ versus for $\epsilon = 0.005 \times 10^{-2}$, it seems reasonable to adopt a convergence criterion of $\epsilon = 0.05 \times 10^{-2}$ for this problem.

The optimal graft configuration of course depends on both the axis entry position $\mathbf{P}_1$ and the graft tip position P_2, so the ability to maintain numerical stability during simultaneous adjustments of both of these variables is crucial to successful algorithm execution. However, the axis entry point must lie on the lateral cortex periosteal surface region Γ_L (i.e., a line in the two dimensional models, so the values of P_{1x} and P_{1y} are algebraically coupled. Hence, assuming constant graft diameter, concurrent optimization of the tip and entry positions requires using only three independent design variables: P_{1y}, P_{2x}, and P_{2y}. Not surprisingly, during operation of the iterative loop, the sensitivity vector showed substantially larger magnitudes for the terms involving the graft tip position (i.e., $\partial f/\partial P_{2x}$ and $\partial f/\partial P_{2y}$) than for that involving the graft entry position ($\partial f/\partial P_{1y}$). This behavior required invoking a normalization process to avoid "prematurely" achieving convergence for P_{1y} due to using the relatively large ϵ value (0.05×10^{-2}) that is appropriate for discriminating convergence of the relatively large f changes accompanying graft tip perturbations. The specific normalization employed was to scale the design variable perturbations in inverse proportion to the magnitudes of the corresponding terms of the initial sensitivity vector. For the typical iteration sequence shown in Figure 11 (d = 8 mm, ∂ = 1 mm, $\epsilon = 0.05 \times 10^{-2}$), the ratio of these initial sensitivities

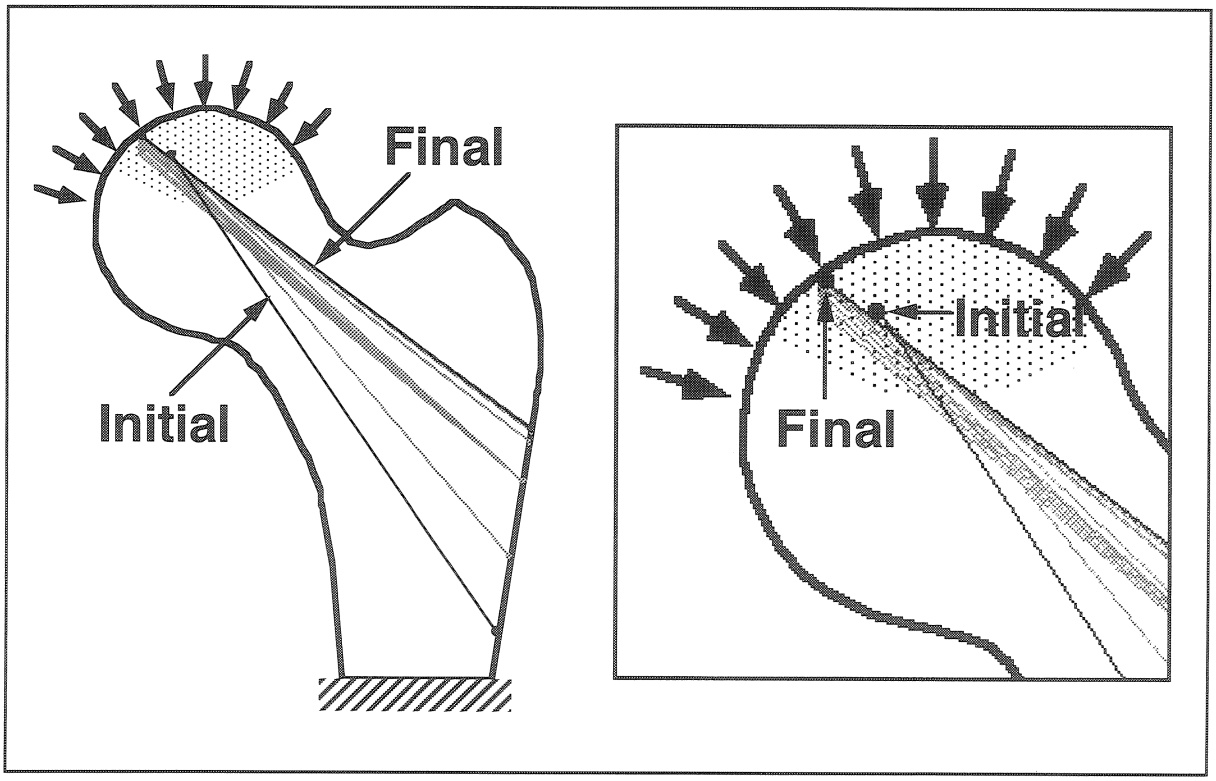

Figure 11. Graft repositioning history during movement toward simultaneously optimal tip and root positions (graft diameter: 8mm)

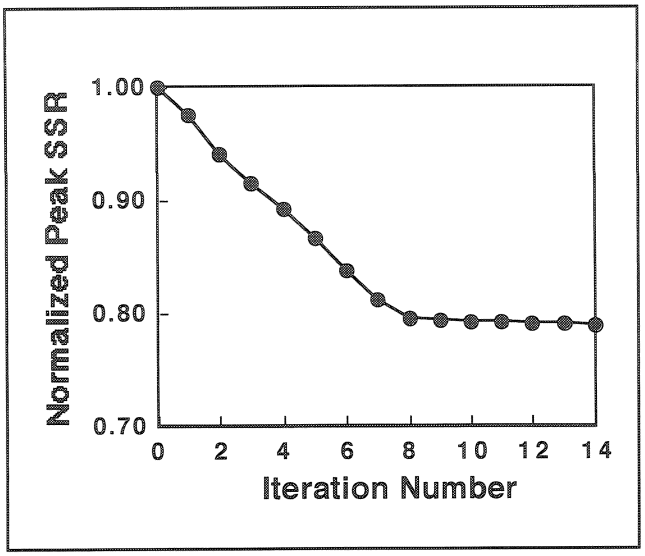

Figure 12. Peak SSR history during simultaneous optimization of graft tip and root position.

for P_{1y} : P_{2x} : P_{2y} was 0.06 : 0.15 : 1.0. In this instance, simultaneous $\mathbf{P}_1$ and $\mathbf{P}_2$ convergence was obtained after 14 iterations, at an intuitively reasonable graft configuration whose peak SSR was approximately 21% below that existing initially (Figure 12).

DISCUSSION

The present exploratory study represents, to our knowledge, a pioneering application of formal mathematical optimization to the problem of attaining maximal structural efficacy in an orthopaedic surgical procedure. While our immediate focus was obtaining mechanically well-positioned fibular grafts for femoral head osteonecrosis, it should be evident that analogous approaches can certainly be invoked to identify construct mechanical optimality for a wide range of other bone and joint surgical procedures. Computer-based surgical planning has become a reality in the last several years[5], although to date actual mechanical stress data have been used in relatively few applications, and only for informal guidance. Clearly, however, the musculoskeletal system's scientific information base will continue to develop, and computational capabilities will continue to advance. For the not-so-distant future of orthopaedic practice, there is no essential conceptual barrier to formal pre-surgical identification of mechanically optimal constructs on a routine basis.

In the interest of efficiently studying optimization algorithm performance per se, it was convenient to introduce several geometrical and material simplifications: planar geometry, regionally homogeneous material properties, perfect graft incorporation, uniformly distributed articular surface loading, and rigid distal support. However, none of these simplifications impacts substantively on the validity of the optimization procedure here demonstrated. In each instance, appreciably more realistic (albeit complex) treatments are well within the present state of the art[7], and fully automated remeshing for the corresponding three-dimensional grafting problem is now technically feasible (Figure 13).

The direct search procedure implemented in the present study is among the more intuitively straightforward of optimization strategies, but it is by no means the most theoretically elegant or computationally efficient[1]. While the optimization process is fully automated, the demonstrated existence of multiple local optima implies that an element of informed human judgement is required to avoid starting with a graft position that will migrate to one of the non-clinically-useful local optima. Combinations of clinical experience[4,9], conventional finite element models[3], and objective function mappings (Figure 6) provide a sound basis for making that judgement.

ACKNOWLEDGMENTS

Financial assistance was provided by NIH Grant AR-35788 and by an Overseas Fellowship Graft from the Educational Ministry of Japan. Dr. M.J. Rudert, Dr. R.A. Brand, and Mr. D.R. Pedersen provided valuable advice and/or technical assistance during conduct of the study.

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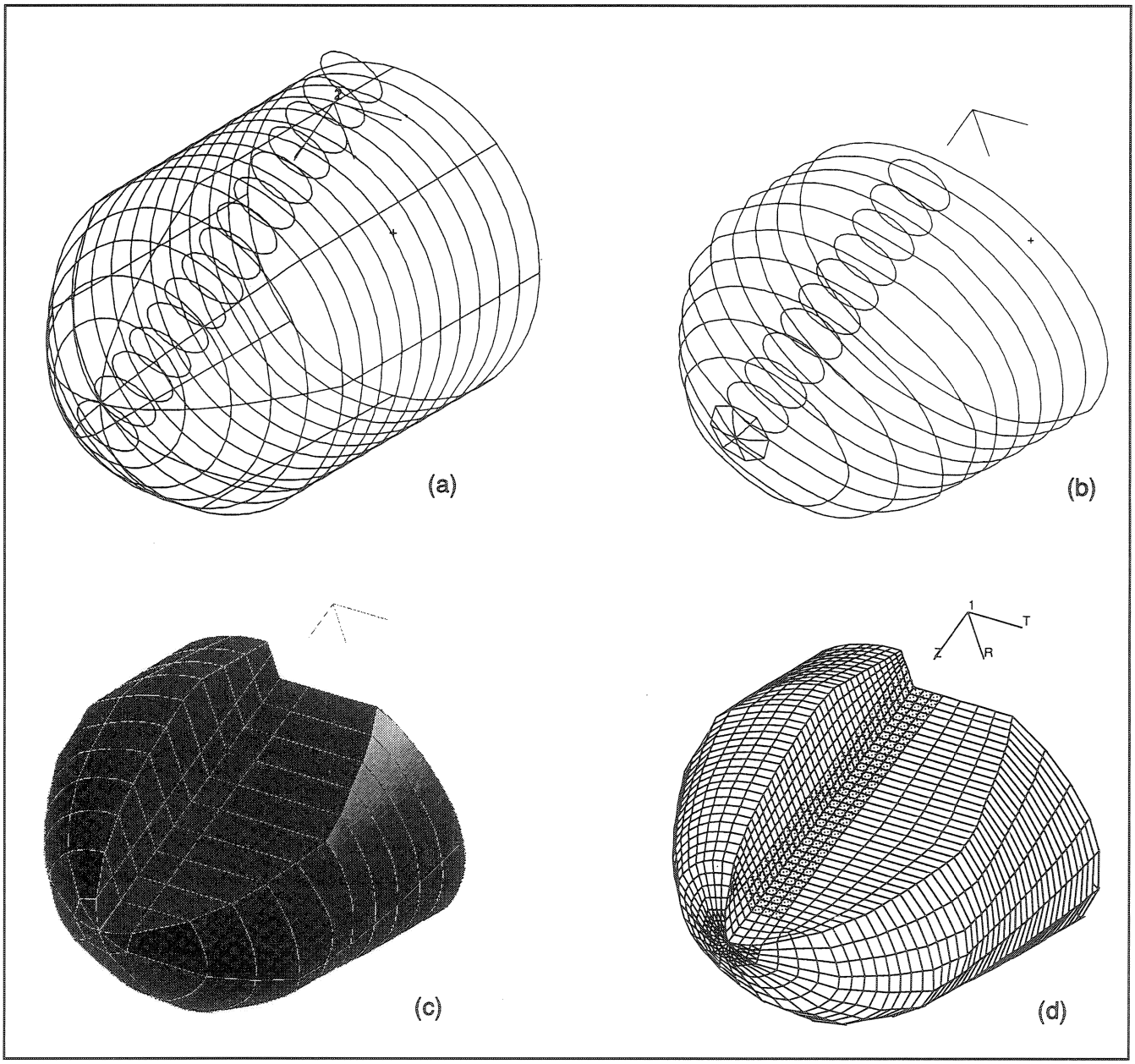

Figure 13. Process of fully automated remeshing for corresponding three-dimensional grafting problem (a) Original rendering curves (b) Rendering curves based on graft coordinate system (c) Solid model (d) Finite element mesh.

BASIC SCIENCE AND CONSERVATIVE CARE OF PATELLOFEMORAL DYSFUNCTION: A REVIEW OF THE LITERATURE

James M. Banovetz, M.D., Ph.D.
Sheila L. Banovetz, M.S., P.T.
John P. Albright, M.D.

INTRODUCTION

As with all other aspects of orthopaedics, our understanding of patellofemoral problems has grown gradually over time and continues to evolve. This paper reviews the development of our knowledge and consequent treatment of patellofemoral disorders, both to understand them better and to get a perspective on the progress that has been made. As is often the case, the basic science is well ahead of the clinical treatment, and closing this gap is one of the goals of clinical research. In reviewing patellofemoral basic and clinical science research, it is important to return to principles of anatomy and kinesiology. Muscular length, strength, endurance and timing are all critically important to this problem.

BASIC SCIENCE—QUADRICEPS FUNCTION

The basic science of patellofemoral dysfunction (PFD) began at the University of Iowa in 1951 with an article by Wheatly and Jahnke[59]. They did an electromyographic study of the thigh and hip muscles and made a number of pertinent discoveries. They used essentially isokinetic movements and found that the vasti fire later than the rectus femoris (RF) and tensor fascia lata (TFL) in active extension (i.e. they begin firing as the subject approaches full extension). Later studies by Brewerton found equal activity of all quadriceps components throughout the range of knee motion, but these used maximal isometric contractions at various flexion angles, which is a very different and less physiologic task[7]. Wheatly and Jahnke also demonstrated that the TFL is a knee extensor (when the knee is near extension— it applies a flexion moment as the knee flexes) and a hip internal rotator[59].

The vastus medialis is divided into two distinct parts—the longus (VML) originating from the shaft of the femur and the obliquus (VMO) originating from the adductor magnus tendon. Hallen and Lindahl also used isometric knee extension to study the function of the VMO fibers in 1967 and found no differences in onset or volume of EMG in varied degrees of flexion, nor did they find subjective weakness or decreased range of motion (ROM) with anesthesia of the VMO[22]. These results are explained beautifully by the anatomic study of Lieb and Perry in which they found that the primary function of the VMO is to counteract lateral pull on the patella by the rest of the quadriceps, rather than contributing to the extension moment[40]. They also found that the last 15 degrees of extension require a 60% increase in extension force. It is in the approach to full extension that the VMO is most needed to counteract the lateral pull of the rest of the quadriceps. Not only is the lateral quadriceps moment at its maximum, but the femoral groove also provides progressively less restraint to lateral patellar motion at this end of the range. Lieb and Perry also did an isometric EMG study in 1971 and found that the VMO had twice the firing rate of the rest of the quads but no relative variation with the degree of flexion; results similar to Hallen and Lindahl[41]. Bose et al. noted that the VMO arises from the adductor magnus tendon, which is relevant to its rehabilitation, as will be discussed later[5].

BASIC SCIENCE—ANATOMIC VARIATION

Although certain types of anatomic variations, such as ITB tightness are more common in people with PFD, there is not a one to one correlation with symptoms[49]. Fairbank et al. studied 446 teenagers and found that there was no difference in joint mobility, Q angle, valgus, or femoral anteversion between normal controls and patients with anterior knee pain, but involvement in athletic activities was highly correlated[12]. Fox looked for VMO hypoplasia in patients presenting for complaints not relating to the knee and found that by his circumference criteria, 42% had VMO hypoplasia[14]. The operative literature certainly has shown success in alleviating patellar instability by tibial tubercle transfer and correction of Q angles. Correcting muscle length problems through rehabilitation protocols has also met with some success in PFD treatment, although this has not been well documented.

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BASIC SCIENCE—VMO FUNCTION

The function of the VMO and its importance near terminal extension was further reinforced by a series of studies in the 1970s beginning with Basmajian et al., who did both open and closed chain extension testing (see Table 1 for an explanation of these terms) and found similar EMG patterns for the RF, VMO, and vastus lateralis (VL) except that the VMO firing increased at terminal extension[2]. Jackson and Merrifield found that all three muscles increased firing with increasing weight and increasing degrees of extension in an open chain paradigm[31]. Francis and Scott trained twenty subjects for eight weeks in open chain knee extension. Ten were prevented from extending the last 15 degrees and ten were allowed full extension. The ten who were prevented from full extension were found not to have increased their thigh circumference in the region of the VMO, but had the same increases higher up the thigh as the group that was trained to full extension, suggesting that terminal extension is needed for VMO hypertrophy[15]. A good review of these early studies was done by Speakman and Weisberg[56].

As research in this area progressed, the increasing awareness of patellofemoral dysfunction led to more clinically relevant studies on the electrophysiology of this problem. Reynolds et al. studied twenty women doing

TABLE 1
STIMULUS FACTORS AFFECTING KNEE EXTENSOR MECHANISM AND MUSCLE CONTRACTIONS

Hip Position:
- Flexion/Extension affects Rectus Femoris.
- Abduction/Adduction affects Tensor Fascia Lata (TFL) and Vastus Medialis Obliquus (VMO).
- Internal/External Rotation affects VMO, TFL, RF, and orientation of femoral groove.

Foot/Leg Position:
- Internal rotation of tibia medializes pull through the extensor mechanism.
- External rotation of tibia lateralizes pull through the extensor mechanism.

Status of Body:
- Weightbearing may affect contraction patterns.
- Need for balance may affect contraction patterns.
- Open chain exercises (i.e. leg extensions) are intended to isolate knee extension but have high patellofemoral contact pressures and do not control motion at adjacent joints.
- Closed chain exercises (foot fixed, as in squat or leg press) are more physiologic.

Associated Contractions:
- Adductor Magnus affects VMO.
- Gluteus Maximus affects TFL through its insertion on the Iliotibial Band.
- Hip Rotators affect position of the femoral groove.
- Hamstring and popliteus contractions affect the position of the tibial tubercle.
- Hamstring and Gastrocnemius contractions affect extensor load.

Type of Motion:
- Isometric stimulates all components maximally with no change in length, and pattern may be determined by knee flexion angle.
- Isotonic causes shortening of muscles with a stable load, and may evoke different patterns through the range.
- Isokinetic causes shortening at a constant rate (and again may evoke different patterns throughout the range) with load determined by the patient.

short squats and found VMO and VL EMG had an equal amplitude of activity[50]. Mariani and Caruso found VMO activity only in the terminal 30 degrees of extension and VL activity primarily in this range[44]. They found that in patients with PFD, the VMO was less active than normal controls, and this was true for both the symptomatic and the non-symptomatic sides. They also found that VMO firing increased after Elmslie-Trillat procedures, although it is not clear whether this was due to the surgery or was a result of the postoperative rehabilitation program. Moller et al. studied twenty eight patients with unilateral PFD using isometric extension at various flexion angles and found no difference side to side in VMO/VL EMG ratio, but generally decreased firing of the symptomatic side[46]. In a fascinating clinical report, Bohannon described a patient who dislocated her patella every time she flexed her knee[4]. Electrical stimulation of her VMO during ROM prevented this dislocation. We performed this test recently on a patient who dislocated in flexion due to an extremely lateralized quadriceps mechanism, and although we were unable to prevent the dislocation, the stimulator did enable her to flex about 30 degrees further before dislocating in a series of trials with the stimulator on or off.

Recent studies have looked more specifically at properties of the EMG output in normal and PFD patients. Souza and Gross compared seven controls with nine patients and found decreased VMO/VL ratios in both knees of PFD patients performing functional activities (agreeing with the findings of the Mariani and Moller papers mentioned above)[55]. Voight and Wieder compared reflex response times of VMO and VL EMG in forty-one normal subjects and sixteen subjects with PFD and found that the VL fires sooner in PFD to a patellar tendon reflex tap[58]. In contrast to the above reports, Boucher et al. studied normal subjects (Q angle = 8.25) vs. those with patellofemoral pain (Q angle = 21.05) using isometric torque at 15, 30, and 90 degrees and found no difference in the EMG of VMO, VML, or VL except that the worst five (out of eighteen patients) had a decreased VMO/VL ratio[6]. MacIntyre and Robertson found similar results in women runners[42]. Karst and Willett compared the "onset" of EMG of VMO and VL with reflex and voluntary tasks and found no difference in either twenty-four normal or twenty-four patellofemoral pain syndrome knees[34]. Tasks included a reflex tap, open chain extension, and stepping up an 8 cm step.

BASIC SCIENCE—QUADRICEPS DEVELOPMENT

The basic science work on quadriceps rehabilitation for PFD has centered on techniques that avoid exacerbating symptoms. Later studies have focused on the VMO as its relative importance has been recognized. Some studies have not differentiated between the longus and obliquus components of the vastus medialis, so their significance is unclear. Knight and colleagues found more EMG activity of the VM as a whole with open chain extension than with SLR in twenty subjects[36]. Open chain knee extension has lost favor in the rehabilitation literature, however, as it has been noted to cause increased symptoms, probably due to increased joint reaction forces[28]. Brownstein et al. found that maximum EMG activity for the quadriceps as a whole was elicited isometrically at 50 degrees for men and 70 for women[8]. Wild et al. studied twenty-six patients with patellar instability and found that straight leg raising (SLR) in full extension stimulates more quadriceps EMG than SLR with a 10-20 degree extensor lag[61]. They also found that adding five pounds to the ankle or rotating the hip did not change the EMG patterns significantly. Soderberg and Cook studied forty subjects and found that quadriceps setting (QS) exercises caused more firing in the vastus medialis (portion not specified), biceps femoris, and gluteus medius, whereas SLR exercises stimulated more RF activity[54]. Karst and Jewett studied twelve subjects doing QS vs. SLR vs. SLR with lateral rotation vs. SLR with adduction and found that QS was best for increasing EMG, whereas adduction or lateral rotation did not increase the VMO component compared to the others[33]. Moller et al. found that isometric quad sets caused increased activity in both the VL and the VMO in normal subjects, patellar subluxators, and those with anterior pain[47]. They made the interesting observation that subluxators decreased their patellofemoral congruence angles with active quadriceps contractions, whereas normal controls and those with isolated anterior knee pain did not.

BASIC SCIENCE—TRAINING THE VMO

With increasing awareness of the importance of the VMO, more studies have focused on attempts to selectively train this muscle. Leveau and Rogers demonstrated in 1980 that using biofeedback and isometric open chain extension at near full extension (at 25% of a maximal contraction), the VMO/VL firing ratio could be increased in ten normal subjects[39]. Grabiner, Koh and Miller studied fatigability in subjects doing isometric or short arc quad exercises from 30 degrees to full extension and found no difference between the VMO and VL in either paradigm, suggesting that these do not selectively work either component[18]. Hanten and Schulthies in a study of twenty-five subjects found that hip adduction exercises activated VMO more than VL[23]. Ingersoll and Knight divided thirty normal women into three categories: VMO/VL biofeedback, daily adjustable progressive resistive exercise (DAPRE), and controls[29]. They measured congruence, rotation, and sulcus angle, and found that DAPRE actually lateralized tracking, while biofeedback medialized it. This suggests that inappropriate rehabilitation can actually make patellofemoral problems worse. Hodges and Rich-

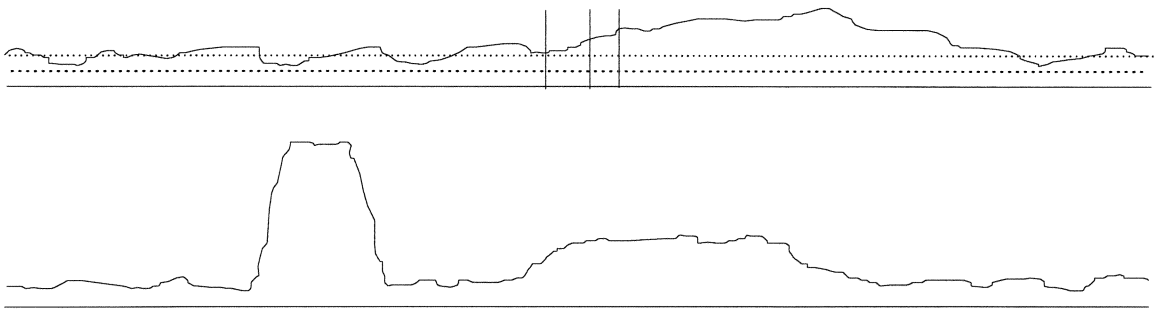
ardson tested VMO and VL EMG in twenty women at 60 degrees of hip and knee flexion with and without weightbearing and with and without hip adduction, at contractions of 100, 50, and 15% of maximum voluntary effort (MVE)[26]. Weightbearing elicited a higher VMO/VL ratio than non-weightbearing, and adduction increased it further. It was not made clear how the data was collected. Although electrical stimulation has been shown to be useful in general quad strengthening and is often used clinically to rehabilitate the VMO, there are no studies in the literature on its use with this muscle[21].

It is difficult to synthesize the often conflicting findings of the above studies on extensor mechanism dynamics, but this is largely due to a lack of uniform approach in study design. The measured variables and the provocative task used both vary considerably and have dramatic effects on the results. The significant factor in patellofemoral dysfunction is the application of force by each element of the system over the course of functional movements. Functional movements are difficult to replicate, but must be used to insure the value of the data. It is virtually impossible to measure the applied force of a muscle, and instead we measure EMG. Unfortunately, there is no simple, direct correlation between EMG and the actual force curve of the muscle.

There are a number of important points to understand in evaluating the above studies and their sometimes contradictory findings. The first is that muscles do not turn on and off like an electrical switch, but rather have baseline activity which can increase or decrease. When discussing the "timing" of muscle EMG, some method has to be chosen to determine when the muscle activity has changed from the baseline, signifying that the muscle has turned "on" or "off." The method used will affect the results obtained. Muscle activity varies dramatically, with long or short bursts, high or low firing rates, and gradual or rapid changes in firing rate. This can make it difficult to compare EMG obtained from different muscles or in different circumstances. Differences in the task used to stimulate a contraction, as shown in Table 1, also affect the resultant EMG.

Other segments of the limb that affect the mechanics at the knee have not been addressed with respect to patellofemoral dysfunction. Although the primary focus has always been the patellar component, the orientation of the femur also effects the axis of the joint. The activity of hip muscles, therefore, may have an impact on patellofemoral dysfunction (see Table 1), and although suggested by clinical anecdote, this remains to be proven. The same is

FIGURE 1
Characteristics of EMG Data--Simulated Rectified EMG Signals



A: Dotted line represents baseline activity. Vertical lines represent potential choices for onset of a contraction. It is a difficult problem to define a contraction and then to define its beginning and end.

B: Two separate contractions differing in onset, peak firing rate, and duration. These would be difficult to compare

true for muscles that affect tibial position. Foot mechanics are also mentioned frequently in the rehabilitation literature, but again, although this relationship is logical, no significant scientific work has been done to establish the relationship.

As shown in Table 1, there are virtually infinite combinations of factors that influence muscle activity and joint mechanics. There is very little work presented in the papers above establishing activity patterns during functional activities such as running, squatting, or stair climbing. These studies need to be done in both normal and symptomatic subjects, both when well rested and fatigued. Improved understanding of pathologic patterns is critical to developing and testing rehabilitation protocols.

In spite of the difficulties with the research methodology, there are a number of salient points that can be drawn from the literature. Knee extension is a complex task involving a variable pattern of quadriceps activation. The VL, RF, and TFL all produce a lateral force moment on the patella. The VMO does not extend the knee, but rather counteracts this lateral force moment. The force required of the quadriceps mechanism increases as the knee nears full extension, and it is here where the lateral pull is the greatest and thus the VMO is most important. Symptoms are not strictly correlated with anatomic variables, but seem to require acute or chronic, overuse trauma to the knee. There may be some decrease in VMO and or VL firing in symptomatic patients.

CLINICAL REHABILITATION STUDIES

Patellofemoral dysfunction was first carefully described as a clinical entity by Hughston in 1968, although he called it "subclinical patellar subluxation that had previously been unrecognized."[27] The clinical literature, like that from basic science, has a plethora of overlapping terms for various anterior knee pain problems. Table 2 lists the relevant rehabilitation articles with their diagnostic terms and treatment protocols. It is clear from the table that there is no consistency to the protocols, and no trend over time. Only seven of them list results, but none of these are carefully, objectively documented. Furthermore, none of them are randomized between differing protocols. None of the studies is carefully grounded in the relevant basic science presented above. Beckman, Craig and Lehman presented a standardized clinical approach to this problem in their 1989 article[3].

TABLE 2. Rehabilitation Protocols by First Author and Year of Publication

Author	Year	Diag.	NSAID	Brace	SLR	Isomet.	VMO	Ham Str	Ret str	Other	Results
			/Rest	/Ice	/AROM	/Isoton		ITB Str	Foot cor		
Hughston[27]	1968	subluxation			X						
Gruber[20]	1979	chondromal	X/X			X/X					
Steadman[57]	1979	PF Problem	X	/X	/X		NS	X	X/X		
DeHaven[10]	1979	chondromal				X					X
Henry[24]	1979	subluxation			X					hip fl/ab	X
Insall[30]	1980	malalign	X/X	X	X						
Paulos[48]	1980	malalign				X/X					
Kettlekamp[35]	1981	malalign	X			X					
Malek[43]	1981	PF pain			X	X					
Abdenour[1]	1983	PF rehab				X					
Fulkerson[16]	1983	PF pain	X		X						X
Wise[62]	1984	PF pain				X/X	biofeed				X
Grana[19]	1985	ext mec d/o	X/X		X	/X			/X		
Fisher[13]	1986	PF pain	X/X	X	X						
Kramer[38]	1986	malalign							X		
McConnell[45]	1986	chondromal					adduct		X/X	taping	X
Brunet[9]	1989	PF rehab			X	X		X		QS	
Henry[25]	1989	subluxation	/X	X	X			X		*	
Whitelaw[60]	1989	an kn pain				X/X					X
Fulkerson[17]	1990	d/o PF alig	X/X	X	X	X/X	NS	X/X	X/X	taping	
Woodall[63]	1990	PF rehab			X	/X	NS				
Shelton[52]	1991	PF dysfunc					NS		X	taping	
Shelton[53]	1992	PF dysfunc					NS		X	taping	
Doucette[11]	1992	lat com syn	X	X/X	X	X/X	NM stim	X	/X	taping	X
Ruffin[51]	1993	PF syn					NS			QS	
Kannus[32]	1994	PF pn syn	X/X			X					
Totals	26		9/7	4/1	11/1	10/7		4/1	6/4		7

chondromal = chondromalacia, PF = patellofemoral, malalign = malalignment, rehab = rehabilitation, ext mec d/o = extensor mechansim disorder, an kn = anterior knee, d/o PF align = disorders of patellofemoral alignment, dysfunc = dysfunction, lat com syn = lateral compression syndrome, syn = syndrome, pn = pain, NS = not specified, biofeed = biofeedback, adduct = adduction exercises, NM stim = neuromuscular stimulation, hip fl/ab = hip flexion/abduction exercises, QS = quadriceps setting exercises, * = QS, Abductor/Adductor strengthening, heel cord stretching.

Hughston's ground-breaking paper was the first to identify the patellofemoral joint as a source of knee problems other than the frank subluxation/dislocation problems that had previously been thought to occur only in overweight, out of shape, knock-kneed females. His population of sixty patients with patellofemoral problems included thirty-five athletes, of which twenty-five were males. Seventeen of the sixty patients had no acute trauma, and the peak incidence was in high school aged athletes. Symptoms included pain, swelling, giving way, locking-popping, grating, and "out of place" in descending order of occurrence. Hughston first described lateral tracking and passive lateral subluxation as important physical exam signs. He also reported tenderness, grating (crepitance), VMO "dystrophy," external tibial torsion, and lateral insertion of the patellar tendon. He recommends AP, lateral and infrapatellar radiographs, but states "cursory physical examination and dependence only on roentgenograms will not lead to an accurate diagnosis." The most common findings are patella alta, lateral tilt and/or subluxation, and a deficient lateral femoral condyle. He does note that radiographs may be completely normal.

Hughston recommended conservative care for all patients except for those with "acute and severe osteochondral fracture or with a severe patellofemoral osteoarthritis." Acute dislocations were casted for six weeks and then started on his rehabilitation protocol, which consisted of straight leg raises but avoidance of resisted knee range of motion, particularly if it was painful. He also recommended activity limitation as needed and laterally padded knee supports. He noted that patients who became asymptomatic on their exercise regimen would often have a return of symptoms when they ceased their exercises, but would again obtain relief with resumption of the program. He goes on to describe surgical care as well, which is outside the scope of this review.

Subsequent articles on this subject have added to Hughston's protocol. Many authors have advocated nonsteroidal medications and various forms of general quadriceps strengthening. Steadman wrote a brief note in 1979 in which he added stretching of the lateral retinaculum and hamstring and gastrocnemius muscles, as well as correction of foot posture[57]. He mentions diminished VMO tone, but does not specify a program to rehabilitate it. DeHaven, Dolan and Mayer were the first group to document results with a conservative program of symptomatic treatment and isometrics[10]. They found that sixty-six out of 100 patients returned to "unrestricted athletics" by eleven months, but of these only forty six were pain free. Henry and Crosland treated 145 patients with six weeks of straight leg raises, hip flexion, and hip abduction exercises and found that 76% improved[24]. Fulkerson treated seventy-eight patients with straight leg raises and found that 74% did well[16].

Wise, Fieberg and Kates introduced biofeedback for VMO rehabilitation into the literature in 1984 and had good results in a small series of six patients[62]. Perhaps the clinical rehabilitation study that is the most relevant to basic science and most specifically oriented towards pathophysiology is that published by Jenny McConnell[45]. She had a study group of thirty-five patients with a mean duration of symptoms of 4.9 years. Twenty-seven of the patients had had previous physiotherapy, primarily quad strengthening, and four had had surgery. She designed a specific protocol for each patient to address their abnormal exam findings. Treatment included ITB and lateral retinacular stretching, McConnell taping, isometric hip adduction, and functional VMO strengthening. She found that at least 83% of her patients had no pain and an additional 8.5% had decreased pain after only 2-8 treatment sessions. There are a number of significant weaknesses in her study. She does not break down her population by specific pathology, and her follow-up was short, with only fourteen of the subjects followed out six months. She reported that all were pain free and back to their sports activities, but there is no specific data to prove this.

McConnell taping, a technique widely used in clinical practice with good anecdotal results, was recently tested in a small prospective study which failed to demonstrate significant effectiveness[37]. It may require a larger study to do so, and it would also be worthwhile to document if it helps alleviate symptoms during the rehabilitation process.

Two subsequent studies have also reported results. Whitelaw and colleagues treated eighty-five patients with a general quadriceps program and found that 68% were better at ten months, but only 57% were able to increase their athletic activity from their status while injured[60]. Doucette and Goble followed fifty-six knees in twenty-nine subjects through a rehabilitation program including quadriceps exercises, taping, and stretching[11]. They found that 84% were pain free at eight weeks. Again, these results are very general and completely undocumented.

The weaknesses in the rehabilitation literature are fairly evident. It is difficult to know which techniques are most effective since there are no studies comparing them. Those articles which present results tend to be short term and do not rigorously explore the patients' functional status pre- or post-treatment. Although no treatments have been rigorously proven effective, the important concepts promoted by the literature as a whole are as follows. Rest, nonsteroidals, and local therapy should be used to relieve symptoms. Rehabilitation and athletic activities should be restricted to those which do not elicit pain. It is important to identify abnormalities throughout

the extremity and treat them specifically. Tight structures, such as the ITB, rectus femoris, hamstrings, or lateral retinaculum should be stretched. The quadriceps should be strengthened, with particular attention to the VMO. This can be done by associating knee extension or straight leg raising with adduction of the hip. Biofeedback can be used to help train the VMO to be active during functional activities, such as stair climbing. Adherence to a rehabilitation protocol is necessary for improvement, and a maintenance program may be needed to keep symptoms under control.

Patellofemoral dysfunction is a complex problem which can often frustrate the orthopedic clinician. The techniques of electromyography and kinesiology are not familiar, and the inconsistency of their application in these studies makes their review difficult. The principles of patellofemoral pathomechanics have been at least partially worked out, although they have to be drawn carefully from the literature as a whole. The rehabilitation studies have not followed closely the basic science developments, and although various programs promote common principles, we still await careful documentation of their effectiveness. It is to be hoped that future outcome studies will be done along these lines, with comparison of techniques and long-term functional results of therapy.

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IMAGING SCAPHOID FRACTURES AND NONUNIONS: FAMILIAR METHODS AND NEWER TRENDS

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INTRODUCTION

The hand and wrist have been the subject of medical imaging since Wilhelm Conrad Roentgen published an image of his wife's hand in 1896[1]. Since then there have been great advances in the number and quality of techniques used to image the hand and wrist. This discussion reviews some of the more useful techniques currently available to the treating surgeon for imaging scaphoid fractures and nonunions, including our most recent experience with helical computed tomography (CT).

CONVENTIONAL RADIOGRAPHY

Plain radiographs remain the single most important imaging modality for the hand and wrist[2]. When an acute scaphoid fracture is strongly suspected on clinical grounds and routine posteroanterior (PA) and lateral projections are normal, additional views are justified. These views are designed to align the plane of the fracture with the central X-ray beam. Most fractures are transverse and occur in a plane perpendicular to the longitudinal axis of the scaphoid. Since the scaphoid is normally flexed approximately 40-60 degrees when the hand is in the neutral position, optimal beam-to-fracture alignment can only be obtained by angling the x-ray tube or dorsiflexing the scaphoid.

Stecher[3] recommended that the hand be placed prone on the x-ray cassette with the fist clenched. This causes the scaphoid to dorsiflex. Placing the wrist in ulnar deviation may further improve visualization of the scaphoid because the scaphoid normally dorsiflexes as the wrist moves into ulnar deviation. However ulnar deviation may cause unwanted distraction of the fracture fragments. In 1949, Bridgman[4] proposed a technique in which the hand, in ulnar deviation, rests on a 17-degree angled board. As a result, the central ray projects perpendicular to the waist of the scaphoid and parallel to the fracture plane. A similar radiograph is obtained if the hand is placed flat on the cassette and the central ray is projected 17 degrees cephalad (Figure 1). This angle of approach may be changed to 20 or even 30 degrees when a fracture is strongly suspected and still not visualized. Scaphoid fractures are not usually visible in the lateral projection. This, however, is an important view because it allows the examiner to evaluate carpal alignment and to detect the presence of carpal instability. This may be especially important when evaluating patients with scaphoid nonunions.

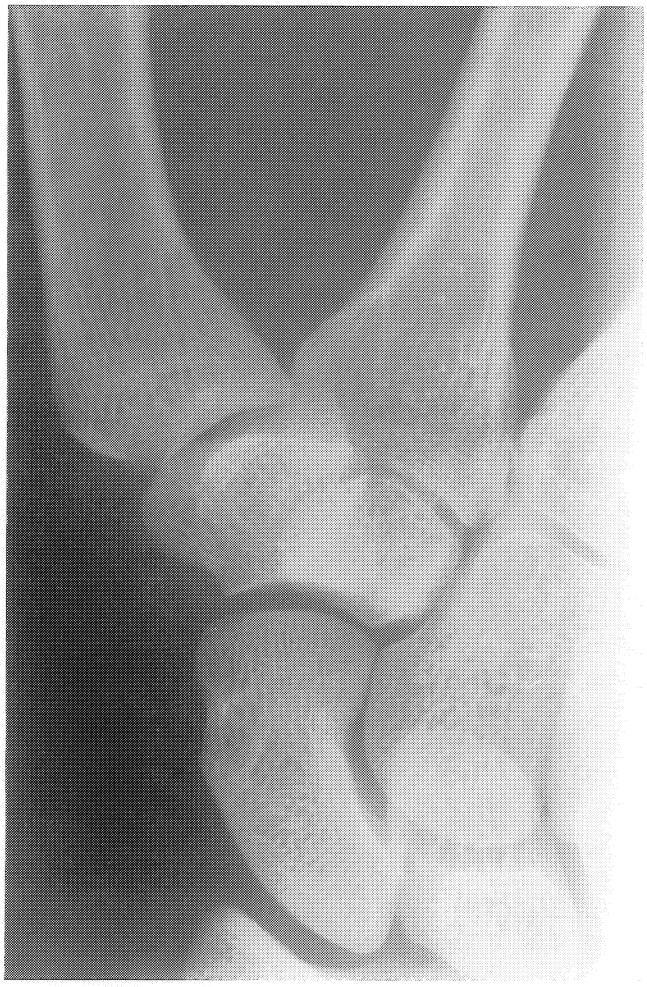

Figure 1. Scaphoid view according to the technique of Bridgman. Note that the body is well demonstrated, particularly the waist.

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PLAIN FILM TOMOGRAPHY

Plain film tomography may be of help in the detection of small, occult, or very early fractures. It is also helpful to determine the degree of healing in cases where fracture lucency persists on plain films. Plain film tomography is an x-ray technique that blurs out the shadows of superimposed structures to show the principal structures being examined more clearly[5]. It does not improve the sharpness of any part of a radiographic image, but rather, it is a process of controlled blurring that leaves some parts of the image less blurred than others. The essential components are an x-ray tube, a film, and a connecting rod that revolves around a fixed fulcrum. When the tube moves in one direction, the film moves in the opposite direction (Figure 2). The plane of interest within the patient is positioned at the level of the fulcrum, and it is the only plane that remains in sharp focus. The simplest type of tomography involves linear motion of the x-ray tube and cassette. However, other motions, such as trispiral or hypocycloidal, are used to improve the tomographic image. When imaging the scaphoid, we typically obtain contiguous 3 mm slices in the PA and lateral projections.

Although plain film tomography can aid in the diagnosis of acute scaphoid fractures and the evaluation of scaphoid nonunions, the technique does have disadvantages. Although the technique can be performed on the casted wrist, image quality is better with the wrist uncasted. The time required to obtain multiple slices can be significant, so that patients often have to remain in a somewhat uncomfortable position for a long time. Finally, the multiple x-ray images taken during tomography result in increased radiation exposure compared with other imaging methods.

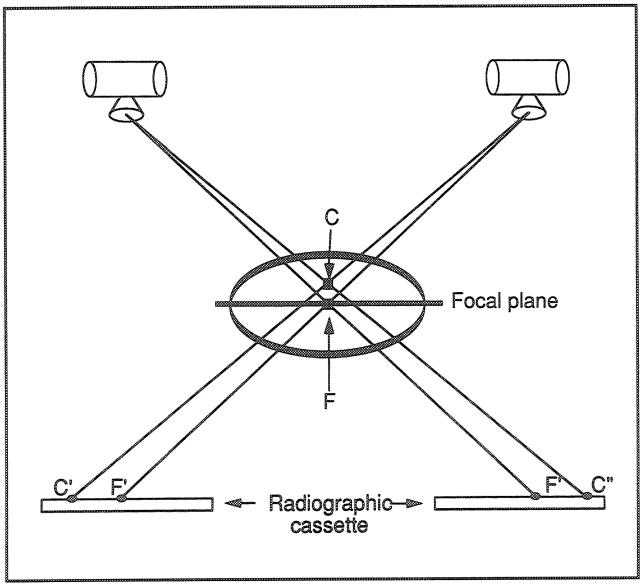

Figure 2. Principles of tomography. The x-ray tube and the radiographic cassette move in opposite directions. Objects within the focal plane (F) remain at the same position on the film (F') and are sharp, whereas objects outside of the focal plane (C) are blurred on the final image (C').

CONVENTIONAL COMPUTED TOMOGRAPHY

CT is a technique where x-rays are projected through the wrist while the x-ray source rotates around the patient. The data is fed into a computer, which then generates a tomographic image. CT is a very valuable method for imaging the scaphoid bone. High quality CT images provide important information regarding fracture plane orientation and location, fracture fragment position, bone resorption and secondary degenerative changes in adjacent joints. Unlike conventional tomography, CT works equally well with casted and uncasted patients. A limitation of CT is that fractures in the plane of the x-ray beam can be difficult to detect. Therefore, optimal fracture detection requires attention to patient positioning when setting up the scan.

A large number of wrist positions have been described for patients undergoing scaphoid CT. Bush et al.[6] and Sanders[7] reported a technique in which the arm is positioned so that the slices are made in a sagittal plane and parallel to the long axis of the scaphoid. Biondetti et al.[8] used a lucite holder to hold the wrist in maximum extension to obtain near-coronal sections of the carpus. Pennes et al.[9] obtained coronal CT images of the wrist without the use of an immobilizing device. This was done by positioning the patient in the prone position with the arm abducted and the elbow flexed at 90 degrees, with minimal flexion of the wrist, and the hand placed vertically so that the palm faced the top of the head. Friedman et al.[10] described a technique in which the wrist was held in a simple immobilizer while scanning the carpus in a variety of planes.

HELICAL COMPUTED TOMOGRAPHY

Conventional CT and helical CT are fundamentally different techniques. Conventional CT scanning involves alternating patient translation and x-ray exposure, whereas helical CT scanning involves simultaneous patient translation and x-ray exposure[11] (Figure 3). The technique is so named because the x-ray can be thought of as tracing a helix or spiral curve on the patient's skin. Helical CT can evaluate occult and complex fractures, assess healing fractures, and evaluate the postsurgical wrist[12].

Both conventional and helical CT can demonstrate scaphoid fractures and nonunions. However, there are advantages of helical CT over conventional CT. Helical CT is faster than conventional CT, not only in the data acquisition phase, but also in the total time to complete the study[13]. This can be a great advantage when working with patients who are in pain, very ill, or victims of trauma. In addition to the total examination time, the speed of data acquisition is important, because this helps to minimize

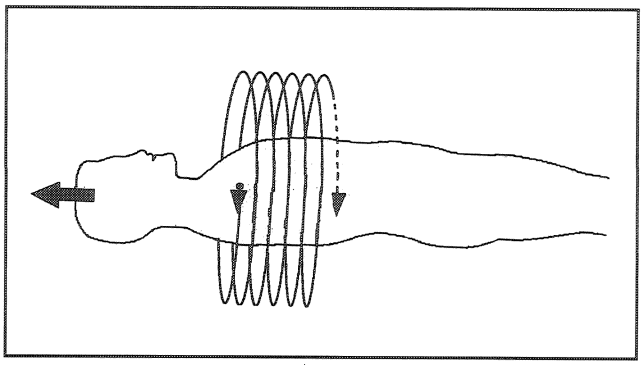

Figure 3. Diagram of helical CT. Helical CT differs from conventional CT in that with helical CT there is simultaneous patient translation and x-ray exposure resulting in a continuous data set.

unintentional intrascan or interscan motion, which can degrade multiplanar and three-dimensional reconstructions. Another advantage of helical CT is that nearly-spaced, overlapping slices can be reconstructed from the basic data set at no additional radiation cost to the patient. This differs from conventional CT where to get closely-spaced slices, radiation dose to the patient increases. Finally, helical CT has the advantage that multiplanar reconstructions, including obique planes, can be generated from the original data. This allows a given fracture to be studied in any plane. The number of scan planes with conventional CT is limited by the ability to position the patient in the gantry.

For both conventional and helical CT it is important to use thin slices (2 mm or less) that are at least contiguous and preferably overlapping. This is necessary as some fractures are not significantly displaced and can be overlooked with thick slices. We typically use 2 mm thick slices obtained every 1 mm.

Figure 4. Sagittal long-axis view by CT for scaphoid nonunion. A. For the sagittal long-axis view, the patient is placed prone on the table with arm overhead and elbow flexed approximately 45 degrees. The wrist is placed in ulnar deviation and the volar surface of the hand rests on the table.

We presently use helical computed tomography to evaluate patients with scaphoid fractures and nonunions. All patients are initially helically scanned with the plane of scan parallel to the sagittal long-axis of the scaphoid. This is done by placing the patient in the prone position on the table with the arm over the head, the elbow flexed to 45 degrees and the volar surface of the hand on the table (Figure 4). The hand is placed in ulnar deviation and the thumb is extended. The scanning plane is parallel to an imaginary line drawn along the central axis of the thumb metacarpal to the tip of the ulnar styloid. A scout image is obtained and if the scaphoid is not parallel to the scanning plane, the wrist is repositioned. Patient position is modified when imaging the casted wrist to align the scan plane with the long axis of the scaphoid.

A second helical data set with the plane of scan parallel to the coronal axis of the scaphoid is then acquired. Using the same scan parameters, the patient remains in the prone position with the arm abducted and the elbow flexed approximately 45 degrees (Figure 5). The forearm is

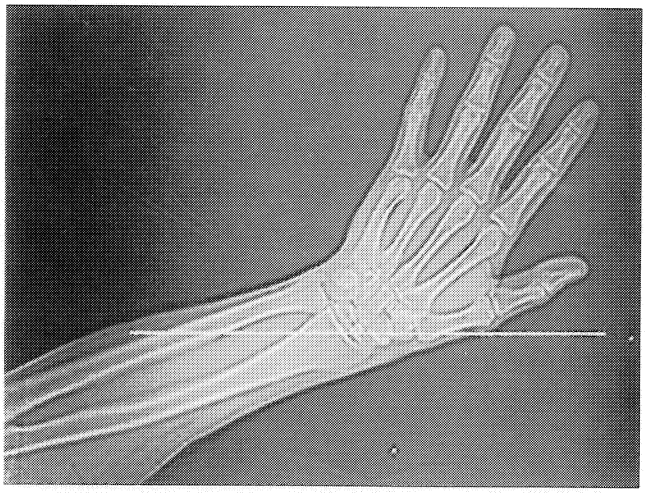

Figure 4B. Scout image from a CT study showing correct position of the wrist for the sagittal long-axis study.

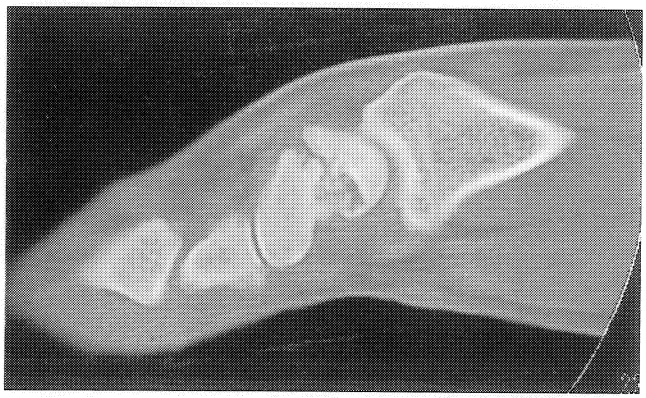

Figure 4C. Sagittal long-axis image from a 22 year old male with a nonunion shows the fracture line with resorption and apex dorsal angulation.

supinated and the wrist is placed in the neutral position so that the palm faces the top of the head. As with the sagittal acquisition, a scout is obtained to confirm proper scanning plane orientation. When indicated, a third oblique sagittal data set acquired with the hand in radial deviation is obtained, similar to the scan in ulnar deviation. Comparison of scans obtained in radial and ulnar deviation allows for an assessment of fracture fragment stability and carpal alignment (Figure 6). We have found that while the sagittal data set can be reformatted to the coronal plane, directly acquired images have better image quality and take less time to acquire than reformatting the sagittal data.

MAGNETIC RESONANCE IMAGING

The first report of magnetic resonance imaging (MRI) of a normal wrist was by Hinshaw et al.[14] in 1977. They did not use a surface coil and were unable to obtain quality images. With the use of surface coils, high-resolution analysis of the wrist by MRI is now possible. MRI is advantageous because it is non-invasive, can obtain images in any plane, is sensitive to changes such as edema, and is free of the streak artifacts seen on some CT examinations[15] (Figure 7). Iameda et al.[16] compared MRI images of scaphoid fractures with those of normal wrists and correlated findings with the healing process of scaphoid fractures. They found that scaphoid fracture lines could be diagnosed as early as two days after injury and continued to be visible for some months longer than on plain radiographs. Low or iso- signal intensity on T1-weighted images and high signal intensity on T2-weighted images of the distal fragment were characteristic of recent scaphoid fractures. They also suggested that the extent of bony union of a scaphoid fracture can be determined by MRI. The distal fragment continues to show a high signal intensity on T2-weighted images up to the time that union is seen on radiographs. Bony union of the distal fragment is indicated by an iso-intense signal on T1- and T2-weighted images. Iameda et al. also noted that the fracture line may remain demonstrable on MRI long after clinical union has occurred and concluded that persistence of a fine line of low signal intensity at a fracture site should not be equated with nonunion.

MRI has been shown to accurately detect avascular necrosis of the carpal bones. In a prospective study, Trumble[17] evaluated the correlation between MRI findings, histology and intraoperative evidence of avascular necrosis of the scaphoid. In this study, the absence or decrease in signal intensity of T1- and T2-weighted MRI images correlated with histologic, fluorescent labeling and intraoperative evidence of avascular necrosis of the proximal pole after a scaphoid waist fracture. Since prognosis for healing in scaphoid fractures depends on whether avascular necrosis is present, MRI can be a useful tool in the evaluation of these patients. Because of its added cost,

Figure 5A. For the coronal long-axis view, the patient remains prone with the arm abducted and elbow flexed approximately 45 degrees and the wrist in supination. The palm faces the top of the head.

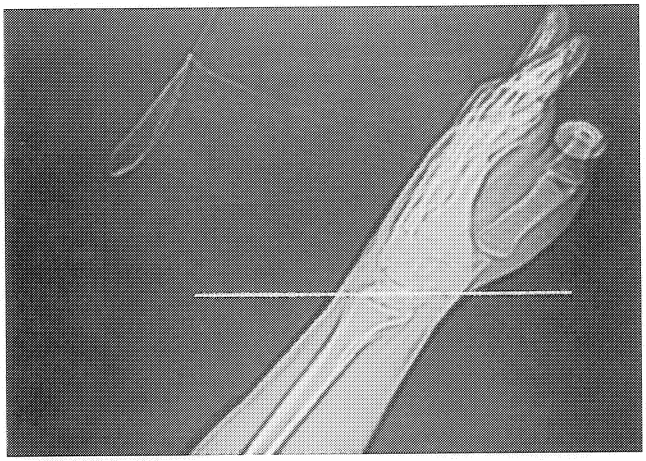

Figure 5B. Scout image demonstrating proper position for the coronal long-axis view.

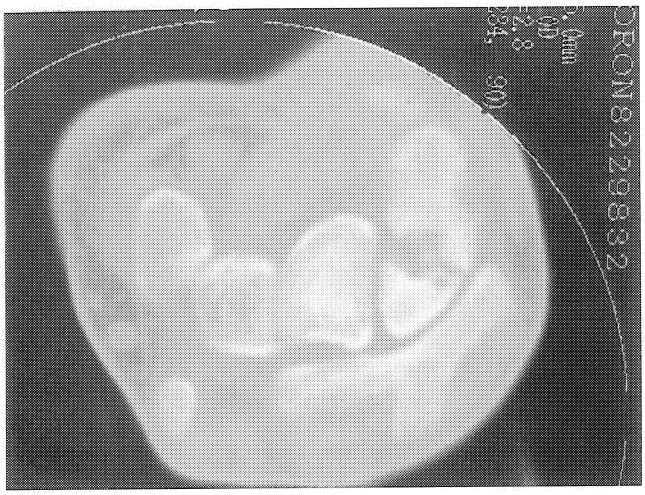

Figure 5C. Coronal long-axis view (same patient as figure 4) demonstrates fracture, resorption, angulation, and sclerosis of the proximal pole.

we do not routinely use MRI to evaluate patients with scaphoid fractures or nonunions. We recommend that the physician rely on the clinical examination and plain films when evaluating patients with suspected acute fractures and conventional tomography or helical CT for patients with suspected or established non-union.

RADIONUCLIDE BONE SCINTIGRAPHY

Plain films and conventional and computed tomography all detect morphological changes of bone, but do not evaluate osteoblastic activity. Radionuclide bone scintigraphy can be used to evaluate the osteoblastic activity of the wrist in patients where a scaphoid fracture is suspected. Early bone scanning has been recommended to increase the accuracy of diagnosis of fractures of the scaphoid[18,19]. In these studies, only the static phase of the bone scan was evaluated. Although the dynamic phase is helpful in determining the degree of overlying secondary hyperemia, the static images are much more reliable in determining the presence of an occult fracture. Tiel-van Buul et al.[20] evaluated the reliability of the dynamic phase of the bone scan alone and found it not to be reliable in the diagnosis of scaphoid fractures.

In a separate study, Tiel-van Buul et al.[21] analyzed two possible strategies for the diagnostic management of clinically suspected scaphoid fractures with initially negative radiographs. The first strategy used repeated plain radiographs up to 6 weeks after trauma. The second strategy employed plain radiographs immediately following injury, followed by three-phase bone scintigraphy 72 hours after trauma if the radiographs were initially negative. Their study suggests that the interobserver agreement as well as the sensitivity of the repeated radiographs is unacceptably low. They maintain that patients with clinically suspected scaphoid fractures and negative initial radiographs should undergo bone scintigraphy rather than repeat radiography.

While bone scans are sensitive for detecting fractures, an inherent disadvantage to bone scintigraphy is a lack of specificity. In particular, it is often difficult to accurately localize an area of intense uptake to a specific carpal bone. Hawkes et al.[22] have devised a system that has a combined display of the X-ray image and isotope bone scan that has an inherent precision of 1 mm. With the advent of these newer technologies, bone scintigraphy may play a larger role in diagnosing future cases of radiographically occult fractures of the scaphoid, but it is uncertain whether these techniques will replace current management of patients with suspected fractures of the scaphoid.

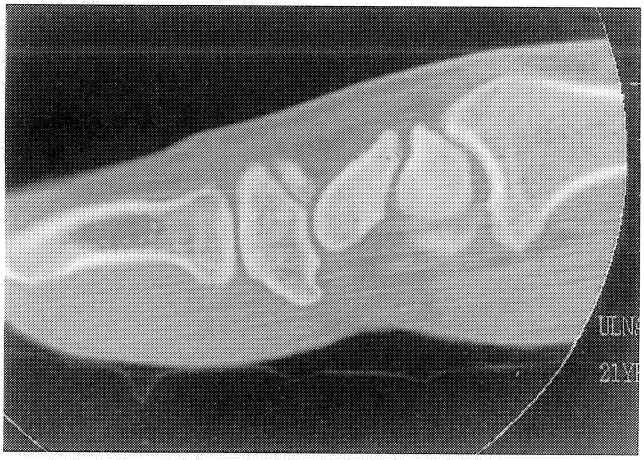

Figure 6. 21-year-old male with scaphoid nonunion.
Figure 6A. Sagittal long-axis view with ulnar deviation of the wrist shows fracture and moderate apex dorsal angulation.

Figure 6B. The radial deviation view is similar to the sagittal long-axis view except that the wrist is radially deviated and the elbow is extended.

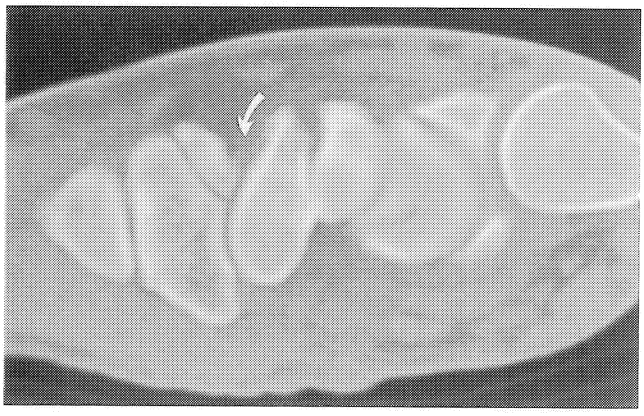

Figure 6C. Image obtained with the wrist in radial deviation shows significant increase in the amount of angulation (curved arrow).

COST CONSIDERATIONS

Each of the modalities presented has advantages and disadvantages for the detection and characterization of scaphoid injuries. One area of concern is the cost of imaging. The least expensive study is the plain radiograph, which in most centers costs less than one hundred dollars. Conventional tomography is roughly twice as expensive as plain films. Computed tomography costs approximately five times as much as plain radiographs. Bone scintigraphy has variable costs depending on whether three-phase versus single-phase studies are ordered, or whether a "limited" bone scan is available. In general, bone scanning is comparable in cost to CT. MRI is the most expensive modality for imaging the scaphoid, and can be twice as expensive as CT, depending on the institution. Additional sequences with gadolinium-based contrast material may or may not add to the patient charge, depending on the institution. Treating orthopaedists may alter ordering strategies for imaging tests of the scaphoid based on costs at their specific institution.

CONCLUSION

Scaphoid fractures and nonunions are important orthopedic problems. Several imaging methods are available for detecting acute fractures as well as evaluating patients with possible or established non-unions; each of these modalities has advantages and disadvantages. Proper selection of the imaging modalities presented in this review will assist the orthopedist in selecting the best treatment strategy.

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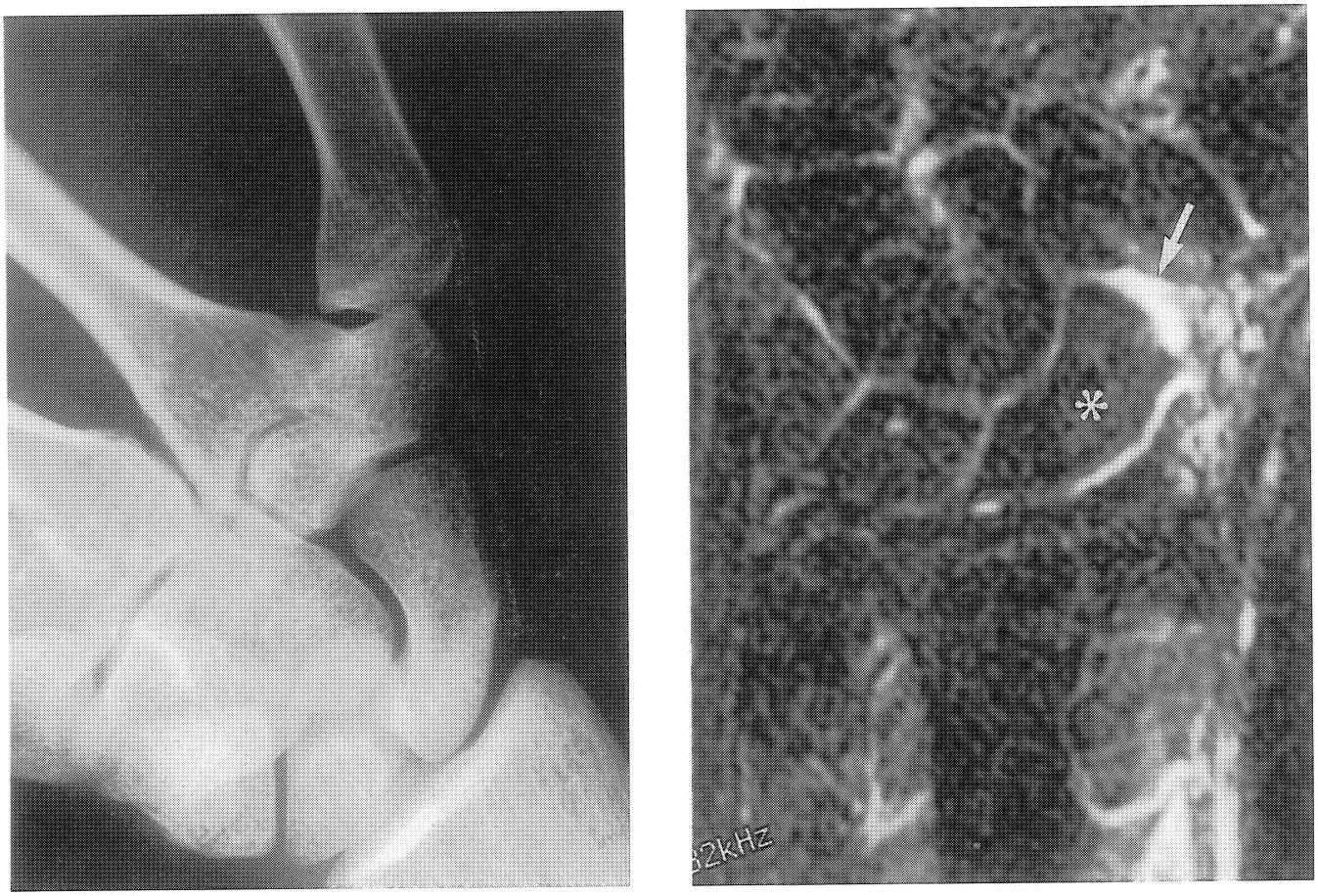

Figure 7. 21-year-old college basketball player with suspected scaphoid fracture.
A. Scaphoid view radiograph is normal.
B. MRI study using inversion recovery technique (TR = 3700 msec, TE = 42 msec, TI = 150 msec) was performed. Inversion recovery images are very sensitive for edema and hemorrhage. Note that there is a small amount of joint fluid adjacent to the distal tip of the scaphoid (arrow), but the scaphoid itself (asterisk) is normal.

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PROSPECTIVE SERIAL TECHNETIUM DIPHOSPHONATE AND INDIUM-111 WHITE BLOOD CELL LABELED IMAGING IN PRIMARY UNCEMENTED TOTAL HIP ARTHROPLASTY

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ABSTRACT

Although technetium diphosphonate (TcMP) and Indium-111 white blood cell labeled (Ind-WBC) imaging are reported useful in identifying aseptic and septic loosening in cemented hip arthroplasty, their usefulness has not been identified in uncemented porous coated total hip arthroplasty. We attempted to define the natural history of TcMP and Ind-WBC imaging in primary P.C.A. uncemented total hip arthroplasty.

Twenty-five hips in 21 patients were scanned immediately postoperatively, at 3 months, 6 months, 12 months, 18 months, and 24 months after surgery with both TcMP and Ind-WBC tracers. Clinical and radiographic follow-up were also obtained at each interval. Intensity and distribution of tracer activity were recorded as well as the time when stabilization occurred around the acetabulum, femoral porous surface areas, and femoral stem tip.

Acetabular cup and femoral porous surface areas stabilized in the first year on both TcMP and Ind-WBC imaging. Focal femoral tip activity continued at 24 months in 72% of TcMP and 24% of Ind-WBC images.

TcMP and Ind-WBC images used to assess uncemented total hip arthroplasty should not be over interpreted. Although persistent intense activity after one year around the acetabulum and porous surface femoral areas should be considered abnormal for both TcMP and Ind-WBC scans, femoral tip activity is present in the majority of patients, with or without thigh pain, at 24 months on TcMP scans. Tip activity can also persist at 24 months on Ind-WBC images and should be interpreted in conjunction with TcMP images.

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INTRODUCTION

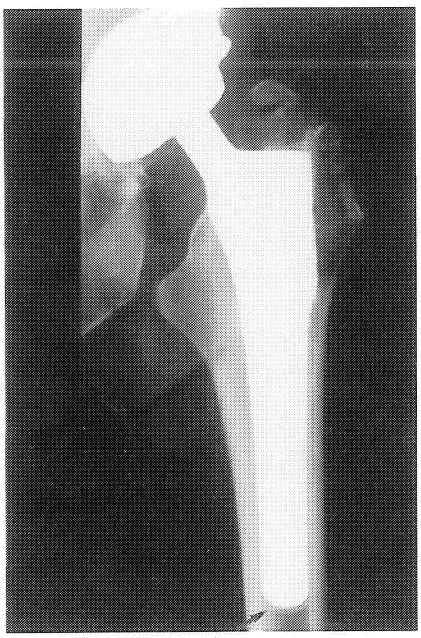
Technetium methylene diphosphonate (TcMP)[3,10] and Indium-111 white blood cell labeled (Ind-WBC)[7,8,11] imaging have been reported useful in the determination of both septic and aseptic loosening of cemented hip arthroplasty. Amstutz has reported the use of technetium methylene diphosphonate in the evaluation of uncemented resurfacing arthroplasty[1]. However, to date, no studies have reported the use of these imaging modalities in uncemented porous coated total hip arthroplasty. In addition, no natural history studies of Indium-111 white blood cell labeled imaging have been reported in cemented or uncemented total hip arthroplasty. This study evaluates the use of serial TcMP and Ind-WBC imaging in an uncomplicated group of patients undergoing primary uncemented porous coated total hip arthroplasty.

MATERIALS AND METHODS

A research protocol which included informed consent was approved by the Clinical Investigation, Human Use, and Radiation Committees of our hospital to evaluate serial TcMP and Ind-WBC imaging after uncemented porous coated total hip arthroplasty. A consecutive group of 21 patients with 25 primary uncemented total hip arthroplasties who completed serial TcMP and Ind-WBC imaging through a 24 month interval were included. The Porous Coated Anatomic (P.C.A.) total hip system was used in all cases. All cases were performed by one of two surgeons, or both. The procedures were performed after the two surgeons had already performed fifty previous procedures with the same device, hence they were well into the learning curve associated with any new procedure[2].

All patients had a preoperative clinical hip rating as described by Harris[6]. At 7 days, 3 months, 6 months, 12 months, 18 months, and 24 months, patients underwent TcMP and Ind-WBC imaging, anteroposterior and frog leg lateral radiographs, and were graded with the Harris hip rating.

In evaluating the clinical rating, both overall score and thigh pain were specifically recorded. Radiographs were evaluated for radiodense lines around the acetabular and femoral components (Figures 1a and b) and sclerosis around the tip of the femoral component (Figure 2). Any



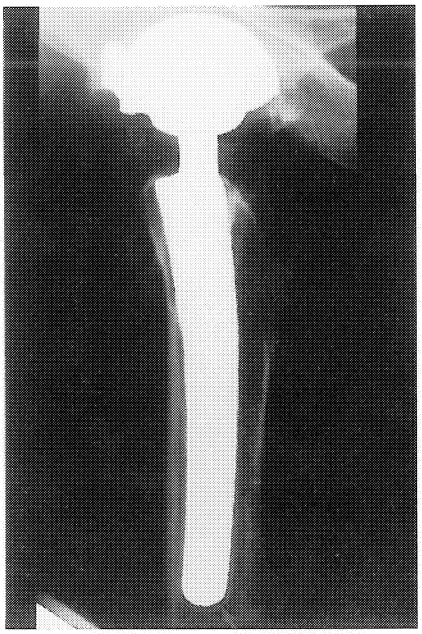


Figure 1. Typical acetabular and femoral radiodense lines (arrows) on anteroposterior (a) and lateral (b) radiographs.

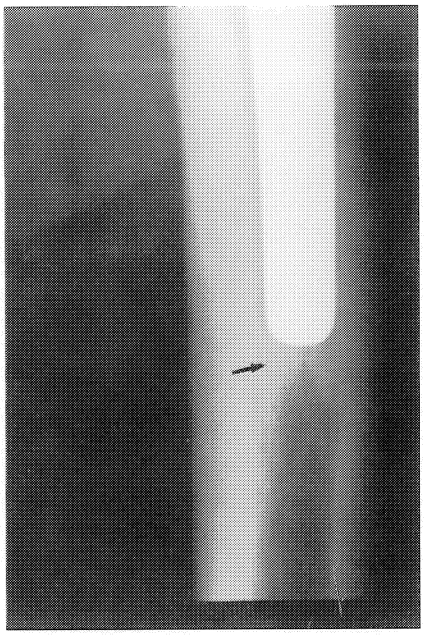


Figure 2. Typical femoral tip sclerosis on radiographs.

line greater than three millimeters wide was considered sclerosis. The distribution of lines and sclerosis were recorded using the three zones of DeLee and Charnley[4] for the acetabulum and the seven zones of Gruen[5] for the femur. In addition, progression of these lines and sclerosis between the one and two year follow-up were noted. Progression was defined as any line or sclerosis that was not present at one year but present at two years, or one that was present at one year and had progressed within the same zone or into another zone by two years.

TcMP and Ind-WBC images were graded for intensity of activity and distribution of activity at each interval. The intensity of uptake on both imaging techniques was graded from zero to four with +4 being complete blackout, +3 - increased activity compared to the iliac crest, +2 - activity of the intensity of the iliac crest, +1 - less than the iliac crest, and zero - the background which was usually equal to the activity in the nonoperated femoral canal. Distribution was recorded according to DeLee and Charnley zones[4] on the acetabulum and Gruen zones[5] on the femur. In addition, the time at which stabilization occurred was documented. Stabilization was defined as the time interval where no further increase or decrease in activity occurred on serial images.

Technique of TcMP Imaging

A dose of 15 mCu of TcMP was injected and the patient was scanned immediately for blood flow images, at thirty seconds to five minutes for blood pool images and two and one half to three hours for static images. For this study only the static images will be reported. If the patient could not void immediately before imaging, the bladder was shielded during the imaging.

Technique of Ind-WBC Imaging

Ind-WBC imaging was performed by obtaining 45 milliliters of the patient's blood, incubating the white blood cells with 500 microcuries of In-111-oxine, and reinjecting the indium labeled white cells back into the patient. Images were obtained 24 hours after reinjection[11].

Patient Population

Twenty-one patients with twenty-five hips who had undergone primary uncemented P.C.A. total hip arthroplasties were included in this prospective, consecutive series study. Seven patients were female and fourteen were male. The average age was 56 years, with a range of 39 to 76 years.

RESULTS

Clinical and Radiographic

Harris hip ratings (H.H.R.) and thigh pain are recorded in Table I for the five postoperative intervals that were evaluated. There were no appreciable differences in scores after 6 months. The only patients with fair or poor results were Charnley Class C patients who scored poorly because of diminished activity level, not pain. A substantial percentage of hips, 28% at two years, had some thigh pain with prolonged activity or when getting up from a chair, but it never required the use of analgesics or anti-

TABLE I
CLINICAL RATING

	3M	6M	12M	18M	24M
H.H.R.	83.6 (66-97)	92 (68-100)	94 (73-100)	93.6 (76-100)	92.7 (53-100)
Thigh Pain	24%	20%	28%	40%	28%

inflammatories and hence was graded slight. It is for this reason that we considered this a group of uncomplicated patients rather than asymptomatic patients.

Overall radiographic findings are illustrated in Table II. The distribution of radiographic lines and sclerosis are demonstrated in Figures 3a-e. As noted, there is a large percentage of patients with progressive lines, 84%, and sclerosis, 52%, around the femoral component with much less progression, 8%, around the acetabular component. These findings are similar to those we reported in our first fifty cases[2]. In no case was there acetabular migration or femoral subsidence.

TcMP Imaging

The intensity and distribution of TcMP activity around the acetabular and femoral components is illustrated in Figures 4 and 5. In addition, the percentage of hips that had stabilized around the acetabulum and femoral tip are recorded by interval. Around the acetabular component, immediate postoperative activity was in the +3 and +4 range and had reduced intensity to the +1 and +2 range by one year. Stabilization had occurred by one year in all but one hip which stabilized by 18 months.

Activity around the porous surfaces of the femoral component postoperatively were in the +2 and +3 intensity range and by one year were in the +1 and +2 range. All femoral porous surfaces had stabilized by one year. Stabilization had occurred by one year in all but one hip which stabilized by 18 months.

Femoral tip activity immediately postoperatively was in the +1 to +3 range and continued in this range during the entire 24 month period. Stabilization only occurred in 4% of hips at 3 months, 8% at 6 months, 12% at 12 months, and 28% at 18 and 24 months.

TABLE II
RADIOGRAPHIC RATING

Femoral Lines 96%	Progressive Femoral Lines 86%
Femoral Sclerosis 75%	Progressive Femoral Sclerosis 52%
Acetabular Lines 48%	Progressive Acetabular Lines 8%

Ind-WBC Imaging

The intensity and distribution of Ind-WBC around the acetabular and femoral components are illustrated in Figures 6 and 7. In addition, the percentage of hips that had rating. We postulate continued bony remodeling or micromotion at the tip of the prosthesis at two years. The stabilized in the acetabular and femoral tip area are recorded by interval. Around the acetabular component, immediate postoperative activity was of the +1 to +3 intensity. By one year, activity was in the 0 to +2 intensity. All acetabular components had stabilized by one year.

Ind-WBC activity around the porous surfaces of the femoral component were in the 0 and +1 range throughout the 24 month period with stabilization in all cases by one year. Femoral tip activity immediately postoperatively was in the 0 to +3 range and remained in the 0 to +2 range throughout the entire 24 month interval. Stabilization occurred in the scans of 28% of femoral tips by 3 months, 44% by 6 months, 64% by 12 months and 76% by 18 and 24 months. In only one case was the Ind-WBC activity greater than TcMP activity after one year.

CASE ILLUSTRATIONS

Case 1

The serial TcMP and Ind-WBC images of a 65 year old patient with a right hip arthroplasty are illustrated in Figures 8 and 9. The patient was completely asymptomatic during the entire 24 month interval and had a Harris hip rating of 100 from 6 months to 24 months post surgery. The TcMP scans (Figure 8) illustrate the typical persistent femoral tip activity at two years. His indium scans show no focal indium accumulation throughout the entire 24 month period.

Case 2

The images in Figure 10 are the serial TcMP scans of a patient who was asymptomatic until 18 months postoperatively when she developed thigh pain which resolved within 3 months after going back to using a cane. The 18 month TcMP image showed increased femoral tip activity which decreased by 24 months. This finding became apparent only because the patient had serial images from before and after the 18 month thigh pain incident.

Case 3

The 24 month TcMP and Ind-WBC images of a 72 year old patient who underwent bilateral uncemented hip arthroplasties are illustrated in Figures 11 and 12. The patient had slight thigh pain bilaterally with activity throughout the entire 24 month period but no symptoms of infection. His hip ratings remained at 91 points. He had

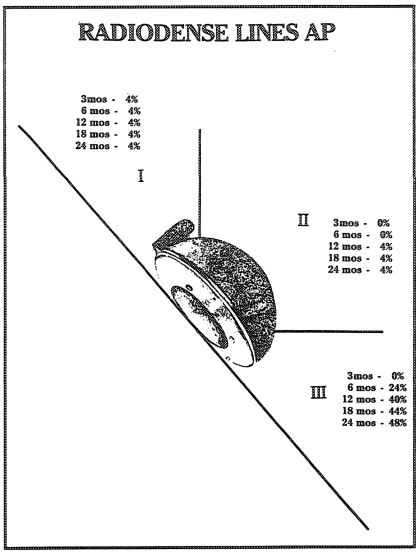


Figure 3a. Distribution of acetabular radiodense lines on serial radiographs.

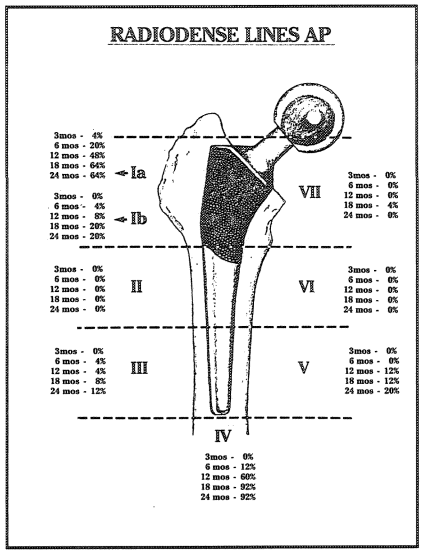


Figure 3b. Distribution of femoral radiodense lines on anteroposterior serial radiographs.

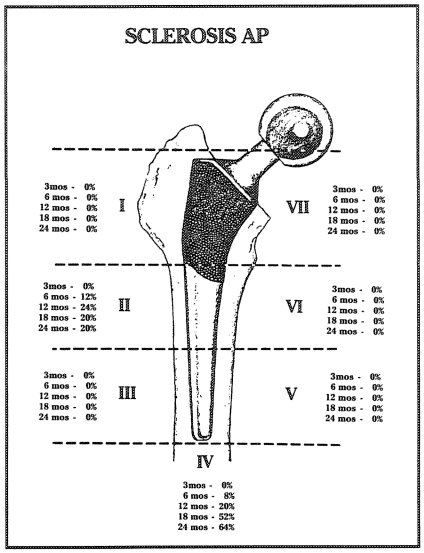


Figure 3d. Distribution of femoral sclerosis on anteroposterior serial radiographs.

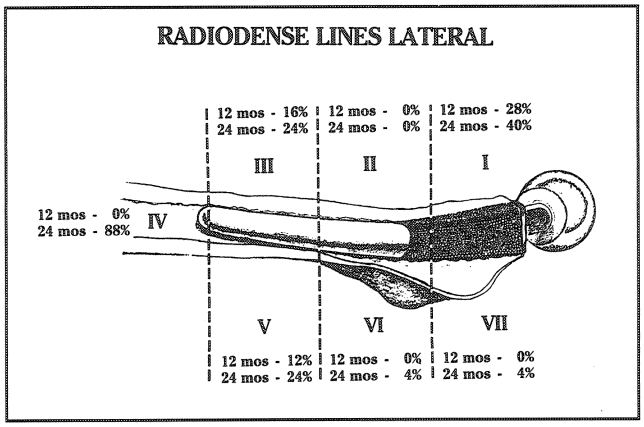


Figure 3c. Distribution of radiodense lines on lateral serial radiographs.

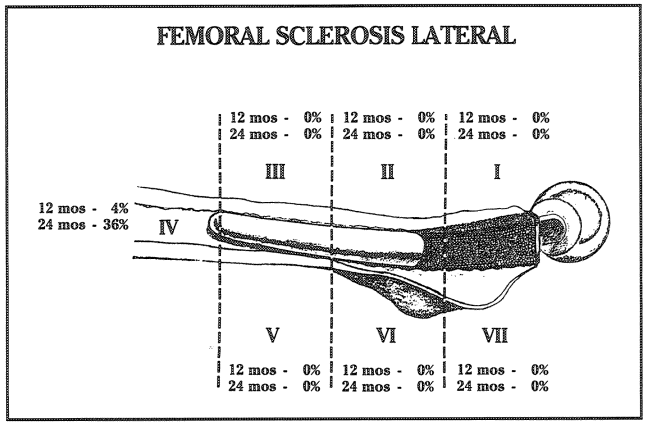


Figure 3e. Distribution of femoral sclerosis on lateral serial radiographs.

persistent focal femoral tip activity on both TcMP and Indium-111 imaging throughout the entire 24 month period. His Ind-WBC activity was equal to or greater than TcMP activity after one year. Serial Ind-WBC images of this patient also revealed the intermittent appearance of lymph node activity which was seen in 20% of scans and could be misinterpreted as focal Ind-WBC uptake consistent with infection.

Case 4

Serial Ind-WBC images of an asymptomatic patient during her entire 24 month follow-up are illustrated in Figures 13a-c. Note the focal accumulation of Ind-WBC on the 13 month image in the area of the greater trochanter which is not present before or after this time. Once again, this could mistakenly be interpreted as representing infection if serial scans were not obtained.

DISCUSSION

Uncemented total hip arthroplasty is a relatively new procedure which continues to be investigated. In cemented total hip arthroplasty, TcMP bone imaging[3,10] and Ind-WBC imaging[7,8,11] have been reported to be helpful in identifying septic and aseptic loosening. In a comprehensive serial evaluation of TcMP imaging in cemented total hip arthroplasty, Utz et al.[10] found that scans in asymptomatic patients usually stabilized by one year, and that only 10% had any increased activity at two years. When present, this was in the area of the greater trochanter or

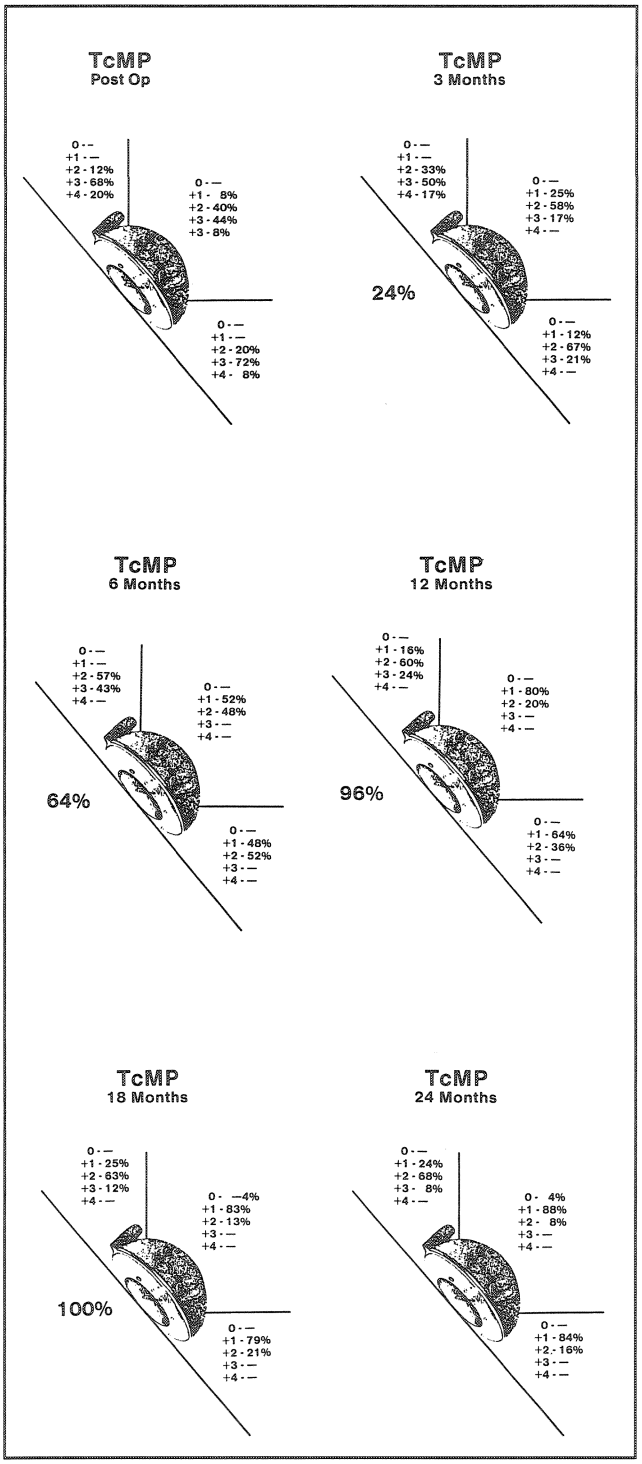


Figure 4. Distribution and intensity of TcMP activity around the acetabulum. The bold type percentage is the percentage of acetabular components stabilized at each interval.

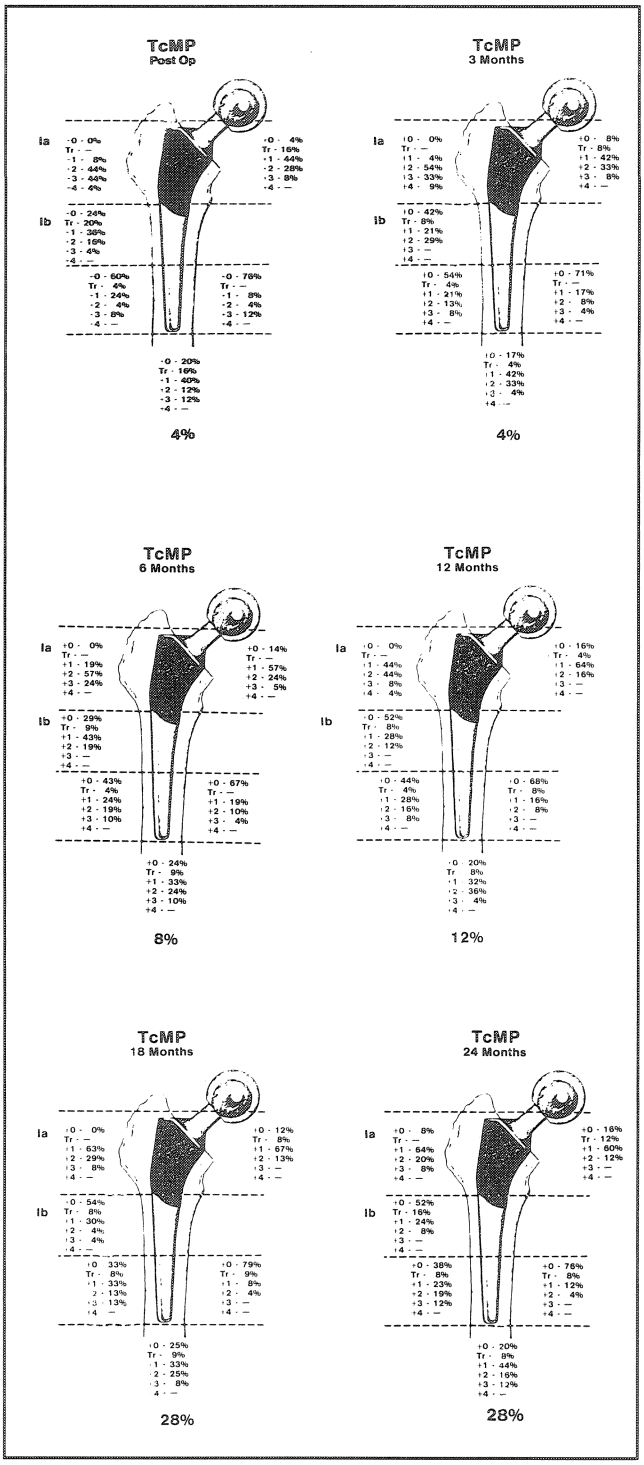


Figure 5. Distribution and intensity of TcMP activity around the femoral component. The bold type percentage is the percentage of femoral stem tips that had stabilized at each interval.

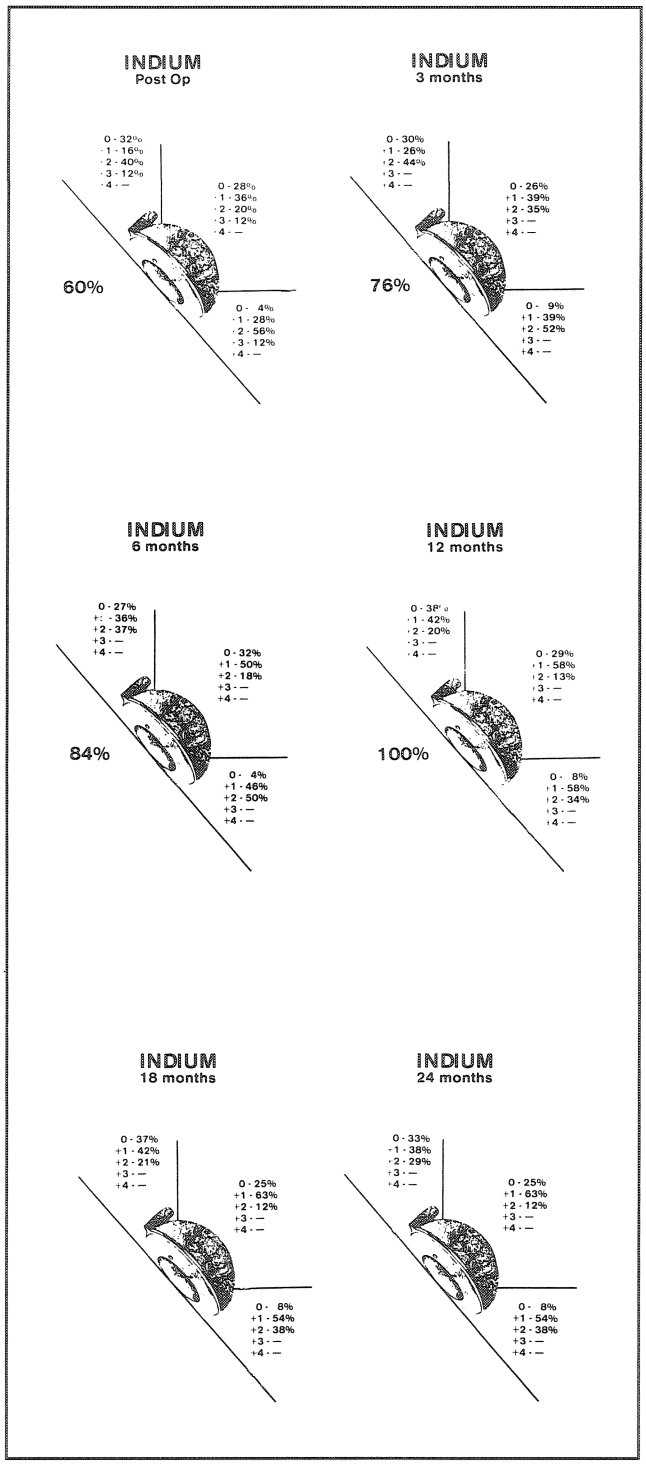


Figure 6. Distribution and intensity of In-WBC activity around the acetabulum. The bold type percentage is the percentage of acetabular components stabilized at each interval.

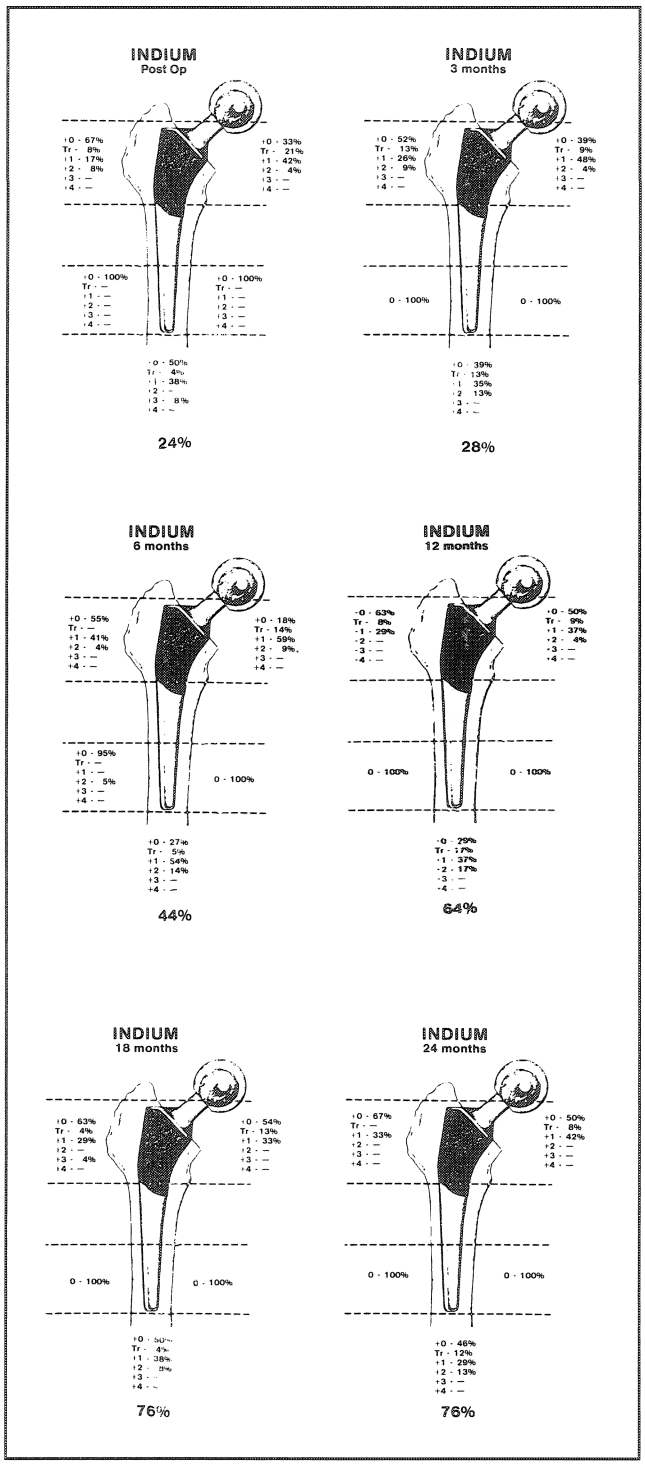


Figure 7. Distribution and intensity of In-WBC activity around the femoral component. The bold type percentage is the percentage of femoral stem tips that had stabilized at each interval.

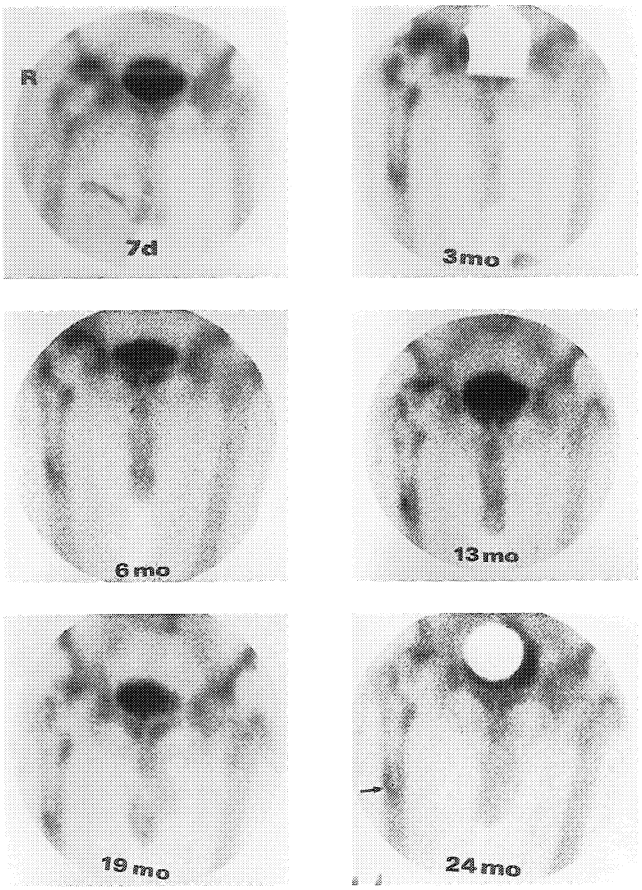


Figure 8. Serial TcMP images of a patient with a right hip arthroplasty that was asymptomatic throughout the 24 month follow-up. Note the persistent femoral tip activity at 24 months (arrow).

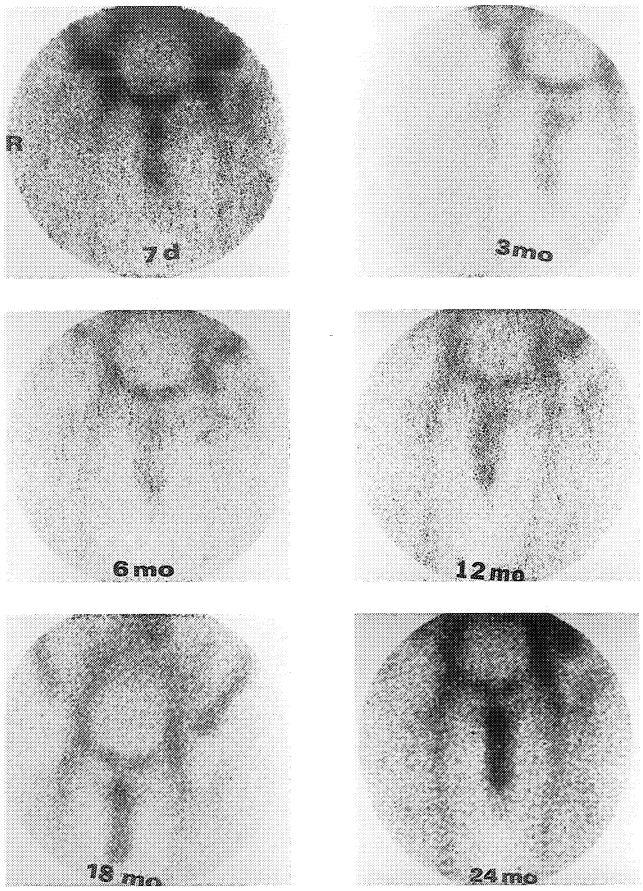


Figure 9. Serial In-WBC images of the same patient as Figure 8. There is no focal indium accumulation during the entire 24 month period.

femoral tip. Merkel et al.[7] and Wukich et al.[11] have demonstrated the efficacy of Ind-WBC imaging in cemented total hip replacement. Merkel used the criteria of any focal activity being indicative of infection. Wukich, however, did find a significant number of false positive scans with focal accumulations of Ind-WBC in noninfected cases. No natural history studies of Ind-WBC imaging have previously been performed in uncemented total hip arthroplasty patients.

Spector et al.[9] have evaluated bone imaging in the animal model. However, the use of serial TcMP and Ind-WBC imaging in uncemented porous coated total hip arthroplasty in humans has not been documented other than in uncemented resurfacing devices[1]. By serial TcMP and Ind-WBC imaging over a two year period in a consecutive series of uncemented primary total hip patients, using a particular prosthesis, we hoped to define the natural history of these tracer activities in uncemented hip arthroplasty patients. We hoped that this serial imaging would better define the biologic response occurring at the porous surfaces and better define any remodeling occurring in the femur over the time interval involved.

On TcMP imaging a definite pattern of activity evolved with time. The acetabulum and femoral porous surface areas stabilized by one year. However, at the femoral tip, activity persisted and did not stabilize in 72% of hips during the entire two year interval. This is definitely different than the TcMP imaging of cemented hip replacements. In addition, it did not correlate with thigh pain or Harris hip findings of stabilization by one year in the acetabulum and continued activity at two years at the stem tip does correlate with the low incidence of progressive acetabular lines, 8%, and the high incidence of progressive femoral lines, 84%, and sclerosis, 52%, on radiographs.

During the first year some focal indium tracer accumulation was common around acetabular and femoral components. At two years 24% of hips still had some femoral tip tracer uptake. However, in only one case was indium uptake greater than TcMP uptake. This data supports the previous findings of Wukich that in cemented arthroplasty focal Ind-WBC uptake alone may not be indicative of

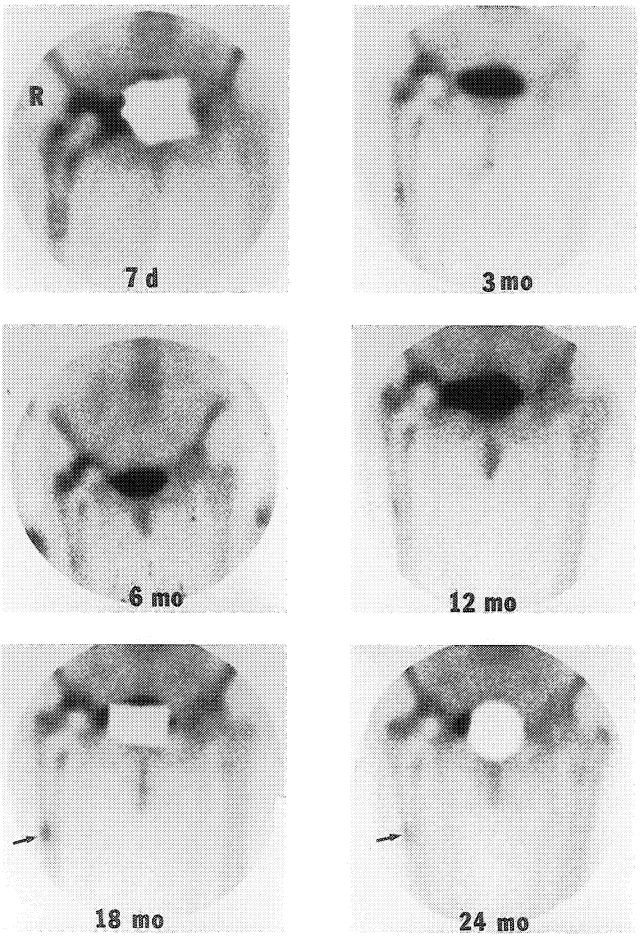


Figure 10. Serial TcMP images of a patient who developed the late onset of thigh pain 18 months after her right total hip arthroplasty. The arrow denotes the increase tip activity at 18 months with decrease activity by 24 months.

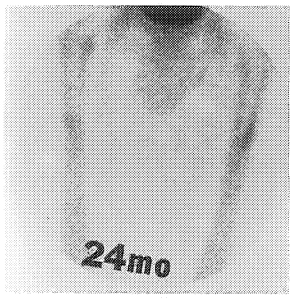


Figure 11. 24 month TcMP images of a patient who had bilateral hip arthroplasties and has slight bilateral thigh pain but no evidence of infection. This is trace TcMP activity at the right stem tip and +2 activity at the left.

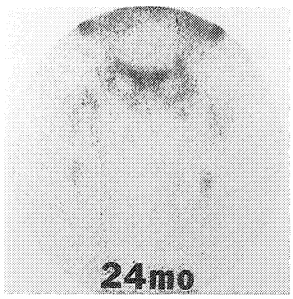


Figure 12. 24 month In-WBC image of the patient in Figure 11 demonstrates continued focal tracer activity at the stem tips which is equal to or greater than TcMP intensity. This response represents a false positive study.

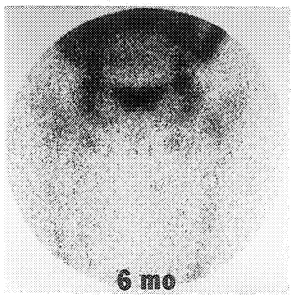


Figure 13a

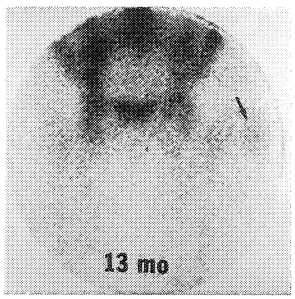


Figure 13b

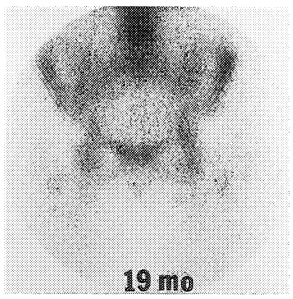


Figure 13c.

Figure 13. Serial In-WBC images of an asymptomatic patient with bilateral uncemented total hip arthroplasties. Patient developed focal In-WBC activity near the left greater trochanter at 13 months (b) denoted by the arrow with no symptoms and no activity before (a) or after (c) this time.

infection, and that combined interpretation of TcMP and Ind-WBC is warranted. If this interpretation is used, only one false positive interpretation would be present in our study, assuming no patients from the group were infected. We feel comfortable making this assumption because all patients had been followed for at least two years without suspicion of infection clinically or radiographically.

CONCLUSIONS

This study attempted to define the natural history of TcMP and Ind-WBC imaging in primary uncemented P.C.A. total hip arthroplasty by serially scanning a consecutive series of patients without postoperative complications. We recommend caution as to the possible over interpretation of both TcMP and Ind-WBC scans in uncemented patients. TcMP activity around the acetabulum and femoral porous surface areas should be stabilized by one year and if activity is present at a later time the scan should be read as abnormal. However, femoral tip activity at two years should not be considered abnormal as it was present in 72% of hips. Although we do not recommend routine imaging in uncemented hips because of the cost of radiation, serial imaging is helpful in the patient with

persistent pain or the patient with late onset pain in order to determine if activity, especially stem tip activity, diminishes with time (Case 2).

Ind-WBC images should be interpreted in conjunction with TcMP. During the first postoperative year focal indium accumulation is common and one should not over interpret any focal activity as an indication of infection. However, any acetabulum or femoral porous surface area activity should be considered abnormal and suspicious for infection after one year. Focal stem tip activity can persist at two years, 24% of hips, and this activity should be compared to TcMP activity before interpreting the scan as suspicious for infection.

These findings should help the orthopaedic practitioner in his use of bone imaging to evaluate the patient with a painful uncemented hip prosthesis. It should help prevent the over interpretation of tracer activity of both TcMP and Ind-WBC imaging. One must also remember that some of these findings may be prosthesis specific and may not apply to devices other than the original P.C.A. total hip arthroplasty design. We continue to evaluate this group of patients with yearly imaging to determine the long term natural history.

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WHEN IS TOTAL HIP ARTHROPLASTY A FAILURE? THE PATIENTS' PERSPECTIVE

Edward J. Hellman, M.D.
Judy R. Feinberg, Ph.D.
William N. Capello, M.D.

ABSTRACT

Total hip arthroplasty (THA) patients (186 primary, 92 revision) were surveyed regarding their satisfaction, their expectations regarding longevity of the hip implant, and their perspective on the potential or actual need for revision surgery. The vast majority of patients were glad they had the original THA, would do it again if faced with a similar choice, and would recommend it to others. One-third of patients believed their current implants would last the rest of their life. The most common responses to either potential or actual failure were happiness it lasted as long as it did, accepting it as "one of those things," and disappointment. No primary THA patients and only 7% of revision THA patients indicated that they would consider the primary THA a failure when revision surgery was indicated.

Key words: hip arthroplasty, revision, patient perspective, outcome

Total hip arthroplasty (THA) is the most common adult orthopaedic reconstructive procedure performed in the United States with greater than 123,000 primary THAs performed each year[1,2]. The vast majority of patients who have undergone this procedure report an overall improvement in quality of life (i.e., decreased pain and improved function)[3]. New advances in technology have decreased risk and improved long-term success of the artificial hip. One-third of THA patients are under the age of 65 years[4], thus making longevity of the implant an extremely important research consideration. It is estimated that at least 1% of the THAs performed each year will require revision; and within this group, 5-9% will need re-revision[4]. With the increase in number of primary THAs, the number of revision procedures is also increasing dramatically.

As a rule, clinical outcome studies of THA define failure as the need for revision surgery. Others may determine failure based on radiographic loosening or the patient's degree of pain. Regardless of what definition is used, the patient's assessment of success or failure is not generally considered. The term "failure" is entirely surgeon-determined.

It is apparent to orthopaedic surgeons that an operation to replace a hip implant is considered a failure of the original THA, but there is no data in the literature addressing the patients' perspective on this issue. The purpose of this study was to obtain information from the patient regarding their satisfaction with their THA, the expected longevity of the implant, and their feelings regarding the potential or actual need for revision surgery.

METHOD

Data was collected using mailed patient survey instruments. The instruments were developed by two experienced orthopaedic arthroplasty surgeons and a rehabilitation psychologist. Two similar survey instruments were developed: one for patients who had undergone primary THA and one for patients who had undergone revision THA. The only difference between the surveys was that the primary patients were asked about possible future revision surgery and the revision patients were asked about their actual revision surgery. The majority of both surveys consisted of questions with multiple choice answers. The initial surveys were field tested with patients outside our study population to ensure completeness of choices and to limit misunderstanding of questions. The average time to complete a survey was ten minutes.

Each survey requested demographic information and information on the patient's current functioning (e.g., degree of hip pain, limp, and functional limitations). All patients were asked on a five-point scale how satisfied they were with their present hip replacement, if they would have the procedure again, and if they would recommend the procedure to others. They were also asked if they had been given an estimate by their surgeon of how long the hip implant would last and how long they felt it should last. Primary patients were asked to indicate how they would feel if their artificial hip would need to be replaced before the designated time and also how they would feel if it required revision after the stated time. Revision patients were asked to comment on how they felt when they were told they needed revision surgery and how they would feel if they required yet another revision in the future. Patients were asked to select as many

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Angry
Frustrated
Sad or depressed
Disappointed
Glad it lasted as long as it did
Accept(ed) it as just one of those things
Surgery was successful, but time limited
Surgery was a failure
I might have done something to cause the problem
My surgeon might not have done the operation totally correctly
The artificial joint might have been flawed in some way

TABLE 1. List of respondent choices regarding potential or actual need for revision surgery

responses as applied based on the same list of emotional and cognitive responses (Table 1).

The study sample was drawn from the hospital THA database. All primary and revision patients were operated on by a single surgeon at one institution, although the primary procedure in revision patients had often been performed elsewhere. There were no restrictions relative to component fixation or number of revision procedures in the revision group. The only inclusion criteria were that the patient had to be a minimum of two years out from either the primary or the most recent revision procedure, and the patient had to be less than 90 years old at the time of the survey. The purpose of the age limitation was to minimize the loss of subjects due to death.

After stratification into primary and revision groups, the sample population was randomly selected proportional to the total number of primary and revision THA patients in the database (e.g., two primary patients for each revision patient). Study numbers were assigned to each subject to allow for patient anonymity in responding to the survey. After three weeks, patients who had not responded were identified by a research assistant and contacted by telephone, requesting return of the completed survey. To obtain an error of estimation at the .05 level, a total sample size of approximately 334 was necessary[5]. Considering the response rates of well-designed surveys with a follow-up reminder are typically 65-75%[6,7], a total sample of 450 patients (300 primary and 150 revision hips) were selected to receive the survey.

Statistical analysis of data included descriptive statistics on all variables by subgroup. Differences between primary and revision THA patients and differences between those who completed the surveys and those who did not were analyzed using t-tests for continuous variables and chi-square analysis for categorical variables.

RESULTS

Of the 450 surveys mailed, 47 (10%) were returned by the postal service as undeliverable. These patients were considered lost to follow-up. We received notification that an additional 18 (4%) patients had died. Of the 385 patients who presumably received the survey, completed surveys were returned by 186 (73%) primary THA patients and 92 (71%) revision THA patients. There were no differences in demographic characteristics between the dropout group and those who completed the survey with the exception of postoperative time. The dropout group was an average of 17 months further postoperatively than the group that completed the surgery ($p<0.05$).

Demographic characteristics of the primary and revision patient groups are provided in Table 2. Although age and gender were similar between groups, a greater percentage of patients in the primary THA performed group were married, employed, and in better general health. Since most of the revision patients had their primary THA performed elsewhere, the original diagnoses were not known with certainty. Time since primary surgery for the unrevised group and time since revision in the revision group were similar. Revision patients had their primary THA an average of seven years prior to their first revision surgery. There was no difference between groups with

	Primary (n = 186)	Revision (n = 92)
Current Age (years)	64 (22-87)	66 (25-87)
Gender		
Male	83 (44%)	39 (42%)
Female	105 (56%)	53 (58%)
Marital Status		
Married	132 (71%)	54 (59%)
Single/Divorced/Widowed	52 (29%)	37 (41%)*
Employment Status		
Full or Part Time	63 (34%)	20 (21%)
Retired	88 (47%)	54 (59%)
Housewife/Student	34 (19%)	15 (20%)*
General Health		
Excellent/Very Good	85 (46%)	29 (31%)
Good	64 (34%)	33 (36%)
Fair/Poor	38 (20%)	30 (32%)**
Diagnosis		
Osteoarthritis	132 (71%)	NA
Avascular Necrosis	27 (15%)	NA
Inflammatory Arthritis	15 (8%)	NA
Dysplasia	6 (3%)	NA
Other	6 (3%)	NA
Time Since Primary THA (years)	6.1	13.0***
Time Since Revision THA (years)	NA	5.1

* $p<0.05$
** $p<0.01$
*** $p<0.001$

TABLE 2. Demographic characteristics of survey respondents

	Primary (n=186)	Revision (n=92)
No or Slight Hip Pain	82%	62%**
No or Slight Limp	89%	68%***
No Support to Walk	82%	50%***
No or Slight Difficulties in Daily Activities	76%	49%***
Community Activities Limited Due to Hip	9%	44%***

** $p<0.01$
*** $p<0.001$

TABLE 3. Current functional status of survey respondents.

regard to component fixation. Fifteen (16%) revision patients had undergone at least one re-revision operation.

Table 3 shows the subjective clinical results of both groups with respect to their present THA. The primary THA group reported better function in all surveyed areas than the revision group. Of those patients who reported limited ability to get out into the community, 9% of primary patients and 44% of revision patients indicated that their limitations were due to their hip ($p<0.001$).

Figure 1 shows the patients' responses to questions about changes in function and satisfaction with their initial THA. Patients in the primary group were asked about their current THA whereas patients in the revision group were asked to recall their level of satisfaction with their original hip replacement. The percentage of patients who were definitely satisfied with the degree of pain relief, improvement in ambulation, and improvement in community access, was greater in the unrevised patient group ($p<0.0001$). The unrevised group was more likely to report that they were glad they had the primary THA done ($p<0.0001$). Poorer general health ($p<.01$) and functional limitations due to the hip ($p<0.001$) correlated with less than total satisfaction in the primary THA group. In the revision group, those who were less than totally satisfied with their primary THA also reported more hip pain ($p<0.05$) and limitations of function ($p<0.001$) due to their hip after revision. There were no significant differences between groups regarding those who would definitely undergo primary THA again if they had it to do over (92% primary vs. 87% revision) and those who would definitely recommend the procedure to others (94% primary vs. 84% revision).

The majority of both primary and revision patients (79% and 82% respectively) indicated that their surgeon had given them an estimated longevity of their primary THA. The average estimated implant longevity as recalled by the unrevised group was 15 years (range 5-50) compared to

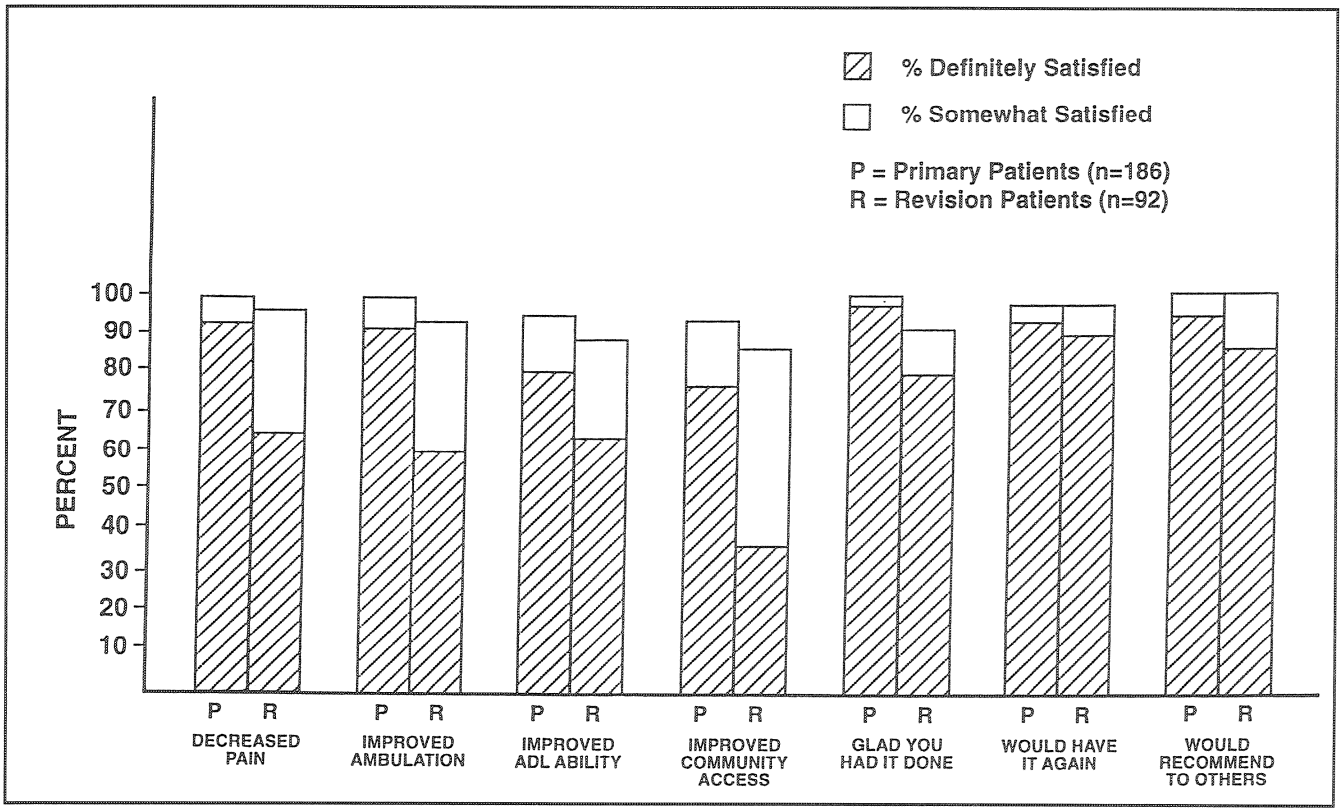

FIGURE 1: Patient Satisfaction with Primary Total Hip Arthroplasty

12 years (range 2-25) for both the primary and the revision arthroplasties in the revised group. About one-third of each group (37% primary, 35% revision) thought their current hip implant would last the rest of their life. The average age of these patients did not differ between groups as a whole. No comparisons were made between the estimated longevity stated by the surgeon and that stated by the patient due to the large number of patients simply reporting "the rest of my life" or "I had no idea how long the implant would last."

Primary THA patients were asked to indicate how they would feel if they were told they would require revision surgery prior to the time the surgeon indicated. Their responses were compared to the responses of the revised group as they recalled their feelings when told they needed revision surgery (see Table 4). The three most common responses were similar in both groups although not in the same rank order. The most common response (51% of patients) by those in the primary group was that they would be glad it lasted as long as it did, whereas only 25% (third in rank order) of patients who actually experienced revision felt the same way ($p<0.0001$). The possibility of surgeon error was more likely to be considered by the revised group ($p<0.0001$). Six (7%) patients in the revised group but no patients in the unrevised group would consider the need for revision surgery a failure of the primary surgery ($p<0.001$). Four of the six patients who felt their primary procedure was a failure underwent revision within four years of their primary procedure. The other two failures were reported by a 47 year old female whose initial arthroplasty was revised 13 years postoperatively and an 84 year old male whose initial arthroplasty was revised after 18 years. The most frequently used responses by the primary group to the theoretical need for revision at the time estimated by their surgeon and for revision patients who might need yet another revision were similar. The top four responses in each group were: glad it lasted as long as it did, accept it as just one of those things, successful but time limited, and disappointment. The only difference in response to premature versus the late need for revision was that patients would be less likely to be disappointed if the revision occurred later than anticipated.

	Primary	Revision
Glad it lasted as long as it did	1 (51%)	3 (25%)
Accept it as just one of those things	2 (30%)	1 (35%)
Disappointed	3 (25%)	2 (29%)
The surgery was successful, but the results were time limited	4 (19%)	8 (11%)
Sad or depressed	5 (12%)	4 (17%)
Frustrated	6 (11%)	5 (16%)
I might have done something to cause the problem	7 (6%)	6 (14%)
Angry	8 (2%)	9 (7%)
The surgeon might not have done the operation totally right	9 (<1%)	7 (13%)
The artificial joint may have been flawed in some way	10 (<1%)	11 (5%)
The surgery was a failure	11 (0%)	10 (7%)

TABLE 4. Comparison of the rank order and group percentages of primary patients' responses to theoretically needing a revision surgery in the future and revision patients' responses to actually being told revision surgery was needed.

DISCUSSION

The purpose of this survey was to document the outcome of THA from the patients' perspective and assess their feelings to the theoretical or actual need for revision surgery. The data clearly show that THA provides significant improvement in pain and function, and that patients are quite satisfied with the procedure. Revision patients are somewhat less enthusiastic about the improvements they obtained from their primary THA. However, revision patients also report more hip-related functional limitations than the primary THA patients, and this decrement in current function may be reflected in their responses to questions about their original hip arthroplasty.

Patients seem to accept that their well-functioning hip may require revision at some time in the future. One could argue that asking a theoretical question about the need for revision surgery to a patient who has not been faced with that situation may not elicit an accurate response. Yet in this study we included a group of revision patients for comparison and found that the majority of their responses were similar. The most common responses in each group appear to reflect a rather pragmatic, cognitive response to a situation in which the patients have few or no alternatives. Even their emotional responses are moderate, with more reporting feelings of disappointment and sadness than of frustration or anger. A greater percentage of those who had undergone revision surgery compared to those in the unrevised group chose to place blame on either the surgeon, the implant, or themselves, although these were not common responses in either group.

Perhaps what is most interesting is that no patient with a primary THA indicated that they would consider the original surgery a failure if they were told that revision surgery was necessary. In addition, very few (7%) patients who had undergone revision surgery felt that the

original surgery had been a failure. This is in contrast to the surgeon's perspective in which the need for revision surgery constitutes failure of the primary THA. Certainly one of the aims of research with THA is to increase the longevity of the implants. However, it may be the mind-set of the consumer that nothing is forever, and that a hip implant may last for only a given time before requiring revision. Even when faced with what would be considered a premature failure or a revision failure, more patients stated they would be disappointed, but very few felt the previous surgery was or would be a failure.

In summary, based on the results of this survey of both primary and revision THA patients, a THA provides significant pain relief and improves function. The vast majority of patients in both groups were glad they had the primary procedure done, would do it again if faced with the choice, and would recommend it to others. No primary THA patients indicated that they would consider the original surgery a failure regardless of when a revision would be necessary. This was corroborated by the majority (93%) of revision THA patients who had experienced revision surgery and still did not consider the primary surgery a failure. The orthopaedic surgeons' definition of THA failure, the need for revision surgery, is clearly different from the patients', who rarely consider revision THA a failure. Perhaps we should reconsider the definition of THA failure.

ACKNOWLEDGMENT

This research was supported in part by a Grant-in-Aid of Research awarded by the Faculty Development Office of Indiana University - Purdue University at Indianapolis.

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SEMITENDINOSUS AND GRACILIS TENDON GRAFT FIXATION FOR ACL RECONSTRUCTIONS

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INTRODUCTION

In the immediate postoperative period, the fixation site is the weakest point in an ACL reconstruction. While the interference screw is the standard for bone-patellar tendon-bone graft fixation, there is no general agreement as to optimal fixation for hamstring grafts.

Previous studies have focused on techniques not commonly used for ACL reconstructions, such as a single washer, single staple, or spiked plate. Kurosaka et al. tested various tendon graft materials using staple fixation, tying sutures over buttons, and screw fixation using six different grafts removed from human cadavers[2]. With staple fixation, either using patellar tendon, iliotibial band, or semitendinosus tendon, the grafts failed at the staple sites in all the cases. The patellar tendon grafts fared better than the iliotibial band and semitendinosus grafts regardless of fixation methods. Among the different methods of fixation with the patellar tendon, those fixed with a screw placed between the bone block of the graft and the tunnel (to achieve interference fit as previously described by Lambert[3]) tolerated higher loads before failure than those fixed with staples.

Robertson et al. compared the failure strengths of various methods of soft tissue fixation[5]. They tested the barbed staple, stone staple, suture techniques, screw with spiked plastic washer, and the screw with spiked soft tissue plate. They used three different types of soft tissue specimens and loaded the specimens in a cyclical fashion until fixation failure occurred. They concluded that the stone staple was the poorest technique and that the screw with the spiked plastic washer or soft tissue plate proved superior overall for all tissue types.

In a direct comparison between hamstring and patellar tendon ACL grafts, Steiner et al. determined that among fixation methods of gracilis-semitendinosus tendon grafts, the strongest technique had the tendons doubled and secured with two screws and two metal washers[6]. This was superior to suture methods, the washer technique with single-stranded grafts, and interference fit screw fixation. They used a custom fixture to maintain the femur and tibia at 20 degrees of flexion and prevented axial rotation about the long axes of the bones during the failure testing on an Instron 1331 Materials Testing Machine. They then translated the tibia anteriorly with respect to the femur in a manner similar to a Lachman test instead of the distraction technique used in previous studies.

While all the mentioned studies provide some measure of the relative strength of the fixation of these grafts, many of these methods have been outdated and replaced by newer techniques. Two currently used techniques are the double staple belt buckle and the double screw and spiked washer figure-8. The purpose of this study was to compare these two methods in a bovine model which closely resembles the fixation of a combined semitendinosus and gracilis tendon graft to the femur in an ACL reconstruction.

METHODS

Two bovine tendons similar in area and appearance to young adult human semitendinosus (14.0 mm^2) and gracilis (7.2 mm^2) tendons were used. The tendons were measured with an area micrometer and used if within 10% of these values. Twenty pairs of tendons and ten femurs were harvested at sacrifice and stored at 20°C until testing. The pairs of tendons were fixed to the distal metaphysis of bovine femurs and the free ends were securely held in a Cryojaw (MicroStrain Corp., Burlington, Vermont) soft tissue clamp[1]. This device utilizes a dry ice reservoir to freeze the tissue within the clamp, preventing slippage. Each femur was used for both techniques, one on the medial aspect of the femur, and the other on the lateral side. The tests were randomized between sites. In the double screw and spiked washer figure-8 technique, both cortices were drilled and tapped 15 mm apart (Figure 1). In order to minimize friction at the bone-screw interface, the screws were repeatedly inserted until less than 0.226 Nm of torque was lost to friction. This technique enabled us to apply consistent pressure on the tendons. The tendons were wound around two bicortical 6.5 mm cancellous screws with Synthes 13.5 mm plastic spiked washers. A 225 g weight was attached to each tendon and the screws were tightened, proximal first, to 0.678 Nm with a torque wrench.

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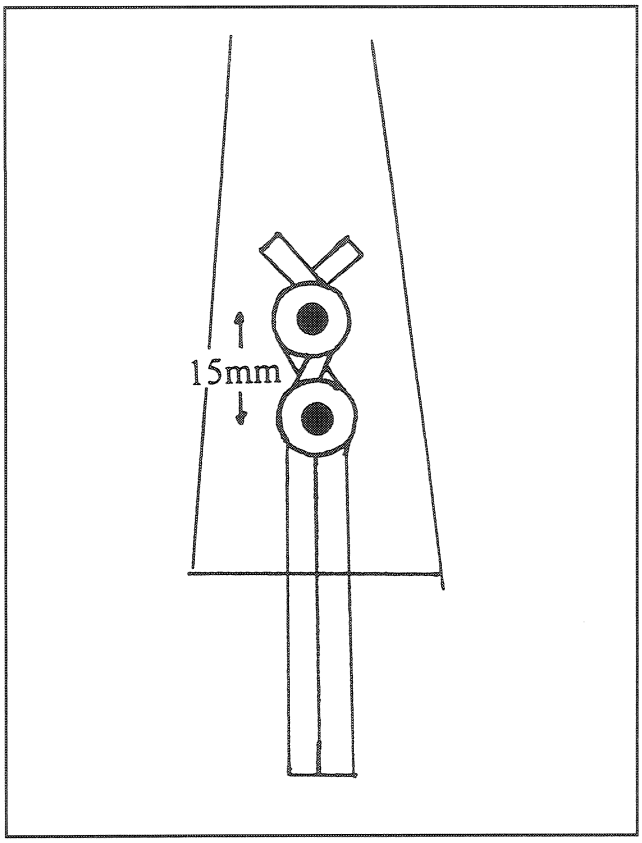

Figure 1. The spiked washer figure-8 soft tissue fixation technique. The tendon was fixed to the distal metaphysis of the femur by wrapping it around two bicortical 6.5 mm cancellous screws with 13.5 mm plastic spiked washers placed 15 mm apart in a figure-8 pattern. The free end was attached to the Cryojaw tissue clamp to perform the testing protocol. The distance between the distal screw and the Cryojaw tissue clamp was measured to be equal to the corresponding distance of the tendon used in the double staple belt buckle fixation technique.

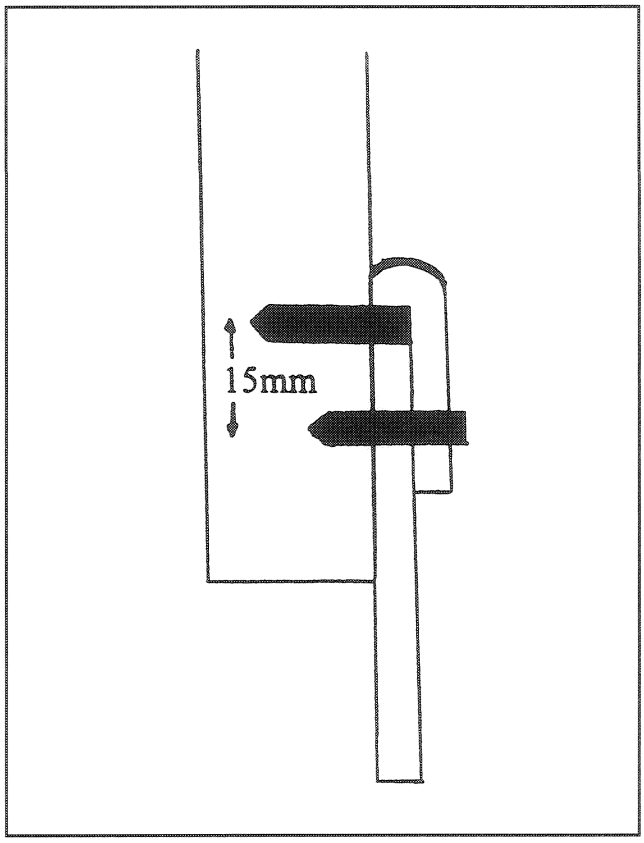

Figure 2. The double staple belt buckle soft tissue fixation technique. The tendon was fixed to the distal metaphysis of the femur using two 7.9 mm spiked staples by securing the proximal staple first. The tendon was then doubled back onto itself and the distal staple was inserted over the double thickness of the tendon 15 mm from the proximal staple. The free end was attached to the Cryojaw tissue clamp to perform the testing protocol. The distance between the distal staple and the Cryojaw tissue clamp was measured to be equal to the corresponding distance of the tendon used in the spiked washer figure-8 fixation technique.

The double staple belt buckle technique used two small (7.9 mm) Richards spiked staples which were placed in 2 mm pilot holes as per the manufacturer's recommendation (Figure 2). The 225 g weights were hung from each tendon, and the proximal staple was secured. The tendons were turned back towards the joint. Another staple was inserted 15 mm distally over the double thickness of tendons. The staples were seated to consistent depths as gauged by barbs on the staples. A caliper measured the depth of insertion after testing.

Both methods of fixation were evaluated by applying a load to failure at a rate of 25 mm/sec. Load, displacement, and time were recorded by a computer at 200Hz. The yield load was determined by performing a regression analysis on all data lying between 20 and 80% of ultimate failure load. Failure was taken as a 5% reduction in load from the regression line.

RESULTS

All specimens failed at the fixation site. The mechanism of failure was different for each technique. The tendons pulled through the spiked washers, shredding the tendon. A partial rupture occurred at the proximal staple, where the tendons turn 180 degrees. The ultimate failure load value of the double staple belt buckle technique (mean = 875 N) was found to be significantly greater than the double screw and spiked washer figure-8 technique (mean = 539 N); $p<0.005$. Similarly, the yield load was significantly greater for the staple technique (737 N) in comparison to the spiked washer technique (451 N); $p<0.005$ (Figure 3). The pattern of the load-displacement curve varied between techniques. The washer technique was found to yield slowly, the friction between the device and tendon keeping force almost constant. As the staple caused a tear in the tendon, an abrupt drop in load would occur. After this initial failure, the load would often

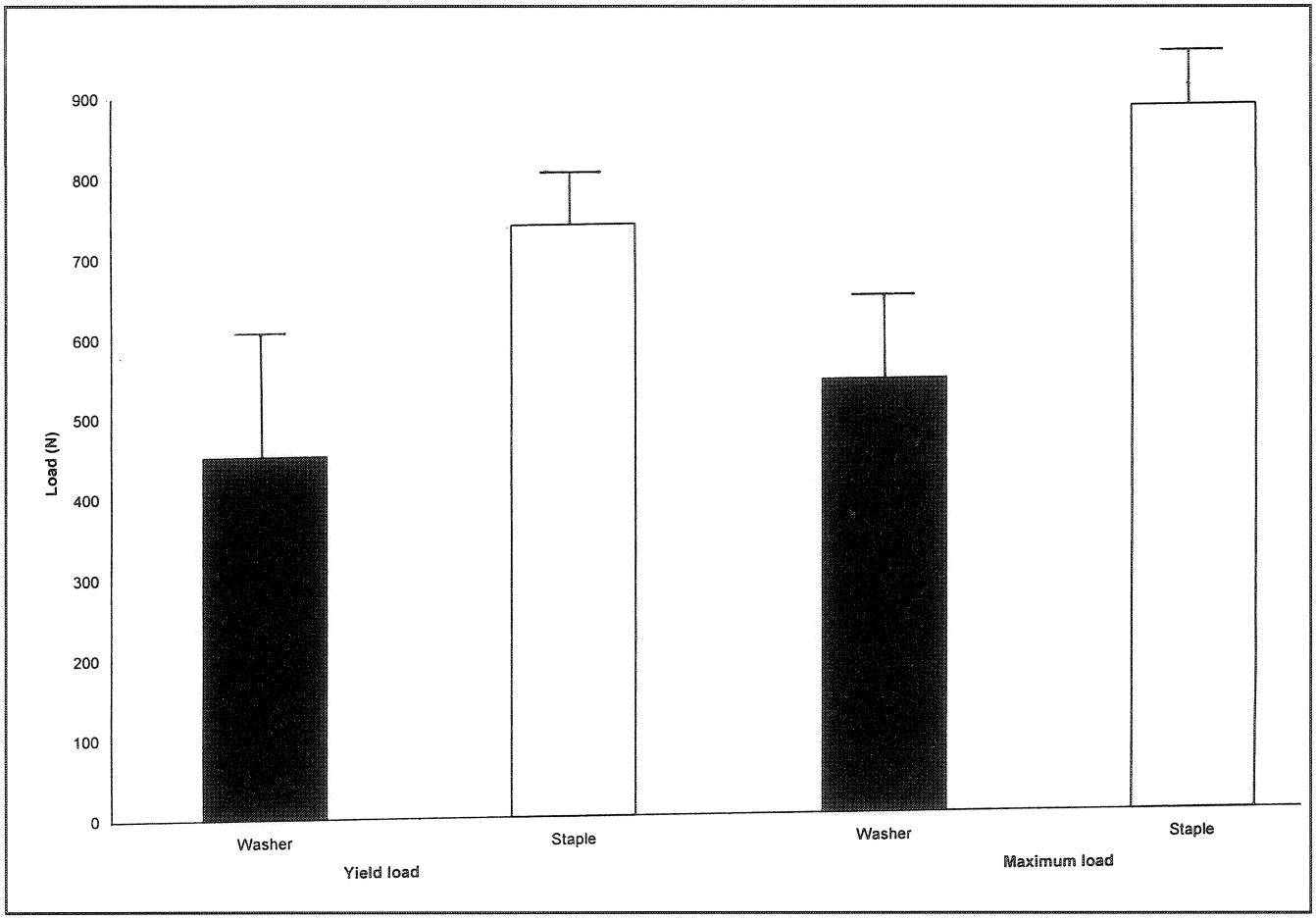

Figure 3. Mean failure loads (washer vs. staple)

increase above the yield point. The difference between maximum load and yield point was 88 N for the washer, 138 N for the staple technique.

DISCUSSION

Both techniques were satisfactory and compared favorably to the fixation measured *in vitro* using the interference screw technique. The double staple belt buckle fixation was found to be the stronger of the two methods for single cycle testing, both in maximum load and yield load. In comparison to previous work, our results did not present any significant discrepancies. The maximum load values for the double screw and spiked washer figure-8 technique compared favorably with the results reported by Steiner et al[6]. The previously reported results by Robertson et al. using similar tissue and a barbed staple probably differed significantly because we used a belt buckle technique with two staples instead of a single staple. They reported that the mechanism of failure was the tearing of the tissue from beneath the staple at the bony interface and not failure of the staple fixation to the bone. We also experienced failure of the staple technique by tearing of the tendon through the staple and not failure of the staple-bone interface. From the clinical perspective, the yield load is probably more relevant, as it represents the load at which permanent deformation occurs. The relative ability of these two methods to resist repetitive physiologic loads and to maintain strength over time still needs to be evaluated before definitive recommendations can be made.

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CHRONIC POST-TRAUMATIC HYPEREXTENSION INSTABILITY OF THE THUMB METACARPOPHALANGEAL JOINT: RESULTS OF THE VOLAR CAPSULODESIS PROCEDURE

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INTRODUCTION

Capsular injuries of the thumb metacarpophalangeal (MP) joint are common. If these injuries are misdiagnosed or inappropriately treated they can result in chronic disability with limited precision pinch and power grip. Chronic instability after a hyperextension injury to the thumb MP joint is well recognized but relatively uncommon. A hyperextension injury can result in a spectrum of injury that includes MP joint sprain, dorsal subluxation of the proximal phalanx, or a dorsal dislocation of the proximal phalanx. Subluxation or dislocation injuries result in avulsion of the MP joint volar plate and collateral ligament complex. When recognized acutely, the majority of thumb MP subluxations or dislocations can be successfully treated with appropriate immobilization and rehabilitation. However, despite appropriate treatment, some patients can present with a persistently painful, unstable thumb that limits their ability to pinch and grasp. Various operative techniques have been described to address such persistent symptomatic instability. In general, the results of these procedures has been satisfactory. We report two patients with post traumatic chronic hyperextension instability of the MP joint of the thumb who were successfully treated with a volar capsulodesis as originally described by Filler[4].

CASE REPORT

Case 1

A 28 year old right hand dominant man sustained a hyperextension injury to the metacarpophalangeal (MP) joint of his nondominant left thumb (Figure 1). Initial treatment consisted of closed reduction and application of a short arm thumb spica cast. Two weeks later the cast was removed, a removable thumb spica splint was fashioned, and a range of motion program was begun. Six weeks post injury, splinting was discontinued and the patient was allowed to resume activities as tolerated. He experienced persistent painful hyperextension instability of the thumb MP joint during activities that involved thumb pinch and grasp, and was subsequently referred to our institution for evaluation.

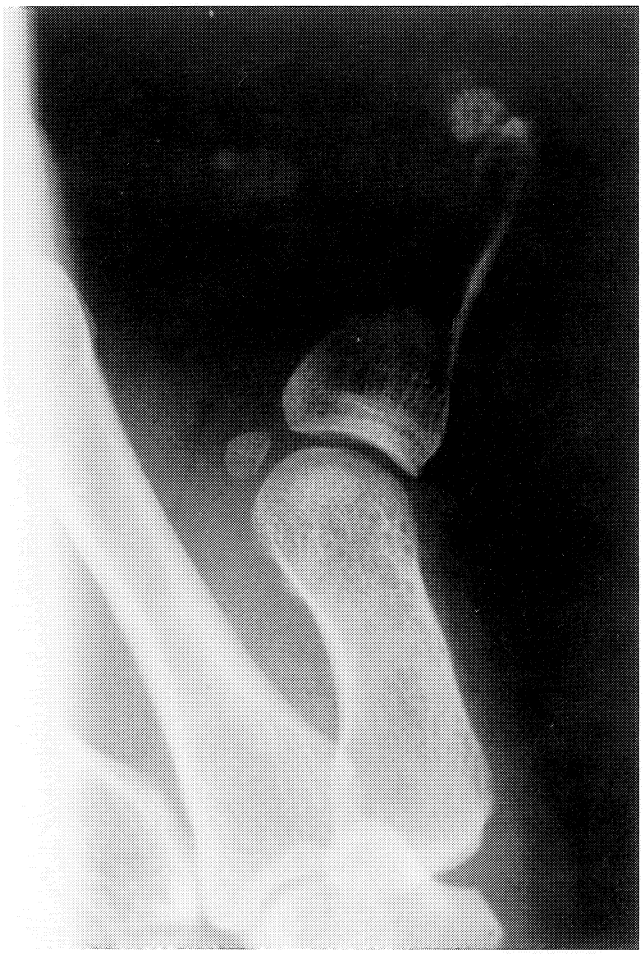

Figure 1. Lateral radiograph of the injured thumb. Note the hyperextension deformity of the metacarpophalangeal (MP) joint.

Physical examination revealed mild swelling of the thumb MP joint with pain and asymmetric volar instability on passive extension of the joint. The radial and ulnar collateral ligaments were intact. Persistent hyperextension instability with rupture of the volar plate was diagnosed and the patient was placed in a custom fabricated forearm based thumb spica splint maintaining the MP joint in 20 degrees of flexion. Two months later, the forearm

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splint was converted to a hand based splint that was worn only for protection during daily activities. The patient noted dramatic improvement in his symptoms. Three weeks after being placed in the splint he sustained a second hyperextension injury to the thumb when the splint was inadvertently torn off his hand. Thumb spica cast immobilization was reinstituted for one month but, following removal of the cast, the patient continued to experience MP joint pain and instability with activities of daily living.

Six months after his original injury, the volar plate was reattached to the first metacarpal and the joint temporarily stabilized with a Kirschner wire. A short arm thumb spica cast was applied for six weeks. A gentle progressive range of motion program was then begun. Four months postoperatively the patient was permitted to return to work.

At the six month follow-up, the patient denied any MP joint pain or instability. Physical examination revealed a grip strength of 30 kilograms (kg) on the involved left side compared with 42 kg on the dominant right side. Key and chuck pinch strengths were 6.2 kg and 6.0 kg respectively on the left and 9.0 kg and 8.0 kg respectively on the right.

Case 2

A 24 year old right hand dominant man presented with a two year history of pain and instability at the MP joint of his nondominant left thumb following a forced hyperextension injury to the thumb. Original treatment consisted of splinting for one week followed by unrestricted resumption of all activities. He continued to experience MP joint pain, weakness, and instability with pinch and grasp. Intermittent periods of splinting were performed without improvement in his symptomatology. He presented to our institution two years following the original injury with the aforementioned complaints limiting his ability to work and perform activities of daily living.

Physical examination revealed pain with passive hyperextension of the thumb MP joint. The MP joint could be passively hyperextended approximately 45 degrees as confirmed on a hyperextension stress radiograph (Figure 2). This finding was not present on the contralateral asymptomatic extremity.

Because of the persistent pain and instability, the MP joint was explored and the volar plate reattached to the palmar aspect of the metacarpal neck with a pull out suture. A short arm thumb spica cast was worn for six weeks at which time the stabilizing wire was removed. A passive range of motion program was begun and three months post-surgery, the patient resumed all activities without restriction. At the six month follow-up, he denied any further episodes of painful instability and was pleased with his operative outcome.

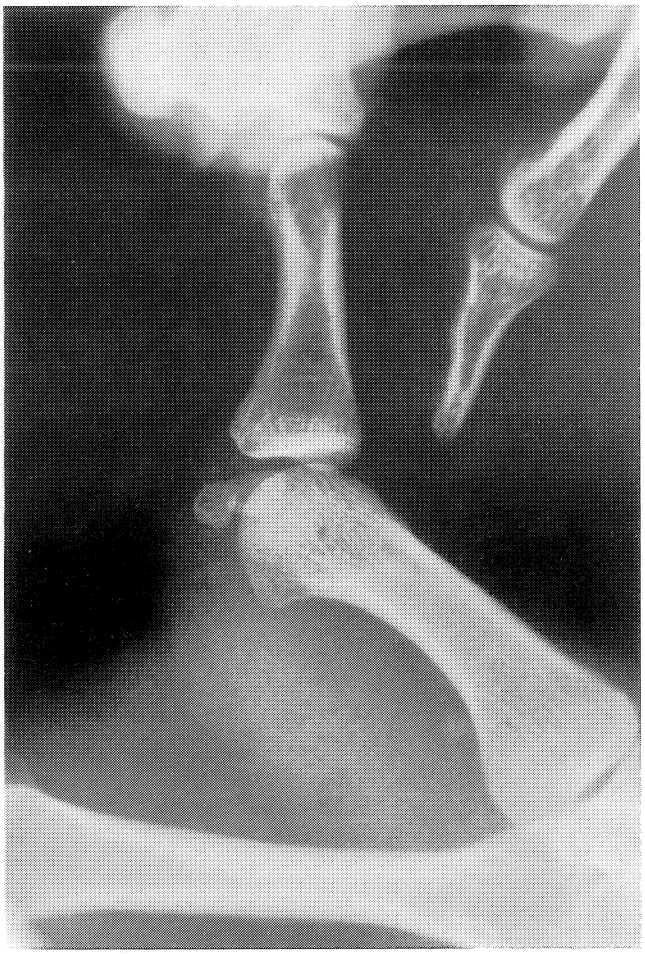

Figure 2. Hyperextension stress radiograph demonstrating volar instability of the MP joint.

OPERATIVE TECHNIQUE

A volar zig-zag incision centered over the metacarpophalangeal joint flexion crease is used to expose the flexor tendon sheath of the thumb. The neurovascular bundles are protected as the flexor pollicis longus (FPL) sheath is exposed and opened in the midline directly over the MP joint. The proximal portion of the A1 pulley is preserved. The FPL tendon is retracted and the volar plate and collateral ligament complex of the MP joint exposed. The volar plate is typically found to be avulsed off the metacarpal neck. Scar tissue is debrided to allow visualization of the MP joint and first metacarpal neck. The volar plate is defined and the joint inspected prior to reattachment of the volar plate. A 3-0 prolene suture is then placed through the volar plate using a modified Kessler technique. Parallel drill holes are created in the metacarpal neck in a distal-volar to proximal-dorsal direction. The volar aspect of the metacarpal neck is gently roughened with a burr or curette to create a cancellous bed, or trough, for volar plate adherence following reattachment. With the MP joint reduced and flexed approxi-

mately 30 degrees, a 0.045 Kirschner wire is drilled obliquely across the joint for stability. The prolene suture is then passed through the bone holes in the metacarpal neck. The volar plate is pulled snugly against the cancellous trough in the metacarpal neck and the suture is tied over a button placed on the skin overlying the metacarpal neck. The A1 pulley is repaired followed by wound closure and the application of a short arm thumb spica splint. The sutures are removed at 10-14 days and the patient is placed in a short-arm thumb spica cast with the interphalangeal joint free for six weeks. At six weeks postoperatively, the Kirschner wire and cast are removed and a custom forearm based thumb abduction splint is fabricated. The patient is instructed in active and active-assisted flexion exercises of the MP joint until twelve weeks after surgery when they are permitted to resume all activities, including athletic participation, without restriction.

DISCUSSION

The anatomy of the thumb MP joint has been described in great detail[1,5,11,12,13,14]. The volar plate has a strong bony attachment distally at the base of the proximal phalanx but a loose fibrous attachment proximally at the metacarpal neck. The proper collateral ligaments insert into the lateral tubercles of the base of the proximal phalanx while the accessory collateral ligaments insert directly into the volar plate and sesamoids. As described by Stener, the MP joint of the thumb receives additional support and resistance to hyperextension from the surrounding musculotendinous structures including the adductor pollicis, which inserts on the ulnar sesamoid, and the flexor pollicis brevis, which inserts on the radial sesamoid[14]. Hyperextension instability of the MP joint is thus prevented by each of these ligamentous, soft tissue, and musculotendinous restraints. For persistent instability to occur, one or more of these structures must fail, as originally proposed by Stener[14]. By far the most common injury pattern responsible for chronic hyperextension instability is avulsion of the volar plate from the metacarpal neck. Occasionally, the volar plate is avulsed from the base of the proximal phalanx. Rarely, hyperextension instability can result from fracture of one of the sesamoids.

Chronic volar instability of the thumb involves painful MP joint hyperextension that on examination is greater than the asymptomatic contralateral side. Thus, physical examination should always include both thumbs. Acutely, well localized tenderness and swelling on the volar aspect of the MP joint is usually present in the involved thumb. Stress examination, by definition, must reproduce the patients symptoms and complaints of pain, weakness, and instability. Increased passive hyperextension with lack of a firm end point is always present in these patients. The stress examination may need to be performed under anesthesia and should always be performed with the interphalangeal joint flexed to relax the flexor pollicis longus tendon. Failure to do so may mislead the examiner.

Evaluation of the patient with possible chronic instability following a hyperextension injury to the MP joint of the thumb should always include radiographs. Radiographs are necessary to rule out sesamoid fracture, assess for degenerative changes, and to determine the location of the volar plate avulsion. Stener proposed that if the sesamoids are located adjacent to the proximal phalanx on a stress view, the volar plate avulsion is proximal to the sesamoids. If they remain adjacent to the metacarpal, the volar plate avulsion is distal to the sesamoids[14].

A hyperextension stress view is often helpful in confirming the diagnosis of instability (Figure 2). Close evaluation is necessary to rule out sesamoid fracture as a cause of volar plate disruption and subsequent instability. Oblique views may be necessary to enhance visualization of the sesamoids[7].

If a hyperextension injury of the MP joint is recognized acutely, initial treatment involves closed reduction, using the method described by Green, followed by six weeks of cast immobilization[5]. Operative treatment is indicated only for irreducible, or complex dislocations[6,7,15]. The majority of patients recover uneventfully. The incidence of persistent MP joint instability is unknown. However, Coonrad reported that 2 of his 26 patients with acute dorsal dislocations that were initially treated with immobilization demonstrated findings and symptoms of chronic hyperextension instability[1]. Ishizuki also had two patients, in their series of 26, who required operative treatment following failed conservative treatment of hyperextension instability of the thumb MP joint[7].

Initial management of chronic instability should include analgesic and/or anti-inflammatory medication, immobilization, and activity restrictions[1,11,12]. Surgery is indicated only if conservative management fails in a cooperative and compliant patient.

Various techniques have been advocated for the surgical correction of volar instability of the metacarpophalangeal joint of the thumb. Milch described the use of a free tendon graft woven between the metacarpal and proximal phalanx to act as a check-rein to hyperextension[10]. The volar capsulodesis procedure was originally described by Zancolli who performed the operation in patients with instability of the digital MP joints secondary to ulnar nerve paralysis[16]. Using this principle, Stener recommended repair of the volar plate in acute hyperextension injuries to the thumb MP joint[14]. Filler successfully performed a volar capsulodesis in 13 cerebral palsy patients with MP joint hyperextension instability[13]. He recommended temporary Kirschner wire stabilization of the MP joint in this particular patient population. Kessler advocated strength-

ening the volar plate with the extensor pollicis brevis tendon weaved through the MP joint[9]. This method of joint stabilization with a local soft tissue restraint was found to be excellent in terms of resisting the deforming hyperextension forces. Similarly, Eiken proposed using the palmaris longus as a free graft to stabilize the MP joint[3]. Posner described advancement of the conjoined tendon of the abductor pollicis brevis and flexor pollicis brevis, claiming to increase MP joint stability by increasing flexion forces[12]. Eaton advocated suturing only the radial portion of the volar plate to the dorsal aspect of the thumb to address the compensatory hyperextension of the MP joint associated with trapeziometacarpal arthritis[2]. However, the durability of this procedure has been questioned.

The volar capsulodesis procedure has gained renewed interest with several series reporting excellent results. Schuurman performed ten volar capsulodeses in eight patients with volar instability secondary to trauma or cerebral palsy. At a minimum six month follow up, all thumbs were found to be stable and pain free[13]. Ishizuki treated two patients with post-traumatic instability with a volar capsulodesis using a pull-out wire. He described satisfactory results in both cases. The outcome of our two patients adds further credibility to the conclusions of these authors.

The volar capsulodesis procedure is an excellent operation to correct hyperextension instability of the thumb MP joint. The operation is logically sound in that the normal anatomy is restored without the need for ligament transposition or transplantation. The operation is technically challenging and we prefer to use a pull-out suture that is protected by temporary Kirschner wire stabilization of the MP joint. Alternatively, a pull out wire or one of the many commercially available suture anchors may also be used. Although we recognize that some MP joint extension is sacrificed with this technique, this has not resulted in any functional limitations or complaints in our patients. The volar capsulodesis procedure has proven to be durable with excellent patient satisfaction and no report of persistent instability.

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MASTOCYTOSIS PRESENTING AS A SKELETAL DISORDER

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ABSTRACT

Mastocytosis is a rare disease of mast-cell proliferation with involvement of the reticuloendothelial systems including skin, bone, gastrointestinal tract, liver, lungs, spleen, and lymph nodes. Systemic mastocytosis is characterized by a combination of symptoms that relate to the mast cells' release of vasoactive substances, such as histamine. These symptoms include urticaria pigmentosa, flushing, syncope with hypotension, headaches, nausea, vomiting, diarrhea, and occasional bronchospasm. The diagnosis of mastocytosis is typically based on the presence of the characteristic extraosseus manifestations. A well recognized roentgenographic feature seen in 70-75% of patients with mastocytosis is diffuse osteolysis and osteosclerosis, affecting primarily the axial skeleton and the ends of the long bones. Rarely, the bony involvement consists of generalized osteoporosis, which may lead to pathologic fracture, or solitary lesions (mastocytomas) which may cause symptoms of localized pain. Four patients with previously undiagnosed systemic mastocytosis had unusual skeletal lesions. Clinical and laboratory evaluation of these patients eventually led to the correct diagnosis of systemic mastocytosis. We report these four cases to emphasize the need for thorough evaluation of unusual musculoskeletal findings in association with extraosseus symptoms that are characteristic of mastocytosis. Knowledge of a wide differential diagnosis of unusual skeletal lesions should include systemic mastosytosis.

INTRODUCTION

Mastocytosis is a rare disease of unknown etiology, characterized by mast cell proliferation in skin and other reticuloendothelial systems including lymph nodes, spleen, liver, bone, bone marrow, gastrointestinal tract, and lungs[27]. It most commonly exists in a cutaneous form, urticaria pigmentosa, characterized by a violaceous maculopapular rash over the neck and trunk. The rash demonstrates dermatographism, and urticates with stroking, a phenomenon known as Darier's sign.

Urticaria pigmentosa is more frequently seen in children and resolves spontaneously by puberty. In approximately 10-30% of cases the disease may persist into adulthood, developing into the systemic form[27]. The disease rarely presents following adolescence.

Systemic mastocytosis is characterized by mast cell proliferation in several organs of the reticuloendothelial system and is often associated with clinical features attributable to histamine release, such as episodic flushing, hypotension, headaches, nausea, vomiting, and diarrhea. Approximately 94% of patients with systemic mastocytosis have urticaria pigmentosa[27]. The systemic form may give rise to malignancy in the form of leukemia or mast-cell sarcoma[9,15,17]. Patients with the adult onset form of the disease are thought to have a higher risk of developing leukemia[17].

Skeletal lesions occur in 70-75% of patients with systemic mastocytosis. This association was first described by Sagher et al.[26] in 1952. The most common pattern of bony involvement is mixed osteosclerosis and osteolysis, affecting primarily the axial skeleton, pelvis, and proximal ends of long bones. The lesions can be mistaken for metastatic disease, Paget's disease, hyperparathyroidism, lymphoma, or myelofibrosis. Skeletal lesions in systemic mastocytosis demonstrate increased uptake in technetium-99 bone scans, particularly when the lesions involve the proximal ends of long bones[31,34]. Although bone pain is a relatively frequent symptom, pathologic fractures are rare[23]. The pattern of multiple mastocytomas occurring as discrete lytic lesions surrounded by a sclerotic halo has been described. Rarely a patient may present with a solitary mastocytoma[26]. The diagnosis of skeletal lesions is typically a secondary finding in patients with known mastocytosis.

Due to the often nonspecific nature of the symptoms of systemic mastocytosis, patients with this disease may go undiagnosed for many years until the disease manifests itself in the form of skeletal lesions. These lesions may cause symptomatic bone pain, or may be discovered as an

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incidental finding on a routine roentgenographic exam. The evaluating orthopedist is thus faced with a diagnostic dilemma and should consider systemic mastocytosis when treating patients with unusual skeletal lesions and extraosseous symptoms characteristic of this disease.

The following four cases are examples of mastocytosis presenting as a skeletal disorder. None of the patients had known disease prior to being seen by the orthopedist. They all demonstrated unusual clinical and roentgenographic findings that with further laboratory and clinical investigation, led to the diagnosis of mastocytosis.

CASE REPORTS

Case 1:

A 52-year-old woman with a history of three prior thoracic spine compression fractures and osteoporosis was admitted for evaluation and treatment of acute back pain. Her past medical history was significant for a total abdominal hysterectomy and oophorectomy at the age of 48. She gave a 15-year history of a violaceous, maculopapular rash over the neck and trunk which was often pruritic and aggravated by sun exposure, stress, alcohol, and friction. She had been treated in the past for peptic ulcer disease and asthma.

Physical examination was remarkable for a brownish-red maculopapular rash over the neck and trunk that urticated with stroking. She had mild hepatosplenomegaly and expiratory wheezes. Her thoracolumbar spine was tender to palpation and painful with ambulation and forward bending. Roentgenographs revealed granular irregularity throughout the axial skeleton and pelvis with mixed areas of osteosclerosis and osteolysis, consistent with diffuse metastasis or metabolic bone disease (Figure 1). Laboratory studies revealed a slightly elevated alkaline phosphatase and no other abnormalities. A technetium-99 bone scan revealed increased activity throughout the axial skeleton and pelvis with some uptake in the hips and distal femora. The presumptive diagnosis at this time was widespread metastatic disease of unknown primary. A transiliac bone biopsy revealed mast-cell proliferation with fibrous changes in the bone marrow. Toluidine blue staining confirmed the presence of mast cells with their purple staining histamine-rich granules. At this time, a 24-hour urine collection was markedly positive for histamine, 70 μg/24h (normal < 50 μg/24 h).

Six years after the diagnosis the patient suffered a minor fall, resulting in a supracondylar femur fracture. Her hospital course was complicated by a flare in her disease, resulting in marked urticaria and bronchospasm. Prior to undergoing open reduction and internal fixation of her femur fracture, the patient was treated with cimetidine and chlorpheniramine, which resulted in resolution of the bronchospasm and urticaria. The femur fracture healed uneventfully and two years later she was pain free and ambulating without assistance.

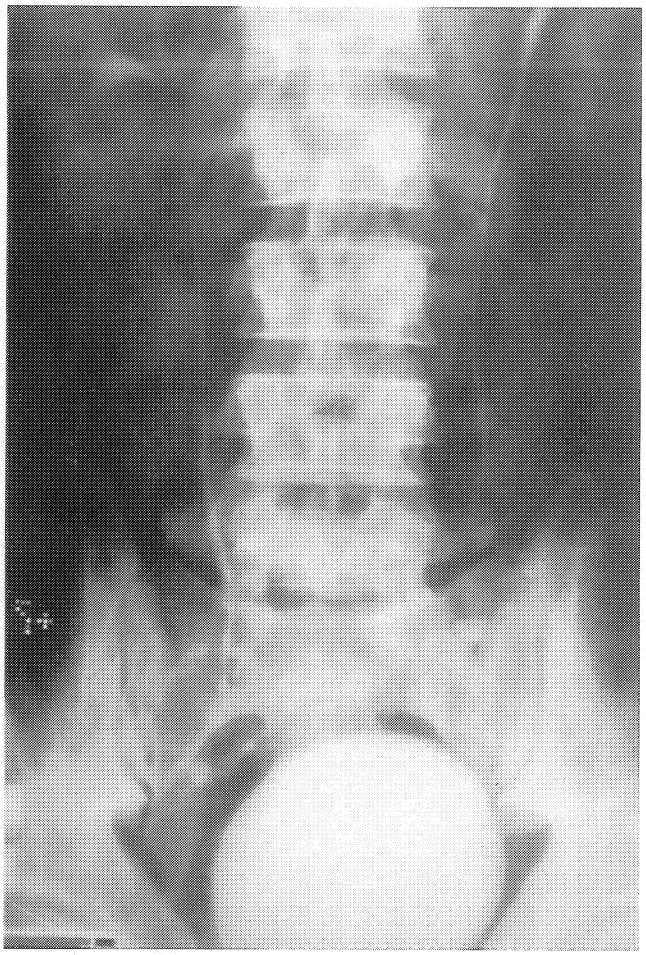

Figure 1. An anteroposterior roentgenograph of the lumbosacral spine at time of intravenous pyelogram reveals granular irregularities with mixed osteosclerosis and osteolysis.

Case 2:

A 62-year-old woman was evaluated for complaints of low back pain. She denied any particular history of trauma and described the onset as gradual. Her past medical history was significant for occasional nausea and vomiting with crampy abdominal pain, diarrhea, and peptic ulcer disease. She was treated for this with a Bilroth II procedure. She gave a history of easy bruisability and cutaneous flushing associated with a rash over the neck and trunk present for over 20 years. She stated that the rash was exacerbated by stress, heat, extreme cold, and ingestion of alcohol.

Routine laboratory studies were all within normal limits. Roentgenographs of her spine and extremities revealed diffuse osteoblastic and lytic lesions, consistent with metastatic disease (Figures 2A-C). A chest roentgenograph was unremarkable. She underwent a transiliac bone biopsy

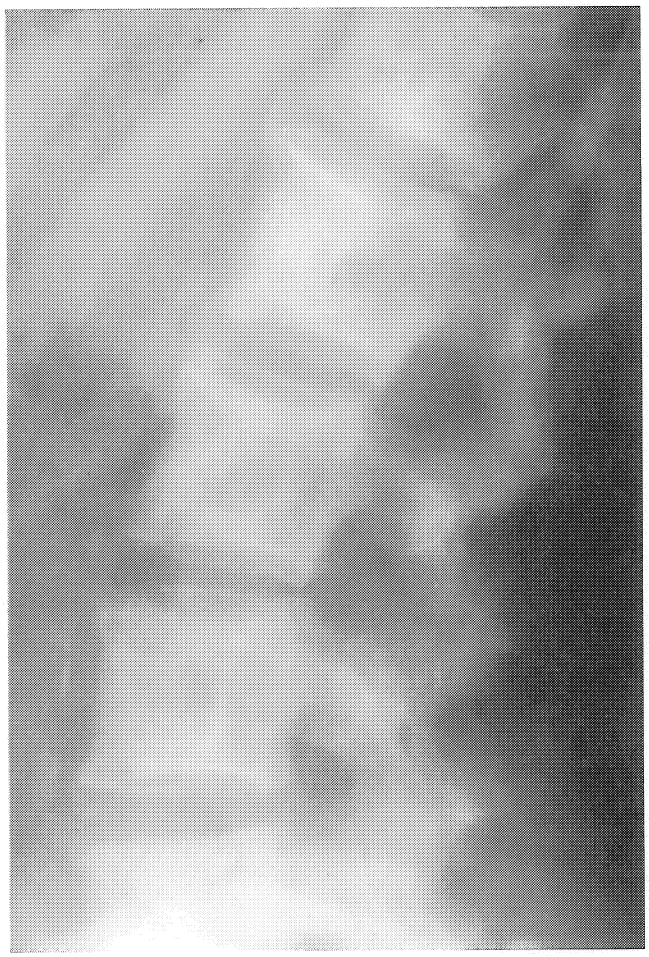

Figure 2A

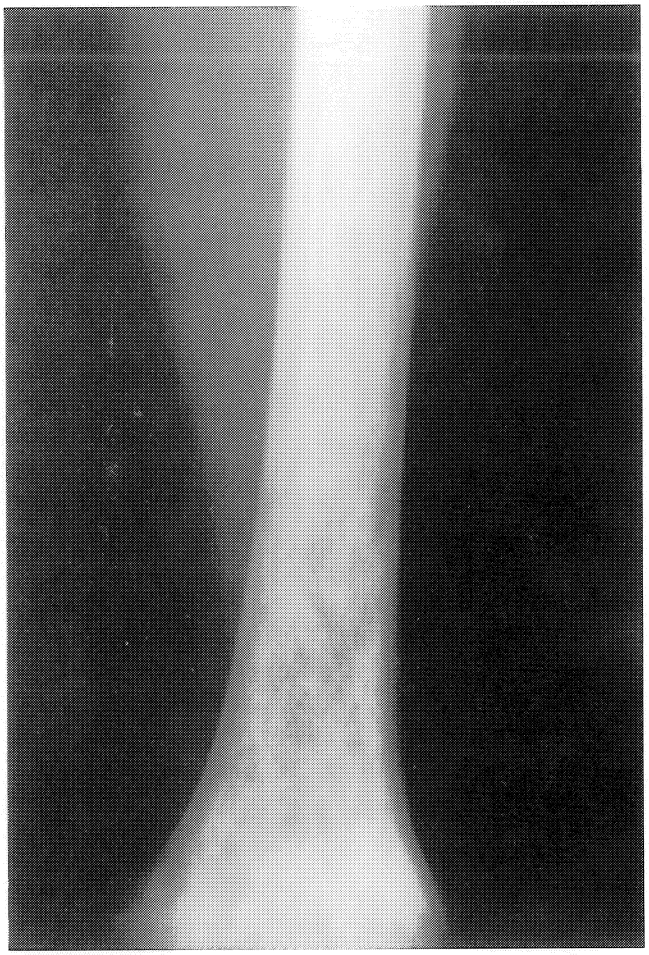

Figure 2C

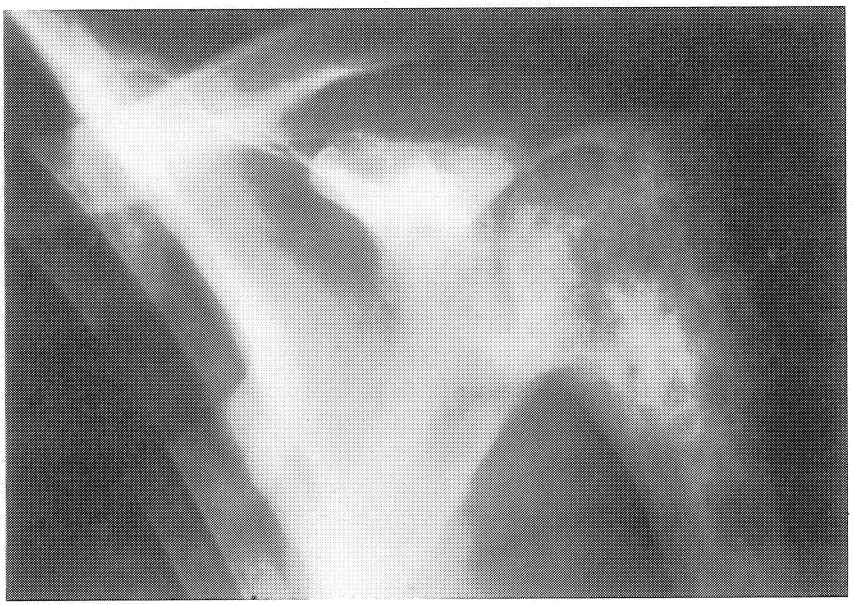

Figure 2B

Figure 2. (A) A lateral roentgenograph of the lumbar spine reveals diffuse osteoblastic and osteolytic changes. (B) Roentgenographs of the proximal humerus and scapula, and (C) distal femur exhibit a permeative pattern of patchy sclerosis and osteolysis with no evidence of cortical expansion or destruction.

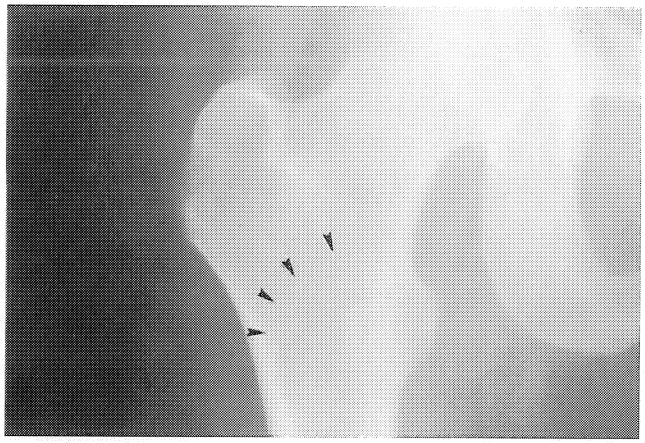

Figure 3A

Figure 3B

Figure 3. (A) Anteroposterior and (B) lateral radiographs of the proximal femur reveal a lytic lesion surrounded by a sclerotic margin, with no cortical disruption, periosteal reaction, or soft-tissue mass. Location of the lesion highlighted by arrows on both views.

which was interpreted as a spindle-cell-sarcoma. She was treated with chemotherapy for two years with no apparent clinical or roentgenographic progression of the disease. Subsequently, a repeat biopsy was interpreted as myelofibrosis with no evidence of malignancy. Chemotherapy was discontinued and the patient's back pain was treated symptomatically. She was presumptively diagnosed as having an atypical form of myelofibrosis without the usual hepatosplenomegaly, anemia, and thrombocytopenia.

During the subsequent five years, the patient required multiple admissions for back pain, nausea, vomiting, and crampy abdominal pain. In order to alleviate her symptoms of bone pain, she was treated with calcitonin with no improvement. Repeat bone scan and roentgenographs revealed no change from prior studies. The differential diagnosis at this time included Paget's disease and myelofibrosis with osteosclerosis even though the patient had normal alkaline phosphatase and urinary hydroxyproline levels.

The patient was readmitted a year later with complaints of continued back pain and an urticarial rash. The rash was diagnosed as neurodermatitis and treated with antidepressants. She was subsequently admitted to the psychiatry service for chronic anxiety, hives, depression, and back pain. Eight years after her initial presentation a repeat biopsy showed marked fibrotic replacement of the bone marrow with numerous eosinophils and histiocytes.

The patient's roentgenographs were placed in a teaching file of myeloproliferative disorders where they were reviewed by one of the authors (J.D.). On chart review, the suspicion of mastocytosis was raised based on the patient's constellation of symptoms. The patient's original bone biopsy slides were recut and stained with toluidine blue, revealing classic mastocytosis with fibrous replacement of the marrow and early myeloid metaplasia.

Case 3:

A 35-year-old-woman was referred for evaluation of right hip pain and a lytic lesion in the proximal aspect of the right femur. The patient gave a history of the gradual onset of pain in the right inguinal area, which was exacerbated by weight bearing. She denied any history of asthma, gastrointestinal, vasomotor, or constitutional symptoms. She described the presence of an allergic rash over her neck and chest which was aggravated by exposure to sun, stress, and alcohol.

Prior to performing an incisional biopsy, routine screening laboratory tests were all unremarkable. Roentgenographs revealed a lytic lesion in the proximal femur, lateral to the lesser trochanter (Figures 3A and 3B). The lesion was surrounded by a sclerotic halo with no periosteal reaction, cortical disruption, or soft-tissue mass. A bone scan revealed an area of increased uptake that corre-

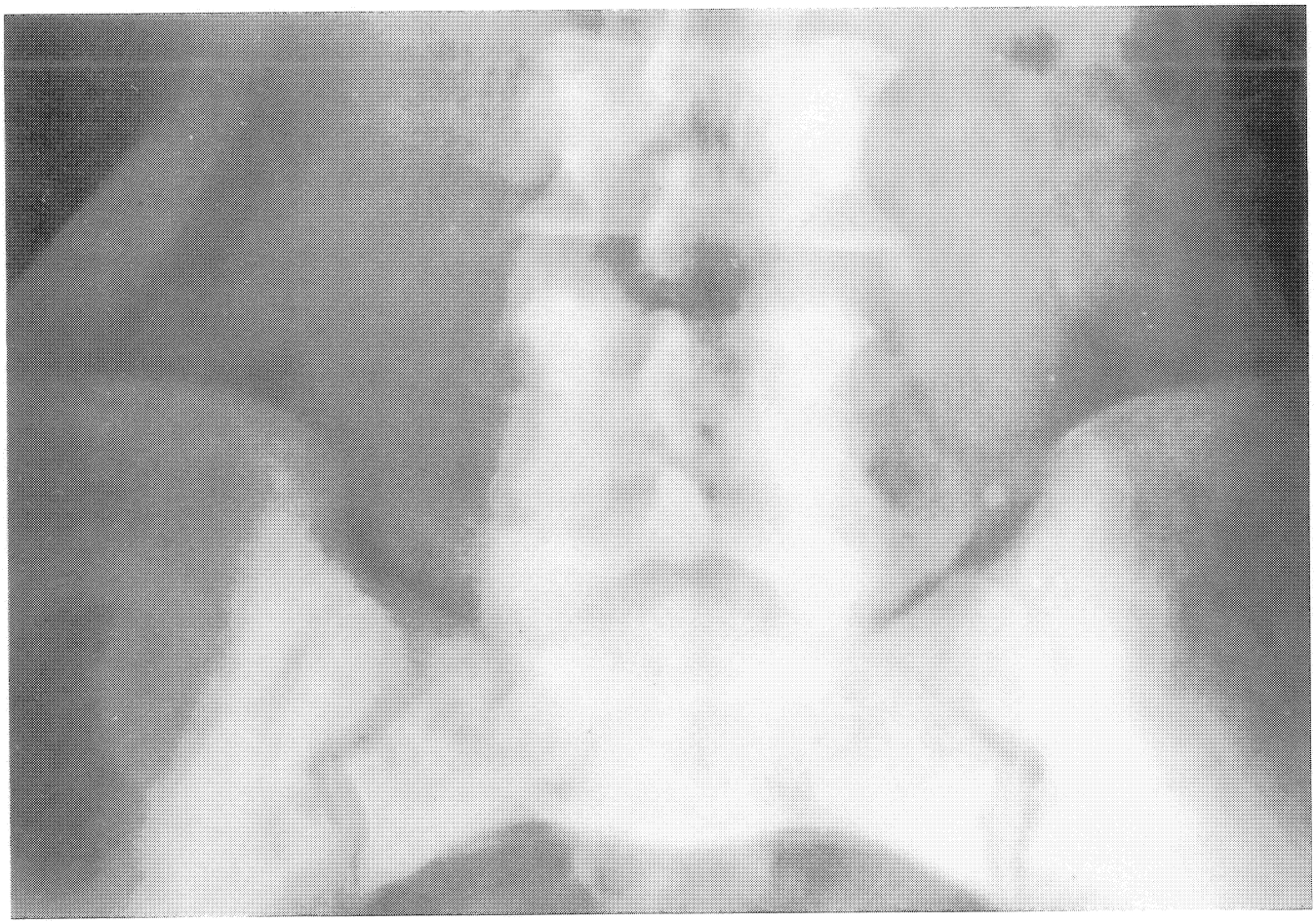

Figure 4. An anteroposterior roentgenograph of the lumbar spine and sacroiliac region of the pelvis reveals a diffuse osteoblastic pattern with focal lytic areas involving the lumbar vertebrae, sacrum, and posterior ilium bilaterally.

sponded to the lytic lesion. No other sites of abnormal uptake were noted. The lesion was further evaluated by computed tomography which confirmed the radiographic findings, showing the lesion to be lytic with sharply defined borders and no evidence of cortical destruction, periosteal reaction, or soft tissue mass extending from the lesion.

Tissue obtained from the incisional biopsy revealed a benign tumor with numerous histiocytes and eosinophils. The preliminary diagnosis was that of an eosinophilic granuloma, and the lesion was treated with curettage and bone grafting. Review of the permanent sections stained with hematoxylin and eosin showed that the cells thought to be histiocytes on frozen section were indeed mast cells. The specimen was then stained with toluidine blue and Giemsa stains which demonstrated the metachromatic purple-staining histamine granules within the mast cells, confirming the diagnosis of mastocytosis; existing in this case as a solitary mastocytoma. Following the tissue diagnosis, the patient's skin lesions were biopsied, revealing classic urticaria pigmentosa with mast-cell invasion in the dermis. Postoperatively, the femoral lesion healed with complete incorporation of the bone graft and resolution of symptoms.

Case 4:

A 67-year-old man was admitted for elective peripheral vascular surgery. His past medical history was significant for hypertension, diabetes mellitus, peripheral vascular disease, and a 30-year history of an urticarial rash, aggravated by alcohol and stress. He admitted having symptoms of occasional flushing, without syncope.

Except for elevations in serum triglyceride and cholesterol levels, admission laboratory studies were within normal limits. His admission chest roentgenograph raised the suspicion of metastatic or metabolic disease based on a permeative granular pattern seen in the ribs and thoracic spine. Further roentgenographic investigation of the spine revealed mixed osteosclerotic and osteolytic lesions throughout the vertebral bodies and posterior elements (Figure 4). The differential diagnosis at the time included fibrous dysplasia, Paget's disease, hyperparathyroidism, myelofibrosis, or diffuse metastatic disease. A bone scan

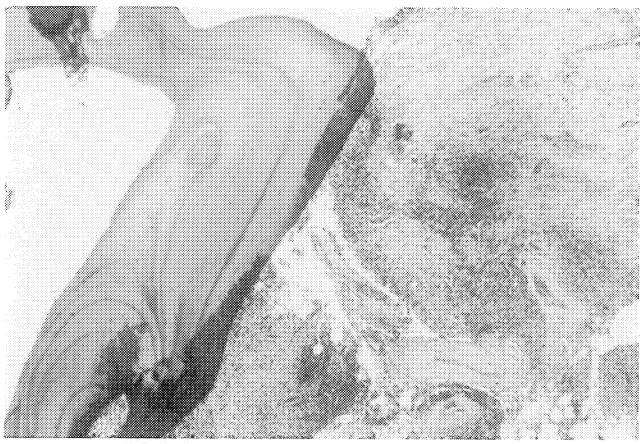

Figure 5A

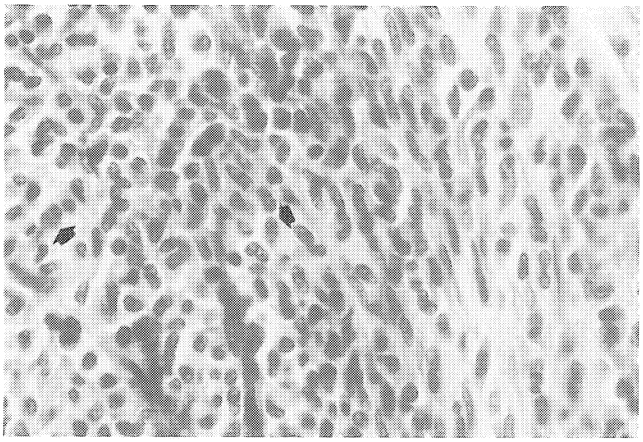

Figure 5B

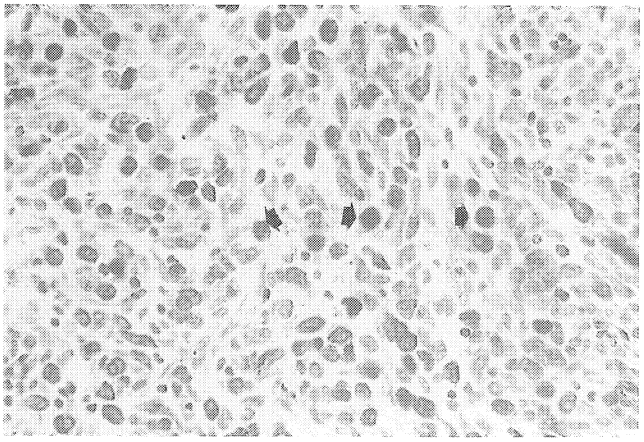

Figure 5C

Figure 5. (A) A photomicrograph of a bone marrow biopsy at low power shows mast cell proliferation, replacing marrow elements. Clumps of mast cells characteristically appear in peritrabecular and perivascular locations. (stain, Hematoxylin and eosin, original magnification = 100X.). (B) A high-power view reveals histiocytes and eosinophils replacing normal marrow elements, with occasional mast cells (arrows). (Stain, Hematoxylin and eosin, original magnification = 400X.). (C) After staining with toluidine blue, the mast cells are readily identifiable within the lesion (arrows). (original magnification = 400X).

revealed uneven, patchy uptake throughout the axial skeleton, ribs, and pelvis. The patient's elective surgery was postponed. He underwent bone marrow and iliac crest biopsy which demonstrated clumps of mast cells invading the bone marrow, with occasional eosinophils and fibroblasts. Subsequent biopsy of the patient's skin lesions revealed mast-cell invasion in the dermis, confirming the diagnosis of systemic mastocytosis.

DISCUSSION

Mastocytosis is characterized by mast-cell infiltration of skin and other reticuloendothelial systems. The cutaneous form of the disorder was discovered by Nettleship in 1869[21] and later termed urticaria pigmentosa by Sangster et al. in 1878[29]. Mastocytosis exists as variable clinical entities, from the more common cutaneous form seen in children that resolves spontaneously, to the systemic form with multiple organ involvement[16,20,35]. The symptoms of mastocytosis, whether in the cutaneous or systemic form, are primarily attributable to the mast cell's release of histamine and other vasoactive substances[24]. Recurrent flushing is a prominent feature of systemic mastocytosis and is occasionally associated with hypotension and syncope.

Table 1
Mast Cell Mediators

Histamine	Platelet Activating Factor
Serotonin	Eosinophilic Chemotactic Factor
Leukotriene	Neutrophilic Chemotactic Factor
Heparin	Chondroitin 4 and 6-Sulfate
Chymase	Dermatan Sulfate
Prostaglandin D2	

The mast cell has been described as a unicelluar endocrine gland since it is capable of elaborating many substances that can cause significant systemic symptoms (Table 1)[12]. Mast cells are thought to originate from the monocyte lineage and can be found in virtually any tissue or organ system. They have been identified in the endosteum of bones of calcium-deficient rats by special staining with hematoxylin-eosin-azure II[37]. The cell's prime mediator is histamine, which is stored in metachromatically staining secretory granules which are released by a host of stimulatory agents including sun, stress, alcohol, and narcotics.

Systemic mastocytosis may affect many organ systems, particularly the skin, skeleton, liver, spleen, gastrointes-

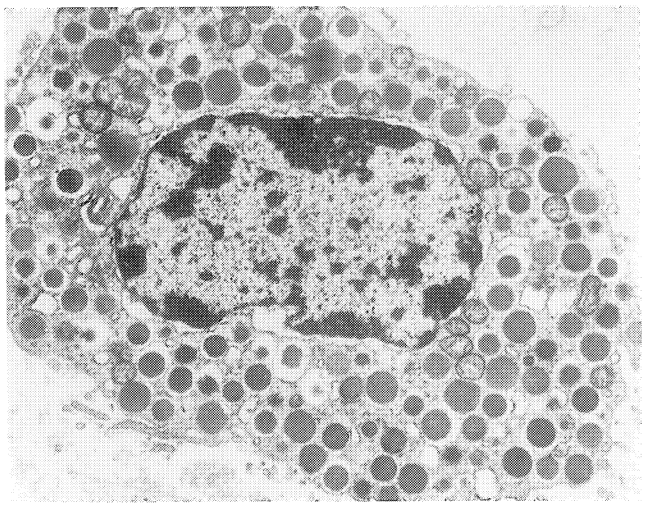

Figure 6. An electronmicrograph shows a typical mast cell with prominent secretory granules, (original magnification = 10,000X).

tinal tract, and lungs. The degree of organ involvement may be variable with the most frequent combination involving the skin, hematopoietic organs, and skeleton[27]. The diagnosis of mastocytosis is typically based on the presence of the aforementioned clinical features and is confirmed by skin or bone marrow biopsy.

The histologic appearance of bone marrow in systemic mastocytosis characteristically reveals clumps of mast cells in peritrabecular and perivascular locations[33] (Figure 5A). The cellular infiltrates can be confused with eosinophilic granuloma, lymphoma, or myelofibrosis (Figure 5B), as identifying features of mast cells can be subtle. Mast cells contain numerous secretory granules which exhibit metachromatic properties, displaying a purple hue when stained with toluidine blue or Giemsa stains (Figure 5C). Without using these special staining techniques, the presence of mast cells may be overlooked or misdiagnosed as myelofibrosis. On electron microscopy, mast-cell granules are a prominent and readily identified feature (Figure 6). Mast cells are found throughout the marrow along with fibroblasts, eosinophils, and histiocytes, resembling histiocytosis X or eosinophilic granuloma. Some areas may be mistaken for myelofibrosis or agnogenic myeloid metaplasia[4,36,39]. Reticulin staining of such a specimen would prove markedly positive for reticulin fibers, which may lead to an incorrect diagnosis of myelofibrosis[17,36]. Biopsy material may be mistaken for malignant lymphoma although Reed-Sternberg cells that may be seen in Hodgkin's lymphoma are characteristically lacking. Histologic examination of the skin reveals marked mast-cell infiltration in the dermis and subcutaneous tissue.

Additional laboratory values that aid in establishing the diagnosis include elevated urinary histamine excretion, mild to moderate eosinophilia, anemia, and thrombocytopenia. Liver function studies and coagulation profiles are normal and urine analysis for vanylmandelic acid and catecholamines, to rule out pheochromocytoma, is negative.

Cutaneous lesions, most commonly in the form of urticaria pigmentosa, are the most overt manifestation of systemic mastocytosis, being present in greater than 90% of cases. These lesions typically flare with sun exposure, stress, and ingestion of alcohol, spicy foods, or narcotics. The presence of such lesions may lead the physician to suspect the diagnosis and search for other organ involvement. In many cases, the skin lesions are overlooked and very rarely may be absent, leading to a delay in diagnosis. Skin lesions were present in each of these cases, but were considered inconsequential, and hence overlooked.

Skeletal lesions occur in 70-75% of patients with systemic mastocytosis. This association was first described by Sagher et al in 1952 and subsequently has been documented by several authors[1,2,3,5,6,14,19,22,23,26,28,31,33]. The pattern of bony involvement is typically that of mixed osteosclerosis and osteolysis, affecting the axial skeleton and the ends of long bones. The lesions may resemble diffuse metastatic disease[2], thus leading to an incorrect diagnosis and treatment as in Case 2. Other diseases that may share a similar roentgenographic appearance include Paget's disease, hyperparathyroidism, lymphoma, myelofibrosis, and agnogenic myeloid metaplasia. Rarely, osseous involvement may present as solitary or multiple lytic lesions surrounded by sclerotic halos. This type of lesion is usually seen in the vertebral bodies and near the ends of long bones. Case 3 demonstrates this rare form of osseous involvement in the proximal femur. Generalized osteoporosis has been reported in patients with systemic mastocytosis and is thought to be related to elevated levels of heparin, a known secretory product of mast cells[13,27]. Despite elevated heparin levels, abnormal bleeding does not occur.

Skeletal lesions are frequently painful, possibly secondary to microscopic stress fractures. On occasion, the lesions may be asymptomatic as demonstrated in Case 4. Here, previously unrecognized systemic mastocytosis was diagnosed after investigation of skeletal lesions found incidentally on routine roentgenographs. Pathologic fractures are rare[23] and, as demonstrated in Case 1, heal uneventfully when treated by conventional means.

The lymphatic and hematopoetic systems are invariably involved in systemic mastocytosis. Splenic infiltration may result in mild splenomegaly with occasional anemia and thrombocytopenia, similar to but less severe than that seen in myelofibrosis. Abdominal and para-aortic nodes are invariably involved, as is bone marrow. Bone marrow is heavily involved with mast cell invasion in the majority of cases of systemic mastocytosis. Gastrointestinal involve-

ment may occur in patients with systemic mastocytosis. Some patients have symptoms of nausea, diarrhea, and crampy abdominal pain. Peptic ulcer disease is seen in 10% of patients with systemic mastocytosis[18]. This is thought to be due to increased gastric acid production stimulated by elevated levels of histamine. Mast-cell proliferation can be seen in the liver, with mild to moderate hepatomegaly. However, liver function studies usually remain within normal limits, and synthesis of clotting factors is rarely significantly compromised.

Pulmonary symptoms are infrequent, and range from mild rhinitis and bronchospasm to severe asthma. These symptoms are exacerbated by environmental allergens as well as the previously mentioned stimuli which cause flaring of the disease.

Determination of prognosis in mastocytosis is variable depending on the age of onset and the degree of systemic involvement. The most common form of the disease is present in childhood, involves only the skin, and resolves spontaneously by puberty. On rare occasion, the disease may persist into adulthood as the systemic form. Urticaria pigmentosa may present in young adulthood and is associated with a 15-30% risk of developing systemic disease[10,27]. Marked enlargement of the liver and/or spleen is associated with a poor prognosis for survival with a fatal outcome in 50% of cases[27]. Late adult onset of systemic mastocytosis can occur and is associated with the greatest risk of developing leukemia, lymphoma, or mast-cell sarcoma[8,15,17].

There is no known cure for systemic mastocytosis, hence therapy is directed towards symptomatic relief. This has been accomplished through the use of cromolyn sodium which stabilizes mast cell membranes[7,11,32,38], and the combination of the H1 and H2 antagonists, chlorpheniramine and cimetidine, which together act to block the effects of histamine by binding to histamine receptors[11]. Medical therapy is crucial during episodes of acute exacerbation of symptoms, and may at times include intravenous epinephrine for refractory symptoms of hypotension and bronchospasm[25]. Perioperative precautions must be taken to avoid administration of known mast-cell degranulators and to relieve emotional stress[30]. The patient in Case 1 suffered an acute flare of her disease preoperatively which was successfully managed with cromolyn sodium, chlorpheniramine, cimetidine, and inhaled bronchodilators. These agents were used throughout the perioperative period and discontinued at the time of discharge.

Mastocytosis is a rare condition with symptoms that may be overlooked or considered inconsequential by physicians who are unfamiliar with the disease. As demonstrated by the cases in this report, the skeletal changes seen in mastocytosis may be the first recognized manifestation of the disease, bringing the patient to the attention of an orthopedist. Quite frequently, the disease may be mistaken for other systemic or metastatic diseases. In treating patients with unusual roentgenographic findings, particularly in the presence of other manifestations of mastocytosis, one must be alerted to the possibility of such a systemic disease and ensure that the proper histologic techniques, such as Giemsa or toluidine blue staining, are performed to establish the correct diagnosis. A better awareness of this disease and its characteristic clinical features may aid in making the diagnosis and selecting appropriate treatment.

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UPPER EXTREMITY MUSCULOSKELETAL MANIFESTATIONS OF DIALYSIS-ASSOCIATED AMYLOIDOSIS

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"Dialysis-associated amyloidosis" is a recently identified complication of uremic patients undergoing long-term hemodialysis[1-6]. The inability of both cuprophane dialysis membranes to filter, and renal failure patients to catabolize the plasma protein β-2 microglobulin, results in the accumulation and subsequent conversion of this substance to amyloid fibrils. Amyloid is deposited predominantly in the musculoskeletal system in synovial, articular and periarticular tissue, resulting in a variety of conditions. The precise mechanism of β-2 microglobulin conversion to amyloid and the reason for the preferential accumulation of amyloid in the musculoskeletal system is unknown. The prevalence of these associated musculoskeletal conditions is directly proportional to the duration of dialysis. Thus, with the increasing length of survival of hemodialysis patients, those involved in their orthopaedic care will begin encountering this unusual, yet distinct, form of secondary amyloidosis. The purpose of this paper is to review the clinical, roentgenographic and pathologic features of dialysis-associated amyloidosis as seen in the upper extremity.

CARPAL TUNNEL SYNDROME

Many reports document a high incidence of hand pain and numbness in chronic renal failure patients on long term hemodialysis[7-15]. The proposed mechanisms for these symptoms include carpal tunnel syndrome (CTS) caused by edema of the flexor retinaculum, venous pooling associated with superficial vein valvular destruction distal to the fistula, and amyloid deposition in the transverse retinacular ligament. Hand numbness can also result from median nerve ischemia caused by radial steal syndrome or occur as part of the generalized peripheral neuropathy that is commonly seen in this patient population[16,17]. Although all of the above mechanisms are accepted and may occur independently or in association with one another, compression of the median nerve by amyloid in the transverse retinacular ligament (TCL), specifically by β-2 microglobulin deposits, is the most consistent finding and the most thoroughly investigated phenomena. Indeed, β-2 microglobulin is deposited in other tissues surrounding the median nerve, including tenosynovium and within the flexor tendons, but has not been found in the nerve itself or in the epineurium[18,19].

The incidence of dialysis associated carpal tunnel syndrome is directly related to the duration of dialysis therapy. The reported rates for patients who have undergone dialysis for less than 14 years of therapy are between 12% and 37%[14]. After 14 to 20 years of dialysis the incidence increases to 60%. Finally, in those patients surviving 25 years of dialysis, all have evidence of carpal tunnel syndrome[11,19].

Diagnostic evaluation includes neurophysiologic testing to assess median nerve conduction velocity across the wrist. Neurophysiologic testing is particularly helpful in differentiating the symptoms of carpal tunnel syndrome from other causes of pain and paraesthesias such as radial steal syndrome, which causes a painful ischemia of the digits deprived of shunted blood, or the generalized metabolic polyneuropathy that is seen in uremic patients[15].

The natural history of CTS in chronic dialysis patients is not known but the majority of studies indicate that compression of the median nerve is progressive. An exception is noted in those patients with dialysis associated CTS who undergo renal transplantation. Renal transplantation has been reported to improve CTS symptoms in isolated cases. The results of conservative treatment of CTS secondary to amyloidosis are also not well defined. Most studies suggest that surgical treatment is inevitable in the hemodialysis patient with CTS. Encouraging results have been reported by various authors. Those patients with a shorter duration of symptoms (defined as less than 2 years) appear to have a better outcome after surgery[15,20,21].

ARTHRALGIAS AND DESTRUCTIVE ARTHROPATHY

Destructive arthropathy is a common feature of dialysis associated amyloidosis[10,23,24]. This arthropathy appears to

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be related to β-2 microglobulin deposition in both the bone and surrounding soft tissues of the joint[23]. Congo red staining and immunohistology have documented amyloid β2-microglobulin deposition in synovial fluid and joint lining[18].

Amyloid has not been detected in destructive arthropathies involving the small joints of the hand[24]. However, the precise role of β-2 microglobulin in the pathogenesis of a destructive arthropathy is unknown. Joint involvement is usually symmetric and most frequently involves in the glenohumeral joint. Shoulder pain is the presenting symptom in 25%-50% of patients and is eventually present in up to 80% of patients. Other joints, including the thoracic and lumbar spine, knee, wrist, and small joints of the hands may also be involved[10,23,24]. Patients typically present with joint pain, swelling and loss of motion. Like many of the other musculoskeletal manifestations of hemodialysis associated amyloidosis, the prevalence of these various joint arthropathies appears to increase with the duration of dialysis, and has been reported to occur in up to 80% of patients who have been on dialysis for more than 10 years. Interestingly, renal transplantation has been shown to reduce the incidence of arthralgias. However, transplantation does not seem to reverse existing bony changes or prevent further joint deterioration once the initial cartilage destruction has occurred.

BONE CYSTS

Direct amyloid invasion with replacement of subchondral bone results in the formation of cysts that are often referred to as "intraosseous amyloidomas"[25]. These bone cysts are usually juxta-articular and appear as multiple lytic, well defined lesions that are invariably surrounded by a thin sclerotic margin. Cortical destruction and extension of the cyst to the articular surface can occur. The most common upper extremity "amyloidoma" locations include the distal clavicle, anatomical neck of the humerus, and carpus[15,24-27]. However, amyloid bone cysts have also been reported in the cervical spine, glenoid, radius, ulna, metacarpals and phalanges[15,24-27]. Carpal cysts tend to localize to the radial side and most commonly involve the scaphoid and lunate (Figure 1).

Amyloid bone cysts may be asymptomatic in up to half of involved patients. The cysts first appear between five and fifteen years after the commencement of hemodialysis and after ten years of dialysis, it has been estimated that 50-60% of all hemodialysis patients will have evidence of amyloid bone cyst formation. Pathologic fracture through amyloidomas have been reported in both the upper and lower extremities[27]. Most fractures heal uneventfully with standard treatment methods, however, nonunion can occur but seems to be unique to pathologic femoral neck fractures[27].

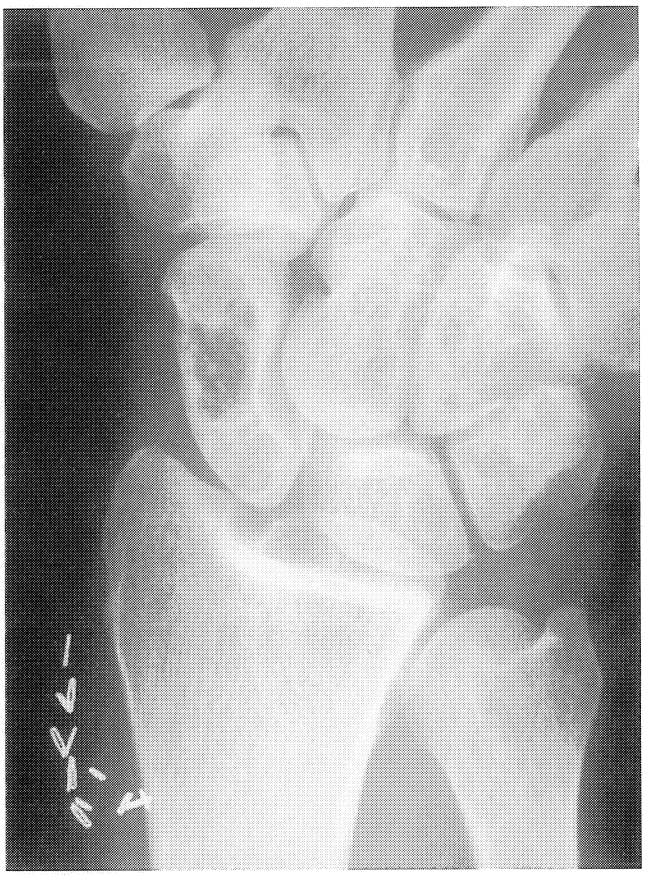

Figure 1. AP view of the hand demonstrating classic amyloid cysts throughout the carpus and distal radius. Note the large cyst in the scaphoid, a typical location and appearance.

The management of asymptomatic amyloid bone cysts is serial observation. Painful lesions may require curettage and bone grafting. The majority of cysts enlarge over time and an annual skeletal survey involving posteroanterior views of both hands and wrists and anteroposterior views of both shoulders and the pelvis has been recommended to avoid the morbidity of pathologic fracture[24,27] (Figures 2 and 3). The diagnosis of amyloid bone cyst formation is usually presumptive in the hemodialysis patient with the aforementioned characteristic radiographic features. However, definitive diagnosis may be required in atypical lesions and is usually performed via a computerized tomography guided biopsy. Congo red staining should be performed to identify amyloid deposition. Such specialized staining helps distinguish amyloidomas from brown tumors of secondary hyperparathyroidism, multiple myeloma, and metastatic disease which can also occur in the hemodialysis patient.

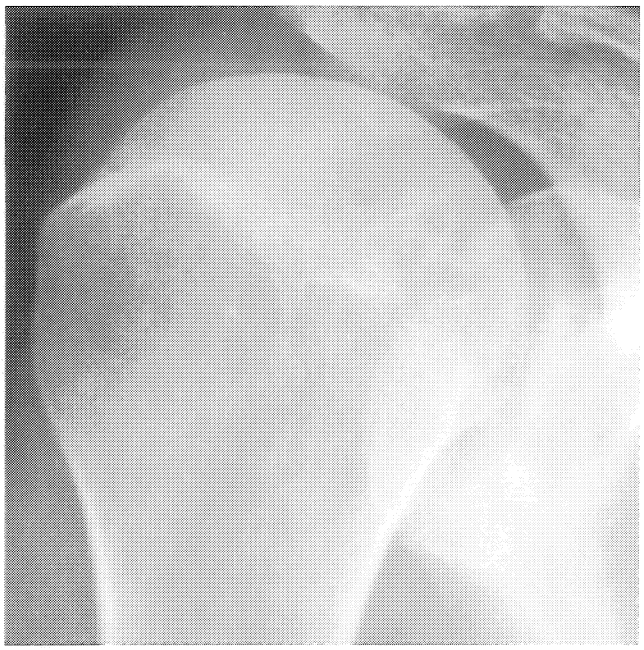

Figure 2. AP view of the shoulder illustrating biopsy proven amyloid cysts in the humeral head.

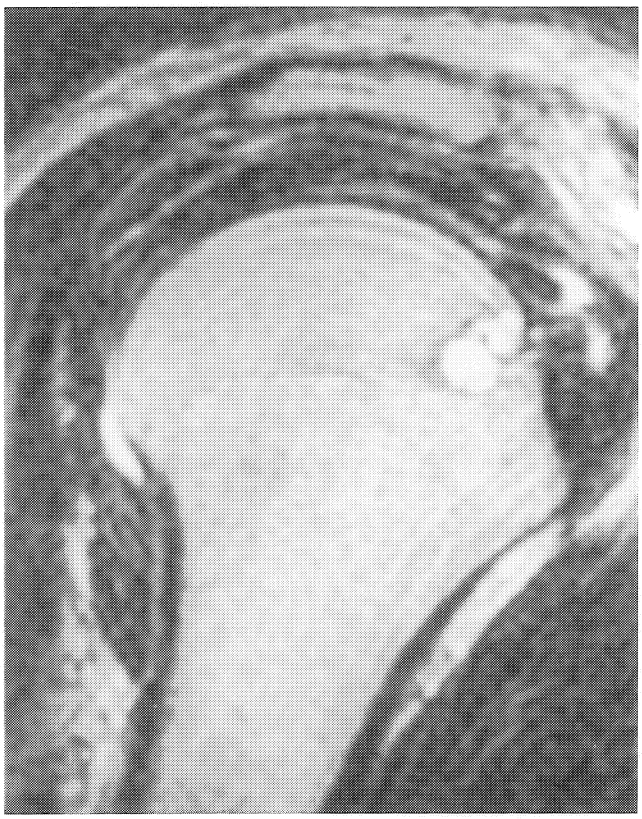

Figure 3. Corresponding MRI image of the patient in figure 2.

TENDON RUPTURE

Spontaneous tendon rupture is uncommon in dialysis associated amyloidosis, but has been reported by several authors. The largest series was reported by Kurer et al., who evaluated 83 renal failure patients who had undergone dialysis for more than 10 years. Six patients had various tendon ruptures; two involved the upper extremity with a digital flexor tendon and extensor tendon rupture. In both cases, the ruptured tendons were repaired primarily with good results. Interestingly, amyloid infiltration was identified at the site of rupture in both patients[15].

FLEXOR TENOSYNOVITIS AND JOINT CONTRACTURES

Flexor tenosynovitis is a common finding in dialysis associated amyloidosis patients. β-2 microglobulin is deposited in both flexor tenosynovium and the soft tissues of the palm. This palmar deposition results in a subcutaneous mass which produces displacement and prominence of the digital flexor tendons during range of motion. This has been referred to as the "guitar string sign" and supposedly, if seen in a renal failure patient on dialysis who has shoulder pain, is considered pathognomonic of β-2 amyloidosis[24].

Bardin evaluated 18 patients on chronic hemodialysis who had biopsy proven β-2 amyloidosis involving various components of the musculoskeletal system[3]. Four patients had tenosynovitis of the digital flexor tendons. Typical findings included palmar swelling, digital pain and limited extension of the involved digits. Ogawa performed Congo red staining on tissue obtained from 45 hemodialysis patients who had undergone a carpal tunnel release and flexor tenosynovectomy[28]. Approximately 80% of the tissue samples were positive, revealing the presence of amyloid fibrils.

Kurer identified four patients with flexor tendon contractures and finger stiffness from a series of 83 patients who had been dialyzed for a minimum of 15 years[15]. On surgical exploration, gross amyloid infiltration within the tenosynovium was found. The infiltration was so extensive that complete excision was often not feasible. Despite the significant improvement in hand function occurring in the majority of patients following partial amyloid excision, residual weakness and joint contractures were common[15].

Flipo identified two patients with destructive arthropathies of the metacarpophalangeal and interphalangeal joints who had associated flexor tenosynovitis[23]. The presence of both a destructive arthropathy and tenosynovitis was significantly debilitating and resulted in persistent pain and swelling and an inability to fully extend the involved digit.

TRIGGER FINGER

Digital triggering occurs secondary to amyloid tenosynovitis. Standard nonoperative treatment includes steroid

injection into the flexor tendon sheath and splinting. Recalcitrant cases often require surgical release of the A1 pulley of the flexor sheath combined with a tenosynovectomy. In chronic hemodialysis patients with carpal tunnel syndrome, triggering of the digits appears to occur at a significantly higher rate than in non-dialysis patients.

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SEVERE GROWTH RETARDATION FOLLOWING LIMB LENGTHENING: A CASE REPORT

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Anthony C. Carantzas, M.D.**

ABSTRACT

The first limb lengthening was reported by Codivilla in 1905. Common complications reported with this procedure include pin tract infections, angulation deformities, joint contractures, and residual limb length discrepancies. Growth arrest or severe growth retardation in the lengthened bones has rarely been reported. We report a case of a skeletally immature patient with right sided hemihypertrophy who underwent lengthening of the left lower extremity by the method of DeBastiani using Orthofix external fixators. Postoperatively he developed severe growth retardation in the lengthened limb, as evidenced by the Mosely straight line graph, and recurrence of his limb length discrepancy. The reason for this growth retardation remains unknown; however, we hypothesize that premature closure at one or more of the physes occurred because of increased tension across the growth plates. The practice of overlengthening a congenitally short limb prior to physeal closure should be questioned because resumption of growth after lengthening may be unpredictable.

The first limb lengthening was reported by Codivilla in 1905[5]. Numerous modifications have been reported since that time and all have been accompanied by frequent complications. Current methods of limb lengthening have reduced the number of procedures required, and have reduced the overall incidence of complications; however, complications continue to occur. Complication rates from 75-200% have been reported[4,14]. Common complications have been pin tract infections, angulation deformities, joint contractures, and residual length discrepancies[4,14]. Rarely has growth arrest or severe growth retardation in the lengthened bones been reported[15,19].

Pouliquen et. al. reported on children with poliomyelitis who underwent lengthening. They found that growth retardation or arrest occurred and was related to the percentage of lengthening. Eighty percent of patients who had lengthening of greater than 20% of the original bone length had a retardation or cessation of growth[15]. Shapiro found that the growth rate of the femur increased after lengthening, while that of the tibia decreased regardless of the percentage of lengthening[17]. A recent report by Sharma, Mackenzie, and Bowen also found a decrease in tibial growth rate after lengthening in patients with total fibular hemimelia. Femoral growth rates diminished as well[19]. The pathophysiology for these alterations in growth rate remains unknown. Several recent studies point to the increasing tension that develops across the physis as a possible mechanism of injury[9,13,16].

In this report, we describe a case of severe growth retardation following limb lengthening in a patient with right sided hemihypertrophy. This case calls into question the practice of overlengthening a short limb prior to physeal closure because resumption of growth may be unpredictable.

CASE REPORT

A six month old male was noted to have a lower limb length discrepancy (LLD) of two centimeters, right longer than left. At the age of six years and 3 months his LLD by scanogram measured 3.3 cm. Sequential scanograms and skeletal age determinations were obtained (Table 1) and plotted on a Moseley straight line graph (Figure 1). This predicted the patient would have a 5.5 to 6.0 cm LLD at maturity if left untreated. Possibilities of distal femoral and proximal tibial epiphysiodeses or limb lengthening were discussed. After several follow-up visits, the patient and his family elected to proceed with limb lengthening. At 13 years and 4 months, his LLD was 4.6 cm by scanogram. Lengthening of 6.0 cm was planned which allowed for 1.0 cm of overlengthening so predicted lengths would be equal at maturity.

At the age of 13 years and 6 months, simultaneous lengthening of the femur and tibia was initiated. The method of DeBastiani was employed using Orthofix (EBI, Parsippany, NJ) external fixators.

Within four days, he was discharged home on toe touch weightbearing. On postoperative day nine, 0.25 mm

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TABLE 1. Data of sequential leg lengths determined by scanogram, limb length discrepancies, and skeletal age.

Date	Leg Length By Scanogram (cm) Left	Right	Limb Length Discrepancy (cm)	Skeletal Age
7/84	56.0	59.6	3.6	7+0
6/85	59.0	62.4	3.4	8+0
8/86	62.7	66.7	4.0	10+0
8/87	65.0	69.0	4.0	11+0
4/88	66.6	70.8	4.2	*
11/88	68.5	72.8	4.3	11+6
7/89	70.0	75.1	5.1	11+6
11/89	70.9	75.5	4.6	12+6
4/90	72.6	77.2	4.6	12+6
	Surgery for lengthening Initial lengthening 3 cm/bone			
11/90			0.5 (L > R)	12+6
	Additional 0.5 cm lengthening			
3/91	80.0	80.6	.6	*
11/91	81.6	82.6	1.0	14+0
3/92	82.5	84.2	1.7	15+0
	Distal Femoral and Proximal Tibial Epiphysiodeses			
4/92	82.9	84.8	1.9	15+9
11/93	83.5	85.0	1.5	*

* = Data Not Available

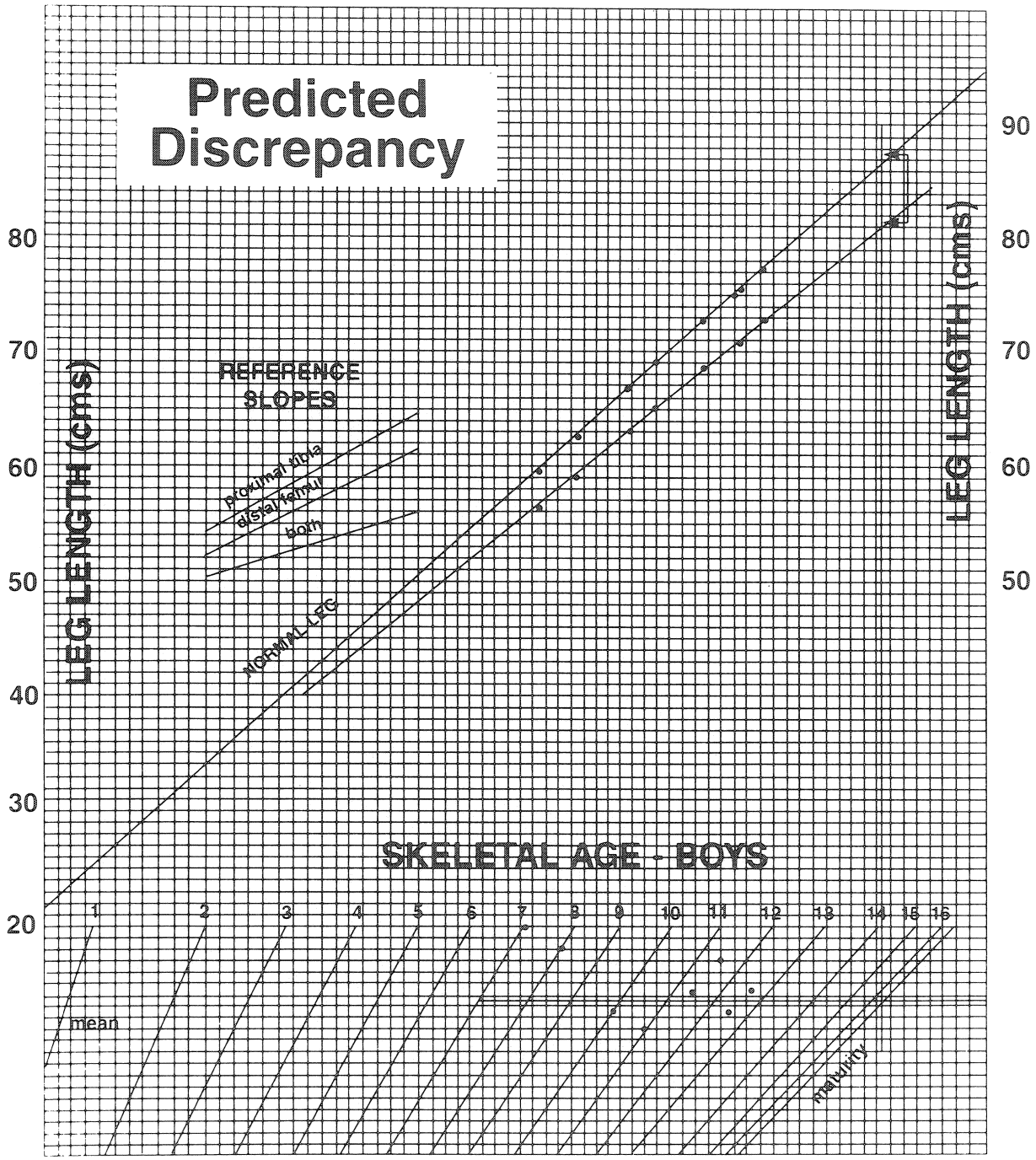

FIGURE 1. A Moseley straight line graph reveals expected age of skeletal maturity (vertical line) and expected limb length discrepancy of 5.5 to 6 cm (arrows) at skeletal maturity.

lengthenings were begun four times a day. Gentle knee range of motion exercises were instituted.

Four weeks postoperatively, the LLD was measured as 1.5 cm by scanogram. He was partial weight-bearing with crutches. A mild pin tract infection was present, treated with oral antibiotics, and resolved. Lengthening was continued.

Six weeks postoperatively, scanograms revealed that each bone had been lengthened 3.0 cm for a total limb lengthening of 6.0 cm. Good callus was noted at this time. The distraction bar was removed and a dynamization collar was applied.

Four months postoperatively, the femoral external fixator body was removed but the pins were left in place for a twenty-four hour observation period. The tibial external fixator was left in place due to delayed consolidation of regenerate bone. The following day the femoral pins were removed. Four days later the patient fractured his left femur through the regenerate bone. This was treated with closed reduction and reapplication of the external fixator. Five months postoperatively and 1 month after the femoral fracture, a scanogram revealed a LLD of 0.5 cm, with the lengthened left leg being longer. An additional 0.5 cm of lengthening was undertaken. At the end of the treatment the initially short left leg was 1.0 cm longer than the right leg.

Six months postoperatively the tibial external fixator was removed. A week later he developed a non-displaced tibial fracture. A long leg cast was applied after closed reduction. The femoral regenerate bone showed good callus and the femoral external fixator was dynamized.

Eight and one-half months after the index procedure, 16 weeks after his femoral fracture, and 10 weeks after his tibial fracture, the femoral external fixator body was again removed but the pins were left in place. One week later he sustained a second fracture through his femoral regenerate bone. Realignment under anesthesia with reapplication of the external fixator to the pins in the femur was performed. Iliac crest bone marrow was aspirated and injected into the left femoral and the left tibial lengthening sites. A short leg cast was applied. Two weeks later EBI bone growth stimulators were instituted on his left femur and tibia.

Eleven months postoperatively and five months after fracturing his tibia, the short leg cast was removed. Radiographs indicated good healing of the femur and tibia.

Thirteen months from the index procedure and five and one-half months after his second femoral fracture the femoral external fixator was removed.

Fourteen months postoperatively, leg lengths were clinically equal. The patient was full weightbearing. A scanogram taken seventeen months postoperatively, however, revealed a 1.0 cm LLD with the lengthened left leg being shorter. The rate of growth of the lengthened left leg had decreased or completely stopped. On his next visit, four months later, and now 21 months since the index procedure, a scanogram showed that the discrepancy had increased to 1.7 cm. Radiographs of the left knee demonstrated thinning of the physeal plates. At this time right distal femoral and proximal tibial epiphysiodeses were performed to prevent progression of this recurrent LLD.

At the last follow-up visit, 29 months after the index procedure and 7 months after epiphysiodeses, he was skeletally mature. The patient's LLD was 1.5 cm indicating little or no additional growth from the distal femur and proximal tibia of the lengthened limb.

DISCUSSION

As previously reported, a high rate of complications is common when performing limb lengthening procedures; however, severe growth retardation and growth arrest in the lengthened bones has not been frequently reported. In the patient presented, growth arrest occurred following simultaneous femoral and tibial lengthening for right sided hemihypertrophy.

As demonstrated by the Moseley straight line graph, the expected discrepancy at maturity for this patient was 5.5 to 6.0 cm (Figure 1). Lengthening of 6.5 cm was performed. As described by Moseley, the rate of growth (slope of line) postoperatively should be equal to the preoperative growth rate[11]. The line is merely displaced upwards an amount reflecting the amount of lengthening. Had this occurred and assuming our calculations were correct, the patient would have had correction of his LLD as depicted in Figure 2. However, as depicted in Figure 3, it is obvious that the slope of the line representing the left leg decreased from its preoperative value. This indicates retardation of growth and possible premature growth arrest at one or more of the physes. This resulted in recurrent LLD which ultimately required epiphysiodeses.

The reason for this patient's growth retardation remains unknown. One possibility is that he may have lost length due to his fractures. However, radiographic and clinical documentation indicate that length was maintained following reduction and fracture healing. However, scanograms after fracture healing demonstrated severe growth retardation. The fractures were not physeal injuries and were caused by low energy trauma, and should not have caused physeal arrest. It is well documented that fractures in long bones often result in overgrowth of the fractured bone[2,6,18]. Shapiro took this concept one step further by stating that the repair process involved in lengthening of the diaphysis is more extensive and longer than repair after fracture of the diaphysis[17]. Thus overgrowth, not growth retardation, is expected.

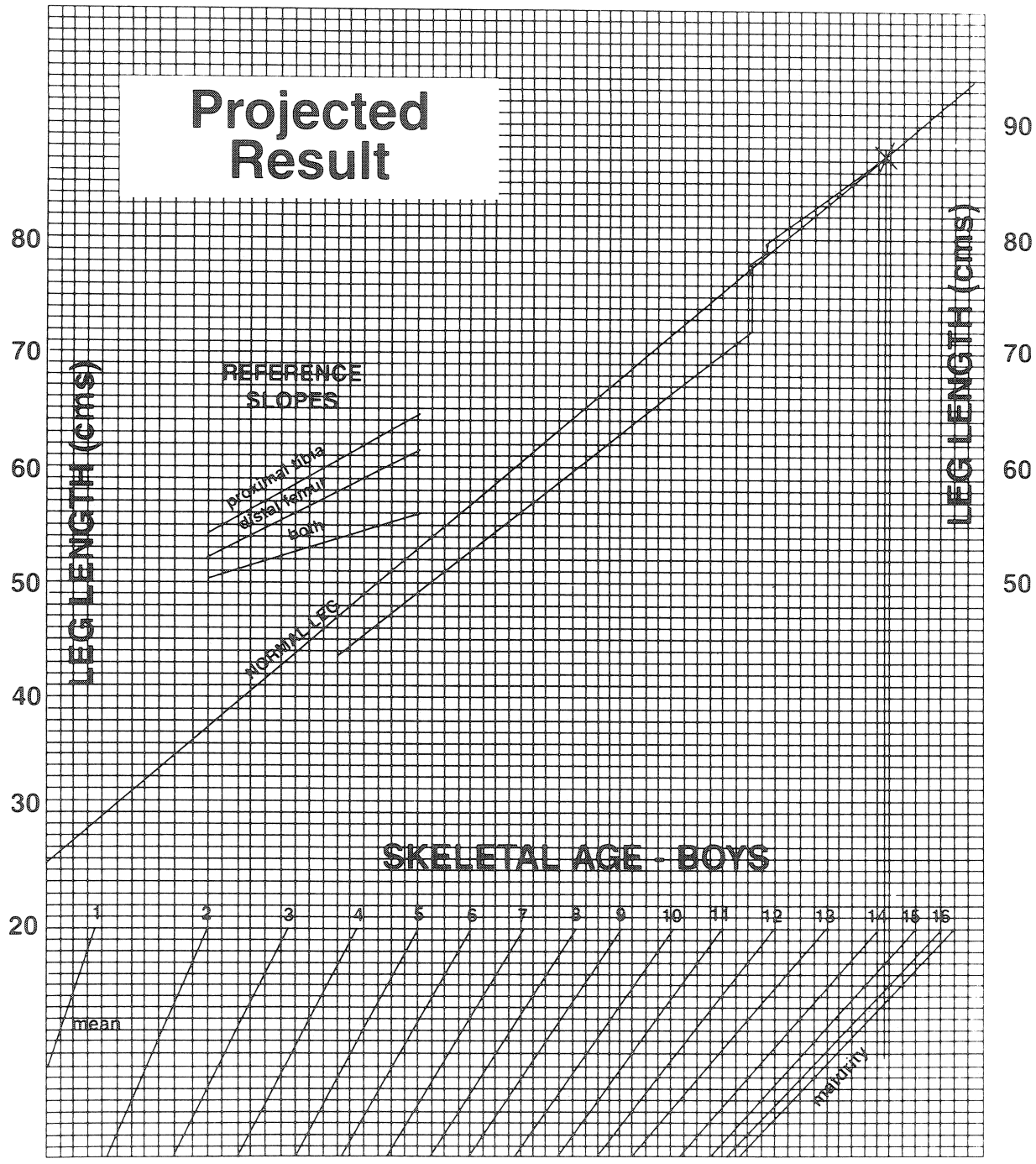

FIGURE 2. A Moseley straight line graph after initial lengthening of 6.0 cm and subsequent additional lengthening of 0.5 cm reveals expected post-lengthening growth resulting in equal limb lengths at maturity.

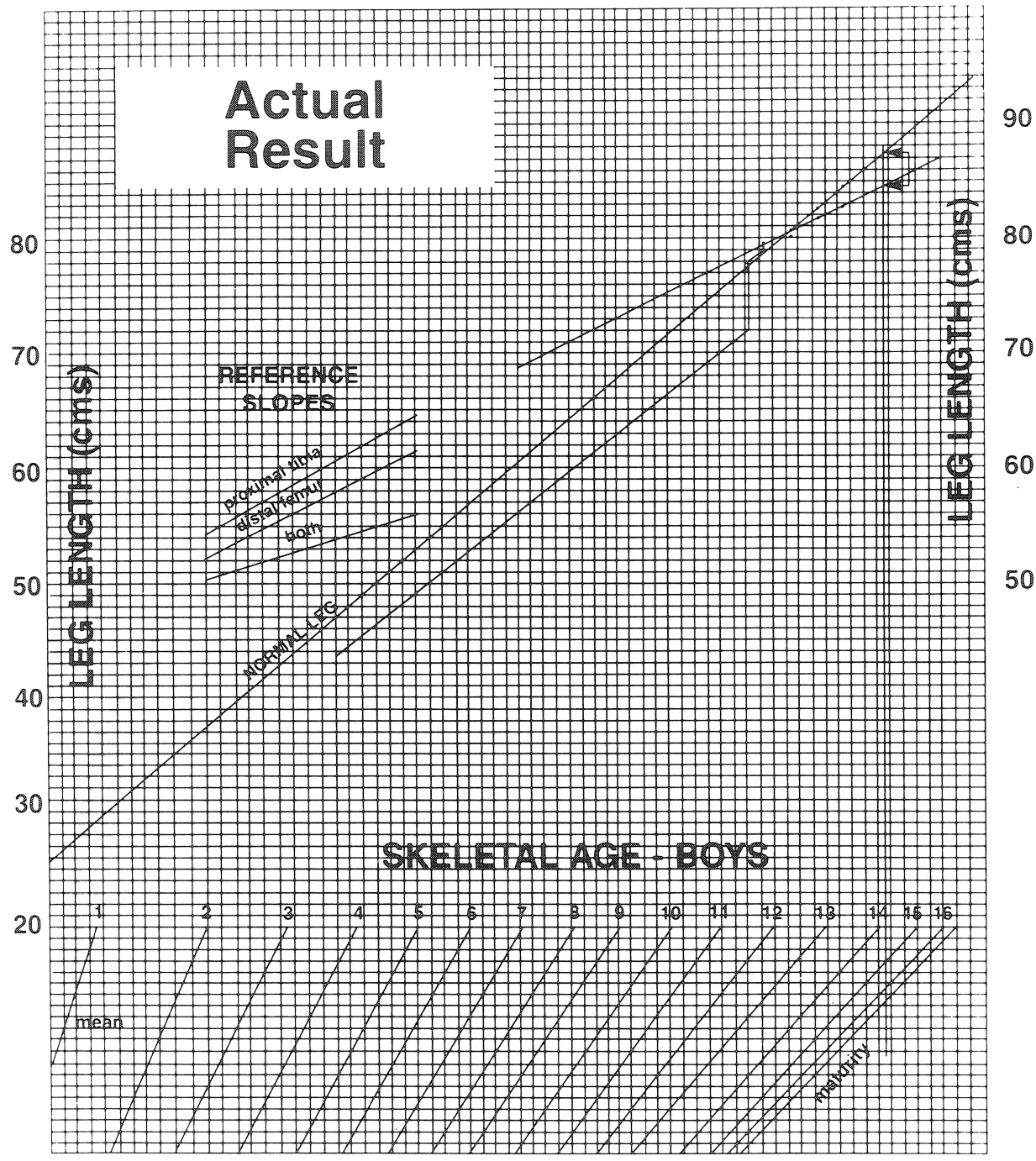

FIGURE 3. A Moseley straight line graph after lengthening showing actual growth rates and resultant residual limb length discrepancy of 3 cm which would have occurred without epiphysiodeses.

Another theory to explain growth patterns following fracture or lengthening procedures concerns the periosteum. In the 1970's, Crilly studied chicken radii and their response to longitudinal or circumferential division of the periosteum accompanying osteotomy. He found marked overgrowth of the radii upon healing in the subjects which had circumferential periosteal division. From these observations he advocated the periosteal tension hypothesis. As the periosteum is firmly attached to the cartilage beyond the growth plate, tension in growing bones compresses the physis and slows growth[7]. Sectioning of the periosteum mechanically releases tension across the physes resulting in overgrowth. Other authors have noted the same phenomenon and have concluded that the periosteum mechanically checks physeal growth[8,10,12,20,22-24]. Chan et. al. took this concept one step further and advocated periosteal stripping to stimulate bone growth in short limbs[3].

Strobino et. al. tried to verify this theory experimentally. They placed a tension creating device across the growth plate in the long bones of rats. As the bone grew, the tension increased. At the point at which the mechanical and growth forces equalized, they theorized that growth would cease. They found this to be true but only under large amounts of tension well in excess of body weight[21]. This concept is similar in theory to the Heuter-Volkmann law which refers to pressure inhibition of physeal growth and is similar to the concept of physeal stapling for epiphysiodesis. Pressures applied in directions parallel to the direction of epiphyseal growth inhibit the rate of such growth[1].

Current methods of limb lengthening emphasize the need to preserve periosteum. This may be beneficial for bone formation but may increase the risk of premature physeal closure.

In the patient presented, growth retardation occurred following a routine limb lengthening procedure. The periosteum was preserved in this patient. Only small 3.0 cm longitudinal incisions were made in the periosteum to allow pin placement. The lengthening was 8.7% of the initial femoral length and 9.6% of the initial tibial length. This was within the limits suggested by Pouliquen to avoid growth retardation or arrest with limb lengthening[15]. However, the patient developed retardation of growth of his lengthened left leg. One possible explanation is that this occurred because the pateint's discrepancy was congenital rather than acquired. Support for this theory comes from Kenwright et. al. They measured tension created by the DeBastiani lengthening technique and found that patients with congenitally short limbs reached higher force peaks during distraction and had higher resting levels of tension between distraction intervals[9].

We hypothesize that the intact periosteum transferred increased tension across the growth plate via the periosteal sleeve and either resulted in a premature epiphyseodesis or a crushing injury to the growth plate. As Shapiro points out, the remaining soft tissues including the muscles, tendons, fascia, and skin are resistant to distraction and likely exert some compressive forces across the growth plate[17]. Wilson-MacDonald et. al. feel that distraction may cause premature fusion of the growth plate secondary to vascular damage as well[24].

Although the etiology remains unclear, we believe severe growth retardation and growth arrest should be considered a potential complication following leg lengthening, and, in some cases, may necessitate a contralateral epiphyseodesis. The practice of overlengthening a congenitally short limb prior to physeal closure should be questioned because resumption of growth after lengthening may be unpredictable. We have suspected this complication in other patients who had similar procedures, but we have been unable to clearly document the sequence of events in these patients. Tension created by the intact periosteal sleeve may be responsible. Further research needs to be conducted to more clearly delineate the cause of this complication in order that appropriate changes be made in surgical procedure to prevent it.

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ALTERATIONS OF FRACTURE HEALING IN THE DIABETIC CONDITION

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Fracture healing normally proceeds in a highly reproducible manner to yield a tissue morphologically and functionally indistinguishable from original bone; however, in several pathological conditions such as diabetes the progress of fracture healing can be substantially impaired. The mechanisms responsible for impaired healing in a given pathological state are not immediately obvious. A better definition of the cellular and molecular regulatory processes underlying fracture healing may aid our understanding of these pathological situations, and lead to the development of more effective methods to treat or prevent the associated clinical complications.

Exhaustive research has documented that diabetes mellitis causes complications in nearly every human tissue. In diabetic bone disease there is retardation of bone development leading to osteoporosis. Recently, insulin has been recognized as a regulator of bone metabolism. Understanding the morphologic events of fracture healing, and conditions which may impair fracture healing, aid in explaining those mechanisms in diabetes which create such clinical consequences as delayed union and nonunion. While little has been written about the effects of insulin on fracture healing, many inferences may be made from the abundant data available regarding insulin effects on bone metabolism. This review examines recent work on the effects of insulin as it relates to fracture healing, with specific emphasis on the role of cytokines and local growth factors.

BIOLOGY OF FRACTURE REPAIR: FROM HISTOLOGICAL CHANGES TO MOLECULAR EVENTS

Fracture healing has been described as a process of tissue regeneration where the site of injury is replaced by bone, not scar tissue. In normal fracture healing, a sequential cascade of events occurs which fulfills three functions[3]: removal of debris, re-establishment of the damaged vascular supply, and restoration of the skeletal matrix. Four histological stages of fracture repair, which follow a discrete timetable, have been described: (1) inflammation, (2) soft callus formation, (3) hard callus formation, and (4) remodeling. The first (inflammatory) stage begins at the time of fracture and persists in human long bone for up to five days[18]. At the fracture site, a hematoma forms which is then invaded by neutrophils and macrophages to remove tissue debris, and subsequently by fibroblasts which establish a provisional collagenous matrix. At the same time, subperiosteal bone formation occurs near the fracture site to provide additional mechanical support for the forming callus. In the second stage, which lasts from 5 to 40 days, the hematoma is replaced by a recognizable fibrocartilaginous callus. Resorption of the hematoma occurs via the concerted action of macrophages and fibroblastic cells[32], while cartilage formation occurs by the differentiation of mesenchymal cells into chondrocytes, and the subsequent elaboration of a type II collagen-based matrix. The cartilage matrix calcifies, and then is progressively replaced by bone synthesized by osteoblastic cells. Finally, subperiosteal new bone forms as an initial support structure, which eventually replaces calcified cartilage. Remodeling of the fracture site occurs by continuous cycles of resorption and formation. Ultimately bone's original architecture is restored (Figure 1).

MECHANISMS IN IMPAIRED FRACTURE HEALING

Diabetes and Absence of Insulin

Insulin, a peptide 6000 Daltons in size, is secreted in the beta cells of the islets of Langerhans in the pancreas[2]. It promotes marked stimulation of bone matrix and formation of cartilage. When systemic levels of insulin are reduced, there are late complications in nearly every body tissue[1], including bone. Diabetic bone disease is characterized by osteopenia. This is likely caused by primary disturbances of calcium, chronic malnutrition, and chronic vascular disease. Given the multifaceted interactions of diabetes and bone metabolism, it is difficult to assess the individual contribution of any single dysfunction for it's role in impaired fracture healing.

Insulin has been well known to have a combination of direct and indirect action on bone[4]. Research has found that insulin directly increases collagen production by osteoblasts[9]. The presence of insulin promotes insulin-like growth factor 1 (IGF-1) which stimulates both collagen synthesis and cell proliferation in osteoblasts[6,10]. IGF-1 treatment has been shown to correct the osteopenia of diabetes.

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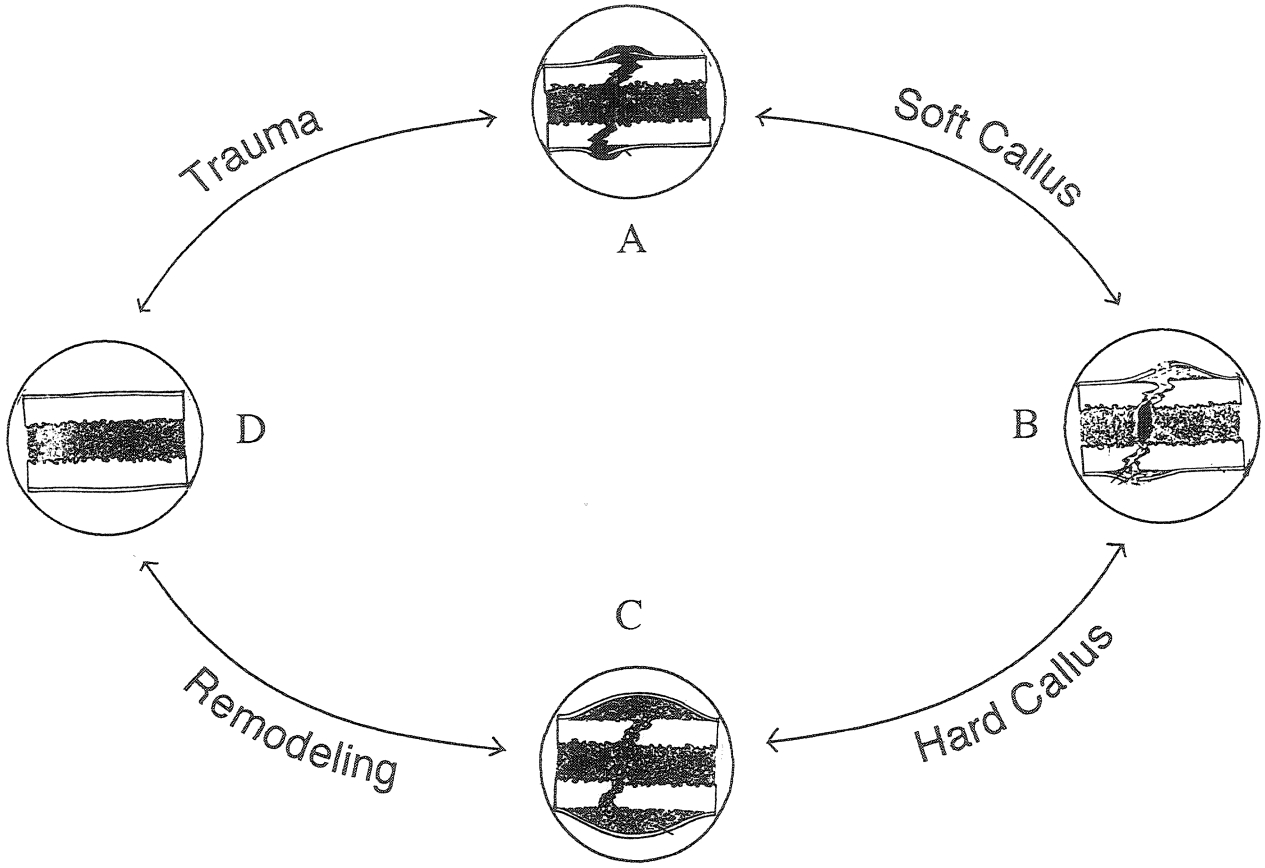

Marker	Stage	Time	Event
[A]	Inflammation	0-5 days	Fracture formation Hematoma Granulation tissue Osteogenesis
[B]	Soft Callus	5-21 days	Fibrous matrix removal Chondrogenesis
[C]	Hard Callus	10-42 days	Chondrocyte hypertrophy Cartilage mineralization Cartilage replacement by bone Marrow formation
[D]	Remodeling	21-50 days	Resorption and remodeling

Figure 1. The sequence of morphogentic events in fracture healing

The fracture healing process in diabetic rats produces smaller and mechanically weaker callus. Further analysis shows a reduced collagen to DNA ratio with diminished collagen productivity per cell[18,33]. These alterations are observed with insulin doses which fail to modify or correct blood glucose levels.

In vitro studies of osteoblasts and chondrocytes in an insulin deficient environment demonstrates reduced calcification and ossification, and delayed cartilage formation[7]. The absence of insulin eliminates production of sulfated glycosaminoglycans, thereby reducing the size of proteoglycans. Collagen formation is impaired due to glucose-mediated impairment of cross-linking, rendering collagen unstable and more easily digested by collagenase[6]. Other studies suggest a 50 to 55% decrease in collagen synthesis in untreated diabetic rats. The diminished amount of collagen in this fracture callus is associated with diminished mechanical strength[9].

METABOLIC ALTERATIONS OF CALCIUM AND VITAMIN D

There are different metabolic factors which play a role in diabetic osteopenia including alterations calcium and vitamin D metabolism, chronic malnutrition, and vascular insufficiency[8,15,27] (Figure 2). Animal studies reveal impaired duodenal calcium absorption in the presence of decreased systemic insulin levels, associated with lower levels of calbindin D-K9 concentration[29]. Active, but not passive, calcium transport is virtually eliminated.

As in human studies, studies of diabetic rats[25] found levels of calcium in the urine up to 13 times normal. This may be related to glomerular filtration, osmotic diuresis, or an insulin-dependent decrease. Some suggest the effect of diminished active calcium transport can be offset by a high calcium intake with an overall increase in food intake.

VITAMIN D

Vitamin D is important in both bone resorption and bone formation. Its synthesis is characterized by a well orchestrated cascade of events[4,11]. Hydroxylated in the liver, vitamin D3 is converted to 25-hydroxyvitamin D3 (25-OH-D3) and metabolized in the kidney to the active metabolites 1,25 $(OH)_2$ D3 and 24,25 $(OH)_2$ D3. Alterations in vitamin D metabolism contribute to such clinical situations as osteomalacia, osteoporosis and osteosclerosis. Although few studies of vitamin D demonstrate a direct role in fracture healing, much is known of its involvement with growth factors and regulation of bone volume.

New concepts of vitamin D action have developed in the past several years. The major change is reflected in its consideration as a steroid hormone rather than as a vitamin. As a steroid, vitamin D's target tissues are bone, intestine, and kidney.

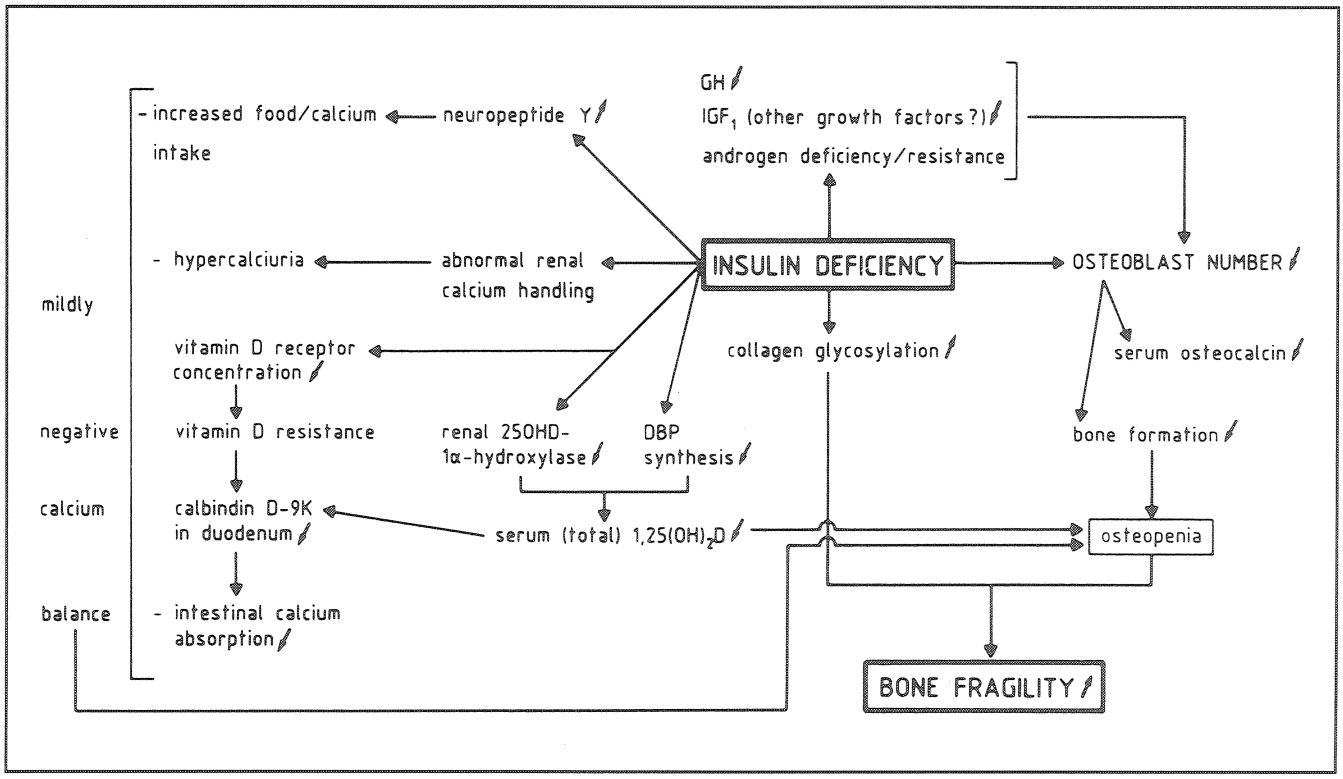

Figure 2. Schematic integration of the effects of insulin deficiency on mineral and bone metabolism in insulin-deficient diabetes mellitus. Copied with permission from Bouillon, R.: Diabetic Bone Disease. *Calcif. Tissue Int.*, 49:155-160, 1991.

In the cytoplasm of the osteoblast, a 1,25 $(OH)_2$ D3 receptor protein has been identified. The receptor has also been seen in intestine, perhaps contributing to calcium influxes in the duodenum. This active metabolite, 1,25 $(OH)_2$ D3, is important for bone remodeling and bone resorption, where osteoblasts act as a relay for osteoclast activation[25]. For its role in bone formation, 1,25 $(OH)_2$ D3 promotes osteoblast differentiation and may influence systemic PTH and calcium levels.

Growth factors such as IGF-1, IGF-2 and TGF-β_1 have a complex relationship with vitamin D3 and its metabolism, resulting in a continuous balance between bone resorption and formation[1,19,20,30]. Both IGF types offer some mitogenic activity for all cells of osteoblastic lineage and enhance type I collagen synthesis and decrease collagen degradation in calveriae. IGF-2 differs from IGF-1 in its greater abundance in bone matrix, and it does not recruit osteoclasts. 1,25 $(OH)_2$ D3 stimulates production of IGF-1 and IGF-2 in osteoblast-like cultures. TGF-β has been implicated in vitamin D metabolism of bone[31].

Additional research has demonstrated that the diabetic state may create a derangement of vitamin D homeostasis. Using streptozotocin-induced diabetic rats, alterations in vitamin D metabolism contributed to fasting hypercalcemia and impaired intestinal calcium absorption[34]. Plasma vitamin D binding protein levels were 62% lower in diabetic rats. Prolonged streptozotocin-induced diabetes in rat studies resulted in growth arrest and reduced bone turnover. The implication of these changes are far from clear regarding a role in impaired fracture healing.

CURRENT ANALYSIS OF DIABETIC FRACTURE HEALING

A recent paper by Kawaguchi et al.[16] emphasizes the paracrine role of insulin in diabetic fracture healing. Basic fibroblastic growth factor (bFGF), a potent stimulator of proliferation in mesenchymal cell types including fibroblasts and osteoblasts, was depleted in early stages of fracture callus formation in diabetic rats. Insulin treatment restored bFGF in the diabetic tissue. Moreover, direct application of a single dose of bFGF immediately postfracture increased callus size and mineral content and enhanced the mechanical properties of callus in normal as well as diabetic animals. A further consequence of bFGF treatment was an increase in transforming growth factor beta (TGF-β) levels in fracture callus. TGF-β, a known stimulator of both chondrogenesis and osteogenesis, may mediate some of the longer term effects of bFGF.

Kawaguchi et al. reinforces earlier findings by Macey et al.[18] that the effects of diabetes on fracture healing begin at a very early stage. They also provide evidence that intervention (using bFGF) at that point, presumably to increase the number of mesenchymal precurser cells available for subsequent formation callus tissue, can partially offset the effects of diabetes at much later stages of fracture healing. They also suggest that in normal bone the ability to form callus is suboptimal and can be further stimulated by appropriate treatment. It should be noted however, that Kawaguchi's study involved fracture of the fibula, a bone that is not a significant weight bearing bone. Mechanical stress may facilitate callus formation in those systems, and a more nearly "optimized" system could show a smaller response to exogenous bFGF[23]. Interestingly, a subsequent study showed that systemic injection of bFGF could stimulate endosteal bone formation in intact bone, apparently by a similar mechanism involving enhanced recruitment and differentiation of osteoblastic precurser cells[9, 10].

Additional studies on the effects of diabetes on fracture healing have focused on collagen synthesis in rat fracture callus. Topping et al.[33] studied type X collagen, a molecule synthesized in hypertrophic chondrocytes. Histologic patterns in normal and diabetic fracture callus found no significant difference in the amount or distribution of type X collagen by immunoflorescent staining. However, the *in vitro* studies did reveal a substantially reduced amount of type X collagen with diabetic fracture callus when compared with controls. Differences of other collagen types were not discussed in this study. The marked effect of diabetes on type X collagen synthesis suggests that this molecule is sensitive to insulin action, but needs to be further reconciled with the similar patterns of immunoflorescent staining seen in prior studies. Perhaps the differences result from a lower turnover rate effected by diabetes.

CONCLUSIONS

There appears to be consensus within the literature that insulin has a role in fracture healing. It is also evident that many of the insulin effects are the result of indirect as well as direct actions on skeletal tissue. Even in the light of these findings many questions remain unanswered. The body of data available on the effects of diabetes on fracture healing and remodeling are inferential, but lead to testable hypotheses concerning fracture healing. As our understanding of molecular events and the role of cytokines in bone development improve, future investigations may be able to clarify the role of diabetes and insulin in fracture healing.

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FAMILIAL CONGENITAL PSEUDOARTHROSIS OF THE CLAVICLE: CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Congenital pseudoarthrosis of the clavicle (CPC) is a rare condition of unresolved etiology and pathogenesis. Familial occurrence of this anomaly has been documented but the pattern of genetic transmission remains obscure. Two cases involving a father and daughter are presented here with a review of the literature. These cases add support to the genetic basis for CPC.

INTRODUCTION

Congenital pseudoarthrosis of the clavicle (CPC) is a rare clinical defect. Although most orthopedic surgeons have had experience with this abnormality, its familial nature is not commonly noted. This study offers a review of the literature and presents two cases of CPC involving a father and daughter.

The condition was initially described by Fitzwilliams as a distinct variant of cleidocranial dysostosis in 1910[6]. CPC has since appeared in a number of studies involving more than 100 patients. This malformation appears to be 25-80% more common in females than males[10,14]. Usually the condition is unilateral and occurs on the right side, but in approximately 10% of the cases involvement is bilateral. Left sided unilateral involvement is extremely rare and is often associated with dextrocardia or cervical ribs[1,11,19,21].

The malformation is fully present at birth, but most often the diagnosis is made a few months to several years later. The patient usually presents with a painless swelling over the middle third of the right clavicle. This swelling often becomes more evident with maturation. Full range of shoulder motion is present. The adult with CPC may complain of fatigue or discomfort related to strenuous activity. CPC markedly shortens the distance from the sternum to the acromion due to an absence of a small portion of the bone. This separates the clavicle into two parts. The larger sternal segment always overlies and is anterior to the proximal end of the acromial segment[10,15,16]. There is often a small depression on the "inferior aspect of the outer end of the medial segment into which the blunted inner end of the lateral segment fits"[14]. The swelling associated with CPC is attributed to the enlarged ends of the bones, the overlapping of the fragments, and the cephalad angulation of the fragments. The bony ends are never pointed as they are in pseudoarthrosis of the tibia[10]. With palpation, a discontinuity in the substance of the clavicle and mobility between medial and lateral fragments is evident[21]. Often cervical ribs or abnormally elevated first ribs are present along with the abnormal clavicles.

CASE REPORT

A 9-year-old girl was seen for evaluation of congenital pseudoarthrosis of her right clavicle. Gestation, labor, and delivery were atraumatic and uneventful. Separation of the right clavicle was noted one day after birth. This was initially thought to be a birth fracture, but never healed. The patient has remained very healthy and her developmental milestones have been normal. At age 9 the mid-clavicular swelling had become more prominent and surgical correction was contemplated. She reported no pain or difficulty with normal activity, although extreme physical exertion, such as throwing a baseball, occasionally produced pain in the region of the defect.

Examination revealed a prominent mid-clavicle on the right, consistent with the diagnosis of CPC. There was no tenderness. She demonstrated full range of motion of the shoulder and had normal strength in the right upper extremity. No other musculoskeletal abnormalities were noted.

Radiographs were reviewed and revealed a 1 to 2 cm defect in the middle third of the clavicle (Figure 1).

The patient's father also had a diagnosis of a right CPC. A bone graft at the age of 10 did not successfully heal his pseudoarthrosis. He experiences right shoulder pain with strenuous activity, such as weight lifting or overhead work. Radiographs of the father's clavicle revealed a pseudoarthrosis with callus formation in the middle third of the clavicle (Figure 2).

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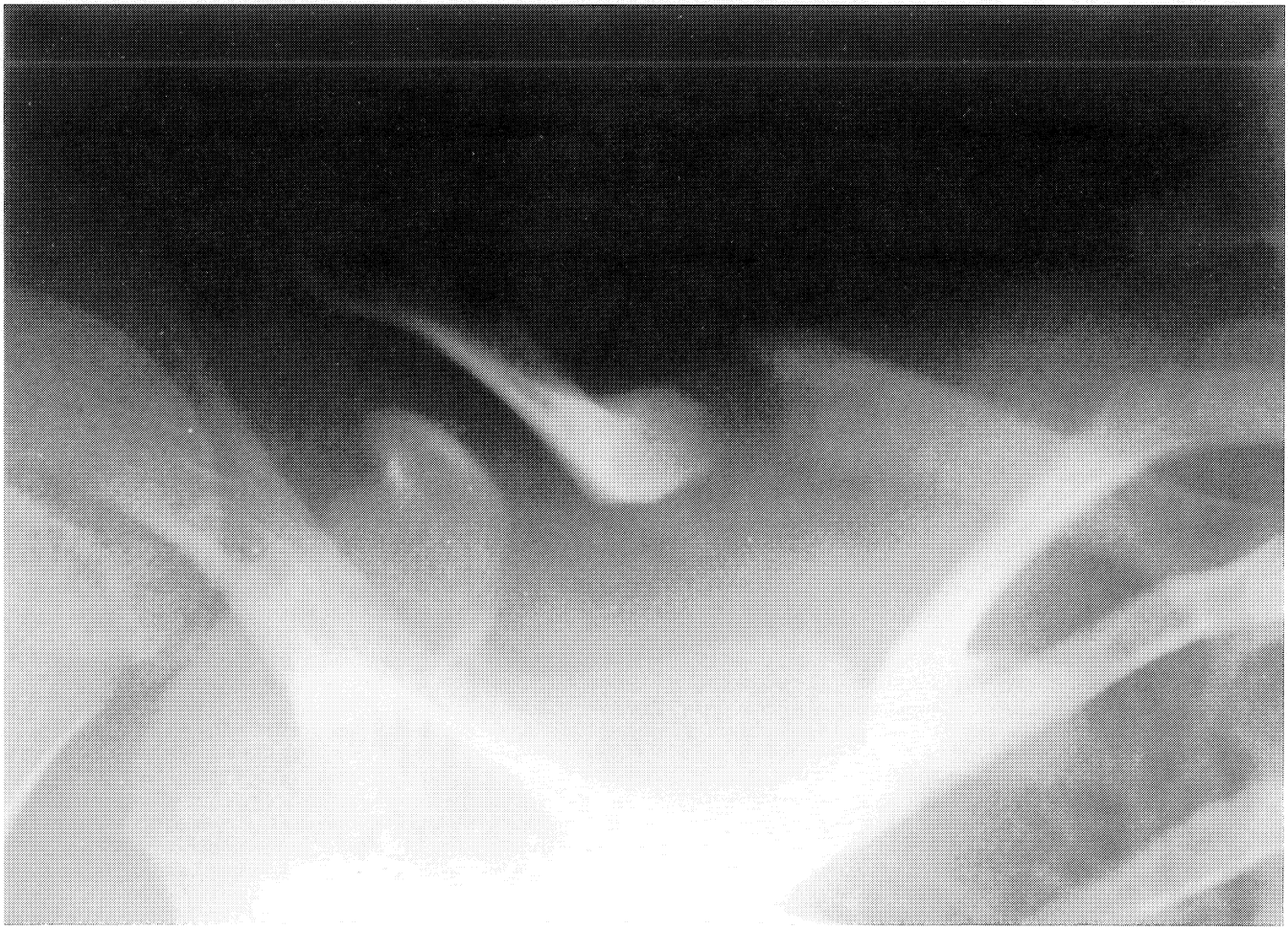

Figure 1. 9 year old female with pseudoarthrosis of the right clavicle.

DISCUSSION

Genetics

Gibson and Carroll were among the first researchers to document familial incidence of CPC. One patient gave a history of an aunt and grandfather who also had the same anomaly. A second family appeared to display an autosomal dominant type of transmission. However, minor bone abnormalities in this family suggest that these patients may have had a variation of cleidocranial dysostosis rather than CPC[7].

Some researchers suggest that environmental factors rather than genetic factors influence the development of CPC. The high prevalence of unilateral CPC exclusive of any family history or additional anatomical abnormalities support this hypothesis[3,9,21].

However, documented cases of familial CPC exclusive of other bone abnormalities have been reported. Owen reported a father and daughter with identical right sided lesions[18]. Toledo noted a positive family history in two sisters whose mother also had a typical right-sided CPC[20]. Alberink studied a man whose three sisters and father had right clavicular pseudoarthrosis[2]. Although the majority of reported cases of CPC show no family history of this disorder, these reports suggest a possible autosomal dominant inheritance pattern in families. Clearly a familial basis for some cases of CPC exists, but a definite genetic pattern has not been established.

Pathogenesis

The anatomical basis of CPC is still questionable. Theories of development of CPC include nonunion of two ossification centers in the embryonic clavicle, exogenous or mechanical causes, and abnormalities of the vasculature surrounding the developing clavicle[3,17,21].

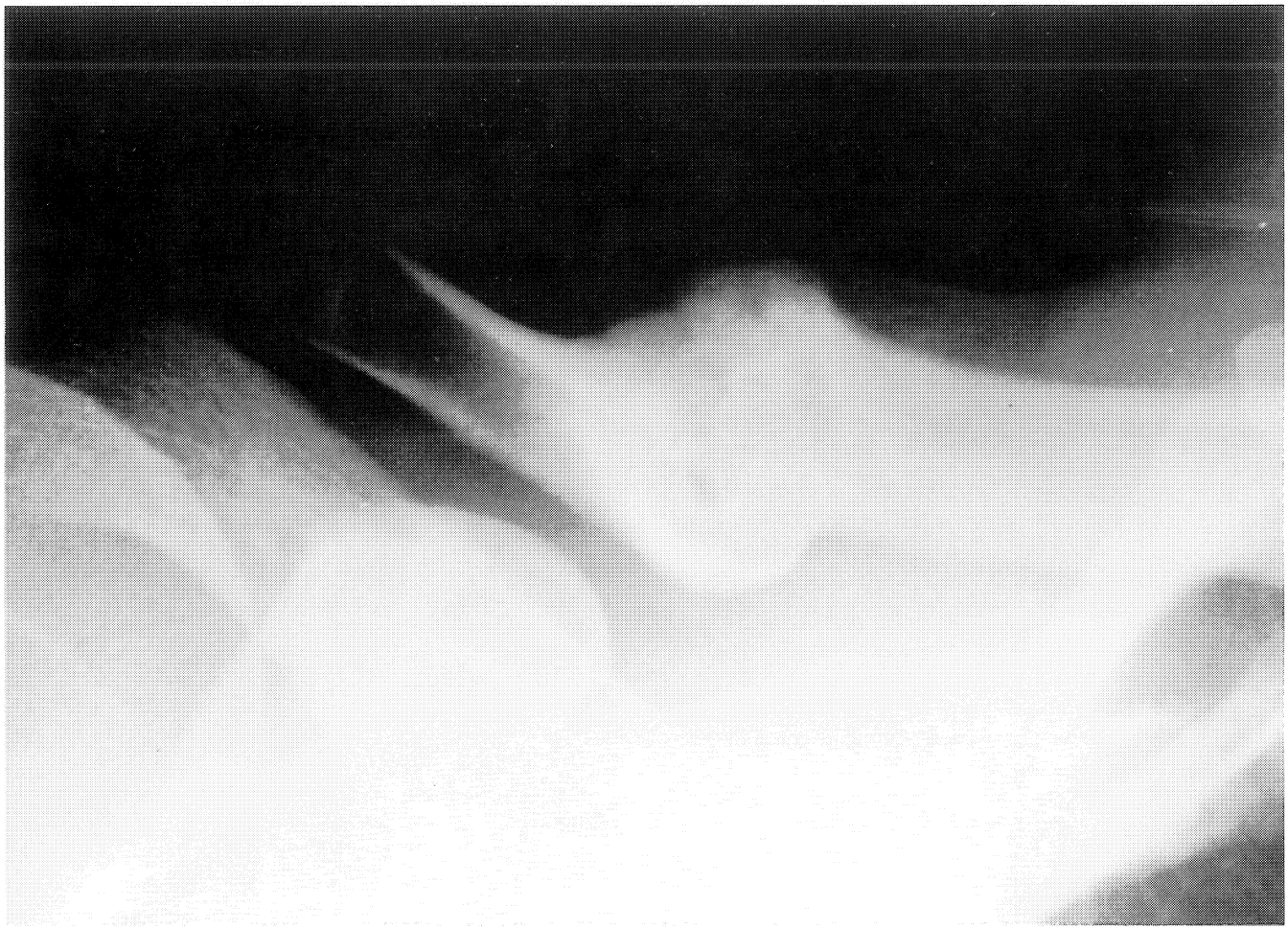

Figure 2. Child's father demonstrates pseudoarthrosis of the same side 30 years after attempted surgical correction.

Studies of clavicular ossification centers by Mall and Wall indicated that the embryonic clavicle forms from two ossification centers[12,21]. Many researchers believe that CPC results from a failure of these two centers to fuse. Gibson and others studied the embryology of the clavicle and believed that it developed from one ossification center[7]. Ogata and Uhthoff have conducted research on embryologic ossification of the clavicle which supports previous studies claiming that the clavicle forms from two membranous ossification centers[17]. After reviewing previous reports, they concluded that the two centers had already fused prior to the histological study by researchers claiming development from only one center. However, Ogata and Uhtoff also indicated that these two centers are more lateral than the usual location of CPC. Thus, failure of fusion of ossification centers may not explain the etiology of this malformation.

A theory based on exogenous or mechanical causes suggests that the prevalence of right sided lesions could be directly attributed to the more common left occipitoanterior presentation during birth. This position may place the right shoulder at a higher risk for trauma. Thus, CPC may result from birth fracture and nonunion of the clavicle rather than an abnormality during clavicle development[3,18].

Another theory strongly supported by Lloyd-Roberts et. al. suggests that certain anatomical abnormalities are predisposing factors for CPC[11]. It is believed that cervical ribs or "vertically disposed and elevated upper ribs" may cause compression of the subclavian artery between the clavicle and first rib. The exaggerated arterial pulsations and pressure which result could influence the development of a pseudoarthrosis. This theory suggests that the increased incidence of right sided CPC may be related to the fact that the right subclavian artery is normally elevated to a point directly underneath the clavicle. Anatomical abnormalities such as cervical ribs may influence the relationship of the subclavian artery to the right clavicle[11]. Although not all cases document predisposing abnormalities[4], cervical ribs have been reported in roughly 15% of the

literature on CPC[8,14,20]. Further support is offered by the fact that dextrocardia or large left sided cervical ribs were present in many reported cases of left-sided CPC[7,11]. In such instances it is suggested that the level of the left subclavian artery may exceed the right[11]. Rib abnormalities are known to have a familial occurrence. If abnormally elevated ribs are a predisposing factor for CPC then this would lend further support to a possible genetic etiology of CPC[11].

CONCLUSION

The father and daughter reported here add support to a genetic basis for CPC. The association of CPC with other familial anatomical anomalies also supports familial inheritance. However, the frequent occurrence of CPC without family history prevents the formation of a conclusive genetic explanation. While researchers are beginning to agree that the clavicle develops from two primary ossification centers, patterns of clavicular formation seem unrelated to formation of CPC. Associated anatomical abnormalities are infrequently documented but may provide some explanation for the formation of CPC. The etiology and pathogenesis of this condition remain obscure. It is our opinion that genetic and anatomical factors play a role in the development of CPC.

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POSTEROLATERAL APPROACH FOR ANTERIOR RESECTION AND POSTERIOR STABILIZATION OF THE UPPER CERVICAL SPINE: A CASE REPORT

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ABSTRACT

Surgical approaches to atlanto-axial lesions are generally accomplished by either anterior (transoral) or posterior approaches as dictated by the location of the lesion. In certain patients, these approaches are combined, either in a single or staged procedure. Mechanical stabilization is much more readily accomplished posteriorly, as this allows easy incorporation of the occiput. While the transoral approach allows excellent exposure of the bodies of C1 and C2, it entails substantial surgical trauma. We describe the case of a woman with destruction of the anterior portions of the C1 and C2 vertebrae by metastatic breast cancer addressed by simultaneous anterior tumor debulking and posterior instrumentation through a posterolateral approach to the upper cervical spine.

INTRODUCTION

Both anterior (transoral) and posterior surgical approaches to the upper cervical spine have been described, with the preferred approach determined by the site of pathology.[1,7,8] Posterior stabilization is mechanically superior, as it allows access to the occiput for hardware stabilization and incorporation of the fusion mass. Simultaneous or staged anterior and posterior procedures are indicated for certain patients with anterior tumor or fracture requiring posterior stabilization. In addition, some patients require posterior stabilization following anterior procedures if resection of anterior supporting structures exacerbates instability or if anterior stabilization fails.

While surgical management of patients with metastatic carcinoma of the atlas and axis has been described, we are aware of no reports of patients undergoing anterior tumor debulking and posterior instrumentation through a single surgical approach. We report a case of metastatic breast carcinoma with anterior destruction of both the C1 and C2 vertebrae, treated by excision of the tumor mass and posterior stabilization through a single posterolateral surgical approach.

CASE REPORT

A 68 year old woman complained of neck and occipital pain for approximately three months, increasing in severity over several weeks. For one week prior to admission, she had been unable to sit or stand due to posterior vertex headaches. She denied weakness, paraesthesias, and bowel or bladder incontinence.

Physical exam revealed cranial nerves II-XII were intact. Both upper and lower extremities were neurologically intact with no pathologic reflexes or clonus.

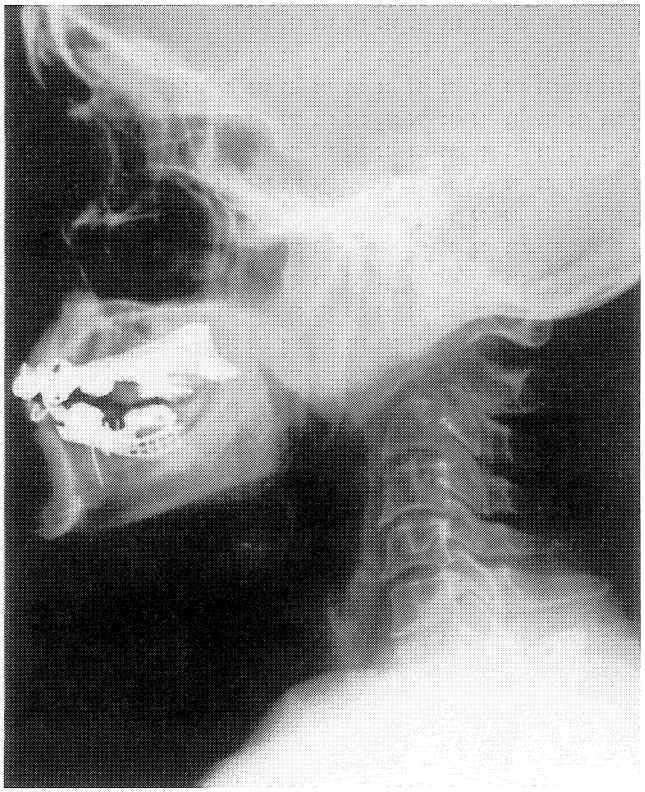

Figure 1. Initial lateral cervical spine radiograph. Resorption of the anterior portions of the atlas and axis with mild cranial settling is noted.

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Cervical spine radiographs showed diffuse destruction of the anterior portions of both the C1 and C2 vertebrae with mild anterior cranial settling (Figure 1).

Further physical exam revealed the left breast to be entirely occupied by a mass. A 2 cm ulceration with purulent discharge was noted with surrounding *peau d'orange*. The patient was placed in a hard cervical collar and admitted to the hospital for a modified radical mastectomy.

A computerized tomography (CT) scan showed extensive destruction of the anterior ring of C1 including the facets, as well as extensive destruction of the body, dens, and facets of C2 (Figure 2). MRI images showed compression of the cord at the C1-C2 level, but no increased signal within the cord on the T2 sagittal image (Figure 3). Angiography documented normal flow within the vertebral arteries. A bone scan revealed no other bony metastases. Staging CT scans of the chest and abdomen revealed no metastases.

The patient was felt to have impending instability of her cervical spine secondary to the extensive destruction of both C1 and C2 accompanied by cranial settling. It was felt that radiation or chemotherapeutic treatment alone was insufficient treatment, so the patient underwent surgical stabilization on the 23rd postoperative day from her mastectomy.

OPERATIVE TECHNIQUE

The patient was placed prone in skeletal cervical traction on the operating table. A midline posterior hockey-stick incision was used, extending upwards to the right mastoid tip. The posterior cervical elements were exposed from C1 to C5. The posterior elements of C1 and C2 were resected. The approach was then deepened on the right, exposing the involved lateral masses of C1 and C2. These were resected, with dissection of tumor mass directly off the vertebral artery. Sufficient resection of tumor was performed to allow mobilization of the vertebral artery and the C1 and C2 nerve roots laterally and anteriorly.

Extensive extradural tumor was visualized anterior to the cord. This was debulked using curettage and aspiration. This was continued cranially to the foramen magnum

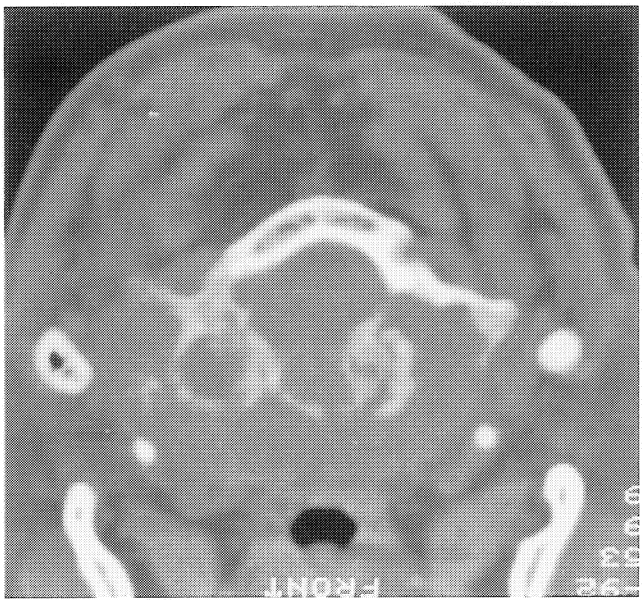

Figure 2. Axial CT image. Extensive destruction of the anterior portion of the ring of C1, the dens, and lateral masses of C2 is demonstrated. Small posterior portions of the C1 and C2 vertebrae are preserved.

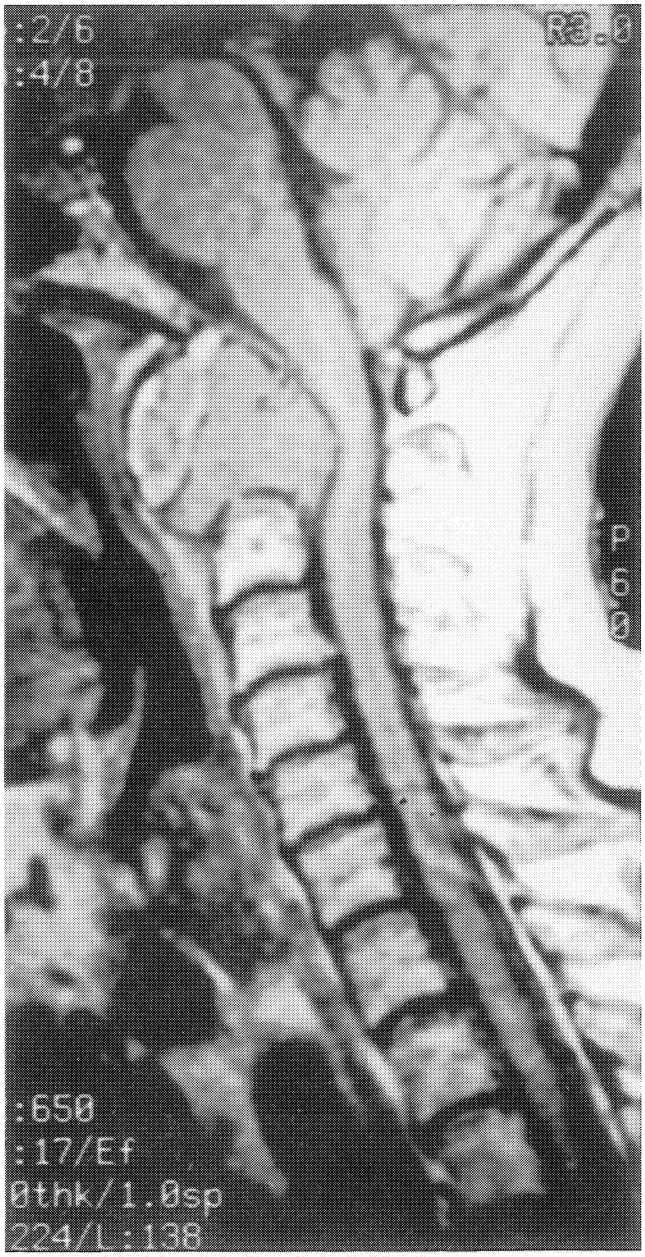

Figure 3. Sagittal MRI image. Substantial spinal cord compression is present due to the soft tissue mass occupying C1 and C2.

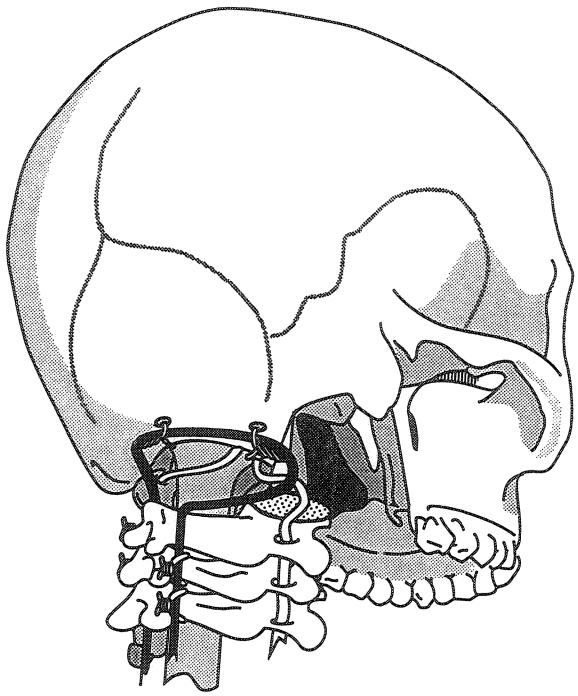

Figure 4. Representation of looped Steinman pin with occipital and sublaminar wires.

and caudally to the disc space of C2-C3 and well past the midline. Following tumor debulking, the cord remained deformed over the dorsally tented posterior longitudinal ligament. This was pushed anteriorly following retraction of the dural sac with a right angle dural separator. Intraoperative ultrasonography was then performed showing good pulsation of the spinal cord and cerebrospinal fluid as well as clear anterior and posterior subarachnoid spaces.

Permanent stabilization was then achieved through a contoured loop formed from a threaded 5/32 inch Steinman pin. This was adapted to the horizontal segment of the occiput and the inferior facets of C3 and C4. The loop was then fixed in position with two braided 22 guage wires on either side of the occiput and with sublaminar fixation to C3 and C4 bilaterally (Figure 4). Postoperatively, the patient was returned to a hard cervical collar (Figure 5).

Radiation therapy was started on the 22nd postoperative day, using lateral ports to avoid interference with wound healing. Her course was complicated by a 1 cm occipital decubitus ulcer that required hyperbaric oxygen therapy. Her activity level gradually improved and she was discharged seven weeks postoperatively in a soft collar, ambulatory and essentially pain free. She remained active with minimal pain, despite involvement of the C4 vertebral body noted 16 months postoperatively. Twenty months

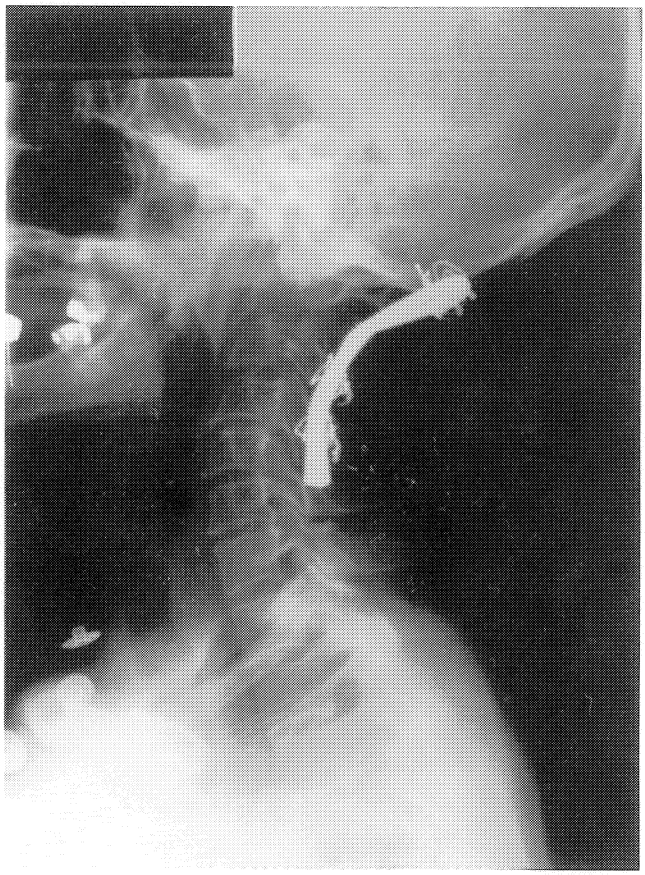

Figure 5. Immediate postoperative lateral cervical spine radiograph. Posterior fixation from occiput to C3/C4 was accomplished with a contoured, looped Steinman pin and 22 gauge wire.

postoperatively she developed a central cord syndrome due to continued enlargement of the tumor mass, and died from respiratory insufficiency one month later. The fixation remained stable.

DISCUSSION

Although indications for surgery in patients with cervical spine tumors have received attention in the literature, guidelines are difficult to establish due to the small number of patients and the unique situation each patient presents.[1,9] Myelopathy due to compression or instability warrants decompressive spinal surgery with fusion, if needed, when significant survival is expected.[4,6] Palliation of pain is a surgical indication when medical therapy has failed.[2] While it remains difficult to precisely define clinical instability, we feel that impending instability is also an indication for stabilization, either surgically or with a halo vest. In this case, the extensive destruction of both C1 and C2, as well as cranial settling, were felt to place the patient at significant risk of catastrophic instability.

Both posterior and anterior approaches to the craniocervical junction have been described.[7,8] The standard posterior approach readily allows immediate stabilization

incorporating the occiput. Anterior stabilization is not only more technically difficult, potential complications from hardware, bone cement, or bone graft are more serious than with posterior placement. However, patients with anterior lesions of the atlas and axis may require debulking of these tumors for pain relief and to prevent neurological compromise. These requirements have led to the use of both approaches in a single anesthetic or as a two-stage procedure.[7] However, this increases the patient's operative exposure, and entails the substantial discomfort of the transoral approach.

While a lateral approach to the anterior foramen magnum has been described,[3,5,6] its application to simultaneous resection of anterior cervical spine tumors and posterior instrumentation has not previously been reported. While the approach described here does not allow as thorough a resection as the anterior transoral approach, and would not be useful in trauma patients requiring anterior fusion, we feel there are many patients for whom this posterolateral approach will decrease the operative exposure, allow a more rapid recovery, and offer excellent pain relief.

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SNAPPING KNEE FROM ANOMALOUS BICEPS FEMORIS TENDON INSERTION: A CASE REPORT

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INTRODUCTION

Symptomatic snapping of the biceps femoris tendon over the fibular head due to an anomalous insertion is a rare condition. We report a case of painful biceps femoris snapping elicited with knee range of motion with tibial internal rotation. Failing conservative treatment, the patient had anatomic repositioning of the tendon with resolution of snapping and symptoms at follow-up.

CASE REPORT

A sixteen year old male presented with a two week history of right knee pain following a soccer match. Pain was localized to the lateral side of the knee, and was exacerbated with activity. On physical examination a palpable pop was localized to the lateral side of the knee during a McMurray test. Closer inspection demonstrated the snap to be localized over a tender fibular head. The snap was elicited when knee motion was ranged from 120 to 100 degrees and only with internal tibial rotation. The remaining knee examination was normal; however, a similar snap was appreciated on the asymptomatic contralateral knee using the same maneuver. Routine radiographs were normal. The patient was treated with ten days of rest and anti-inflammatory medication with partial resolution of his symptoms.

Three years later the patient returned complaining of steadily increasing pain and functional limitation that was refractory to activity modification. Radiographs and magnetic resonance imaging were normal. The patient was diagnosed with snapping biceps femoris tendon and was indicated for exploration and tendon stabilization.

At surgical exploration the biceps femoris tendon inserted onto the anterolateral aspect of the proximal tibia. No attachment to the fibular head was appreciated. The diagnosis was confirmed as the biceps femoris tendon was noted to displace over the fibular head during knee extension from 120 to 100 degrees with internal tibial rotation (Figures 1 and 2). The tendon was reinserted into the posterolateral fibular head through a ten millimeter drill hole and sutured to bone and periosteum. Postoperatively, the patient was immobilized for four weeks in a long leg cast in five degrees of flexion. Rehabilitation progressed uneventfully and at one year follow-up the patient was asymptomatic without snapping and no appreciable hamstring weakness.

DISCUSSION

Although anatomical variations to the biceps femoris insertion are known,[6] symptomatic snapping of the biceps femoris tendon due to an anomalous insertion is a rare clinical entity; only two previous cases have been reported[3,4]. Other causes of symptomatic knee popping in adolescents include semitendinousus snapping,[3] meniscal tears, discoid meniscus,[1] and congenital snapping of the knee[2]. In congenital snapping, articular dysplastic features are associated with subluxation in extension and reduction or snapping with flexion. As in the current report, both previous cases of snapping biceps femoris tendon were bilateral with symptoms on only one side[3,4]. In contrast with the other cases published, snapping was only appreciated with internal tibial rotation during range of motion from 120 to 100 degrees. During this maneuver, rotation tightens the tendon and places the fibular head in a prominent anterolateral position; both of which enhance the snapping.

During surgical exploration the complete biceps femoris tendon was noted to insert entirely on the anterolateral aspect of the proximal tibia. The tendon was reinserted into the posterolateral fibular head with resolution of symptoms and snapping. Kristensen et al, reported a similar anomalous insertion of the tendon; however, surgical treatment in this case involved fibular head resection[3]. The possibility of inadequate bone resection and continued symptoms is contrasted with our anatomic positioning and concordant risks of tendon rupture. Lokiec et al, reported an abnormal insertion of the tendon on the anterior aspect of the fibular head; in that case, symptoms were relieved after partial repositioning to the anatomic position[4].

In conclusion, lateral knee snapping from an anomalous insertion of the biceps femoris tendon may become symptomatic. Although rare, the incidence is not truly known as asymptomatic cases do exist as evidenced in the current and previous reports. Failing conservative treatment the authors prefer anatomic repositioning.

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Figure 1. Intraoperative photograph demonstrates anterolateral tibial insertion and anterior positioning of the biceps femoris tendon over the fibular head at 90 degrees of knee flexion.

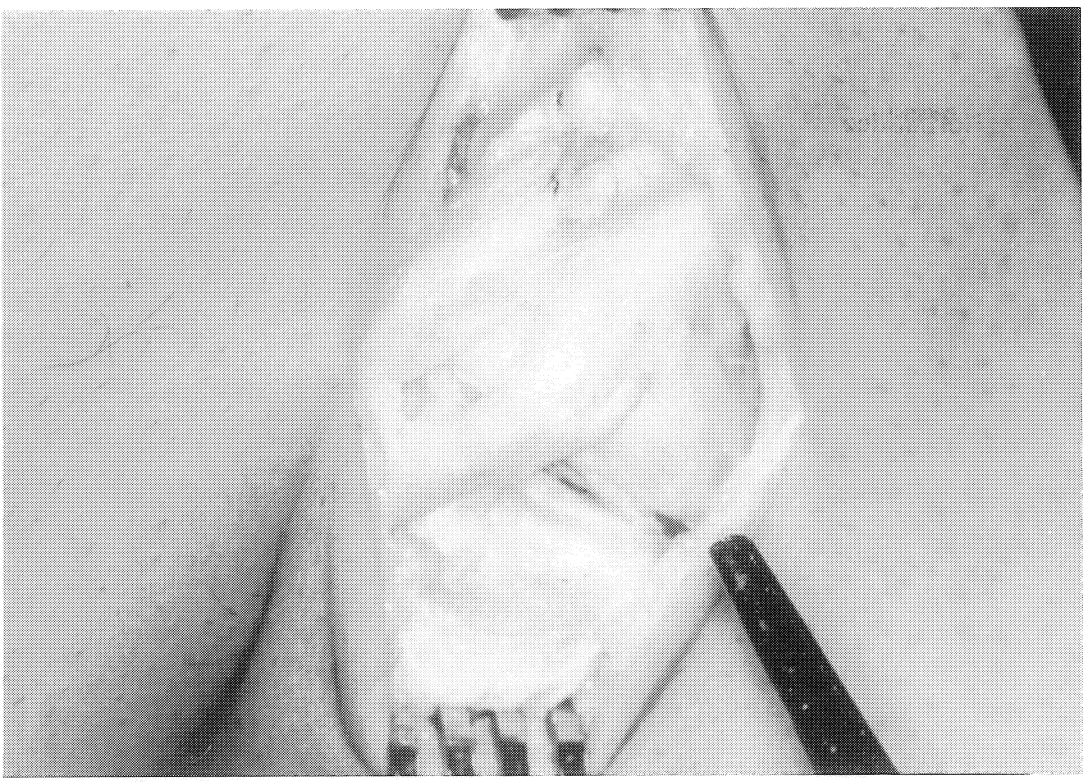

Figure 2. Intraoperative photograph demonstrates that with further flexion greater than 120 degrees and internal rotation the biceps femoris tendon displaces over the fibular head.

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THE NATURAL HISTORY OF THE NEGLECTED FELON

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Peter J. L. Jebson, M.D.

A felon is a closed-space abscess of the distal pulp of a finger or thumb most frequently caused by *Staphylococcus aureus*[1]. The etiology is controversial but is felt to be secondary to direct inoculation since the majority of patients have a history of preceding penetrating trauma. Treatment involves incision and drainage combined with antimicrobial therapy. The neglected or untreated felon is extremely rare and only anecdotally mentioned in the literature[1-3]. Recognized complications include skin slough, osteomyelitis, septic arthritis and flexor tenosynovitis, although the latter is extremely rare. We describe a case of a neglected felon that has all of these features in addition to the previously unreported 'triad' of osteomyelitis, secondary suppurative tenosynovitis and flexor pollicis longus tendon rupture.

CASE REPORT

A 51 year old African-American, right hand dominant male presented with a one month history of progressive right thumb pain and swelling. The symptoms were initially noted in the distal most pulp region of the thumb and began insidiously with no history of penetrating trauma. The patient ignored his symptoms until fourteen days later when the pain became intolerable and the swelling had progressed to involve the entire thumb. He then sought treatment from his family physician who unsuccessfully attempted to aspirate and drain the thumb. Four days later, the patient presented to our clinic with an intensely swollen and painful thumb (Figure 1).

The patient's past medical history was significant for non-insulin dependent diabetes mellitus and he was poorly compliant with medical management. He also had a history of tobacco, alcohol and intravenous drug abuse.

On physical examination, the patient was afebrile and the vital signs were stable. The entire thumb was diffusely swollen and the distal pulp and overlying epidermis was necrotic. The thumb was held in slight flexion at both the metacarpophalangeal (MCP) and interphalangeal (IP) joints. There was no evidence of a puncture wound or draining sinus. The entire volar surface of the thumb from the distal pulp space to the MCP crease was exquisitely tender to palpation. Attempts at passive extension of both joints exacerbated the patient's pain. Neurovascular examination of the right upper extremity was normal. The thenar eminence and volar forearm were non-tender to palpation. There was no evidence of an ascending lymphangitis, epitrochlear or axillary adenopathy.

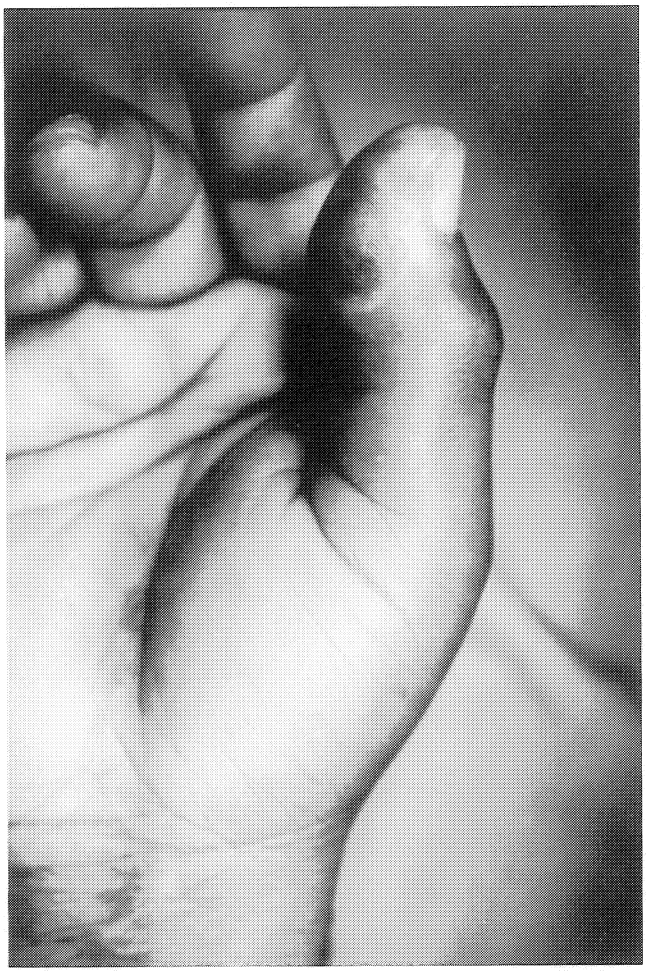

Figure 1. The thumb at presentation.

Radiographs of the thumb revealed diffuse soft tissue swelling, osteopenia and erosions of the distal phalanx (Figure 2). Admitting laboratory values included a blood glucose of 588 mg/dl (normal 65 - 110), white cell count of 14,800 cells/mm^3 with a left shift, and a c-reactive protein of 11.2 mg/dl (normal 0 - 0.4).

A presumptive diagnosis of a neglected felon with secondary osteomyelitis of the distal phalanx and a suppurative tenosynovitis of the flexor pollicis longus (FPL) tendon sheath was made. Emergent exploration of the thumb was performed through a volar Bruner-type inci-

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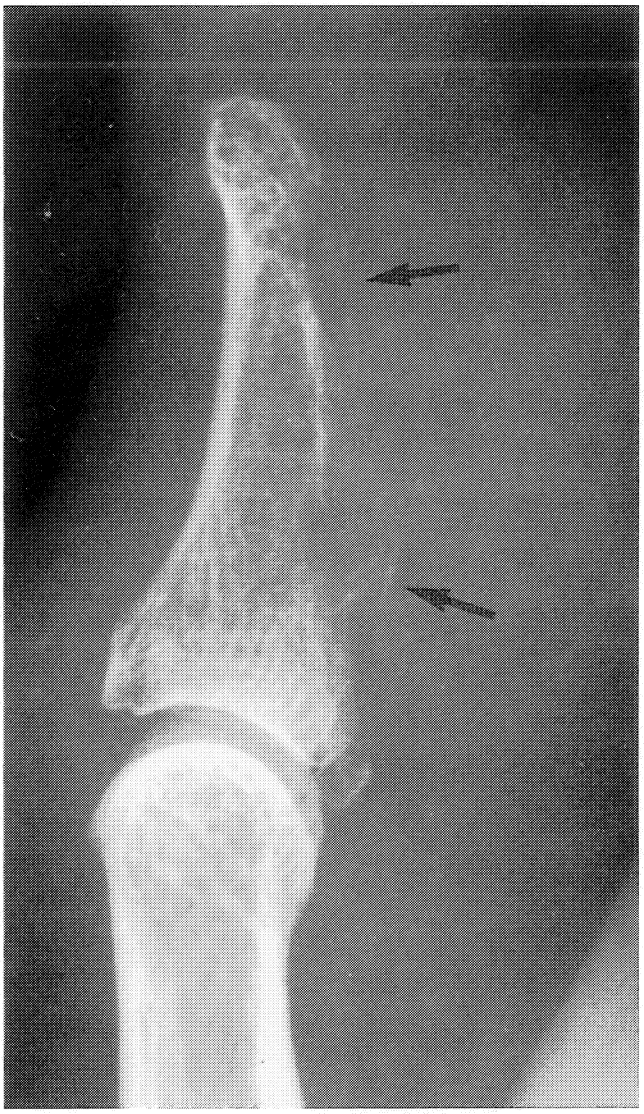

Figure 2. Lateral radiograph of the right thumb illustrating the diffuse soft tissue swelling and erosions involving primarily the volar aspect of the distal phalanx (arrow).

sion. Purulent, necrotic material was encountered throughout the pulp space and volar subcutaneous tissues to the level of the MCP crease. The infection had eroded through the periosteum of the distal phalanx, into the medullary canal. The FPL tendon sheath was also eroded distally with purulence noted within the sheath itself. The FPL tendon was ruptured just proximal to its insertion on the distal phalanx (Figure 3). Debridement of the soft tissues followed by amputation through the DIP joint was performed. The FPL tendon sheath was then irrigated through a separate carpal tunnel incision. Tissue specimens were sent to microbiology for culture and sensitivities. An intraoperative gram stain revealed gram positive cocci in clusters. The wounds were left open. The patient

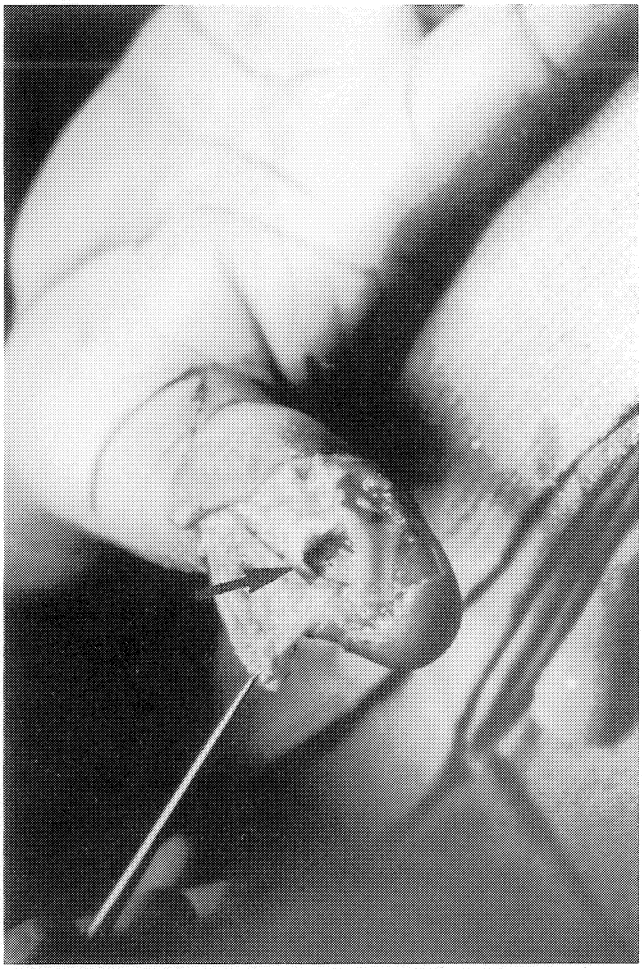

Figure 3. Intraoperative view of the thumb demonstrating erosion of the FPL tendon sheath (arrow) and absence of the FPL tendon.

was started on Unasyn (ampicillin/sulbactam) 3g IV Q6°. Repeat debridements were performed at 48-72 hour intervals until there was no evidence of active infection or necrotic tissue. Because the wound could not be closed in a delayed primary fashion, whirlpool treatments and dressing changes were initiated to promote secondary healing. Final wound cultures revealed a polymicrobial infection involving *Staphylococcus aureus, Enterobacter cloacae,* and *Klebsiella* species. The antibiotic regimen was subsequently changed to nafcillin and gentamicin based upon antibiotic sensitivity studies. The thumb wound failed to completely close by secondary intention, and, as a result, the proximal phalanx and overlying periosteum became desiccated and necrotic. The thumb was subsequently amputated through the MCP joint and the wounds closed primarily. Intraoperative bone and tissue cultures remained negative and the antibiotic regimen was discontinued. Two months later, all wounds were healed without evidence of recurrent infection (Figure 4). The patient complained of the inability to grasp objects and thus, a web

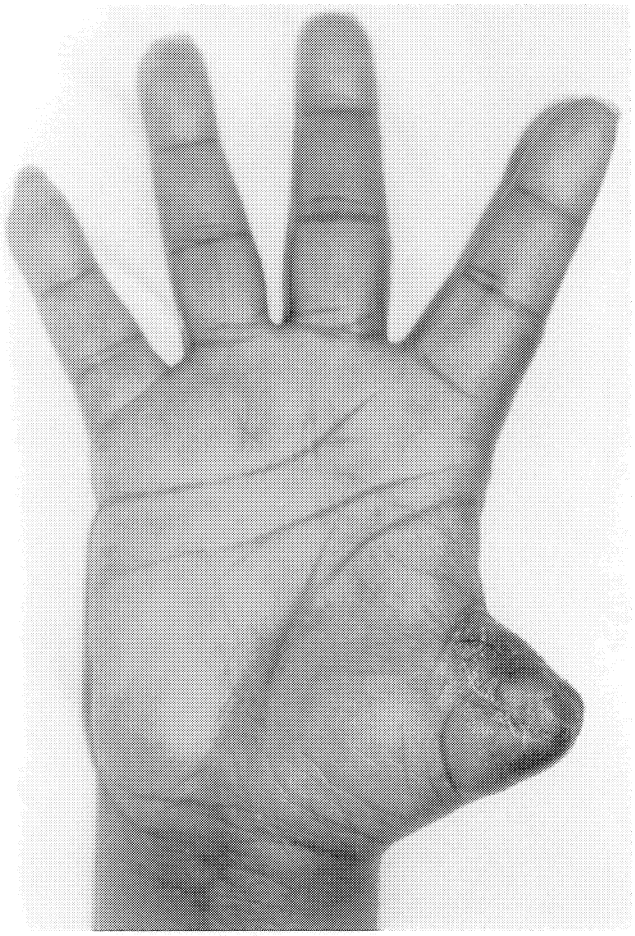

Figure 4. Appearance of the thumb remnant three months following amputation through the metacarpophalangeal joint.

space deepening procedure was being considered to enhance function.

DISCUSSION

The natural history of the untreated or neglected felon has been briefly described in the literature[1-3]. The associated complications of skin necrosis, osteomyelitis, septic arthritis and tenosynovitis appear to occur in a predictable pattern. As the untreated abscess persists and increases in volume, pressure within the noncompliant pulp space increases vascular congestion and causes ischemia of the adjacent structures. The infection may spontaneously decompress through a sinus tract and/or erode into the periosteum of the terminal phalanx. Direct invasion of the phalanx itself can follow, resulting in osteomyelitis. With continued spread into the adjacent interphalangeal joint, a secondary septic arthritis can ensue. If the volar cortex of the terminal phalanx becomes involved, the infection can spread into the adjacent flexor tendon sheath producing a suppurative flexor tenosynovitis. This mechanism of "spontaneous" sheath infection, however, is described as rare or "never encountered," with those sheath infections reported in patients with pulp space infections the result of inadvertent penetration of the sheath during overzealous incision and drainage[1,2]. Finally, and only in extremely unusual cases with persistent infection, radial and/or ulnar bursal infections occur followed by a "fibrosed, useless hand"[1].

Our patient demonstrated several uncommon features of the neglected felon including suppurative tenosynovitis that appeared to result from direct invasion of the flexor pollicis longus (FPL) sheath. While it is conceivable that infection of the sheath may have been produced iatrogenically during 'aspiration' of the thumb, the intraoperative findings support the 'spontaneous' mechanism of direct invasion from the adjacent phalanx. Osteomyelitis of the terminal phalanx with erosion of the FPL insertion also resulted in the previously unreported complication of secondary tendon rupture. Additional unusual features include absence of penetrating trauma to the thumb, the polymicrobial nature of the infection and the significant morbidity of this relatively common hand infection when neglected or not recognized. Our report emphasizes the need for recognition and expedient and appropriate treatment of a pulp space infection, particularly in the immunosuppressed or 'high risk' patient.

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KIRNER'S DEFORMITY: A CASE REPORT AND REVIEW

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CASE REPORT

This patient is a 12 year old girl who was brought by her mother for evaluation of her hands. The parents had noticed a painless, progressive and symmetrical curvature of her little fingers over the past year. There was no history of trauma or infection and she was otherwise healthy. There was no family history of a similar deformity.

Physical examination showed symmetrical palmar and radial curving of the distal phalanges of the little fingers. There was no tenderness, redness or swelling. The nails were deformed with a pronounced convexity. The FDS, FDP and extensor mechanisms all functioned normally. Radiographs are shown in Figure 1. Treatment options were discussed. Since the deformity was painless, continued observation was indicated. The patient will be followed periodically until skeletal maturity.

DISCUSSION

Kirner's deformity is an uncommon condition first described in Germany in 1972[8]. It differs from both clinodactyly (usually radial deviation at the DIP joint) and camptodactyly (flexion deformity at the PIP joint). Over sixty cases have been reported in the literature with a variety of treatments provided.

The deformity consists of a palmar-radial deviation of the distal phalanx associated with a varying degree of rotation. It usually becomes obvious between the ages of eight and fourteen years. Some authors report a mild deformity at birth, but this is an inconsistent finding. There is an approximate two to one female to male predominance. Bilateral, symmetrical involvement is most common, although the right side is occasionally more severely involved.

Inheritance has been described as an autosomal dominant trait with incomplete penetrance[2]. However, many cases arise sporadically[3]. No specific genetic linkage has been identified.

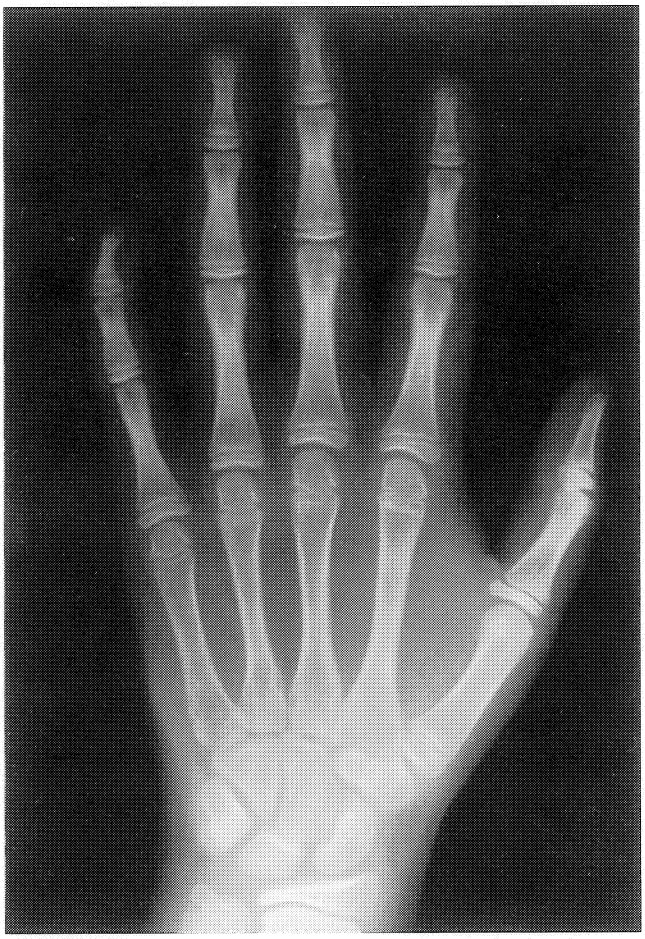

Figure 1A.

The deformity progresses over months to years and is sometimes associated with redness and swelling at the base of the nail. Correlation with a growth spurt may be noted. Pain is a rare finding[12] and functional limitations are usually minimal, confined to difficulty typing or playing musical instruments. Progression halts with closure of the physis.

There have been many associated musculoskeletal abnormalities reported with Kirner's deformity. These include absence of the flexor digitorum superficialis tendon in the little finger[5], Downs syndrome[13], pes cavus[14],

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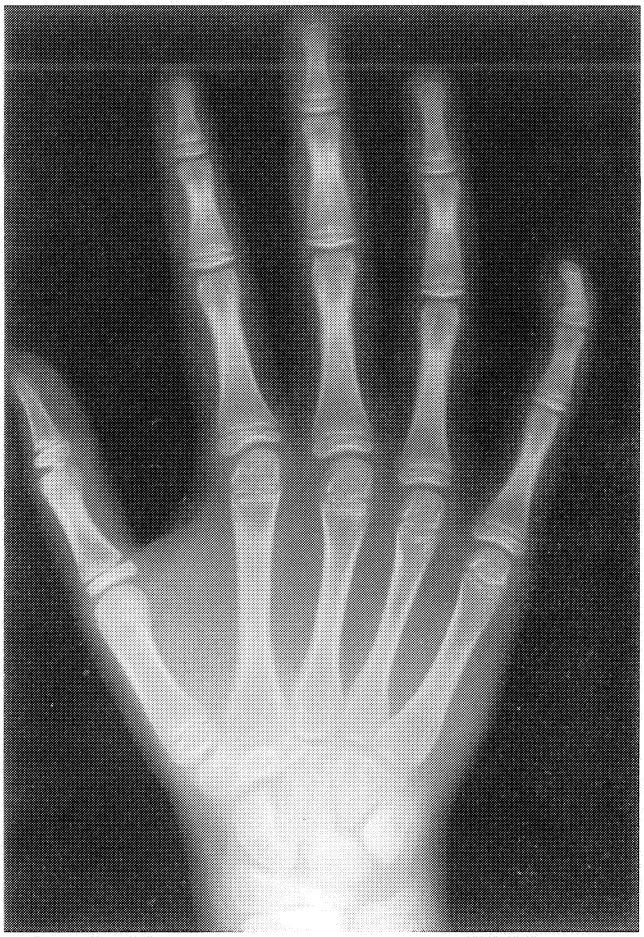

Figure 1B.

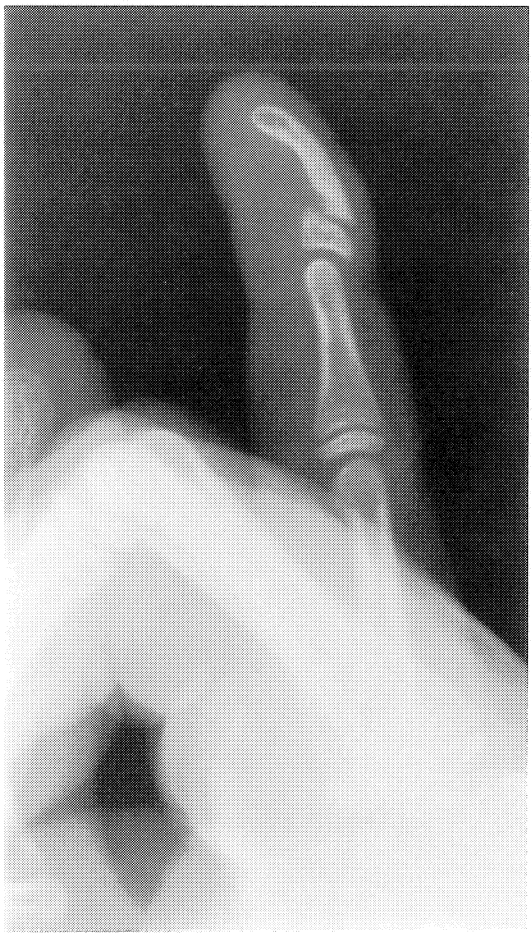

Figure 1C.

Figure 1. AP views of both hands show typical bilateral Kirner's Deformity with radial deviation of the fifth distal phalanx (a and b). Lateral view of the fifth digit demonstrates the typical palmar deviation (c).

progressive myositis ossificans[12], Turner's syndrome[2], Silver syndrome and Cornelia de Lange syndrome[11].

Flatt pointed out that frostbite may produce a similar deformity by causing premature closure of the distal phalanx physeal plate[4]. However, this tends to involve additional fingers and may also affect the middle phalanx. Trauma and infection can be excluded by history.

Radiographic features are quite consistent[11] and show deviation of the diaphysis with preservation of the epiphyseal, metaphyseal and articular alignment. The physeal plate tends to be widened and the diaphysis is sharply narrowed with a loss of normal trabecular bone structure. With closure of the physeal plate, the diaphysis regains its width and trabecular structure, but the deformity, usually 10-50 degrees, persists. No spontaneous resolutions of the deformity have been reported.

The etiology of Kirner's deformity is not well understood. Only one pathology specimen has been described[7]. This showed lysis between the diaphysis and epiphysis of the terminal phalanx. It has been proposed that the deformity is aggravated by the pull of the flexor digitorum profundus tendon[13]. A systemic cause seems unlikely with such isolated findings. Serum calcium, phosphate and alkaline phosphatase levels were normal in a patient under observation throughout the progression of her deformity[3].

Observation, splinting and osteotomy have been recommended for treatment of Kirner's deformity. Dykes reported on the use of "flat night splints" for patients with pain[3]. The splints did not influence the progression of the deformity and the efficacy of pain relief was not addressed.

Freiberg and Forrest reported a single case of custom splinting, including the DIP joint[5]. The patient was splinted full-time for sixteen months with monthly splint revisions to ensure adequate fit. Some straightening of the diaphysis was noted at the conclusion of treatment and closure of the physis.

Carstam and Eiken recommended an osteotomy to correct the "cosmetic problem . . . especially because of its female predilection."[1] However, their only documented case was of a seventeen year old who was willing to

undergo amputation to correct the "unsightly" deformity. Surgery was delayed until physeal closure in order to prevent recurrence of the deformity. They advised one or more volar osteotomies, leaving an intact dorsal periosteal hinge with K-wire stabilization[6].

Finally, epiphysiodesis and corrective osteotomies have been reported in the German and French literature. Niederwieser and Segmuller reported a microscopic hemi-epiphysiodesis without further progression and with normalization of the nail form[10]. Mantero described the Bonola technique of a dorsal closing wedge osteotomy with intra- or extraosseous K-wire fixation[9]. Both authors felt further trials were warranted.

SUMMARY

Kirner's deformity is an uncommon "characteristic" palmo-radial curvature of the distal phalanx of the little finger. Splinting may be beneficial for pain relief and, if used early, may retard progression of the deformity. Disability is usually minimal and treatment to correct the deformity may be delayed to prevent recurrence.

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Not Pictured:	Ignacio V. Ponseti, James N. Weinstein, James V. Nepola, Charles L. Saltzman, Peter J.L. Jebson, Laura J. Prokuski, Todd M. Williams, David K. Sneller, Lisa M. Morton