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# THE IOWA ORTHOPAEDIC JOURNAL

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# IOWA ORTHOPAEDIC JOURNAL

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## INSTRUCTIONS FOR AUTHORS

Any article relevant to orthopaedic surgery, orthopaedic science and 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 1993 journal is February 1, 1992.

Articles published and their illustrations become the property of *The Journal*.

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 this copy of *The Journal* and follow style exactly.

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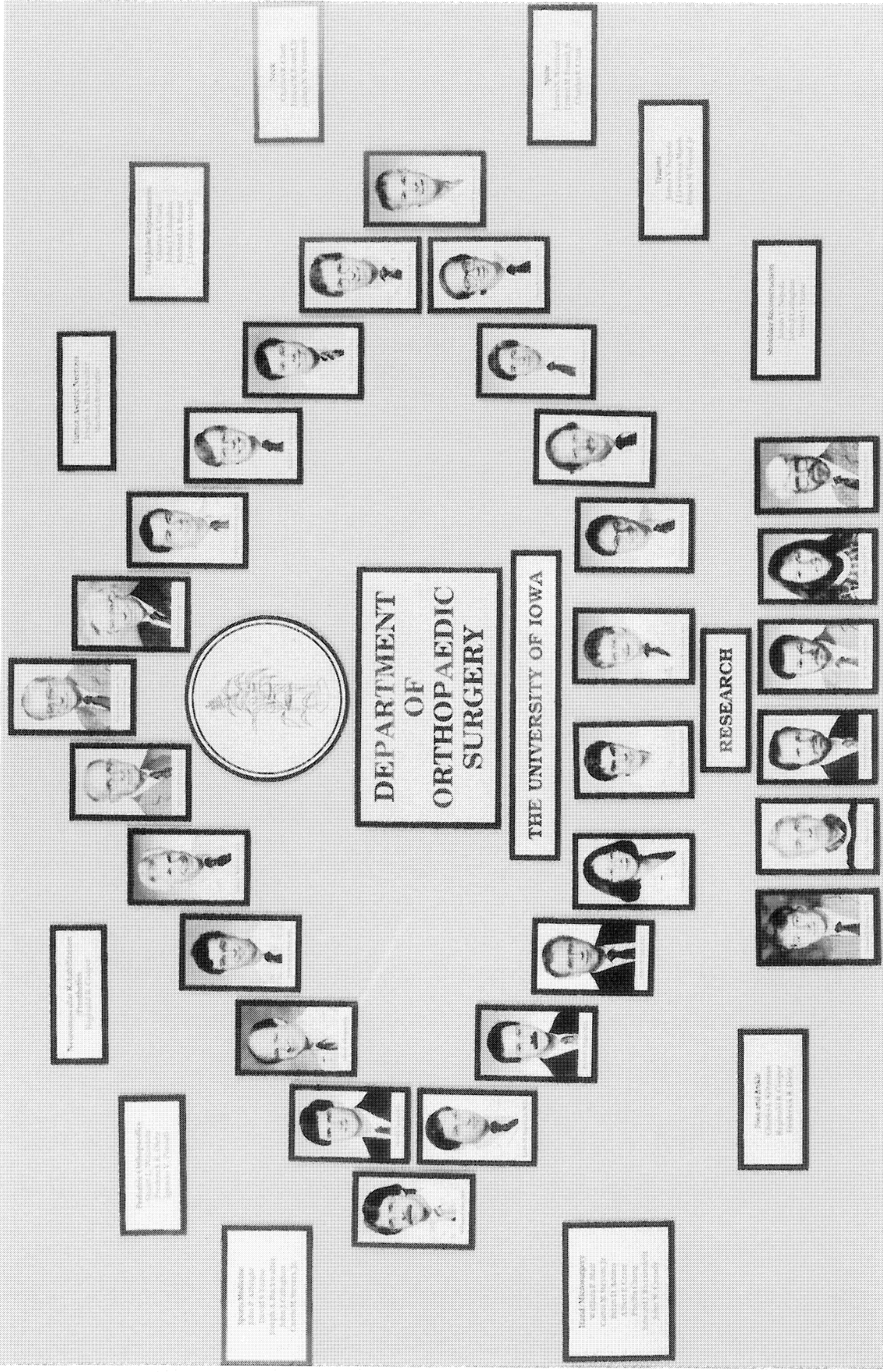
a. *Black-and-white glossy prints* of photographs. Give *magnification* of photomicrographs.

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**Preparation of manuscript:** Manuscripts must be typewritten, double-spaced with wide margins. Write out figures under 100 except percentages, degrees, or figures 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 less than two years' follow-up be accepted.

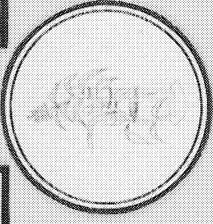
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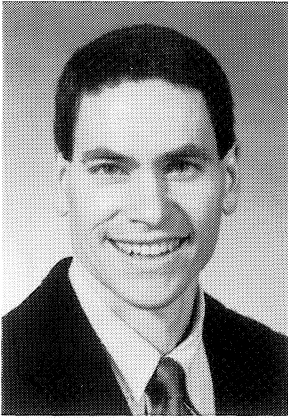
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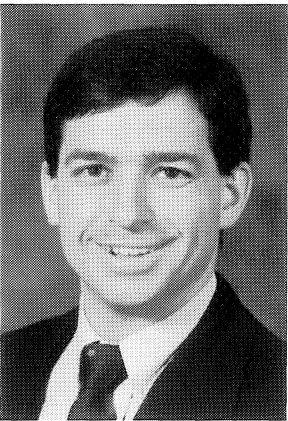
## 1994 GRADUATING SENIOR RESIDENTS



**Lacy Eugene Thornburg, M.D.**

Gene grew up in the western mountains of North Carolina. After graduating from high school, he worked for several years in the logging camps of Alaska. Upon returning, he enrolled in the University of North Carolina at Chapel Hill, where he obtained a bachelors degree in Zoology in 1985. While at Chapel

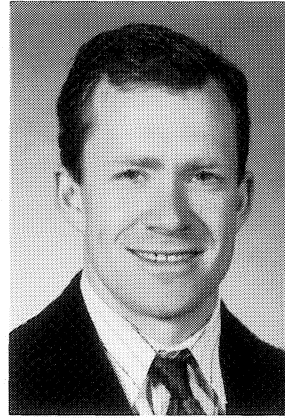
Hill, he met his wife Shannon. Shortly after their marriage, they moved to Boston, where he entered Harvard Medical School and received his M.D. Subsequently, he, along with Shannon and their new son Jesse, moved to Iowa where Gene began his Orthopaedic residency training in 1989. While in Iowa City the Thornburg family has had two additions, son Luke and daughter Whitney. After residency, Gene plans a year of advanced training in Hand and Microsurgery at the Indiana Hand Center.



**Kary R. Schulte, M.D.**

Kary was born in Waukon, Iowa on February 11, 1963, and raised in Cedar Rapids, Iowa. He graduated from the University of Notre Dame in 1985, with a B.S. in Pre-Professional Studies. He attended the University of Iowa College of Medicine, and received his M.D. in 1989. Next year, Kary, his wife Kathleen, and daughter Mary Kate, will be

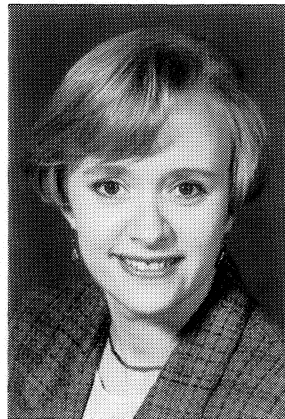
relocating to Pittsburgh, PA, where Kary will be a fellow in Sports Medicine at the University of Pittsburgh.



**Kenneth J. Noonan, M.D.**

Ken is a native of Iowa City; however, home is Rochester, Minnesota where he and his family moved during his childhood. He graduated in 1985 from Luther College with a bachelors degree in Biology and Mathematics. He received his M.D. in 1989 from the University of Iowa College of Medicine. Next year he and his wife

Carrie will remain in Iowa City while Ken completes an NIH Research Fellowship. After further training in Pediatric Orthopaedics, Ken hopes to practice in an academic center.



**Susan K. Bonar, MD.**

Sue is a native of Kansas City. She graduated from the University of Chicago in 1985 and received her M.D. from Yale University in 1989. Next year she and her husband, Dave, will move to Atlanta where she will be a Fellow in Foot and Ankle Surgery at Emory University. She hopes to practice in an academic center in the midwest.

## EDITORS' NOTE



This edition marks the fourteenth year of publication for the *Iowa Orthopaedic Journal*. Each edition has served to solidify a growing tradition focused on education. As editors, our goal was to continue this proud tradition.

The success of the *Journal* depends on the generous support of the contributing authors. This volume contains review articles, case reports and articles in the fields of basic science and clinical research. We hope you find the content stimulating and worthy of discussion.

This year we were fortunate to publish Dr. Jacob's reflections on the early days of Iowa Orthopaedics. It is clear that our department's tradition of excellence would not have evolved without the significant contributions of the great scholars and teachers like Steindler, Larson, Ponsetti, Bonfiglio, and Flatt.

Last year Dr. Adrian Flatt returned to Iowa as a visiting professor for the annual hand lecture series. His greatness as a teacher and expertise in hand surgery were evident to all of us. In recognition of Dr. Flatt's contributions to our department and to the field of hand surgery, we have chosen to dedicate this issue of the *Journal* to him.

Later this year it is anticipated that the Department of Orthopaedic Surgery will move into new facilities in the Pappajohn Pavilion. Those who have worked closely with the architects, particularly Drs. Cooper and Blair and Mr. Paul Etre have ensured that the tradition of our department will not be lost. The Carroll B. Larson Conference Room will be an expanded facility with the capability of accommodating our larger department. State of the art multimedia equipment will serve to enhance our quality conferences, meetings and symposia.

The Steindler Library will remain the central resource in the Department of Orthopaedics for learning and research. The new library will nearly double in size, and will be adjoined by a facility devoted solely to resident education and departmental research. Its continued growth will depend on the generous contributions from many individuals. One individual whose support has been unwavering is Martin Steindler, nephew of Dr. Arthur Steindler.

Finally, we would like to thank Mr. Paul Etre, Ms. Laura Cole, and our faculty advisors, Drs. Cooper, Clark and Buckwalter, who made this publication possible.

James Krieg  
George Emodi



## ADRIAN E. FLATT - SURGEON AND SCHOLAR

Joseph A. Buckwalter, John J. Callaghan & William F. Blair  
University of Iowa Orthopaedics Department  
1201 Carver Pavilion  
Iowa City, IA 52242

The great clinical and scientific achievements of the University of Iowa Orthopaedics Department and its prominent national and international role in advancing the specialty result primarily from the efforts of a small group of talented faculty members. Each one of these people chose to commit decades of their professional lives to patient care, teaching, research and the advancement of Orthopaedics at the University of Iowa. Adrian E. Flatt, M.A., M.D., M. Chir, FRCS, FACS (Figure 1) is among this group of remarkable Orthopaedic surgeon scholars that includes Arthur Steindler, Ignacio Ponseti, Carrol Larson, Michael Bonfiglio and Reginald Cooper.

During Dr. Flatt's 22 years at the University of Iowa he used his clinical skills to improve the quality of life for thousands of patients and developed an academic hand surgery program which contributed innovative concepts in

clinical care, basic biomechanical research involving the upper extremity and educational opportunities for students, residents and hand fellows (Figure 2). Dr. Flatt's nearly 200 publications and three books<sup>1</sup> increased understanding of disorders of the hand and their treatment for physicians throughout the world. The more than 50 hand surgeons he accepted as his Hand Fellows from 14 different countries have carried his teachings and commitment to scholarship throughout the world.

Although Dr. Flatt has spent most of his professional life in the United States and has been a United States citizen since 1960, his heritage and formal education are English. The Flatt family traces their ancestors to the Vikings who raided England's eastern seaboard beginning in the eighth century. By the last decades of the ninth century the Scandinavians had established permanent farming settlements in Eastern England, and one family group adopted the name Flatt from the flat lands of the Thames estuary.

Adrian's father, Leslie Neeve Flatt, a railway engineer, married Barbara Allen, a commercial artist, on September 3, 1918 in the Church of all Souls in London. Born August 26, 1921 in Frinton-on-Sea England, Adrian Ede Flatt, along with his sister Penny, spent much of his youth in the town of Frinton overlooking the North Sea. At the time of

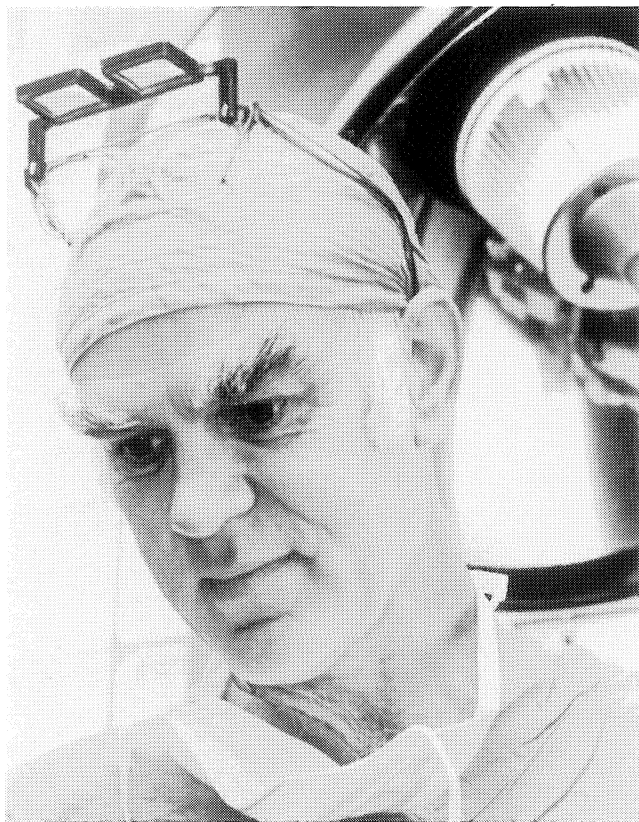


Figure 1

Dr. Adrian Flatt in the operating room at University of Iowa Hospitals and Clinics



Figure 2

Dr. Adrian Flatt and Dr. Carrol Larson at the 1978 senior residents picnic located in the courtyard of the Steindler building.

Adrian's birth, his father was working as an engineer for the British Railways in India. As a result, soon after his birth Adrian and his mother left for India where he spent the early years of his life watched over by a personal attendant.

Adrian's first memorable encounter with a physician came as a result of a skeletal deformity he contracted in India. At about two years of age he developed progressive knock-knees. His mother took him back to England by sea to have the deformity evaluated by Dr. Still, the internationally recognized English pediatrician whose accomplishments include the description of Still's Disease. Dr. Still briefly observed Adrian and then informed his mother, "Your kid has rickets Mam. Feed him milk. That will be five guineas, please." Adrian left impressed by Dr. Still's ability to quickly provide a definitive diagnosis and treatment.

By age five Adrian Flatt had returned to live in England, although his father continued to work in India for periods as long as two and one half years. Adrian began his formal education at the age of 5 in the Connaught School in Frinton-on-Sea. His report for the term ending December 1926 shows that he was only "fairly good" at "drawing and colourwork," but a "neat worker" in paper-cutting, and "promising" in play-acting. His skills in the latter two subjects served him well as a surgeon and teacher. At age eight, he began attending a residential school where he lived in a dormitory with his classmates for nine months of the year (Figure 3). In 1935, at age 14, he entered Haileybury College<sup>2</sup> where he completed his pre-collegiate education. His academic performance at Haileybury documented his intellectual talents and he was accepted to Cambridge University in 1939.

At Cambridge Adrian Flatt joined a class of about 200 students pursuing an education in medicine<sup>3</sup>. In 1942 he graduated from the University with his bachelors degree in



Figure 3

Adrian Flatt holding the soccer ball as the captain of his residential school soccer team.

Natural Science, and moved from Cambridge to London where he completed his medical school education at the Royal London Hospital in 1946. He then served a rotating internship at the London Hospital before becoming a resident surgical officer to the plastic surgery unit at Stoke Mandeville Hospital in 1947. During his time as a house officer in London, Dr. Flatt worked with two of the most important and influential orthopaedic surgeons of the era, Sir Reginald Watson-Jones and Sir Henry Osmond-Clark. His exposure to these two charismatic personalities and their work showed him the great potential of orthopaedic surgery for improving musculoskeletal function. In 1948, Dr. Flatt completed his plastic surgery training and published his first scientific paper: a report of his doctoral studies on the successful storage of skin grafts in refrigerators<sup>4</sup>.

In early 1949 Dr. Flatt applied to become a surgeon in the Royal Air Force, and solicited the support of Watson-Jones and Osmond-Clark. Sir Reginald Watson-Jones wrote:

Dr. Adrian Flatt spent six months as house surgeon in my department at the London Hospital when he had many opportunities of gaining experience in the treatment of casualties, fractures and orthopaedic conditions. He proved to be a very keen, alert and stimulating house surgeon and I formed a high opinion of his capabilities.

Sir Osmond-Clark wrote:

I have the great pleasure in writing this testimonial for Mr. Adrian Flatt, who was my House Surgeon at the London Hospital for six months. I found him extremely efficient, keenly interested in his work, with a quick perception and above average intelligence. He combined with these qualities a very courteous and delightful personality.

Perhaps partially as a result of these recommendations, Dr. Flatt became a flight lieutenant (Captain) surgical specialist at the RAF Hospital in Ely England and was trained and appointed as the surgeon in charge of a parachute rescue team. He was soon promoted to squadron leader (Major), and in late 1949, he became the surgeon assigned to all armed forces in Ceylon (Sri Lanka), at the RAF Services Hospital (Figure 4). He had responsibility for all surgery performed at this hospital and also served as a visiting consultant to RAF stations throughout Ceylon, Singapore, Malaya, Indo China and Hong Kong (Figure 5). During this time he participated vigorously in the activities necessary for maintaining the proper British Military presence overseas including rugby, Military balls and other events that required consumption of appropriate liquid refreshments<sup>5</sup>. In 1950 he returned to England to become a demonstrator of anatomy at Cambridge University and subsequently the Royal College of



Figure 4

An open orthopaedic ward at an RAF Services Hospital in Ceylon in 1949.

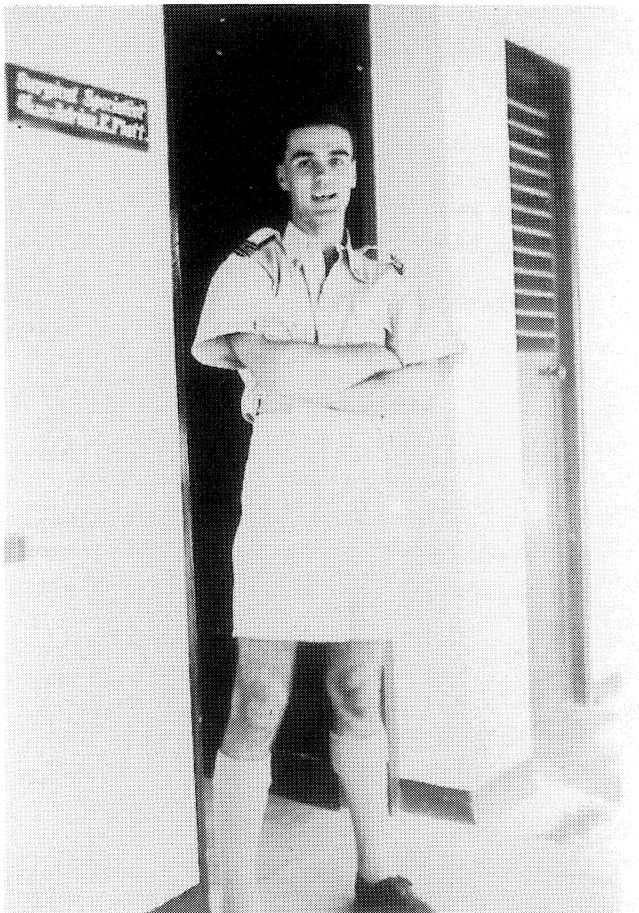


Figure 5

Dr. Flatt, a squadron leader surgical specialist stationed in Ceylon in 1950.

Surgeons. He received his doctorate (M.D.) in 1951 for his studies of autogenous skin grafts, and in 1953 he was named a fellow of the Royal College of Surgeons.

Having completed doctoral studies on skin grafts, plastic surgery training, military service and three years of teaching anatomy, Dr. Flatt continued to develop his keen interest in hand anatomy, mechanical function of the hand and upper extremity and the surgical treatment of hand injuries and diseases. After considering the option of continuing as a plastic surgeon, he decided that training in orthopaedics would provide a better education for a surgeon who wanted to improve the function of hands impaired by injury or disease. He applied to Sir Reginald Watson-Jones for the position of first assistant<sup>6</sup> in the Orthopaedic and Accident Department service of Sir Reginald Watson-Jones and Sir Henry Osmond-Clark and promptly received a telegram that read, "Many congratulations on breadth of experience and personal character acknowledged by all that made your appointment today easy—Reginald Watson-Jones".

During 1954 and 1955, Dr. Flatt received support from a Fulbright Scholarship in the United States to study surgery of the hand. He took advantage of this opportunity working for six months as a fellow with Dr. J. William Littler at Roosevelt Hospital in New York City and for three months as a fellow with Dr. Daniel Riordan in New Orleans at the Carville Leprosarium. He spent the other three months of his scholarship lecturing and visiting clinics throughout the United States.

In 1955, Dr. Flatt rejoined Watson-Jones in London. Shortly after his return to England, Carroll Larson, Chairman of Orthopaedics at the University of Iowa, offered Dr. Flatt the opportunity to develop an academic hand surgery program in Iowa City. Partially because of the limited level of salary support in England, approximately £1000 per year, and partially because of the opportunity to develop a hand surgery program in a well recognized Orthopaedics Department, Dr. Flatt accepted a position as an Associate to the Department of Orthopaedic Surgery at the University of Iowa Hospitals in Iowa City, Iowa. Sir Watson-Jones initially disapproved of Dr. Flatt's decision, but in less than two years agreed that it may have been a wise choice for his former first assistant (Figure 6).

Dr. Flatt first saw Iowa City on June 30, 1956, when he arrived at the Iowa City airport in a DC3 with his wife, three month old son, no furniture or other household necessities and a total of \$83. July 1, 1956, Virgil Hancher, the president of the University, formally appointed Dr. Flatt "Associate in Orthopedic Surgery" with a salary of \$6,000 per year. Arrangements were made for him and his family to live in a Quonset hut next to the football stadium. These corrugated metal shelters had been built as temporary housing for the naval flight school based in Iowa City during World War II. Following the war, to help accommodate students and young faculty, the University converted the Quonset huts to family housing by dividing each

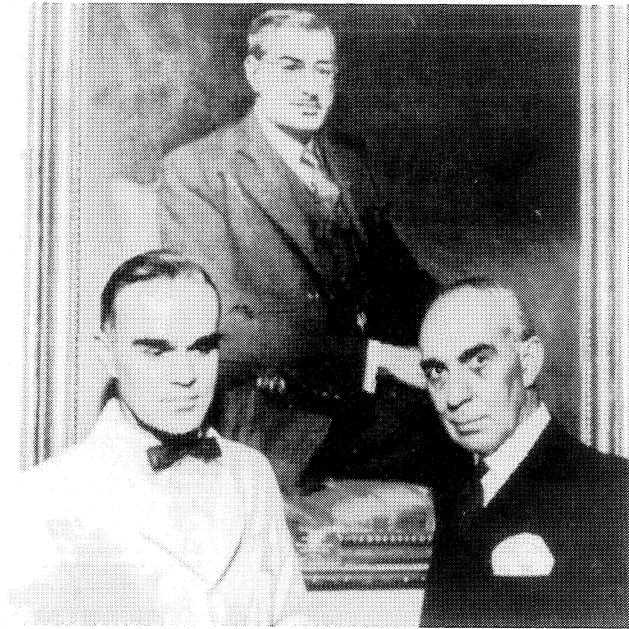


Figure 6

Dr. Flatt with Sir Reginald Watson Jones standing before the portrait of Arthur Steindler in Steindler Library in Iowa City. (1963)

hut in half with a small kitchen in each unit. The units were bitter cold and drafty in the winter and insufferably hot and humid during the summer, but the rent was minimal and it was a short walk to the hospital.

In 1956 the Orthopaedic Department was housed in Children's Hospital. The structure had been completed in 1917 primarily due to the efforts of Arthur Steindler, the first Chairman of Orthopaedics at Iowa<sup>7</sup>. The faculty consisted of the Chairman, Dr. Carroll Larson, and Drs. Michael Bonfiglio and Ignacio Ponseti. The program accepted four residents a year. The clinics, operating rooms, physical therapy facilities and inpatient areas, located within Children's Hospital, served large numbers of patients suffering from post-polio paralysis and deformity, or bone and joint tuberculosis.

Dr. Flatt's initial responsibilities included general orthopaedic clinics and teaching anatomy to the orthopaedic residents, a task he continued with great enthusiasm throughout his career at Iowa. The development of an academic hand surgery program soon replaced his general orthopaedic practice. Initially he focused much of his clinical effort on the evaluation and treatment of patients with acute minor hand injuries (Figure 7). Based on his experience with these patients and his previous work in England he wrote his first book, "The Care of Minor Hand Injuries," published in 1959. His practice soon included increasing numbers of patients with disorders of the hand due to rheumatoid arthritis and congenital anomalies. His studies of the evaluation, treatment methods and outcomes of treatment for these patients provided the basis

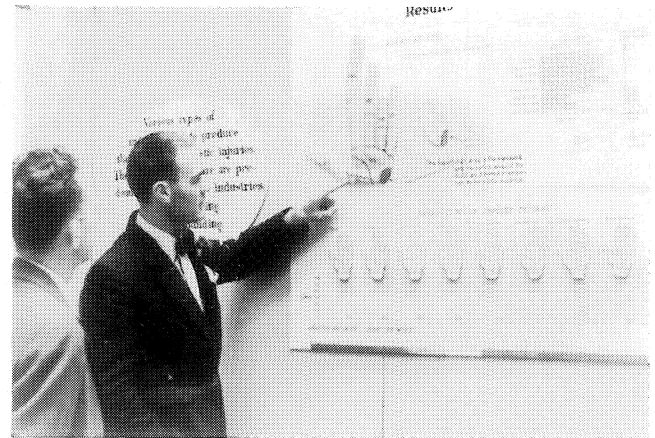


Figure 7

Dr. Flatt with an exhibit he prepared at the University of Iowa on the types and anatomic distribution of hand injuries.

for his second book, "The Care of the Rheumatoid Hand," published in 1963, and his third book, "The Care of Congenital Hand Anomalies," published in 1979.

Dr. Flatt's expertise as an anatomist led the Anatomy Department to ask him to lecture in their medical school courses. Within a short time, few students would miss a lecture given by Dr. Flatt, even if they felt they had already mastered the anatomy of the hand. Long after most of them forgot the origins and insertions of the intrinsic muscles of the hand, they remembered his stimulating demonstrations of the function of the human hand. As a result of Dr. Flatt's contributions to the Anatomy Department he was appointed a full Professor of Anatomy.

In 1961, Dr. Flatt began the development of a laboratory for the study of biomechanics of the upper extremity. He was particularly interested in the problems of the pathomechanics of ulnar drift and the mechanics of the normal wrist and metacarpal-phalangeal joints. As part of the laboratory development, he helped organize MD/Phd and masters programs with the College of Engineering and the Department of Anatomy. Graduates of these programs include Gary Fisher, Professor of Engineering, Youngil Youm, Professor and Chairman, School of Mechanical Engineering, Prohang, Korea and Richard Berger, a member of the Mayo Clinic Hand Surgery Staff (Figure 8). In 1972, the American Academy of Orthopaedic Surgeons recognized Dr. Flatt's innovative studies of hand and upper extremity biomechanics and their relationship to ulnar drift by awarding him the Kappa Delta Award for outstanding orthopaedic research.

In 1962, he started a post-residency training program in surgery of the hand. He restricted appointments to individuals who were Board qualified surgeons in general, plastic or orthopaedic surgery. Although the majority of surgeons who completed the program were United States citizens, he also trained individuals from Argentina, Aus-



Figure 8

Dr. Flatt posing with his co-workers in the hand and upper extremity biomechanics laboratory in the basement of the Steindler Building.

tralia, Brazil, Canada, India, Great Britain, Japan, Malaysia, Mexico, Poland and Puerto Rico. In addition to their clinical experience, Dr. Flatt's hand fellows conducted research projects, many of them related to congenital and rheumatoid hand problems.

Dr. Flatt's clinical experience, combined with his basic biomechanical studies of the problems of rheumatoid hand deformities, led him to investigate corrective procedures including crossed intrinsic transfers and flexor tendon transfers along with prosthetic joint replacements. Based on these studies he developed prostheses for the wrist and finger joints that were recognized by United States patents<sup>8</sup>. One of his implants, a steel hinge with two prongs projecting from each side of the hinge (the Flatt prosthesis), gained national attention in a December 15,

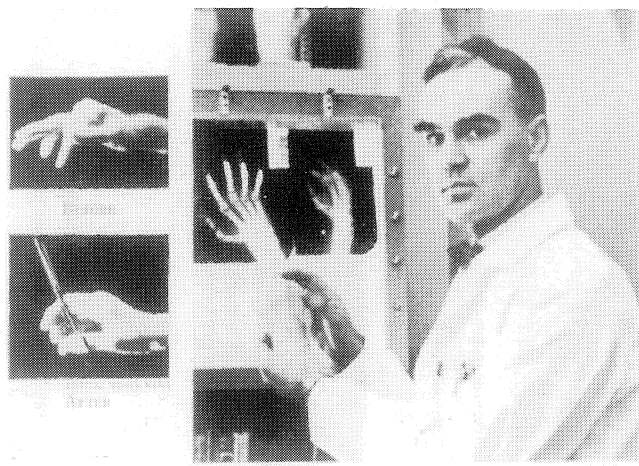


Figure 9

Photographs showing a barber's hands before and after replacement of his metacarpal-phalangeal joints with Flatt prostheses and Dr. Flatt standing in front of radiographs showing Flatt prostheses.

1961 TIME magazine article entitled "Steel Knuckles" (Figure 9). Inventors of other prosthetic joints frequently asked Dr. Flatt to evaluate their devices in his laboratory. In many instances the results sent the implants back to the design stage or encouraged their inventors to buy a commercially available device. Dr. Flatt pioneered the clinical use and evaluation of these devices, and his studies of the long term results of prosthetic replacement of finger joints are among the most important in this area of clinical investigation.

Dr. Flatt's interest in congenital disorders of the hand and upper extremity led him to develop a registry for patients with these problems. This program, jointly supported by the Children's Bureau in Washington D.C. and the Iowa Crippled Childrens Service, included a comprehensive investigation into all aspects of the incidence, prevalence and anatomic patterns of congenital hand deformities. The registry made it possible to conduct a series of critically important clinical studies of congenital



Figure 10

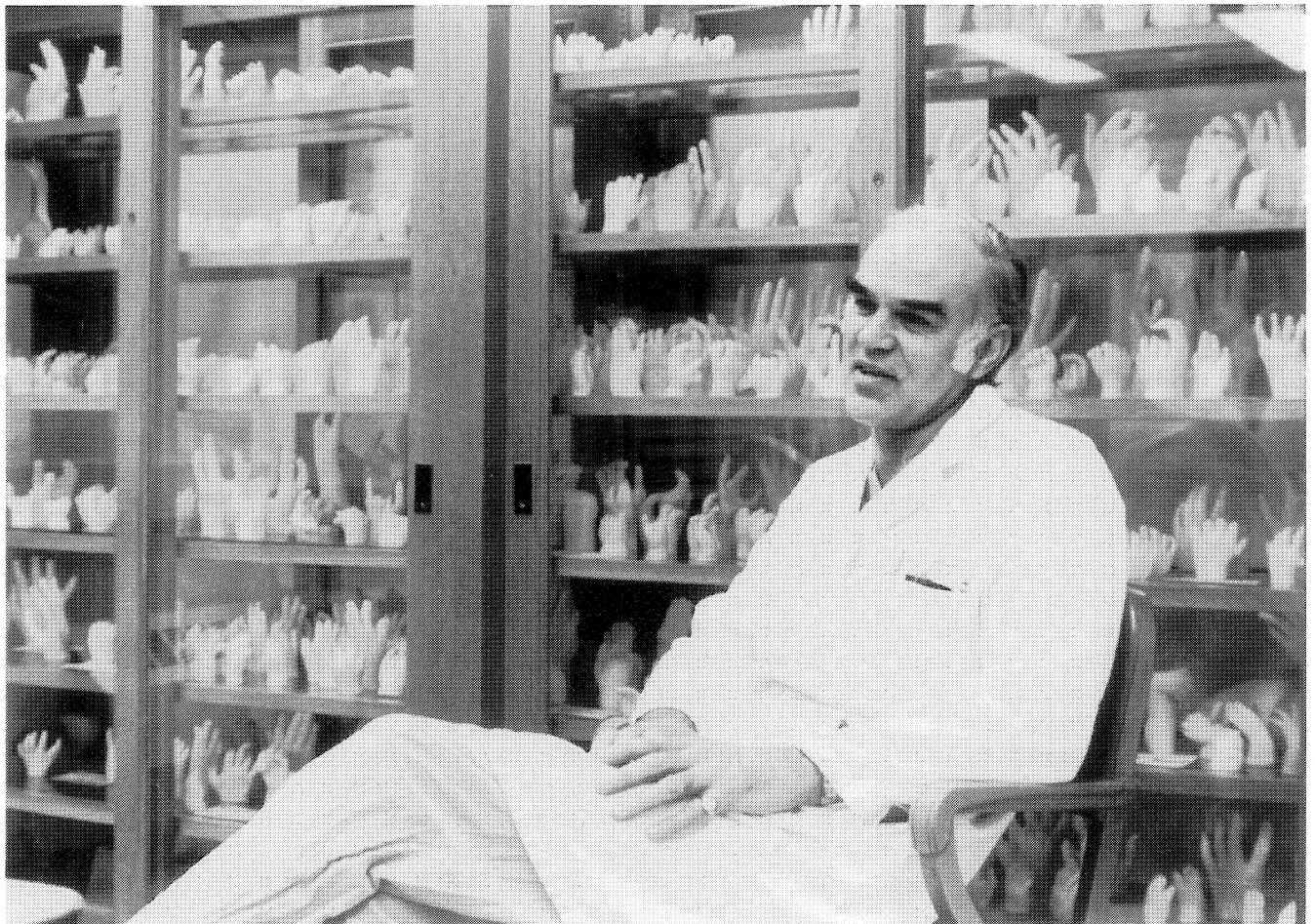
Newspaper photograph showing Dr. Flatt making a mold of President Harry Truman's hand.

hand deformities. The two volume final report of this more than 10 year project was published in 1980.

Among his hobbies, Dr. Flatt includes making and collecting hand casts of patients and public figures (Figure 10). To create the casts, he first makes an alginate mold of the individual's hands. Then he pours a thin layer of methacrylate into the mold, carefully rotating the mold to capture the finest features of the hands, including the finger prints, hair and pores. He finishes the hardened cast by applying bronze paint and then treating them with an antiquing process. Dr. Flatt's residents and fellows had the opportunity to learn this technique under his direction and critical evaluation. His collection of patient hand casts includes almost every known congenital anomaly of the hand (Figure 11), and the collection of hand casts from surgeons, scientists, artists, actors and actresses, astronauts, athletes, and politicians consists of more than 80 pairs of hand casts including those of the Mayo brothers, Harvey Cushing, Arthur Steindler, Sir Reginald Watson-Jones, Christian Bernard, Werner von Braun, Jonas Salk, Linus Pauling, Norman Rockwell, Walt Disney, Andrew

Wyeth, Jamie Wyeth, Andres Segovia, Isaac Stern, Van Cliburn, Katharine Hepburn, Paul Newman, Joanne Woodward, Neil Armstrong, Buzz Aldrin, Alan Shepard, Arnold Palmer, Joe DiMaggio, Mickey Mantle, Wilt Chamberlain, Chris Evert-Loyd, Dan Gable, Winston Churchill, Margaret Thatcher and Presidents Harry Truman, Dwight Eisenhower, Lyndon Johnson, Gerald Ford, Jimmy Carter, Ronald Reagan and George Bush (Figure 12).

In the clinics, Dr. Flatt consistently showed a masterful ability to gain the confidence and admiration of patients and their families, after a brief encounter many of them would speak of him in awe. His authoritative professional manner and definitive answers to the most complex questions left them comforted and confident that they had received the best possible treatment even if there was little or nothing that could be done to help their medical problem. Although he progressively focused his clinical activities on problems of the rheumatoid hand and congenital hand, he did not always restrict himself to these areas, and patients with many other types of orthopaedic problems sought his help.



**Figure 11**

Dr. Adrian Flatt seated in front of his collection of hand casts from patients with congenital hand deformities. (1976 at the University of Iowa)

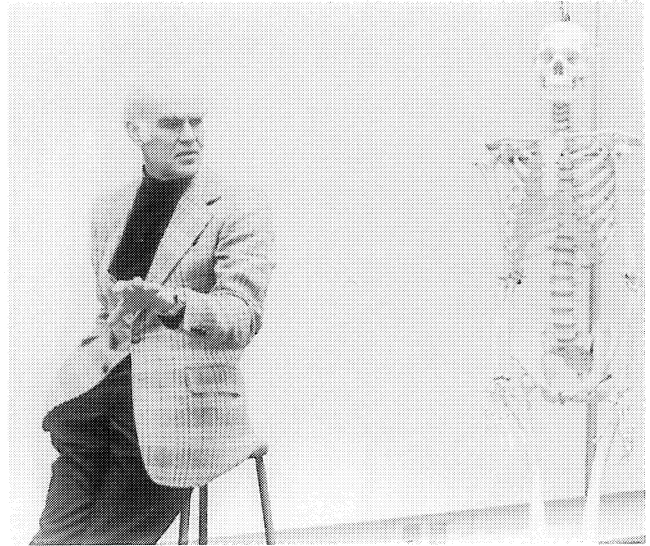


**Figure 12**

Dr. Flatt with his casts of Wilt Chamberlin's and Willie Shoemaker's hands.

One of these patients, a promising track athlete, ruptured his Achilles tendon in 1966. Dr. Flatt recommended the definitive care (immobilization in a plaster dressing) and advised the patient that his body obviously was not intended to "take this sort of abuse," therefore he should follow pursuits other than athletic competition. Despite Dr. Flatt's limited sports medicine practice, his professional manner convinced the patient that Dr. Flatt's advice was correct and the patient abandoned his athletic career<sup>9</sup>.

As a teacher, Dr. Flatt inspired and motivated students and residents (Figure 13). He had unique talents for simplifying and clarifying complex problems and for making the simplest topics fascinating. His lectures entertained as well as educated. When he conducted a surgical indications conference the residents dared not drift off to sleep or even assume a relaxed posture for fear of being rudely



**Figure 13**

Dr. Flatt enjoying one of his favorite activities, lecturing to an attentive audience.

awakened by a series of penetrating questions. When Dr. Flatt began to quiz them, even residents who were fully awake and had prepared assiduously for the conference found themselves struggling to avoid the appearance of having severely limited knowledge of the subject under discussion. When asked a question, Dr. Flatt always provided a sharp clear answer, usually correct and always memorable. On one occasion when challenged by an apparent contradiction between his teaching in the clinic and the text of one of his books, Dr. Flatt advised the students, "Don't believe everything you read."

During Dr. Flatt's tenure at Iowa no resident completed the program without vivid memories of Dr. Flatt's osteology tutorials in the departmental library. A few minutes after the residents had assembled, always on time or ahead of time, he would walk in briskly, and make himself comfortable in a large chair with his worn copy of Frazer's *Anatomy of the Human Skeleton* open in his lap. After appropriate greetings, he would pause while paging through the book, peering through his half glasses at illustrations kept hidden from the residents. Then, consulting the anatomic drawings and his own extensive knowledge, he would proceed to make the most knowledgeable residents extremely uncomfortable by quizzing them on the precise location and significance of every crevice, protuberance, ligament and capsular attachment or relationship to a nerve or blood vessel of a given bone; no feature of a bone's structure, function or development was too obscure to avoid intense discussion.

He took special pleasure in the osteology of the carpal bones. During these sessions he would instruct a resident to close his or her eyes, and then place a carpal bone in his or her open hand. Somehow the bones he selected never

included the hamate or the pisiform. He would give the residents a few seconds before asking them to identify the bone without opening their eyes. If the resident passed this test, he or she was asked to identify each of the surfaces, grooves and contours of the bone while keeping their eyes closed. Residents lucky enough to receive the lunate often did relatively well for a few minutes, but at that point, to help the resident avoid the problem of overconfidence Dr. Flatt would ask if the bone came from the right or left wrist and the likely profession and political affiliation of its owner.

In addition to his contributions to clinical care, teaching and research, Dr. Flatt advanced the field of Hand Surgery nationally and internationally. He helped found the Second Hand Club which evolved into the British Society of Surgery of the Hand. He served the American Society for Surgery of the Hand on multiple committees as well as President; and he was instrumental in the founding of the Journal of Hand Surgery and served as its editor-in-chief for ten years.

In 1979, Dr. Flatt left the University of Iowa to become Clinical Professor of Orthopaedics at Yale University. In 1982, he became Chairman of Orthopaedics at Baylor University Medical Center in Dallas, Texas, a position he held until his retirement from clinical practice in 1992. The medical students in the class of 1995 at University of Texas Southwestern Medical School in Dallas recognized his skill and impact as a teacher by electing him outstanding teacher of the year.

Dr. Flatt currently is the coordinator of research at the Tom Landry Sports Medicine and Research Center, Baylor University Medical Center, Dallas. Despite his busy schedule he is finding time to work on revisions of his three landmark books in hand surgery in addition to teaching anatomy, preparing a new book on surgical anatomy of the hand and giving lectures and surgical demonstrations at medical centers throughout the world.

As a result of his many scholarly achievements Dr. Flatt has received a variety of national and international honors. He has been an invited lecturer at universities throughout the United States, Europe and South America and is an honorary member in the Groupe D'etude De La Main, the British Society for Surgery of the Hand, the Venezuelan Hand Society and the British Association of Plastic Surgery. He was appointed a Hunarian Professor of the Royal College of Surgeons in 1962 and in 1986 was appointed the Sir Reginald Watson-Jones lecturer by the Royal College, being the first of Sir Reginald Watson-Jones' trainees to be so honored<sup>10</sup>. In 1992, he was honored as an international pioneer of hand surgery by the International Federation for Societies of Surgery of the Hand, and he currently serves as a trustee of the Foundation of the Royal College of Surgeons in England.

These well deserved national and international honors are impressive, but they do not recognize some of Dr. Flatt's enduring accomplishments. He helped move forward the excellent Orthopaedic Program at the University of Iowa, especially in the area of hand surgery. His innovative concepts and dedication to academic hand surgery continue to influence the Orthopaedic Department's hand surgery program. The upper extremity biomechanics laboratory continues investigations of wrist kinematics and recent clinical research projects include studies of the functional outcomes of surgical reconstruction of rheumatoid hand deformities and congenital anomalies of the hand. These studies incorporate patients that Dr. Flatt treated and included in his clinical investigations. The gratitude these patients feel for Dr. Flatt's care and the records of his indications for surgery, surgical treatments and post-operative therapy have provided an invaluable foundation for ongoing investigations. In addition to his clinical and academic contributions to the University of Iowa, he attracted students, residents and fellows and transmitted to them an enthusiasm for study and practice of orthopaedics. Many of these individuals, partially as a result of Dr. Flatt's inspiration as a surgeon and scholar, have also contributed to advances in the understanding of the musculoskeletal system and the treatment of patients with musculoskeletal diseases and injuries.

#### ENDNOTES

1. The Care of Minor Hand Injuries, The Care of the Rheumatoid Hand and The Care of Congenital Hand Anomalies
2. Colleges like Haileybury are the equivalent of High Schools in the United States
3. In the English system, physicians must first earn a bachelors degree (B.A.). Those who complete medical school receive a degree as a Bachelor of Medicine and Surgery (M.B.B. Chir.). About five percent of the students who receive a M.B.B. Chir. conduct a major research project and then write and defend a thesis. Successful defense of the thesis leads to a doctorate in medicine (M.D.).
4. "Refrigerated Autogenous Skin Grafting" *Lancet*, August 14, 1948, pp. 249-251
5. Good beer was rare, but British Gin traveled well
6. The equivalent of a senior resident or fellow in the United States
7. Children's Hospital was later renamed the Steindler Building to recognize Arthur Steindler's contributions to the University.
8. The University of Iowa paid Dr. Flatt one dollar for each patent. He framed the one dollar check he received for the first patent and the one dollar bill he received for the second.



9. This method of managing athletic injuries has gone out of favor, but it had the great advantages of relieving the doctor of any responsibility for returning the athlete to their former level of performance and of allowing the athlete to tell people how great he could have been if only the doctor had allowed him to continue to compete as one of the authors can attest.

10. In 1992, the Royal College appointed one of Dr. Flatt's former students and residents as the Sir Reginald Watson-Jones Lecturer.

# APPLICATION OF MULTIPLANAR LIGAMENTOTAXIS TO EXTERNAL FIXATION OF DISTAL RADIUS FRACTURES

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## INTRODUCTION

### Distal Radius Fractures

Of all fractures that can occur in the hand and wrist, those of the distal radius have the greatest potential to impair function. Fractures of the distal radius are most frequently generated by the high-energy impact to the outstretched hand as a result of a fall. Numerous classification systems for distal radius fractures have been devised<sup>14,18,19,26,30,31,33,37-39,41</sup>. As first described by Colles<sup>13</sup>, these extra-articular fractures of the distal radial metaphysis are associated with dorsal displacement of the distal fragment. In many of these fractures, the dorsal cortex and distal fragment are comminuted. Frequently, serious disruption of the distal radial articulations occurs and malalignment of the distal radioulnar joint may result from fracture displacement<sup>34</sup>. Anatomic reduction of comminuted fractures of the distal radius is often a challenge, as these inherently unstable fractures are prone to collapse and displacement<sup>14,22,24,55</sup>.

### Ligamentotaxis

Fractures of the distal radius have the potential to disrupt the mechanical function of the hand drastically. Unfortunately, the same ligaments and tendons that surround the fracture and serve to mold its reduction frequently present barriers to open reduction. The molding of fracture fragments into alignment by traction force applied across the fracture through the surrounding soft tissue is known as ligamentotaxis<sup>52</sup>. Although the capsular and ligamentous structures generally are preserved following comminuted fractures of the distal radius<sup>53</sup>, they can be injured at the time of fracture<sup>35</sup>. Because of these injuries, the integrity of the wrist ligaments may not always be adequate to maintain reduction by ligamentotaxis<sup>55</sup>. Böhler<sup>7</sup> employed uniplanar ligamentotaxis in the form of skeletal traction to maintain length during fracture healing. Anderson and O'Neil<sup>3</sup> were first to maintain fracture reduction with an external fixator using the same principle of ligamentotaxis. DePalma's cadaveric study<sup>16</sup> and numerous clinical studies<sup>8,14,15,22,30,48,49</sup> have documented the value of intact soft tissue in obtaining and

maintaining skeletal length during the fracture healing process. However, clinical studies have frequently revealed that longitudinal traction alone cannot adequately restore joint congruity and palmar tilt to the distal radius. These clinical observations have been confirmed in a cadaveric study in which traction forces applied parallel to the radius could not restore the distal radial articular surface to normal<sup>5</sup>.

## TREATMENT OF DISTAL RADIUS FRACTURES

Both osteoarticular<sup>14</sup> and soft tissue<sup>14,45</sup> complications can result from distal radius fractures. Cooney et al.<sup>14</sup> reported a complication rate for distal radius fractures of over 31% and found that the incidence was correlated with the severity of the fracture. In addition to complications resulting from the initial injury, some complications are related to the method of treatment. The adequacy of fracture reduction is one factor that affects the complication rate<sup>57</sup>. The most favorable treatment of distal radius fractures requires techniques that avoid such complications as hand stiffness, carpal tunnel syndrome and fracture malunion. This section briefly outlines historical approaches to the treatment of distal radius fractures and is followed by a discussion of the anatomical and biomechanical principles upon which state-of-the-art fracture reduction and fixation technology is based.

### Plaster Cast

An early standard approach to the treatment of distal radius fractures, as well as other wrist fractures, was closed reduction and immobilization with a plaster cast<sup>17</sup>. This technique does not always adequately maintain fracture reduction as fixed traction is not obtained. A common problem is shortening of the radius at the fracture site. Active finger motion and use of the hand are compromised by the cast.

### Pins and Plaster

As early as 1929, Böhler<sup>7</sup> recommended that fracture reduction be maintained by the use of transfixing pins incorporated into a plaster cast. This method uses skeletal traction to maintain length during healing of the fracture.

Although this technique usually does not restore normal palmar tilt<sup>15,22</sup>, it is still in wide use today<sup>12,22,29,42,46</sup>.

### Open Reduction and Internal Fixation

There are some instances when bone stability or articular congruity are not obtained by closed reduction of the distal radius fracture<sup>40</sup>. In these instances, open reduction followed by internal fixation is an option. This approach to fracture management is often technically difficult and is frequently accompanied by numerous complications.

### Closed Reduction and External Fixation

Numerous complications were associated with the early use of an external fixator for fracture management. These complications were, in part, related to a lack of understanding of the biomechanical principles involved in the proper use of external fixators. During the past four decades, numerous external fixator designs have evolved<sup>9,24,25,27,52,54</sup>. These designs were often based upon the principle of uniplanar ligamentotaxis by which longitudinal traction was applied to maintain skeletal length. Anderson and O'Neil<sup>3</sup> were first to report incorporation of the principle of ligamentotaxis into maintenance of fracture reduction by external fixation. In combination with pins and plaster or external fixation, a multitude of studies have demonstrated the merit of ligamentotaxis in maintaining skeletal length during fracture healing<sup>8,14,15,22,30,48,49</sup>. Although many commercial devices that utilize longitudinal traction are available, clinical<sup>5,19,33,50</sup> and cadaveric<sup>5</sup> observations have revealed that they are often unable to restore joint congruity and normal palmar tilt of the distal radial articular surface.

### Biomechanics of Multiplanar Ligamentotaxis

Inherent to the design of any external fixator that allows restoration of joint congruity and palmar tilt to a fractured distal radius is a detailed understanding of the biomechanical principles of multiplanar ligamentotaxis. Multiplanar ligamentotaxis extends the principle of uniplanar ligamentotaxis to include translation of the hand to bring the distal fragment(s) of a fractured radius into alignment. Not only does the design of the fixator allow longitudinal ligamentotaxis, it also integrates the capacity to utilize ligamentotaxis independently in the dorsal-palmar and radial-ulnar planes. This technologically advanced design, which is based on the sound biomechanical principles defined below, allows the surgeon to improve the quality of fracture reduction by permitting fracture reduction in several planes<sup>1</sup>.

### Biomechanics of Longitudinal Ligamentotaxis

A typical Colles' type fracture is depicted in Figure 1A. Note the displacement of the dorsal fragment. Application

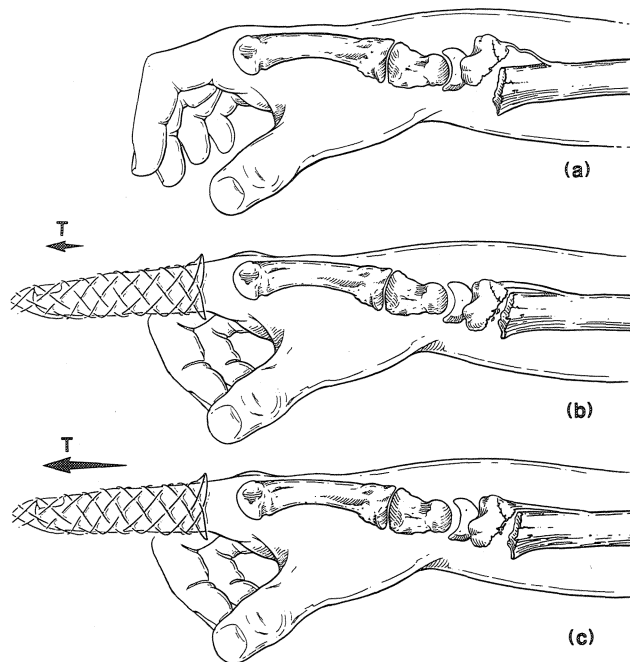


Figure 1  
A. Typical displaced Colles' type fracture. B. Restoration of skeletal length by longitudinal traction. C. Increased dorsal tilt resulting from excessive longitudinal traction. (Courtesy of Hand Biomechanics Lab, Inc., Sacramento, CA.)

of longitudinal traction, shown in Figure 1B, initially tilts the dorsal fragment palmarly and restores skeletal length. However, if excessive traction is applied (Figure 1C), dorsal tilt may actually be increased as the distal fragment pivots on the intact dorsal soft tissue hinge. The biomechanical forces responsible for the adverse effects of excessive longitudinal traction on dorsal tilt are shown in Figure 2. The dorsal tilting force on the distal fragment (M<sub>DF</sub>) is a product of the traction force (T) and a moment arm (DRP) created by the distance between the line of transmission of the force down the shaft of the radius (R) and the pivot point (P) defined by the dorsal hinge.

An external fixator using the principle of longitudinal ligamentotaxis is shown in Figure 3. Skeletal length is restored using skeletal traction with the wrist in a neutral position. The fingers extend as skeletal length is restored.

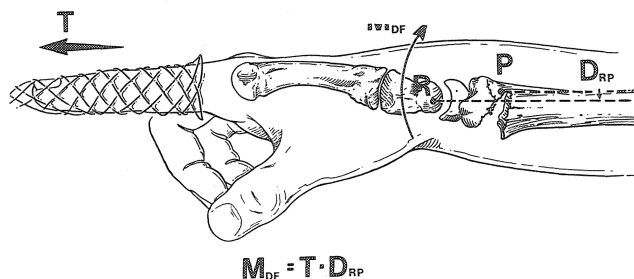


Figure 2  
Dorsal tilt of the distal fragment resulting from excessive traction. (Courtesy of Hand Biomechanics Lab, Inc., Sacramento, CA)

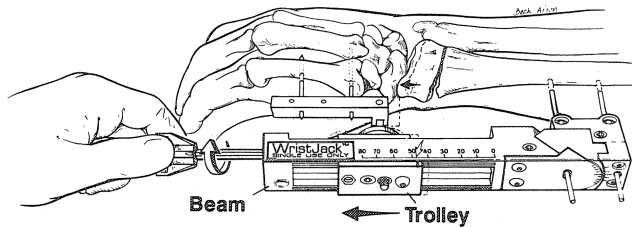


Figure 3

External fixator restoration of skeletal length using the principle of longitudinal ligamentotaxis. (From Agee, J.M.: Agee-Wrist Jack Surgeon's Manual. Sacramento, CA. Hand Biomechanics Lab, Inc., 1993, p.25; with permission.)

This extension reflects the wrist distraction and increased carpal height required for ligamentotaxis to pull the fragments into position. Metacarpophalangeal (MP) joint extension and extrinsic finger extensor tendon tightness are useful indicators of skeletal length. Excessive distraction force applied in increments is accompanied by concomitant degrees of finger MP joint extension. Progressive separation of the carpal bones with each degree of extension can be demonstrated radiographically.

### Biomechanics of Dorsal-Palmar Ligamentotaxis

Clinically, longitudinal traction is combined with thumb pressure and wrist flexion to restore palmar tilt. However, it is only when the dorsal ligaments tighten, as the limits of wrist flexion are approached, that palmar tilt will be effectively restored. Persistent advanced wrist flexion is necessary to maintain palmar tilt, but this position cannot be used safely during fracture healing due to subsequent adverse effects on hand function<sup>4,10,23</sup>.

Two types of ligamentotaxis used for reduction of distal fractures are shown in Figure 4. Longitudinal traction, as discussed above, restores skeletal length. Palmar translation of the hand on the forearm produces forces that act to sublux the midcarpal joint, creating a force that is transmitted through the proximal carpal row to the distal radial fragment, which tilts its articular surface palmarly. As forces are transmitted from the third metacarpal and capitate distally to the lunate and distal radius proximally, a volar intercalated segmental deformity of the carpus is created that is not unlike the volar wrist collapse described by Linscheid et al<sup>32</sup>. The capitate impinges on the lip of the lunate to create the rotatory force necessary to tilt the lunate and distal radial fragments palmarly. The mechanical link for the ligamentotaxis that restores palmar tilt is provided by the capsular ligaments common to the carpus and distal radius. In the operating room, reduction is achieved as follows. While the wrist is held in neutral position and longitudinal traction is used to maintain skeletal length, a translating force is used to displace the hand palmarly. Palmar translation rotates the distal frag-

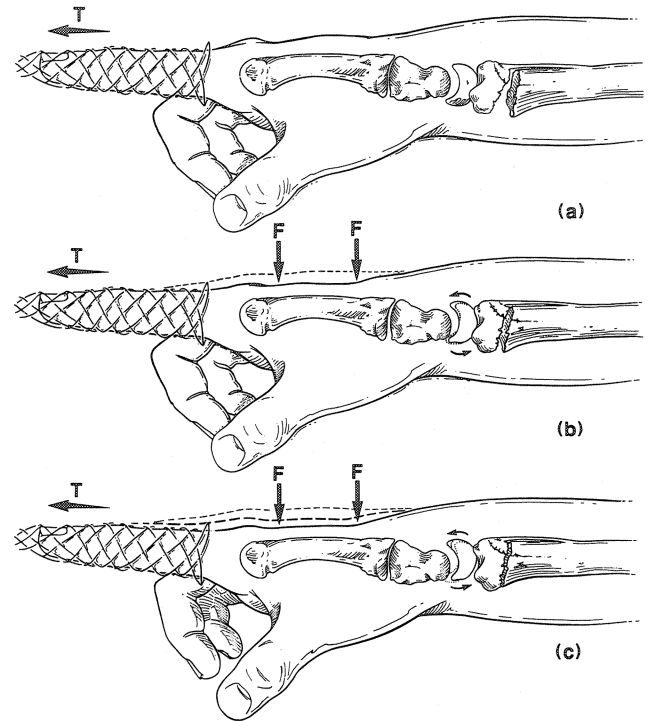


Figure 4 A.

Biomechanics of ligamentotaxis obtained by longitudinal traction to restore skeletal length.

Figure 4 B,C.

A palmar translating force (F) tilts the distal radius articular surface palmarly. (Courtesy of Hand Biomechanics Lab, Inc., Sacramento, CA.)

ment and palmar tilt is restored as the tightened dorsal periosteal hinge impinges on the shaft fragment. Use of excessive force to restore palmar tilt must be avoided as it can tear the soft tissue hinge and convert the Colles' fracture into a palmarly displaced Smith's fracture<sup>50</sup>.

After restoration of skeletal length following fracture of the distal radius, an adjustment is made to the multiplanar external fixator to decrease any excessive longitudinal traction. This adjustment prevents a dorsal tilting moment on the distal fragment that would prevent palmar translation of the hand from restoring palmar tilt to the distal fragment (Figure 5). A worm gear adjustment on the fixator carries the hand, wrist and distal radius fragment through an arc centered on one of the radial shaft pins. Distracting forces combine with an intact dorsal periosteum to tilt and displace the distal fragment(s) dorsally (Figure 5A). The fragment(s) aligns dorsally as the periosteum is reaposed (Figure 5B), and additional palmar translation rotates the distal fragment(s) and tilts its articular surface palmarly (Figure 5C).

### Biomechanics of Radial-Ulnar Ligamentotaxis

The hand can also be translated on the forearm in a third geometric plane; i.e., in a radioulnar plane. With ulnar translation, the first and second extensor compartment

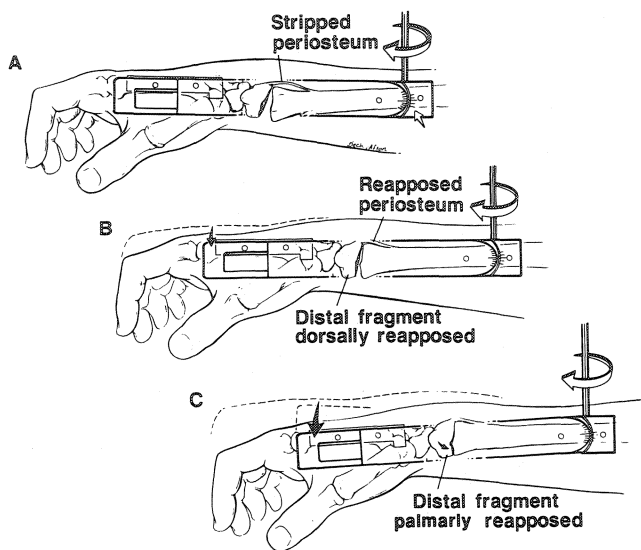


Figure 5

External fixator restoration of palmar tilt using the principle of dorsal-palmar ligamentotaxis (From Agee, J.M.: Agee-WristJack Surgeon's Manual. Sacramento, CA. Hand Biomechanical Lab, Inc., 1993, p. 29; with permission.)

tendons and their associated retinaculum create a radial soft tissue hinge. This forms the basis of the mechanism for the ligamentotaxis of ulnar translation to restore ulnar tilt.

If the fracture is malaligned in the radial-ulnar plane, further selective fracture reduction is accomplished using the radial-ulnar gear adjustment of the external fixator as ligamentotaxis is used to obtain optimal alignment of the distal fragment. Although this is an infrequently used adjustment of the fixator, it can be used to improve appositional alignment of the fracture fragments when the fractures extend to involve the distal radioulnar joint.

### Excessive Distraction and Wrist Position During Fracture Healing

An understanding of the biomechanics of the hand related to distraction forces and wrist position during fracture healing is absolutely essential to prevent the external fixator-induced complication known as claw hand.

External skeletal fixators for distal radius fractures are most frequently applied with pins inserted proximally into the radial shaft and distally into the index and long finger metacarpals<sup>2,3,11,15,44,47</sup>. The applied traction forces pass dorsal to the axis of rotation of the wrist, producing fixator-induced wrist flexion and reciprocal MP joint extension. When excessive distraction is applied and maintained during fracture healing, the carpal bones remain distracted and there is increased skeletal length (Figure 6). The increased length transmits tension forces into the extrinsic digital muscles, producing clinically evident clawing of the fingers. Thus the "claw hand" produced by



Figure 6

Excessive distraction induced by an external fixator leads to clinically evident clawing of the fingers. (Courtesy of Hand Biomechanics Lab, Inc., Sacramento, CA.)

external fixation results from fixator-induced wrist flexion and excessive distraction.

Research on the biomechanics of external fixation led to the design of a fixator<sup>1</sup> that permits wrist flexion and extension about an axis that projects through the center of rotation of the wrist<sup>56</sup>. When the wrist is in extension, tension on the finger extensor tendons is relatively relaxed so as to prevent contractures of MP joint extension. MP joint flexion facilitates active finger range-of-motion exercises and hand use during fracture healing. This fixator, that has evolved from the basic biomechanical studies, permits the wrist to be flexed and extended without loss of fracture reduction. These positions can be independently adjusted to restore palmar tilt and to select a "wrist-extended" position to avoid hand stiffness.

### DISCUSSION

Colles' fractures are frequently treated inadequately<sup>4,13,14</sup>. Complications that occur during and following treatment can be osteoarticular<sup>14</sup>, associated with the soft tissues<sup>14,45</sup> or both<sup>14</sup>. As discussed by Semel<sup>57</sup>, an increased complication rate is correlated with inadequate reduction.

Charnley<sup>10</sup> emphasized that the classical method of treating Colles' fractures with plaster violates two basic principles of fracture treatment: 1) the plaster slab is not mechanically sound, and 2) the position of flexion of the

wrist is not optimal for function of the hand. Gupta<sup>23</sup> found that for distal radius fracture patients treated with plaster, the best functional results, including the least finger stiffness, occurred in those patients with their wrists immobilized in an extended rather than a neutral or flexed position.

External fixation of a fractured distal radius allows the surgeon to place the wrist in an extended position during healing while maintaining reduction. In a study of 20 Frykman Type V to VIII fractures<sup>1</sup>, a "wrist-extended" position (average, 8.4 degrees) was used without any loss of reduction when an external fixator designed to maintain only longitudinal ligamentotaxis was used. Subsequent to this study, the design of the fixator was enhanced to create a multiplanar fracture reduction system. This design extended the principle of ligamentotaxis obtained by traction in one plane<sup>3,5</sup> to ligamentotaxis in two additional planes in which both appositional and rotational (tilting) alignment in dorsal-palmar and radial-ulnar planes are achieved. Beckenbaugh et al.<sup>6</sup> used this new system to treat 20 Colles' fracture patients. They found that the average postreduction measurement of palmar tilt was 5.4 degrees and ulnar inclination was 22.6 degrees. They reported that in addition to maintaining fracture reduction, the fixator accurately restored both radial length and palmar tilt to the distal radius with only minimal or no wrist distraction. Their study using a fracture reduction system based upon the principle of multiplanar ligamentotaxis has provided the best clinical evidence to date of the value of palmar translation to restore palmar tilt. The ability of this system to restore palmar tilt in laboratory-created fractures has also been reported<sup>36</sup>.

The best functional results following fracture of a distal radius are obtained when the radius is restored to its original anatomy<sup>12,15,18,22,26,42,49</sup>. The quality of fracture reduction is measured objectively by palmar tilt, radial length, ulnar inclination (radial tilt), radial shift and dorsal shift<sup>51</sup>. The multiplanar fracture reduction system allows the surgeon not only to perform the initial fracture reduction and fragment alignment, but it permits additional adjustments to be made early during postoperative care if follow-up radiographs indicate the necessity.

A similar approach to the design of external fixators has been reported by others<sup>43,44</sup>. By using multiplanar ligamentotaxis external fixators the need for immobilization of adjacent joints is eliminated, allowing for early mobilization and an earlier return to function. The basic purpose of external fixation is to maintain distraction forces during fracture healing. Fixators that maintain excessive traction produce delayed fracture healing and clawing of the fingers with associated hand stiffness. Although initial overdistrac-tion of the fracture may be necessary to disimpact and align the fragments, continued use of excessive distraction

is detrimental to the functional outcome<sup>28</sup>. The clinically recognizable clawing of the hand, an "intrinsic minus position," is actually an "extrinsic extensor plus" position of the fingers. The degree of this deforming force can be estimated by passively flexing the finger tips to the distal palmar crease, with the index finger being the most sensitive indicator of this biomechanical imbalance. While some distraction is required for an external fixator to maintain skeletal length, the detrimental effects can be lessened by placing the wrist in a neutral or extended position. The lowest carpal tunnel pressures occur with the wrist in a neutral position<sup>20,21</sup>. Based upon this finding, it seems that placement of the wrist in extension in excess of 20 degrees may improve hand tendon mechanics and decrease the risk of finger stiffness at the risk of increased carpal tunnel pressures on the median nerve.

Although the treatment of Colles' fractures has been significantly improved through application of multiplanar ligamentotaxis via dorsal-palmar and radial-ulnar translation, severe fragment displacement and the absence of soft tissue attachment often require additional treatment. When displaced fragments cannot be reduced with external fixation alone<sup>26,30</sup>, external fixation with percutaneous pin manipulation of key fragments or open reduction and internal fixation may be the option of choice. Bone grafting may be of benefit when voids created by crushed metaphyseal bone are evident<sup>26,34</sup>. In those injuries that extend to involve the distal radioulnar joint, a long arm splint may be of benefit if forearm supination improves reduction and stability of that joint.

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# POST-BURN HETEROTOPIC OSSIFICATION AT THE ELBOW

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## INTRODUCTION

In a prospective radiographic study, heterotopic ossification around joints has been reported to occur in up to 23% of patients after significant burns<sup>10</sup>. Retrospective studies generally report a much lower incidence of 0.38% to 3.3%<sup>2,4,6</sup>. The elbow is the most frequently involved joint in most series<sup>4,6</sup>.

The etiology of this heterotopic bone formation is not clear. It tends to be related more to the severity of a burn than to its extent or location<sup>4,5,7,10</sup>. Munster reported that 22% of patients with third-degree burns demonstrated X-ray evidence of heterotopic ossification versus 2.4% of those with second-degree burns<sup>7</sup>. The periarticular tissues do not have to be directly involved by the burn. When the burn does involve the periarticular tissues, it is usually confined to the skin without direct injury to the deep tissues where the bone formation takes place. Histologic studies of the deep tissue beneath burns has demonstrated chronic inflammation<sup>4</sup>.

Cases in which post-burn heterotopic ossification occurs without direct involvement of overlying tissues lend support to the theory that humeral factors play a role. Metabolic phenomena have also been considered; however, serum levels of calcium, phosphate, and alkaline phosphatase are not predictive<sup>1,4,7</sup>. Trauma to the joint from the initial injury or possibly during the patient's rehabilitation, has also been implicated but never proven<sup>1</sup>. Other factors implicated include tissue hypoxia, infection, circulatory stasis, increased protein intake with calciuria and antibody reactions<sup>9</sup>. Immobilization of the elbow and the amount of time at bed rest have not been shown to be statistically significant factors<sup>7</sup>. Evans felt people with burn ankylosis were physically or psychologically resistant to physical therapy<sup>4</sup>. This, however, has been disputed by Munster<sup>7</sup>.

Radiographically, the formation of bone is first observed within 12 to 20 weeks<sup>10</sup>. Positive bone scans may precede the plain X-ray changes by three weeks<sup>9</sup>. The quantity and location of the bone is variable. Several patterns of bony deposition have been described<sup>6</sup>.

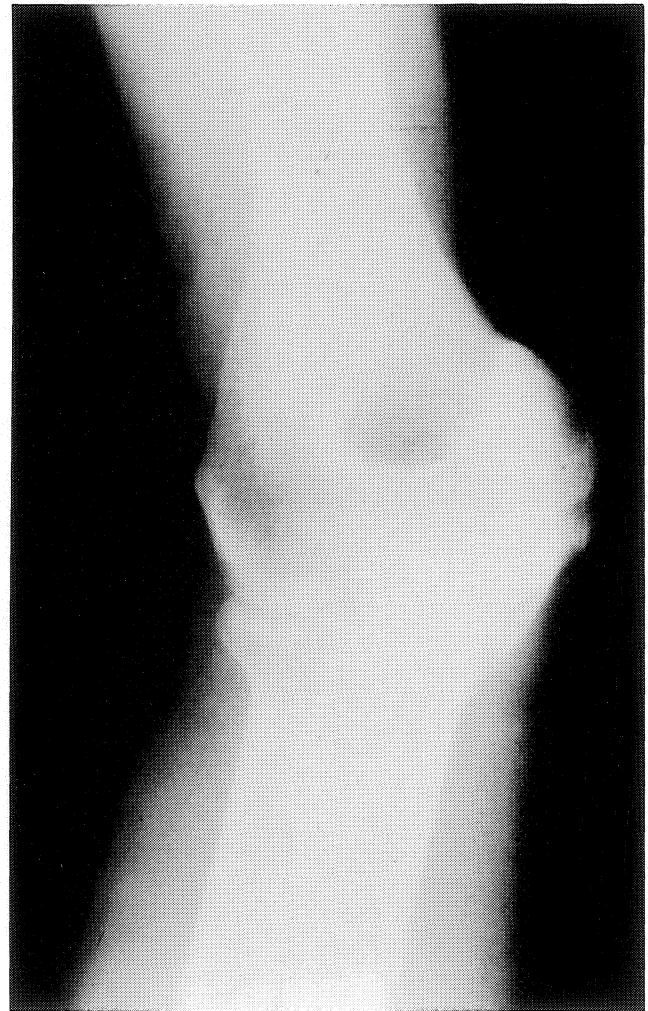


Figure 1A

AP radiograph of elbow showing mature heterotopic bone.

1. **Linear Ossification:** Ossification in the plane of the ligaments or tendons that is not connected to the bone<sup>3</sup>.
2. **Periosteal New Bone:** This is found directly under burned areas and resolves after the skin is healed.

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Figure 1B

Lateral radiograph of elbow showing mature heterotopic bone.

**3. Limited Exostosis or Osteophytes:** This bone forms from the joint margins and may bridge the joints.

**4. Extensive Calcification and Ossification:** This may lead to bony ankylosis and become a significant functional problem.

The extensive ossification lies deep to the muscles, but does not replace them. This bone may completely bridge the joint, but spares the joint surfaces in adults<sup>3</sup>. In children extensive intra-articular ossification and joint destruction may be observed; however, this is considered to have a bacterial etiology<sup>3,4</sup>.

Bone deposition and ankylosis of the elbow occur in a predictable pattern. Ossification typically begins deep to the triceps tendon adjacent to the medial condyle and humeral shaft and extends distally and medially (Figure 1A and 1B). The ulnar nerve is commonly encased in the cubital tunnel (Figure 2). The radial head is seldom [radiographically or clinically] involved<sup>7</sup>. The bone initially has an amorphous or cloudy appearance on radiographs. The limits of the bony margins then become well delin-

eated and eventually mature cortical margins and cancellous bone become apparent<sup>4</sup>.

Physical and occupational therapy are used extensively in the rehabilitation of the burned patient. Splinting with gentle active and active-assisted range of motion are employed to help maintain and reestablish motion of the extremities. The onset of pain or stiffness in the elbow may be the first warning of impending heterotopic ossification. In this situation, it is imperative to continue gentle active range of motion exercises. As the active phase of bone formation ceases, pain should decrease as well. When radiographs demonstrate mature bone, intensive physical therapy should resume. Some patients may improve their range of motion with physical therapy and bone may actually resorb if it does not completely bridge the joint. This phenomenon is particularly remarkable in children.

Patients who have failed physical therapy and still have restricted motion at the elbow may be candidates for surgical resection. For optimal results patients should



Figure 2

Cubital tunnel radiograph demonstrating bony encasement of the cubital tunnel.

have functionally limited range of motion, radiographically mature heterotopic ossification, well-healed skin and no signs of inflammation<sup>2,3,9</sup>.

### MATERIALS AND METHODS

Four patients (six elbows) with an average age of 41.5 years (range 25 to 64 years old) at the time of their burn were operated on at an average of 8.3 months after their burn. Information was obtained from chart review, radiographs, clinical examination, and telephone interviews. Two left elbows and four right elbows were involved. Each patient had third-degree burns and fulfilled the previously listed criteria for surgery. Four of the six elbows were completely ankylosed, while one patient had 10° of active motion and another had 30°. In patients with bilateral involvement, the second elbow was approached two to four months following the release of the first. All patients had varying degrees of ulnar dysesthesias preoperatively. EMG and nerve conduction studies were not performed routinely. The average follow-up for these patients was 79 months with a range of 27 to 126 months.

### SURGICAL TECHNIQUE

The elbows were approached posteromedially in five of the six patients, while one patient (S.R.) underwent excision through a posterior approach. The triceps tendon was reflected sharply for exposure of the medial humeral condyle and the posterior aspect of the elbow. After the extent of the heterotopic bone was clearly delineated, osteotomes and ronguers were used to remove the heterotopic bone. The ulnar nerve in the cubital tunnel was encased in bone in every case and a subcutaneous anterior ulnar nerve transposition was performed in each patient. The radial head and lateral aspect of the elbow did not require exposure. Patients were splinted until the second post-operative day when therapy for active range of motion of the elbow was initiated. Continuous passive motion was not used in this group of patients. Patients averaged nine days in the hospital (range five to seventeen). One extensive seventeen day stay was due to a partial wound breakdown. Patients were then followed as out-patients by the hand therapists two to three times per week depending on their progress.

### RESULTS

The results of our four patients are shown in Table 1. Range of motion was recorded preoperatively, at the time of surgery and at clinical intervals. The average active range of motion improved from six degrees to over ninety degrees postoperatively. Range of motion improved most significantly during the first six months then plateaued. All elbows obtained at least 100° of active motion with the average of 121°. Cubital tunnel symptoms improved in all patients. Supination and pronation were not problems in these patients.

### COMPLICATIONS

A partial wound dehiscence following a posterior approach in patient S.R. occurred early in the series. The wound responded to dressing changes and healed. There were no wound problems in patients who underwent posteromedial incisions. Patient R.B. requested reoperation on his left elbow after initially achieving a range of motion of 100 degrees (35°-135°). Due to his avid interest in archery, he desired to obtain terminal extension to allow him to use a bow. The second operation improved his extension to -5 degrees and enabled him to use a bow.

### DISCUSSION

A review of previously reported surgical results of post-burn ankylosis has revealed an average total arc of motion from 73.6 to 110 degrees<sup>5,10</sup>. In agreement with our study, the published series of Hoffer and Peterson demonstrated continued improvement in their patients four

TABLE 1

PATIENT	ARM	AGE AT BURN	TIME OF OP AFTER BURN	PREOP MOTION	FINAL ROM	FOLLOW-UP	COMPLICATIONS
R.B.	L	33 yrs.	5 mo.	ankylosed at 35°	35-135°	10 yr. 6 mo.	None
			14 mo.*	35-135°	5-140°		
	R	33 yrs.	9 mo.	ankylosed at 50°	5-140°	10 yr. 2 mo.	None
D.L.	R	25 yrs.	10 mo.	70-80°	25-140°	2 yr. 3 mo.	None
S.R.	L	64 yrs.	6 mo.	ankylosed at 45°	40-145°	8 yr. 7 mo.	Wound dehiscence
			8 mo.	ankylosed at 45°	30-130°		
	R	64 yrs.	8 mo.	ankylosed at 45°	30-130°	8 yr. 5 mo.	None
K.D.	R	30 yrs.	12 mo.	65-95°	10-145°	2 yr. 8 mo.	None

\*Patient elected to attempt second operation to gain terminal extension to allow him to use a bow and arrow.

to six months postoperatively. During their follow-up of fifty-two months, no significant recurrence occurred. Our study population is small; therefore, we are unable to make any comments regarding patient age and outcome. Patient S.R., however, at age 64, obtained 102 degrees compared to the average of 130 degrees in the younger group of patients who averaged 27 years of age.

The literature points out the importance of operating only when the heterotopic ossification is mature<sup>2,3</sup>. Radiographs are a reasonable method to determine this. Bone scans may be helpful in the early diagnosis of heterotopic ossification, but like alkaline phosphatase and calcium levels, bone scans have not been shown to be helpful in judging maturity<sup>1,4,5</sup>. We concur with the importance of waiting until the skin has completely healed and matured. These two points appear to be important in preventing recurrence.

Peters reported good results for a patient with a positive bone scan who underwent excision of heterotopic bone at the elbow. The procedure was felt to be necessary due to a progressive ulnar nerve deficit. The patient did receive Didronel (Norwich Eaton, Norwich, New York); however, the efficacy of this drug has not been proven.

The immediate postoperative range of motion seems to be a reasonable indication of final range of motion. Every effort should be exhausted to obtain full range of motion at the time of surgery. The postoperative course is difficult, and requires close follow-up and supervised occupational therapy or physical therapy. We are pleased with our results of excision in this small series, and are optimistic that we can improve the function of patients' upper extremities following severe burns that result in bony ankylosis of the elbow due to heterotopic ossification.

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# CHRONIC PERILUNATE FRACTURE DISLOCATIONS AND PRIMARY PROXIMAL ROW CARPECTOMY

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Perilunate dislocations and fracture-dislocations are rare injuries that result from high-energy trauma to the wrist. A variety of injury patterns have been described and their mechanisms delineated<sup>1,2,3,4</sup>. Fracture-dislocations are more common than pure dislocations and dorsal injuries occur more frequently than volar. The dorsal trans-scaphoid fracture-dislocation represents the most common perilunate injury overall<sup>2,3</sup>. The particular injury pattern encountered is dependent on several factors, including the direction and magnitude of the deforming forces, the integrity of the involved ligaments, and anatomy of the carpal bones.

Perilunate fracture-dislocations can be complex and challenging. The management of such injuries remains controversial. Recent studies have confirmed that early open reduction and internal fixation with restoration of all osseous and ligamentous damage provides the best functional outcome<sup>2</sup>. However, despite continued emphasis on the recognition and management of perilunate injuries, patients occasionally present undiagnosed, resulting in a chronic form of injury.

Patients who present within six weeks of injury have been treated in a manner similar to that of acute injuries<sup>2,3,4</sup>. For those injuries that are more than six weeks old, open reduction and internal fixation as well as alternative treatment methods such as proximal row carpectomy, intercarpal or radiocarpal arthrodesis have been proposed<sup>4-9</sup>. This report describes the course of two patients who presented late to our institution with perilunate fracture-dislocations which were treated by primary proximal row carpectomy.

## CASE REPORTS

### Case 1.

A 45-year-old right hand dominant male laborer fell on his outstretched right wrist on September 14, 1977.

Radiographs were obtained and interpreted as normal. The patient was placed in a volar splint for four weeks and repeat radiographs were obtained. A perilunate injury was subsequently recognized and a short-arm cast was applied

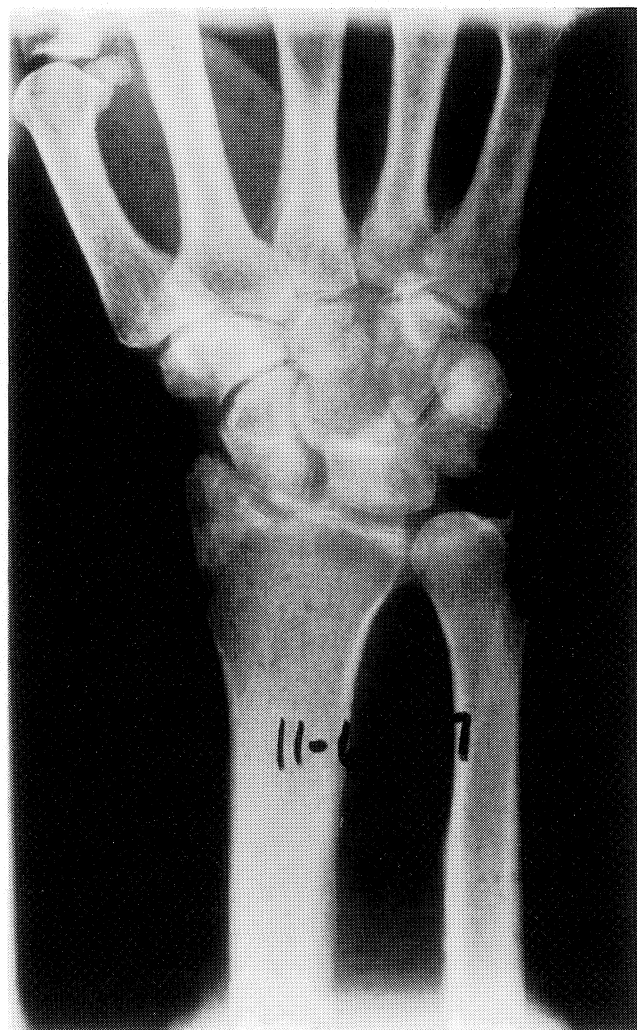
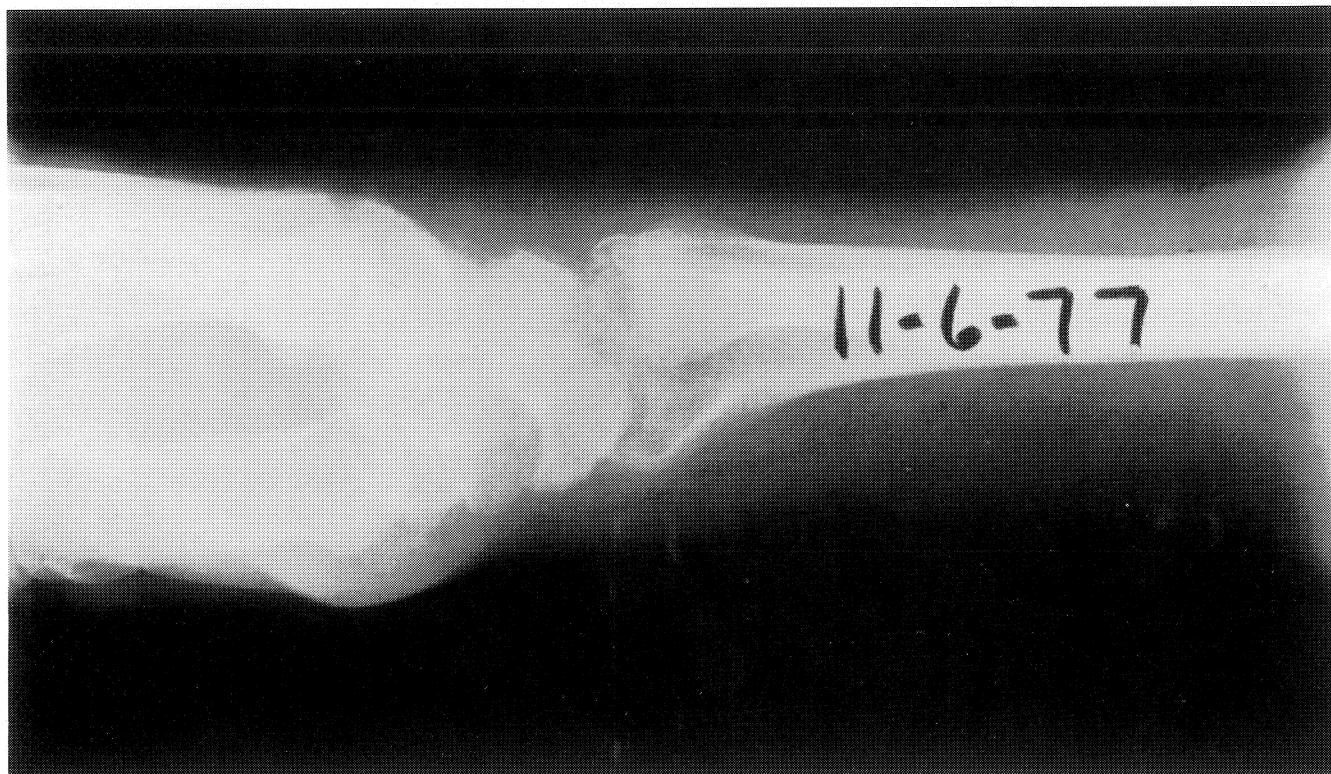


Figure 1A

AP roentgenogram demonstrating a trans-radial styloid perilunate fracture dislocation. Note settling of scaphoid at the fracture site and the incongruity of the midcarpal arc.

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**Figure 1B**

Lateral view revealing the dorsally dislocated capitate "perched" on the lunate.

without any attempts at reduction. He was referred to the hand clinic approximately eight weeks post-injury. Physical examination revealed tenderness of the right wrist with a palpable dorsal wrist prominence and limited, painful range of motion. There was moderate thenar atrophy and hypesthesia of the thumb and index finger. Roentgenograms demonstrated an unreduced trans-radial styloid perilunate fracture-dislocation (Figs. 1A and 1B). He was admitted to the hospital, and the following day underwent surgery through a combined dorsal and volar approach. The transverse carpal ligament was released and the median nerve was found to be hyperemic and contused. A dorsal incision was subsequently utilized and scar tissue around the dorsally dislocated capitate was excised. The scapholunate ligament was ruptured and the scaphoid was malrotated and volarly subluxed. Similarly, the lunotriquetral ligament was disrupted and the interval filled with scar tissue. There was no evidence of injury to the articular surface of the proximal capitate or lunate fossa of the distal radius. The radial styloid fracture extended into the scaphoid fossa, and was associated with a 3 x 5 mm area of articular depression. An unsuccessful reduction of the dislocation was attempted. A proximal row carpectomy was subsequently performed.

Postoperatively, the patient was immobilized in a long-arm thumb spica cast. Four weeks later the radial styloid

fracture was noted to be healed and occupational therapy was begun. At three months post-injury, range of motion of the right wrist was 25° dorsiflexion, 20° palmar flexion, 5° radial deviation, and 15° ulnar deviation. Sensibility testing revealed normal two-point discrimination in all digits. The patient denied pain and was able to return to work in a paper mill. At nine years post-injury, the patient noted intermittent discomfort of the wrist, but rated his surgical result as excellent. Range of motion was 50° dorsiflexion, 30° palmar flexion, 15° radial deviation, and 30° ulnar deviation. Grip strength was 105 pounds on the right and 135 pounds on the left. Radiographs revealed subchondral cysts beneath the scaphoid fossa of the distal radius (Figs. 2A and 2B).

The patient returned on request sixteen years post-injury. He continued to be employed at the paper mill, and denied pain in his wrist or functional limitations. Range of motion of his right wrist was 40° dorsiflexion, 30° palmar flexion, 5° radial deviation, and 10° ulnar deviation. Grip strength was 90 lbs on the right and 110 lbs on the left. Radiographs (Figs. 3A and 3B) revealed the proximal capitate to be in good position in the lunate fossa with a dorsal radial osteophyte, a remnant of the distal scaphoid, and persistent cystic changes in the scaphoid fossa of the distal radius.

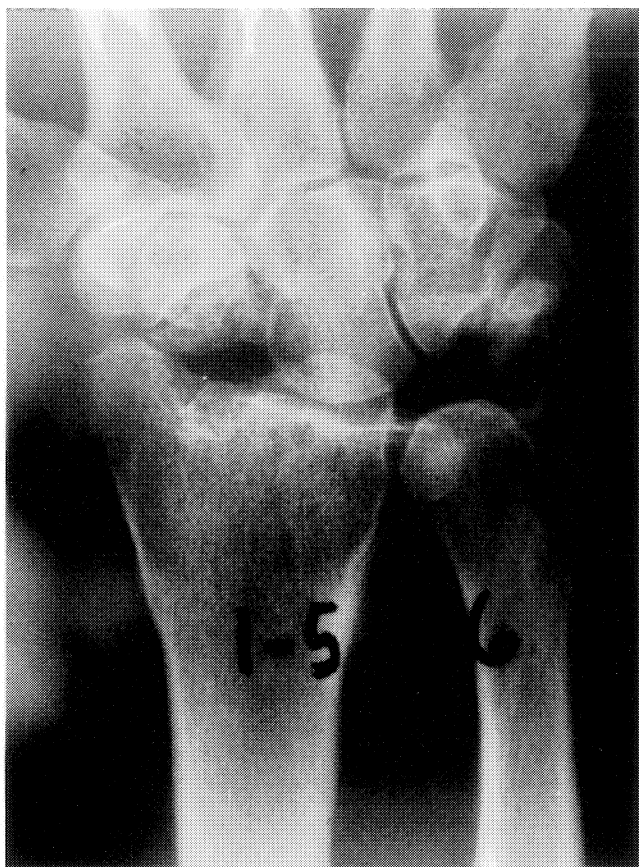


Figure 2A

Nine years post-surgery. Note articular defect at previous radial styloid fracture site.

### Case 2.

A 32-year-old right hand dominant male was involved in a motor vehicle accident. He sustained an isolated left wrist injury, and presented to a local emergency room where radiographs were obtained. The patient was diagnosed with a scaphoid fracture, splinted, and instructed to follow-up with an orthopaedic surgeon. The patient was subsequently imprisoned and was therefore not evaluated until 11 weeks post-injury. Upon presentation to the hand clinic, the splint was removed and the wrist examined. There was moderate tenderness and painful, limited range of motion of the wrist. The neurovascular examination was unremarkable. Roentgenograms demonstrated an unreduced dorsal trans-scaphoid perilunate fracture dislocation (Figs. 4A and 4B). The patient subsequently underwent a primary proximal row carpectomy through a dorsal midline incision. Significant synovitis and scar tissue were present throughout the radiocarpal and midcarpal articulations. The scapholunate ligament remained intact, but the lunotriquetral ligament was clearly disrupted and the interval filled with prominent scar and synovial tissue. The scaphoid fracture was examined and the fragments excised along with the lunate and triquetrum. The lunate fossa of

the distal radial articular surface was found to have no chondral damage. The proximal articular surface of the capitate was noted to have a 5 mm stellate-shaped chondral fracture and a 2 x 6 mm linear chondral defect with exposed subchondral bone. The scaphoid fracture was examined and the fragments excised along with the lunate and triquetrum. The capitate was positioned in the lunate fossa and the dorsal capsular rent repaired. The hand was immobilized in a short-arm cast for four weeks and then occupational therapy was begun. Shortly thereafter, the patient was released from prison and failed to return for therapy and follow-up.

### DISCUSSION

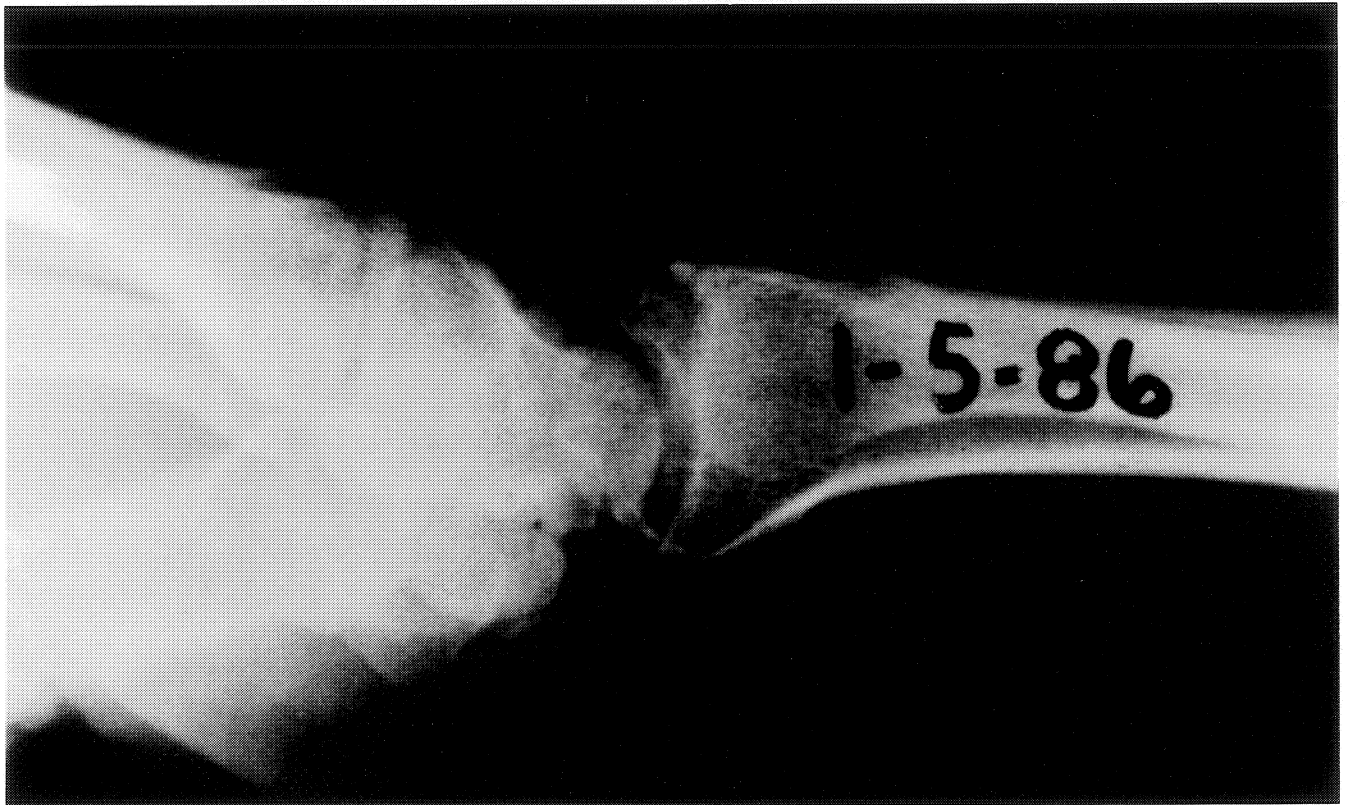
Chronic perilunate fracture-dislocations are defined as those that remain unreduced for a minimum of six weeks<sup>5</sup>. If left untreated, these injuries can result in post-traumatic arthritis, incapacitating wrist pain, diminished grip strength, limited wrist range of motion, carpal tunnel syndrome, and attritional flexor tendon rupture<sup>2,10</sup>. These sequelae of neglected perilunate injuries have led to the recommendation of surgical intervention for *all* late diagnosed injuries, regardless of symptoms<sup>2</sup>. However, the optimal management remains controversial and current recommendations are based upon studies that involve small series of patients with limited follow-up. As a result, numerous procedures with varying outcomes have been advocated.

Russell<sup>11</sup> and Wagner<sup>12</sup> recommended wrist fusion for neglected perilunate injuries. Wagner believed that arthrodesis was necessary because the extensive dissection needed for reduction invariably produced an arthritic wrist.

Campbell advised primary proximal row carpectomy<sup>7</sup>. Neviasser reported his results with such treatment in ten patients with untreated or incompletely reduced trans-scaphoid perilunate fracture-dislocations<sup>6</sup>. Significant improvement in pain, range of motion, and grip strength were found.

Fisk<sup>13</sup>, Green and O'Brien<sup>4</sup>, and Siegert et al.<sup>5</sup> have all recommended that open reduction and internal fixation (ORIF) be attempted regardless of delay. Anecdotal reports of the success ORIF in injuries up to 15 weeks old have been described<sup>8,9</sup>. In addition, Fernandez has described the combined use of ORIF and external fixation for both the reduction and immobilization of neglected perilunate injuries<sup>14</sup>. However, a recent large multicenter study described the unsatisfactory outcome of patients who underwent late ORIF<sup>2</sup>.

Because of the morbidity of untreated perilunate injuries, particularly fracture-dislocations, we agree with Herzberg et al.<sup>2</sup> that some form of surgical intervention should be encouraged. The prolonged immobilization necessary after extensive ligamentous and osseous repair;



**Figure 2B**

**Lateral roentgenogram demonstrates seating of the capitate in the lunate fossa of the distal radius.**

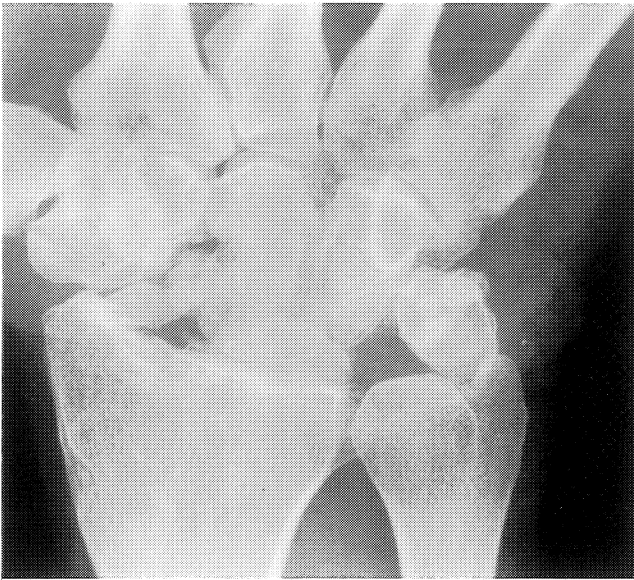
the risk of carpal avascular changes, the potential for late carpal instability, the high incidence of post-traumatic arthritis and the risk of pin tract infection with late ORIF, with or without external fixation, have led us to prefer primary proximal row carpectomy for chronic, neglected perilunate injuries. We have found proximal row carpectomy to be a durable, motion-preserving procedure for various wrist problems, including perilunar instability. However, we acknowledge that wrist arthrodesis may be necessary in those wrists with an eroded or badly damaged proximal capitate and/or lunate fossa, which may contraindicate proximal row carpectomy. Our second patient had a small chondral defect on the proximal capitate, but we did not feel that the size of osteochondral defect warranted arthrodesis. Proximal row carpectomy was performed in an attempt to preserve motion with the understanding that arthrodesis may be necessary if the patient developed symptomatic radiocapitate degenerative joint disease. The follow-up of this patient was too short to determine the long-term results of such rationale.

The long-term follow-up of our first patient was encouraging. His post-op course demonstrated the durability of the benefits of proximal row carpectomy.

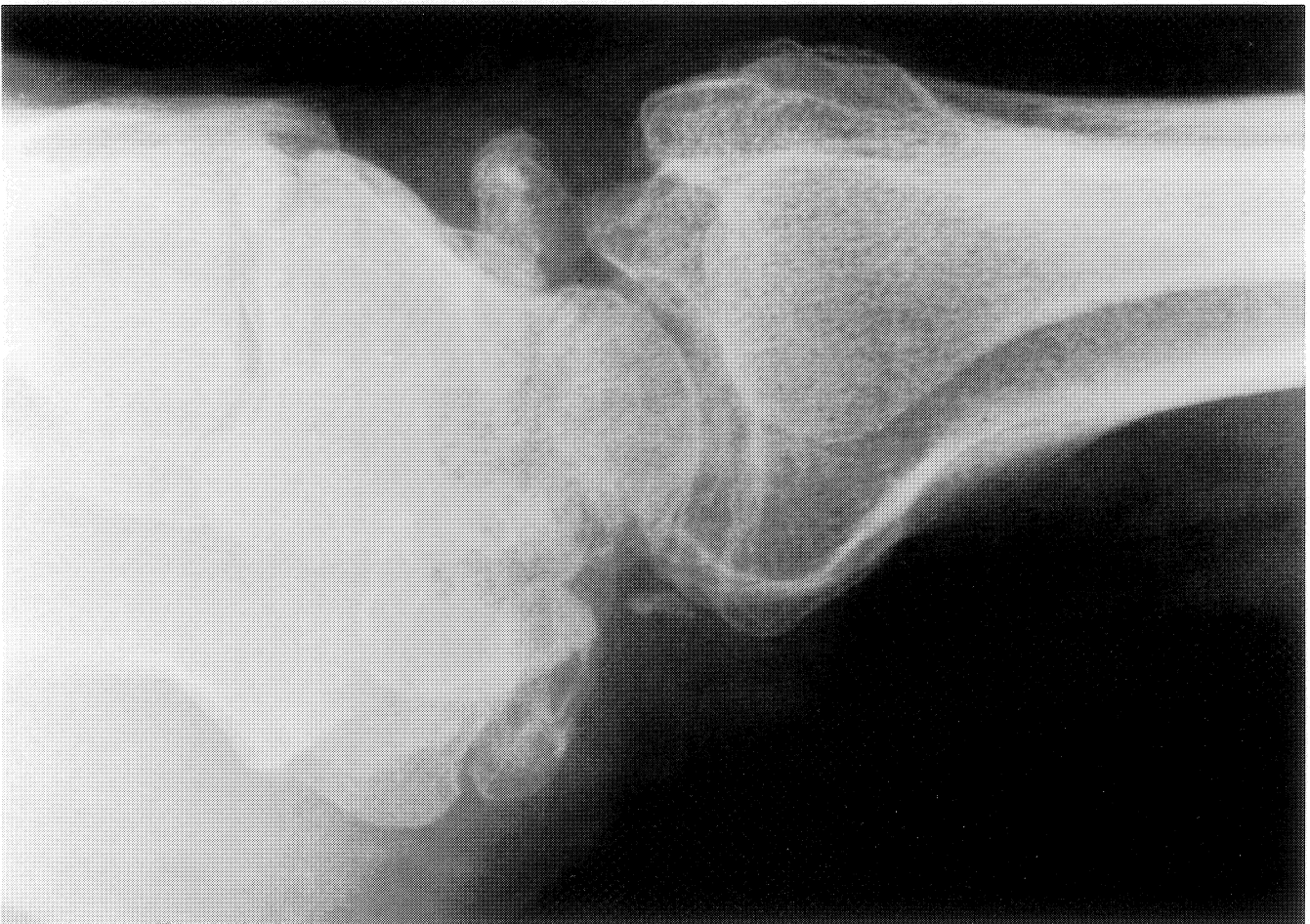
The surgical management of neglected perilunate injuries is challenging. Many surgical procedures have been

recommended, but the optimal management remains controversial. We believe that the selection of the appropriate procedure should be based upon the injury pattern, patient and surgeon expectations, and the intra-operative findings. When possible, we prefer to perform primary proximal row carpectomy because of the predictable pain relief, preservation of wrist motion, and the durability of the results.





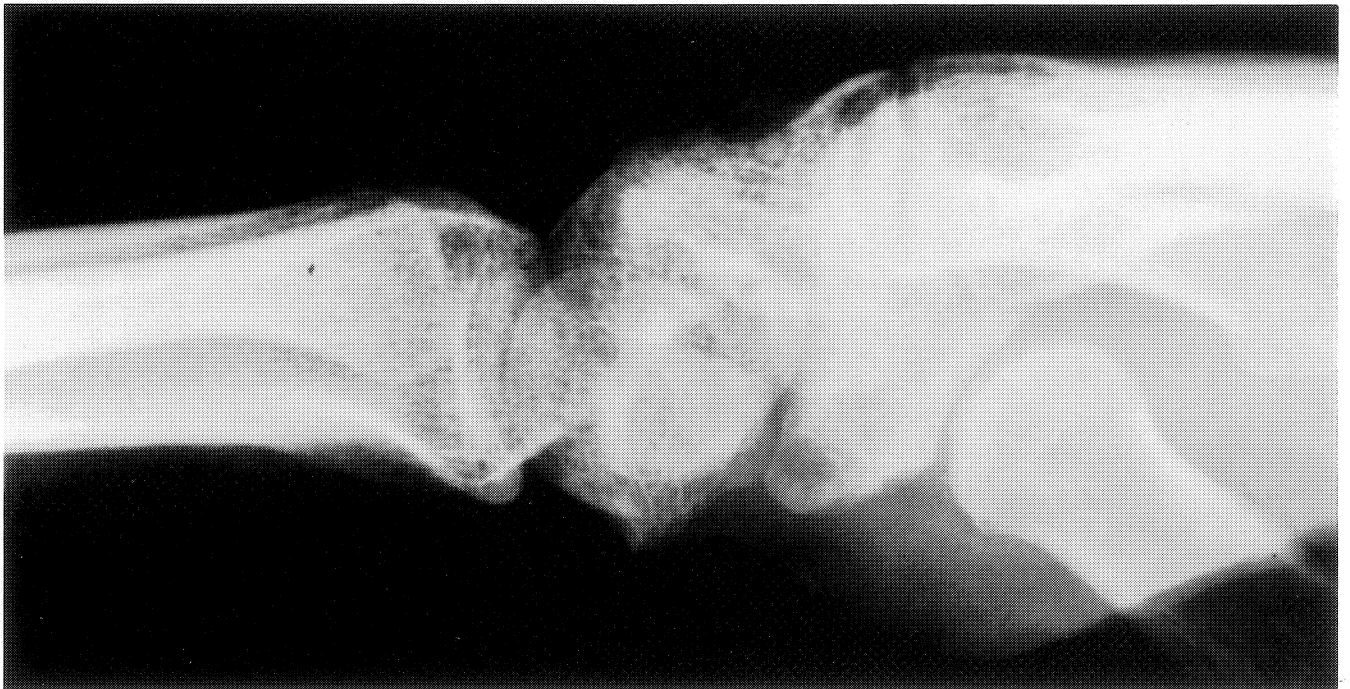
**Figure 3A**  
PA radiograph at sixteen years post-surgery. Capitate is seated in the lunate fossa and appears to lack degenerative changes. A remnant of the distal scaphoid is seen near the radial styloid.



**Figure 3B**  
In the lateral view a dorsal rim osteophyte can be seen above the proximal pole which appears to be seated well within the lunate fossa.



**Figure 4A**  
A scaphoid fracture and overlapping carpus at the capitolunate articulation are seen indicative of a trans-scaphoid perilunate injury.



**Figure 4B**  
The scaphoid fracture is obscured in the lateral view which clearly demonstrates the dorsal midcarpal dislocation.

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# THE "DON'T OPERATE ON ME" SIGN

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## INTRODUCTION

The presence of a bony prominence and limited elbow motion in a child does not always point to radial head dislocation. We report a case of a patient with a distal humeral bony prominence and limited motion. Surgical reconstruction was attempted and failed to restore motion. The presence of an unusual bony prominence in this case probably represents a displaced medial condyle.

## CASE REPORT

H.N. was referred at six months of age for evaluation of a dislocated right radial head. His past medical history was significant for cleft palate repair at the age of three and a half months.

On examination, the right upper extremity had forty to eighty-five degrees of elbow motion and very limited supination and pronation. Radiographs of the elbow revealed an absence of formation of the medial condyle with medial subluxation of the ulna (Figure 1). The lateral film demonstrated elongation of the radial neck, with posterior subluxation of the radial head in relation to the distal humerus (Figure 2). Elbow arthrogram was performed and the radial head showed posterior subluxation with flattening of the anterior portion of the unossified proximal radial ossification center. There was communication of the joint space with the space along the proximal ulna. No trochlea could be identified and there was gross distortion of the proximal ulna and the ossified portion of the distal humerus (Figure 3).

At eight months of age he underwent exploration and attempted reconstruction through an anterior approach. The dysplastic capitellum angled posteriorly approximately ten degrees. It articulated with an angulated flat radial head which fit the capitellum best in the neutral position (Figure 4). The radial head was slightly longer than normal, but was not specifically subluxed posteriorly as had appeared in the radiographs. Inspection of the medial side of the joint showed an extremely dysplastic trochlea. It was lying two centimeters proximal to the capitellum.

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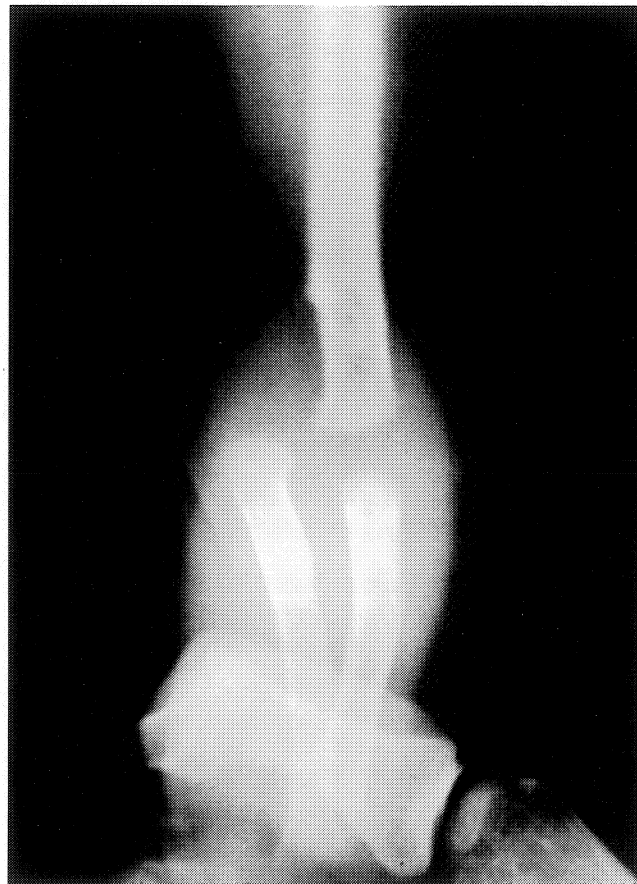


Figure 1  
AP radiograph of the elbow demonstrates absence of medial condyle with medial subluxation of the ulna.

Motion was present between the trochlea and the humeral shaft, possibly representing a loose fibrocartilaginous union. A band of cartilage passed from the olecranon posteriorly and attached firmly to the rim of the trochlea. A cleft appeared between this cartilage and the medial shaft of the distal humerus. A remnant of the joint was present between the olecranon and the trochlea posteriorly. Range of motion of the elbow at surgery improved to forty-five to 120 degrees. However, the motion was markedly incongruous at the radial capitellar joint and the



Figure 2

Lateral radiograph of the elbow demonstrated a mass of bone anteriorly on the distal humerus (The Don't Operate On Me Sign). The radial head appears subluxed posteriorly.

ulnar trochlea joints. Because reconstruction of the joint was not possible, the procedure was complete and the skin reapproximated.

The child has continued to be followed in our clinic without any final improvement in preoperative motion. Radiographs demonstrated continued development of the elbow (Figure 5).



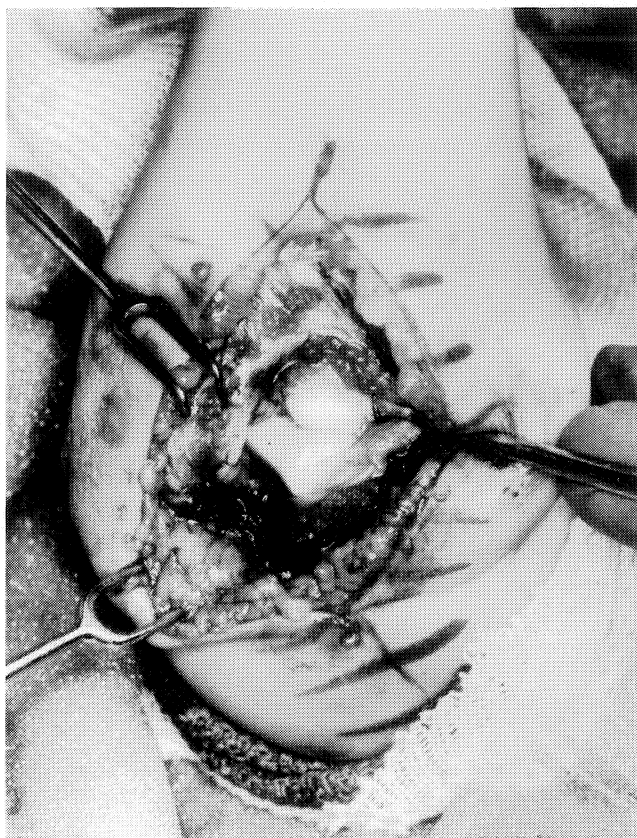
Figure 3

Arthrogram of the elbow.

### DISCUSSION

The bony prominence on the humerus possibly presents the displaced medial condyle. The abnormality could be a variant of humeral-ulnar synostosis although, in this particular case, the elbow did not become solidly fused. A similar mass is noted in an example of humeral-radial synostosis; the prominence is likely a displaced rudimentary ulna (Figure 6).

From this case study it is reasonable to assume that when one sees his abnormal bony ossification in a child, that surgery will not be helpful, "The Don't Operate On Me Sign".



**Figure 4**  
Operative exposure of the dysplastic radiocapitellum articulation.



**Figure 5**  
AP radiograph of the elbow one year after surgery. Note ossification of the trochlea.



**Figure 6**  
Radiograph of humeral-radial synostosis with a large bony prominence. A variant of the "Don't Operate On Me Sign."

# ANTERIOR LABRUM RECONSTRUCTION WITH MINI-CAPSULAR SHIFT PROCEDURE

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John L. Pinkowski, M.D.

## ABSTRACT

An anterior labrum reconstruction and mini-capsular shift (ARMS) procedure was performed in 64 patients (69 shoulders) with traumatic anterior or anterior-inferior glenohumeral instability between 1984 and 1990. Sixty-three of the sixty-nine stabilizations were performed for recurrent dislocation and six were performed for recurrent subluxation. Eighty-eight percent of the patients (61 shoulders) were available for clinical follow-up at an average of 36 months (range 28-78). There were 44 males and 12 females with an average age of 28 years (range 15-46).

Excellent range of motion was recorded at follow-up for the operated shoulder with an average of 180 degrees of forward elevation, 72 degrees of external rotation with the arm at the side, 92 degrees of external rotation with the arm in the 90 degree abducted position and 90 degrees of internal rotation with the arm in the 90 degree abducted position. The range of motion of the normal shoulder was 180 degrees, 76 degrees, 101 degrees and 94 degrees respectively. Utilizing the rating scale from the American Shoulder and Elbow Society, pain improved from 3.1 to 4.4, stability improved from 1.1 to 4.5, and function improved from 2.5 to 3.8 on the average. Subjective rating revealed 95% of the patients to be satisfied with the operative procedure. According to the criteria of Rowe et al, 90% of the results were good or excellent. Five patients (8%) suffered a recurrent dislocation at an average of 32 months after the surgery. Four resulted from significant trauma (6%). One patient (2%) complained of a single episode of subluxation during early recovery. Two patients required shoulder manipulations to improve motion. No other complications occurred. The patients reviewed in this study were actively involved in sports. The majority of pre- and postoperative Tegner ratings exceeded 7.0.

## INTRODUCTION

The importance of an anterior glenohumeral ligament complex avulsion in the pathogenesis of recurrent anterior instability was first described by Perthes in 1906<sup>12</sup>. In 1938, Bankart reconfirmed the importance of the lesion and detailed a technique for its repair using sutures

through drill holes placed in the anterior glenoid rim<sup>2</sup>. Since that time there have been a number of procedures described and applied to recurrent anterior instability of the shoulder. Besides the Bankart repair, staple capsulorrhaphy (Rayo-DuToit)<sup>14</sup>, subscapularis tightening (Putti-Platt)<sup>13</sup>, subscapularis transfer (Magnuson-Stack)<sup>6</sup>, bony block (Eden-Hybbinette)<sup>11</sup>, coracoid transfer (Bristow-Helfet)<sup>22</sup> and other stabilization procedures have been published in the orthopaedic literature<sup>10,19,24</sup>. More recently, a small number of reports on arthroscopic capsular reconstructions, labrum staplings and suture repairs have been published<sup>3,4,7</sup>. Large differences in success rates (65%-99%) have been reported for these various procedures. Patient profiles, activity scales and accurate range of shoulder motion have been, for the most part, ignored in these reports, and thus, it is difficult to accurately compare the success rates of one procedure to another.

Rowe et al<sup>17</sup> in 1978 described a modification of the Bankart technique and reported its use in 161 patients with a 4% recurrence rate. Although one of the most detailed reports to date, patient follow-up details such as resultant range of motion were not clearly provided in this review, nor were patient-activity profiles. In 1989<sup>21</sup>, Thomas and Matsen further modified the Bankart-Rowe procedure in an effort to simplify the technique and minimize trauma to the subscapularis, thus improving range of motion. Their achieved range of motion was excellent as predicted and they reported a 97% good to excellent overall result. Unfortunately, only 62% of the patient population was available for follow-up and no activity profiles were determined. Potential arguments against Bankart capsular procedures, because of late failure and stretching of capsular tissues, were refuted by these long term studies.

The reasons for failure of anterior stabilization procedures have been evaluated by Morrey and Janes<sup>8</sup>, and Rowe et al<sup>16</sup>, among others. These authors found that the presence of a Bankart lesion and excessive laxity of the joint are major factors that contribute to recurrence. Whereas the use of hardware (staples and screws), bone blocks (including coracoid transfers) and muscle-shortening procedures result in good to excellent stability, they lead to poor functional results secondary to loss of motion, pain and glenohumeral arthritis. In our opinion, one of the greatest obstacles to comparing previous

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**TABLE I**  
**Rating scale from American Shoulder and Elbow Society**

<i>Pain</i>	<i>Stability</i>	<i>Function</i>
(5) None	(5) Normal	(4) Normal
(4) Slight	(4) Apprehension	(3) Mild Compromise
(3) After Unusual	(3) Rare Subluxation	(2) With Difficulty
(2) Moderate	(2) Recurrent Subluxation	(1) With Aid
(1) Marked	(1) Recurrent Dislocation	(0) Unable
(0) Complete Disability	(0) Fixed Dislocation	

reports of surgical procedures is the fact that atraumatic, multidirectional instability of the shoulder was not well-recognized nor described prior to Neer and Foster's report<sup>9</sup> in 1980. It is now well-recognized that there is a continuum of pathology, beginning with the traumatic recurrent anterior dislocators (TUBS) and proceeding to the opposite extreme in those patients who present with atraumatic multidirectional instability (AMBRI)<sup>16</sup>. Distinguishing the degree of capsular laxity, either constitutional or acquired, can be extremely difficult and this is perhaps one of the reasons why so many procedures have been developed to tighten the anterior capsular structures in deference to repairing the Bankart lesion. Recognizing the importance of repairing the anterior glenohumeral ligament complex (including the labrum), and the difficulty of assessing and treating anterior and anterior-inferior capsular laxity, the senior author (LEP) has used an anterior labrum reconstruction and mini-capsular shift (ARMS) procedure in the treatment of traumatic and atraumatic anterior and anterior-inferior instability.

We diagnose *anterior instability* in patients who present with documented glenohumeral dislocations or subluxations with the arm maximally abducted and externally rotated. These patients have apprehension in this arm position, and often have radiographic evidence of anterior glenoid and posterior humeral head trauma. These patients have no inferior or posterior patholaxity. Patients with *anterior-inferior instability* have similar findings, but *also* demonstrate an increased amount of inferior laxity (positive sulcus sign). The inferior laxity may be uni- or bilateral, but no posterior patholaxity exists in this group.

The ARMS procedure has been used since 1984, and permits selective anterior and inferior capsular tightening at the time of surgery. The ARMS procedure consists of a mini-approach through the inferior one-third of the subscapularis tendon and capsule, repair or reconstruction of the Bankart lesion and a shift of the anterior-inferior capsule superolaterally. This approach affords the opportunity to address the Bankart lesion and any capsular redundancy while preserving motion and subscapularis function. The technique and the first six years of experience are described in this report.

## MATERIALS AND METHODS

Sixty-seven patients underwent an ARMS procedure for recurrent, *traumatic* anterior or anterior-inferior glenohumeral instability between 1984 and 1990. Three patients died, leaving 64 available for independent review. We examined 39 patients and conducted a chart review and phone interview on another 17 patients, resulting in an 88% follow-up. The surgeon was excluded from all evaluations. There were a total of 61 stabilizations performed in these 56 patients. Forty-four shoulders experienced instability as a direct result of sporting activities and 17 resulted from a motor-vehicle or motorcycle accident or a fall. There were 44 men and 12 women. The average age was 28 years (range 15-46). The operation was performed on 40 right shoulders and 21 left shoulders. Thirty-nine procedures were performed on the dominant and 22 on the nondominant shoulder. The average follow-up was 36 months (range 28-78).

The cases were reviewed using a detailed questionnaire and clinical examination. The questionnaire, based on the rating scale of the American Shoulder and Elbow Society, specifically addressed pain, stability and function (TABLE I). We also used a rating sheet for Bankart repair as described by Rowe et al<sup>7</sup> to allow comparison with other reports (Table II). Pre- and postoperative patient activity levels were quantitated using the Tegner Activity Level Scale<sup>20</sup> (Table III). Recovery time and overall subjective satisfaction were also recorded. The clinical examination documented range of motion in forward elevation, external rotation at the side and in the 90 degree abducted position, and internal rotation with the arm in the 90 degree abducted position. Stability was assessed in 90 degrees of abduction and maximum external rotation to elicit an apprehension sign.

The range of motion and stability of those patients not examined clinically were assessed over the phone. The patients were instructed to perform the following maneuvers with their backs against the wall. In order to assess forward elevation, they were asked to lift their hands over their heads with their elbows fully extended. Internal and external rotation was assessed in 90° of abduction by having the patients flex the elbow to 90° and then rotate

**TABLE II**  
**Rating Sheet for Bankart Repair**

Scoring System	Units	Excellent (100-90)	Good (89-75)	Fair (71-51)	Poor (50 or Less)
<b>Stability</b>					
No recurrence, subluxation	50	No recurrence	No recurrence	No recurrence	Recurrence of dislocation or
Apprehension when placing arm in certain positions	30	No apprehension when placing arm in complete elevation and external rotation	Mild apprehension when placing arm in elevation and external rotation	Moderate apprehension during elevation and external rotation	Marked apprehension during elevation
Subluxation (not requiring reduction)	10	No subluxations	No subluxations	No subluxations	
Recurrent dislocation	0				
<b>Motion</b>					
100% of normal external rotation, internal rotation and elevation	20	100% of normal external rotation, complete elevation and internal rotation	75% of normal external rotation, complete elevation and internal rotation	50% of normal external rotation, 75% of elevation and internal rotation	No external, 50% of elevation (can get hand to face) and 50% of internal rotation
75% of normal external rotation, normal elevation and internal rotation	15				
50% of normal external rotation, 75% of normal elevation and internal rotation	5				
50% of normal elevation and internal rotation, no external rotation	0				
<b>Function</b>					
No limitation in work or sports; little or no discomfort	30	Performs all work and sports; no limitation in overhead activities; shoulder strong in lifting, swimming, tennis, throwing; no discomfort	Mild limitation in work and sports; shoulder strong; minimum discomfort	Moderate limitation doing overhead work and heavy lifting; unable to throw, serve overhead in tennis, or swim; moderate disabling pain	Marked limitation; unable to perform overhead work and lifting; cannot throw, play tennis, or swim; chronic discomfort
Mild limitation and minimum discomfort	25				
Moderate limitation and discomfort	10				
Marked limitation and pain	0				
Total units possible	100				

their arms so that their hands pointed toward the floor and then the ceiling respectively. External rotation was also assessed with the arms adducted at their sides by having the patients outwardly rotate the arm with the elbow flexed 90°. The patients were then instructed to compare

sides. Assessment of anterior apprehension was made by asking the patients to place their arms in a cocking position and simulate a forceful throwing maneuver. This method of data acquisition was also applied to those patients who were clinically examined and was found to only correlate

TABLE III  
Tegner Activity Level Scale — Definitions

<b>LEVEL 10</b>	<b>COMPETITIVE SPORTS</b> —Soccer, football, rugby (national elite)	<b>LEVEL 5</b>	<b>RECREATIONAL SPORTS</b> —Jogging on uneven ground at least twice weekly
<b>LEVEL 9</b>	<b>COMPETITIVE SPORTS</b> —Soccer, football, rugby (lower division) —Ice hockey —Wrestling —Gymnastics	<b>LEVEL 4</b>	<b>WORK</b> —Moderately heavy labor (truck driving, etc.)
<b>LEVEL 8</b>	<b>COMPETITIVE SPORTS</b> —Racquetball or bandy —Squash or badminton —Track & field, athletics (jumping, etc.) —Downhill skiing	<b>LEVEL 4</b>	<b>RECREATIONAL SPORTS</b> —Cycling —Cross-country skiing —Jogging on even ground at least twice weekly
<b>LEVEL 7</b>	<b>RECREATIONAL SPORTS</b> —Soccer, football, rugby —Bandy and ice hockey —Squash and racquetball —Athletics (running) —Cross-country running (competitive or recreational)	<b>LEVEL 3</b>	<b>WORK</b> —Light labor (nursing, etc.)
<b>LEVEL 6</b>	<b>RECREATIONAL SPORTS</b> —Tennis —Handball —Basketball —Racquetball —Downhill skiing —Jogging at least 5x per week	<b>LEVEL 3</b>	<b>COMPETITIVE/RECREATION/SPORTS</b> —Swimming
<b>LEVEL 5</b>	<b>WORK</b> —Heavy labor (construction)	<b>LEVEL 3</b>	<b>BACKPACKING/HIKING</b>
<b>LEVEL 5</b>	<b>COMPETITIVE SPORTS</b> —Cycling —Cross-country skiing	<b>LEVEL 2</b>	<b>WORK</b> —Light labor (mailman, etc.)
		<b>LEVEL 2</b>	Walking on uneven ground possible but impossible to hike or backpack
		<b>LEVEL 1</b>	<b>WORK</b> —Sedentary work (secretarial)
		<b>LEVEL 1</b>	Walking on even ground possible
		<b>LEVEL 0</b>	Sick leave or disability pension because of knee problems

within 10 degrees of the motion actually measured by the examiner; thus, motion assessment over the phone was not as accurate.

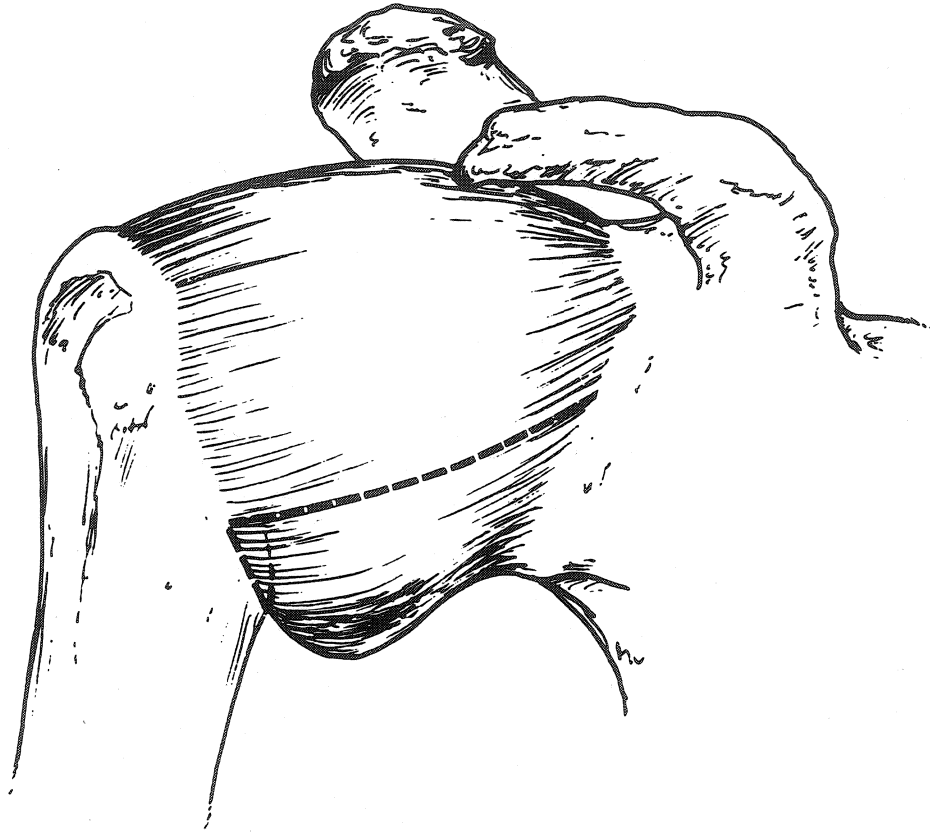
#### TECHNIQUE

The ARMS procedure has been used since 1984. It combines aspects of the methods described by Rowe et al<sup>7</sup> and Neer and Foster<sup>4</sup>. The labrum is reattached from inside the joint with the aid of the drill holes or suture anchors. If the labrum is deficient or absent, it can be reconstructed by imbricating the capsule medially and then securing it with the same methods described above. Once

the labral insufficiency has been addressed (or if there is none present) the anterior-inferior capsular redundancy is treated by utilizing a superolateral shift of the inferior limb of the capsule. All the operations were performed by or under the direction of the senior author (LEP).

The indications for this procedure include a patient history and physical examination, and radiographic evidence confirming a recurrent anterior or anterior-inferior instability. Findings of significant multidirectional or posterior laxity are a contraindication to the operation.

The surgical technique is performed under general endotracheal anesthesia. Examination under anesthesia is



**Figure 1**

The subscapularis and its underlying capsule are vertically incised 1.5 cm from the insertion of the subscapularis onto the lesser tuberosity through the inferior fibers, reflecting approximately one-third of the inferior subscapularis muscle.

carried out with careful attention to the extent of anterior, inferior, and posterior laxity. A significantly positive sulcus sign ( $\geq 2$  cm) with greater than 50% anterior and inferior subluxation of the humeral head over the glenoid rim indicate the need for a Neer inferior capsular shift.

The patient is placed in the “beach-chair” position with the waist and legs slightly flexed. A small bolster is placed just medial to the scapula to allow more posterior displacement of the humeral head during the procedure. Arthroscopy may be indicated to confirm the diagnosis and assess other pathology in selected cases.

A standard deltopectoral approach is made using a 6 cm incision from the coracoid to the axilla. A short (4 cm) axillary incision may be used in patients when cosmesis is more of a concern. The deltopectoral interval is identified along with the cephalic vein. The vein is retracted laterally. The coracoid and conjoined tendon are identified and the conjoined tendon is retracted medially. It is not necessary to osteotomize the coracoid nor partially release the conjoined or pectoralis tendon. The arm is externally rotated revealing the subscapularis. The vessels at its inferior border are identified and cauterized or retracted out of the operative field.

The rotator interval between the subscapularis and the supraspinatus tendon is identified. If the patient demonstrated a significant sulcus sign under anesthesia and if the interval is excessively large ( $\geq 15$  mm) or it splays open when downward traction is applied to the arm, then it is closed. O-Dacron sutures are used to close the interval after exposing the biceps tendon and taking care to avoid placing sutures in it. This will add to inferior stability of the reconstruction by tightening the superior capsule and its associated coracohumeral ligaments. The interval was closed in about 40% of our patients. A vertical incision is made in the inferior one-third of the subscapularis tendon, starting approximately 1.5 centimeters medial to the insertion of the subscapularis onto the lesser tuberosity. The incision is carried into the joint (Figure 1). A horizontal split is then made through the subscapularis tendon and capsule extending medially to the anterior glenoid rim (Figure 2). Care is taken to protect the axillary nerve. The lower one-third of the subscapularis muscle is then dissected off of the capsule and tagged with multiple #1 Vicryl sutures (Figure 2). The capsule is dissected off its humeral insertion using a periosteal elevator. This is performed while externally rotating the arm. This facili-

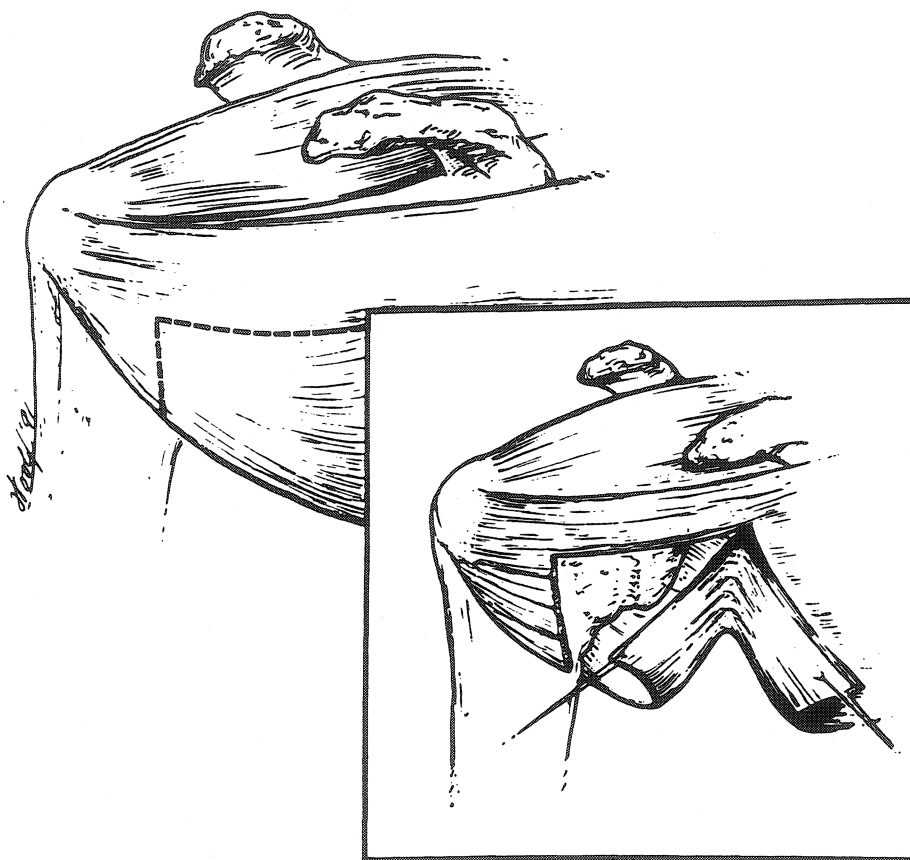


Figure 2

After the subscapularis and capsule tissues are reflected, the capsule can be more easily separated from the overlying subscapularis muscle. The capsule is then released from the humeral neck as the humerus is gradually externally rotated. Care is taken not to harm the axillary nerve which lies directly inferior to the capsule.

tates release of enough of the inferior capsular attachment and it helps to protect the axillary nerve.

The extent of capsular stripping from the humeral neck is determined by the degree of inferior capsular laxity and the size of the inferior capsular pouch. A normal inferior capsular pouch should not allow an index finger to occupy the inferior glenoid space.

Generally speaking, those patients who required "interval closure" also required more shift of the inferior capsule. If significant inferior patholaxity was not present, then the capsule was only stripped for a distance of one centimeter. A humeral head retractor is inserted along with a labral retractor so that the anterior labrum can be better visualized. The labral defect is repaired with #2 Ethibond sutures through bony holes in the anterior glenoid rim or with the use of suture anchors (Figure 3). The labrum can be repaired to soft tissue or with a screw and washer if there is a large bony fragment. The repair site is prepared by roughening the anterior glenoid rim and neck with a burr and rasp. Initially, in this series, holes were utilized to repair the labral defect. These were made using a small drill to create parallel holes which were then

connected to the outer cortex with a tenaculum and small right-angled awl. A #5 Mayo needle was used to pull the suture through these holes. More recently, suture anchors have been used to repair the labrum. The insertion holes for the anchors are prepared *on* (not below) the glenoid rim using the specifically designed drill and drill guide. The anchors are fully inserted and set. Usually three anchors are needed for the repair, but this number varies depending on the size of the defect. Once the sutures are set, they are brought through the capsule and tied outside the joint. This gives excellent "tissue-to-bone" approximation with the suture knots outside the joint (Figure 3 inset).

After the labral lesion is repaired, the capsular shift is performed if indicated. Sufficient capsular release off of the humerus is assured by pulling the inferior capsule superiorly and digitally probing the inferior pouch. If the inferior recess is obliterated, then the dissection is adequate. If the recess is still present, then further external rotation and release of the capsule from the humeral neck is indicated. Using a burr, the cortical bone along the inferior

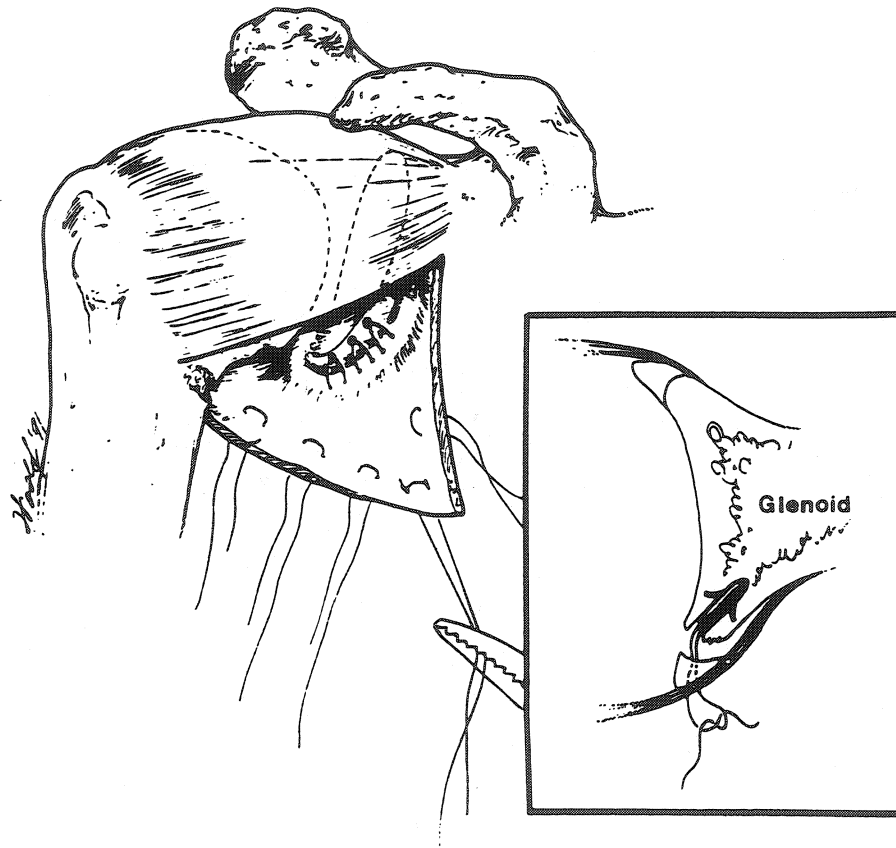


Figure 3

Abrasion of the glenoid neck is carried out to enhance capsule-to-bone healing. The Bankart lesion is repaired using suture anchors near the articular surface. Care is taken not to place the anchor too far medial to the lip of the glenoid. Multiple sutures are then placed in a progressive manner, proceeding from inferior to superior along the lateral capsular edge. This facilitates anterolateral advancement of the inferior capsule.

neck is roughened. This will create a bleeding surface to promote the attachment of the advanced capsule.

Multiple #2 Ethibond stay sutures are placed going outside-to-inside and then inside-to-outside along the inferior flap of the capsule. Placement of these sutures is critical. A stay-suture is placed at the apex of the inferior flap. This will be the leading edge of the inferior capsule when it is shifted superiorly. Next, multiple sutures (usually three) are placed laterally along the vertical edge of the flap, proceeding inferiorly and posteriorly. These sutures will further advance the capsule superiorly and laterally (Figure 3).

The stay-sutures are then tied. It is important for the arm to be held in 30 degrees of abduction and 0 degrees of external rotation to assure postoperative external rotation is near normal. The apical suture is brought underneath the superior capsule and out through the superolateral attachment of the subscapularis (not in the muscle itself). The vertical sutures are brought up in a similar fashion just inferior to the apical suture. This suture placement shifts the inferior capsule laterally and superiorly (Figure 4),

without "tethering" that part of the subscapularis muscle which was not detached.

The inferior one-third of the subscapularis tendon is then reattached anatomically with nonabsorbable sutures and the deltopectoral interval is reapproximated.

After the operation, the shoulder is immobilized in a sling for one to three weeks, depending on the amount of capsular laxity at the time of surgery. Passive range of motion is initiated one week postoperatively. The goal for external rotation is 5-10 degrees per week starting at 0 degrees after week three. Active range of motion is implemented on week three. Isotonic exercises are started on week six. Return to contact sports is not allowed until six months postoperatively.

## RESULTS

We reviewed the operative findings of the 61 shoulders. All of the patients had a Bankart lesion with varying degrees of capsular laxity. In four shoulders, there was an associated fracture of the glenoid rim (6%). These patients were treated with fixation of the bony fragment using a 3.5 mm short lag cancellous screw with a washer, as well as

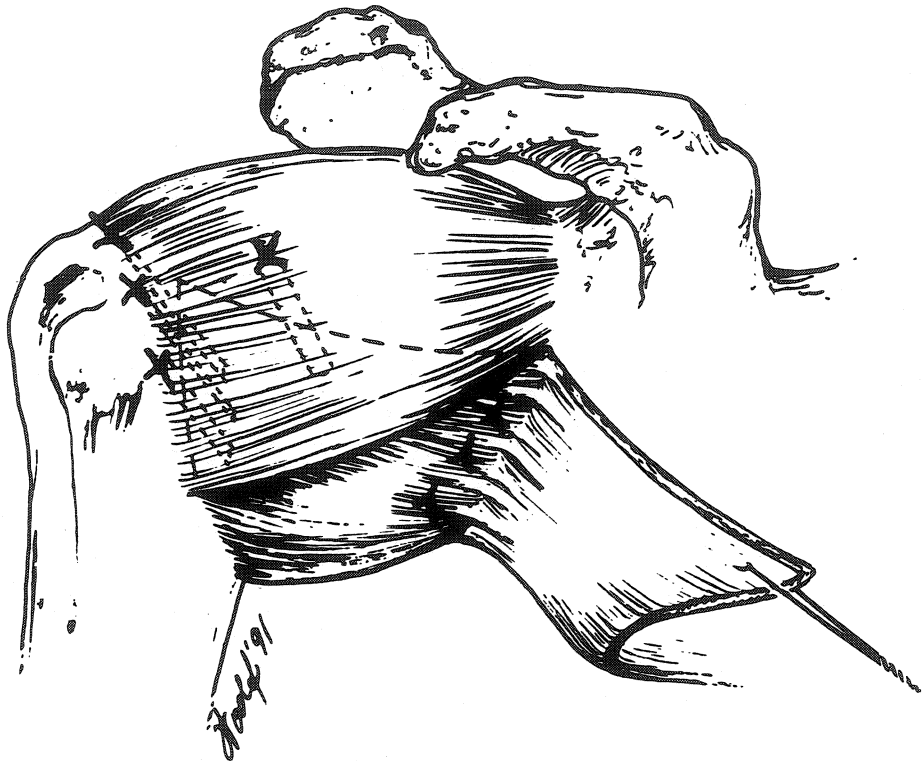


Figure 4

When the anterior-inferior capsule is shifted, care is taken not to tenodesse the remaining subscapularis by placing the sutures medially. Also, the intra-articular portion of the long head of the biceps must be avoided as the sutures are placed superiorly through the subscapularis and capsule. The arm should be held in 30 degrees of abduction with a bolster behind the scapula so that the prominence of the humeral head prevents overtightening. After the inferior capsule has been secured, the subscapularis tendon fibers can be returned to their normal anatomic position using nonabsorbable sutures.

soft tissue repair of the labrum. A posterolateral Hill-Sach's lesion was noted in 29 shoulders (50%), but this did not alter treatment. A redundant inferior recess was noted in 42 shoulders (61%), but was felt to be significant in only 28 of these patients (40%). This was observed arthroscopically in many cases as well as by open visualization. Fixation of the labrum was accomplished with drill holes through bone in 26 shoulders (42%) and with suture anchors in 25 shoulders (42%). No difference in clinical results was noted between these two groups. However, operative time was reduced significantly (average 28 minutes) by using suture anchors. The average number of drill holes or anchors required to repair the lesion was 2.6 with a range of one to four. Six shoulders had soft tissue repair of the labral defect with sutures (10%) and four (6%) had repair of the labrum through fixation of the bony Bankart Lesion using a screw and washer. There were no immediate perioperative complications.

The postoperative range of motion demonstrated an average forward elevation of 180 degrees (range, 170 to 180), an average external rotation at the side of 72 degrees (range, 10 to 95 degrees), an average external rotation at 90 degrees of abduction of 92 degrees (range,

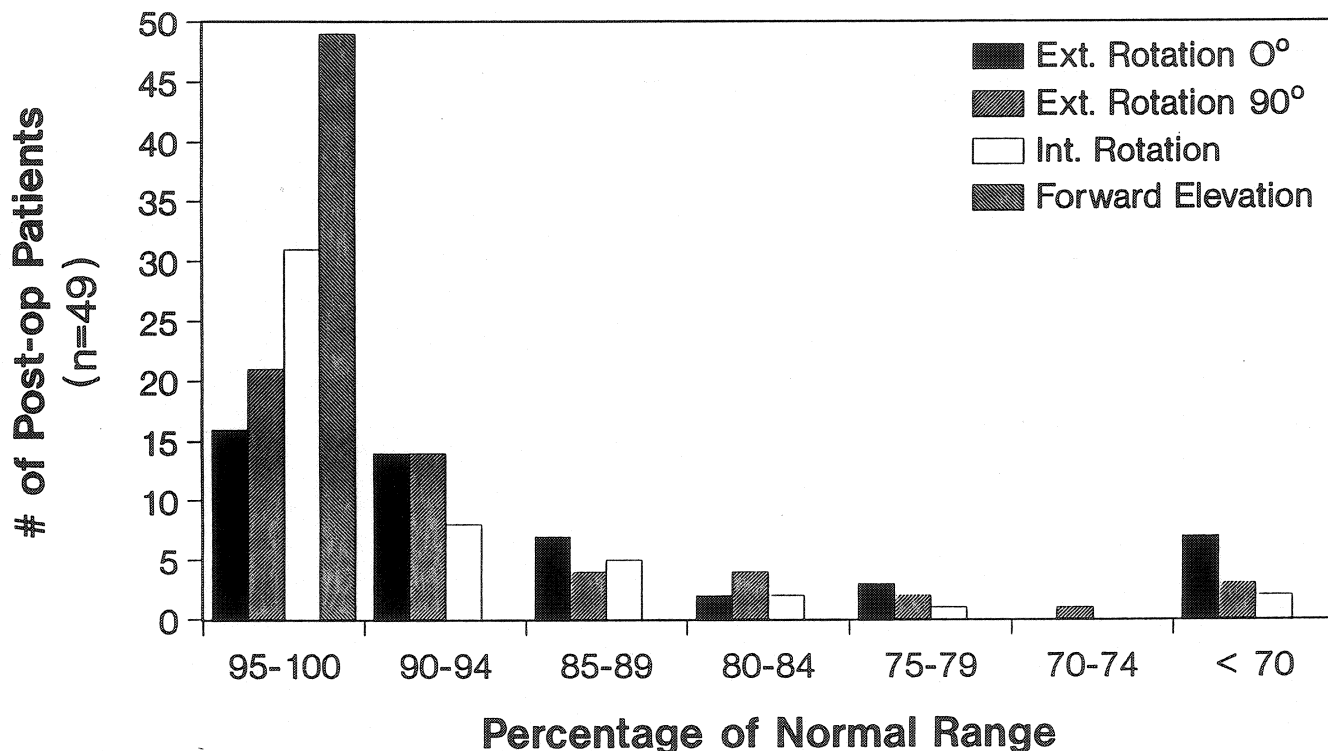
45 to 120 degrees), and an average internal rotation of 90 degrees (range 65 to 125 degrees). When compared to the opposite shoulder, this represents normal forward elevation, a loss of four degrees of external rotation at the side, a loss of nine degrees of external rotation at 90 degrees of abduction, and a loss of four degrees of internal rotation (Table IV).

The recovery time, as determined by return to full sports and activities without limitation, averaged 6.5 months (range 3-12). Fourteen patients (23%) considered themselves to be fully recovered by three months and forty-one patients by six months.

Manipulations were required in two patients (3%). Both patients had failed to progress despite aggressive physical therapy. One patient had a manipulation three months after surgery because forward elevation was only 75 degrees and external rotation was 0 degrees. At 57 months follow-up, this had improved to 180 degrees of forward elevation and 90 degrees of external rotation. The other patient required a manipulation at four months after surgery. The pre-manipulation motion in forward elevation was 133 degrees and external rotation was 27 degrees. At 45 months follow-up the patient had 180 degrees of

TABLE IV

## Range of Motion



forward elevation and 90 degrees of external rotation. Of note was the fact that many patients gained motion as late as two years postoperatively. Thus, we are now less concerned with slow progress in physical therapy during the first year.

Based on the rating scale from the Shoulder and Elbow Society, pain indicators improved from an average 3.1 (range 1-5) to an average 4.4 (range 3-5). Thirty-five patients (57%) reported no pain and 19 patients (32%) reported only slight occasional discomfort with sports. The patients with more marked pain experienced episodes of recurrent instability or had a greater limitation of motion.

Function, as it relates to activities of daily living and sports, improved from an average of 2.5 (range 1-4) to an average of 3.8 (range 2-4). Fifty-one patients (84%) reported normal function. Nine patients (15%) had mild limitations and one patient had significant compromise. The limitations in function related directly to recurrent instability and/or the presence of pain. Using the Tegner Activity Scale<sup>20</sup> to rate patient activity levels revealed an average preoperative score of 6.0 (range 3-9). Over 60% of the patients returned to their previous level of work or

sports participation. None reduced their activity level because of their shoulder.

Stability scores improved from an average of 1.1 (range 1-2) to an average of 4.5 (range 1-5). There were 55 patients (90%) with excellent stability and no apprehension signs. Five patients (8%) redislocated and one patient experienced one episode of subluxation. Four of the five patients who redislocated did so secondary to significant trauma. This represents a 2% recurrence rate due to procedure failure and a 6% recurrence rate due to serious trauma, for a total of 8% failures.

According to the criteria of Rowe et al, there were 49 excellent, five good, two fair, and five poor results. Recurrent dislocation was the cause of the five poor results. Subluxation or excessive loss of motion resulted in the two fair results. Fifty-seven shoulders (93%) had normal or near-normal motion. Four patients had 50% or less of normal external rotation. Thirty-six patients had normal function of the shoulder, 18 had mild limitation, and 7 patients had moderate limitation.

We analyzed the patients who had recurrent instability. The average age of these patients was 25 years (range 22-38). The average time to recurrence was 32 months



(range 12-66). The nondominant shoulder was involved in four out of six patients. Of those patients who redislocated, one patient redislocated twice and the others only once. All these dislocations spontaneously reduced. As previously noted, significant trauma was the cause of the redislocation in four of five patients. Three occurred from a serious fall while snow skiing, one from a high-speed fall while water skiing, and one from a less-forceful maneuver when the patient was pulled up onto a roof by his involved arm.

Re-operation for recurrent instability has been performed in two patients. One patient was found to have a disruption of a previously repaired labrum that had been fixed with three suture anchors. The labrum had torn away from the firmly attached anchors. The second patient had sustained a large bony fracture of the anterior glenoid (through the Bankart suture tunnels) which required open reduction and internal fixation.

One patient experienced one episode of subluxation during early recovery (12 months). This occurred while playing basketball. The patient was dunking the ball and hit the backboard, forcing his arm into abduction and external rotation. This degree of trauma may have been adequate to cause the recurrence, but in review, it was noted that this patient is mildly ligamentously lax with a mild sulcus sign and a history of involvement of both shoulders. He has had no recurrent instability symptoms at 28 months.

## DISCUSSION

Recently, recurrent glenohumeral instability has been classified into two groups. The first group, referred to by the acronym TUBS, has a traumatic etiology, is unidirectional, has a Bankart lesion and typically requires surgery to restore stability. The second group, referred to by the acronym AMBRI, has an atraumatic etiology, is multidirectional, is often times bilateral, and is usually treated by aggressive rehabilitation prior to employing surgery<sup>16</sup>. When surgery is necessary, reconstruction using an inferior capsular shift type of procedure is indicated<sup>9</sup>. Although this classification facilitates discussion, many patients fall somewhere between a true unidirectional instability and a multidirectional instability. Thus, the distinction between these two groups is at best difficult and a surgical procedure specifically designed to address one extreme or the other may be inappropriate. It is our feeling that *both* capsular laxity and labrum insufficiency may be prominent features of unidirectional instability. The ARMS procedure was developed to simultaneously address labrum insufficiency and/or capsular redundancy when necessary. The indication for the ARMS procedure is a patient with predominantly anterior or anterior-inferior patholaxity (as previously defined) and no posterior patholaxity. Thus, the patients reviewed in this series demonstrated a torn anterior labrum (100%); a positive appre-

hension sign (85%); a positive sulcus sign (61%); *no* posterior labrum tears, and *no* posterior patholaxity. By employing the same surgical approach and exposure, the treatment is simplified. By selectively shifting the anterior-inferior capsule superolaterally, the surgeon has better control over the ultimate anterior-inferior laxity that is achieved. We still recommend the inferior capsular shift for the multidirectional instability patient who presents with those clinical features first delineated by Neer in 1980<sup>9</sup>.

By only reflecting the inferior one-third of the subscapularis tendon, its resultant function is minimally affected. Thus, the use of this procedure in throwing athletes is preferred. Jobe et al described an anterior capsulolabral reconstruction for these throwing athletes<sup>5</sup>. A small capsular shift and labral reconstruction were performed on the glenoid side of the joint. Our preference is to reconstruct or reattach the labrum and then shift the capsule on the humeral side of the joint. In the authors opinion, this better reinforces the inferior capsule and affords specific control of the capsular shift with less likelihood of damage to the axillary nerve. Additionally, healing of the capsule to bone is more likely and there is more capsular tissue to work with. The two patients who required repeat surgery were found to have significant labral injuries and confirmed our impression that this surgical technique effects excellent humeral and glenolabral healing without sacrificing motion or function.

As previously noted, our technique is designed to address the Bankart lesion and any moderate to severe capsular redundancy that is constitutional or the result of multiple dislocations. It allows healing of the lateral capsule to bone and tightens the anterior-inferior capsule when the inferior limb is shifted superolaterally. This acts as a check-reign to anterior-inferior displacement. Other advantages of this technique are important to point out. Since most of the procedure is performed laterally, we have not found it necessary to osteotomize the coracoid. Also, by minimizing trauma to the subscapularis muscle and reattaching the lower portion anatomically, the "tenodesis" effect is avoided, resulting in excellent motion. Maintenance of external rotation was evidenced by the fact that 93% of the patients had normal or near-normal motion, with average external rotation of 72 degrees with the arm at the side and an average external rotation of 92 degrees with the arm in the 90 degree abducted position. These motion results appear equal to those reported arthroscopically with a higher success rate for stability. Similar results to our own were achieved by Altchek and Warren in their 1991 report<sup>1</sup>.

Admittedly, the ARMS procedure can be significantly "more surgery" than simply reattaching a torn labrum and entails a higher risk of motion loss. However, it is our contention (and observation) that anterior instability is a

mixture of pathologic constitutional types. The ARMS procedure allows the surgeon flexibility in addressing the various "types" without altering the surgical approach.

Over-tightening the capsule or closing the rotator cuff interval in those patients that do not require these steps may result in significant loss of motion. We had four patients early in our series who lost approximately 50% of their external rotation. We believe over-tightening of the capsular tissues and failure to push aggressive range of motion exercises were the causes.

The advent of the suture anchor has made this procedure easier. A review of our operative procedures revealed an average savings of 28 minutes of operative time. A recent study assessing shoulder reconstructions with suture anchors also demonstrated excellent results without complications<sup>15</sup>. We found no difference in the results between the patients treated with drill holes through the anterior glenoid and the patients treated with suture anchor fixation. A possible exception to this, however, was noted in the recurrent dislocation group where a fracture through glenoid drill holes was found after a classic Bankart-type repair versus a similar case wherein the labral tissues had simply torn away from the glenoid and the suture anchors. Complications of using hardware about the shoulder have been reported<sup>25</sup>, but use of the suture anchor does not appear to be associated with these. Furthermore, in our experience, if failure of the initial procedure occurs, the presence of previously placed anchors does not preclude their use again as a fixation device as they do not significantly compromise bone stock.

Including our traumatic redislocations, we observed an 8% recurrent instability rate at an average of 36 months follow-up. This rate is higher than the results of 3-4% reported by Rowe et al<sup>17</sup> and Thomas and Matsen<sup>21</sup>. We feel this difference may be due to several factors. First, our patient selection included patients with unidirectional anterior instability and/or anterior-inferior instability as demonstrated by a positive sulcus sign. Thus, our study group contained patients with more constitutional laxity, and was closer in comparison to Altchek and Warren's group. Their recurrent instability rate was 9% with a similar follow-up time and retrieval rate<sup>1</sup>. Second, our average follow-up time was slightly longer and a higher percentage of patients returned for evaluation. Third, the "at-risk" activity level of our patients appears to be significantly higher than most studies due to the large snow-skiing population in our geographic area. Seventy-five percent of patients in this series sustained the initial subluxation or dislocation as a result of sports and over two-thirds of these were secondary to snow skiing.

In conclusion, we feel that the anterior labrum reconstruction with mini-capsular shift (ARMS) procedure offers the advantages of near-normal range of motion and

function, while providing excellent stability. It facilitates evaluation and correction of any capsular redundancy, as well as the repair of the Bankart lesion, with minimal trauma to the subscapularis muscle.

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# MRI OF TENDON INJURIES

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Tendons connect muscle to bone and transfer the force of muscle contraction to bone. Tendons have a distinct structural organization including cells (e.g. tenocytes) and an extracellular matrix composed of collagens, proteoglycans, non-collagenous proteins, and water. As a result, they are able to withstand larger forces than muscles<sup>2,19</sup>. Some tendons are enclosed by a synovial membrane called the tendon sheath, which consists of a visceral and a parietal layer separated by a film of fluid. The lining of the tendon sheath is highly cellular and quite vascular. It produces synovial fluid and reacts to inflammation by cellular proliferation and formation of excess fluid. This reaction may result in adhesions and restriction of the normal gliding motion between the visceral and parietal synovial layers<sup>19</sup>. Some tendons have no true synovial sheath and are instead encased by a layer of fibrous tissue called the paratenon. Tendons which follow a curved course are subject to greater strain than those following a straight course. Sesamoid bones improve the mechanical advantage of tendons across joints. Certain tendons such as the popliteus and the long head of the biceps are intracapsular.

With the advent of magnetic resonance imaging (MRI), our knowledge and understanding of tendon problems has greatly improved. Prior to MRI, tendons were evaluated by plain radiography, ultrasound, computed tomography (CT), and tenography. Although these modalities have advantages and continue to be used, they also have drawbacks. Plain radiography has limited tissue contrast and individual tendons are difficult to identify. Ultrasound is highly operator dependent, displays a small field of view, and has limited soft tissue contrast. CT achieves better tissue contrast than plain radiography or ultrasound, but CT lacks the multiplanar capabilities of MRI, which is key in studying muscles and tendons. With CT, images are typically acquired in the axial plane which is not always ideal for studying muscles and tendons. Tenography is invasive and difficult to interpret, and has variable accuracy. Because MRI is non-invasive, displays images in multiple planes and has excellent tissue contrast and

spatial resolution, it is now the preferred imaging modality for studying a large variety of musculoskeletal problems.

## ACHILLES TENDON

The Achilles tendon is the largest tendon in the body. The gastrocnemius and soleus combine to form the Achilles tendon. It has no true synovial sheath, instead it is encased by a layer of fibrous tissue called the paratenon. Its vascular watershed region is about 2-6 cm above its insertion on the calcaneus. This area of the tendon is susceptible to ischemia and is frequently the site of tendon degeneration and rupture. Injuries to the Achilles tendon cause changes ranging from mild inflammation of the paratenon to severe structural changes, including complete rupture. Paratenonitis is characterized by thickening and edema of the fatty areolar tissue between the paratenon and the tendon, fat necrosis, and connective tissue proliferation. This process impairs the normal gliding between the tendon and the paratenon. Tendinosis or tendinopathy denotes degenerative changes within the tendon substance, commonly present within the mid-substance. Histologically, areas of focal fibrous proliferation or poorly organized scar tissue, which weaken the tensile strength of the tendon, are seen. Tendon rupture is the most serious injury. It usually occurs between 2 and 6 cm proximal to its calcaneal insertion. This injury seems to be occurring with increasing frequency, and it is between 2 and 12 times more common in males<sup>13</sup>. Rupture of the Achilles tendon typically occurs in poorly conditioned, middle-aged men who engage in intermittent strenuous activity<sup>9,12</sup>. There is a high incidence of Achilles tendon rupture in individuals with type O blood<sup>9</sup>. The mean age of patients with Achilles tendon rupture is 35 years. Sudden jumping is a common cause, with basketball accounting for more than half the ruptures<sup>1,3</sup>.

The majority of complete Achilles tendon ruptures usually can be diagnosed clinically. However, local hemorrhage and edema may obscure the clinical findings and up to 20-30% of cases may be missed<sup>11,23</sup>. For these patients, MRI has proven to be an effective diagnostic tool. Like all other tendons in the ankle, the Achilles tendon is best imaged in the sagittal and axial planes. Normal tendons appear dark on all spin-echo sequences because of their high collagen concentration and low water content<sup>14,20</sup> (Figure 1). On the axial sections, the normal

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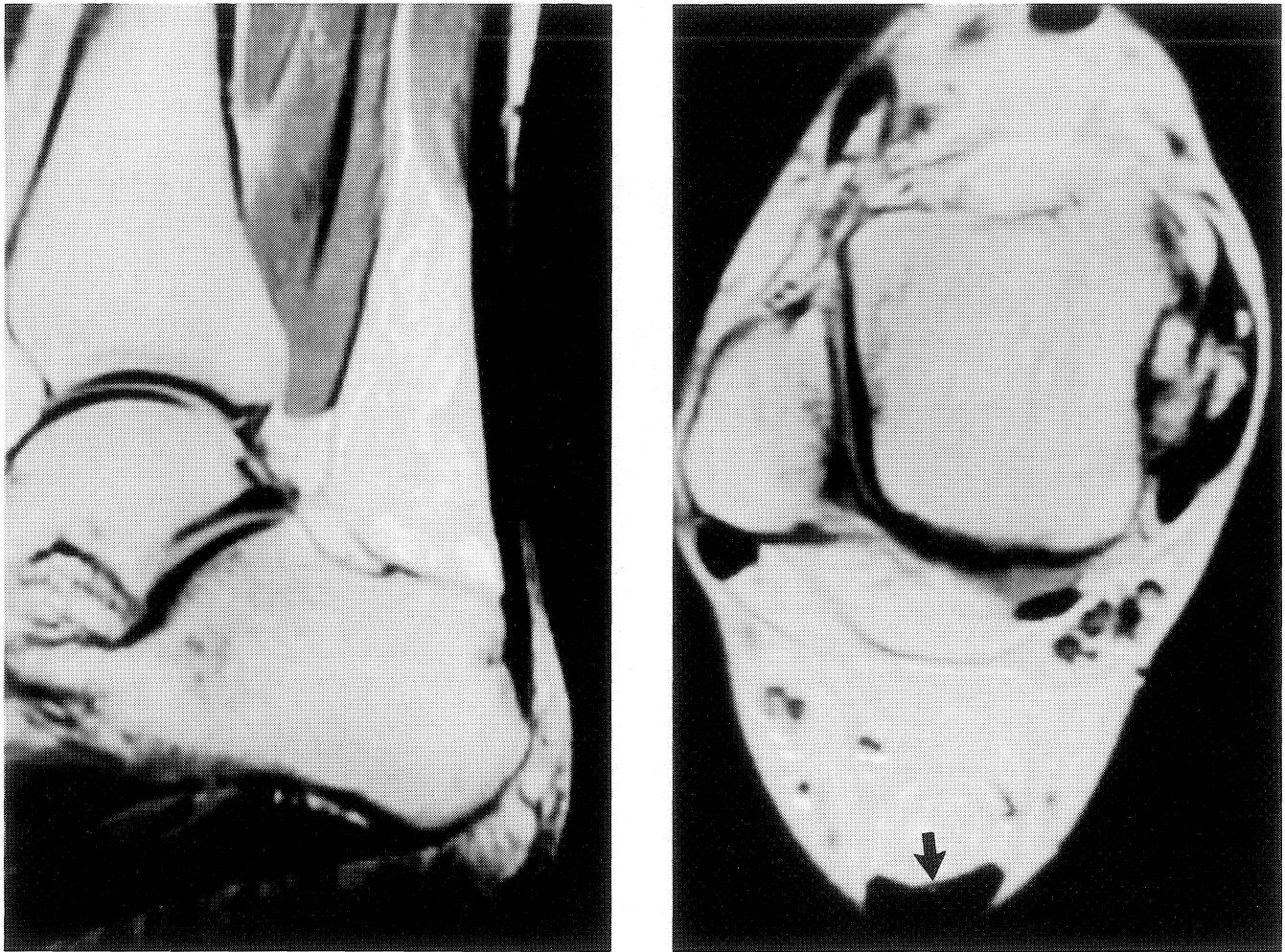


Figure 1: Normal Achilles Tendon.

A: Sagittal proton density (TR = 2000 msec, TE = 20 msec) image demonstrates uniform tendon thickness, lack of intra-substance signal and sharp tendon margins. B: Axial proton density (TR = 2000 msec, TE = 20 msec) image shows the normal crescentic shape, concave anteriorly (arrow).

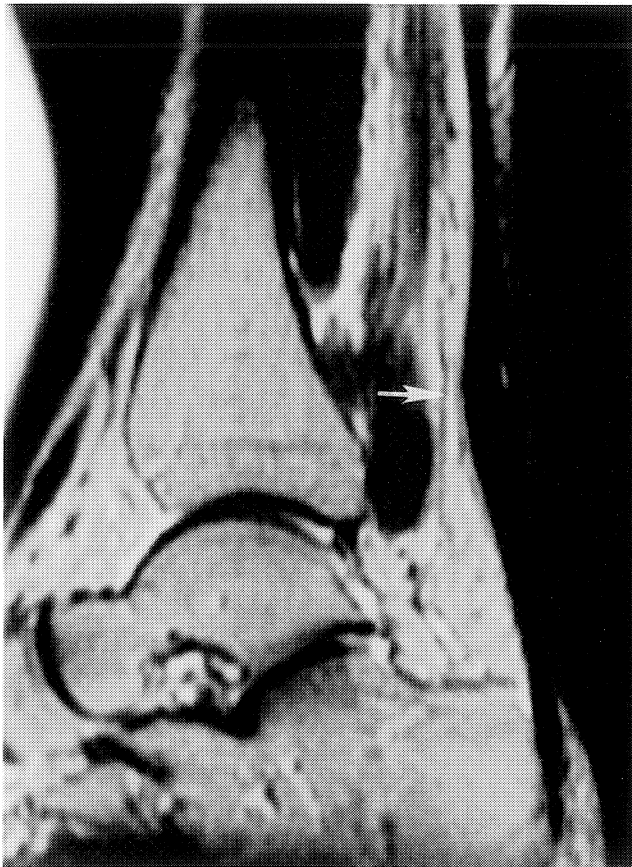
Achilles tendon is roughly crescentic in shape; it has a convex posterior surface and mildly concave or flat anterior surface. Patients with tendinosis show fusiform thickening of the tendon with or without intra-substance increase in signal intensity due to longitudinal splitting and mucoid degeneration (Figure 2). In complete rupture, the tendon becomes discontinuous, and associated areas of increased signal intensity on T2-weighted images are seen due to edema and hemorrhage<sup>14</sup> (Figure 3).

It is not clear from the literature whether conservative therapy or operative treatment is more effective for acute ruptures of the Achilles tendon<sup>2,18</sup>. With either treatment, some tendon remodeling occurs, but persistent tendon thickening is observed even at 12 months after the injury<sup>20</sup>. MRI studies of tendons treated operatively often

show persistent intra-tendinous fluid collections which can complicate interpretation in patients in whom infection is suspected<sup>20</sup>.

#### POSTERIOR TIBIAL TENDON

The posterior tibial tendon is one of the main stabilizers of the hindfoot. It inverts and plantar flexes the foot and actively supports the medial longitudinal arch. The tendon primarily inserts at the navicular tuberosity, with fibers extending to insert on the plantar surface of the cuneiforms and base of the second, third and fourth metatarsals. Partial tearing and degeneration can be of variable severity. Complete rupture presenting as an acquired flat foot deformity is most commonly seen in females in their 5th and 6th decade. Tendon rupture may also be seen in young athletes engaged in sports requiring rapid



**Figure 2: Partial rupture of Achilles tendon.** T2-weighted (TR=2000 msec, TE=20msec) sagittal image demonstrates fusiform swelling in watershed zone of tendon with abnormal intra-substance signal (arrow).

changes in direction<sup>15</sup>. Twenty-five percent of the ruptures occur at its navicular insertion, but the rupture occurs more commonly due to mechanical erosion behind the medial malleolus<sup>10</sup>. With partial tears of the tendon, MRI shows thickening of the tendon and possible longitudinal splitting. Fluid will be present in the surrounding tendon sheath (Figure 4). Tendon sheath fluid, however, is commonly seen in asymptomatic patients, especially within the flexor tendons, so fluid alone is not a reliable sign of tendon damage<sup>25</sup>. Complete rupture on MRI is seen as discontinuity of the tendon with an empty tendon sheath (Figure 5). There is a high association between posterior tibial tendon rupture and the presence of an os tibiale externum (accessory navicular)<sup>24</sup>.



**Figure 3: Complete Achilles tendon rupture.** Sagittal T1-weighted (TR=600 msec, TE=20 msec) image shows marked thickening of the tendon, increased signal and complete disruption five centimeters proximal to the tendon insertion.

### ANTERIOR TIBIAL TENDON

The anterior tibial tendon inserts on the first metatarsal and medial cuneiform bones. It acts as a dorsiflexor and inverter of the foot. As it crosses the ankle joint, it is the most medial tendon. This tendon is much less commonly injured than the foot flexors possibly because: (1) it primarily functions in an unloaded state (during swing phase of gait), (2) its course is fairly straight, and (3) it has a better blood supply than the flexor tendons. There are few cases of anterior tibial tendon disruption reported in the literature. Rupture of this tendon is typically the result of a laceration, but spontaneous ruptures have been reported<sup>21</sup>. This generally occurs at the level of the inferior extensor retinaculum. Patients with tibialis anterior tendon rupture present with foot drop and are often misdiagnosed as having a neurological problem<sup>2</sup>. With MRI, ruptures of the tendon are best demonstrated in the sagittal plane. The tendon will be discontinuous with some retraction of the proximal tendon and an empty tendon sheath (Figure 6). In the acute setting, associated hemorrhage may be present.

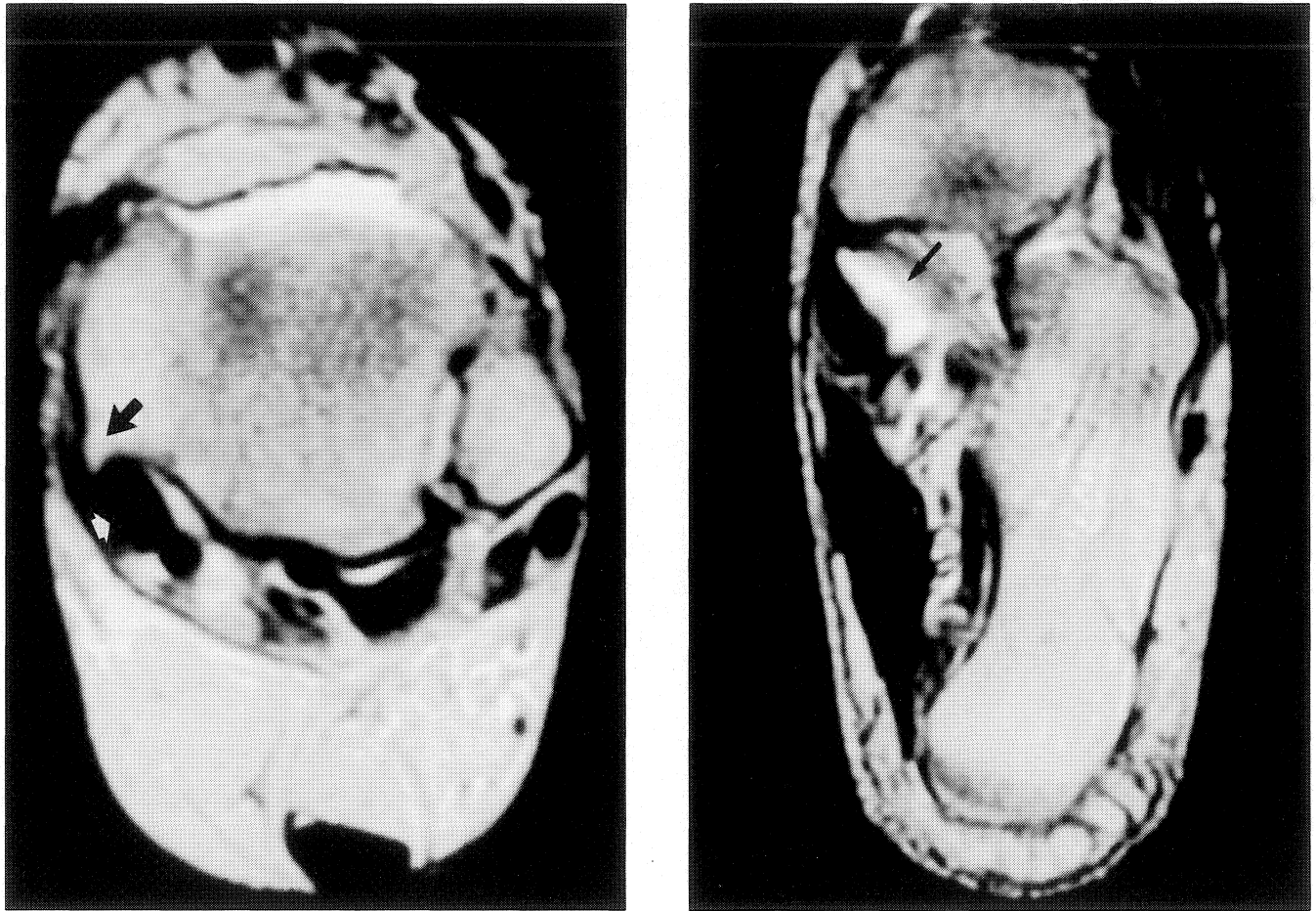
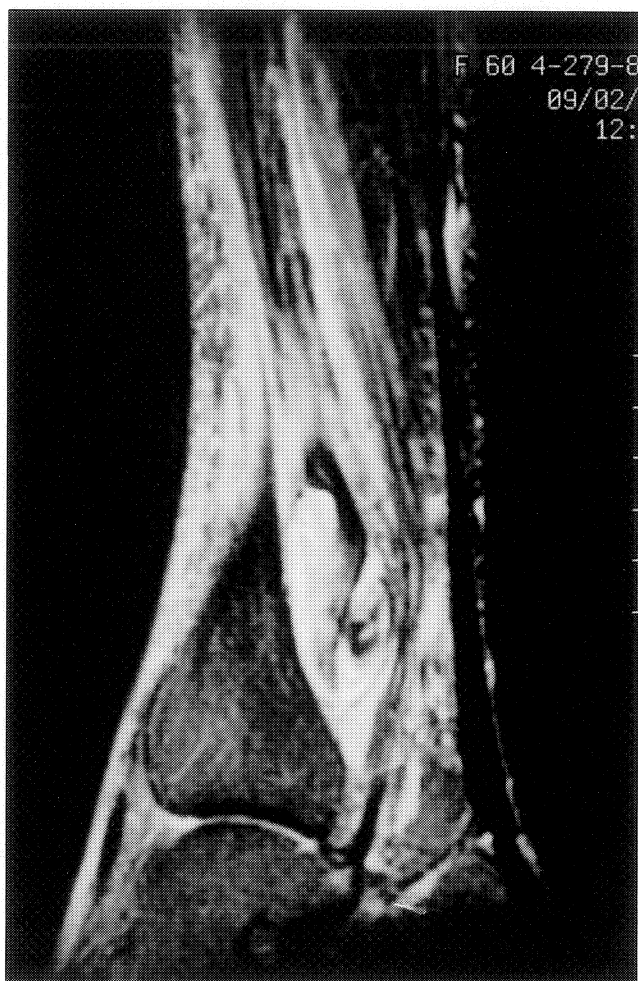


Figure 4: Tibialis posterior tendonopathy.

A: Axial T2-weighted (TR=3000 msec, TE=102 msec) image of ankle shows enlargement of tibialis posterior tendon (white arrow). Note spur off posterior aspect of tibia (black arrow). B: Axial T2-weighted (TR=3000 msec, TE=102 msec) image distally demonstrates tendon sheath distended by fluid but that the tendon is intact (arrow).



**Figure 5: Complete tear of tibialis posterior tendon.**  
A & B: Sagittal and axial T2-weighted (TR=4000 msec, TE=95 msec) images show retraction and balling up of the disrupted tendon.



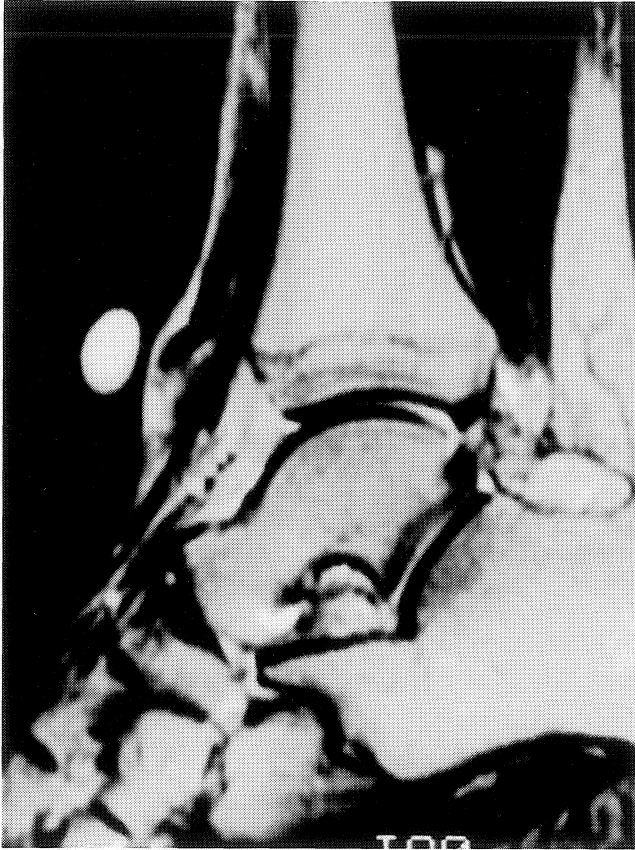


Figure 6: Tibialis anterior tendon rupture.

Sagittal T2-weighted (TR = 2000 msec, TE = 90 msec) image shows disruption of tendon. Figure 7: Normal peroneal tendons. Axial proton density (TR = 2000 msec, TE = 20 msec) image demonstrates peroneus brevis (black arrow) medial and anterior to peroneus longus (white arrow) as they travel around the distal fibula.

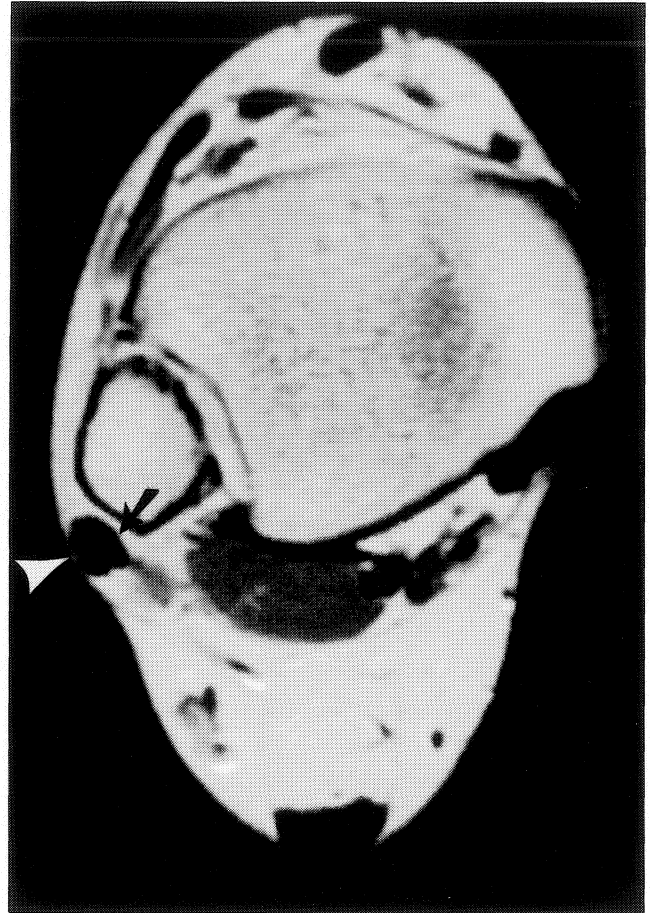


Fig. 7: Normal peroneal tendons.

Axial proton density (TR = 2000 msec, TE = 20 msec) image demonstrates peroneus brevis (black arrow) medial and anterior to peroneus longus (white arrow) as they travel around the distal fibula.

### PERONEUS LONGUS AND BREVIS TENDONS

The peroneus longus and brevis function as dynamic stabilizers of the lateral ankle and hindfoot. The peroneal muscles pronate and evert the foot<sup>9,22</sup>. The tendons are most prone to injury and subluxation where they course around the lateral malleolus (Figure 7)<sup>14</sup>. The peroneus longus tendon courses posterior and lateral to the peroneus brevis tendon at the lateral malleolus, travelling in the peroneal groove, restrained by the superior peroneal retinaculum. Subluxation or dislocation is the most common injury to the peroneal tendons, often seen with lateral

malleolar fractures or severe ankle sprains originally described in skiers. Surgical exploration usually reveals a capacious superficial peroneal retinaculum, with lifting of the periosteum off the lateral malleolus<sup>26</sup>. Partial tearing and degeneration typically occurs in the peroneus brevis and is seen on MRI as tendon thickening and longitudinal splitting<sup>26</sup> (Figure 8). Progression to tendon attenuation and complete tear is also seen in more severe cases, but only a few cases are described in the literature.

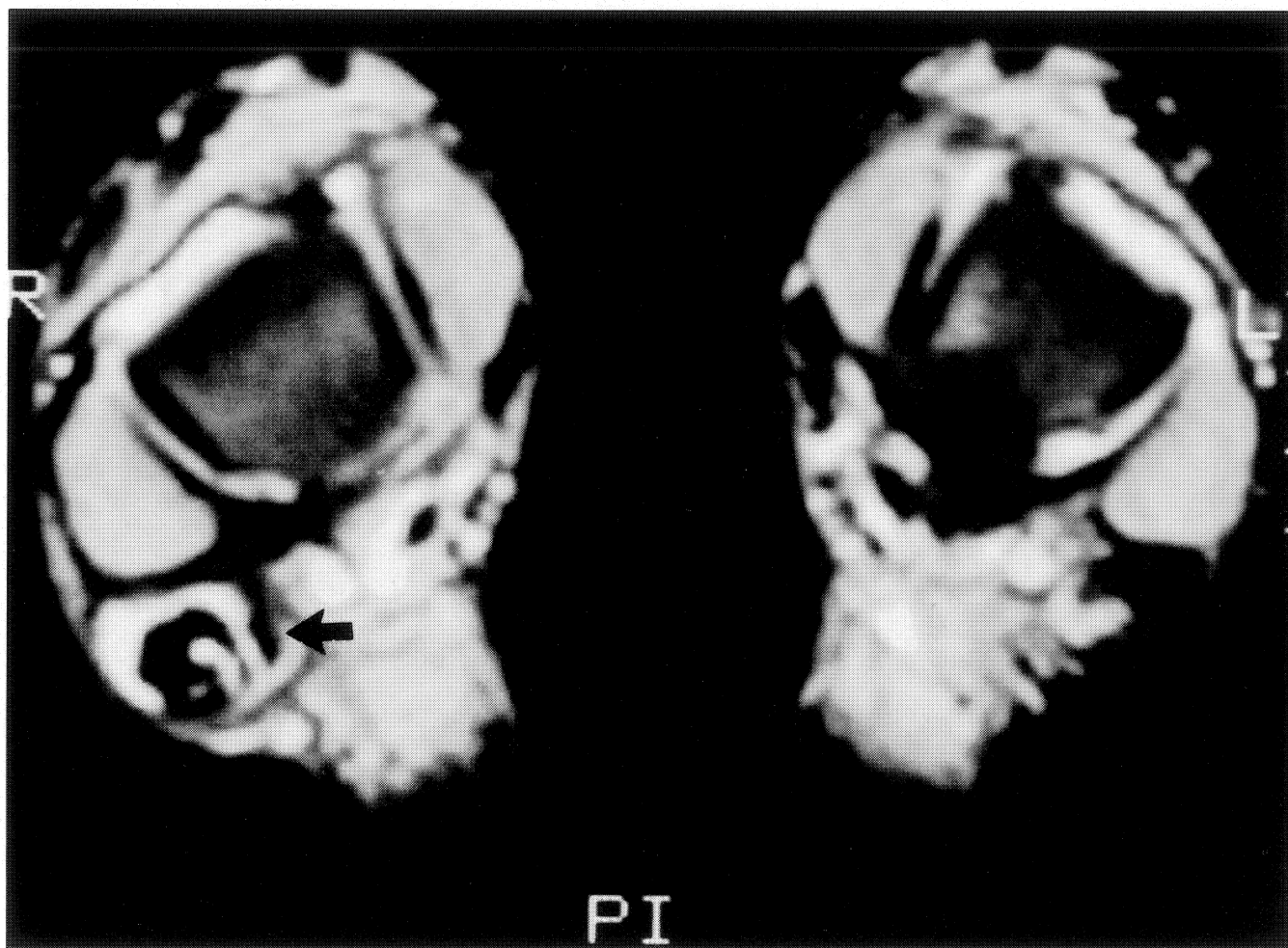


Figure 8: Peroneus brevis longitudinal splitting.

A: Axial T2-weighted (TR=4000 msec, TE=95 msec) image shows thickening of the right peroneal tendons (arrow) when compared to the normal left side. Intra-tendon signal is present.



Figure 8 B: Sagittal T1-weighted (TR=350 msec, TE=16 msec) image demonstrates the longitudinal splitting (arrows).



Figure 8 C: Intraoperative photograph confirms longitudinal splitting of peroneus brevis.

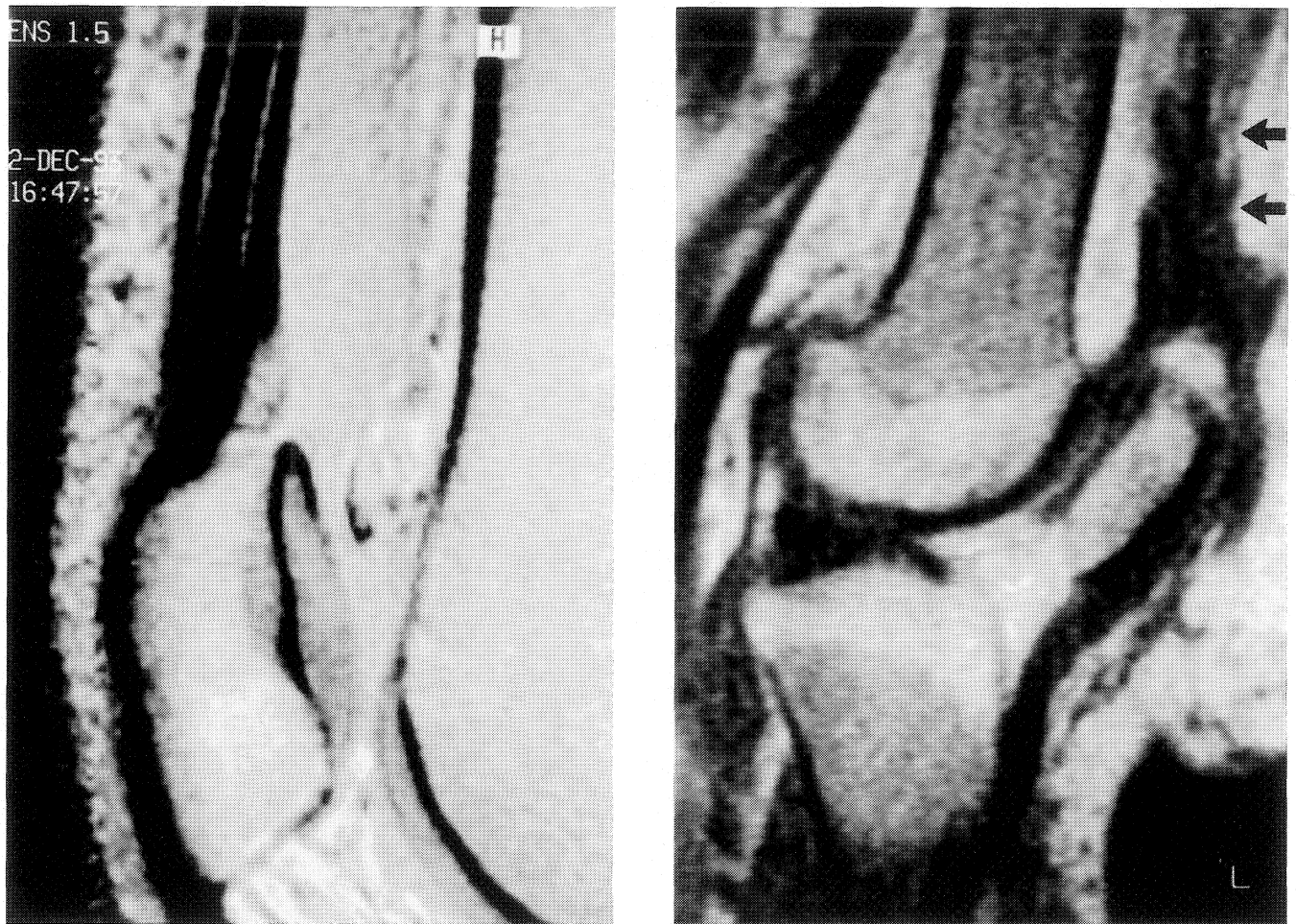


Figure 9: Quadriceps muscles.

A: T2-weighted (TR=2950 msec, TE=90 msec) sagittal image of the knee shows the normal striated appearance of the quadriceps muscles.  
 B: 39 year old woman on long term steroids has disruption of the quadriceps muscles (arrows) with tilting and inferior retraction of the patella on sagittal T1-weighted (TR=516 msec, TE=20 msec) image.

### QUADRICEPS MECHANISM

The quadriceps mechanism includes the rectus femoris, vastus lateralis, vastus medialis, vastus intermedius, medial and lateral retinacula, as well as the patella and patellar ligament. It does not insert focally onto the tibial tubercle; rather, it inserts as a broad band onto the anterior surface of the tibia. Trauma resulting in an isolated tear of the quadriceps tendon or patellar ligament or fracture of the patella will not result in complete loss of extension because the expansions of the medial and lateral retinacula will support the traumatized structure and maintain some degree of function. The normal quadriceps mechanism does not rupture under stress. However, the quadriceps mechanism can rupture even with minor stress when

weakened by muscle attrition in the elderly, fatty infiltration in obese individuals, or in association with one of several systemic diseases<sup>16,17</sup>.

Injury to the quadriceps tendon and associated structures of the quadriceps mechanism such as the retinacula can be evaluated with MRI. Sagittal and axial images are most useful for detecting injuries in this region. The quadriceps mechanism normally has a striated appearance, typically showing three layers<sup>29</sup> (Figure 9). MRI findings in quadriceps tendon tears parallel those seen in the patellar ligament, but may be associated with inferior retraction of the patella rather than superior retraction as may be seen with complete rupture of the patellar ligament.

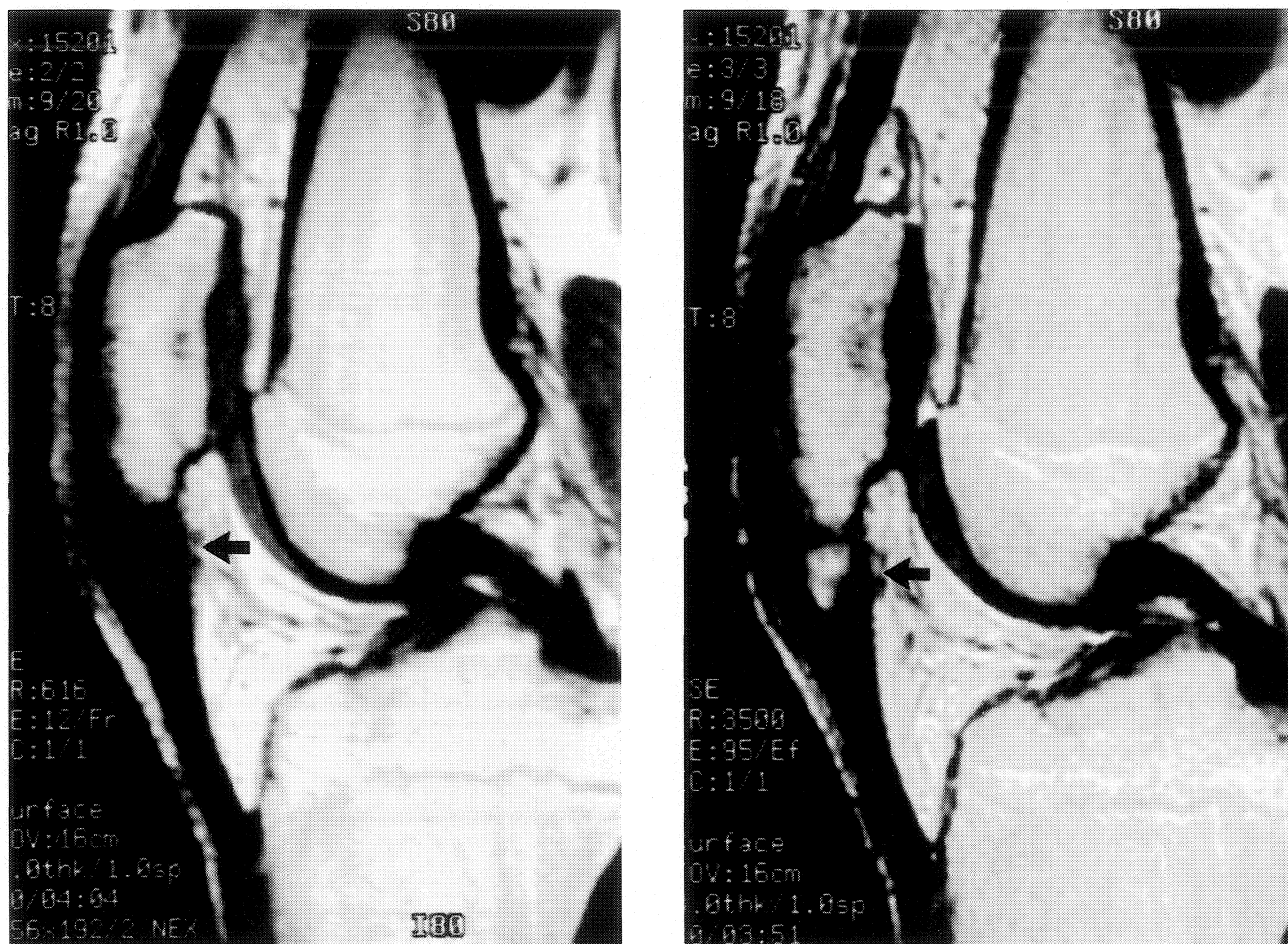


Figure 10: MRI of patellar tendonitis.

A & B: T1-weighted (TR=616 msec, TE=12 msec) and T2-weighted (TR=3500 msec, TE=95 msec) sagittal images of the knee show increased thickness and signal of proximal patellar ligament with indistinct posterior margin (arrow).

### PATELLAR LIGAMENT

The patellar ligament rarely ruptures in healthy individuals. Rupture usually is the end stage of partial tearing, often called patellar tendinitis or jumper's knee, which results from the cumulative effect of repetitive trauma and microtearing of the patellar ligament. Radiographic findings are often unremarkable but may show loss of the cortical margin and irregularity of the inferior pole of the patella early in the disease. Later findings on plain radiographs include elongation of the inferior pole of the patella.

It is possible to evaluate the patellar ligament on standard MR images of the knee. The normal patellar ligament has uniformly low signal intensity on T1-weighted, T2-weighted and proton density images; it

increases slightly in anteroposterior diameter from proximal to distal, and displays distinct margins. The AP diameter of a normal tendon, in its proximal portion, should not exceed 7 mm. Females generally have thicker proximal tendons than males<sup>5</sup>. In patellar tendinitis or partial tearing, the ligament may show increased signal intensity on T1-weighted, T2-weighted and proton density images; it also shows increased AP diameter proximally and the margins of the affected ligament become indistinct, especially posterior to the thickened segment<sup>5</sup> (Figure 10). With complete rupture, the ligament is totally separated from the inferior pole of the patella and it is easy to diagnose clinically and to confirm by MRI.

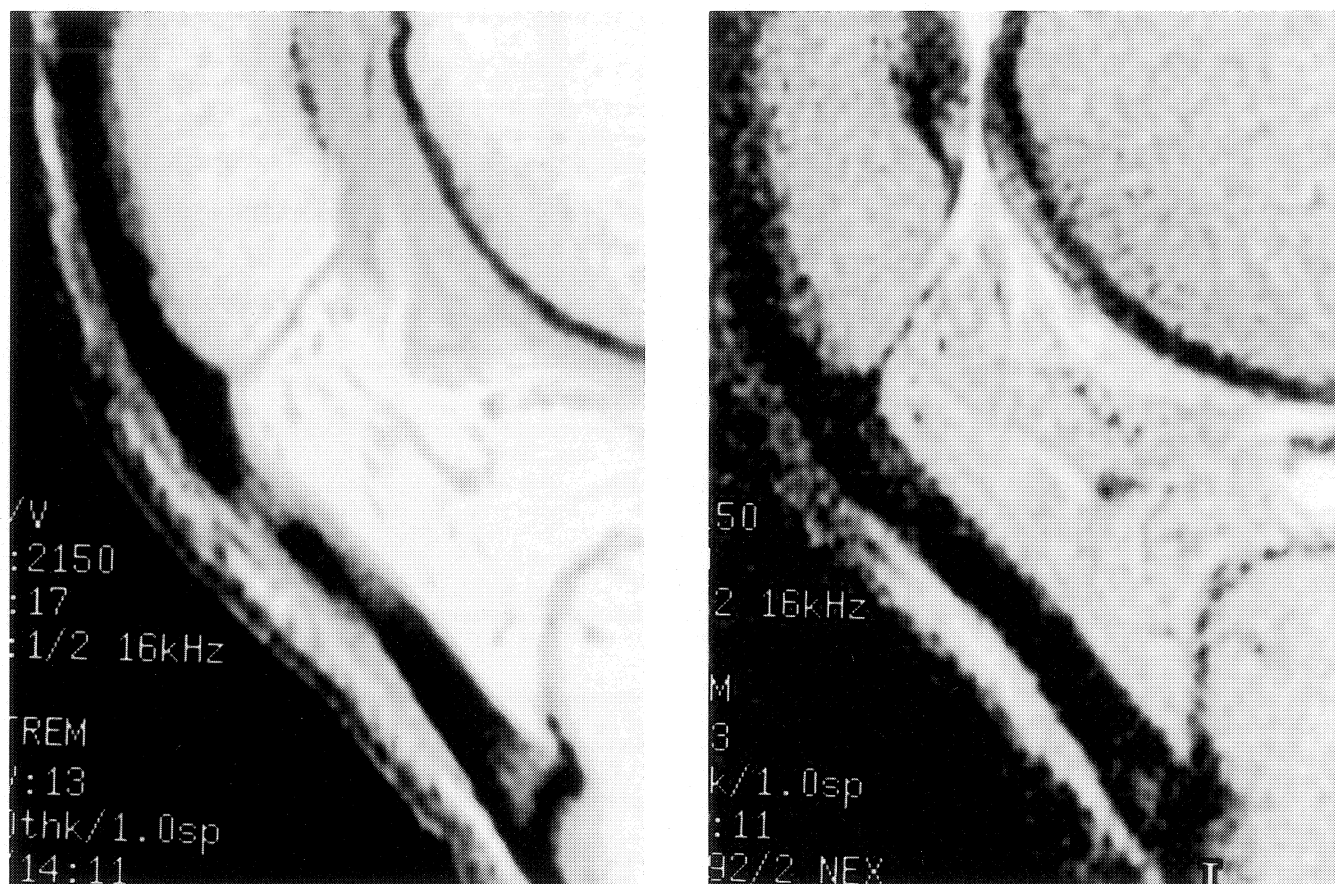


Figure 11: "Magic Angle" effect.

A: Proton density (TR=2150 msec, TE=1750 msec) image of patellar tendon has mottled increased signal throughout. B: T2-weighted (TR=2150 msec, TE=80 msec) image is normal.

One must be aware of the "magic angle" effect when evaluating any ligament or tendon because it could simulate a tear. When tendons are oriented at 55 degrees from the main magnetic field, tendons can have increased signal on short TE (echo time) sequences, such as T1-weighted and proton density sequences. The effect is diminished at longer TE, so that T2-weighted images will not show increased signal<sup>6</sup> (Figure 11).

### ROTATOR CUFF

The rotator cuff is made up of the tendons of the subscapularis, supraspinatus, infraspinatus and teres minor muscles. The subscapularis inserts on the lesser tuberosity while the other tendons insert on the greater tuberosity. The supraspinatus tendon inserts most superiorly on the greater tuberosity. There is a hypovascular zone 2 cm proximal to the insertions of the tendons, called the critical zone. Most tears occur in this location.

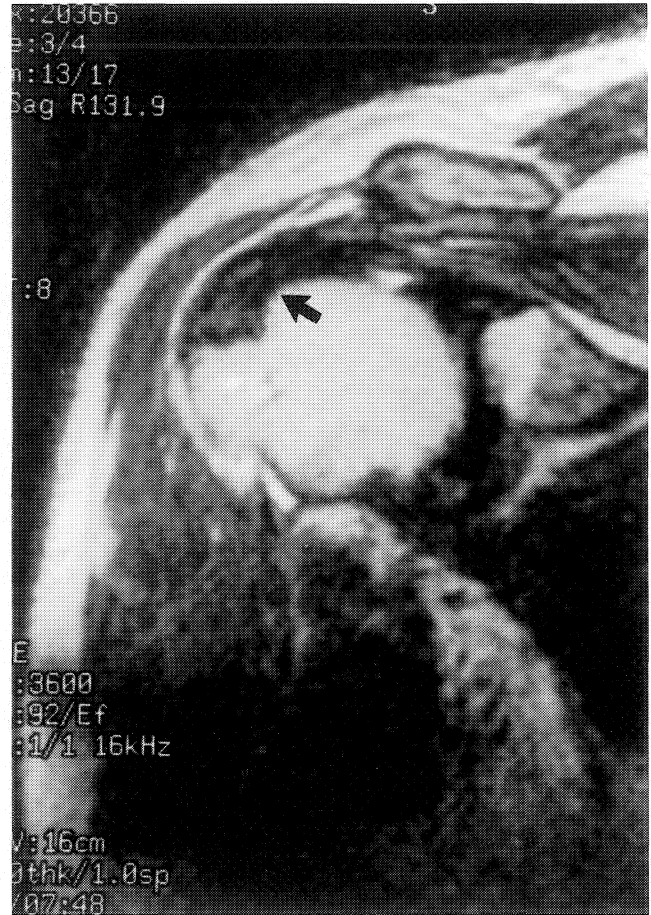


Figure 12: Rotator cuff tendonopathy.

A & B: Coronal T1-weighted (TR = 600 msec, TE = 17 msec) and T2-weighted (TR = 3600 msec, TE = 92 msec) images show abnormal increased signal of supraspinatus tendon (arrows).

Shoulder pain is often related to problems with the rotator cuff tendons. In younger patients, overuse, such as with overhead sports, can cause tendonopathy<sup>28</sup> (Figure 12). Anatomic variants of the acromion and degenerative changes at the acromioclavicular joint have been implicated as well in the impingement syndrome<sup>4</sup>. This

tendonopathy can progress to partial tears or complete disruption of the tendons of the rotator cuff, which most commonly involve the supraspinatus tendon. Complete rotator cuff tear is more commonly seen in patients over the age of 50, and it is usually due to tendon degeneration, thinning and, finally, attrition<sup>4,28</sup> (Figure 13).

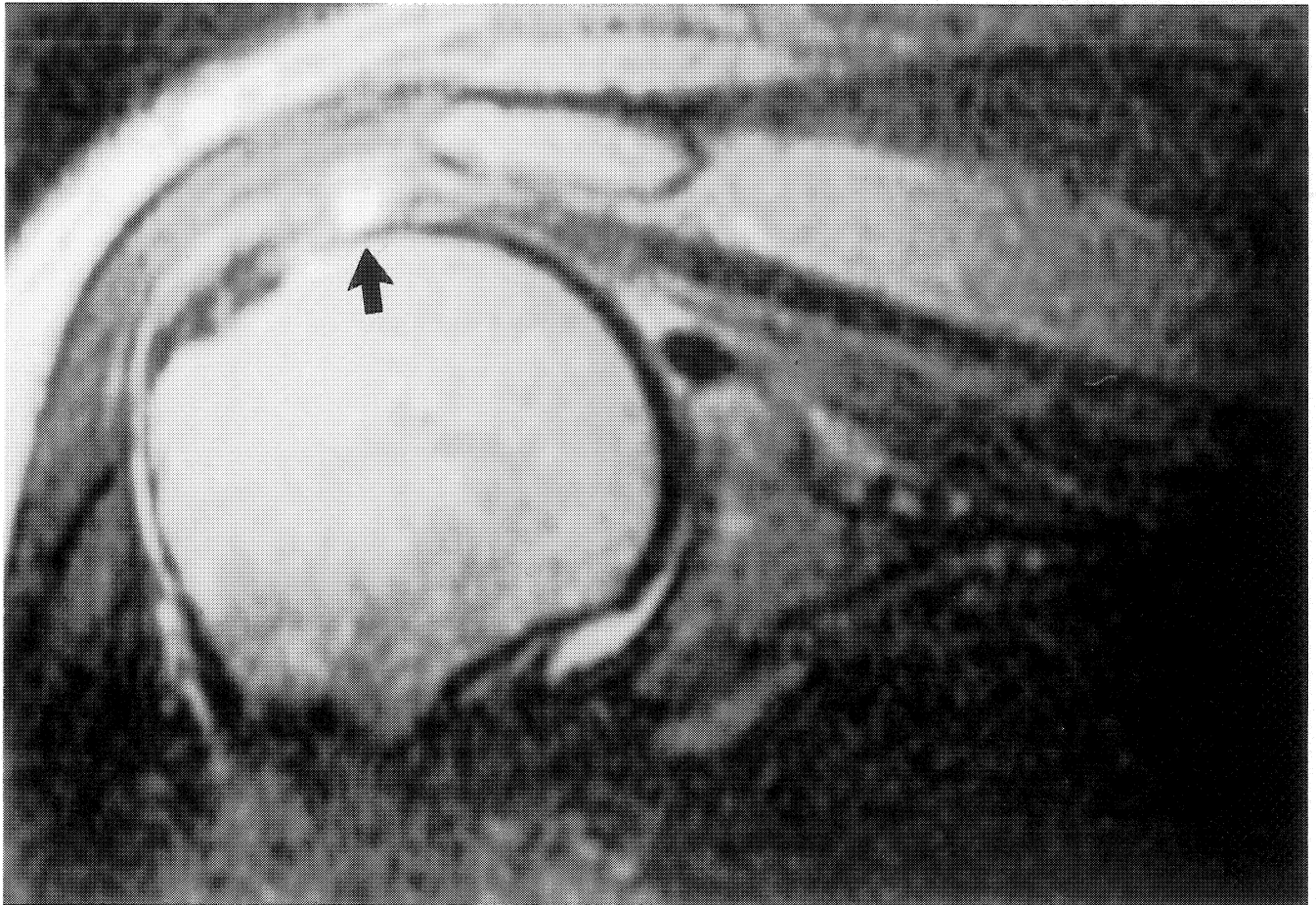


Figure 13: Complete rotator cuff tear.

Coronal T2-weighted (TR=2000 msec, TE=80 msec) image shows full thickness increased signal within supraspinatus tendon (arrow) in a patient with rotator cuff tear.

### TENDON OF THE LONG HEAD OF THE BICEPS BRACHII

While the short head of the biceps brachii muscle originates at the coracoid process, the long head originates at the superior aspect of the glenoid and labrum. It has an intra-articular course within the glenohumeral joint and curves around the lesser tuberosity to run in the bicipital groove. Proximally, this groove is covered by a number of structures, including pectoralis major, the transverse humeral ligament, and slips from the subscapularis tendon

which help to prevent medial subluxation or dislocation of the tendon<sup>7</sup>. Damage to these structures can be associated with medial subluxation or dislocation of this tendon. This is well demonstrated on coronal and axial MR images which show the tendon positioned medially, anterior to the glenoid<sup>4</sup> (Figure 14). Absence of the tendon from its expected position in the proximal bicipital groove is best visualized in the axial plane. Rupture of the long head of the biceps tendon can also be easily diagnosed by MRI, but is usually a straightforward clinical diagnosis.



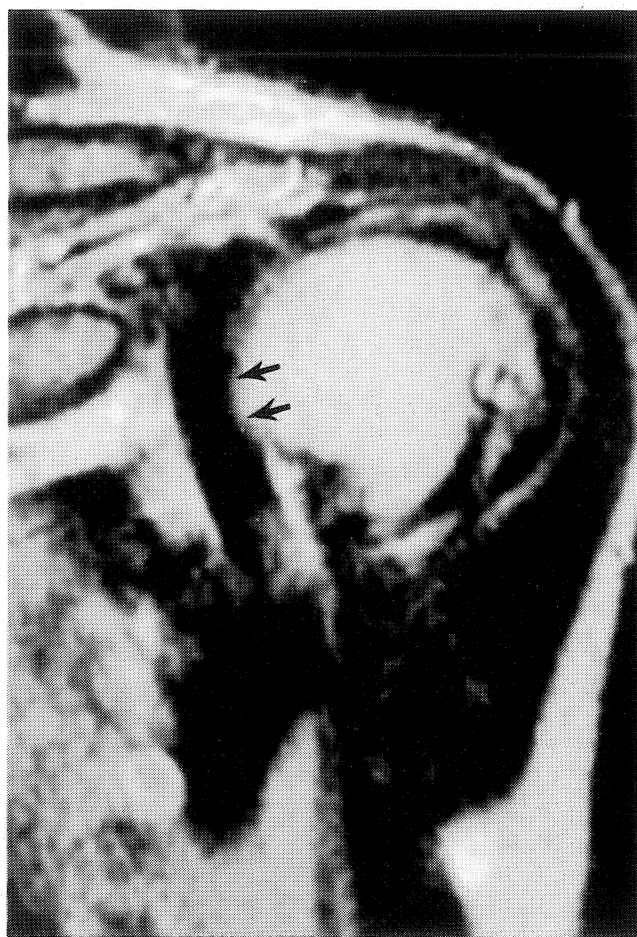


Figure 14: Dislocation of tendon of long head of biceps brachii.

A: Axial T1-weighted (TR=500 msec, TE=11 msec) image with empty bicipital groove (arrow). The tendon is dislocated medially (arrowhead), anterior to the glenoid labrum. B: Coronal T2-weighted (TR=2000 msec, TE=90 msec) scan shows medial dislocation as well (arrows).

### HAMSTRING MUSCLES

The hamstring muscles consist of the semimembranosus, semitendinosus, and the biceps femoris. These muscles originate at the ischial tuberosity as a conjoined tendon. Distally, the semitendinosus courses medially as one of the tendons of the pes anserinus. The semimembranosus inserts mainly on the posteromedial tibia 1 to 2 cm distal to the medial plateau. The biceps femoris inserts laterally on the fibular head. Tendon injuries in this group almost always occur in track athletes, during strenuous activity. In skeletally immature patients, the ischial apophysis can be avulsed. In the healing phase, this area can have florid new bone formation, simulating neoplasm<sup>8</sup>. In adults, the ischial tuberosity is spared but the conjoined tendon ruptures (Figure 15). MRI readily demonstrates the avulsion, best seen with coronal and axial images. Distally, the biceps femoris insertion avulses, pulling a bone fragment off the fibular head as well<sup>27</sup> (Figure 16). Typically, this occurs in association with acute trauma.

### CONCLUSION

The diagnosis and treatment of tendon problems constitute an important part the orthopedist's practice. As more and more people engage in athletic activities, there will continue to be injuries to tendons throughout the body. We are increasingly able to accurately characterize these injuries. MRI has revolutionized the imaging of tendons, aiding in prompt diagnosis and guiding treatment algorithms. MRI continues to advance with new pulse sequences and techniques, and promises to continue to allow better characterization of the soft tissues of the musculoskeletal system.



Figure 15: MRI of hamstring conjoined tendon avulsion. A: Coronal T1-weighted (TR = 683 msec, TE = 20 msec) image with normal conjoined tendon attaching to left ischial tuberosity (arrow) and avulsed right conjoined tendon (arrowhead).

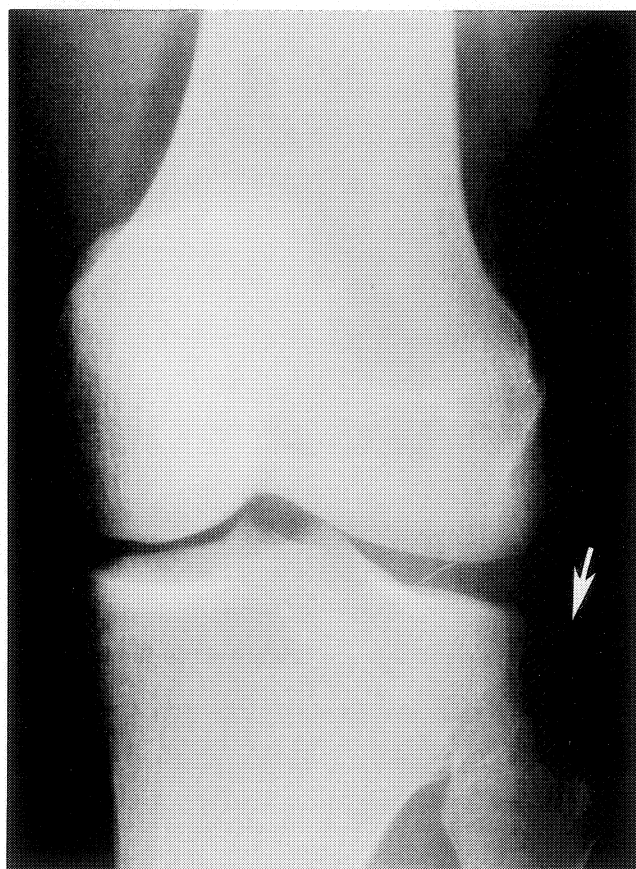


Figure 16: Biceps femoris avulsion. A: Internal oblique radiograph with fibular head avulsion and retraction of bone fragment (arrow). B: T1-weighted (TR = 500 msec, TE = 12 msec) coronal MR image shows that the biceps femoris tendon is attached to the retracted fragment of bone (curved arrow). El-Khoury, Brandser, and Saltzman El-Khoury, Brandser, and Saltzman

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# SPORTS FRACTURES

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## ABSTRACT

Fractures occur in athletes and dramatically influence performance during competitive and recreational activities. Fractures occur in athletes as the result of repetitive stress, acute sports-related trauma and trauma outside of athletics. The literature provides general guidelines for treatment as well as a variety of statistics on the epidemiology of fractures by sport and level of participation. Athletes are healthy and motivated patients, and have high expectations regarding their level of function. These qualities make them good surgical candidates. Although closed treatment methods are appropriate for most sports fractures, an aggressive approach to more complicated fractures employing current techniques may optimize their subsequent performance.

## INTRODUCTION

There are three general categories of fractures that occur in athletes including fatigue fractures, acute sports-related fractures, and fractures that occur outside of sports<sup>2,3,7,12,16,17,18,21,23,26,31,39,44,45</sup>. Nearly half of the articles in the literature on sports fractures deal with fatigue (stress) fractures. Acute fractures that occur during sports result from directly overloading bone (e.g. boot top tibial shaft fractures in skiing) or from ligament or tendon avulsion (e.g. tibial tubercle avulsion in long jumping). Injuries sustained outside of sports (e.g. open tibial shaft in car wreck) also occur in athletes. Their optimal treatment is influenced by the patient's athletic demands. This article will review the epidemiology of sports fractures. The general principles of diagnosis and treatment of fatigue fractures and traumatic fractures are presented and illustrated by common examples.

## EPIDEMIOLOGY

Three hundred thousand sports fractures occur in the U.S. each year<sup>47</sup>. An estimated 38,160 fractures per year

occur in high school football<sup>38</sup>. The fracture rate in skiers has been reported to be 0.64 per 1000 skier days<sup>33</sup>. The overall incidence of sports fractures in athletes is 0.12% per year<sup>34</sup>. Stress fractures comprise 10% of all sports injuries and 15% of all injuries to runners<sup>25</sup>. The majority of stress fractures occur in runners<sup>35</sup>.

Table 1 lists the distribution of fatigue fractures in athletes. Fatigue fractures tend to follow a sport-specific distribution pattern (Table 2). The level of competition also affects the distribution of fractures. In competitive athletes the tibia is most often injured, while in recreational athletes the metatarsals and pelvis are more likely to be injured<sup>15</sup>.

**Table 1. Distribution of fatigue fractures in bone scan-positive athletes<sup>25,34</sup>**

tibia	49.1%
tarsals	25.3%
metatarsals	8.8%
femur	7.2%
fibula	6.6%
pelvis	1.6%
sesamoids	0.9%
spine	0.6%

**Table 2. Common sites of fatigue fracture according to sport**

Sport	Bones Fractured
Running	tibia, fibula, metatarsal <sup>11,43,46</sup>
Ballet	tibia, metatarsal <sup>27</sup>
Football	fifth metatarsal <sup>30</sup>
Basketball	tibia, fibula, patella, tarsal navicular <sup>20,40</sup>
Soccer	tibia, fibula, patella <sup>29,48</sup>
High jump	tibia, fibula, patella <sup>29,48</sup>
Tennis	ulna <sup>28</sup>
Softball	ulna <sup>28</sup>
Volleyball	ulna <sup>28</sup>

Age is also a factor. The proximal tibial metaphysis accounts for the majority of fatigue fractures in skeletally immature athletes. The radiographic appearance of these injuries is often concerning. The intense periosteal reaction may suggest a more aggressive process like neoplasia. However, young patients respond relatively quickly to treatment and the healing time is generally shorter than for adults.

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Pediatric athletes are at greater risk of acute fracture in sports than adults. Athletes between the ages of 8-11 and 14-18 years have twice the fracture risk and 11-14 year old athletes have five times the risk of fracture compared to all other ages<sup>36,50</sup>. The adolescent's maximum fracture incidence occurs at or shortly before the period of maximum growth velocity<sup>1,4</sup>. One theory accounting for these observations is that mineralization lags behind longitudinal growth, resulting in weaker bone.

Acute fractures in athletes are found to have a sport-specific distribution pattern (Table 3). The cause of some fractures is related to acute stress placed by participation (e.g. humerus fractures in baseball pitchers), while the cause of other fractures is not so intuitively obvious (metacarpal fracture in cross country skiing). Sports with higher rates of acute fractures tend to involve higher energy activities (downhill skiing).

**Table 3. Common sites of acute fracture according to sport**

Sport	Bones Fractured
Downhill skiing	tibia, fibula, metacarpals <sup>22</sup>
Cross country skiing	fifth metacarpal <sup>3</sup>
Snowboarding	radius, ulna, tibia, fibula, humerus, metacarpals <sup>37</sup>
Weight lifting	scaphoid <sup>40</sup>
Baseball	humerus <sup>6</sup>
Softball	humerus, phalanges, metacarpals <sup>6,14</sup>
Arm wrestling	humerus <sup>32</sup>
Soccer	talus, scaphoid <sup>19,40</sup>

### CATEGORIES

Over half of the articles on sports fractures in the literature relate to fatigue failure from repetitive stress. Fractures occur when bone is subjected to a number of cycles of repetitive loading that exceeds the fatigue threshold. Bone is a dynamic tissue, and is much less apt to fail by fatigue than inert structures because it remodels in response to applied loads. The deposition of new bone in high stress regions occurs relatively slowly and decreases, but does not eliminate, the risk of fatigue failure. Athletic performance is enhanced by training to build strength, endurance and coordination. Effective training regimens typically include thousands of repetitive cycles of applied load. For example, 2.5 million cycles of 3 times the body weight are applied to the metatarsals of an athlete running 10 miles per day for 3 months<sup>13</sup>.

Variables that increase the risk of fatigue fractures include an abrupt increase in the number of cycles or amount of load, (e.g. going from 3 miles per day to 10 miles per day), lower limb malalignment, nutritional deficits, and endocrine abnormalities (e.g. amenorrhea). Fa-

tigue fractures can effectively be prevented by well designed graduated training programs, orthotics, nutritional counseling, and correction of underlying metabolic or endocrinologic disorders. For example, a regimen of alternating days of marching in military recruits dramatically reduced the incidence of fatigue fractures<sup>42</sup>, as did marching in running shoes instead of boots<sup>15</sup> and the use of shock absorbing insoles<sup>41</sup>.

Acute fractures result from directly overloading bone and from ligament or tendon avulsions. The usual mechanism of injury of an avulsion fracture is a sudden violent muscle contraction or a sustained muscle contraction across an open apophysis. Avulsion injuries are common in the younger athlete, occurring primarily between the ages of fourteen and twenty-five<sup>51,52</sup>. Tendinous avulsion fractures typically occur in soccer and football players, sprinters and jumpers<sup>9</sup>. Football finger has been used to describe an avulsion fracture of the flexor digitorum profundus from the ring finger distal phalanx that occurs during tackling. Ligament avulsion fractures can occur when a sudden load is applied to a joint. Five percent of knee ligament injuries are actually bony avulsions. The anterior cruciate ligament, posterior cruciate ligament, medial collateral ligament, and lateral collateral ligament can avulse fragments of the bone from their insertions<sup>15</sup>. Avulsions of the lateral tibia plateau (a lateral capsular sign) from posterolateral complex injuries have also been reported, especially in combination with ACL tears<sup>55</sup>. The slower the rate of loading of a ligament complex, the greater the chance of avulsion fracture<sup>9</sup>. Physeal avulsion fractures occur in skeletally immature patients due to the relative weakness of the surrounding bone to shear stresses (e.g. iliac crest, tibial tubercle)<sup>50</sup>.

Fractures which occur outside of sports, e.g. from a motor vehicle accident, affect the athlete as much as sports-related injuries<sup>10,50</sup>. Short term problems common to all fractures in athletes include stiffness and pain, which can prevent training and competition. The long term complications of fractures, nonunion, malunion and degenerative joint disease may be devastating to an athlete but are fortunately uncommon. Immobilization and subsequent disuse atrophy are more frequently observed, and may interrupt athletic participation for weeks to months after the fracture has healed. In light of limited years of eligibility, every effort should be made to minimize the deconditioning associated with fracture treatment.

### TREATMENT

A physician managing fractures in athletes should take into consideration the type of sport and level of involvement. An elite athlete may require more aggressive treatment than a recreational athlete in order to return to their previous level of function<sup>24</sup>. The goals of treatment should be thoroughly discussed with the athlete in order to

improve compliance and outcome. A timely return to participation must be balanced against risk of refracture and other potential long term complications. Operative treatment which delays rather than accelerates return to competition (e.g. thumb ulnar collateral ligament repair in a football lineman) is not well accepted by athletes. The goals of the athlete should be considered in making treatment recommendations. When possible, consideration should be given to end of season surgery. Many athletes attempt to return to sports before adequate bone healing or soft tissue reconditioning has occurred, increasing the risk of reinjury<sup>53</sup>. This underscores the importance of employing treatments which minimize immobilization time and hasten rehabilitation. Athletes differ from the general fracture population in that they are usually younger and in better health. Their risk of operative complications is less than the general population and their high motivation make them relatively good surgical candidates<sup>22</sup>.

There are special considerations for athletes with fractures when operative treatment is chosen. Extremes of physical size are commonly encountered, and require additional preoperative planning. For example, standard size intramedullary nails will be inadequate when treating tall basketball players and petite woman gymnasts with very narrow medullary canals. Due to high loads during competition, load sharing fracture fixation is preferred to minimize the risk of subsequent fracture related to stress shielding. Implant removal is recommended after completion of a phase of eligibility or at the end of the first full season of competition one year after injury.

### CONCLUSION

Fractures in athletes are common and sport-specific. Their injuries should be approached from a different perspective than those that occur in the general population. Athletes have different demands and goals. Recommended treatment should minimize immobilization time and allow early return to conditioning and competitions.

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# POPLITEUS FUNCTION IN ACL-DEFICIENT PATIENTS

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## SUMMARY

Anterior cruciate ligament (ACL) injuries commonly result in anterolateral instability, resulting in a "pivot shift" phenomenon. Given that popliteus muscle stimulation results in a pivot shift, others have postulated that the popliteus muscle has a role in the pivot shift phenomenon. We hypothesized that patients with instability from ACL injuries may have excessive popliteus muscle activity. Therefore, we studied the EMG activity (using fine wire electrodes) of the popliteus muscle in sixteen normal subjects and ten ACL-deficient subjects. We recorded the EMG in six activities (level walking and jogging, ascending walking and jogging, and descending walking and jogging). Pearson's Product Moment Correlations were above 0.7, except in the case of ascending the treadmill ( $r=0.427$ ) and ascending jogging ( $r=0.645$ ), suggesting that the timing of the signals was similar for injured and uninjured limbs. Variance ratios for the injured and uninjured limbs were statistically similar, suggesting similar variability of patterns. Thus, we observed only minor popliteus EMG signal differences in this group of patients. We conclude that the popliteus muscle does not contribute to instability in the studied activities.

## INTRODUCTION

Anterior cruciate ligament (ACL) injuries commonly result in anterolateral instability (i.e., anterior subluxation of the lateral tibial plateau). Such subluxation occurs frequently during athletic activities and can be elicited on clinical exam, i.e., the "pivot shift" phenomenon<sup>6,7,8</sup>. Peterson et al.<sup>20</sup> demonstrated that electrical stimulation of the popliteus muscle (an internal rotator of the tibia) causes an "active pivot shift" in ACL-deficient patients, and reported one patient who could voluntarily contract his popliteus muscle (documented by EMG) and elicit the pivot shift phenomenon. They therefore postulated a role of the popliteus in patients with this problem.

In order for the popliteus muscle to cause a pivot shift, there must be either 1) activity of increased magnitude but normal timing, 2) activity of normal magnitude but abnormal timing (including abnormal duration), or 3) activity of normal or decreased magnitude in the presence of structural instability and/or reduced antagonistic activity. This study was designed to evaluate the first two possibilities.

## METHODS AND MATERIALS

The study was conducted with 16 normal subjects (15 males and 1 female) and 10 ACL-deficient patients (6 males and 4 females). The ACL injury must have occurred more than one year previously for inclusion. No patients had reconstructive knee surgery performed, although four patients had diagnostic arthroscopy. Two had a complete and one had a partial medial meniscectomy. Prior to recording the EMG data, the study patients completed the Cincinnati rating system questionnaire<sup>19</sup> and underwent a standard knee examination, torque measurements on a strength-testing machine (Lido Active, Loredan Biomedical Inc., Davis, CA), and drawer measurements using an instrumented stability testing device (KT-1000)<sup>9</sup>. Each physical examination was performed by the same physician. During the exam range of motion, effusions, alignment, and crepitus were recorded. The results of Lachman's, anterior drawer, posterior drawer and pivot shift tests were also noted. Inclusion criteria for the ACL-deficient group were a positive anterior drawer sign, Lachman test, and pivot shift test. All KT-1000 measurements were performed by the same individual (MW). KT-1000 testing was performed at 30° of knee flexion with a twenty pound force. Subjects with greater than 3 mm more varus or valgus laxity compared to the contralateral knee were excluded.

We recorded the activity of the medial hamstring, lateral hamstring, vastus medialis, vastus lateralis, medial gastrocnemius, lateral gastrocnemius, and the popliteus muscles bilaterally. Popliteus activity was recorded with two

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fine wire electrodes (fifty micron Teflon coated stainless steel) inserted into the muscle with a twenty-seven gauge needle<sup>3</sup>. The distal 1.5 mm of each wire was exposed and the distance between the exposed tips was approximately one mm. The site of insertion was located at the base of the medial tibial flare, approximately eight cm below the medial joint line. Placement of the electrodes into the popliteus muscle belly was performed by inserting the needle just posterior to the medial edge of the tibia, directing it toward the fibular head, and advancing it three cm into the tissue<sup>3</sup>. The exposed tips of the electrodes were barbed which allowed removal of the placement needle without displacing the electrodes. The exposed external wire endings were connected to a preamplifier located adjacent to the electrode site. Placement of electrodes was confirmed two ways. First, seated subjects with knees flexed 90 degrees and feet flat on the floor were instructed to internally then externally rotate their feet. With proper placement, the EMG showed consistent and constant activity while rotating the foot inward, and consistent inactivity while rotating the foot outward<sup>2,4</sup>. Second, toe raising activated the gastrocnemius muscles while the popliteus remained silent.

We used bipolar surface electrodes with a built-in preamplifier to collect the EMG signals of the other muscles. The electrodes were made of eight mm diameter silver discs whose centers were separated by twenty mm. After establishing the site of electrode placement<sup>4</sup> the skin was shaved if necessary, cleansed with alcohol, and dried. Symmetrical placement of electrodes was assured by measurement of distance from the knee joint line and by visual inspection. Electrodes were placed over the above-mentioned muscle bellies on both legs. Double adhesive-backed tape was used to attach the surface electrodes to the skin and electrode gel was used to fill the wells in the tape over the silver discs, allowing EMG signal transmittance. Once all electrodes were in place, the leg was lightly wrapped with self-adhering bandages to protect the electrodes and cables from inadvertent displacement. Pressure sensitive foot switches, were taped to the bottom of the subjects shoes. The foot switches allowed accurate recordings of heel and toe contact in order to define the gait cycle.

Each subject was instructed to walk on a treadmill at a self-selected comfortable walking pace and a comfortable heel-to-toe jogging pace. At each speed, measurements were taken during level walking, a seven degree incline and an eight degree downhill grade. The subjects were allowed to become comfortable on the treadmill (two to five minutes) before data collection began. Data was accumulated over several ten second trials until a total of twenty to thirty cycles was obtained for each exercise. To insure that any potential differences between EMG re-

cordings were not owing to alterations in speed between groups, we determined differences in speeds for the two sets of subjects with a non-parametric, unpaired, two-tailed test of significance (Mann-Whitney).

A small sixteen channel junction box (fourteen EMG channels and two foot switches channels) was strapped to the subjects' back. A ten meter cable connected the box to an amplification console containing an oscilloscope and an interface to a laboratory computer.

Surface and fine wire electrode signals were pre-amplified ( $A = 100$ ) by a low bias current (2-pA), low noise (8 nV-Hz-1/2) JFET differential amplifier contained in each electrode assembly. The console provided additional amplification ( $10 < A < 1000$ ), and bandpass filtering (20 to 500 Hz) by cascaded low-pass/high-pass filters with a Butterworth response. This conditioned AC EMG signal was converted to a linear envelope by a true RMS-to-DC converter that had an averaging circuit time constant of twenty-five mSec. The resulting EMG envelopes were digitized by a twelve-bit A/D converter in the laboratory computer, sampled at one hundred Hz per channel, and stored on disk.

A semi-automated interactive sorting procedure detected all heel strike events for both left and right sides. Successive heel strikes for a given side were used to mark the beginning and ending of individual gait cycles. Each individual gait cycle was normalized to one hundred time points through linear interpolation<sup>11</sup>. The amplitudes of these one hundred time points were then normalized to the maximum amplitude observed in that muscle throughout the testing session. We excluded from subsequent analysis cycles for which heel strike could not be precisely identified (occasionally heel strike was ambiguous as when the subject scuffed the heel).

Ensemble averaged envelopes (with standard deviations) for each muscle from individual subjects were computed from all included cycles (typically twenty to twenty-five with a minimum of twelve) at each of the one hundred points. Individual ensemble averages were excluded from further statistical analysis when the standard deviations were large at times when the mean was near zero. Such signals generally reflect a high noise to signal ratio on individual cycles or spurious individual cycles with a high, constant baseline. We also eliminated individual ensemble averages of the popliteus when a portion closely resembled that of the medial gastrocnemius (i.e., crosstalk). "Grand" ensemble averages were then created for each muscle and activity using the ensemble averages for all subjects.

The degree of symmetry was established using two measures: Pearson's Product Moment correlation coefficient "r" and variance ratios. The former measure was used by Arsenault et al.<sup>1</sup> in their study of symmetry. The

variance ratio was reported by Hershler and Milner<sup>10</sup> as a statistical descriptor for repeatability of EMG patterns; we used a slightly modified formulation suggested by Kadaba et al.<sup>11</sup> as a measure of day-to-day constancy (agreement) of surface EMG signals. Pearson's "r" values provide a measure of profile shape similarity (concurrence of peaks and valleys) between two cycles. As adapted from Hershler and Milner<sup>10</sup>, the variance ratio reflects the variation in subject-to-subject ensemble averages with larger ratios suggesting greater variability. Pierotti, et al.<sup>21</sup> argued that EMG pattern comparisons resulting in a high correlation coefficients and low variance ratios are indeed similar profiles.

The statistical differences between variance ratios of the injured and uninjured limbs for all activities were determined with a nonparametric, paired, two-tailed test of significance (Wilcoxon signed rank test).

### RESULTS

The normal subjects walked at a speed of  $3.8 \pm 0.2$  m/sec while the injured subjects walked  $3.5 \pm 0.3$  m/sec ( $p=0.10$ ). The normal subjects level and uphill jogging speed averaged  $7.5 \pm 0.9$  m/sec and the injured subjects  $6.9 \pm 1.0$  m/sec ( $p=0.33$ ).

In the ACL-deficient patients, the time since initial injury averaged  $9.2 \pm 5.4$  years, with a range from one to 15.5 years. Scores from the Cincinnati Rating Scale averaged  $76 \pm 11.6$ , with a range from 55-94. When these patients were asked to score their knee function based upon a percentage function, their responses averaged  $71\% \pm 21\%$ , ranging from 35% to 97%. The displacements with the hand-held stability testing machine averaged  $11.7 \text{ mm} \pm 3.2$  on the injured side (range 5.7 to 15.0 mm) and  $5.3 \text{ mm} \pm 2.3 \text{ mm}$  (range 2.0 to 9.5 mm) on uninjured limbs.

In the normal subjects, a biphasic peaked envelope characterized the popliteus muscle for all activities (Figure 1-4). A short burst occurred just after toe-off and a longer burst at the swing-to-stance transition. We observed this pattern in more than 90% of individual envelopes. Jogging effected an earlier second peak (Figure 2). Variability, as reflected in the variance ratios ranged from 0.362-0.595.

Grand ensemble averages of the EMGs generally revealed minor discrepancies between injured and normal extremities (Figures 1-4) for most activities. Both injured and control limbs exhibited the burst just after toe-off and swing-to-stance phase transition activity for all activities.

Pearson's Product Moment correlations for the uninjured and injured sides were 0.767 for level walking (Figure 1), 0.809 for level jogging (Figure 2), 0.427 for ascending walking (Figure 3), 0.645 for ascending jogging, 0.785 for descending walking (Figure 4), and 0.812 for descending jogging. In ascending walking, the toe-off bursts in the injured limbs were substantially smaller than

in the normal limbs (Figure 3). The timing and duration of the two peaks for all activities remained similar for uninjured and injured suggesting that the timing of contraction of the popliteus remained normal in the ACL-deficient patient, but that the force of contraction might be reduced during some activities.

The variance ratios tended to be lower ( $p=0.06$ ) for the injured side for five of the six activities (Figures 1-3), except in descending walking when the unaffected limb registered a slightly lower (i.e., 0.454 vs. 0.469) average variance ratio (Figure 4).

### DISCUSSION

Some reported evidence, as well as intuition, suggests that the popliteus muscle might play a role in the pivot shift phenomenon in ACL-deficient patients. Our study, suggests that if this is the case, it is not due to popliteus hyperactivity.

Most studies of the popliteus muscle infer its function from its origin and insertion<sup>2,13,14</sup>. Applications of fine wire EMG have made it possible to study the muscle's activity; however, only "on-off" determinations have been conducted for this muscle in the normal gait cycle<sup>3,16,23</sup>. "On-off" signals reflect nothing about pattern differences, and even the results of timing differences are subject to an arbitrary choice of threshold activity.

Before considering our data, we must note several limitations common to our study and other similar studies. First, crosstalk is always a potential problem while studying a small muscle that it is surrounded by larger muscles<sup>15</sup>. The only true ways to designate which part of the envelope represents cross-talk are to denervate surrounding muscles or to electrically isolate the muscle, neither of which is possible in a clinical setting. Cross-talk can be reduced by introducing a high-pass filter, since at a given electrode site the high frequency content of adjacent muscles is attenuated relative to the high frequency content of the muscle of interest. However, low frequency information of interest may be lost by doing this. Another approach is to simultaneously record the patterns of the larger surrounding muscles. By then overlapping the envelope of the muscle being studied to those envelopes of nearby muscles, gross cross-talk can be identified and the data excluded. An example of this may be observed in our data by comparing the popliteus and medial gastrocnemius envelopes. In some instances, our popliteus envelopes had large relative amplitudes in the ascending activities and had a large spike of activity in stance phase resembling the morphology of the stance phase spike present in the simultaneously recorded medial gastrocnemius envelope. In those subjects who had a stance phase spike and large amplitudes in the ascending activities, activity could be observed in the popliteus lead when the subject performed isolated plantar flexion against resistance. In the other

subjects this activity was not present. This strongly supports the concept of crosstalk, therefore, we excluded any envelopes which contained a spike in mid-stance phase (resembling the simultaneously recorded medial gastrocnemius envelope) and which always had maximum amplitude in the ascending activities.

Second, kinematic differences are present in overground versus treadmill walking<sup>5,18</sup>. However, the envelopes we produced in the medial gastrocnemius, lateral gastrocnemius, medial hamstrings, lateral hamstrings, vastus medialis, and vastus lateralis, were all morphologically similar to published patterns of overground walking<sup>21,22,25</sup>. We therefore suspect that the treadmill does not artificially cause changes these activity patterns, although no previously published EMG envelope of popliteus activity in overground walking are available for comparison.

Third, normalizing magnitudes to the peak activity for each muscle allows only inferences about relative magnitude differences. While it might seem preferable to normalize to maximum voluntary contraction, this is problematic with the popliteus muscle for several reasons. First, it is difficult to instruct a patient to maximally contract the popliteus muscle—most would not know exactly what to do. Furthermore, it is not clinically possible to isolate popliteus function, as the medial hamstrings contribute greatly during active internal rotation of the tibia on the femur. The anatomic location of the popliteus confounds matters even more. Finally, the position or activity in which maximal popliteus EMG occurs is unknown. Thus, in the absence of a means to obtain maximal contraction, we accepted that we could determine only relative differences during the various activities.

Fourth, our fine wire recordings of the popliteus demonstrated greater variability than surface recordings of other muscles<sup>12,21</sup>. This may be due to relative movement of the fine wire tips during locomotion, or to inherently greater variability. We are unaware of any direct comparisons of similarly processed fine wire to surface EMG.

Fifth, most of our patients had adapted satisfactorily to their injury, were relatively functional in recreational pursuits, and had not sought further treatment. A more recently injured and dysfunctional patient population (i.e., consistent with Noyes "law of thirds" categorization of ACL injuries<sup>19</sup>) tends to have more problems with instability. It remains possible that this latter population of patients could exhibit excessive popliteus muscle activity associated with more frequent instability.

Sixth, treadmill locomotion did not engender feelings of instability in our patients, and may not be the best test for excluding the possibility of excess popliteus activity. Abnormal activity may occur only during activities that are capable of eliciting a pivot shift (e.g., cutting). However,

recording EMG in these situations would result in variable patterns and it would be difficult to establish "normal" patterns, i.e. develop reproducible ensemble averages of EMG patterns.

Two studies report popliteus EMG activity during normal gait. Using only "on-off" descriptions, Mann and Hagy<sup>16</sup> observed monophasic popliteus activity just prior to heel strike (91-100 per cent of the gait cycle) and through most of the stance phase (0-57 per cent of gait cycle). In contrast, Stern et al.<sup>23</sup>, reported biphasic popliteus activity characterized by two brief bursts of activity during level walking, one at the moment of heel-strike and a second near toe-off. This second burst at toe-off was present only in some of our subjects. During jogging, they noticed a burst of activity throughout the first half of stance phase often followed by a second period of activity in mid swing phase.

Our data during level treadmill walking is consistent with the study of Stern et al.<sup>23</sup>. When activity representing potential crosstalk was eliminated, the popliteus muscle demonstrated two periods of activity. The longer period of contraction occurred during the swing-to-stance transition, although in some subjects the magnitude of activity was higher in the stance-to-swing transition. However, contrary to the findings of Stern et al., we found biphasic activity during jogging corresponding to the two peaks during level walking. The toe-off spike was located at a lower percentage of the entire gait cycle, corresponding to the decreased stance phase at jogging pace. We agree with the conclusion of both Mann and Hagy, as well as Stern et al., that popliteus activity generally corresponds to the two portions in the gait cycle during which the tibia rotates inwardly relative to the femur<sup>24</sup>.

A possible explanation for the discrepancy in our findings and those of Mann and Hagy is the possibility of cross talk in their study. They found the popliteus active throughout most of stance phase. If the popliteus electrodes detect cross-talk with the medial gastrocnemius lead, this is the pattern to be expected.

In comparing normal popliteus activity to that in ACL-deficient patients, we found only minor discrepancies in most of the six activities. Thus, our results were not consistent with our hypothesis that this group of ACL-deficient patients would exhibit an increase in popliteus activity. However, if functionally significant, the decrease in relative magnitude of the short burst at toe-off in injured limbs may reflect a protective decrease in contraction of the popliteus muscle. Since artificially stimulating the popliteus causes the pivot shift in a passive setting, ACL-deficient patients may reduce popliteus activity to avoid instability. The notion of an adaptive response is supported by in our patients' ability to pursue desired recreational activities without reconstructive surgery.

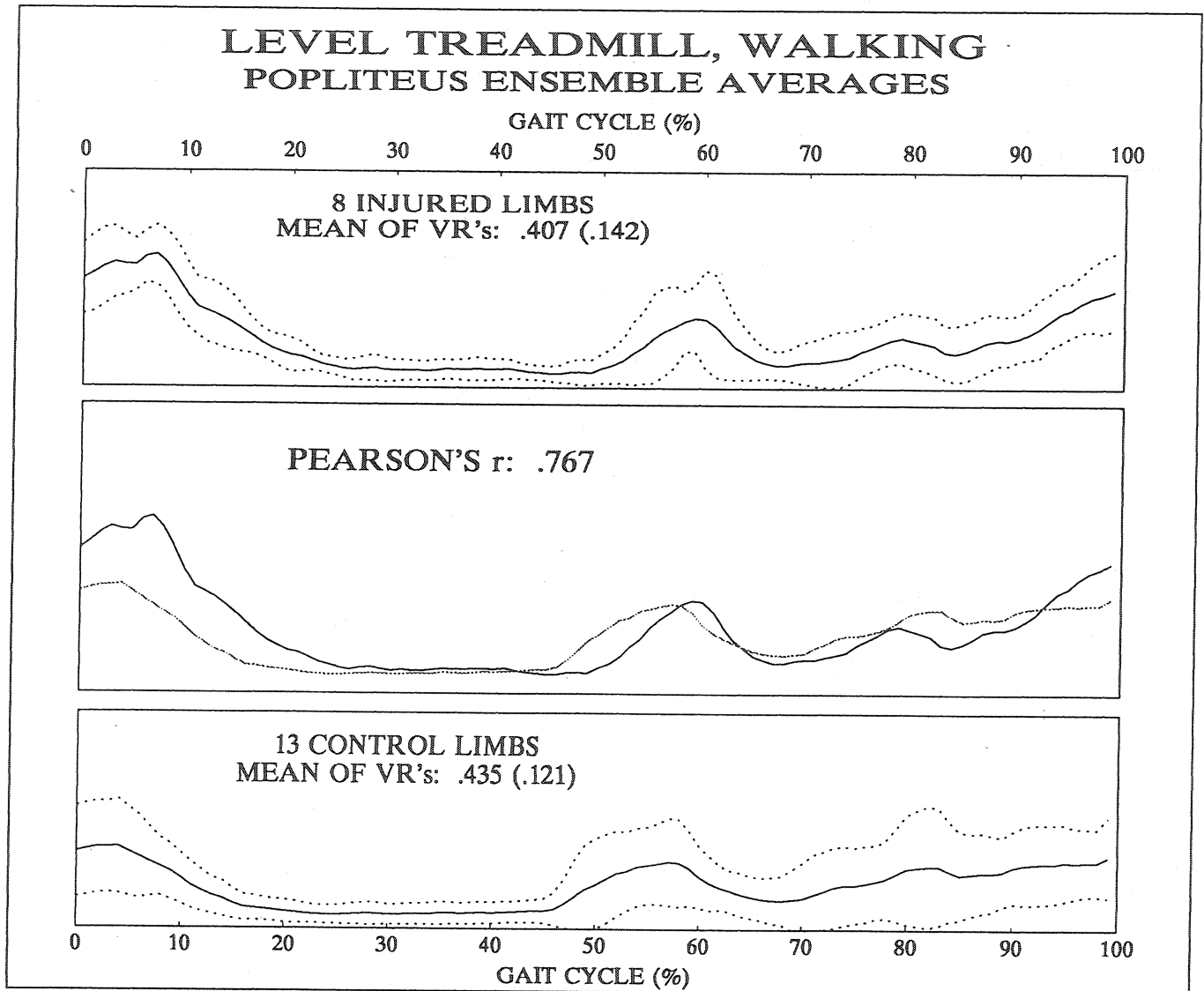


Figure 1.

Grand ensemble average ( $\pm 1$  standard deviation) for level treadmill walking for injured limbs (top window) and control limbs (bottom window); variance ratios shown with  $\pm 1$  standard deviation in parenthesis. Grand ensemble averages for injured and control limbs with Pearson's Product Moment Correlation "r" (middle window). Injured limbs tended to show less activity in late stance and swing, but the lower mean values always fell within the  $\pm 1$  standard deviation range of the control activity.

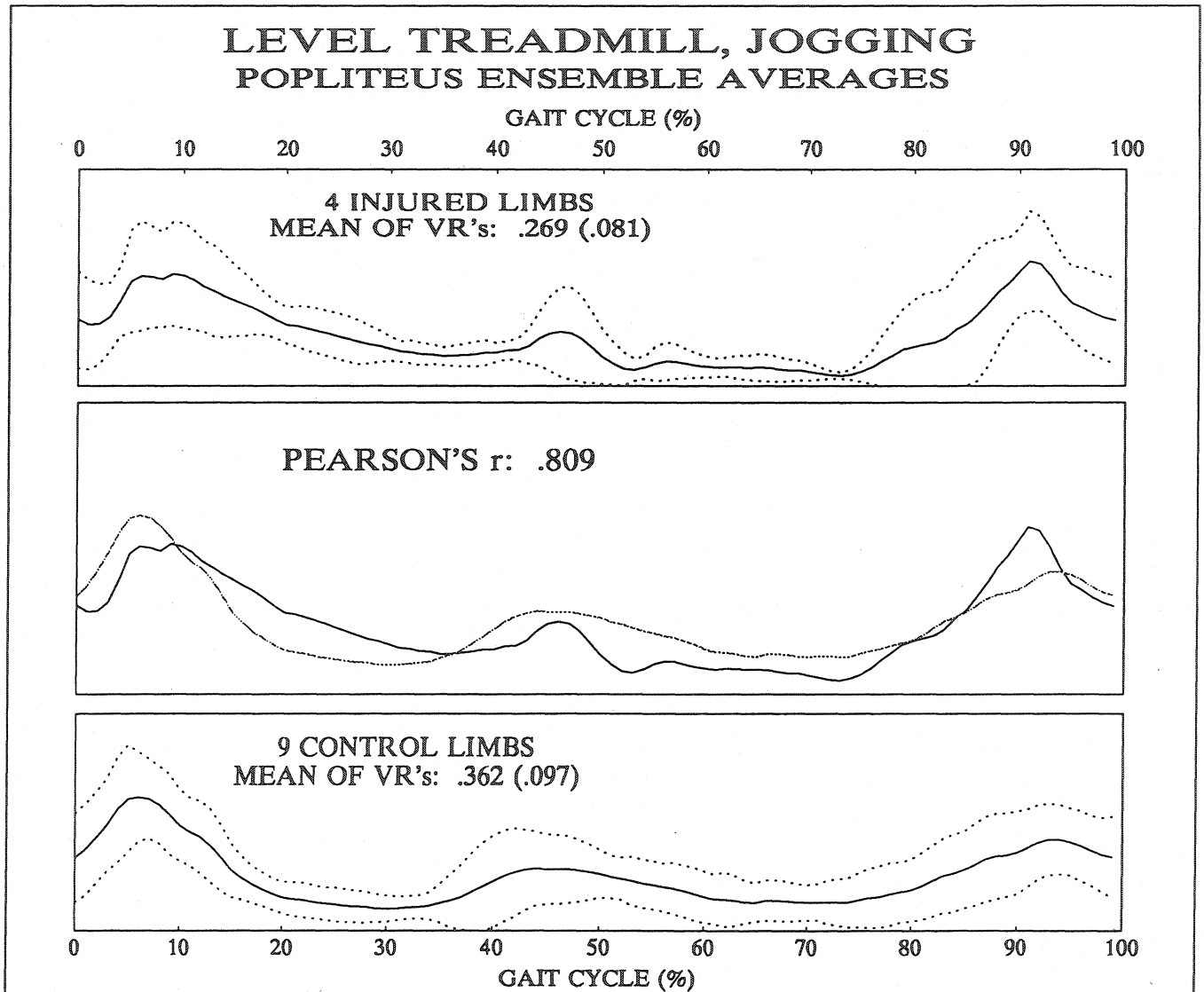


Figure 2.

Grand ensemble average ( $\pm 1$  standard deviation) for level treadmill jogging for injured limbs (top window) and control limbs (bottom window); variance ratios shown with  $\pm 1$  standard deviation in parenthesis. Grand ensemble averages for injured and control limbs with Pearson's Product Moment Correlation "r" (middle window). Injured limbs tended to show more activity in late swing, but the higher mean values always fell within the  $\pm 1$  standard deviation range of the control activity.

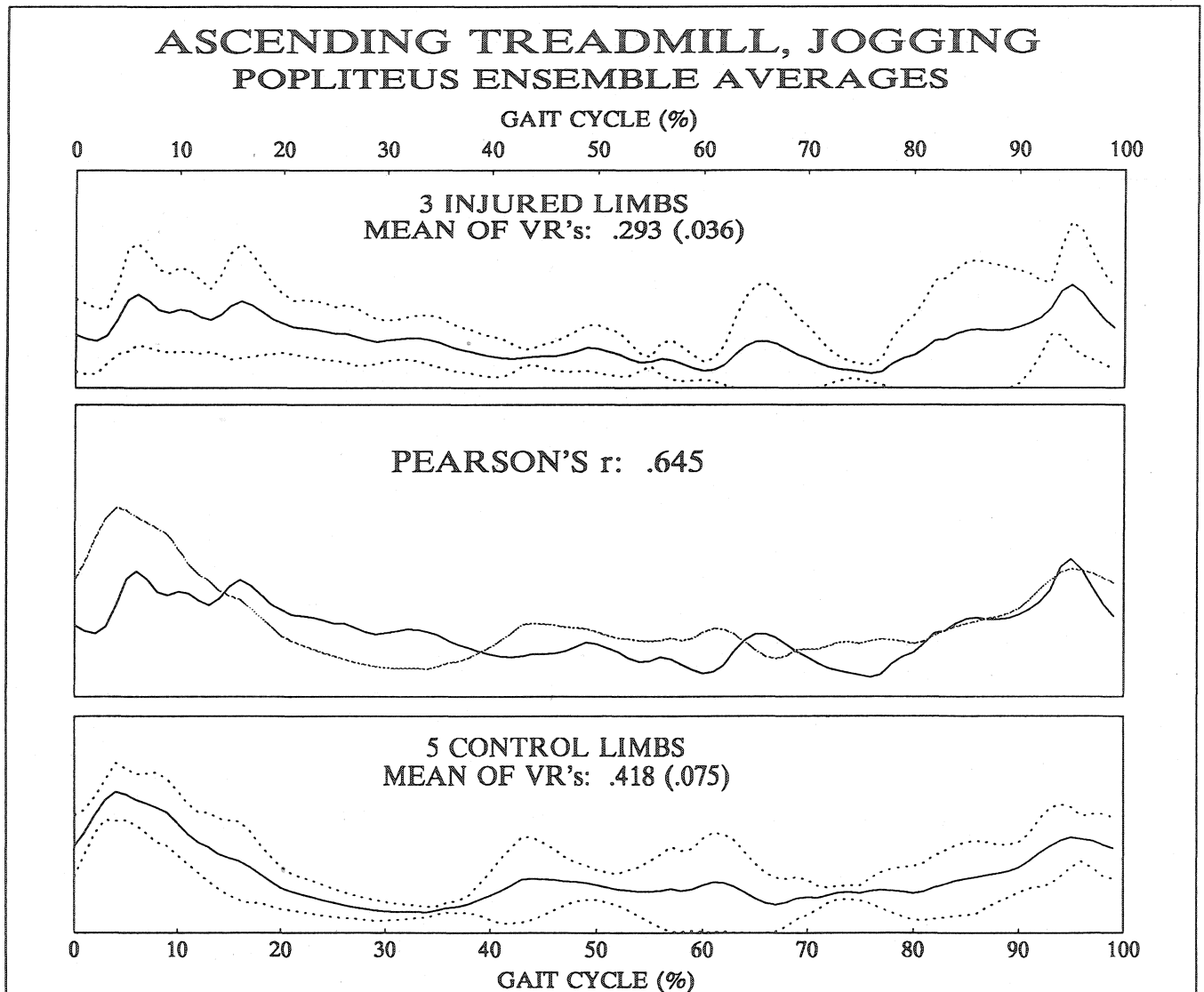


Figure 3.

Grand ensemble average ( $\pm 1$  standard deviation) for ascending treadmill walking for injured limbs (top window) and control limbs (bottom window); variance ratios shown with  $\pm 1$  standard deviation in parenthesis. Grand ensemble averages for injured and control limbs with Pearson's Product Moment Correlation "r" (middle window). Injured limbs tended to show less activity in late stance and swing, but the lower mean values generally fell within the  $\pm 1$  standard deviation range of the control activity.

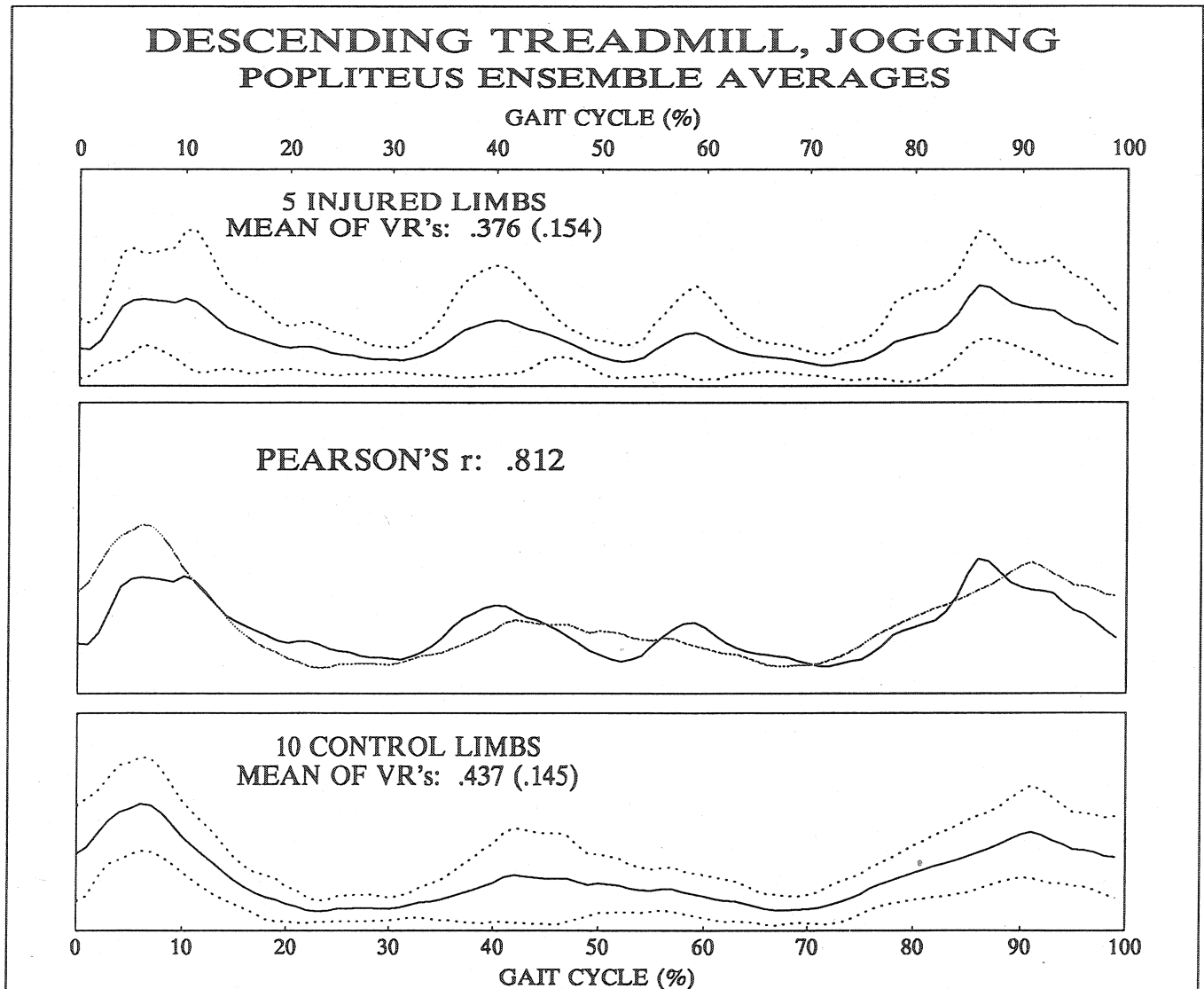


Figure 4.

Grand ensemble average ( $\pm 1$  standard deviation) for descending treadmill walking for injured limbs (top window) and control limbs (bottom window); variance ratios shown with  $\pm 1$  standard deviation in parenthesis. Grand ensemble averages for injured and control limbs with Pearson's Product Moment Correlation "r" (middle window). Injured limbs tended to show higher activity in late swing, but the higher mean values always fell within the  $\pm 1$  standard deviation range of the control activity.

### ACKNOWLEDGMENTS

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# MANAGEMENT OF CHRONIC POSTEROLATERAL INSTABILITY OF THE KNEE: OPERATIVE TECHNIQUE FOR THE POSTEROLATERAL CORNER SLING PROCEDURE

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The term posterolateral rotatory instability (PLRI) of the knee describes a spectrum of pathologic states of ligamentous laxity in which the lateral tibial plateau subluxates posterior to the lateral femoral condyle when an external rotational force is applied to the knee (Figure 1). When the point of subluxation is reached, the patient will recognize the associated weakness and posterolateral pain. In its mildest form, it is merely a physical exam finding of a gliding motion which is determined to be excessive only when it is compared to the patient's opposite side. When a clinically significant degree of laxity exists, the examiner can hold the foot in external rotation, apply valgus pressure and move the knee from a fully extended position into 20 or 30 degrees of flexion to demonstrate the subluxation. This subluxation will often present as the sudden translational acceleration of the tibia on the femur that the patient associates with episodes of giving way and that we know as a reverse pivot-shift phenomenon. Further flexion with the leg in valgus and external rotation will increase the patient's complaint of pain and weakness in the posterolateral corner of the knee.

Anatomically, PLRI and the reverse pivot-shift episodes are associated with injury to the structures at the posterolateral corner of the knee, known collectively as the arcuate complex. The arcuate complex consists of the arcuate ligament, popliteus tendon, the lateral head of the gastrocnemius, lateral collateral ligament, and the joint capsule. Clinically, functional instability is known to result either acutely from hyperextension-varus injuries; or gradually, associated with other ligament injury patterns. Patients with mild PLRI present with an insidiously developing entity which is commonly overlooked on physical examination. Patients with the most severe degree of laxity present a dramatic clinical picture where violent and painful pivot-shift subluxations are experienced with every step. It is this severe chronic state that is known to be most difficult to correct surgically.

In the late 1970's, a novel soft tissue procedure named the "posterolateral corner sling procedure" (PLCS), was developed that has, in my experience, proven to be a very reliable method for eliminating PLRI and the reverse pivot-shift phenomenon. The results of an outcome study of this surgery have been presented recently<sup>1</sup>. With the results-oriented article committed to publication elsewhere, the purpose of this paper is to describe the operative technique of the PLCS procedure. The procedure involves creation of an extraarticular sling that extends from a point on the posterior tibia, immediately medial to the proximal tibio-fibular articulation, anteriorly and superiorly to an isometric point on the femur.

## PREOPERATIVE EVALUATION AND OPERATIVE PLANNING

In isolated cases of severe arcuate complex pathology the following clinical findings are usually present; varus laxity, a positive dial test at 30 and 70 degrees of flexion, a posterior lateral drawer test, and a reverse pivot-shift. The degree of direct lateral laxity due to lateral collateral ligament (LCL) deficiency must be carefully evaluated because a separate surgical correction may also be required. The presence of intraarticular pathology is highly likely. It should be documented and treated arthroscopically prior to the extra-capsular dissection. The coexistence of other planes of instability is also quite frequent. In my experience, the most frequent ligamentous deficiency

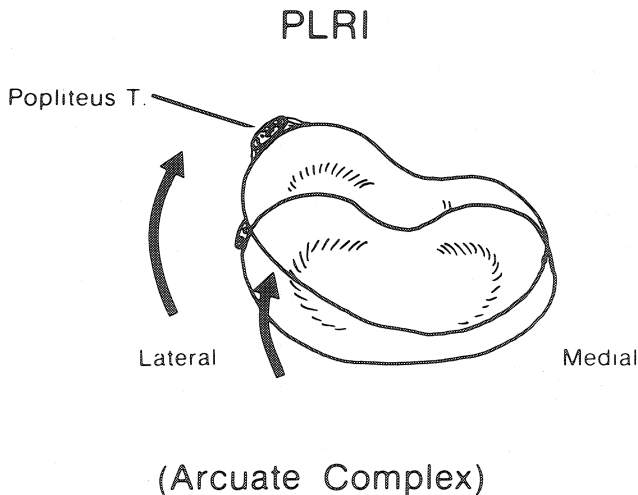


Figure 1

Top view of pathologic posterior displacement of the lateral tibial plateau on the lateral femoral condyle with laxity of the arcuate complex.

to accompany PLRI is anterior cruciate ligament (ACL) deficiency with anterolateral rotatory instability (ALRI). Less frequently posterior cruciate ligament (PCL) deficiency with direct posterior laxity is present. The combination of PCL and the arcuate ligament complex insufficiency makes for the most dramatic findings on dial and reverse pivot shift testing. The dial test is then likely to be greater at 70 degrees than at 30 degrees.

In any case of multidirectional instability of the knee, it is recommended that surgical correction of each component of instability be planned preoperatively. This is particularly important for estimating the duration of the entire operation and in judicious use of the tourniquet. Diagnostic arthroscopy and the PLCS procedure can be accomplished together in under two hours. However, each additional finding that requires surgical correction will add to the total length of the case. For instance, when an ACL reconstruction with patellar tendon autograft, a single meniscus excision/repair and an LCL reconstruction are added to the above two step procedure, a target time of 5 hours should be anticipated. In this instance, a split surgical team approach could be used to cut down operative time.

## DESCRIPTION OF OPERATIVE PROCEDURE

### The approach

With the knee flexed 45 degrees, the skin incision courses from just distal to Gerdy's tubercle (Figure 2), along the iliotibial band (ITB) onto the mid-lateral aspect of the distal thigh. Before the posterolateral corner is dissected it is important to identify and protect the peroneal nerve. It is most readily identified three finger breadths below the fibular head where it becomes superficial as it wraps around the fibula (Figure 2). Maintenance of the knee in a flexed position keeps the nerve out of the operative field during the procedure described below. The most important anatomical structures that need to be exposed for this approach are best located by first identifying the LCL from its femoral to its fibular attachment (Figure 3). Lying immediately posterior to the LCL is the tendinous portion of the lateral head of the gastrocnemius. The plane between the tendon of the lateral head of the gastrocnemius (Figure 4) and the LCL is developed from the femoral insertions to 2-3 cm below the lateral joint line. The gastrocnemius is retracted posteriorly to expose the structures deep to it. Careful dissection is then carried out in order to identify the capsule and the popliteus tendon.

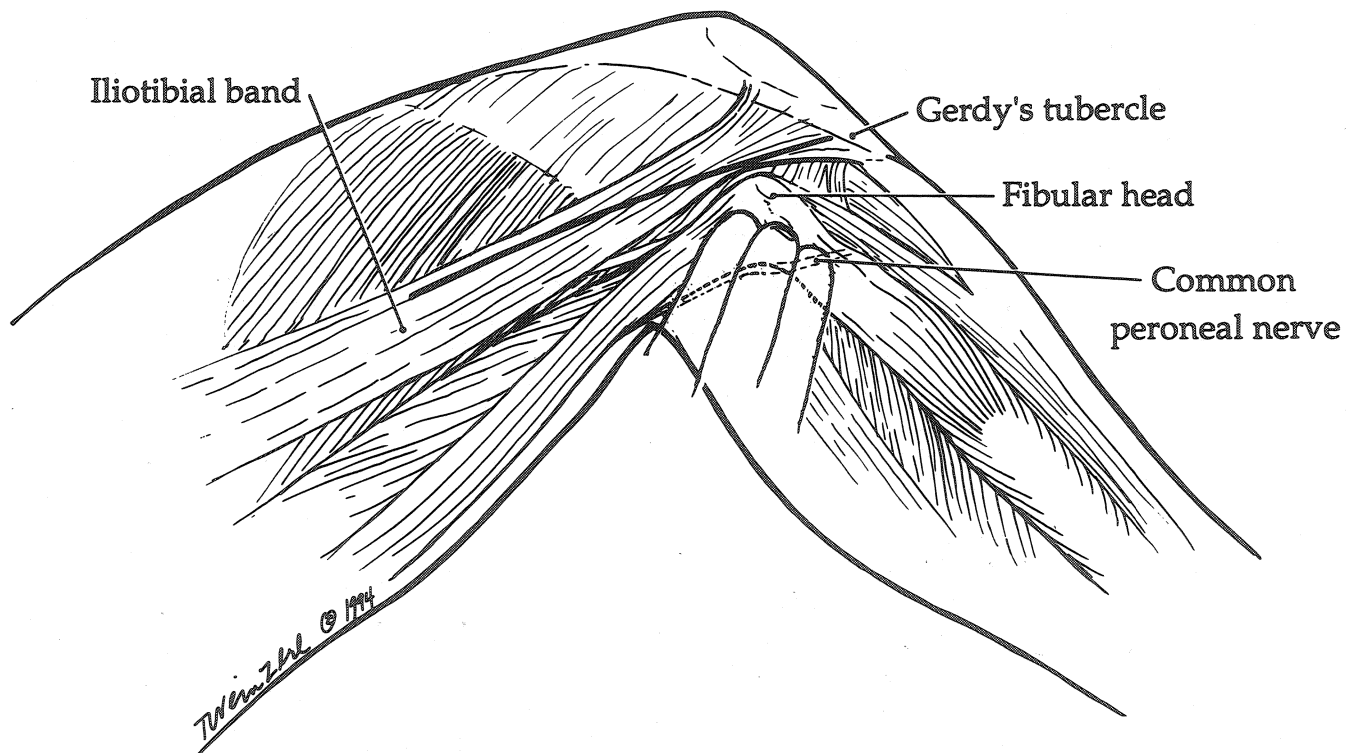


Figure 2

Lateral view of knee flexed 70-80 degrees. The incision is shown as it begins at Gerdy's tubercle and extends proximally up the mid thigh region. The location peroneal nerve can be identified as it becomes superficial to the fibular shaft approximately 3 finger breadths from the fibular head.

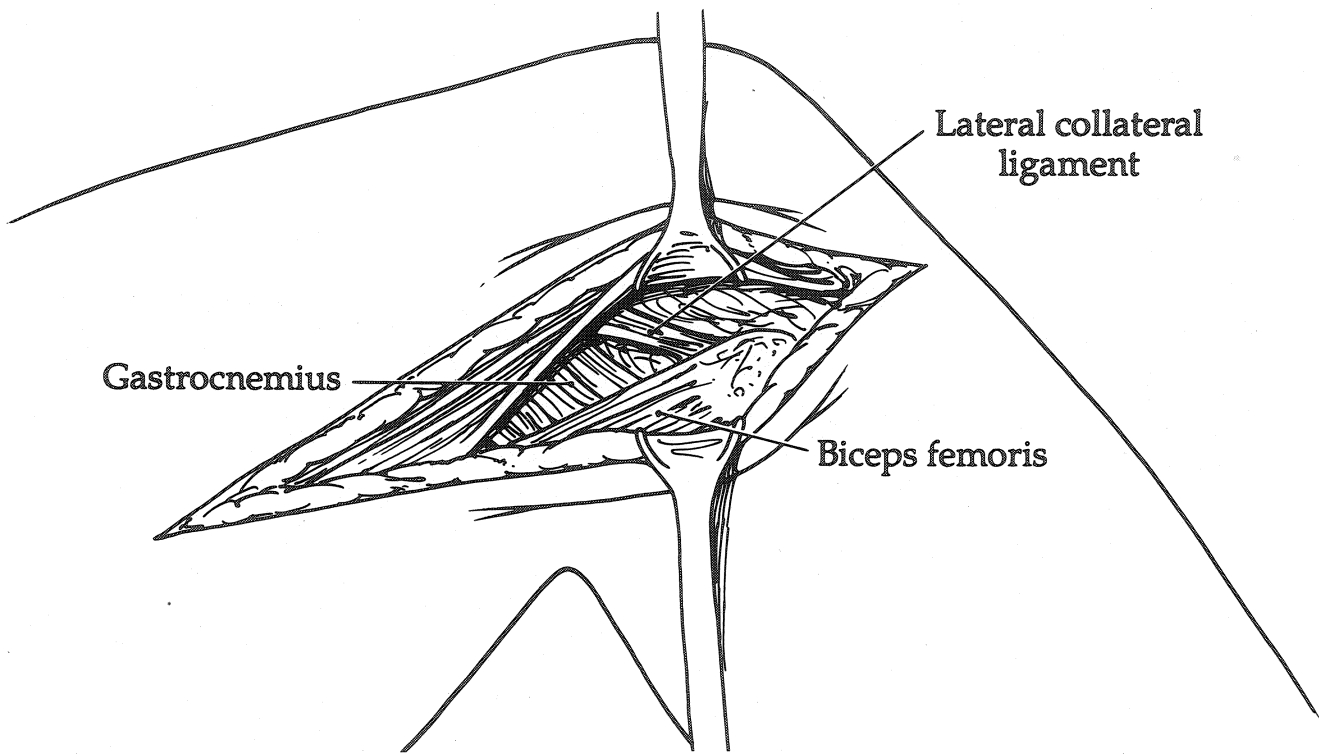


Figure 3

Beneath the ITB the LCL and the tendinous portion of the lateral head of the gastrocnemius lie adjacent to each other.

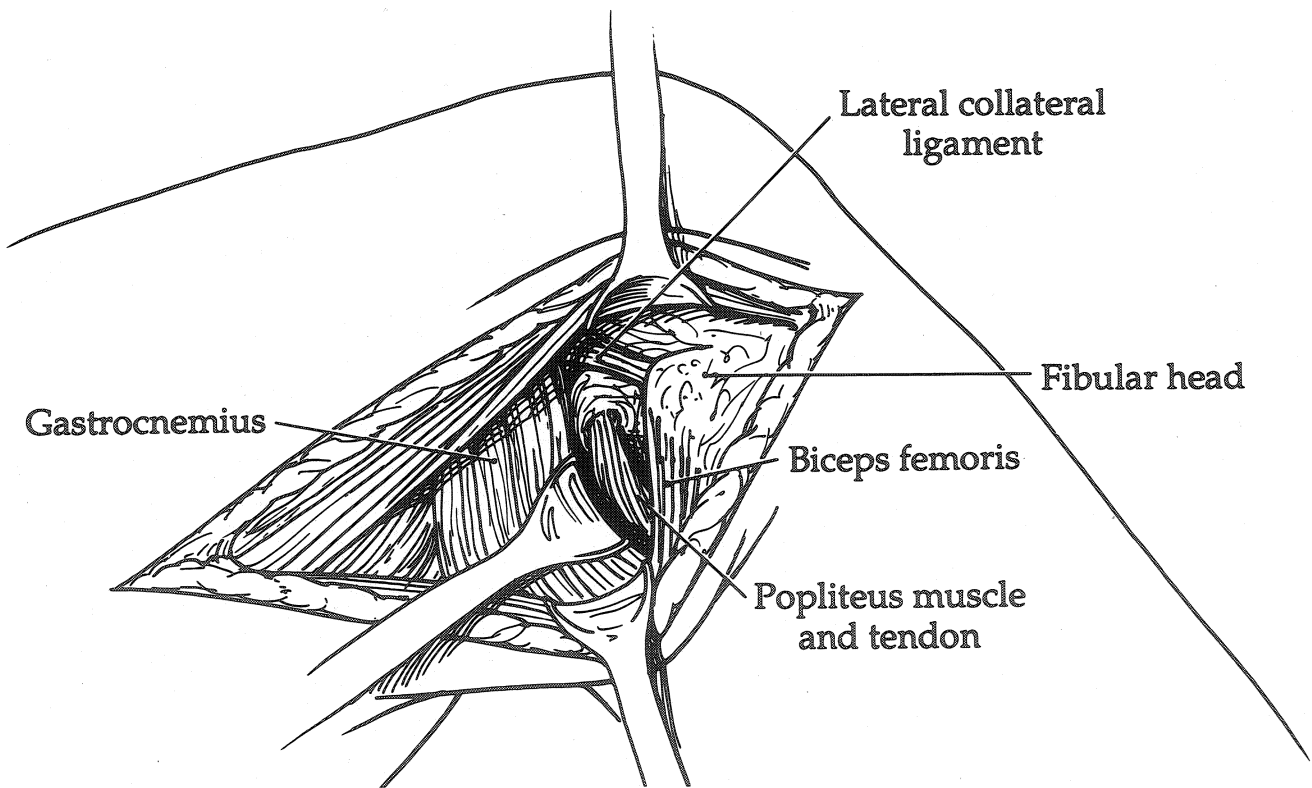


Figure 4

When the gastrocnemius is retracted, the popliteus can be found coursing obliquely. The muscle originates on the posterior aspect of the proximal tibia and inserts on the femur just inferior and anterior to the LCL.

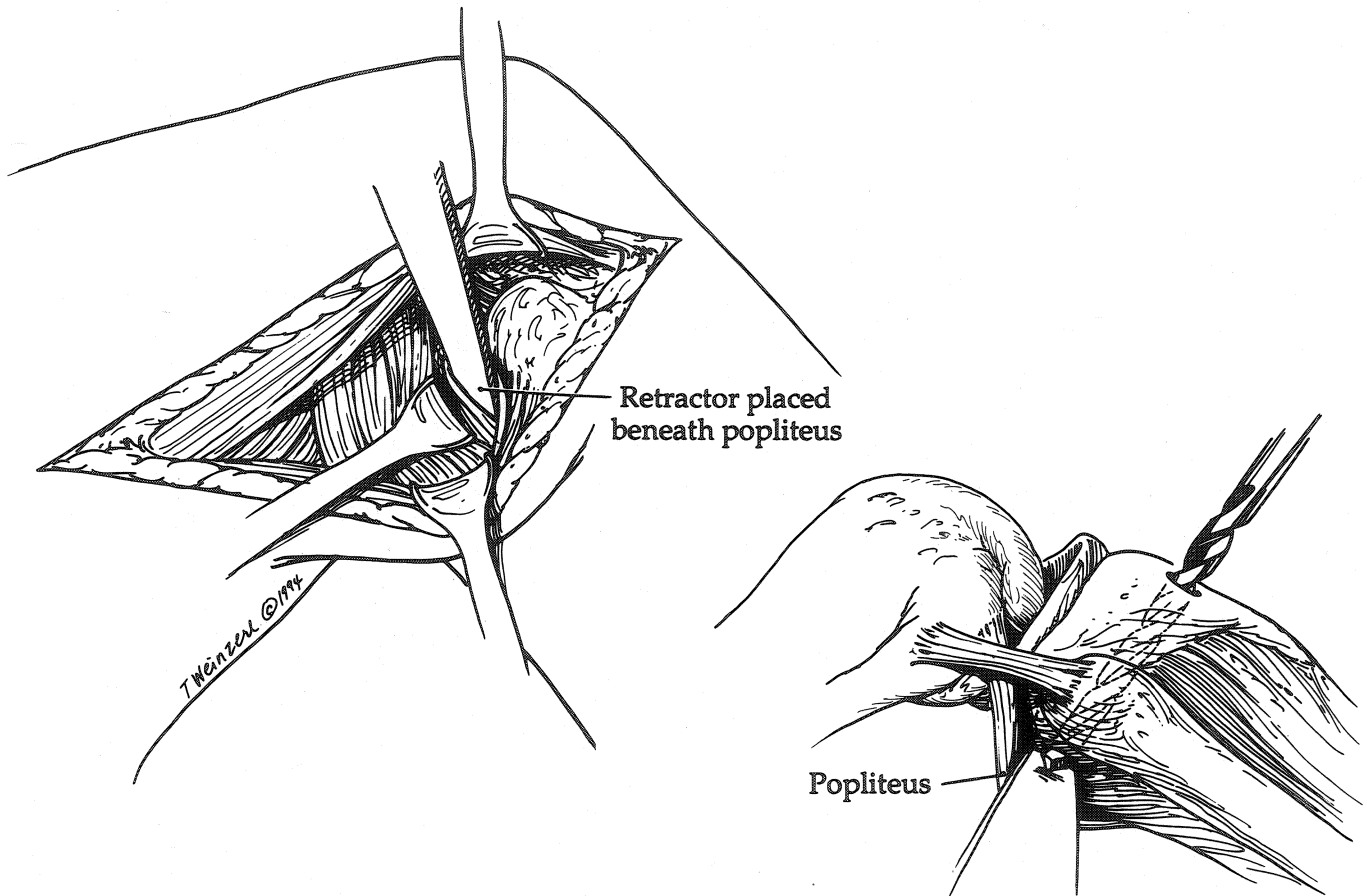


Figure 5

Once the popliteus is mobilized posteriorly, a retractor is inserted to expose the site where the tunnel will exit.

The popliteus tendon should be clearly delineated as it courses obliquely across this interval. In the deep portion of the wound it fans out as a wide muscle belly to its origin on the posterior aspect at the tibia distal to the capsule. The popliteus tendon disappears near the joint line as it dives deep to a thickening in the capsule, the popliteal foramen. This can be observed from above during the arthroscopic inspection of the lateral gutter and the posterior corner of the lateral meniscus. If help is needed to locate the popliteus tendon, it is most readily visualized from the top of the foramen through a vertical incision into the joint capsule at the anterior edge of the LCL. The narrowest of the Cave retractors (or a hemostat) can then be passed retrograde through the foramen to help identify the course of the tendon in the area of interest. Once having identified the portion of the tendon inferior to the joint line, an incision is made along its anterior border and then the entire tendon is bluntly freed from the adjacent tissues. This maneuver is continued until the popliteus can be swept posteriorly enough to expose the posterior lateral corner of the tibia.

### The Tunnel

A retractor is then placed beneath the popliteus to identify the target area on the tibia and to serve as a soft tissue protector from the exiting drill while the tunnel is being created (Figure 5). With the posterior tibia exposed, a 3/32 guide pin is passed from near Gerdy's tubercle to the desired point on the exposed tibia. The pin should exit at least 1-1.5 cm beneath the articular surface of the lateral tibial plateau and at least 1 cm medial to the proximal tibio-fibular articulation (Figure 6). This placement may be accomplished free hand or with the help of one of the latest large arc versions of the tibial guides available for endoscopic ACL reconstruction. Having achieved proper guide pin placement, a 6-8 mm diameter tunnel is established with a cannulated drill. The size of the tunnel depends on the size of the patient as well as of the graft but usually is 6, 7, or 8 mm in diameter. The location of the anterior starting point of the tunnel must be sufficiently inferior to the tibial articular surface and medial to the tibio-fibular joint to avoid compromising their integrity. If it is necessary to incorporate an anterior Losee type extracapsular sling to help control ALRI (due to a concomitant ACL deficiency) the starting point should be at, or slightly medial to, Gerdy's tubercle.

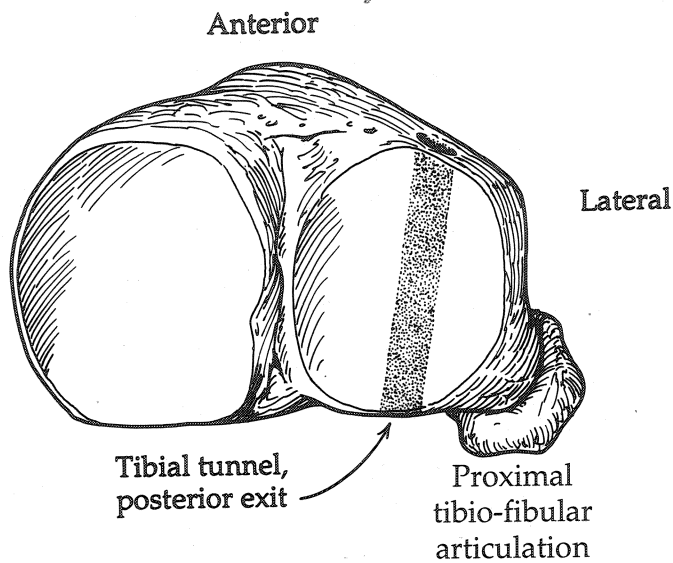


Figure 6

Superior view of the tibia to illustrate relationship of tunnel to proximal tibio-fibular joint. When using an ITB autograft, the anterior aspect of the tunnel should be in line with Gerdy's tubercle as shown here. When the PLCS is to be combined with a Losee type anterior sling, the entry portal should be medial to Gerdy's tubercle the interval between the tubercle and the patellar tendon.

### The Graft

Allografts and autografts have proven successful for this procedure. Freeze dried and fresh frozen (and irradiated) ITB and Achilles tendon allografts have been used. At least at the time of implantation, fresh frozen tissue is notably stronger than the freeze dried grafts. Among the allografts, Achilles tendons are the easiest to work with and probably the stronger of the two alternatives. For autografts, the central slip of the ITB is the most convenient to use because of both its length and its attachment to Gerdy's tubercle. If ITB autograft is being used, care must be taken to make sure that sufficient length of graft is harvested. While usually around 18 cm, an accurate estimate of the length needed can be made by running a suture through the tunnel and up to the proposed femoral attachment site. If an ITB or Achilles tendon allograft is used, anterior tibial fixation must be achieved prior to searching for and securing the isometric femoral attachment.

### Creating the Sling

The extracapsular posterolateral corner sling is created by passing a graft through the tibial tunnel from front to back with lead end anchor sutures and suture passers. The graft then exits its bony tunnel to emerge anterior to the lateral head of the gastrocnemius and popliteal tendon (Figure 7). From this point, the graft runs to a point on the

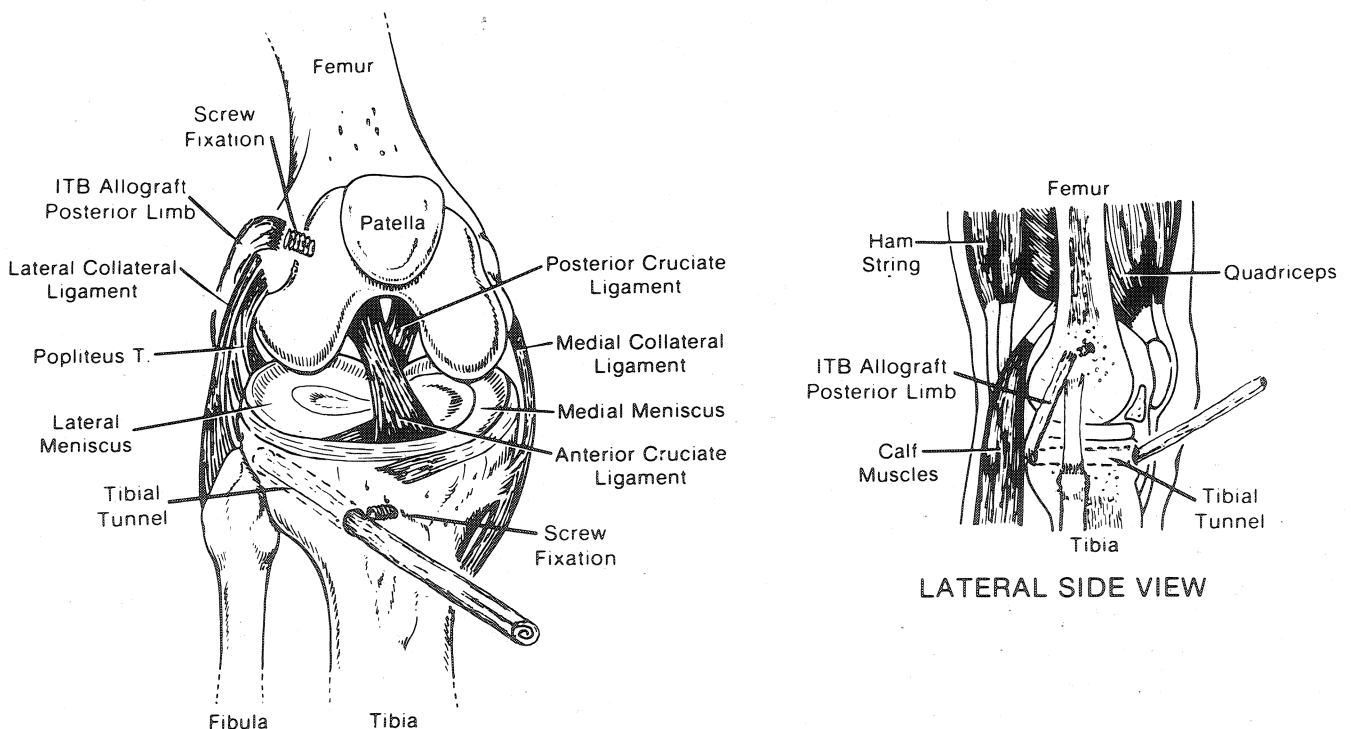


Figure 7

The posterior sling is accomplished by passing the graft from anterior to posterior in the tunnel. (Shown here is an allograft which must first be fixed to the tibia.) The posterior limb that exits the tunnel is brought up to an isometric point on the femur.

femur that will prevent pathologic posterior subluxation of the lateral tibial plateau in an isometric fashion. This isometric point is located by placing a Steinman pin slightly superior and anterior to the femoral attachment of the lateral collateral ligament. The proposed graft placement site is then tested for isometry by first placing the knee at 90 degrees of flexion, rotating the foot 10-15 degrees internally and then tensioning the soft tissue unit over the Steinman pin locator.

**Placement Testing**

Two tests are performed. The first test is performed by maintaining the internal rotation of the foot while the knee is run through a complete range of flexion and extension. Successful placement will result in equal tension of the graft throughout a complete range of motion from at least 0 to 120 degrees of flexion. In the second test, the thigh is supported and the knee is flexed to the degree (usually at least 45 degrees of flexion) that produced the most dramatic reverse pivot-shift preoperatively. By grasping the foot (without varus stress) the reverse pivot shift phenomenon is recreated when there is no tension placed on the PLCS graft. Tightening the graft should eliminate the posterior subluxation. This test can be repeated throughout the entire range of flexion. Care must be taken to make sure that any varus laxity from lateral joint line opening is taken up in the tensioning process.

**Considerations in the presence of complex multidirectional laxity: ACL Deficiency with ALRI**

Most frequently PLRI is seen in conjunction with ACL deficiency and ALRI. In this situation, concomitant intra-articular reconstruction of the ACL is imperative. This can be accomplished in any manner the surgeon wishes. Even an over-the-top graft should not preclude anchoring the extra capsular sling on the lateral femoral condyle. If the ACL tunnel is to exit on the lateral femoral cortex, then care should be taken to insure that it is located as high as possible above the flair of the distal femoral metaphysis. This is particularly important to avoid a conflict between the ACL graft tunnel location and the screw fixation of the PLCS.

Until the arthroscopically assisted intraarticular ACL reconstruction is completed, it is particularly important to maintain a water-tight joint by avoiding any incisions into the lateral capsule. Because it is difficult to establish a neutral point for the lateral compartment in the presence of both anterior and posterior lateral rotary instability, the manner in which the grafts are tightened is critical. It is my experience that the best way to find this neutral tibio-femoral relationship is to achieve ACL graft fixation first with the knee held in full extension. This should be accomplished without varus deformity and in neutral to very slight external rotation. Once the proper positioning and graft tension are identified in this position, the knee is

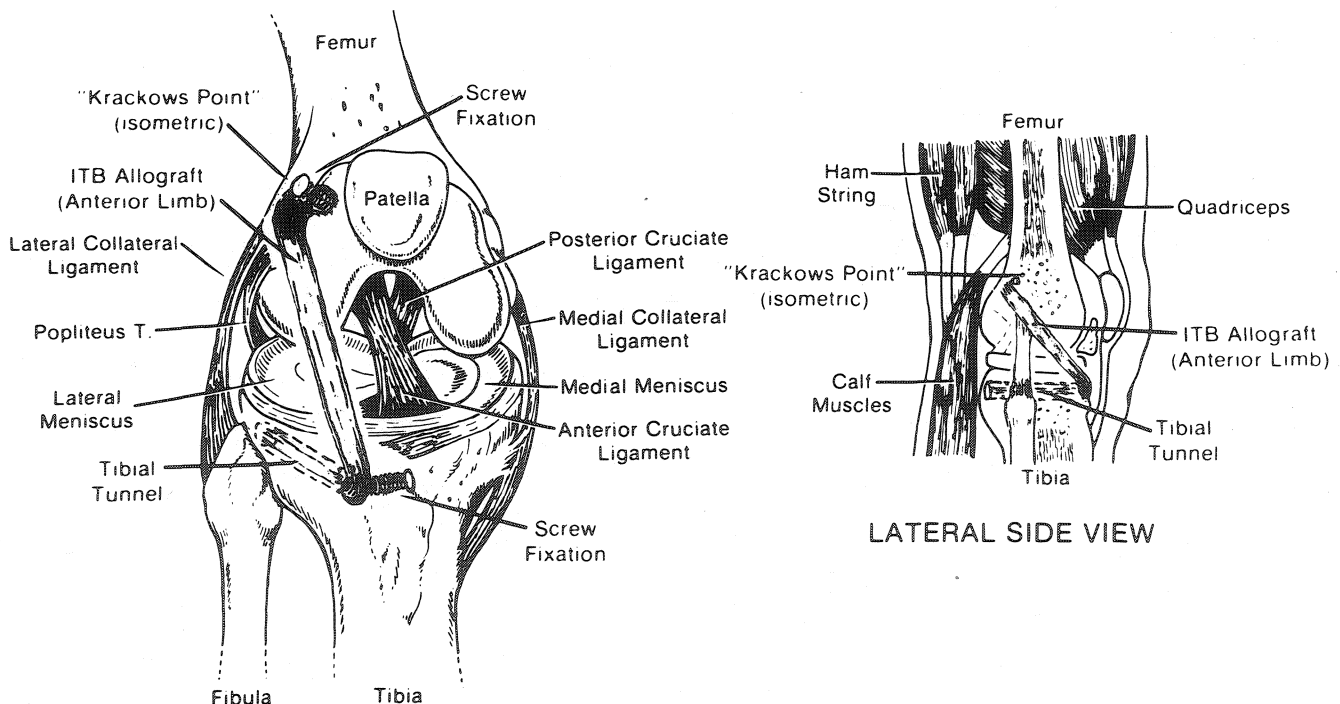


Figure 8

If an anterior extracapsular sling is needed to help control ALRI, the anterior limb of the allograft is taken back to Krachow's point.

maintained in the same rotation as the knee joint is flexed to about 20 degrees and the graft is tightened and secured with interference screws. Once having achieved this fixation, the PLCS procedure can be tensioned against the ACL.

If an allograft is being used for the PLCS and a Losee type extracapsular reinforcement of the ACL reconstruction is desired, an anterior extracapsular sling is run from the anterior tibial tunnel exit to a point near Krackow's isometric point on the femoral condyle (Figure 8). This sling is secured after the PLCS has been secured. The isometric point is also located with and tested over a Steinman pin. However, in this situation, neutral and even slight external rotation is maintained throughout the range of motion. Krackow's point was described as isometric in relationship to the ITB attachment at Gerdy's tubercle. It is located posterior to the femoral attachment of the LCL and near the gastroc tendon. More specifically, it is usually found to be 1 cm distal and posterior to the septum for the lateral thigh.

#### **Varus Laxity**

If varus laxity presents a significant problem, attention should also be directed to the fibular collateral ligament. Unless there is extreme varus alignment, soft tissue reconstruction should be successful obviating the need for a tibial osteotomy. If the ligament is intact but just lax, it is tightened by the proximal advancement of its femoral attachment on a cortical bone block. If there is insufficient LCL tissue or if there is a need for augmentation of an acutely ruptured ligament, this can be accomplished in one of two ways. First, as described by Clancy, all or part of the biceps tendon can be left attached to the fibula and dissected proximally after the peroneal nerve has been identified at its posterior border. The tendon is then screwed to the femoral origin of the LCL. Another alternative to this reconstruction is to use an Achilles tendon allograft. This method first involves the shaping of a tapered 6-7 mm bone plug cylinder from the calcaneus. This is such a small bone plug that shaping with a Midas Rex bur is recommended over the use of rongeurs in order to avoid shattering the bone. A taper is designed with the narrow end at the tendon attachment and the wider

diameter located at the calcaneal end. This graft is then run from front to back through a tapered tunnel in the fibular head (of 6 tapering to 5 mm) that runs parallel to the lateral knee joint line. The proximal portion is then fixed over a soft tissue screw and washer at the LCL femoral attachment.

#### **PCL Deficiency with Posterior Laxity**

Similar to the situation of ACL deficiency, the PCL should be reconstructed prior to the PLCS procedure. The most difficult situation I have dealt with yet has involved both ACL and PCL deficiency as well as medial collateral ligament (MCL) and LCL laxity. Simultaneous tightening of both cruciate constructs with repeated testing of AP laxity is a critical first step. The PLRI and varus components can be addressed only after intraarticular stability has been achieved.

#### **POSTOPERATIVE MANAGEMENT**

Postoperative management should be individualized, but the plan should generally include a moderate early range of motion (e.g., 0-90), protection from external rotation and avoidance of varus stress for at least 6-8 weeks. This includes keeping the patient non-weight bearing if there is varus alignment. Often, a cast brace is indicated to maximize rotational and medial-lateral control during this critical period. A functional knee brace with straps placed in a manner similar to those used for the posterior cruciate reconstructions and a lateral heel and sole wedge are prescribed for use at 6-8 weeks postoperatively. Progress should be cautious. The patient must be warned that any loosening may mean that they go back into a cast. A 9-12 month rehabilitation time period is anticipated.

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# BILATERAL OSTEOCHONDRITIS DISSECANS IN A FEMALE PITCHER A CASE REPORT AND REVIEW OF THE LITERATURE

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## INTRODUCTION

### **A case of bilateral elbow osteochondritis dissecans in a female pitcher is presented.**

Osteochondritis of the elbow is a well known disorder affecting pitchers and other individuals that place high demands on their upper extremities. Elbow osteochondritis has been described infrequently in females. Increased participation of female athletes in organized sports is likely to increase the incidence and reporting pattern of this disease.

## CASE REPORT

The patient is a 17 year old right handed female multi-sport athlete. She was active in high school volleyball, basketball, and was a softball pitcher. She presented with the complaint of several years of intermittent right elbow pain and swelling. Approximately three months prior to presentation she noted pain and swelling, with new symptoms of grinding, locking and decreased range of motion. She denied a history of acute trauma. She continued sports with no loss of playing or practice time.

Past medical history was negative for any other joint pathology or endocrine disorders. Family history was negative for any bone or joint problems. Examination of her right elbow revealed no obvious swelling. There was slight tenderness to palpation anteriorly over the antecubital fossa. There was no joint line tenderness to palpation. Active range of motion was 0-130 degrees with audible grinding. There was loss of extension compared to the opposite, asymptomatic elbow. She was able to demonstrate full active pronation and supination. The only other significant finding was pain on active wrist extension against resistance. This reproduced her elbow pain. Neurovascular examination was normal.

Roentgenograms of the right elbow revealed a large loose body with a defect in the distal capitellar articular surface. A zone of increased density was notable at the base of the crater-like defect. Flattening of the capitellum was demonstrable on the lateral view. Olecranon spurs were visible on both the AP and lateral views (Figures 1 and 2). Because of the presence of mechanical symptoms, limited range of motion, and loose body formation, surgery was indicated.



Figure 1

Intraoperative findings revealed multiple loose bodies. The capitellar defect came in contact with the radial head in full extension. The radial head articular surface appeared normal. A medial trochlear condylar defect was noted. With elbow flexion to 90 degrees, the trochlear defect abutted the coronoid process of the olecranon.

Diffuse synovitis and olecranon osteophytes were present. The operative procedure included removal of loose bodies, debridement of the capitellar and trochlear defects and partial synovectomy. On follow-up she resumed full activities, including pitching without pain, catching or functional limitation.





Figure 2

The patient returned 14 months after her initial presentation. She was now participating in intercollegiate basketball. She stated that her right elbow was "as good as new." She had a new complaint of left elbow pain. There was no history of acute trauma. She complained of catching, grinding, and limited range of motion. She felt that these symptoms were identical to those that had been present in the right elbow prior to surgery.

On examination no obvious swelling or tenderness was present. Active range of motion was 0-140 degrees. She demonstrated full active pronation and supination. Neurovascular examination was normal. Roentgenograms revealed a loose body with a crater like defect in the distal capitellum (Figures 3 and 4). The patient wished to have surgery performed but deferred the procedure until the conclusion of basketball season.

#### DEFINITION

Initially the term osteochondritis dissecans was used to define a pathological process involving inflammation of the bone and joint with desiccation of the osteochondral fracture. More precise histological analysis has shed new light on the pathological process that takes place in osteochondritis. In 1953 Green described microscopic finding of aseptic necrosis of the subchondral bone. There were no inflammatory cells present<sup>7</sup>.

The progression of macroscopic changes in osteochondritis has been well documented. Initially, subchondral bone undergoes necrosis. Cartilage maintains its nourishment from the synovial fluid. But, necrotic bone is resorbed and the cartilage loses its mechanical support. This places it at high risk for fracture and displacement from its base. With repetitive trauma, softening, degeneration and

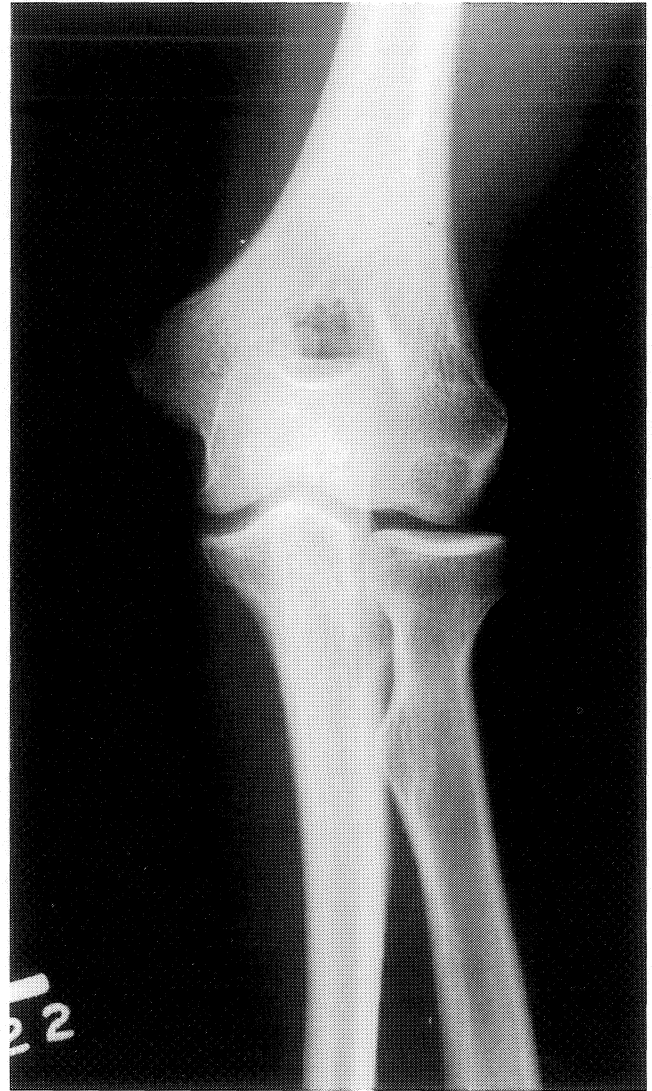


Figure 3

eventual fragmentation of the joint surface occurs. If protected, necrotic bone is resorbed and the osteochondral fragment may go on to heal.

#### ETIOLOGY

Several theories on the etiology of osteochondritis dissecans have emerged<sup>7,15</sup>. One theory suggests an ischemic insult to a localized area of subchondral bone. Histopathological architecture is similar to that found in infarction. Hyperemia and edema are common findings. Late stages of osteochondritis follow one of two routes. Repair is more likely to take place if the articular surface remains intact. Restoration of normal architecture is often the end result. If the articular surface is fractured, the necrotic osteochondral segment may become detached and form a loose body.

Repetitive trauma has been suggested as the etiology of



Figure 4

osteochondritis of the elbow. Albright described side-arm pitching mechanics that contribute to elbow joint pathology<sup>1</sup>. Tullos and King also analyzed the mechanics of pitching<sup>20</sup>. They felt that the forces across the radiocapitellar joint played a part in development of osteochondritis of the capitellum. During the acceleration phase of pitching, the shoulder and arm move forward, the forearm and hand are left behind, and the elbow assumes a position of extreme valgus. The elbow flexor mass, medial collateral ligament, and the medial joint capsule become attenuated leading to abnormal compressive forces across the lateral joint space. Medial epicondyle avulsion fractures can occur in skeletally immature patients. Flexor muscle mass ruptures are uncommon but can occur in adults. Medial collateral ligament sprains may lead to elbow joint instability. Osteochondral fractures and osteochondritis dissecans are common sequelae of chronic repetitive micro-trauma to the lateral joint space<sup>5,6</sup>.

Heredity may play a role in the development of osteochondritis. There have been several reports of osteochondritis dissecans occurring in several generations of the same family<sup>3,19,22</sup>.

### INCIDENCE

Osteochondritis dissecans usually occurs during the second decade of life, during the growth spurt<sup>11,12,17,22</sup>. The dominant elbow is most commonly affected. Bilateral

involvement has been described infrequently. Repetitive trauma is identified as the etiology of osteochondritis in over 50 percent of reported cases<sup>10</sup>. Involvement of multiple joints has been described<sup>7,8,19</sup>.

Osteochondritis primarily involves the knee, but elbow involvement occurs in about 6 percent of all cases<sup>11</sup>. The evaluation of 1000 elbow x-rays in an adult population revealed an incidence of 0.4 percent<sup>14</sup>. Of all cases of osteochondritis reported in the literature, 98 percent have been in males. Osteochondritis has been reported infrequently in females. The largest series to date describes ten cases of osteochondritis in seven female gymnasts<sup>9</sup>.

### CLINICAL FEATURES OF OSTEOCHONDRITIS DISSECANS

#### History

The typical patient is an adolescent or young adult complaining of elbow pain and swelling. Usually he or she is active in sports or activities involving repetitive, ballistic upper extremity motion. Often pain is present which is aggravated by activity and relieved with rest. The patient may complain of a grinding, or catching sensation with limited range of motion.

#### Physical Examination

An effusion is usually present. Cubitus valgus is a common finding in pitchers. Palpation of the lateral joint line can be painful. Pronation and supination are often limited. Crepitus with active and passive range of motion is usually present in patients with radiographic evidence of a loose body. In many asymptomatic pitchers a 5-15 degree flexion contracture is common. With acute symptoms, look for loss of flexion as well as a more pronounced flexion contracture.

#### Diagnostic Tests

Roentgenograms should include AP and lateral views of the elbow. Findings on the AP view include an irregular area of decreased density in the subchondral bone of the capitellum. This lesion is usually surrounded by a well circumscribed zone of increased density. Cystic changes in the capitellum are often present. If the osteochondral lesion has been shed from its base, a loose body is usually apparent. On the lateral view, flattening of the capitellum is visible. The ipsilateral radial head may be larger when compared to the opposite, unaffected side. Early physal closure is a common finding when comparison views are obtained. Osteophyte formation and joint space narrowing in young adults with chronic symptoms are not uncommon.

It is important to evaluate the integrity of the articular surface and the displacement of the osteochondral fracture. Arthrography can be used to assess integrity of the

articular surface. Dye will collect in the articular surface defect, and surround the defect if it is completely detached. Tomography is useful during treatment. It can help identify areas of healing and areas where a fracture line persists. MRI may help define the extent of a capitellar defect. Its diagnostic and prognostic utility compared to other less costly modalities has yet to be determined<sup>13</sup>.

### DIFFERENTIAL DIAGNOSIS

Panner's Disease is defined as osteochondrosis of the capitellum. This is a disease involving the ossification centers in skeletally immature patients. Degeneration and necrosis is followed by regeneration and reossification. It occurs primarily in boys, ages 7-12, during active ossification of the capitellar epiphysis. Patients present with pain and limitation of elbow extension. Swelling and lateral joint line tenderness are present. The entire capitellar ossification center is fragmented with sclerosis and alternating rarefaction. The capitellar outline may be irregular and smaller than the opposite unaffected side. With time, the epiphysis heals with a normal radiographic appearance. The condition is self-limited with rest as the only prescribed treatment. Ischemic bone diseases, endocrinopathies, collagen vascular diseases, multiple epiphyseal dysplasia are just a few disease processes that should be included in the differential diagnosis.

### TREATMENT AND PROGNOSIS

Treatment is based on clinical and radiological findings. Pappas described findings and treatment based on age and clinical findings<sup>16</sup>. In-younger patients the articular surface is usually intact. Sometimes a partially detached, nondisplaced lesion is present. With protection, healing of the fragment occurs in the majority of cases. Treatment should include rest and possibly immobilization. After a three to four week period of immobilization, range of motion exercises are initiated. The elbow is protected from trauma until radiographic evidence of healing is noted.

In adolescents, healing is not as predictable. Evaluation of the integrity of the articular surface is important. The lesion may be partially or completely detached from its subchondral bed. Non-operative modalities include activity modification and short term splinting. A program designed to improve range of motion with forearm flexor and extensor muscle stretching follows. Mechanical symptoms of catching and grinding with limited range of motion suggest a loose body. If confirmed radiographically, operative intervention is indicated<sup>2,9,12,16</sup>. Operative options include removal of the loose body, an attempt at reattachment of the loose body, and drilling or curetting the crater base with loose body removal.

The treatment of an articular surface defect is controversial. Bauer recently evaluated the long term follow-up of elbow osteochondritis dissecans<sup>2</sup>. Thirty-one patients were followed an average of twenty-three years. Twenty-three of thirty-one patients had surgery that consisted of loose body removal. Sixty-one percent of the patients had degenerative joint disease at ten year follow-up. When osteochondritis presented in childhood, the prognosis was better. Long term results revealed degenerative joint disease in only twenty-five percent of cases presenting in childhood.

Basic science literature allows us to evaluate articular cartilage defects and healing in the animal model. Cartilage response to injury depends on several factors. The depth of the lesion determines the healing response<sup>4,21</sup>. Partial thickness defects demonstrate no significant change in dimensions with time, and essentially no evidence of early or late repair.

Shapiro has recently documented the healing response to 3mm full thickness defect in the rabbit model<sup>18</sup>. Full thickness defects to bleeding subchondral bone expose the defect to undifferentiated marrow mesenchymal cells. This allows the inflammatory cascade to begin. Fibrin clot formation is followed by mesenchymal cell infiltration. These cells undergo differentiation to chondroblasts. Full thickness articular defects fill in with hyaline cartilage superficially and mature subchondral bone in the deeper layers.

Short term findings are not as encouraging. Fibrillation and degeneration at the repair site are evident at twelve weeks after injury. Progressive erosion and destruction of the newly formed articular cartilage layer are evident at forty-eight weeks. The data allow us to predict that attempts at articular surface repair in full thickness defects are likely short-lived. This supports Bauer's clinical finding of osteoarthritis in the long-term follow-up of surgically treated elbow osteochondritis<sup>2</sup>.

In summary, exposure to repetitive micro-trauma is a well known cause of elbow osteochondritis in throwing athletes. Treatment of the nondisplaced osteochondritic lesion is initially non-operative with immobilization or rest followed by range of motion. A completely detached but non-displaced lesion should be treated non-operatively. Initial restriction of activity is important for healing. In patients with refractory symptoms, drilling may encourage healing of the lesion back to its base. The primary indication for surgical intervention is loose body removal in patients with mechanical symptoms.

Long term follow-up suggests a relatively poor prognosis in patients with osteochondritis presenting after childhood. Osteoarthritis is a common finding on follow-up in this group of patients. Preventive measures include evaluation and correction of abnormal throwing mechanics in

pitchers. Patient and parental education play an important role in the young athlete. In athletic patients with osteochondritis and evidence of degenerative joint disease, it may be prudent to suggest changing to a sport that decreases the risk of further articular cartilage damage. It is important that the patient and parent are made aware of the late sequelae of osteochondritis. They need to play an active role in the decision whether or not to continue the sport or activity that is responsible for repetitive micro-trauma to the elbow. Halting the progression of articular cartilage destruction and subsequent degenerative joint disease should be the overall goal of the physician, coach, parent and patient.

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# PSYCHOMOTOR EDUCATION IN ORTHOPAEDICS: A RECONSIDERATION

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The shift from clinic to operating room as the primary educational venue for orthopaedic surgeons gives reason to reconsider psychomotor education from both theoretical and practical perspectives, that is, the educational principles involved, and how they are currently implemented in orthopaedic teaching programs.

## PRINCIPLES

Conceptually, education begins with the development of a curriculum, for which Lippert has described six steps: 1) formulation of a philosophy; 2) establishment of needed competencies; 3) identification of resources; 4) development and sequencing of courses; 5) determination of teaching strategies; and 6) evaluation.<sup>1</sup>

Formulation of a philosophy entails recognition of the fact that surgical outcomes are determined more by thought and attitude than by motor skill; for that reason, psychomotor education must inculcate requisite knowledge and values as well as technical skills.

Competencies needed for orthopaedic surgical procedures include restoration of three-dimensional anatomic configuration, fitting surfaces together, internal and external fixation, insertion of replacement components, cementing techniques, and soft tissue reconstructions.

Minimal resources include an accessible area for studying x-rays and videotapes, dissecting specimens, practicing with power tools, and preoperative planning. Necessary equipment may be obtained from the department, hospital, instrument companies, grants, and private donations. Funding sources must also be secured for maintenance of the area and equipment.

Courses are sequenced to proceed from simple to complex movements in the use of instruments, followed by execution of basic and advanced procedural skills in which movements are linked and practiced until uninterrupted flow is achieved. In general, the more complex the procedure, the greater the need for laboratory simulation prior to performing it in the operating room. Where possible, procedures performed in the skills laboratory should be integrated with related aspects of the cognitive curriculum.

During the learning experience, the trainee should be supervised by an enthusiastic, non-threatening, and knowledgeable instructor who watches and comments constructively as necessary.

Evaluation is accomplished by means of a checklist, which includes the cognitive and attitudinal domains as well as motor skills. Throughout, principles of adult learning must be observed, i.e., the exercises must be problem-oriented, meaningful to the learner, and interactive.

Because it avoids the tensions of the operating room, a skills laboratory offers a model setting for the isolation and practice of motor skills. Possessing the needed motor competencies, a surgeon is better able to adjust the alterations in the surgical plan dictated by unexpected operative findings; and being more confident of his own skills, he or she is able to devote more attention to team interactions.

A transitional and perhaps underutilized arena for psychomotor education is the animal surgery laboratory, which our colleagues in general surgery have utilized more often and more effectively than the orthopaedists.

The final stages of learning occur with the progression from second to first assistant to surgeon in the operating room, where improved motor skills lessen time, tension, and complications.

Preoperative planning and postoperative review are also critical to the enhancement of psychomotor skills. In the preoperative phase, the surgeon must become fully acquainted with the anatomy and operative details, visualizing in a three-dimensional mind's eye each step of a procedure, and anticipating as many unexpected intraoperative findings as possible, along with alternative operative modifications for each. Use of the skills laboratory may be particularly valuable at this time. In the early postoperative period, the operator should review the procedure, making a frank self-appraisal of his or her performance. Special attention should be given to those areas of the procedure that did not go smoothly, and to means by which they may be avoided when the procedure is next performed.

## CURRENT STATUS

To determine the present state of psychomotor education, a questionnaire was sent to all 163 orthopaedic program directors. One hundred eight programs (66%), responded with the following findings:

Fifty-eight percent of psychomotor education takes place in the operating room, followed by roughly 10% each for courses (e.g. A.O.), cadaver or dry bones exercises,

and hands-on demonstrations by representatives from instrument companies; 9% was carried out in skills laboratories.

Sixty-four percent of the reporting programs used a skills laboratory. These facilities occupied an average of 603 square feet, with a range of 50 to 7000. The most frequently taught skill in these laboratories was fracture fixation (67%), followed by arthroscopy (45%), "basic" (32%), arthroplasty (19%), and miscellaneous skills (7%).

The average annual cost for maintenance and operation of a skills laboratory was \$9,992, 55% of which was provided by the department, 13% by hospitals, 11% by instrument companies, 8% by a combination of these sources, and 13% by "other" sources. The *total* annual cost—including attendance at courses—for teaching psychomotor skills to residents averaged \$11,486.

Eighty-five percent of respondents indicated that they spent 1-5 hours per week teaching psychomotor skills outside the operating room. Eleven percent did no additional teaching and 4% devoted more than five hours to this activity. Fifty-seven percent of the reporting programs believed that the amount of time given to psychomotor education was about right, whereas 33% felt they should commit more and 9% less time to this activity.

When asked if they would send their residents to a central facility designed, equipped, and staffed to teach basic orthopaedic psychomotor skills, estimating a course length of one week and a tuition of \$500, 54% of respon-

dents replied "yes," 14% "maybe," and 32% replied "no." They felt that the most important competencies to be taught in such a facility were fracture fixation (23%); arthroscopy (19%); joint replacement (12%); use of power tools, basic equipment, technique and rationale (11%); osteotomy (8%); microsurgery (7%); and intramedullary nailing techniques (7%).

These data must be viewed in light of the probability that the programs that responded to the questionnaire were those with more comprehensive and structured programs in psychomotor education.

If one accepts the premise that procedural events will pay an increasingly dominant role in the professional lives of orthopaedic surgeons, educational programs should consider how to meet this need. Given the collision between the value and cost of psychomotor skills laboratories, provision of a centralized facility, such as the Orthopaedic Skills Learning Center being developed adjacent to the American Academy of Orthopaedic Surgeons could provide resident education in basic motor and surgical skills, as well as hands-on components of continuing medical education courses.

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# A RANDOMIZED CLINICAL TRIAL COMPARING CEMENTED TO CEMENTLESS TOTAL HIP REPLACEMENT IN 250 OSTEOARTHRITIC PATIENTS: THE IMPACT ON HEALTH RELATED QUALITY OF LIFE AND COST EFFECTIVENESS

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## SUMMARY OF CLINICAL RELEVANCE

Randomized clinical trials are considered mandatory before a new pharmaceutical agent can be released for public consumption. Surprisingly, randomized clinical trials are virtually unheard of in assessing a new surgical procedure or device. In this study, funded by the Medical Research Council of Canada, 250 osteoarthritic patients with mainly unilateral osteoarthritis of the hip were randomized as to whether they received a cemented or cementless total hip replacement. Patients were stratified for age (under 60 or over 60 years) and surgeon (RBB or CHR). All operations were done in the same operating room, utilizing the same direct lateral approach and surgical technique. Post-operative care was the same. The patients and the two clinical observers (KL and RB) remained blinded as to whether a cemented or cementless device had been inserted. Accurate patient cost was documented for each patient's in-hospital stay, as well as out-patient costs during the first year. Cost to quality adjusted life year data was then generated, such that comparisons could be made to other medical interventions (i.e. coronary artery bypass).

Several clinically relevant results emerged from this study, which the authors believe is the first randomized clinical trial comparing two orthopaedic implants.

- 1) Surgical patients are willing to be part of a meaningful randomized clinical trial. In this study, 78% of eligible patients agreed to participate.

- 2) Total hip replacement is an amazingly efficacious procedure, converting a patient with severe compromise of their quality of life to near normal health.
- 3) Total hip replacement compares favorably to virtually any medical or surgical treatment modality in terms of cost to quality adjusted life years.
- 4) Cemented and cementless total hip replacements are virtually identical in terms of patient specific, disease specific, global, utility, functional capacity and cost related outcome measures.
- 5) In an era of cost containment, data such as this study will have an important effect in insuring that cost effective treatments remain funded.

A 60 year old woman presenting with osteoarthritis of the hip in 1976 might have been offered a resurfacing arthroplasty of the hip. This procedure was considered an appropriate surgical intervention at that time. An investigation of the literature of the day suggested that surface replacement was indeed an efficacious procedure for the surgical management of osteoarthritis of the hip<sup>18</sup>. Its advantages were "intuitively obvious" and many surgeons in Europe and North America championed this device as a "conservative" total hip replacement. It was released by the manufacturers without substantial clinical trials and as a result it was several years before its deficiencies, namely those of acetabular loosening, femoral neck fracture and massive osteolysis, became apparent to the orthopaedic surgical community. This so called "conservative" surgical procedure turned out to be a radical procedure, making revision total hip more difficult because of major bone defects created by the attendant osteolysis. There are many other examples of failures and disasters in the orthopaedic literature, including the Mittelmeier hip as well as heat pressed polyethylene<sup>14</sup>. The question one has to ask is, "Could these failures and disasters have been

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prevented?" All orthopaedic surgeons are in agreement that cemented total hip arthroplasty has revolutionized the treatment of patients suffering from arthritis of the hip; nevertheless, with patients living longer and longer, surgeons, in conjunction with the orthopaedic industry, have attempted to improve the results of total hip arthroplasty by employing cementless implants. Is the new technology of cementless total hip replacements justified? To date, the efficacy of cementless arthroplasty, its cost effectiveness and its impact on health-related quality of life have not been investigated. As health care providers and as orthopaedic surgeons it is incumbent upon us to be able to justify to third party payers as well as our patients why we are doing what we are doing. Comparative studies comparing cemented to cementless total hip joint replacements are rare<sup>11</sup>. To date, all have been retrospective and a randomized clinical trial to assess the efficacy of cemented and cementless has not been reported. Randomized clinical trials are used extensively to assess new medical interventions and new pharmacological interventions; however, it is rare for a randomized clinical trial to be used to assess a surgical intervention. The advantage of a randomized clinical trial, comparing two different operative procedures, is that it allows investigators to minimize bias by ensuring that most prognostic factors such as age, sex, weight and femoral type are similar in the two groups.

A randomized clinical trial comparing cemented to cementless total hip joint replacement has been performed at the University of Western Ontario. The purpose of the trial was two fold. 1) What effect does elective total hip joint replacement have on health-related quality of life when a cemented implant is compared to a cementless implant? 2) What is the cost-effectiveness of total hip replacement?

### METHODOLOGY

A randomized clinical trial comparing cemented to cementless total hip joint replacement has been funded by the Medical Research Council of Canada. All surgery was performed under the direct supervision of the two senior authors (CHR/RBB). The Mallory Head Implant (Biomet. Inc.) was chosen for purposes of this study. The implant was chosen primarily because the geometry of the cemented and cementless implants were similar. In addition, the authors felt, in 1987, that a titanium implant (with cobalt chrome heads) was preferable to a cobalt chrome implant. All patients with osteoarthritis of the hip, either primary or secondary, between the ages of 18 and 75, presenting to the University Hospital beginning in October 1987 were asked to be part of the study. The exclusion criteria included other causes of hip joint arthritis, severe arthritis of the knees or the other hip, or any other condition which might be expected to affect rehabilitation.

Patients who had a revision hip on the other side or a poorly functioning contralateral hip replacement were also excluded. Individuals with medical illnesses which were likely to lead to death within five years were also excluded. All patients with osteoarthritis of the hip meeting the inclusion criteria were asked to be part of the randomized clinical trial. After appropriate explanation of the study, the patient was asked to be blinded as to whether a cemented or a cementless total hip arthroplasty had been implanted. The duration of blinding requested was five years. Of the individuals approached, 78% agreed. Of the 22% who refused, the principle reason for refusal included a preadmission bias to a cementless prosthesis (41%), no interest in the study (37%), and a small group who wished to know their implant (22%). Those patients entering the study were stratified by surgeon (CHR/RBB), as well as age - over 60 and 60 and under. They were randomized within each stratum. Patients were assessed preoperatively by an expanded role nurse (KL or RB), the study explained and the consent obtained. Post-operatively, all patients were reviewed in an identical manner at 3 months, 6 months, 12 months and yearly thereafter. Outcome measures analyzed included disease specific (Harris Hip score, d'Aubigne, WOMAC, MACTAR), global outcomes (sickness impact profile), and time trade-off techniques as a measure of utility and functional capacity (six minute walk). The first patient was operated on October 13, 1987 and the 250th on January 14, 1992<sup>9</sup>.

Health-related quality of life measures and the six-minute walk test were administered by a blinded study nurse preoperatively, and at each follow-up visit. Neither the nurse nor the patient were aware of the type of prosthesis the patient had received. In addition, at each follow-up visit, the patients were informed of their response to the quality of the questionnaires of the *previous* visit. This has been shown to decrease variability without decreasing responsiveness<sup>5</sup>.

The health-related quality of life measures (HRQOL) used included each of the following.

1) *The Harris Hip Score*

This is a commonly used instrument that was developed for patients with traumatic hip disorders and contains questions about pain, function and range of motion<sup>7</sup>. The best possible score is 100. Pain and mobility account for the majority of the score (44 and 47 points respectively).

2) *The d'Aubigne Score*

This instrument consists of three dimensions that receive equal weight, namely pain, mobility, and ability to walk<sup>13</sup>. The best possible score is 18.

3) *The WOMAC Osteoarthritis Index*

The WOMAC is a disease specific questionnaire developed for patients with arthritis of the hip or knee<sup>1</sup>. It



consists of three dimensions (pain - 5 questions, stiffness - 2 questions, physical function - 17 questions). The questionnaire is reproducible and has been shown to be responsive to change in clinical trials<sup>2</sup>. The best score for each item is zero using a 10 centimeter visual analogue scale.

4) *MACTAR*

The MACTAR is a patient specific questionnaire that has been developed and used in patients with arthritis. Prior to surgery patients are asked to identify the five physical activities that are most adversely affected by their hip joint arthritis. Thus each patient has his/her own set of specific activities that are assessed pre- and postoperatively using a 10 cm visual analogue scale with zero being the best possible score.

5) *The Sickness Impact Profile (SIP)*

The SIP is a behavior based questionnaire consisting of twelve dimensions<sup>3</sup>. The ambulation, mobility, body care and movement dimensions can be aggregated to form a global physical score. The SIP scores range from zero to 100 with zero being the best possible score. The SIP was developed in the general population and has good internal consistency and reproducibility and has been previously used to evaluate hip replacement. Because of concern about the length of time that each patient will be willing to be interviewed, three dimensions considered irrelevant for these patients were excluded (eating, concentration and alertness behavior).

6) *Time Trade-Off*

The time trade-off is a utility measure which reflects the improvement in over all health-related quality of life. Utility measures are used to calculate the cost per quality adjusted life years. In this study, utilities were derived using time trade-off technique which essentially asks patients "how many of their current years of life they are willing to give up in order to achieve full health". Prior to asking patients about their current health, patients were asked to rate three hypothetical scenarios which were designed to represent patients with mild, moderate and severe arthritis of the hip. The scenarios each covered six items: pain and stiffness, use of walking aids, analgesic use, night pain, ability to do house work, and socializing.

7) *Six Minute Walk*

The six minute walk is a measure of functional capacity in which the patient walks as far as possible on the same track with identical prompts over a six minute time frame<sup>6</sup>.

Two hundred fifty (250) patients were entered in this study (124 cemented and 126 cementless). In the cemented group there were 64 males and 60 females and in the cementless group 68 males and 58 females. Three

patients in each group did not return for follow-up; however, all of them report good function and none have come to revision. A total of six patients died since the initiation of the study, three in each group.

## RESULTS

Of the 250 patients entered, 206 have been followed one year, 164 two years, 106 three years and 50 four years. For purposes of this paper, only the two year data will be presented.

1. *HARRIS HIP SCORE*

An analysis of the Harris Hip score preoperatively revealed no difference when the cemented group and the cementless group were analyzed (43 vs 42). Using this instrument to assess postoperative recovery, there was no statistical difference in the Harris Hip scores when cement was compared to cementless at any of the follow-up periods up to two years. At two years, the average Harris Hip score for the cemented patients was 96 and for the cementless patients 97. (Fig. 1)

2. *D'AUBIGNE SCORE*

An analysis of the d'Aubigne revealed no difference preoperatively between the cemented and the cementless patient, 9 vs 9. Postoperatively and at each follow-up period there was no statistical difference in the d'Aubigne scores up to two years when cemented hips were compared to cementless (17.4 vs 17.5). (Fig. 2)

3. *WOMAC*

The WOMAC osteoarthritis index is a *disease specific* questionnaire employing a visual analogue scale which assesses three dimensions - pain, stiffness and physical function. Preoperatively the WOMAC pain score averaged 6.0 in both groups. There was dramatic improvement when cement and cementless were compared at each follow-up visit out to two years where the pain score was 1.0 in both groups. (Fig.3) Similar findings were noted for each of the dimensions analyzed by the WOMAC.

4. *MACTAR INDEX*

The Mactar employs a set of *patient-specific activities* that are most adversely affected by this patient's hip disease. Hip disease affects individuals in different ways; however, the disabilities chosen by patients as being most important included difficulty with walking, difficulty with shoes and socks, difficulty with stairs, difficulty standing, night pain, insomnia, aching and soreness. When one analyzed these specific measures and compared cemented to cementless total hip replacement using the Mactar questionnaire, preoperative scores of 7.8 and 7.7 were reduced to scores of 1.0 and 0.67 when cemented and cementless patients were compared at two year follow-up. (Fig. 4)

FIGURE 1  
HARRIS HIP SCORE

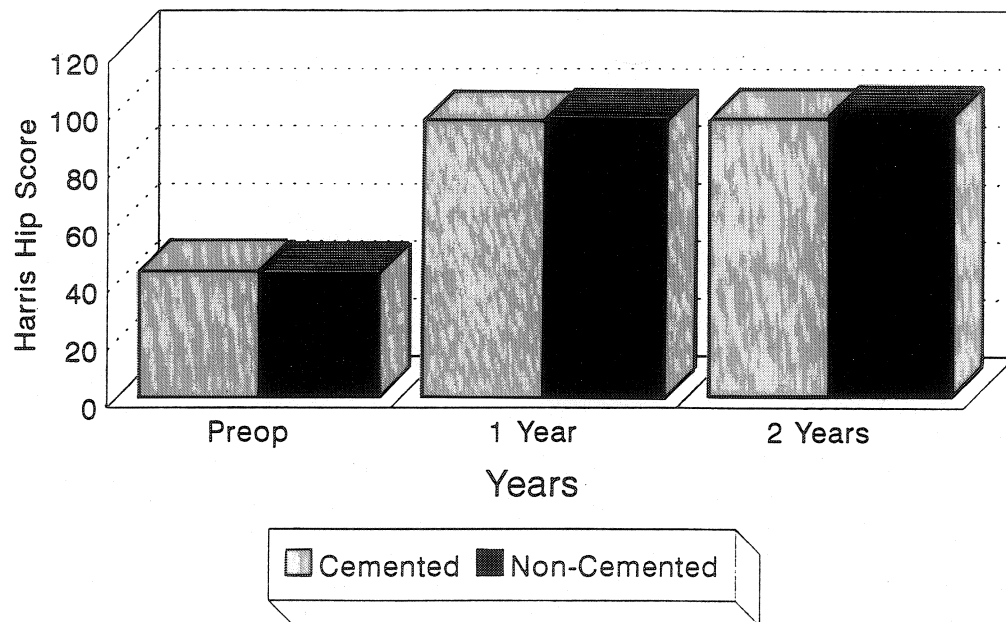
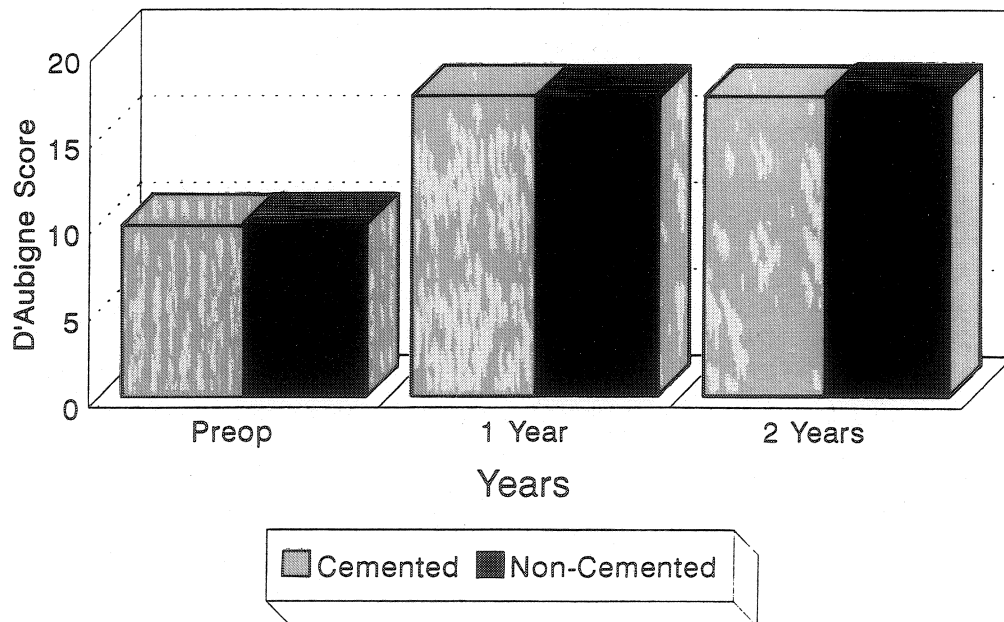
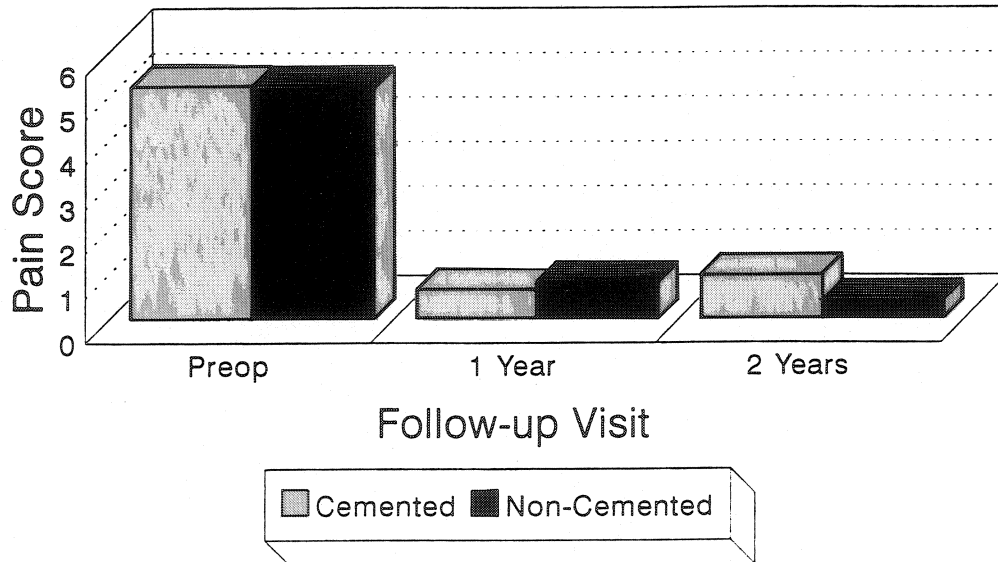


FIGURE 2  
D'AUBIGNE



**FIGURE 3**  
**WOMAC**  
**Pain Score**



**FIGURE 4**  
**RCT: MACTAR PATIENT**  
**SPECIFIC MEASURES**

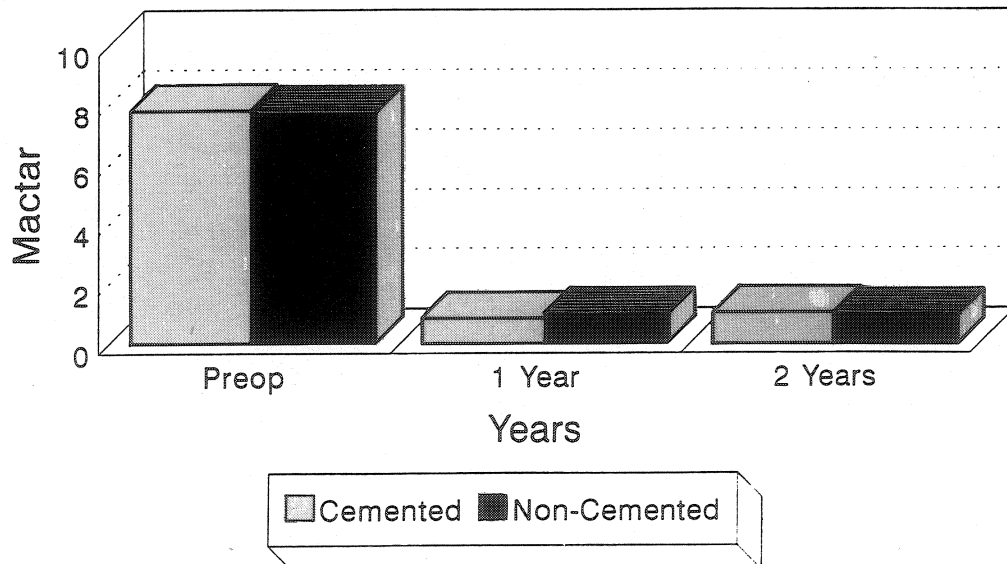


FIGURE 5  
SIP: GLOBAL PHYSICAL

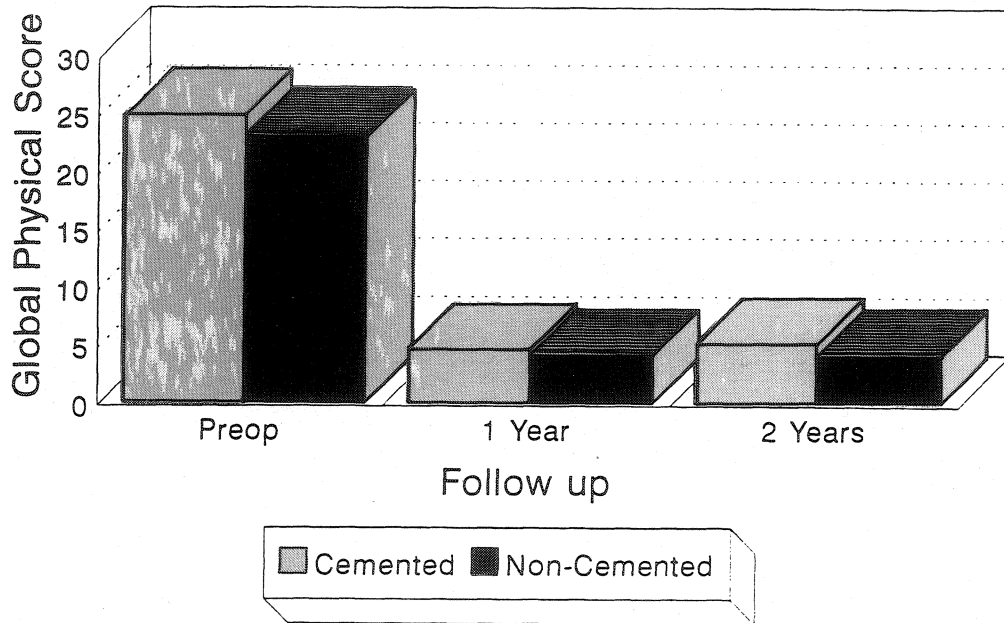


FIGURE 6  
SIP: SLEEP / REST

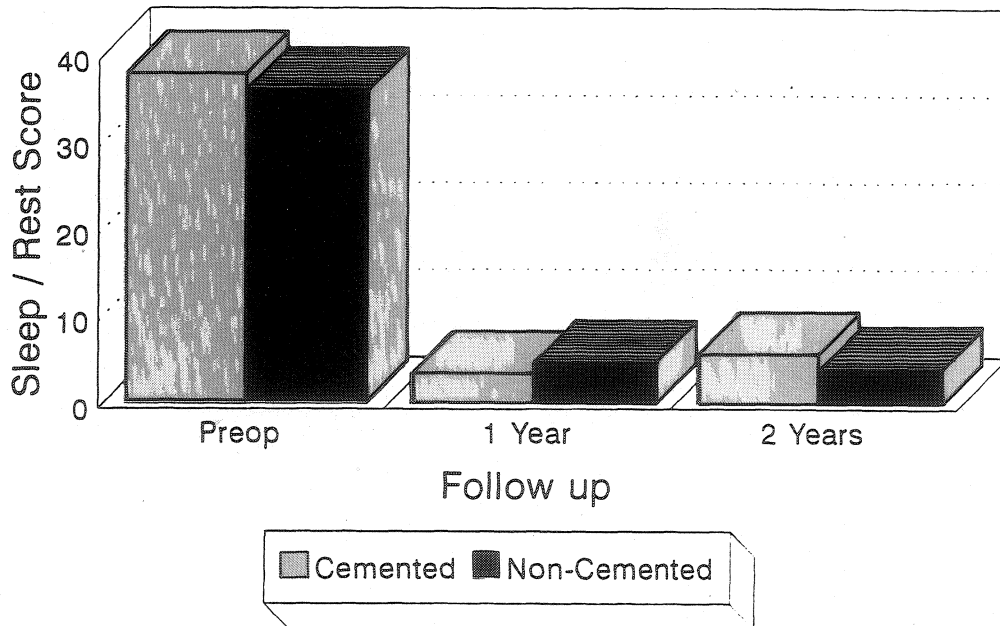


FIGURE 7  
TIME TRADE OFF SCORES

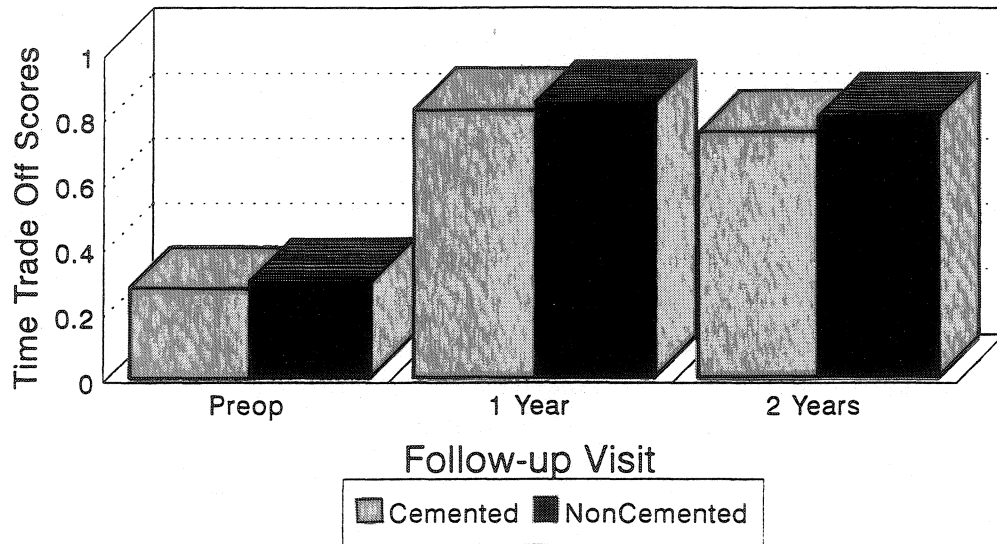
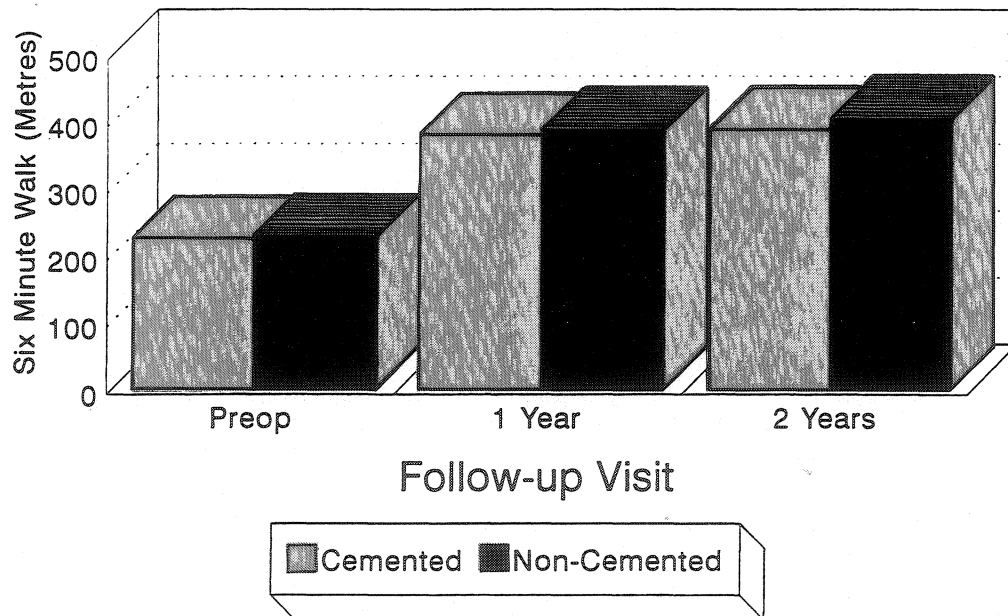


FIGURE 8  
SIX MINUTE WALK



#### 5. SICKNESS IMPACT PROFILE

The sickness impact profile measures the *patient's overall health-related quality of life* by analyzing 12 behaviorally-based dimensions including three physical dimensions (ambulation, mobility and body care) which can be computed to a global physical score. The preoperative global physical score comparing cemented to cementless was 25.2 vs 23.3. An analysis of the postoperative data at each follow-up period failed to reveal any statistically significant difference when cemented was compared to cementless out to two years (5.2 vs 3.2). (Fig. 5) When individual components of the Sickness Impact Profile were analyzed including recreation and past time, sleep and rest, similar results were noted. Total hip replacement, whether it were cemented or cementless, resulted in dramatic improvement in all categories. Preoperatively, it was evident that the patients' osteoarthritis had a dramatic effect on their ability to sleep and rest comfortably with scores of 37.9 for the cemented group preoperatively and 36.1 for the cementless group. Postoperatively, the scores for the same patients were reduced to 5.7 for the cemented patients and 4.1 for the cementless patients at two years. (Fig. 6) Similar findings were noted with each of the behaviorally based dimensions analyzed by the Sickness Impact Profile.

#### 6. TIME TRADE OFF

The time trade off utility is a unidimensional global health-related quality of life measure with a value that ranges from 0.0 (indifference between life and death) and 1.0 (equivalent to perfect health). Patients were asked to rate the three hypothetical scenarios described in the "Methods" section, and also to rate their own current health state. Once again, cemented or cementless total hip replacement had a dramatic effect when this utility was used as an outcome measure. Preoperatively, the TTO averages for current health were 0.28 for cemented and 0.30 for cementless. At two-year follow-up the results were 0.76 and 0.81 respectively. Once again, there was no statistically significant difference when cemented were compared to cementless total hip replacements. (Fig. 7)

#### 7. SIX MINUTE WALK

The six minute walk measures the distance a patient is able to walk back and forth along a 30 metre course in six minutes with the same individual administering the test and with the same prompting<sup>6</sup>. This is done preoperatively and at each follow-up visit. Total hip replacement whether cemented or cementless had a dramatic improvement on the distance walked using this measure. Preoperatively, the cemented patients were able to walk, on average, 227.1 metres while the cementless patients were able to walk 229.1 meters.

At two-year follow-up the cemented patients were able to walk on average 392.0 and the cementless 408.5 metres. (Fig. 8) There was no statistically significant difference between the two groups.

#### 8. ECONOMIC

The cost of total hip replacement, comparing cemented to cementless was evaluated from society's perspective. Sixty patients form the basis of the detailed cost analysis for hospital in-patient costs. A cohort of 100 patients provided data on physician charges, follow-up charges and the cost to the Canadian health care system and Canadian society through the first postoperative year. Costs included visits to physiotherapists, to doctors, lost time from work, readmission to hospital, etc. These data expressed in 1988 Canadian dollars indicated the average cost of all patients (cement or cementless) to the system, including all physician fees, implant costs and time in the hospital was \$9,990. When one broke down the cost between cemented and cementless, the cemented implant cost the system \$9,853, and those receiving a cementless implant cost the system \$10,119. This difference relates to the difference in the cost of a cementless implant. An analysis of the outpatient cost for the first year postoperatively demonstrated an average cost to society of \$1,137. Once again, when one compared cemented to cementless there was little difference between the two groups. The average cost to the system for the cemented group was \$975 versus \$1,297 for the cementless group. The difference in costs in the first year postoperatively between cemented and cementless relates largely to the considerably longer distance the cementless patients had to travel to visit their doctor as opposed to the cemented patients. Distance from the University Hospital was *not* stratified as one of the variables and as a result, it evolved that the average cementless patient had to travel 223 kilometers to see their orthopaedic surgeon, whereas the average cemented patient had to travel only 142 kilometers. The number of physiotherapy doctor visits were identical for the cemented and cementless groups and the difference in costs is explained entirely by the travel distances. As well, an outpatient cost analysis including nursing visits, social service visits, readmissions to the hospital, etc. demonstrated no statistical difference when the cemented group was compared to the cementless group for the first year postop<sup>8</sup>. (Table 1)

Total hip arthroplasty seems quite cost effective compared to other interventions. Using our data, it is possible to calculate the cost per quality adjusted life years associated with total hip arthroplasty. Prior to surgery, the average patient assessed utility was ap-

**TABLE 1**  
**Out-Patient Care (1988 \$ Canadian)**

	<i>All Patients</i>	<i>Cemented</i>	<i>Non-Cemented</i>
Physiotherapy	203	199	207
<i>Orthopaedic Clinic</i>			
—physicians' fees	123	123	123
—travel and parking	217	172	262
<i>Visits to Physician</i>			
—physicians' fees	16	17	16
—travel and parking	7	7	7
—opportunity cost	19	20	18
<i>Visits to Patient</i>			
—nurse/physiotherapist	72	44	99
—social services	76	45	107
<i>Admissions to Hospital - Hip Related</i>			
i) London	246	238	254
ii) Non-London	158	110	204
<b>TOTAL HIP RELATED COSTS:</b>	<b>1137</b>	<b>975</b>	<b>1297</b>
<i>Admissions to Hospital:</i>			
<i>Non-Hip Related</i>			
i) London	546	584	509
ii) Non-London	239	170	307

proximately 0.29 which increased to about 0.84 by three months, a change of 0.55. This improvement was maintained for the rest of the first year. Therefore, the average quality adjusted life year (QALYS) gained during the first year was  $0.55 \times 0.75 = 0.41$ . The cost per QALY gained from hip arthroplasty is therefore  $\$11,127/.41 = \$27,139$ . If one assumes that the improvement in health related quality of life is sustained during the first three years (a reasonable assumption based on our data), and that the cost of hip related care is approximately \$500/year for each of the second and third years, then (ignoring discounting) the cost per QALY gained during the first three years after surgery is \$8,031. Using a recently suggested classification system for grading media technologies, hip arthroplasty is a Grade B technology (costs less than \$20,000 per additional QALY)<sup>8</sup>. Grades vary from Grade A (technology is both less costly and more effective than the relevant alternative) to Grade D (technology costs more than \$100,000 per additional QALY relative to the alternative, or is less effective and saves less than \$20,000 per QALY gained) to Grade E (technology is

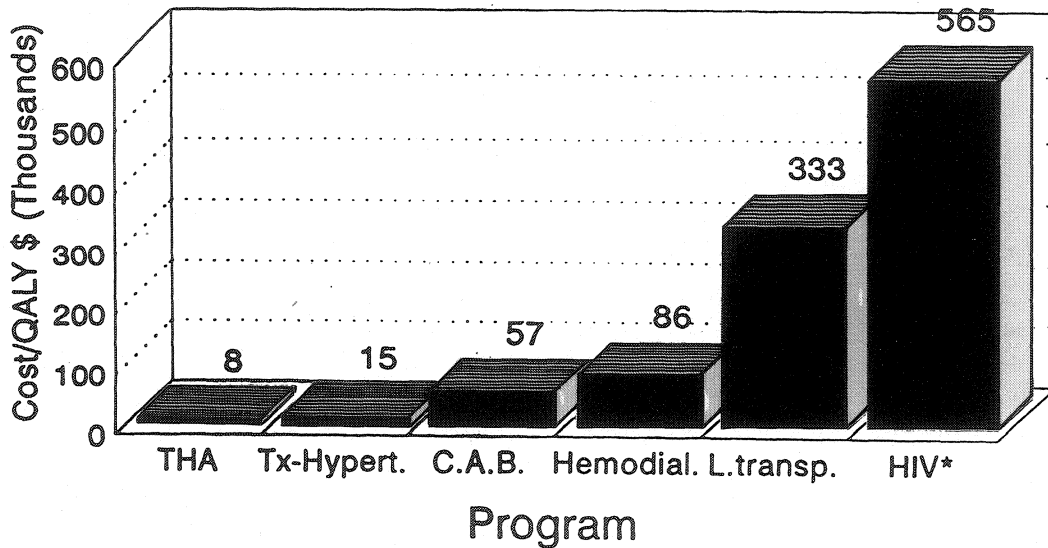
more costly and less effective)<sup>8,10</sup>. Figure 9 graphically depicts the efficacy of total hip replacement compared to other medical interventions (i.e. the treatment of moderate hypertension, coronary artery bypass for angina, hospital hemodialysis, liver transplantation and HIV universal precautions).

### DISCUSSION

This study demonstrated that it is possible to assess the efficacy of surgical intervention using a randomized clinical trial. Its strengths include the fact that the study was randomized, prospective, and has virtually complete follow-up with accurate cost data. To the best of our knowledge a study of this kind has not been reported in the orthopaedic literature.

The preoperative responses to the questionnaires administered to the patient indicate that osteoarthritis of the hip not only causes pain but also severely affects the individual's physical activity, social interactions, and over all health. While we, as orthopaedic surgeons, recognize that our patients are severely disabled, I don't believe that many of us recognize how severely disabled our patients

FIGURE 9  
**COMPARATIVE COST-UTILITY RATIOS**



\*cost/additional life saved

are. The preoperative sickness impact profile and time trade-off scores suggest that patients regard their health related quality of life as being as adversely affected than patients on chronic dialysis.

The improvements in health related quality of life after total hip replacements are rapid and complete, and affect all aspects of the patient's over all well being. For example, the improvement in the time trade-off score was particularly impressive. Two years postoperatively, the mean score was 0.79 which is better than the score these patients assigned to the best hypothetical scenario of a patient with mild arthritis. By comparison, the time trade off score of anemic dialysis patients did not improve once their anemia was corrected with erythropoietin, despite marked improvement in symptoms of fatigue<sup>4</sup>. The likely explanation for the difference is that a successful hip replacement returns most patients to near normal activity, while the dialysis patients are still uremic, on dialysis, and may continue to suffer from co-morbid conditions such as coronary artery disease.

This study has demonstrated that the in-hospital costs of total hip replacement, as well as the outpatient costs during the first year, did not show statistical differences when the cemented patients were compared to the cementless patients. The major costs during the first year postoperatively were for routine out patient visits to an orthopaedic clinic (4 during the first year), and the cost of

readmission for hip related problems (suspected infection and deep vein thrombosis). Seven of the 8 readmissions occurred during the first six months after surgery and thus it is likely that costs will decrease over the ensuing years, provided the hip continues to function well.

These data clearly indicate that comparing the cost of cemented and cementless hip prostheses will only differ significantly if there is a difference in the rate of revision surgery. Even though the cost of revision procedures must be discounted for a time, a large difference in revision rates between the two types of prostheses five or ten years after the original procedure *could* have an important economic impact. The authors plan to follow this cohort of patients for ten years, and thus should be able to address this important question.

The weaknesses of the study include the following. No attempt was made to estimate the cost of maintaining patients on the waiting list prior to coming into hospital, and a study of that is currently underway. The costs of relatives traveling to visit patients during the initial hospitalization were not calculated. In addition, we relied upon patients' diaries to estimate outpatient resource use. Nevertheless, we were impressed by the diligence with which patients recorded hip related events.

A number of clinically relevant conclusions can be reached:



- 1) Surgical patients are willing to be part of a meaningful randomized clinical trial. In this study, 78% of eligible patients agreed to participate.
- 2) Total hip replacement is an amazingly efficacious procedure, converting a patient with severe compromise of their quality of life to near normal health.
- 3) Total hip replacement compares favorably to virtually any medical or surgical treatment modality in terms of cost to quality adjusted life years.
- 4) Cemented and cementless total hip replacements are virtually identical in terms of patient specific, disease specific, global, utility, functional capacity and cost related outcome measures.
- 5) In an era of cost containment, data such as this study will have an important effect in insuring that cost effective treatments remain funded.

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# THE PREVALENCE AND NATURAL HISTORY OF EARLY OSTEONECROSIS (ON) OF THE FEMORAL HEAD

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## ABSTRACT

We performed limited MRI exams of the hips of renal transplant patients to determine the prevalence of osteonecrosis (ON) and the natural history of early lesions. Of 132 subjects, ten patients and 15 hips were considered positive for ON (prevalence = 7.6%, bilaterality = 50%). Eleven of the MRI-positive hips were Ficat Stage 0 (asymptomatic, pre-radiographic) and were followed with serial radiographs and MRI exams. With an average follow-up of 22 months, only one of these early lesions progressed beyond Ficat 0. The other ten hips neither developed progressive MRI changes nor progressed to radiographic stages.

Although our follow-up of 22 months is short, the results suggest that early ON may have a benign course in many cases. This supports the recent work of Kopecky et al., who found that many early lesions in renal transplant patients seemingly stabilize or disappear. While the prevalence was lower than in some previous reports, a significant number of patients did demonstrate previously unsuspected disease.

Given the recent reports of poor results and high complication rates using "prophylactic" surgery such as core decompression for early ON, we recommend further study into the natural history of these lesions to assess the need for such procedures.

In addition, we describe the use of a simple, quick and cost-effective method to screen high risk patients for early ON.

## INTRODUCTION

Osteonecrosis of the femoral head, from whatever cause, is known to have a poor prognosis once it reaches the radiographic stages (Ficat II-IV)<sup>1</sup>. Several studies have shown inexorable progression to collapse and advanced degenerative arthritis in most cases treated non-operatively<sup>2,3,4,5</sup>. The only satisfactory treatment for advanced ON is total joint arthroplasty. However, because the disease most commonly affects young adults who are not good candidates for joint replacement, many joint preserving operations such as core decompression have been recommended. All these procedures are reported to give better results if performed early in the course of ON<sup>2,3,6,7,8,9</sup>. Most authors recommend "prophylactically" treating hips diagnosed prior to radiographic changes to halt progression. Thus an emphasis has been placed on early diagnosis using MRI, bone scans, or biopsy, especially in patients at high risk. This has and will continue to result in earlier diagnoses in many cases. However, the natural history of these early lesions is not known. Prophylactic surgery in effect presupposes that these hips will follow the same course as later stages, but no study has documented this assumption. The scant information available suggests the contrary; that is, many early lesions may have a benign natural history and do not require treatment<sup>10</sup>. In addition, none of the numerous investigations into core decompression and other procedures suggest completely satisfactory results<sup>11,12,13,14,15</sup>. Recent literature questions whether surgery alters the natural history of any stage whatsoever.

## MATERIALS AND METHODS

Our research protocol had the approval of our Institutional Review Board. To recruit subjects, we reviewed the computer records and/or charts of patients selected randomly from the list of our transplant clinic. Patients were contacted by phone and asked to participate in the study if they: 1) Were at least 18 years old, had a functioning renal transplant and were at least three months post transplant

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surgery; 2) Had no previous diagnosis of ON; and 3) Were willing to undergo a brief MRI exam. Patients were not excluded for any other reason. Those with hip pain but no previous diagnosis of ON were included in an attempt to identify both Ficat Stage 0 and I.

Of the approximately 220 patients contacted, 132 underwent the initial MRI screening exam. Those patients not scanned included seven patients already diagnosed with ON while the remainder failed to show, became claustrophobic, or could not fit in the scanner. Prior to the procedure, the exam, and scope and purpose of the project were explained to each patient. Each patient signed informed consent and filled out a questionnaire regarding hip pain and limp, in order to stage positive scans. Data collected from the chart of each patient tested included: age, weight, cause of renal failure, time since transplant, serum creatinine level, current steroid dose, and estimated cumulative steroid dose.

The first 63 patients had scans in the coronal plane; the remaining scans were done in the axial plane to accommodate a related study. Images were obtained with either a Picker Vista (Picker International, Inc., Highland Heights, OH) 0.5 Tesla magnet or a General Electric Signa (General Electric Company, Milwaukee, WI) 1.5 Tesla magnet. For the Picker scanner, scan parameters included 5 mm slice thickness, no interslice gap, a spin-echo pulse sequence with TR = 600 and TE = 20, a 192 x 256 acquisition matrix, and four signal acquisitions. For the General Electric unit, scan parameters were similar with a 5 mm slice thickness, 1.5 mm interslice gap, a spin-echo pulse sequence with TR = 600 and TE = 12, a 192 x 256 acquisition matrix and two signal averages. Total examination time was approximately 15 minutes, including patient positioning. A grant was charged \$326 for each scan based upon an estimate of the resource expenditure, excluding physician fees (no patient was charged).

One of two musculoskeletal radiologists (DR and CW) interpreted the MRI examinations at the time they were performed. They interpreted the scans as either normal, indicative of ON, or equivocal. MR images were considered normal if the signal intensity of the femoral head was uniform throughout. Images were indicative of ON if they demonstrated crescentic areas of low signal intensity in the weight-bearing portion of the head, rings of low signal intensity, or collapse of the femoral head<sup>16,17</sup>. After an interval of six months, the same two musculoskeletal radiologists interpreted all images in conference without knowledge of the original interpretation of the scans. We compared the original and consensus interpretations.

Anterior-posterior and Lowenstein lateral pelvis radiographs were obtained on each patient whose MRI was considered positive or equivocal. These films were also read independently and in consensus by the same two

radiologists. These patients also had a complete history and physical exam of the hips including palpation, ROM, Trendelenberg sign and assessment of gait. The hips were staged according to a modification of the Ficat staging system<sup>7</sup>:

- 0 - asymptomatic, normal exam and normal x-rays
- I - symptomatic and abnormal exam, normal x-rays
- II - trabecular changes on x-rays w/o collapse
- III - radiographic collapse w/o DJD
- IV - radiographic DJD

To differentiate Stage I from Stage 0, a patient had to have more or less constant hip or groin pain with a concomitant exam demonstrating limited hip motion, pain with range of motion, and limp. Those hips diagnosed in Stage II and beyond were excluded from the study and the patient offered referral to a joint reconstructive surgeon. Patients with pre-radiographic stages were followed with repeat plain films every six months, and a repeat MRI every year. All imaging studies were reviewed by the same musculoskeletal radiologists. If a patient progressed into a radiographic stage he/she was offered referral to a joint reconstructive surgeon.

Logistical regression analysis was used to determine whether or not any of the various clinical parameters predicted the occurrence of ON.

## RESULTS

The MRI images in every case were similarly interpreted on the initial reading and later in conference. Two hips were initially read as equivocal by both observers but were later considered normal by consensus opinion. One other initially equivocal reading was rated equivocal by consensus six months later. The femoral head in this case had changes consistent with either necrosis or a bone cyst. This hip was followed per protocol, did not change in appearance on MRI, and no radiographic changes developed; it was not included in the group diagnosed as ON. In no case was a T-2 weighted image considered necessary to confirm or refute the diagnosis; an original exam was repeated to confirm artifact in one case and to confirm ON in another.

Of the 132 tested, ten patients and 15 hips had MR changes consistent with ON. Thus, the prevalence of ON in these patients was 7.6%, and lesions were bilateral in 50% of patients. After a thorough history, physical exam and A-P and Lowenstein lateral radiographs, the hips were staged. Eleven of the 15 were Ficat 0 (four of these eleven hips were described as painful but did not meet the criteria for Ficat 1). Two were Ficat II and two were Ficat III. There were no Ficat Stage I or IV hips. None of the four patients with radiographic lesions had previously sought medical attention for their hip, and they did not desire consideration for surgery. Because three of the four had contralateral pre-radiographic lesions, both hips were

followed in the study for progression. Therefore, all 15 hips were discovered because of the screening exam, and otherwise would have remained undiagnosed. Eleven of the 15 were radiographically normal, diagnosed solely by MRI.

The patient's age, weight, cause of renal failure, time since transplant, creatinine level, current and estimated cumulative steroid dose were not significant predictors of ON, individually or in combination.

The 11 pre-radiographic hips were followed with serial radiographs every six months and an MRI every year. Average follow-up was 22 months (range six to 29 months) and eight hips in six patients had a greater than two year follow-up. Only one hip progressed, from Ficat 0 to Ficat II. This change occurred one year into the study and he remains asymptomatic on that side. He has a Stage III lesion on the contralateral side with moderate symptoms. Every other early lesion remained essentially the same by MRI and did not progress into radiographic stages. None of these patients developed new symptoms and all remained Ficat 0 at the last follow-up.

#### DISCUSSION

Osteonecrosis, most commonly of the femoral head, is a well known complication of renal transplantation. Previous reports of the incidence have ranged from 3 to 41%, with most estimating around 20% based on radiographs and/or bone scans<sup>18</sup>. However, two revolutions have occurred since these studies. First, better immunosuppressants have afforded a reduction in steroid dosing, and some evidence suggests this has lowered the incidence of ON<sup>18</sup>. Second, MRI has emerged as the most sensitive exam for detecting ON, especially in the earliest stages<sup>17,19,20,21</sup>. The incidence or prevalence of ON at this time using MRI is not well known. Some studies have recently used MRI as a screening exam for ON, and others have suggested screening all patients at high risk<sup>10,19,22,23</sup>. A current estimate of the expected prevalence should be known prior to such wide scale screening with an expensive test.

Using an abbreviated screening MRI, we found a prevalence of ON of the femoral head of 7.6%, and lesions were bilateral 50% of the time. Of note, none of the ten patients we discovered had previously sought medical attention for the hip(s), including the four Ficat Stage II and III hips. Eleven of the 15 hips were radiographically normal, diagnosed solely by the screening MRI. Therefore, each patient could potentially benefit from being diagnosed by a screening exam, and specifically the MRI in most cases.

This prevalence of 7.6% agrees closely with that reported recently by Tervonen, et al., who discovered 6.0% of asymptomatic renal transplants had ON using a similar abbreviated MRI<sup>23</sup>. Both of these figures are lower than

previous reports, possibly due to decreased steroid dosing or other factors. This lower estimate should be kept in mind prior to considering any wide scale screening of high risk patients.

Neither in our study nor that of Tervonen, et al., could specific risk factors for ON be identified. Therefore, selective testing of subsets of particularly high risk transplant patients does not seem feasible at this time.

We elected to use an abbreviated T-1 weighted MRI for several reasons. For one, T-2 images are less accurate for detecting ON, and frequently only confirm the diagnosis made by T-1<sup>10,24</sup>. Secondly, scanning in one plane should be adequate to make the diagnosis. This reduced scan cost to \$326, approximately one-third the cost of a standard hip MRI. The exam time of 15 minutes is also one-third the standard exam time. This more cost-effective exam should be considered if screening of high risk patients is undertaken. The fact that each scan was interpreted similarly by the two radiologists and that only one scan was considered equivocal throughout the study supports the use of this limited exam. These relatively quick and inexpensive scans were believed useful to scan for and study the natural history of ON in high-risk groups.

The goal of any surgical intervention is to positively influence an otherwise adverse natural history. This implies that treatment is available to improve the prognosis. In the case of early ON, the natural history has been presumed to be the same as later stages (i.e., progression to collapse and secondary degeneration), and it has been further presumed that intervention such as core decompression with or without grafting would halt this progression. However, the natural history of early lesions is unknown and surgical procedures have had variable reports of success.

Using core decompression, Ficat reported excellent results for Stage I and II only, and stated the "prognosis depends entirely on early diagnosis and effective treatment"<sup>7</sup>. Hungerford and Zizic have reported similar results supporting early diagnosis and core decompression, and recommended against coring beyond Stage II as it did not prevent femoral head collapse<sup>2,3</sup>. These authors advocated a functional exploration of bone on any suspicious, radiographically negative hip. If intraosseous hypertension or evidence of ON existed on venography, they recommended proceeding with core decompression, noting a high correlation of these tests with eventual biopsy results. Steinberg, et al. reported better success in earlier stages using coring with added cancellous bone grafting, with or without electrical stimulation<sup>9</sup>. They advocated using MRI in suspicious cases to diagnose early lesions, but noted that ON could be present even with a negative MRI. Finally, Meyers found limited success using vascularized muscle pedicle grafts for lesions already displaying

a crescent sign, compared to outstanding success in treating early lesions<sup>8</sup>. The initially encouraging results in treating early ON, along with other supportive studies<sup>6,25</sup>, prompted enthusiasm for making the earliest diagnosis and intervening early with core decompression and other procedures.

However, most recent literature has reported higher failure and complication rates for these procedures. Camp and Colwell reported progression in 60% of Stage I and II hips treated with core decompression at a mean follow-up of 18 months and a ten percent incidence of perioperative femoral fracture<sup>11</sup>. Similarly poor success was demonstrated by Hopson and Siverhus, Saito, et al., and Seiler, et al<sup>15,12,26</sup>. Learmonth, et al., reported the highest rate of failure, with radiographic progression in 75% of Stage I and 86% of Stage II hips at a mean follow-up of 31 months<sup>13</sup>. Nelson and Clark reviewed the efficacy of structural bone grafting, and found progression in at least 82% of hips with a minimum of two year follow-up<sup>14</sup>. These studies have tempered the enthusiasm for prophylactic surgery for ON, and the efficacy of surgery to preserve the natural femoral head in any stage has to be questioned.

More importantly, the untreated natural history of these early lesions is largely unknown. The available evidence suggests a more benign course than previously presumed. Kopecky, et al., discovered 14 patients and 25 hips with MR changes consistent with ON by prospectively scanning 106 renal transplant patients serially two years after transplant<sup>10</sup>. The MR images returned to normal in six of these hips and the lesions decreased in size in seven hips in five asymptomatic patients. Only seven hips (28%) in four patients developed pain and radiographic changes over a mean follow-up of 16 months.

In the current study, only one of the eleven Ficat Stage 0 hips progressed into the radiographic stages. The other ten hips have not developed new symptoms, radiographic changes or a change in appearance by MRI. The mean follow-up of 22 months is short, and other hips may progress over time. However, previous studies documenting progression in later stages have shown progression to occur over periods less than this follow-up<sup>4,5</sup>. We will continue to follow these hips for progression to determine their long-term natural history.

In conclusion, we describe a quick, simple and relatively inexpensive limited MRI to screen patients at high risk for ON of the femoral head. Using this exam, ten of 132 patients had 15 hips with ON; these cases would otherwise not have been diagnosed. However, given the rather benign natural history of these lesions, and that prophylactic surgery may not alter the natural history, this study provides additional evidence as to the questionable appropriateness of such procedures.

## ACKNOWLEDGMENTS

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# BILATERAL HUMERAL HEAD OSTEONECROSIS FOLLOWING SPINAL CORD INJURY: A CASE REPORT ILLUSTRATING THE IMPORTANCE OF ADHERING TO THE RECOMMENDATIONS OF THE SECOND NATIONAL ACUTE SPINAL CORD INJURY STUDY

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## ABSTRACT

Five years prior to the 1990 publication of the Second National Acute Spinal Cord Injury Study (SNASCIS), a 24-year-old man sustained traumatic paraplegia, and was treated with 797 mg of dexamethasone over the ensuing 26 days. Within three years he developed symptomatic bilateral humeral head osteonecrosis. Although his total steroid dose was less than one-third of the comparable dose recommended by the SNASCIS, the duration of administration of the steroid was much longer. This case illustrates the importance of adhering to the guidelines established by the SNASCIS, especially regarding the 24-hour administration period.

## INTRODUCTION

The recommendations of the Second National Acute Spinal Cord Injury Study of 1990 established guidelines for the total dose and duration of steroid administration. In order to maximize cord function and avoid steroid complications, high dose, short term steroid administration was recommended<sup>3</sup>.

Previous reports have discussed the relationship between steroid administration and humeral head osteonecrosis in a variety of situations,<sup>1,5,6,7,9,15,21,28</sup> but have not addressed this issue in spinal cord injury patients. In this case report we describe the course of a patient, injured 5 years prior to publication of the SNASCIS, who sustained a spinal cord injury and developed bilateral humeral head osteonecrosis after receiving a relatively small total steroid dose over a relatively long period of time. His course illustrates the importance of adhering to the steroid administration protocol established by the SNASCIS.

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This patient was treated at Holy Family Hospital and subsequently at St. Luke's Memorial Hospital, Spokane, Washington

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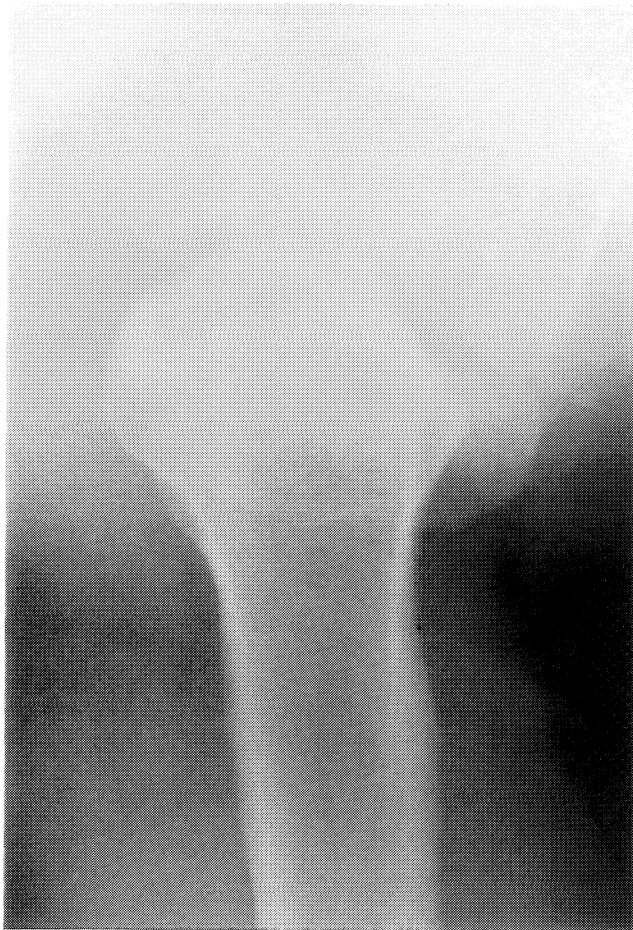
Acknowledgment: Lisha Wilson typed the manuscript

## Case Report

In 1985, a 24-year-old man fell from a tree, landed on an exposed tree root, and sustained an L1 burst fracture. Witnesses noted slight movement of the lower limbs immediately after the injury, but on initial clinical examination the patient had a complete L1 level paraplegia. Myelography revealed a complete block of the dye column at the L1-2 level. A CT/myelogram revealed fractures of the L1 body, pedicles, and left transverse process, with a complete block due to hemorrhage and edema. The canal was intact with no extradural compression.

Due to the possibility of an incomplete cord lesion, the patient received intravenous dexamethasone. Initially he was given 10 mg every 6 hours for 3 days, then 25 mg every 6 hours over the following 4 days, then 4 mg every 6 hours for the next 19 days. The instability of the L1 fracture was apparent after an initial attempt at mobilization in plastic body shells. Therefore, on the twenty-sixth day after injury, a posterior fusion with allograft bone and stabilization with Harrington distraction rods from T10-L4 was successfully performed. Steroids were not resumed post-operatively. His post-operative course was uncomplicated. After two months of rehabilitation, he was discharged from the hospital.

Three years after injury, the patient, employed as an electronics technician, noted the gradual onset of bilateral shoulder pain. There was no history of antecedent trauma. The pain was present when he performed wheelchair transfers and occasionally at night. Initially, it was mild and he was able to continue working. Seven years after injury, the patient returned for evaluation with the complaint of progressive bilateral shoulder pain. Physical examination revealed well-developed shoulder girdle musculature, decreased shoulder abduction bilaterally, and marked pain on external rotation and internal rotation. External rotation was limited to 20 degrees on the right, 10 degrees on the left. Plain radiographs showed collapse and sclerosis consistent with osteonecrosis of both humeral heads (Fig. 1). Magnetic resonance imaging (Fig. 2) delineated the extent of the bilateral humeral head involvement. A radiographic survey of hips, knees, and ankles revealed no further sites of necrosis.



Figures 1A and 1B:

Plain radiographs demonstrate the grade 3 osteonecrosis of the domes of both humeral heads with increased subchondral density, cortical fractures, and slight collapse of the articular surfaces.

Despite the pain, the patient continues to work as an electronics technician. Arthroscopic debridement was considered; however, the patient elected to proceed with nonoperative treatment.

### DISCUSSION

Since the publication of the Second National Acute Spinal Cord Injury Study in 1990, it has become standard practice in patients with acute cord injuries to administer methylprednisolone, initially 30 mg/kg of body weight intravenously as a bolus, followed by infusion of 5.4 mg/kg/hour over 23 hours. Steroid administration beginning more than eight hours after the injury has not been shown to improve neurologic outcome. The study was multi-centered, randomized, double-blind, placebo-controlled, and submitted to rigorous statistical analysis. The recommended steroid regimen has been incorporated into most spinal cord injury treatment protocols.

At the time of injury, this patient weighed 93.2 kg. According to the presently recommended regimen, he would have received 2,800 mg of methylprednisolone

initially, followed by 11,600 mg over the following 23 hours (for a total of 14,400 mg methylprednisolone). This patient's injury occurred in 1985, five years prior to publication of the SNASCIS. He empirically received a total dose of 797 mg of dexamethasone in an attempt to optimize cord function. This dose is equivalent to 4300 mg methylprednisolone, or 30% of the presently recommended dose<sup>24</sup>. However, despite the relatively low steroid dose, this patient received dexamethasone over twenty-six days, rather than the twenty-four hours recommended by the SNASCIS. It is widely known that even low doses of steroid over several days can induce osteonecrosis. Conversely, we were unable to find a single report of osteonecrosis following the administration of high-dose methylprednisolone over the very short period of time recommended by the SNASCIS.

Jones described several prothrombotic factors that increase the likelihood of steroid-induced osteonecrosis<sup>16</sup>. He described a corticoid dose exposure threshold of about 2,000 mg prednisone (this patient received the equivalent of 5,300, mg prednisone). In addition to receiving dexam-





Figures 2A and 2B

Magnetic resonance imaging delineated the full extent of necrosis almost to the epiphyseal line, but with bilateral sparing of small lateral columns of viable bone. There are narrow bands of decreased signal intensity surrounding necrotic bone (intermediate intensity).

ethasone, this patient had other prothrombotic factors that reduced the threshold for osteonecrosis, the spinal cord injury itself, spinal column surgery and cigarette smoking. Rafael noted an increased incidence of osteonecrosis from release of fat emboli and tissue thromboplastins with spinal cord injury, and has reported the highly thromboplastic properties of injured neural tissue<sup>25</sup>. Indeed, this patient's MRI revealed high signal intensity in the distal cord and conus medullaris, indicating cord hemorrhage and edema.

The patient smoked one pack of cigarettes per day at the time of his surgery. Matsuo has estimated that this increases the relative risk of osteonecrosis to 3.9 times that for a non-smokers<sup>19</sup>.

The serum lipids, prothrombin time, partial thromboplastin time, bleeding time, and fibrin-split products were all normal, both before and after surgery. There was a brief period of thrombocytosis after surgery, otherwise the platelet count was normal throughout hospitalization. There were no bleeding problems encountered. Arterial pO<sub>2</sub> was normal in the perioperative period. During surgery, small pieces of thrombin-soaked gelfoam were uti-

lized for local hemostasis. These were used in such small amounts, accompanied by such copious and frequent irrigation, that they were not felt to be a cause of the osteonecrosis. We routinely use thrombin-soaked gelfoam in scoliosis surgery and low back surgery without the problems of thrombosis or osteonecrosis.

This patient never exhibited any symptoms of fat embolus, such as obtundation, dyspnea, axillary petechiae, hypoxemia, or hyperlipidemia. He had taken no medications prior to his injury, had no steroids after surgery, was a rare social drinker, and did not have diabetes.

Anderton and Helm<sup>1</sup> reported a 27-year-old male with increased intracranial pressure and headaches, who had received 112 mg of oral dexamethasone over seven days (a dose about 1/7th the dose our patient received) and two years later developed osteonecrosis of both humeral and both femoral heads. Similarly, Fast and colleagues<sup>9</sup> described a 38-year-old male with cerebral edema due to a head injury who received 366 mg of intramuscular dexamethasone (less than 1/2 our patient's dose) over 19 days, with a maximum daily dose of 36 mg. Only three months

later he developed symptoms of osteonecrosis of both humeral and femoral heads. Cases of multifocal osteonecrosis after short-term steroid therapy have been reported by Good<sup>13</sup>, McCluskey<sup>21</sup>, and Taylor<sup>28</sup>. These complications from such small steroid doses suggest the possibility of an idiosyncratic reaction. These and other authors<sup>10</sup> have suggested that steroid-induced alterations of liver function lead to fat embolization and osteonecrosis.

Solomon<sup>26</sup> believed there was no direct effect of steroids on osseous tissue, but a study of rabbits given high doses of steroids suggested a direct cytotoxic effect on osteocytes<sup>23</sup>. Cruess<sup>6</sup> cited the experimental animal studies of Kawai<sup>18</sup>, which demonstrated that fat emboli caused by corticosteroids led to subchondral osteocyte death. Patterson<sup>22</sup> showed that cortisone caused fatty degeneration of the liver and hypolipidemia. Cruess<sup>8</sup> noted massive amounts of intracellular lipids in the dead osteocytes of steroid-treated rabbits.

Known non-steroid causes of osteonecrosis include alcoholism, hemoglobinopathy, trauma to the affected bone, Gaucher's disease, caisson disease, gout, hyperactivity of the adrenal cortex, systemic lupus erythematosus, and chronic pancreatitis<sup>4,8,11,17</sup>. This patient did not have any of these conditions. We believe he is the first patient to be reported with osteonecrosis due to steroids administered because of a spinal cord injury.

We theorize that prolonged steroid administration, along with the other thrombotic predisposing factors<sup>16</sup> of cord injury, spinal column surgery, and cigarette smoking, were the cause of this patient's osteonecrosis.

As noted by Hayes<sup>15</sup>, limited shoulder ROM in osteonecrosis may be either mechanical (as from an osteochondral fragment interposed between the long head of the biceps and the rotator cuff), or due to pain, as in our patient. Also in agreement with the observations of Hayes, we noted that the subchondral osteonecrosis occurred in the superior portion of the humeral head, and that ordinary activities of daily living are enough to induce subchondral fractures and induce the crescent sign in the necrotic bone. These forces are probably greater in paraplegic individuals, who rely entirely on their upper limbs for mobility.

To our knowledge, there are no reports of humeral head osteonecrosis occurring in patients who have received methylprednisolone administered in accordance with the Second National Acute Spinal Cord Injury Study. The case reported here illustrates the importance of adhering to the recommendations of this study, especially regarding the 24-hour duration of administration.

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# THE EFFECTS OF SPACE FLIGHT ON THE COMPOSITION OF THE INTERVERTEBRAL DISC

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## ABSTRACT

The lumbar annuli of rats flown on a COSMOS space flight were compared with those of three control groups and a ground antigravity tail suspension model. The wet and dry weights of the space flight annuli were significantly smaller than the three control groups. In addition, the collagen-to-proteoglycan ratio was significantly greater in the flight group due to a proportional increase in collagen and a decrease in proteoglycan. Finally, it appears microgravity may have altered the nature of the proteoglycan population as more proteoglycans leached from the annuli of flight animals than control animals when immersed in water.

## INTRODUCTION

Intervertebral discs consist of three separate structures, the nucleus pulposus (NP), the annulus fibrosus (AF) and the cartilaginous endplates (EP). Each consists primarily of collagen, proteoglycans (PG) and water, but the extent of collagen and PG varies markedly among the three structures and is related to the function of each. The EP is hyaline cartilage and is key in absorbing axial loads and in facilitating the diffusion mechanisms necessary for nutrition of the NP and AF<sup>1,10,15</sup>. The AF is primarily composed of types I and II collagen embedded in a PG gel and the NP consists of a high concentration of PG and type II collagen. The NP attracts water and exerts a swelling pressure enabling it to support an applied load while the collagenous AF resists this expansion and enables the disc to sustain the rotation and shear forces associated with movement<sup>7</sup>. Under normal circumstances, the balance between swelling pressure and axial loading determines tissue hydration which relates to the functional and health status of the disc. Temporary changes in environmental conditions, e.g., weightlessness, may alter the composition of the disc which in turn may affect the state of hydration and therefore the ability of the disc to withstand axial loading upon return to normal loading forces.

The effects of weightlessness on the disc have not been well defined. Tyrrell et al.<sup>16</sup> reported fluid loss together with disc deformation as being primarily responsible for changes of nearly 1% in human height between morning and night. Thorton et al.<sup>17</sup> attributed fluid imbibition as being the most likely cause for the 5 cm increase in height associated with spaceflight. In order to investigate the

problem further, in 1988 our laboratory was invited to participate in a joint NASA-COSMOS Biomedical Life Science Conference in Moscow to plan studies for upcoming COSMOS spaceflight missions. At this conference, it was arranged for us to receive intervertebral discs from animals to be flown on the COSMOS 2044 space flight September 29 to October 12, 1989. Ten male Czechoslovakian Wistar rats were flown on this 14-day flight. We received discs from five of these animals and their appropriate counter controls in order to determine the effects of weightlessness on the collagen, PG and water content of the AF.

## MATERIALS AND METHODS

### Mission and Animals.

Ten male Czechoslovakian Wistar rats were flown on the COSMOS 2044 Space Flight for 14 days. Intervertebral discs L3-4 to L6-S1 from rats numbered 6-10 were used in our studies. The Vostok vehicle was launched from the Plesetsk facility in the USSR at 9:30 a.m. on September 29, 1989. The biosatellite made 224 orbits at an inclination of 82.3° with an apogee of 294 km and a perigee of 216 km. During reentry, the animals experienced forces of 3-4G, and on landing the impact reached a maximum of 30G for a few milliseconds. During the flight, ambient temperatures were 23-26.5°C during days 1-11 and on days 12 and 13 for unknown reasons the temperature rose to a peak of 29.4°C. On day 14, the temperature returned to previous levels. The lighting regimen was controlled at 16 hours light to 8 hours dark commencing at 8 a.m. Control animals were represented by five animals from each of the following groups: basal (B), killed at the beginning of the flight; vivarium (V), housed in similar colony cages during the flight time period; and synchronous (S), exposed to simulated flight conditions. In addition, five tail-suspended (T) animals were compared with the flight (F) and control groups. Dietary and water intake were similar for all groups throughout the study.

### Tissue Collection.

At landing, the F rats were transported to the dissection site approximately 30 minutes away by helicopter. The rats were killed by decapitation 3-11 hours after touchdown. The lower spinal segments were harvested, packed

in dry ice and immediately stored at  $-70^{\circ}\text{C}$ . Following recovery of the F tissues, the control tissues were similarly harvested in Moscow and shipped to our laboratories in dry ice. Upon arrival to our laboratories, the L4-5 and L5-6 discs were isolated for biochemical studies and the L3-4 and L6-S1 discs from two animals in each group were used for ultrastructure studies. The L3-4 and L6-S1 disc from a third animal in each group was used for light microscopy and the remaining L3-4 and L6-S1 discs from the other two animals stored as backup tissue. The NP was reamed out from each disc with a 27 gauge syringe needle and all studies were conducted on the AF and the connecting EP.

### Weight of Annuli.

The initial wet weights of the L4-5 and L5-6 annuli were recorded at the time of dissection (Time 0). Each annulus was then immersed in water for two hours and weighed at 30-minute intervals until a stable weight was reached. Each sample was then lyophilized and the dry weight determined. The hydrating medium was saved to determine the amount of PG leaching from the tissue into the imbibing media.

### Light Microscopy.

The L3-4 and L6-S1 discs were thawed while being fixed in a solution containing 1.5% glutaraldehyde (0.05M cacodylate buffer), 1% buffered formalin and 0.5% ruthenium red for 24-48 hours. After fixation, the discs were embedded in paraffin, sectioned at 6 microns and stained with Safranin-O for PG content.

### Electron Microscopy.

The discs for electron microscopy were thawed in 5 ml of cold ( $4^{\circ}\text{C}$ ) 2.5% glutaraldehyde (0.1M cacodylate buffer, pH 7.4) for 30 minutes. Each disc was then divided into anterior, transitional (the area immediately adjacent to the NP) and posterior segments. Small pieces consisting of several laminae were then stripped from each segment, processed with ruthenium red for collagen-proteoglycan relationships and embedded in Spurr embedding resin. Cross sections were examined at 20,000 magnification and longitudinal sections at 60,000 magnification for each sample using a Hitachi H-7000 electron microscope.

### Biochemistry.

The L4-5 annuli from each animal were finely minced, lyophilized and digested with papain at  $60^{\circ}\text{C}$  for 24 hours. Aliquots of each digest containing 150-160  $\mu\text{g}$  of dry tissue were hydrolyzed in 6N HCl at  $130^{\circ}\text{C}$  for four hours. After drying under vacuum, the hydrolyzates were dissolved in 1 ml of water, microfuged and filtered through 45 nm filters. Hydroxyproline was then determined with the use

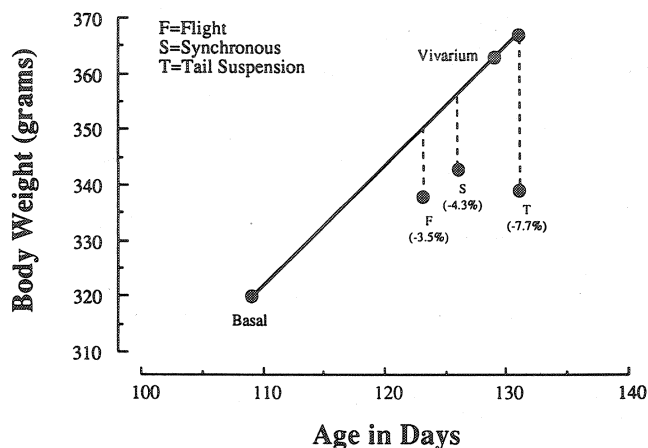


Figure 1.

Body weight-age comparisons of animals involved in COSMOS 2044. If a linear growth rate is assumed between the basal and vivarium groups, F animals are 3.5% below expected values, S controls 4.3% and T animals 7.7%.

of 0.1 ml of the filtered hydrolyzates and 1 ml sample volume according to Woessner<sup>21</sup>.

## RESULTS

### Body Weights.

Due to the demands on the dissection team, the five groups of animals were killed on a staggered schedule. Hence, the B group, sacrificed at the start of the flight, constituted the youngest group and the T group, the oldest group. Assuming a normal linear rate of growth between the B and V groups between 109 and 129 days, the F group was 3.5% less, the S group 4.3% less and the T group 7.7% than predicted by the group curve (Fig. 1). The weights of the F and T groups were not significantly different from the weight of the S group ( $p > 0.05$ ).

### Weight of Annuli.

For each animal, we determined the weights of the L4-5 and L5-6 annuli. The weights of the two annuli were not significantly different; therefore, we used their mean value (L4-6, Table 1) to compare groups. The wet and dry weights of the annuli from the three control groups were not significantly different from each other although the annuli from the youngest group (B) were consistently the heaviest. The annuli from the flight group were 20% to 25% smaller than the annuli from the three control groups ( $p < 0.03$ ), and also significantly smaller than the T group ( $p < 0.002$ ), Table 1.

### Water Content.

The water contents of the annuli were similar among all animals and across all groups. At the time of dissection (Time 0), water represented 52% to 57% of the weight of

**Table 1: Wet and Dry Weights of Annuli of Cosmos 2044 Rats**

GROUPS		BASAL	VIVARIUM	SYNCHRON	FLIGHT	SUSPENDED
Age of Animals (days)		109	129	127	123	131
Weight of Animals (g)		320±5	363±5	343±17	338±5	339±21
Wet Weight of Annuli (mg)						
	L4-L5	12.2±0.5	10.3±2.1	10.3±3.1	7.7±1.1	10.3±1.7
	L5-L6	12.8±3.8	9.8±3.7	10.6±1.3	8.2±2.0	13.6±3.1
	L4-L6	<b>12.5±2.7</b>	<b>10.1±2.9</b>	<b>10.5±2.2</b>	<b>7.9±1.6*</b>	<b>12.0±2.9**</b>
Stable Time						
	L4-L5	21.1±2.4	18.7±4.4	16.5±2.9	15.0±1.5	15.1±1.8
	L5-L6	22.0±4.5	18.2±7.8	19.5±3.4	16.5±3.7	20.5±4.4
	L4-L6	<b>21.5±3.4</b>	<b>18.4±6.0</b>	<b>18.0±3.4</b>	<b>15.8±2.8</b>	<b>17.8±4.5</b>
Dry Weight of Annuli (mg)						
	L4-L5	6.0±1.5	5.0±1.0	4.7±1.3	3.6±0.5	4.5±0.9
	L5-L6	5.5±1.7	4.5±17.7	4.4±0.3	3.6±0.9	6.3±1.5
	L4-L6	<b>5.8±1.5</b>	<b>4.8±1.3</b>	<b>4.5±0.9</b>	<b>3.6±0.7*</b>	<b>5.4±1.5**</b>

All Data = mean ± standard deviation

\*Flight group significantly smaller than all control groups p<.03.

\*\*Suspended group significantly greater than flight group p<.002.

the annuli. When immersed in water, the annuli imbibed water to a constant level representing 70% to 77% of the weight. No significant differences occurred among the groups. It therefore appears that weightlessness had a negative effect on the size of the annulus, but it did not impair the ability of the tissue to imbibe water, expressed as a percentage of the total weight, and to swell.

**Light Microscopy.**

No discernible difference in the light microscopic features of the IV discs as revealed by the Safranin-O reaction for PG was detectable among the five groups. In all specimens there was an intense Safranin-O reaction centrally in the NP and the adjacent transitional zones of AF. Near the periphery, the Safranin-O reaction decreased markedly, with the stain being confined to the pericellular areas (Fig. 2).

**Electron Microscopy.**

Collagen fibril diameters: Mean collagen fibril diameters were determined at the anterior, transitional and posterior regions of the L3-4 discs. One hundred fibrils in each location were measured from each specimen used for electron microscopy, and the fibrils were grouped using 20 nanometer increments ranging from 30 to 270 nanometers. In the anterior and transitional locations, the greatest percentage of fibrils were in the 70-90 nm (80) category in all groups except for the tail suspension group transitional

area in which a slightly higher percentage of fibrils were found in the 50-70 (60) nm class. In the posterior regions, the greatest percentage of fibrils were in the 50-70 (60) nm class in all groups except for the vivarium group in which the peak percentage was found in the 90-110 (100) nm class (Fig. 3). Fibrils in the anterior locations exhibited the greatest overall range in diameters (30- 270 nm). If the frequency distributions are divided into small (<110 nm) and large (>110 nm) components, the anterior regions contain 50% to 70% small fibrils, the posterior area 75% to 90% small fibrils and the transitional area (except for the V group) 90% to 100% small fibrils (Table 2).

**Table 2: Percent Collagen Fibrils Smaller than 100 nm in Anterior, Transitional and Posterior Regions of Lumbar Discs L3-4**

	Anterior	Transitional	Posterior
	L3-4	L3-4	L3-4
Basal (B)	57	93	86
Vivarium (V)	68	65	82
Synchronous (S)	51	98	83
Flight (F)	67	99	75
Tail Suspension (T)	59	100	89

Collagen-proteoglycan relationships: Ruthenium red staining delineated electron dense PG particles in regular

periodic array along the collagen fibrils in all groups. The anterior region exhibited the largest fibrils and fewer particles compared to the transitional and posterior areas. Anteriorly the basal group appeared to have the greatest concentration of granules and the flight group the poorest. In the transitional and posterior regions, there appeared to be an increased density of granules accompanied by a decrease in collagen fibril size (Fig. 4). These areas also exhibited the most intense Safranin-O staining (Fig. 2).

**Biochemistry.**

**Proteoglycans:** It has been reported that PG leach from swollen annuli when placed in an imbibing media<sup>18</sup>. To determine the degree of "leaching out" of the annular PG during two hours of imbibition, we lyophilized the hydrating medium of each sample, redissolved it in 1 ml of water and determined the amount of hexuronate by the



Figure 2.

Light microscopic Safranin-O stain demonstrating intense reaction (red) near nucleus pulposus (NP) with decreasing intensity toward periphery (green), indicating a decreasing PG gradient from the NP to periphery. X90

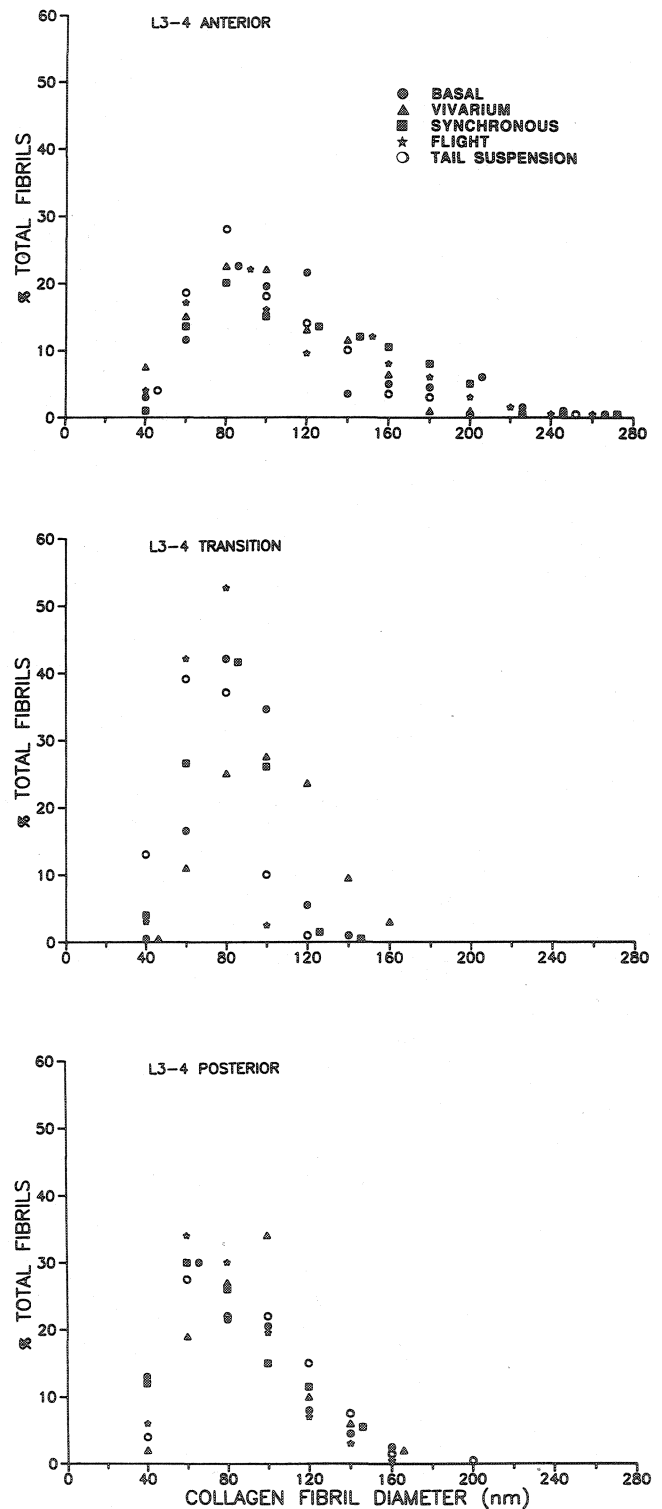


Figure 3.

Collagen fibril diameter frequency distributions in anterior, transitional and posterior regions of the L3-4 discs. Fibrils in the anterior regions exhibit the greatest range in diameters (30-270 nm) whereas transitional fibrils exhibit the smallest diameter range (30-170 nm).

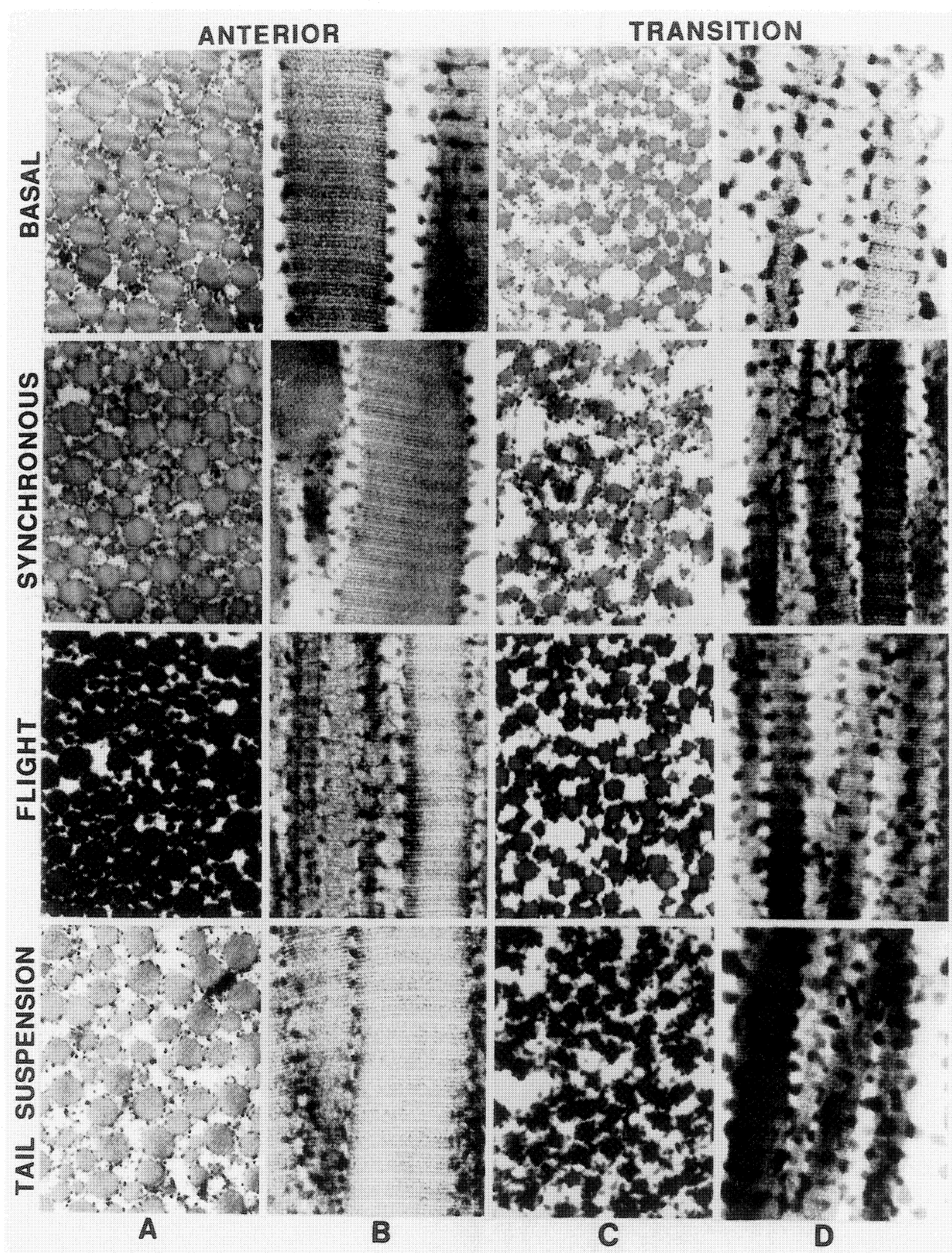


Figure 4. Ruthenium red-stained electron-micrographs demonstrating fibril sizes and collagen-PG relationships in anterior and transit and areas from B, S, F and T groups. Transitional and posterior regions demonstrated an increased PG density and decreased fibril size. Cross-sections X 40,000, longitudinal sections X 90,000.



**Table 3: Hexuronate in the Annuli of Cosmos 2044**

GROUP	TISSUE	MEDIUM		TOTAL	TISSUE	MEDIUM
		(% Dry Tissue)				
Basal (B)	(1.53 ± 0.17)	0.15 ± 0.07		1.68 ± 0.23	91.0 ± 2.7	9.0 ± 2.7
Vivarium (V)	1.53 ± 0.29	0.19 ± 0.03		1.72 ± 0.29	88.9 ± 2.4	11.1 ± 2.4
Synchronous (S)	1.94 ± 0.32	0.11 ± 0.05		2.05 ± 0.32	94.6 ± 2.7	5.4 ± 2.7
<b>Controls (B+V+S)</b>	1.67 ± 0.32	0.15 ± 0.06		1.82 ± 0.32	91.5 ± 0.32	8.5 ± 3.4
Flight (F)	1.35 ± 0.33	0.32 ± 0.14		1.67 ± 0.35	80.8 ± 8.2	19.2 ± 8.2*
Suspended (T)	1.35 ± 0.26	0.20 ± 0.10		1.55 ± 0.35	87.8 ± 3.3	12.2 ± 3.3**

All Data = means ± standard deviations

\*Flight group significantly greater than control gorups combined (B + V + S), p<.001.

\*\*Tail suspension group significantly greater than control groups combined (B + V + S), p<.05.

Blumenkrantz<sup>2</sup> method. The amount of hexuronate remaining in the tissue was determined by the same method after papain digestion of the tissue. The total amount of hexuronate originally present in the annulus was calculated from the amount of hexuronate in the imbibing media plus the amount in the tissue.

The annuli of the B and V groups contained nearly identical amounts of total PG while the annuli of the S group contained nearly 20% more hexuronate. This difference, however, was not statistically significant due to the small sample size. There was also no significant difference between the total hexuronate in the annuli of the F and T groups compared to the control groups individually or when the three control groups (B-V-S) were combined (Table 3).

The amount of hexuronate leaching out from the annuli of the control groups varied from 5.4% in the S group to 11.1% in the V group. The annuli of the F group released 19.2% of their hexuronate into the hydrating media. This was significantly greater (P<.001) than the amount released by the 15 animals in the combined groups (B-V-S). The amount of PG released by the annuli of the T group (12.2%) was also significantly greater (p<.05) than the amount released by the combined (B-V-S) control groups (Table 3).

Collagen: The mean hydroxyproline content of the three control groups (B-V-S) was 5.91 ± 0.80% of the dry weight with no significant difference between groups. The annuli of the F group were significantly more (p<.02) collagenous than the controls with hydroxyproline representing 6.93 ± 0.59% of the dry weight. The amount of hydroxyproline in the annuli of the suspended animals was very similar to the amount in the control tissues and significantly less (p<.04) than that of the F group (Table 4).

**Table 4: Hydroxyproline in the Annuli of COSMOS 2044 Animals**

HYDROXYPROLINE	
(% Dry Tissue Wt)	
Basal (B)	5.71 ± 0.35
Vivarium (V)	5.36 ± 0.45
Synchronous (S)	6.64 ± 0.91
Controls (B + V + S)	5.91 ± 0.80
Flight (F)	6.93 ± 0.59*
Suspended (T)	5.88 ± 0.71**

\*Flight group significantly greater than control groups combined, p<.02

\*\*Suspended group significantly less than flight group p<.04

Collagen-proteoglycan ratio: Based on the assumption that hydroxyproline represents 14% of the collagen molecule and hexuronate 20% of the weight of PG, mean collagen-PG ratios were calculated for each group and for the three control groups (B-V-S) combined. The mean collagen-PG ratio of the F group (7.71) was significantly greater than the three control groups considered individually (p<.04) or combined (p<.001). The T group collagen-PG ratio (6.28) was significantly greater than the S group (p<.05) and the combined control groups (p<.04). No significant differences were found between any of the control groups nor between the F and T groups (Table 5).

Table 5: t-Test-p-Values Comparing Mean Collagen-PG Ratio Between Groups

GROUP	MEAN + S.D.	B	V	S	F	T
Basal (B)	(5.40 ± 0.47)		p<.69	p<.39	p<.02	p<.07
Vivarium (V)	(5.20 ± 1.44)			p<.69	p<.04	p<.19
Synchronous (S)	(4.97 ± 0.91)				p<.02	p<.05
Controls (B+V+S)	(5.19 ± 0.96)				p<.001	p<.04
Flight (F)	(7.71 ± 1.77)					p<.14
Suspended (T)	(6.28 ± 0.84)					

### DISCUSSION

The 14-day spaceflight adversely affected the size of the annulus fibrosus. The wet and dry weights of the annuli from flight animals were significantly smaller than the three control groups and the tail suspended animals. The water content of the tissue expressed as percent of tissue weight was, however, the same in the flight and control groups. The smaller annuli in the flight group could result from a loss of tissue or from a failure to increase the size of the annulus through normal growth. We have no evidence to support either an increase in resorption or a decreased synthesis. The only applicable data, the weights of the annuli, are misleading because their decrease with age is probably related to the closure of the growth portion of the endplate and not to an actual loss of the annulus fibrosus. In previous studies of bone exposed to weightlessness, the reduction in bone has been related to a suppressed growth rate more so than to an increase in resorption<sup>3,8,9,17,19,20,22</sup>. It can be suggested that weightlessness might reduce the contact area between disc and blood supply from the vertebral body, thereby negatively affecting the nutrition of the disc and the metabolic activity of the cells as has been reported to occur in the fused sections of dog spines<sup>6</sup>. It is also plausible that weightlessness might induce hormonal changes that in turn might affect the metabolism of the disc. Further investigations will be necessary to clarify such possibilities.

The water content of the annuli of control and experimental rats at the time of dissection was between 52 and 57% of the weight of the tissue. This is considerably lower than found in 15 samples of human annuli of individuals between the ages of 4 and 76 years where water represents 70% ± 3% of the weight of the tissue<sup>5</sup>. The difference in the water content of rats and human annuli is most probably related to the amount of PG present in the two tissues, the human annulus containing 2.5 times more PG than the rat's annulus<sup>5</sup>. When the rat's annuli were excised from vertebral bodies placed in water for two hours, their water content increased to 70% to 77% of the

weight of tissue while 8.5% of the PG leached out from the annuli of the control animals. Our data are consistent with the imbibition studies of human annuli of Urban and Maroudas<sup>18</sup> who demonstrated that 500 μm thick annular slices immersed in 0.015M NaCl for 70 minutes increase their water content by nearly 30% while losing 15% of their PG to the hydration medium. Under similar conditions 45% of the PG leached out from the corresponding NP. The relatively small loss of PG from the annuli and the greater loss from the corresponding NP during a short imbibition time are related to differences in the structures of the PG and the collagen framework in the two tissues. The NP consists of randomly dispersed collagen fibrils of small diameter without any organized complex structure immersed in a highly hydrated gel of PG, 75% of which are in the monomeric form and easily extracted under associative conditions<sup>14</sup>. In the annulus, PG surrounds the collagen fibrils and nearly 50% of the PG is in the aggregated form which are effectively extracted only with a dissociative medium<sup>14</sup>. The significant increase in the amount of hexuronate leaching out from the annuli of the flight and tail suspended groups in comparison to the three control groups would seem to suggest that the experimental conditions had caused a change in the PG population. Weightlessness and tail suspension deprive the cells of the stimuli related to cyclic application of compressive forces and, like immobilization, might result in a reduced proportion of PG aggregates and an increased amount of PG extractable by water or low salt concentrations<sup>11</sup>. It seems reasonable to suggest that any PG abnormality caused by weightlessness or tail suspension might be entirely reversible, as it is in short-term joint immobilization, through the formation of a normal PG population induced by the return to normal gravitational conditions. In spite of the difference in the amount of PG extracted by immersion in water, ultrastructure examination of the tissues did not reveal an obvious difference between groups in the collagen-PG relationships demonstrated by ruthenium red staining.

If collagen fibrils are to have high tensile strength, they need to be large in order to maximize the possibility of intermolecular cross-links, but if the collagen network is to return to its original form after compression, the network must have sufficient collagen-ground substance interactions to inhibit nonrecoverable creep. This latter property can best be met with small fibrils<sup>12,13</sup>. Hence, tissues like the disc are comprised of a wide variety of collagen fibril sizes to meet both requirements. Our data indicate that within any single disc the largest fibrils are in the anterior region and a greater proportion of small fibrils is in the transitional and posterior regions. This suggests that in the rat, the anterior area might be submitted to higher tensile forces, whereas the transition and posterior regions sustain greater compressive forces. Interestingly, at the L3-L4 disc, only the tail suspension model demonstrated a larger mean fibril diameter in the posterior region compared to the transitional region. This raises the question as to whether altered spinal forces resulting from tail suspension contributed to an increase in posterior region collagen fibril size.

The annulus contains seven types of collagen (I, II, III V, VI, IX, XI), although types I and II represent more than 80% of the collagen and form the fibrous framework of the annulus<sup>4</sup>. Collagen represents 42% of the dry weight of the annuli of the three control groups. The smaller annuli of the flight group contain 49.5% collagen and therefore are significantly ( $p < .02$ ) more collagenous than their normal counterparts. The slightly reduced PG content and the increased collagen content result in a mean collagen-PG ratio in the flight group significantly greater than each of the control groups individually ( $p < .04$ ) and all control groups combined ( $p < .001$ ). The collagen-PG ratio of the tail suspension model is also significantly greater than the ratio found in the controls ( $p = .04$ , Table 5).

If we consider the weight as well as the composition of the annuli, the biomechanical demands placed upon the annulus by nearly identical body weights (340 g) are sustained in the control groups by 455  $\mu\text{g}$  PG and 2110  $\mu\text{g}$  collagen and in the flight group by only 300  $\mu\text{g}$  PG and 1781  $\mu\text{g}$  collagen. This represents 34% less PG and 15% less collagen to sustain identical body weights upon return to 1 g and might affect the tensile stress of the disc, associated with the fibrous component, and/or the compression strength regulated by the ground substance (Fig. 5). Each annulus of suspended animals contains 418  $\mu\text{g}$  PG and 2267  $\mu\text{g}$  collagen, representing 8% less PG but 7% more collagen compared to the controls.

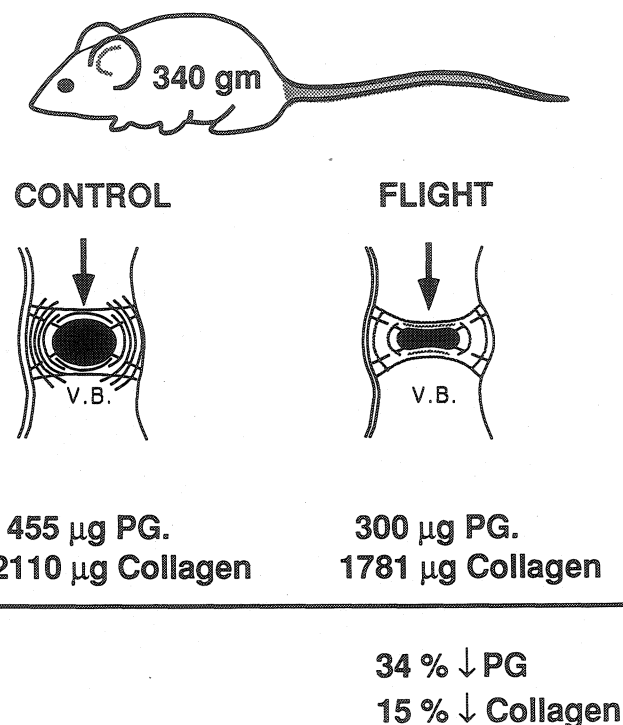


Figure 5. Schematic diagram illustrating changes in flight animal intervertebral discs compared to control animals.

## CONCLUSIONS

Our findings indicate that after 14 days weightlessness the annulus fibrosus is undergoing alterations of its matrix components as indicated by (1) a smaller size, (2) an increased collagen-proteoglycan ratio, (3) an increase in amount of proteoglycan leaching out of the tissue and (4) a proportional increase in collagen content. The tail suspension model does not produce a reduction in the size of the annulus, but the changes in the matrix of the suspended animals may indicate a similar but slower ongoing process that may eventually produce findings similar to those of the flight animals. Future research involving the annulus fibrosus should focus on whether prolonged weightlessness may predispose the disc to injury, whether the observed changes are reversible, and, if so, the time period required to restore the disc to its normal stature.

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# THE EFFECTS OF IMMOBILIZATION ON THE MATURATION OF THE ANTERIOR CRUCIATE LIGAMENT OF THE RABBIT KNEE

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## ABSTRACT

Immobilization-induced alterations occurred in young anterior cruciate ligament (ACL) samples, including the loss of the rounded appearance of the cells. The mature ACL was minimally altered by immobilization at the light microscopy level. In the immobilized young ACL the fibroblasts became elongated and there was loss of the normal pericellular matrix. The immobilized mature ACL differed from controls primarily in the intracellular composition, as there was significantly more rough endoplasmic reticulum (RER) present. Collagen concentrations were reduced only in young immobilized ACL, while no differences were observed in the mature ACL. The collagen synthesis rate in the mature ACL increased with immobilization, although no significant change was observed in the young ACL. The increase in the rate of synthesis of the stress deprived ACL in the mature animals reflected an increase in collagen turnover rather than an increase in accumulation of collagen.

## INTRODUCTION

Treatment of musculoskeletal injuries often includes immobilization as part of the therapeutic regimen. It is well recognized that daily musculoskeletal activity is important for maintenance of connective tissue homeostasis and for general well being of synovial joints. Immobilization has detrimental effects on periarticular ligamentous tissues<sup>4</sup>. The various functional alterations that occur with prolonged immobilization include joint contractures, loss of range of motion secondary to adhesions<sup>11</sup>, and changes in ligament compliance and load-to-failure<sup>17,21,26,31,32</sup>.

Biochemical changes have been described, including loss of collagen mass. The rate of degradation exceeds the increased rate of synthesis<sup>6</sup>. Loss of glycosaminoglycan (GAG) and decrease in hydration have been demonstrated after 9 weeks of immobilization<sup>2</sup>. Recovery from these changes is prolonged and often incomplete<sup>32</sup>. The vast majority of these studies were performed with skeletally

mature animals including dogs<sup>8,16,24</sup>, rats<sup>9,15</sup> and rabbits<sup>1,3,5,6,7,12</sup>. Little work has been reported focused specifically at the effects of immobilization on the periarticular structures such as the anterior cruciate ligament (ACL) (i.e. morphological and biochemical) in the young or more mature age groups.

Changes in connective tissue associated with aging alone have been studied in skin, cartilage and ligaments<sup>7,11,20,22,23,29,33</sup>. Structural morphology, biochemical composition and biomechanical properties have been assessed in these tissues. Observed structural changes include a decrease in cell density and a decrease in the quantity of rough endoplasmic reticulum (RER) within the cytoplasm of the cell. Biochemical changes include a decrease in hydration, collagen content, and collagen synthesis rate. With age one finds an increase in the amount of nonreducible collagen crosslinks (i.e. pyridinoline) and a concomitant decrease in the number of reducible collagen crosslinks, i.e. hydroxylysinonorleucine (HLNL) and dihydroxylysinonorleucine (DHLNL)<sup>7</sup>. In addition, the load to failure of the ligaments is increased in young tissues compared to more mature tissues<sup>33</sup>.

In this study we assessed the effects of prolonged immobilization on the ACL in young (2-month-old) and mature (36 to 40-month-old) New Zealand white rabbits using light microscopy, transmission electron microscopy (TEM), and determinations of collagen concentration and synthesis rates.

## MATERIALS AND METHODS

Thirty-eight New Zealand white rabbits were utilized in this study: 19 were 2 months old ( $\pm 0.25$  mo), and 19 were 40 months old ( $\pm 4$  months). The animals were anesthetized with ketamine and xylazine intramuscularly, and one percent lidocaine with epinephrine locally. The experimental limb (left hind leg) was immobilized in full flexion of  $150^\circ \pm 10^\circ$  in order to take tension off the ACL. A 2.4 mm Steinmann pin was sterilely passed through the tibia, hooked around the femur and locked in this position with a nut anterior to the tibia (Akeson et al. 1973). The right hind limb of each rabbit served as the nonimmobilized control. All animals were then allowed free cage activity for 12 weeks, at which time they were sacrificed with an

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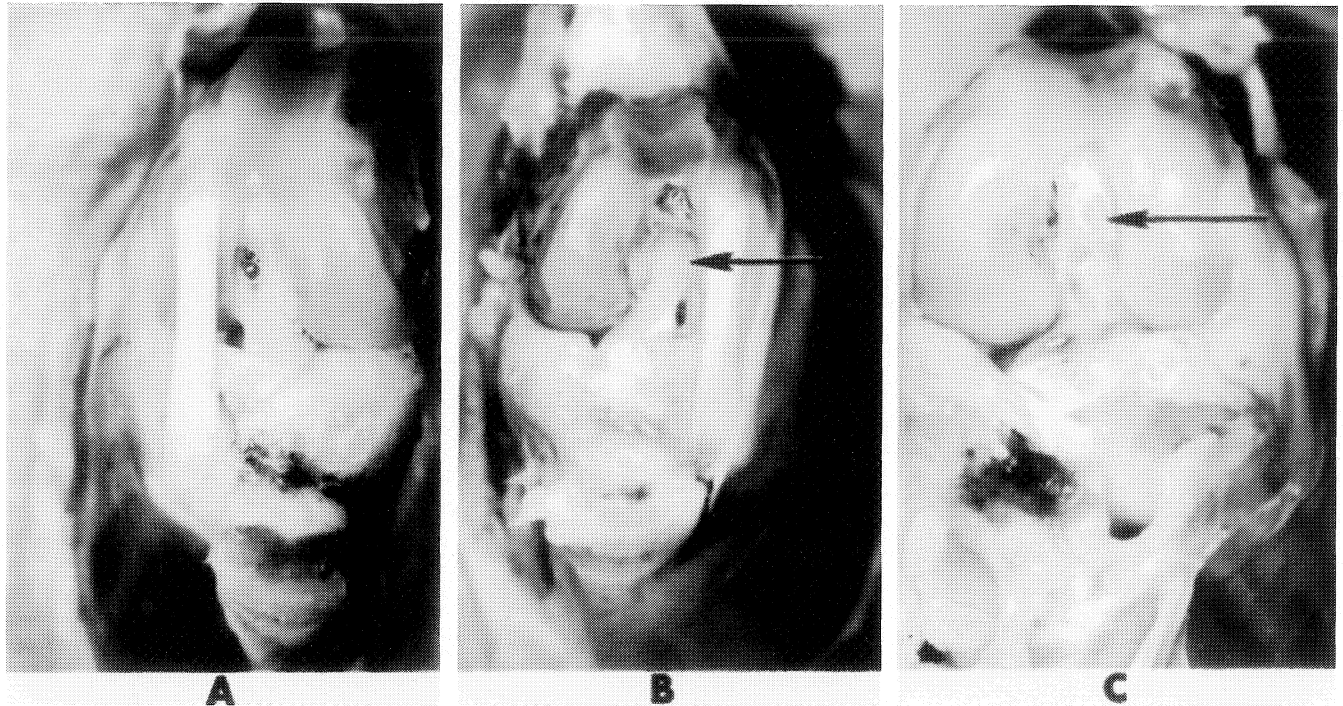


Figure 1

Rabbit ACL adhesion. (A) normal ACL from 20-wk-old rabbit, (B) young animals + 12 wk immobilization, and (c) mature animals + 12 wk immobilization. Note: Adhesion formation in B and C.

intracardiac injection of T-61 euthanasia solution. The animals were randomly utilized as follows:

**Light Microscopy.** The hind limb ACLs of three rabbits from each group were sharply dissected and immediately fixed with 10 percent buffered formalin. Following successive dehydration in alcohol, the tissue was embedded in paraffin and sectioned coronally at a thickness of six microns. The samples were stained with hematoxylin and eosin (H&E).

**Transmission Electron Microscopy (TEM).** The hind limb ACLs of three rabbits from each group were fixed in situ with Karnovsky's fixative, and resected. Only the central third of the ACL (excluding the insertions) was utilized in this part of the experiment. Following further fixation, the tissue was post-fixed in 2 percent osmic acid and cacodylate buffer, dehydrated in alcohol and propylene oxide, and embedded in Spurr. One micron thick pilot sections were stained with toluidine blue to select blocks for thin sections of 600-800 angstroms. Lead citrate and uranyl acetate were used in staining. Three randomly selected sections from each sample were viewed on a Zeiss transmission electron microscope.

**Collagen Content.** The collagen concentration was calculated from the determination of hydroxyproline (hyp) in acid hydrolysates of aliquots of lyophilized ground ACLs from the hind limbs of eight rabbits from each group<sup>6,30</sup>. The percent of collagen was calculated based on the dry weight of the tissue analyzed.

**Collagen Synthesis.** The relative rate of collagen synthesis in control and immobilized ACLs was determined utilizing *in vitro* organ culture. Freshly dissected whole ACLs were placed into media consisting of modified Eagle's media (MEM) supplemented with Earle's salts, 10 percent fetal bovine serum, vitamin E, nonessential amino acids, penicillin/streptomycin, and tritiated proline. Following 48 hours of incubation in humidified air with 5 percent carbon dioxide, the tissue samples were removed from the media and dialyzed for 24 hours against deionized water. The tissues were then lyophilized, pulverized and hydrolyzed in 6N HCl for 24 hrs at 108°C. An aliquot of each hydrolysate was sampled for determination of total hydroxyproline (hyp) content, while the remainder was subjected to cation-exchange high performance liquid chromatography in order to separate proline from hyp. <sup>3</sup>H-labeled hyp (representing newly synthesized collagen) was determined quantitatively by monitoring the column eluate for tritium activity using a radioactive flow detector. Relative synthetic rates were expressed as counts per minute (cpm) per mg of total hyp.

**Statistical Analyses of Collagen Concentration and Collagen Synthesis Rate.** Analyses were performed for both age groups using paired student's t-test, comparing the immobilized with the control ACLs in each rabbit. The level of significance was chosen as  $p < 0.05$ .

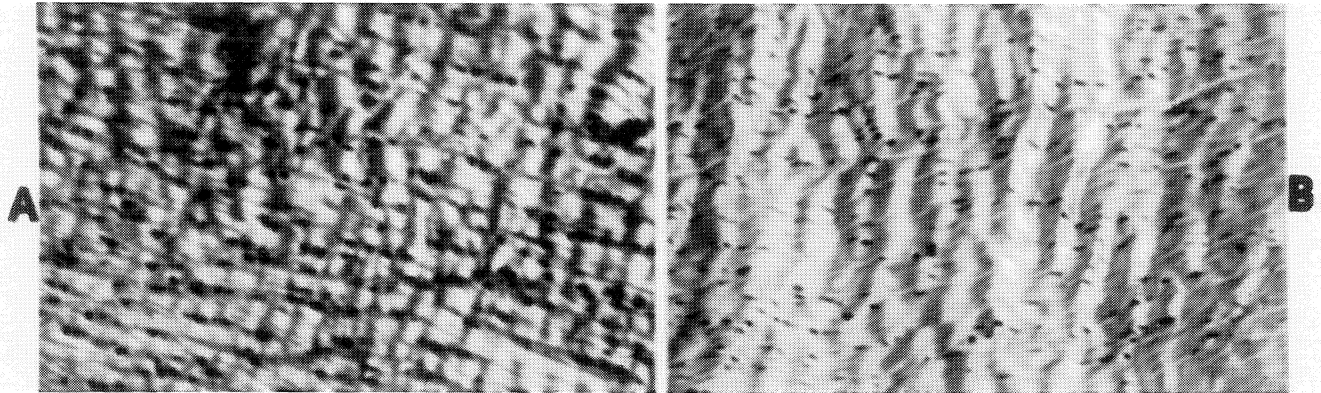


Figure 2

Polarized light appearance (stain, hematoxylin and eosin) (original magnification x50) of (A) young rabbits ACL control, and (B) young rabbits ACL immobilized.

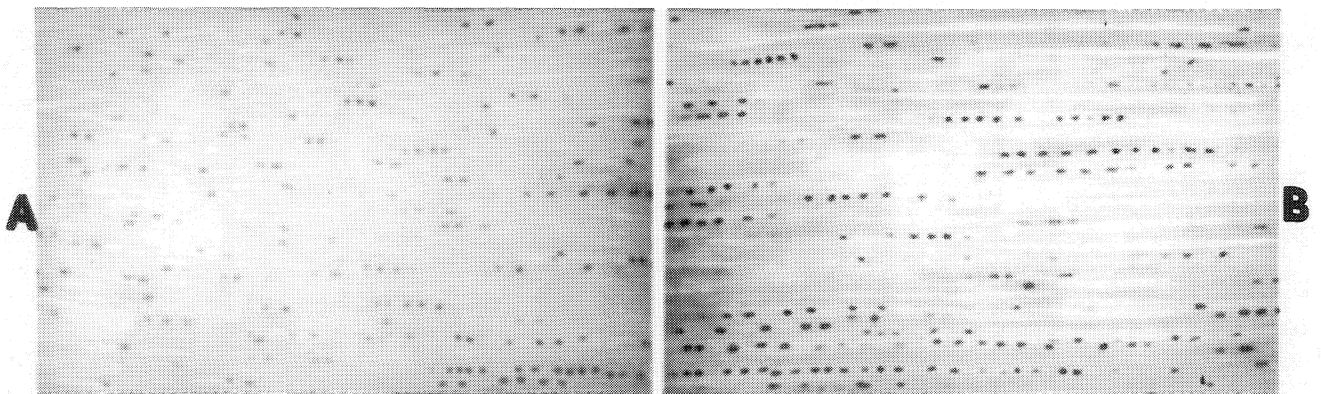


Figure 3

Hematoxylin and eosin stain (magnification x50). (A) mature rabbits ACL control, and (B) mature rabbits ACL immobilized.

## RESULTS

**Gross Morphology.** There was no visually apparent atrophy of the ACL in either experimental group. Adhesions, including adherence of the fat pad to the ACL, occurred in 60-70 percent of the animals of both age groups in the experimental knee. In some cases this process engulfed and embedded the ACL beneath fibrofatty tissue (Figures 1A, 1B, and 1C) for the young and old animals. In approximately 25 percent of the young rabbits the immobilized patellar tendon (PT) underwent degeneration and infiltration by fatty tissue which resulted in a two fold increase in a the A-P thickness of the tendon.

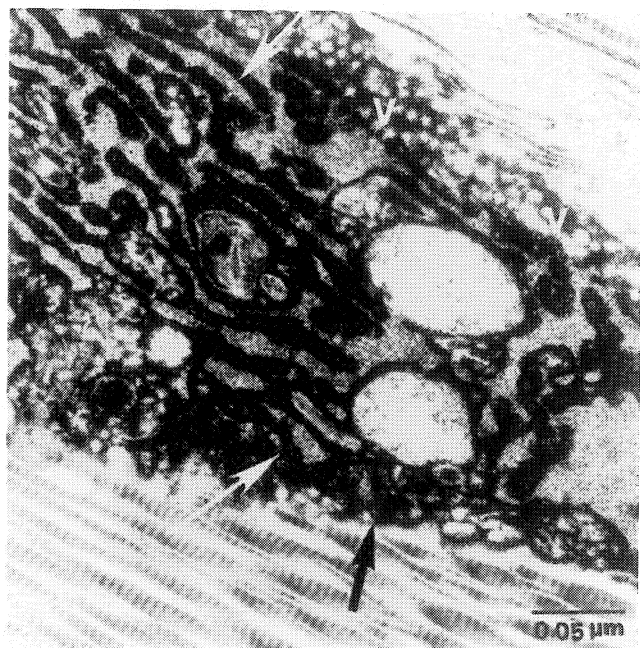
**Light Microscopy.** In the young control ACL the fibroblasts were generally rounded and organized in rows or columns, somewhat independent of the low amplitude, high frequency crimp pattern of the collagen (Figure 2A). The mature tissues had, in general, fewer cells per high-powered field than the young tissue.

Alterations induced by immobilization, as detected at the light microscopy level, occurred in the young ACLs (Figure 2B). The fibroblast took on a spindle shape and the crimp amplitude increased while the frequency decreased.

The mature ACLs were relatively unaffected at the light microscopy level (Figures 3A and 3B).

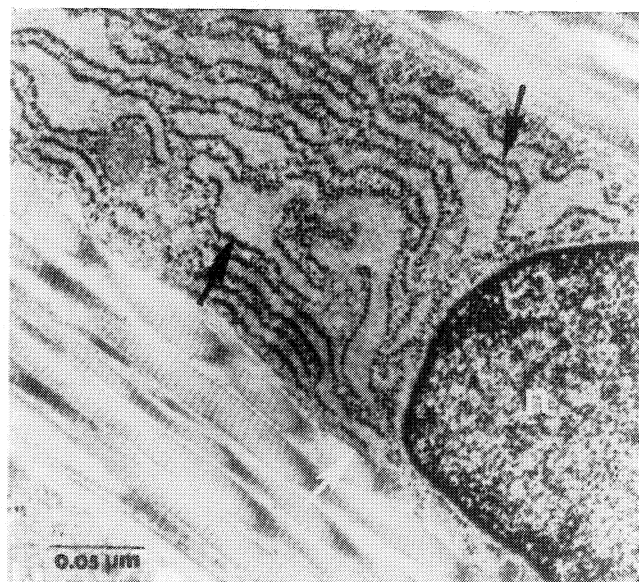
**TEM.** TEM confirmed the light microscopy results. The control young ACL possessed ovoid fibroblasts arranged in columns (like a string of pearls), surrounded by a pericellular matrix. Rough endoplasmic reticulum (RER) was prominent in the cytoplasm (see arrow) as was the Golgi apparatus (Figure 4A). In the immobilized young ACL, the ultrastructure was markedly different. The fibroblasts became elongated and there was less pericellular matrix. The collagen fibers, previously separated from the cell membrane by amorphous matrix, appeared tightly associated with this structure. Intracellularly, several additional changes were noted. The cisterns of the RER were more dilated than in the control samples (see arrow), and contained a proteinaceous substance (Figure 4B).

The control, mature ACL fibroblasts resembled their younger counterparts, though they differed in several respects. The mature cells retained their ovoid shape but had less pericellular matrix (Figure 5A). The matrix contained extensive electron dense lipid particles (see



**Figure 4A**

TEM Nonimmobilized contralateral control young ACL. This higher magnification view emphasizes the prominent RER in the cytoplasm (white arrows). Cell membrane (black arrow) and vacuoles (v).



**Figure 4B**

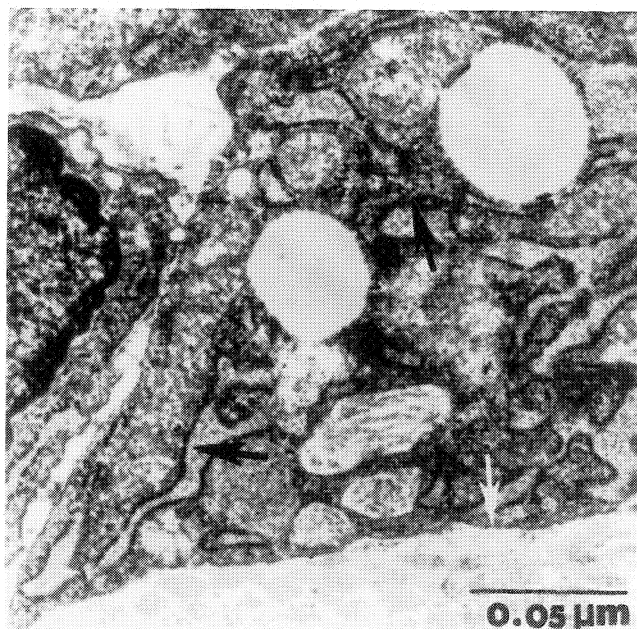
TEM. Immobilized young ACL. This higher magnification view represents the cisterns of the RER which were more dilated (black arrow) than the nonimmobilized contralateral control young ACL. Cell membrane (white arrow) and nucleus (n).

arrow). The cells were nearly devoid of RER, and frequently contained lipid droplets. The immobilized tissues differed primarily in the intracellular composition, as there was significantly more RER (see arrow) found in the ACL



**Figure 5A**

TEM. Mature ACL control. High magnification illustrating matrix contained extensive lysosomal body defects (black arrows). Minimal RER observed (white arrow).



**Figure 5B**

TEM. Immobilized mature ACL. High magnification illustrating more RER relative to the nonimmobilized mature ACL (black arrows), and cell membrane (white arrow).

of the immobilized limbs compared to the controls (Figure 5B). Electron dense particles were observed with some degree of frequency in both the control and immobilized mature ACL, as seen by Roy in his study of articular cartilage<sup>25</sup>.

*Collagen Concentration.* The results of collagen concentration determinations are illustrated in Figure 6. Following immobilization, the collagen concentration decreased significantly ( $p < .05$ ) in the young immobilized ACL, while no statistical change was observed in the mature ACL.



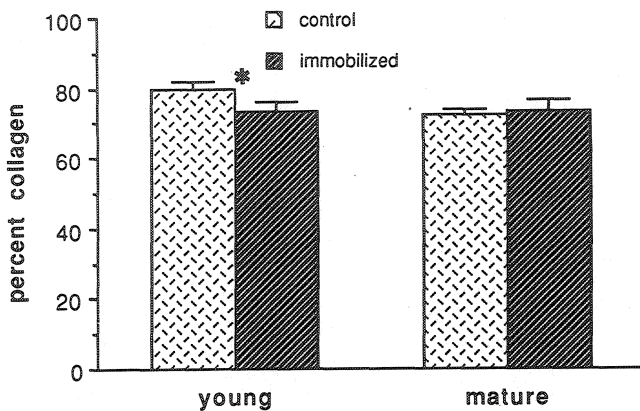


Figure 6

Collagen concentrations in the control and immobilized anterior cruciate ligament from young and mature rabbits. Note: \*Indicates a statistically significant difference between control and immobilized ligaments of  $p < 0.05$ . Error bars indicate standard error of mean.

**Collagen Synthesis.** The relative rates of collagen synthesis in the control and immobilized ACLs of young and mature rabbits are illustrated in Figure 7. The rate of collagen synthesis more than doubled in the mature ACL group after 12 weeks of immobilization, while it did not change significantly in the young ACL group.

## DISCUSSION

In this study, changes in the gross appearance, histology, ultrastructure, and collagen characteristics of immobilized ACLs in young and mature rabbits were studied. The gross appearance of immobilized ACLs and the formation of intra- and periarticular adhesions is in agreement with previous reports<sup>27</sup>. Intraarticular adhesions such as those seen in this study have been reported by Enneking and Horowitz in the immobilized human knee<sup>11</sup>.

The effects of prolonged immobilization were most prominent in the ACL of young rabbits. Growing without the normal combination of active tensile and torsional forces, the immobilized ACL of a young rabbit experiences only the fixed tension originally applied during the immobilization procedure. This fixed tension relaxes with time, yielding an unloaded environment for growth. We theorize that loading is necessary for the normal development of the ACL into a "fibrocartilage" like structure. The lack of a similar degree of change in the mature tissue may again be a factor of differentiation. The mature ACL is perhaps no longer in an active differentiating state; therefore, the well-established cytoarchitecture is preserved, despite being immobilized. The age-related decrease in fibroblast density within ligaments has also been observed by Haus and Reflor in their study of the human ACL<sup>14</sup>. This finding may be due to either a loss of cells with increasing age, a relative increase in extracellular components, or a combination of both factors.

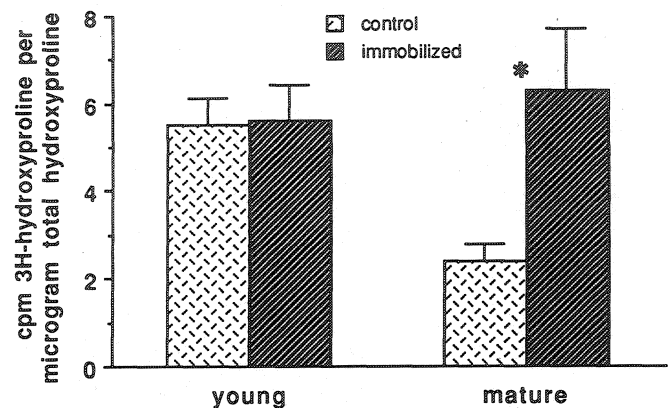


Figure 7

Collagen synthesis rates within the control and immobilized anterior cruciate ligaments from young and mature rabbits. Note: \*Indicates a statistically significant difference between control and immobilized ligaments of  $p < 0.05$ . Error bars indicate standard error of mean.

In prolonged immobilization a loss of the proteoglycan-containing pericellular matrix is observed<sup>10,19</sup>. The young ACL fibroblasts become elongated and intimately associated with surrounding collagen fibers. This is in marked distinction from these cells' normal ovoid morphology, which is strikingly similar to the appearance of chondrocytes. As theorized by Clark and Sidles, the matrix may function in protection of the fibroblasts of the ACL from the compressive forces resulting from the twist induced in the ACL with knee flexion. The lack of these forces may signal the fibroblast to modify protein synthesis and secretion. This phenomenon may be a soft tissue corollary to Wolff's Law.

The ultrastructural changes observed in the present study are similar to, though not as extensive as, those seen by Roy in the study of immobilized cartilage<sup>25</sup>. Changes observed include the dilation of RER cisterns, swelling of mitochondria, lipid droplet accumulation, and electron dense lipid particle accumulation. All of these changes have been characterized as degenerative and several have been associated with both immobilization and the aging process.

While their exact nature is unknown, we theorize that the electron-dense bodies are derived from undegraded cellular lipids, which tend to accumulate with age. These bodies have also been observed in articular chondrocytes<sup>13,25</sup>.

An unexpected discovery was the increase in the RER of the ACL fibroblasts in the mature immobilized specimens. This finding indicates an increase in cell synthetic activity. Indeed, the rate of collagen synthesis increased in the mature ACL upon immobilization, reflecting an increase in collagen turnover rate, rather than an increase in accumulation of collagen<sup>5,15</sup>. As the resting rate of syn-

thesis in the mature tissue is low, even a slight increase in rate would appear substantial in proportion.

In this study, age and stress deprivation have both separate and overlapping effects on periarticular connective tissue. Gross, histologic, ultrastructural, and biochemical changes have been described. With immobilization, the morphology of the young ACL fibroblast becomes less similar to the chondrocyte which it normally resembles.

#### ACKNOWLEDGMENTS

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# THE USE OF THERMOGRAPHY IN SYMPATHETICALLY MAINTAINED PAIN

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## ABSTRACT

This paper reviews the symptomatology, pathophysiology, and treatment of reflex sympathetic dystrophy and sympathetically maintained pain. It is the author's experience that there exists a group of patients who present with chronic, unexplained pain following trauma, but lack the physical findings and positive investigative tests to confirm the diagnosis of reflex sympathetic dystrophy. For these patients, thermography serves as a useful and sensitive test to diagnosis sympathetically maintained pain. This paper presents six case reports in which thermography was used to diagnosis sympathetic dysfunction as the cause of chronic pain.

## INTRODUCTION

During the Civil War, in 1864, Mitchell, et al. observed that soldiers with battlefield nerve injuries sometimes developed persistent "burning" pain and progressive trophic changes in the injured extremity<sup>16</sup>. Mitchell named this disorder "causalgia," derived from the Greek term for "burning pain"<sup>17</sup>. In 1940, Homans used the term "moderate causalgia," and in 1945, DeTakats used "causalgic states" to describe neurologically mediated pain syndromes<sup>4,11</sup>. Since that time, terms, such as "major causalgia," "minor causalgia," "Sudek's atrophy," "sympathalgia," "post-traumatic dystrophy," and "shoulder-hand syndrome" have been used to describe the pain syndrome and the corresponding pathologic changes that occur in limbs following injuries. Evans was the first to use the term "reflex sympathetic dystrophy"<sup>7</sup>. Currently this is an all-inclusive term for a spectrum of syndromes that result from autonomic dysfunction.

The condition of reflex sympathetic dystrophy (RSD) may follow major or minor trauma. The condition is defined by the International Association for the Study of Pain as "continuous pain in a portion of an extremity after trauma, which may include fracture but does not involve a direct nerve injury, that is associated with sympathetic hyperactivity." The hallmark of RSD is chronic pain and swelling out of proportion to the injury or underlying process. The pain often localizes to the area of injury and with time may progress proximally to involve the entire

limb. The pain varies from a dull sensation to severe burning pain that frequently interferes with all activities including sleep.

Classically RSD presents as a spectrum of signs and symptoms which progresses through three stages. Each stage can last from weeks to months<sup>3,18,20</sup>. The first, or acute stage may begin days, weeks, or months after the injury. In the acute stage, the patient experiences a constant aching or burning pain, hyperalgesia, and hyperhydrosis. Pitting edema, hyperthermia or hypothermia, increased hair and nail growth, and associated color changes may be apparent in the involved extremity. Emotional changes can increase the intensity of the symptoms.

The second, or dystrophic stage begins about three to six months after the injury, and may last up to a year. This stage is characterized by severe continuous burning pain. Generally, the pain is more intense than in the acute stage. The edema becomes brawny and fixed. There is frequently hair loss and the nails develop grooves or ridges. There is wasting of the muscles and progressive stiffness of the joints. Radiographs often show early spotty periarticular osteoporosis which can later become polar or diffuse.

The third, or atrophic stage is characterized by irreversible, marked tissue changes. The hyperpathia may diminish or persist. As the swelling resolves, the skin becomes smooth, cool, tight, glossy, and either pale or cyanotic. There are fibrotic changes of the periarticular soft tissues and fascia which cause joint contractures. The fingers and toes appear fusiform and pointed due to atrophy of the palmar fat and beveling of the nails. The muscles of the extremity show marked wasting. Radiological studies in this stage show severe osteoporosis. Radionuclide scintigraphy demonstrates increased uptake.

## PATHOPHYSIOLOGY OF REFLEX SYMPATHETIC DYSTROPHY

Many theories have been proposed to explain the mechanism responsible for RSD. Livingston in 1943, proposed the theory that painful stimuli initiate "reverberating circuits in the spinal cord"<sup>14</sup>. Doupe, et al., proposed the theory that trauma or nerve injury causes the formation of artificial synapses or "cross stimulation" between sympathetic efferent fibers and somatic sensory afferent fibers, at the site of injury<sup>6,13</sup>. Recent studies in animal models have confirmed the existence of "cross stimulation" (ephapses) in injured nerves<sup>5</sup>. This cross-stimulation

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between efferent sympathetic fibers and nociceptive afferent fibers is one way that the sympathetic nervous system may play a central role in the pathophysiology of RSD.

Roberts proposed that sympathetically maintained pain is the result of a "high rate of firing in the spinal wide-dynamic-range (WDR) or multi-receptive neurons<sup>19</sup>." Roberts postulated that pain syndromes, such as causalgia and RSD are mediated by activity in low-threshold, myelinated mechanoreceptors. He suggested that the actions of the sympathetic efferent on the afferent fibers evoke activity in the sensitized WDR neurons to produce a painful sensation. Roberts fostered the term "sympathetically maintained pain" to describe a painful condition associated with sympathetic dysfunction.

The majority of evidence in recent literature strongly supports the theory that the sympathetic nervous system plays a central role in the development of RSD and sympathetically maintained pain<sup>18,20,3</sup>.

### TREATMENT

Sympathetic blockade plays a key role in the diagnosis and treatment of RSD and sympathetically maintained pain. A favorable response to sympathetic blockade confirms the diagnosis of RSD<sup>3</sup>. Early diagnosis is essential as the treatment is most effective in the early stages. The results of treatment are less predictable in the later stages when structural changes are present.

Bonica has stated that if the diagnosis is correct, sympathetic blockade combined with vigorous physical therapy relieves the pain in 80% of patients<sup>3</sup>. The number of repeated sympathetic blocks to render effective treatment varies. Intravenous regional guanethidine and reserpine have also been successful in the treatment of patients with RSD. Several authors have reported up to 80% pain relief with intravenous guanethidine<sup>2,8</sup>.

In addition to sympathetic blockade, adjunctive therapy is important in the treatment of RSD and sympathetically maintained pain. Physical therapy, occupational therapy and transcutaneous electrical stimulation (TENS) have been beneficial<sup>20</sup>. Many patients with long-standing RSD and sympathetically maintained pain undergo emotional and psychological disturbances which require intense psychological support. For these patients, psychological support is of equal importance to the other treatment modalities.

### THERMOGRAPHY IN SYMPATHETICALLY MAINTAINED PAIN & REFLEX SYMPATHETIC DYSTROPHY

The diagnosis of RSD is easily made in patients who present with the classic signs and symptoms. However, many patients with chronic pain syndromes do not present in this fashion. In the author's practice, most of the

patients have few or no classic signs of RSD. It is in this group of patients that infrared electronic thermography is most helpful.

The sympathetic nervous system plays a critical role in the production of RSD and sympathetically maintained pain. The clinical manifestations of these syndromes reflect the dysfunction of the autonomic nervous system, which includes temperature regulation. Hypothermia and hyperthermia of the involved limb, as shown on thermography, provide evidence of sympathetic dysfunction. In a recent study, Bennett and Ochoa obtained infrared thermographic images on rats with experimentally induced nerve injuries<sup>1</sup>. These images were similar to those obtained in patients diagnosed with RSD.

Janig<sup>12</sup> classified reflex sympathetic dystrophy into three subgroups based on their clinical symptoms and physical findings: 1) "Sympathetic Algodystrophy" describes the syndrome in the group of patients who have all the signs and symptoms of RSD. 2) "Sympathetic Dystrophy" describes the syndrome in a group of patients who lack the typical burning pain, but have all the other symptoms of RSD. 3) "Sympathetic Maintained Pain" describes the syndrome in patients who have typical spontaneous pain and allodynia, but present no physical findings of RSD.

In patients with sympathetically maintained pain, the thermogram may be the only objective way to demonstrate an abnormality. Hendler has stated "at a minimum, diagnostic studies that would facilitate the diagnosis of RSD would be thermography and bone scintigraphy"<sup>10</sup>. I agree with Harden, who proposed that the term sympathetically maintained pain should be reserved for the situations in which autonomic abnormality is detected by thermograms alone, and the term reflex sympathetic dystrophy should be used for those cases with classic physical findings and investigational studies consistent with RSD<sup>9</sup>.

Since electronic thermography reflects sympathetic nerve activity, it is an excellent test for the detection of RSD and sympathetically maintained pain. Electronic thermography measures heat emission from the body surface. Thermography is highly sensitive and can detect sympathetic dysfunction even in the absence of clinical findings.

Electronic thermography has led to an increased interest and awareness in sympathetically maintained pain. In my practice, I rarely see patients who manifest all of the classic signs and symptoms of RSD. The majority of my patients who complain of chronic, unexplained pain in an extremity following a major or, more frequently, minor trauma, can be characterized as patients with sympathetically maintained pain, rather than RSD. It is in these patients that thermography has proven itself a sensitive test and useful diagnostic tool.

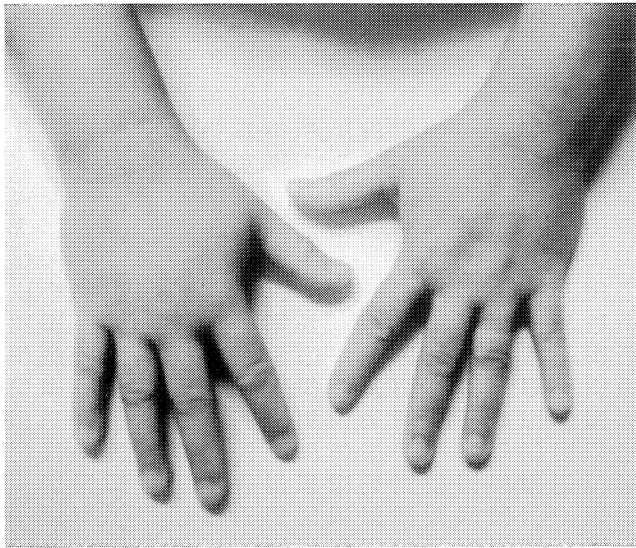


Fig. 1-A  
Photograph of swollen and discolored right hand.

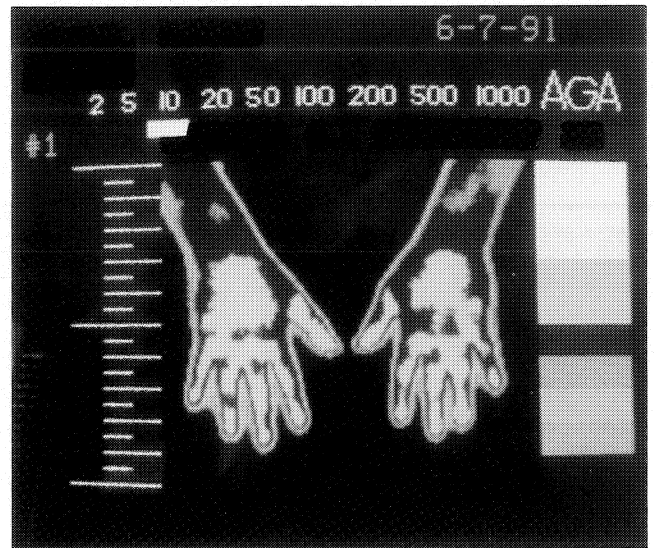


Fig. 1-C  
Thermogram shows symmetry of heat emission of the right and left hands.



Fig. 1-B  
Thermogram shows relative increase in heat emission (hyperthermia) of the right hand.\*

## CASE PRESENTATIONS

### Case 1

S.T., a 37-year-old woman, injured her right hand while using a wrench to loosen a bolt. She was diagnosed as having acute de Quervain's tenosynovitis of the right wrist, and treated with a steroid injection and immobilization. Shortly after returning to work her right hand became painful, swollen, and discolored (Fig. 1-A). An electronic thermogram showed a relative increase in the heat emission of the right hand compared to the left, suggestive of sympathetically maintained pain (Fig. 1-B). Several weeks after discarding the splint and increasing the use of the hand, most of the pain and swelling had resolved. A follow-up electronic thermogram study showed symmetry of the heat emission between the right and left hands (Fig. 1-C).

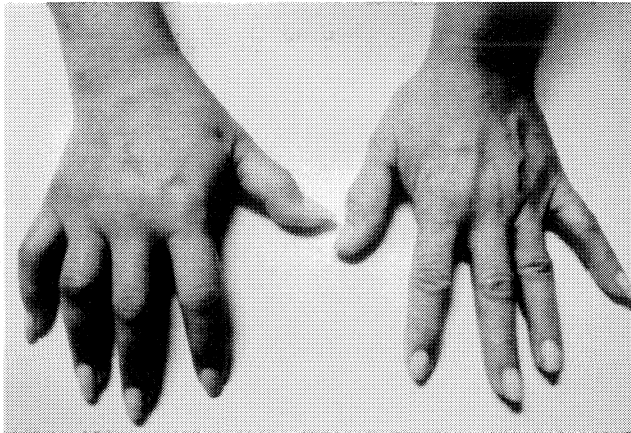


Fig. 2-A

Photograph of swollen and discolored right hand.

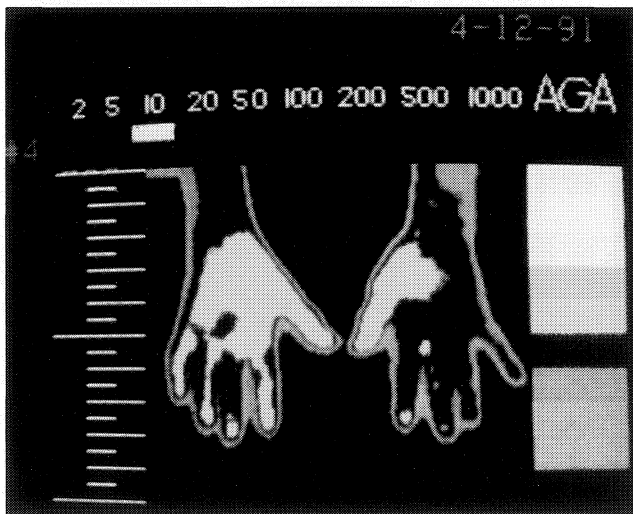


Fig. 2-B

Thermogram shows hyperthermia of dorsum of right wrist and hand.

### Case 2

P.M., a 46-year-old woman, injured her right hand when she slipped on a wet floor. A few days later she complained of pain and swelling of the right hand. The pain persisted despite immobilization and steroid injection. Four months later an EMG study showed evidence of a carpal tunnel syndrome and radiographs demonstrated CMC arthritis of her thumb. Five months after the injury, the patient underwent a carpal tunnel release and a CMC arthroplasty, which failed to relieve her symptoms. Several stellate ganglion blocks did not relieve her hand swelling and pain (Fig. 2-A). An electronic thermogram performed three months after the carpal tunnel release and CMC arthroplasty demonstrated increased heat emission in the right wrist and hand compared to the left, suggestive of sympathetically maintained pain (Fig. 2-B).

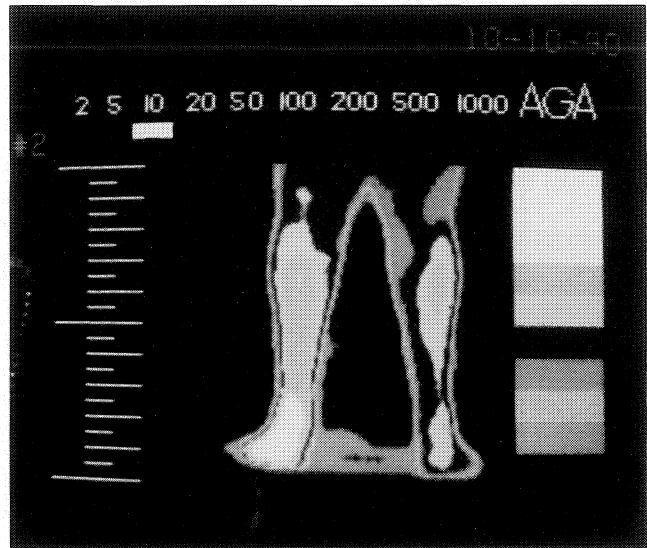


Fig. 3-A

Thermogram demonstrates hyperthermia of right leg.

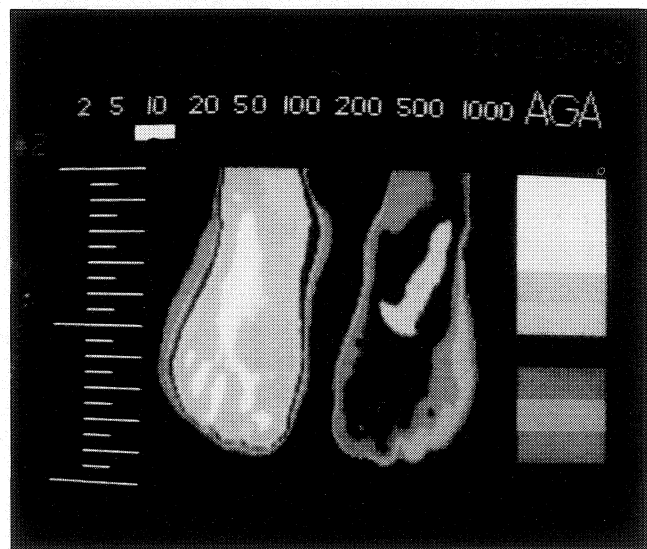


Fig. 3-B

Thermogram shows hyperthermia of right foot.

### Case 3

M.M., a 49-year-old woman, had an uncomplicated excision of an accessory tarsal navicular bone from her right foot. This was followed by immobilization of the right foot in a non-weight bearing cast for six weeks. After removal of the cast and progression to full weight bearing, the patient developed burning pain in the right foot and lower leg. An electronic thermogram three months after the surgery on her right foot showed a relative increase in the heat emission of the right leg and foot suggestive of a sympathetically maintained pain syndrome (Fig. 3-A & 3-B).

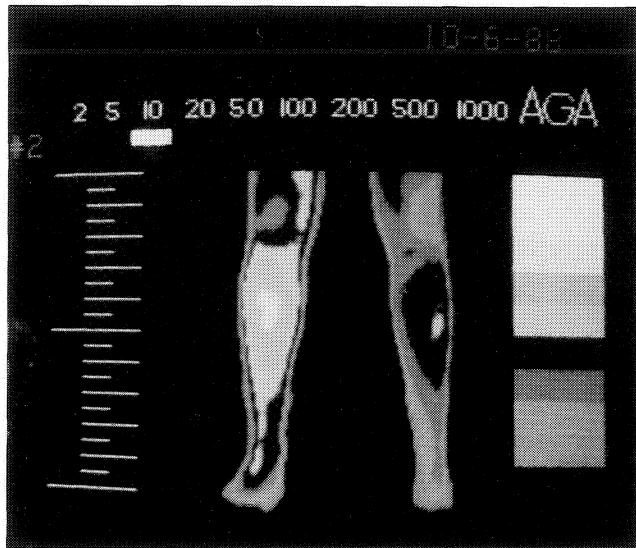


Fig. 4-A  
Thermogram shows hypothermia of lower left leg.

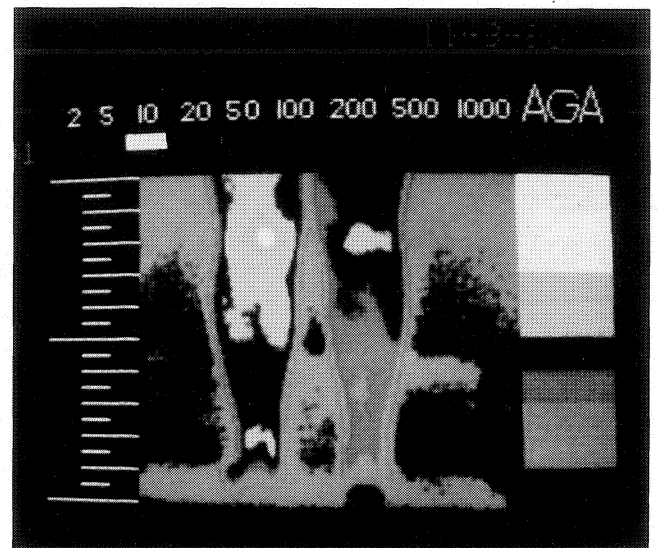


Fig. 5-A  
Thermogram shows hypothermia of right posterior lower leg.

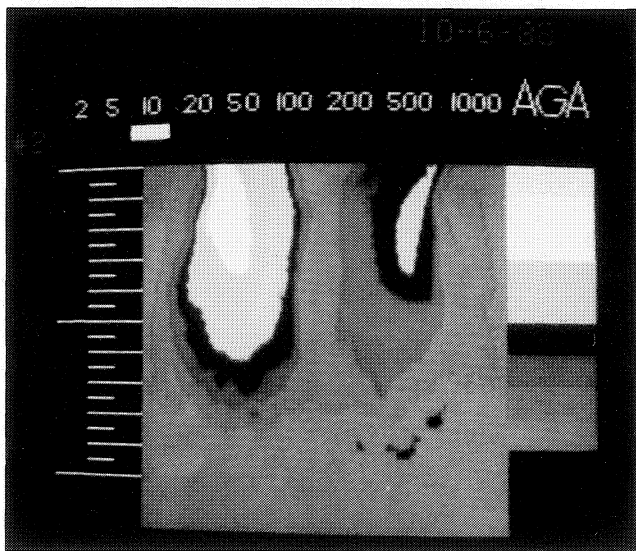


Fig. 4-B  
Thermogram shows hypothermia of dorsum of left foot.

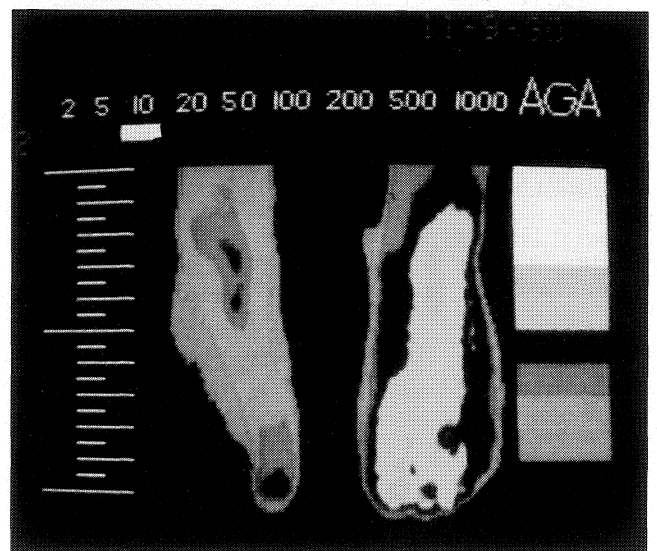


Fig. 5-B  
Thermogram shows hypothermia of right foot.

#### Case 4

C.G., a 30-year-old man, twisted his left knee and sustained a nondisplaced fracture of the medial tibial plateau. His injury was treated with a period of non-weight bearing and immobilization. After several weeks, the patient complained of pain and a cold sensation in the left foot and leg. An electronic thermogram two months after the injury showed a global decrease in the heat emission of the left thigh, leg, and dorsal foot (Figs. 4-A and 4-B). This was suggestive of a sympathetically maintained pain syndrome of the left lower extremity.

#### Case 5

V.W., a 41-year-old woman, injured her right foot in an automobile accident. Two months after the accident, the patient complained of continued pain in the right ankle and foot. An electronic thermogram performed two months after the injury revealed a decrease in the heat emission of the right lower leg and foot (Figs. 5-A and 5-B). These findings were consistent with a sympathetically maintained pain syndrome of the right lower extremity. After a series of three lumbar sympathetic blocks, the pain in the right ankle and foot resolved. Two months later, the pain recurred. A second series of lumbar sympathetic blocks



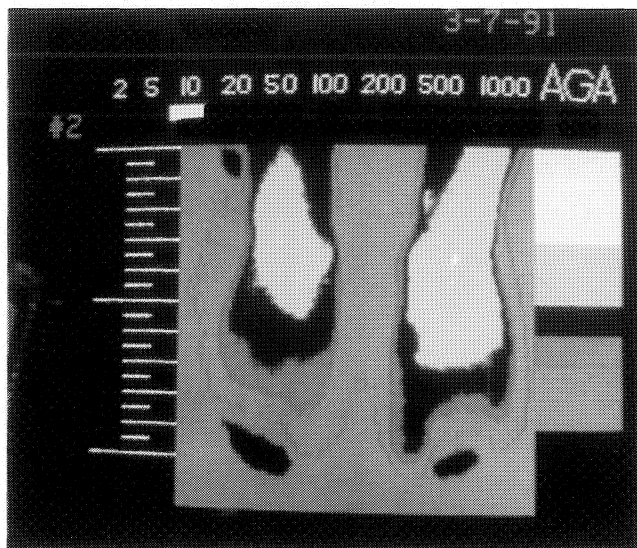


Fig. 5-C

Post-block thermogram show relative symmetry of vascular heat emission of the dorsal aspect of the right and left foot.

resulted in marked relief of the pain. A repeat electronic thermogram one month after the second series of blocks showed an increase in the heat emission of the right ankle and foot (Fig. 5-C). Thus, the repeat thermogram confirmed the clinical improvement following the second series of blocks.

#### Case 6

B.G., a 45-year-old woman, complained of a constant burning pain on the dorsal and plantar surfaces of her left foot and over the lateral aspect of her left ankle. Walking one block exacerbated her pain and caused a purple discoloration in her left heel.

The patient reported that her left foot was injured in an automobile accident when her car was struck from the rear by another vehicle. At a local hospital, radiographs of the left ankle and foot showed no fractures. A splint was applied to the left foot and leg, and she was kept non-weight bearing. The splint was removed after four months and her weight bearing was advanced. She was treated with physical therapy for three months, and eventually released to go back to work as a truck driver. She could not resume this job because she was unable to push the clutch with her foot due to pain. An orthopaedic surgeon suspected RSD and requested a thermogram.

The electronic thermogram demonstrated a global decrease in the heat emission pattern of the left lower extremity from the upper posterior thigh to the plantar aspect of the left foot (Figs. 6-A and 6-B), consistent with RSD.

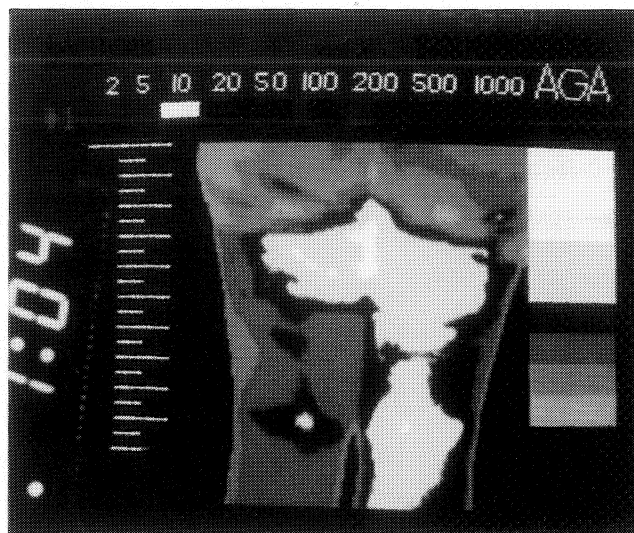


Fig. 6-A

Thermogram shows hypothermia of the posterior left thigh.

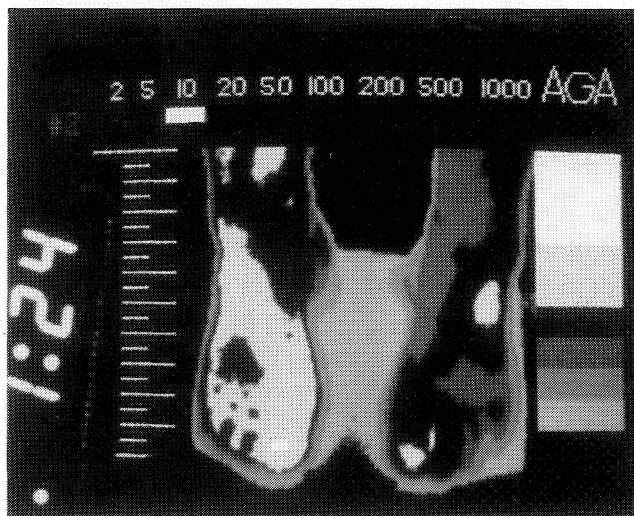


Fig. 6-B

Thermogram shows hypothermia of the dorsum of left ankle and left foot.

#### SUMMARY

Reflex sympathetic dystrophy is a pain syndrome that is associated with a broad spectrum of physical findings caused by dysfunction of the sympathetic nervous system. Classic RSD has been divided into, acute, dystrophic, and atrophic stage based on the chronicity of the physical findings. Various theories have been proposed to explain the pathophysiology of RSD. It has been suggested that there are "reverberating circuits" in the spinal cord that are triggered by intense pain<sup>14</sup>. Another theory is the existence of ephapses (artificial synapses) or cross-stimulation between the sympathetic efferent and somatic sensory afferent fibers<sup>6</sup>. It has also been said that RSD is

caused by "a central nervous perturbation, which once established persists despite the subsequent removal of the cause"<sup>22</sup>.

Early diagnosis is crucial for successful treatment of RSD. Sympathetic blockade followed by intensive physical therapy often alleviates the pain and may prevent progression of the syndrome. Sympathetic ganglion blocks have been the cornerstone of the diagnosis and treatment<sup>3,18,20</sup>. The number of blocks necessary for satisfactory outcome varies, and must be individualized for each patient. The pharmacologic interruption of the function of the nerve fibers only lasts a few hours, but the benefit of interfering with the cycle may last for days before the symptoms return. Alternative treatments to ganglionic blockade include regional intravenous infusions (guanethidine, reserpine, and other agents), somatic sensory nerve blocks, sympathectomies, oral agents, and others. Common adjunctive therapy includes physical therapy, TENS, massage, and functional activities such as stress loading. Physical therapy should always accompany treatment with sympathetic blocks. Cognitive psychotherapy and psychological support are also of primary importance in some cases.

The majority of patients in my practice do not present with the classic signs and symptoms of RSD. Their clinical findings are much more subtle. In this group of patients, the condition is described more appropriately as "sympathetically maintained pain". This diagnosis is facilitated by thermography which reflects the activity of the sympathetic nervous system. The thermogram demonstrates temperature changes of the involved extremity. Local or global hypothermia or hyperthermia of an extremity is indicative of autonomic dysfunction when there are no other identifiable causes.

"Sympathetically maintained pain" is a more appropriate term to describe a syndrome which may be more common than the classic syndrome of RSD. The term RSD should be limited to those cases with typical signs and symptoms, while "sympathetically maintained pain" should be used whenever there is only thermographic evidence of sympathetic dysfunction<sup>9</sup>.

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# STABILIZATION PROCEDURES OF THE HINDFOOT

James V. Bono, M.D.\*  
Richard L. Jacobs, M.D.\*\*

## INTRODUCTION

In the past, stabilization procedures of the hindfoot have been reserved for the treatment of infantile paralysis,<sup>1,2,4,8-26</sup>. Any joint which has been deprived of its stability as a result of paralysis requires additional extrinsic or intrinsic stabilization. Manipulation and bracing were the mainstays of treatment of the paralytic foot in the late 19th century<sup>1</sup>. However, braces at that time were cumbersome, expensive, unsightly, and associated with skin problems. This led many surgeons to favor internal stabilization. The indications for stabilization procedures of the foot, which during the first two decades of the 20th century were limited almost exclusively to poliomyelitis, include talipes equinovarus, traumatic arthritis, rheumatoid arthritis, neoplasm, infection, and neurological disorders<sup>5-8,27,28</sup>.

## Goals

The primary goals of treatment are aimed at restoring functional, painless stability to the hindfoot. Secondary goals include obviating the need for external support, improved cosmesis, and easier shoeing. Once the foot and ankle have been stabilized, the patient will hopefully be relieved of knee, hip and low back pain resulting from an altered gait. With return of function and stability, a patient may return to a productive life style.

## Pathophysiology and Biomechanics

As with most orthopaedic conditions, a distinction must be made between flexible and fixed deformities. In order to retard progression of the deformity and prevent recurrence, a supple deformity may only require soft tissue procedure. Bony procedures are often reserved for fixed deformities. To correct the deforming forces present in an unstable foot and ankle, it is necessary to have an understanding of the muscle forces required to maintain equilibrium of the foot. The four dominant muscle groups

(triceps surae and toe flexors, tibialis anterior and toe extensors, peronei, and posterior tibialis) represent the four columns of support about the foot and ankle. The relative action of these muscle groups is responsible for configuration of the ankle and foot. If one column fails, equilibrium can be reestablished by transposition of the adjoining columns. However, if two columns fail, it is difficult to reestablish equilibrium. If one muscle were to become paralyzed, its antagonist would predominate, resulting in deformity. The degree of muscle imbalance is unique in each case, leading to a variety of unstable configurations.

The varus deformity of the foot is usually the result of paralysis of the peronei, leading to the unopposed contraction of their antagonist, the tibialis posterior. The foot becomes inverted with a supinated attitude, causing the lateral border of the foot to be the principle weight bearing surface. A concomitant paralysis of the tibialis anterior with unopposed contraction of the triceps surae would add an equinus attitude to the foot, resulting in an equinovarus configuration. With weakening of the peronei, tibialis anterior, and triceps surae, the plantar intrinsic muscles are unchecked, resulting in an equinovarus deformity with a cavus attitude.

A valgus deformity of the foot is usually the result of weakness of the posterior tibialis, leading to unopposed contraction of its antagonists, the peronei. The foot becomes everted with a pronated attitude, causing the medial border of the foot to be the principle weight bearing surface. With a concomitant paralysis of the tibialis anterior, the valgus deformity would be accentuated along with abduction of the forefoot. With paralysis of the plantar muscle groups, the arch drops, resulting in a planovalgus deformity.

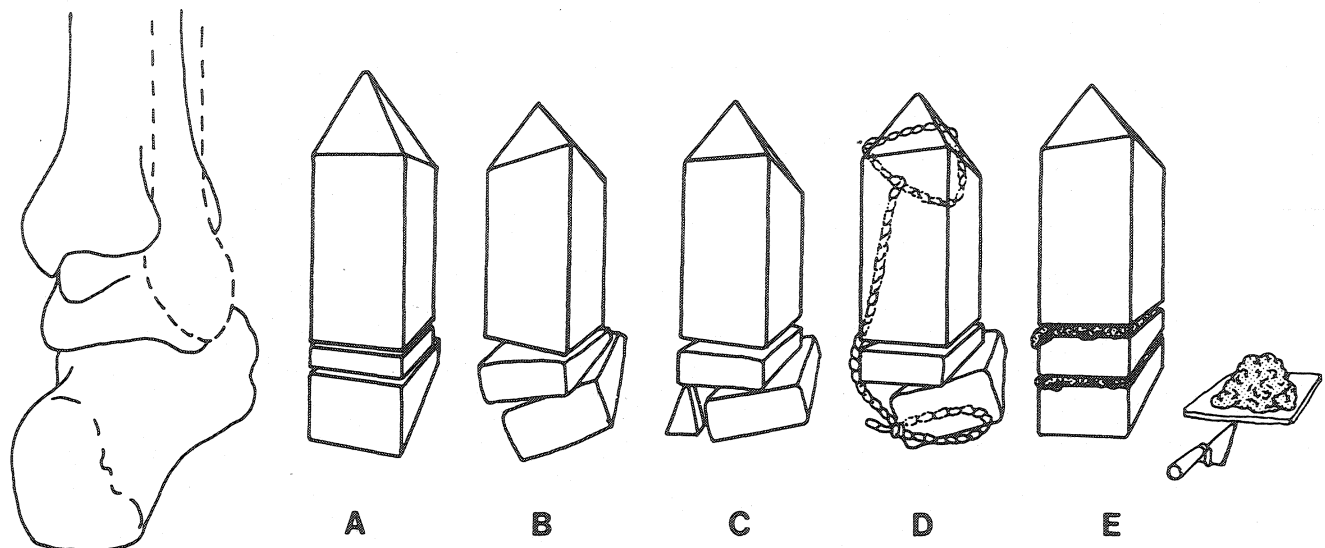
A calcaneus deformity of the foot results from paralysis of the triceps surae, leading to unopposed contraction of its antagonist, tibialis anterior. The pull of the short plantar muscles is unopposed, thereby drawing the tubercle of the calcaneus forward into a near vertical position. A concomitant paralysis of the tibialis anterior and toe extensors would lead to unopposed contraction of the toe flexors with a resultant calcaneocavus deformity.

Equally important as the muscular support of the foot is the bony architecture. Harris and Beath<sup>3</sup> compared the leg and foot to an obelisk (the tibia), based on a plinth consisting of two superimposed foundation stones (the talus and calcaneus) (Figure 1). If the foundation stones are smoothly cut with all surfaces at right angles to each

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after Harrison &amp; Beath

FIGURE 1

The leg and foot may be compared to an obelisk (the tibia), based on a plinth consisting of two superimposed foundation stones (the talus and calcaneus).

other; if they fit accurately and are precisely superimposed upon one another; and if they rest upon a level foundation, the weight of the shaft is carried without danger of collapse. Should the foundation stones be imperfect in their outline, tilted or not accurately superimposed, the weight of the column will be off-center and the structure will be unstable. The stability of the foot is provided by its bony structure, muscle balance and ligamentous support. Weightbearing and propulsion are most efficient when body weight is transmitted directly to the ground; a balance between skeletal structure and soft tissue support is essential.

The less stable the skeletal structure, the greater is the demand on the soft tissues. Conversely, a stable bony architecture demands little contribution from the muscles. Thus the goal of stabilization procedures of the foot and ankle is to restore the harmonious balance between bony stability and soft tissue support. This is a complex task when one compares the ankle and subtalar joints to a "universal joint" which permits movement about three axes. The talocrural joint is a hinge joint which only permits movement in a dorsoplantar axis. The subtalar joint is composed of the anterior and posterior talocalcaneal joints, a hinge joint, and a pivot joint, allowing movement in a mediolateral plane. Lastly, the talonavicular joint may be likened to a ball-and-socket joint, allowing rotatory movement of pronation and supination.

### Reconstruction

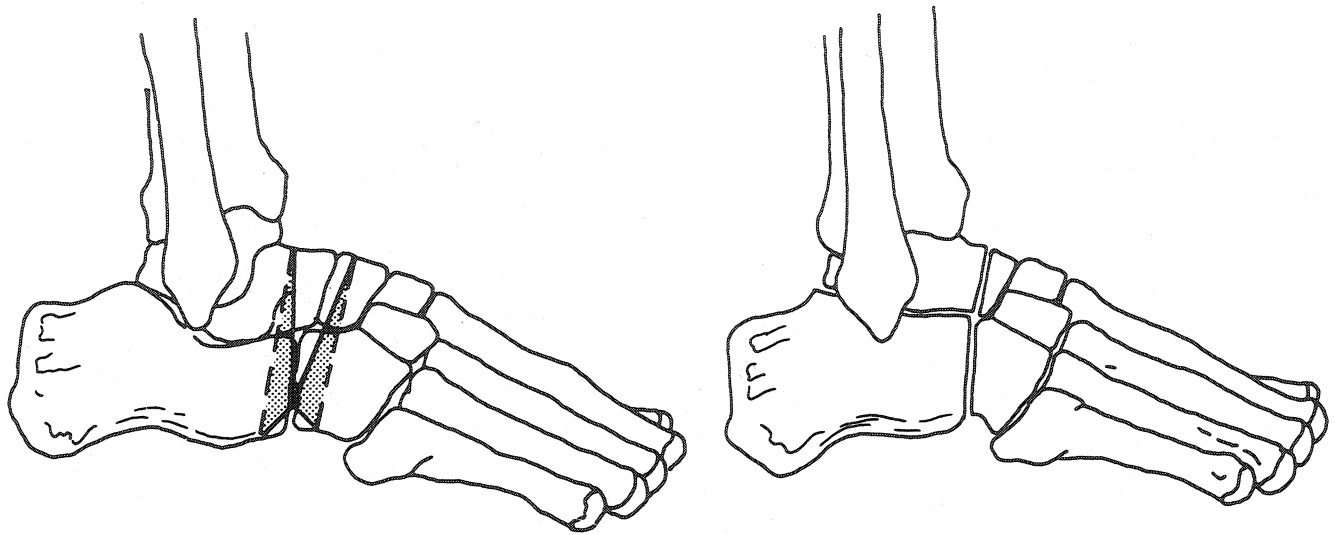
Considering the complex anatomy of the ankle and tarsus, it is not surprising that 56 distinct reconstructive

operations on the skeleton of the foot had been chronicled by Halgrimsson in his extensive review in 1943<sup>1</sup>. The ideal stabilization procedure is one which would obliterate or limit ankle and subtalar motion, effecting a stable and painless transfer of body weight to the midfoot. The first arthrodesis by an American surgeon is credited to Louis Bauer of New York in 1860<sup>1</sup>. Bauer performed an arthrodesis on a 14-year-old boy with a deformed knee by denuding the articular surfaces of cartilage. Arthrodesis then became a consideration for the treatment of deformed feet secondary to poliomyelitis.

In order to attain stability and function of a flail or deformed joint, a combination of soft tissue and bony reconstruction is often required.

### Soft Tissue Reconstruction

The goal of tendon transfer is to substitute for the function of the paralyzed muscle and restore balance to the joint. A tendon is transected and transferred into another tendon or bone; its neurovascular supply is preserved. The transferred tendon must have a similar excursion to the paralyzed muscle it is replacing and the joint across which the tendon is to act must be mobile. The strength of a tendon transfer is proportional to the potential force and excursion of the individual muscle, that is, the muscle to be transferred must have adequate motor strength and contraction to accomplish its newly acquired function. It may be anticipated that one grade of muscle power is to be lost after a muscle tendon is transferred. The functional demands of ankle plantar flexion, for example, far exceed that of ankle dorsiflexion. Consequently, replacing the



### Intra-articular Arthrodesis

FIGURE 2

Intra-articular arthrodesis of the subtalar joint consists of fusion of the subtalar, calcaneocuboid, and talonavicular joints.

function of the plantar flexors requires more strength than that of the dorsiflexors. Effective ankle plantar flexion often requires the additional action of two or three motor groups. The specific constellation of tendon transfers must be individually designed to suit the functional requirements of the limb.

#### Bony Reconstruction

Stabilization of the hindfoot often requires more than soft tissue reconstruction. Arthrodesis about the foot and ankle provides stability, allows for the correction of deformity, and results in increased function of the extremity. Bony stabilization procedures of the foot and ankle can be subdivided into several categories:

1. Intra-articular arthrodesis
  - a. Pantalar
  - b. Subtalar -Triple Arthrodesis
2. Extra-articular arthrodesis
3. Arthrorisis (anterior or posterior bone blocks to limit motion at the ankle joint)

#### Intra-articular Arthrodesis—Pantalar:

A pantalar arthrodesis consists of fusion of the ankle, talocalcaneal, calcaneocuboid, and talonavicular joints. The fusion is achieved by excision of all exposed cartilaginous surfaces, osteotomy and bone wedging to correct deformity, and immobilization in the corrected position until union has occurred. The initial report on pantalar arthro-

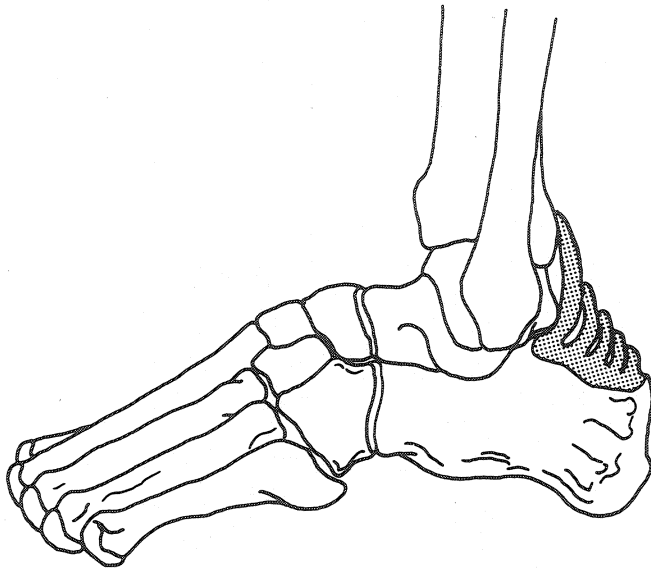
desis was published by Lorthior in 1911. He removed the talus, denuded it and the opposing surfaces of cartilage, and then replaced it in its previous position. By this means he obtained ankylosis of the talocrural, talocalcaneal, and talonavicular joints. Many modifications of this procedure have been made; however, it remains an "effective and satisfying"<sup>12</sup> procedure for providing stability to the flail ankle and hindfoot.

#### Intra-articular Arthrodesis—Triple Arthrodesis:

This procedure<sup>27,29</sup> consists of fusion of the subtalar, calcaneocuboid, and talonavicular joints, (Figure 2) and provides hindfoot stabilization while preserving freedom of motion at the ankle joint. In this manner, muscular function is optimized without the risk of lateral or rotatory displacement. In effect, the "universal joint" of the foot is changed into a hinge joint, permitting movement about a single axis. This procedure obviates the muscular requirements of lateral and rotatory foot stability, allowing existing muscles to be used solely for plantar flexion and dorsiflexion of the foot. In this manner, the efficiency of locomotion is maximized.

#### Intra-articular Arthrodesis—Limited:

In situations where an individual articulation of the tarsus and midtarsus is affected, a limited intra-articular arthrodesis is indicated. For example, in post-traumatic subtalar arthritis secondary to calcaneal fracture, a limited intra-articular arthrodesis of only the talocalcaneal joint is warranted.



**Campbell Procedure 1923, 1925**

### Posterior Arthrorisis

**FIGURE 3**

In the posterior arthrorisis, according to Campbell, a bone block is created on the dorsum of the calcaneal tuberosity, effectively limiting plantar flexion while preserving dorsiflexion.

#### Extra-articular Arthrodesis:

This procedure involves the creation of an extra-articular bony ridge. In this manner, an ankylosis is created about a joint. Since the procedure is extra-articular, growth disturbance is kept to a minimum, making this procedure the alternative to intra-articular fusion in a child. The Grice subtalar arthrodesis is a common example<sup>19</sup>.

#### Arthrorisis:

Arthrorisis describes procedures which are aimed at limiting motion in a joint while maintaining some mobility. The goal is to create a bony endpoint or check rein, past which a joint is not allowed to move. In 1923, Campbell<sup>11</sup> described an operative technique designed to prevent dropfoot secondary to infantile paralysis (polio) (Figure 3). In the posterior arthrorisis, according to Campbell, a bone block is created on the dorsum of the calcaneal tuberosity, effectively limiting plantar flexion while preserving dorsiflexion. In 1922, Putti described a surgical procedure intended to prevent excessive dorsiflexion in a calcaneus foot with paralysis of the triceps surae<sup>30</sup>. In this anterior arthrorisis, a bone wedge was placed into the anterior part of the trochlea tali, thereby creating a bony block which struck against the anterior border of the tibia when the foot was doriflexed.

#### Summary

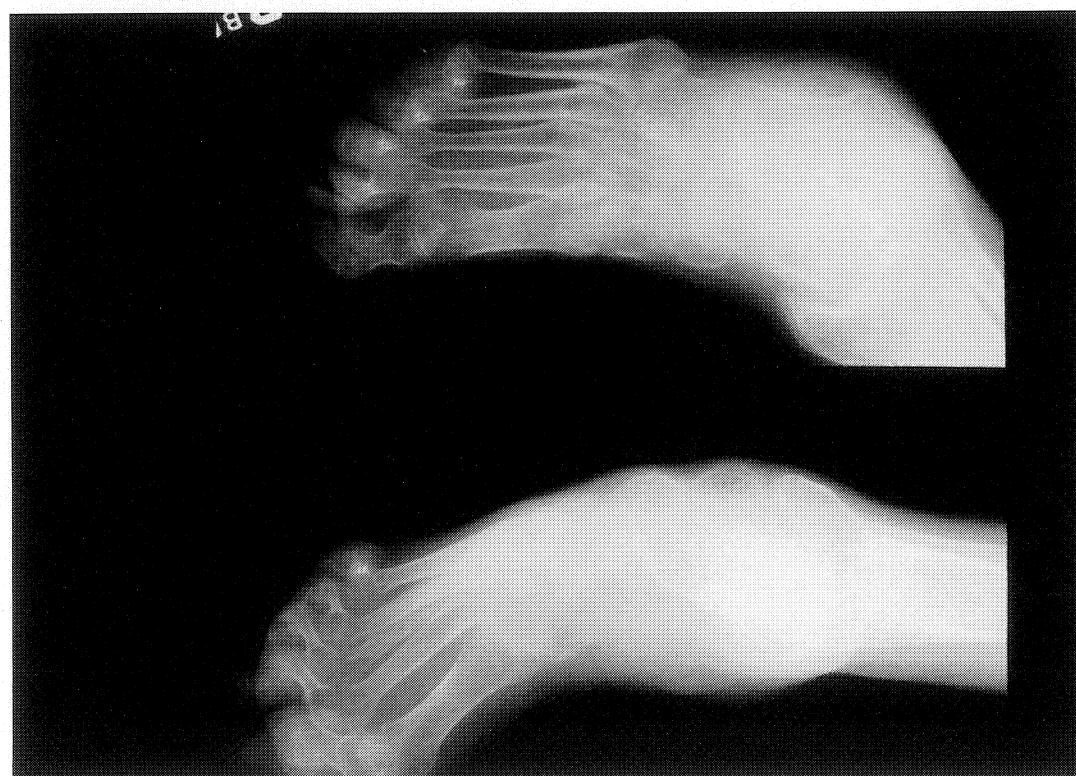
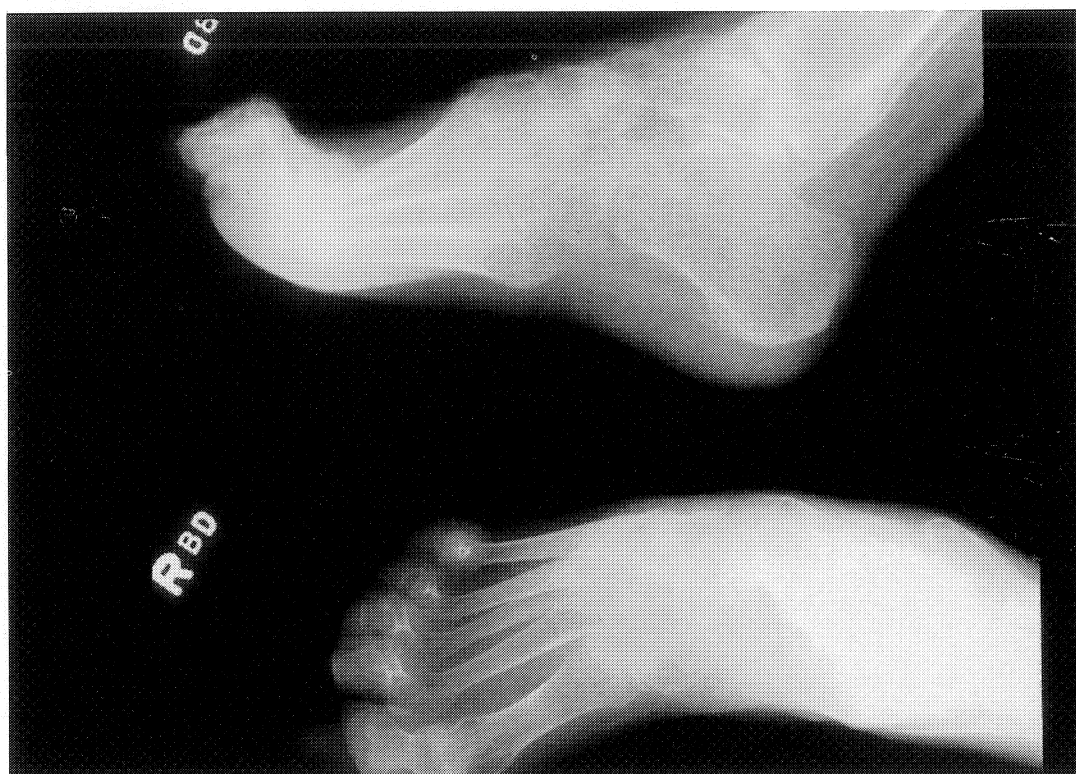
A variety of diseases affecting the lower extremity have led to a development of stabilization procedures of the hindfoot. The muscle paralysis resulting from acute anterior poliomyelitis, the severe arthritis secondary to trauma or the inflammatory arthritides, and the deformity secondary to congenital and neuromuscular disorders, present the orthopaedic surgeon with challenging problems. These problems may be solved by careful biomechanical analysis and operative planning; the soft tissue viability and bony configuration must be carefully scrutinized when attempting to restore equilibrium to the foot and ankle. The ankle and subtalar joint may be compared to a "universal joint" supported by four musculotendinous columns. These columns may be transferred to attain equalization of muscle tension about the foot and ankle. Similarly the talus and calcaneus have been compared to "foundation stones" that support the tibia and provide stability if anatomically aligned. In cases where the musculotendinous columns fail, stability can be restored through a variety of ankylosing procedures that correct the deformity. To optimize function and stability, reconstructive procedures of the flail or deformed foot and ankle may require bony and soft tissue realignment. Tendon transfer may be a critical adjunct to arthrodesis in order to maximize function and prevent recurrence of deformity.

#### CASE ILLUSTRATIONS

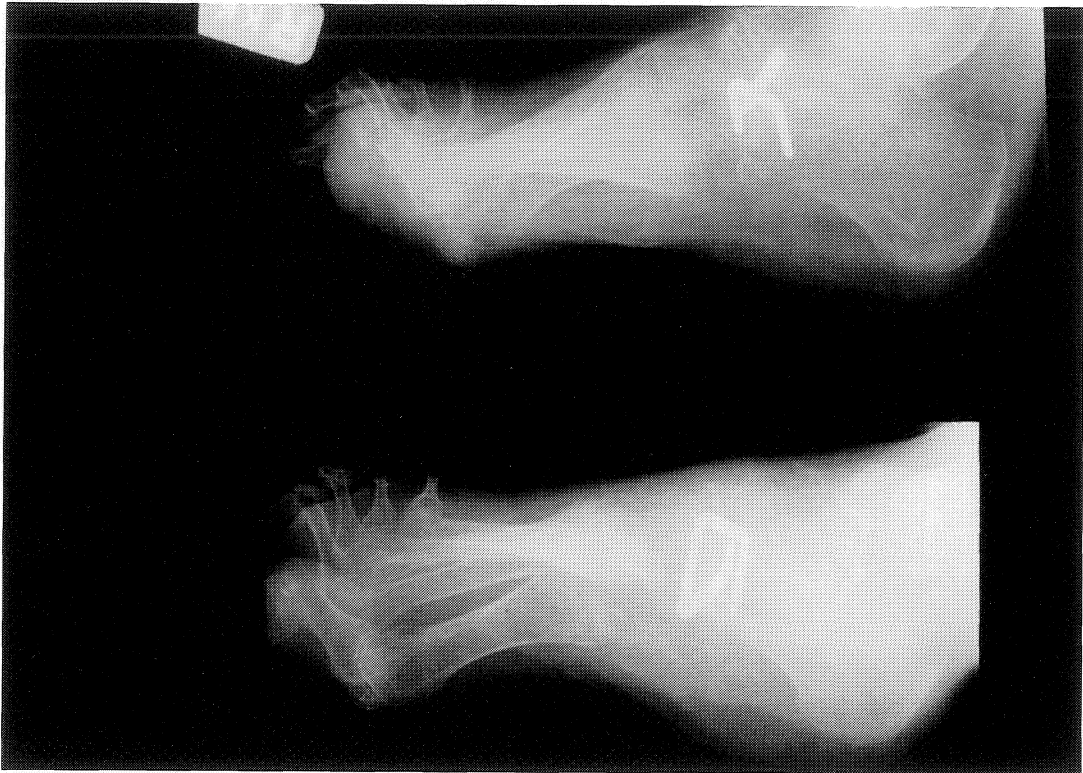
##### Case Illustration #1

Talipes equinovarus—A 58 year old male with Charcot-Marie-Tooth disease (Figures 4A and 4B) complained of increasing pain in the foot due to maldistribution of weight. He had corns and calluses from walking on the lateral side of his right foot and walked in marked varus and 60% of fixed equinus. He also began experiencing low back pain due to his circumducted gait. The patient's walking was affected to the point where he tired easily. The right foot appeared inverted with an extremely tight Achilles tendon which limited the motion of the foot.

The goals of surgery included placing the foot more plantigrade and relieving the pressure on the lateral side in order to improve function and decrease pain. The operation recommended was a heel cord lengthening with a dorsal wedge osteotomy through Chopart's joint to help bring up the forefoot and help correct the varus. The subtalar joint was fused with the heel in slight valgus. Normally a heel cord lengthening is not performed at the same time as a triple arthrodesis, but in this situation, it was warranted to get the best possible position at the time of his fusion.



**FIGURE 4A, B**  
58-year-old male with Charcot-Marie-Tooth disease, showing equinovarus deformity.



**FIGURE 4C, D**  
After triple arthrodesis with dorsal wedge osteotomy and percutaneous heel cord lengthening. Staples were used to cross the calcaneocuboid and talonavicular joints to hold position.



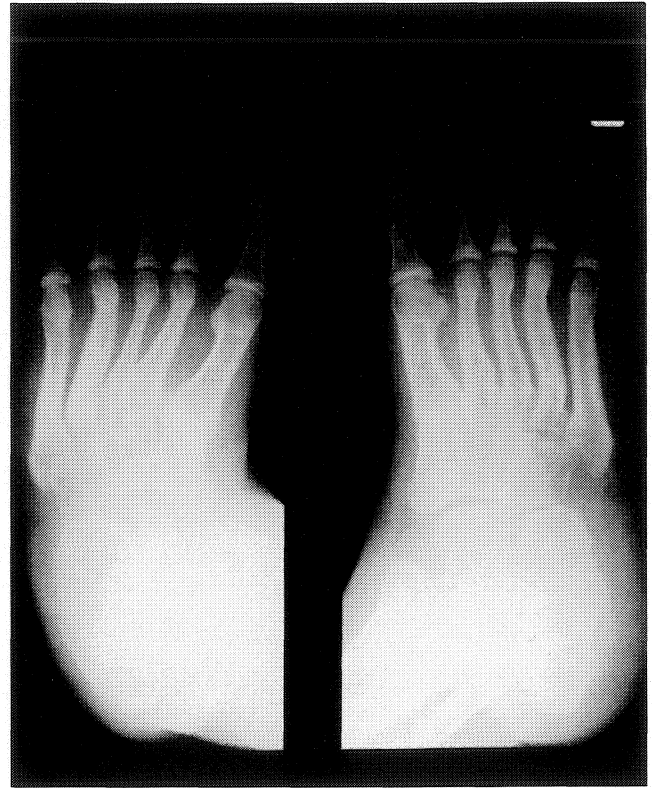
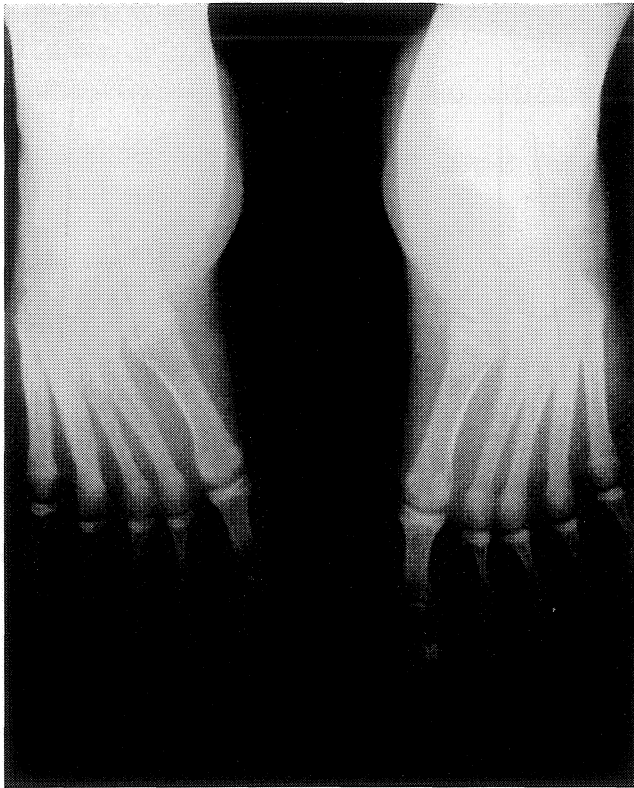


FIGURE 5A, B

A 12 1/2 year old boy with bilateral talipes equinovarus.

**Procedure:** A right foot triple arthrodesis with dorsal wedge osteotomy and percutaneous heel cord lengthening (Figures 4C and 4D). Staples were used to cross the calcaneocuboid joint and the talonavicular joint to hold position. A posterior plaster splint was applied which was circularized to a cast on postop day #2.

**Post-Op Course:** The patient was up on crutches with weight bearing as tolerated in the immediate postoperative period. A Texas Rehabilitation type of ankle-foot orthosis was worn long-term after the surgery.

**Follow-up:** The patient was well satisfied with the results of his triple arthrodesis. The fusion was clinically solid. The foot pain and calluses disappeared. Extra depth shoes were worn to accommodate marked clawing of the toes in both feet.

### Case Illustration #2

Talipes equinovarus—A 12 1/2 year old boy was born with bilateral club foot deformities (Figures 5A and 5B). He was treated with serial casting until the age of 10 months at which time bilateral heel cord lengthenings and medial soft tissue releases of both feet were performed. He had not worn special shoes for the past 5 1/2 years, but had severe problems with shoe wear. He had pain around both ankles as well as the fifth metatarsal head. His symptoms were much worse on the right side. He could

walk no more than five minutes without excruciating pain. He had trouble with his balance because of his feet, as well as difficulty with stairs.

On physical examination the patient was 5'2" tall and weighed approximately 180 lbs. There was marked calf atrophy bilaterally. Motor and sensory function were symmetrically intact. He had bilateral cavus deformities with dropped forefeet. Both feet could be dorsiflexed to neutral position. He had 15° of heel varus bilaterally and diminished subtalar motion. He had no push off and walked with short steps.

A weight reduction program was begun and a triple arthrodesis of the right foot was proposed when the patient reached skeletal maturity.

**Procedure:** A triple arthrodesis of the right foot was performed at age 14 (Figures 5C and 5D). A staple was placed at the calcaneocuboid joint. Postoperatively, a posterior splint was placed with the foot in neutral position. This posterior splint was circularized on postop day #2 to a short leg cast.

**Follow-up:** At three months time, roentgenograms revealed early bony bridging of all three of the joints with the staple across the calcaneocuboid joint. The patient was taken out of plaster and started on flatfooted partial weight bearing as tolerated. At one year, there was persistent nonunion at the talonavicular joint. The patient was fitted



FIGURE 5C, D

A unilateral triple arthrodesis was performed at age 14. A staple was placed at the calcaneocuboid joint. Note the deformity of the dome of the talus.



FIGURE 6B

A 70-year-old female with unexplained peripheral neuropathy, resulting in Charcot changes with peritalar dislocation.



FIGURE 6A



FIGURE 6C

The talonavicular dislocation was reduced and a triple arthrodesis was done.



FIGURE 6D

for an ankle-foot orthosis with molded insoles and showed marked improvement. Note the deformity of the dome of the talus. Ankle fusion may improve his mobility and obviate the need for orthoses.

### Case Illustration #3

Charcot Joints and Peritalar Dislocation (Figure 6A, 6B)—A 70 year old female recalled injuring her right foot when she was walking down a ramp to her house. Since that time her foot was painful and swollen, especially on the medial side. She was placed in a short leg cast for two weeks at which time the cast had to be removed due to an ulceration with infection on the medial side of the foot. She experienced progressive deformity of the right foot with complete dislocation of the talonavicular joint and dislocation of the subtalar joint. The right foot was in marked eversion with the head of the talus very prominent in the long arch of the foot. In a standing position she toed out 60° on the right with the patella pointing straight ahead. There were no ulcerations, but the foot had a red patch over the medial malleolus. In a standing position the head of the talus directly bore weight in the long arch of the foot. She had a very slight decrease in light touch sensation of the fourth and fifth toes. Position sense was markedly impaired as well. Workup of this patient proceeded with a complete medical and neurological evaluation to determine the source of her neuropathy. Mild type II diabetes was found. A protective Texas Rehab type of

ankle-foot orthosis was used to prevent any further injury to the ankle and foot. Eventually, the patient was indicated for corrective surgery to prevent the development of further deformity and ulceration leading to infection and possible limb loss.

**Procedure:** Through medial and lateral incisions, the talonavicular dislocation was reduced and a triple arthrodesis was performed (Figures 6C and 6D). The position of the foot was held postoperatively by staples. The patient required protection in a cast for a long period of time as the fusions consolidated.

The patient was given a Texas Rehabilitation type of ankle-foot orthosis after cast removal to protect her from deformity in the event of the development of a Charcot ankle.

**Follow-up:** Over four years the foot slowly drifted back into valgus position, losing approximately 25% of the reduction. The patient was able to get around enough to perform her activities of daily living and did not have any significant pain. She continued to wear the brace.

### Case Illustration #4

Charcot Ankle—A diabetic 60 year old woman with severe peripheral neuropathy underwent ORIF of the left ankle after sustaining a bimalleolar fracture (Figure 7A and 7B). Plaster immobilization was used for only a short time and the patient subsequently developed a varus deformity of the ankle secondary to fracture of the metal plate and

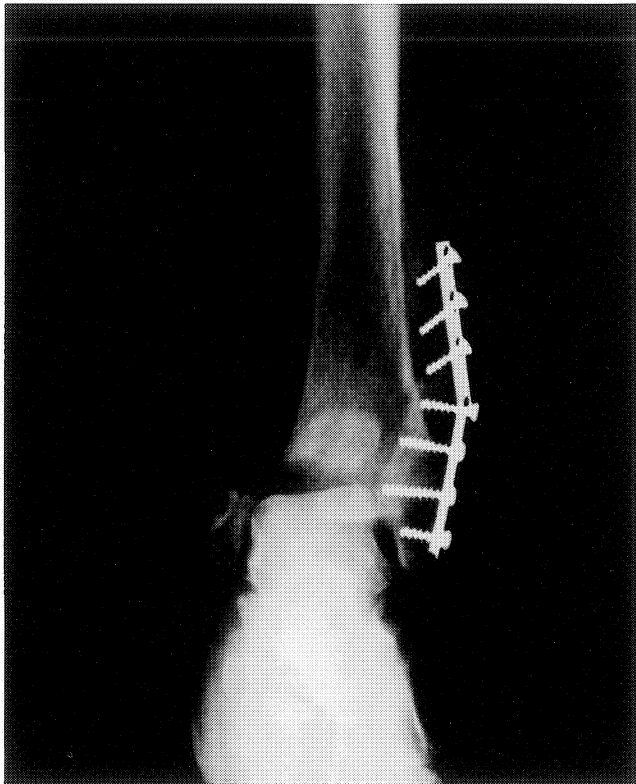


FIGURE 7A, B

loss of reduction. The patient went on to develop an ulcer over the lateral malleolus. The patient had little sensation in her foot and the ankle was grossly unstable.

Although some Charcot joint changes are gradual in nature, this patient had a fairly typical presentation. Her ankle was neuropathic and should have been treated accordingly. Review of the initial roentgenograms showed the ankle and foot to be in a reasonably well aligned position beneath the distal tibia, without migration medially or laterally. The foot and ankle could have been immobilized in a well padded short leg cast until all soft tissue swelling subsided and until all increased warmth around the ankle disappeared.

Plaster immobilization should be continued much longer than in the nonneuropathic patient. A Texas Rehab Institute type of ankle-foot orthosis should then be fitted and worn continuously after the cast removal. If it is possible to keep the foot in a reasonably well aligned position, the patient may maintain a functional extremity. Rarely ulceration of the foot and ankle results from the use of an orthosis. This should be considered an acceptable risk of a required treatment.

In this case increasing deformity developed with ulceration of the foot and instability at the ankle on weight bearing. If left untreated, this situation would have led to deep infection and eventual loss of the extremity. Therefore, an attempt to shift the foot back to a stable, neutral,

position for weight bearing was indicated. It was unlikely that bony fusion would result; however, a fibrous ankylosis with the foot in corrected position would be quite satisfactory.

Procedure: Removal of hardware and "fusion" of left ankle (Figures 7C and 7D). Following the removal of broken hardware, the tibiotalar joint was denuded of cartilage and subchondral cortex and reduced from an unstable to a stable position. Two heavy threaded Steinmann pins were placed in a retrograde fashion through the calcaneus and talus into the tibia. These pins were cut just at the skin level and a well padded short leg cast was applied. The patient was instructed to be nonweightbearing on the affected limb until a bony or fibrous ankylosis occurred. In general, this averages four to six months time. The pins were then removed and an ankle-foot orthosis worn long term.

Follow-up: The pins were removed and the patient was allowed to begin weightbearing with a Texas Rehab type of ankle-foot orthosis at five months. At ten months roentgenograms revealed some callus formation and maintenance of satisfactory alignment of the ankle.

#### Case Illustration #5

Post Traumatic Arthritis—A 25 year old fell from a second story window at home and fractured his right os calcis (Figures 8A and 8B). Initial treatment consisted of a

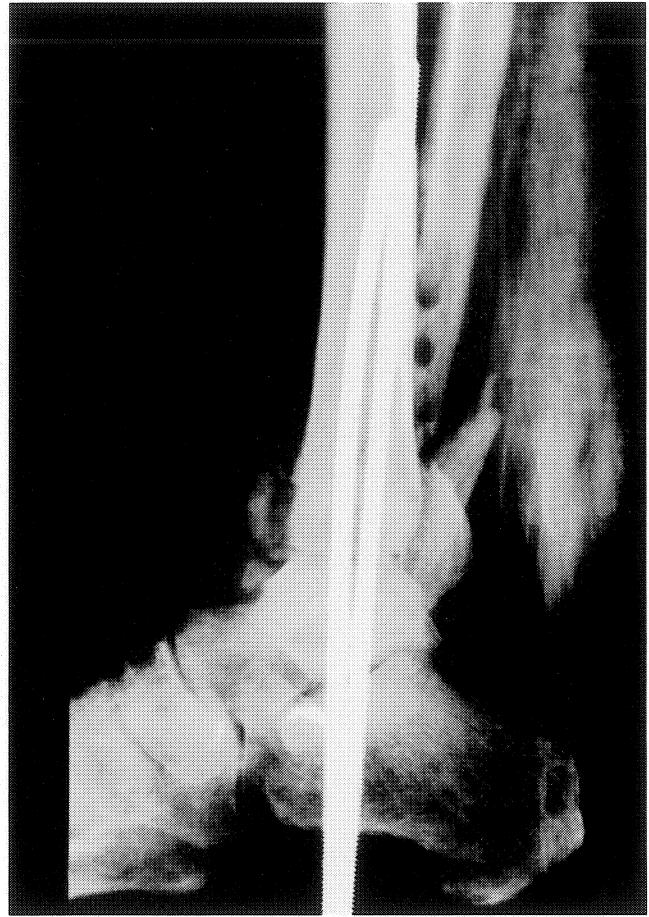


FIGURE 7C, D

Removal of hardware and fusion of the left ankle. Two heavy threaded Steinmann pins were placed in a retrograde fashion through the calcaneus, talus, and into the tibia.

soft pressure dressing on the ankle and foot for six days. A nonweightbearing short leg cast was then applied. Two months after the injury the cast was removed and weight-bearing was advanced. The patient was able to ambulate with the assistance of a cane in his right hand, wearing an ankle brace with steel stays. However, walking on uneven ground was difficult. He was unable to run and noted that stair climbing was very difficult. He could not carry any load and could not walk farther than two blocks before he had to stop and rest because of pain.

There was pain and marked decrease in range of motion at the right subtalar joint. Lisfranc's joints were open bilaterally and nonpainful. The heel was wide and the calcaneal tuberosity rode high. There was a full painless range of ankle motion.

Tomograms revealed impaction of the posterior facet into the bony substance of the os calcis (Figure 8C). There was a spike of bone protruding from the os calcis which crossed the calcaneocuboid joint (Figure 8D).

This patient displayed post-traumatic subtalar arthritis resulting from a calcaneal fracture. Tomograms showed

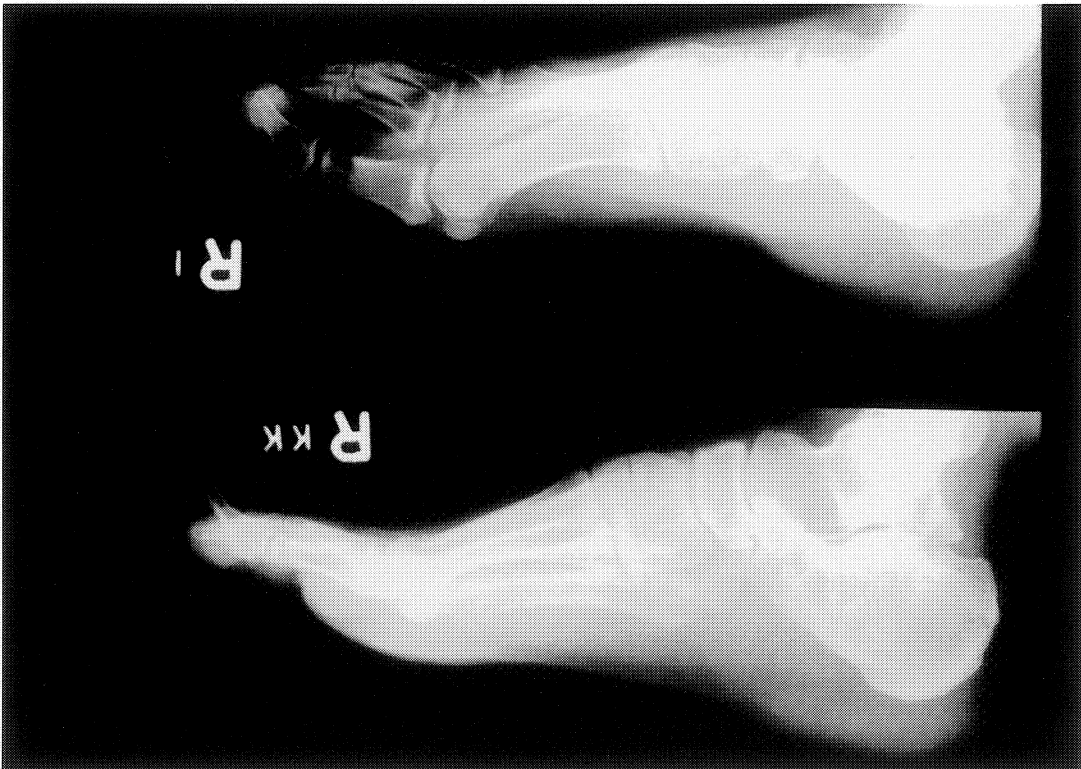
arthritic changes of the subtalar, calcaneocuboid, and talonavicular joints, in decreasing severity (Figure 8E).

**Procedure:** Triple arthrodesis of the right foot (Figures 8F and 8G). Using a combined medial and lateral approach, the talocalcaneal, calcaneocuboid, and a talonavicular joints were denuded and fixed with staples. The foot and ankle were placed in a short leg cast and the patient mobilized on crutches.

**Follow-up:** Patient remained nonweightbearing in a short leg cast until evidence of fusion was demonstrated on roentgenograms. Pain was greatly diminished, and function markedly improved. Activities of daily living were now possible without use of an orthosis or cane.

#### Case Illustration #6

Hindfoot varus with metatarsus adductus—A 43 year old who was involved in a motor vehicle accident in which he sustained a sciatic nerve injury. He underwent a posterior tibial transfer to the dorsum of the foot for a drop foot deformity. Several years later, the patient described increasing pain on the lateral side of the foot, specifically over the calcaneocuboid joint. He noticed swelling on the



**FIGURE 8A, B**  
A 25-year-old with intra-articular fracture of the calcaneus.

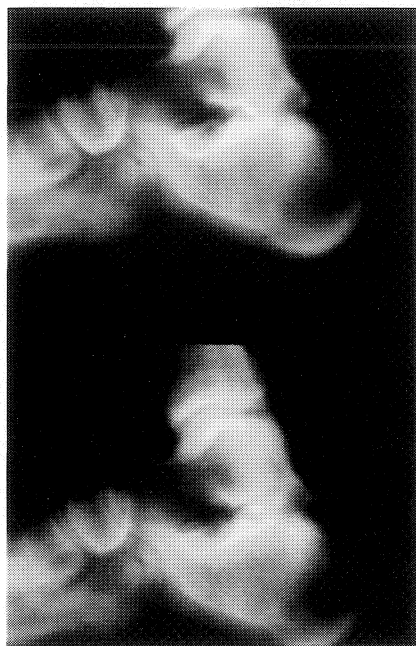


FIGURE 8C

Tomograms revealed impaction of the posterior facet into the bony substance of the os calcis.

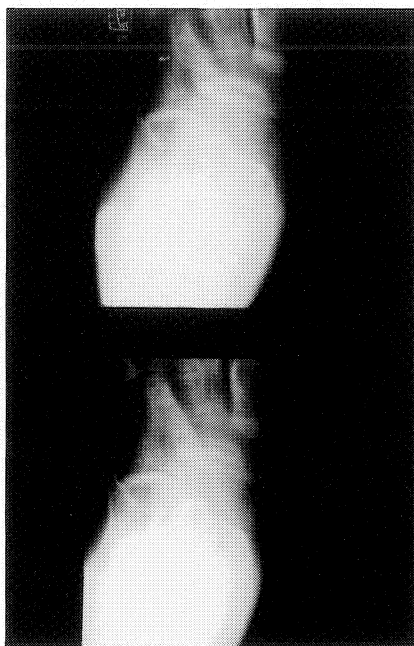


FIGURE 8D

Tomograms reveal a spike of bone protruding from the os calcis which crossed the calcaneocuboid joint.



FIGURE 8E

Tomograms reveals early arthritis of the talonavicular joint.

lateral side of his foot and ankle toward the end of the day and a progressive adduction deformity of his forefoot. He was able to ambulate without assistance but could not run. He went upstairs in a nonreciprocating fashion and felt unstable.

On physical exam the heel was in 10-15° of varus with 15° of metatarsus adductus. The ankle had 0-25° of plantar flexion but no dorsiflexion. There was a small jog of motion in the subtalar joint. Motion at Chopart's and Lisfranc's joints was supple with referred pain to the calcaneocuboid joint region. Active dorsiflexion of the foot (by his posterior tibial transfer) was 4/5 in strength. Plantar flexion was 4+/5. There was no active contraction of the peroneals.

Roentgenograms revealed arthritic changes in the calcaneocuboid joint. The subtalar joint appeared to be normal.

Several treatment plans were entertained. One was to do a simple osteotomy of the os calcis, either with a lateral closing wedge (the Dwyer procedure) or a straight horizontal cut through the tuber, translating the tuber laterally (the Gleich procedure), along with a corrective osteotomy through the metatarsals of the forefoot. A less aggressive approach would be to start by doing a calcaneocuboid fusion and then assess on the operative table the flexibility of the hindfoot deformity. If flexible, the hindfoot should shift out of varus at the same time the forefoot adductus is corrected. If rigid, a formal triple arthrodesis would be indicated.



FIGURE 8F

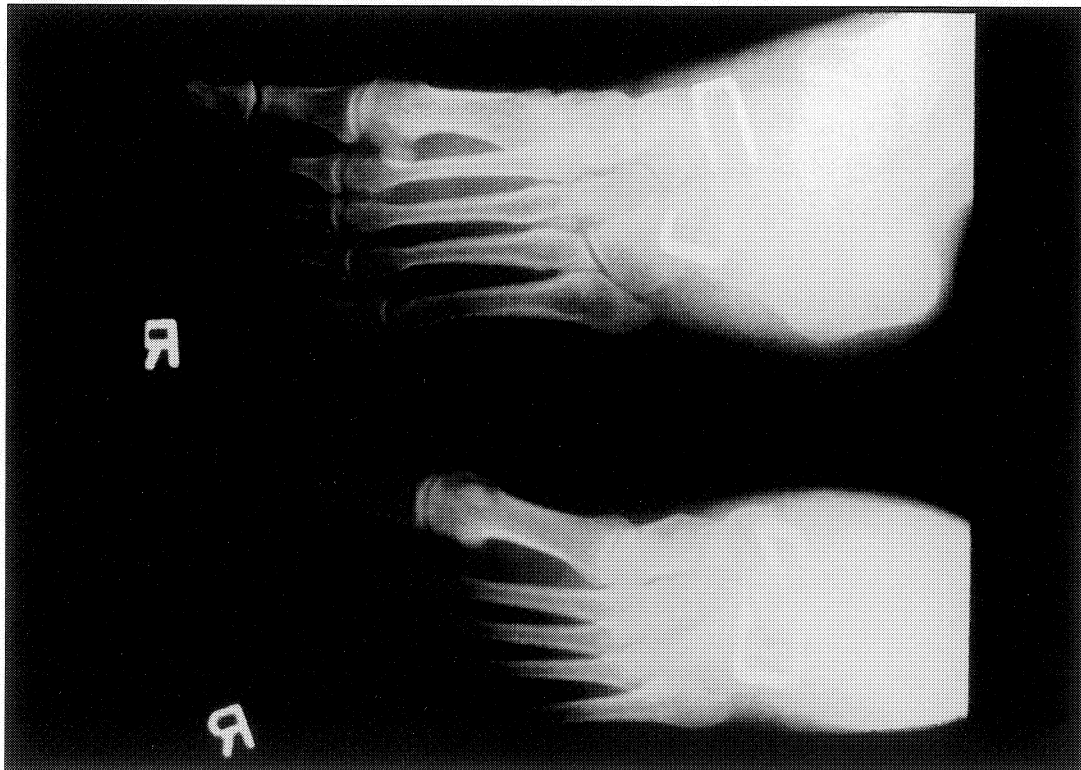


FIGURE 8G



Procedure: A Dilwyn-Evans procedure (Figures 9A and 9B). After medial release, an oblique incision across the calcaneocuboid joint was done and this joint space fused. The heel varus and forefoot adductus were acceptably corrected. The fusion mass was stabilized with a staple. A posterior splint was placed.

Follow-up: At two years there was solid fusion of the calcaneocuboid joint, but persistent pain across the region



FIGURE 9A, B

A 43-year-old with hindfoot varus and metatarsus adductus, following a Dilwyn-Evans procedure.

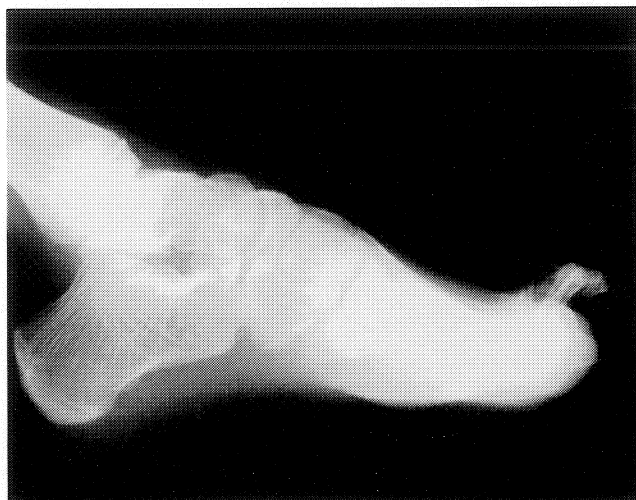
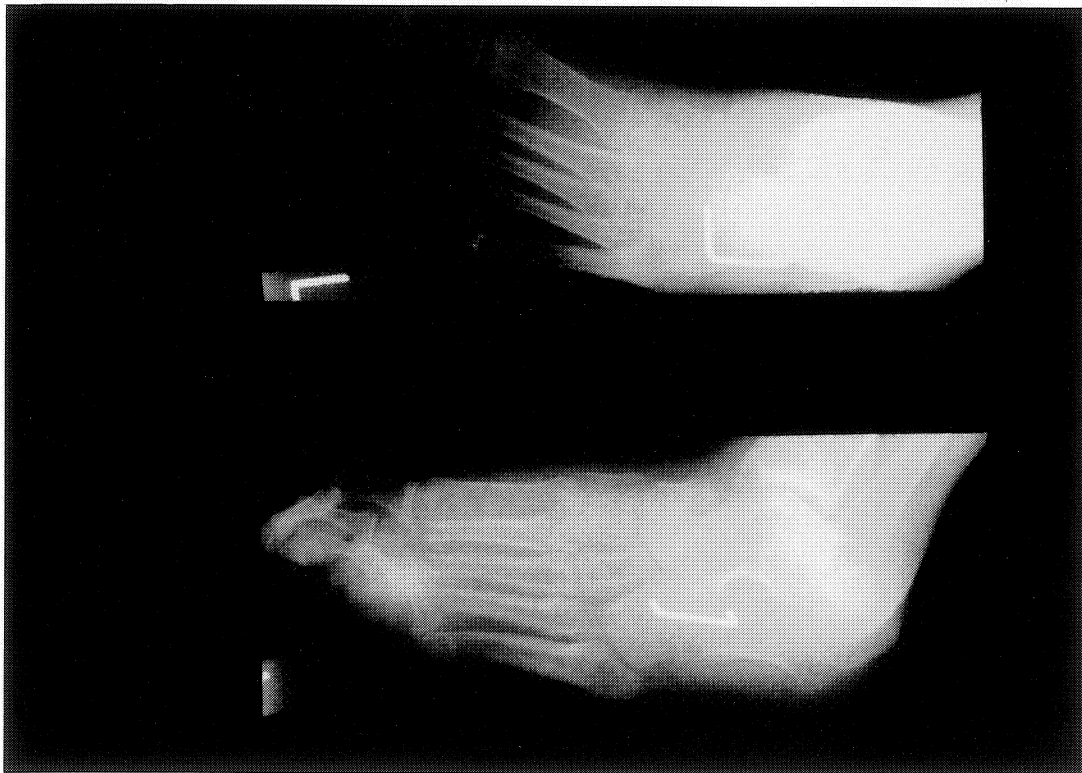


FIGURE 10A, B

A 17-year-old with history of progressive hypertrophic polyneuropathy (Dejerine-Sotta disease) with equinovarus deformity.

of Chopart's joint. The patient remained symptomatic despite wearing a molded insole. A formal triple arthrodesis was offered as a salvage procedure.



**FIGURE 10C, D**  
Following triple arthrodesis. A staple is placed across the calcaneocuboid joint.

### Case Illustration #7

Equinovarus deformity—A 17 year old with history of progressive hypertrophic polyneuropathy (Dejerine-Sotta disease) had painful equinovarus deformities bilaterally (Figures 10A and 10B). There was no limitation in walking endurance, nor were there problems with shoeing. The deformities were progressive and of cosmetic concern to the patient.

On physical examination, the patient had gross sensory loss in both lower extremities with almost total anesthesia of the soles of the feet along with marked diminution of proprioceptive sensation in all of the toes. There was marked weakness of the peronei on both sides and a lesser degree of weakness of the posterior tibial muscles. The patient could walk on his toes reasonably well but he could not walk on his heels. Anterior tibial pulses were weak and a great deal of the dorsiflexion of the foot was accomplished by extensor tendons to the toes. There were no deep tendon reflexes of the ankle or knee on either side.

In this case, consideration should be given to the goals of the proposed procedure. This patient had no problems with shoeing, pain or impairment. His concerns were mainly cosmetic. However, his deformities were likely to progress due to the nature of his underlying condition. Therefore, the main indications for surgical intervention were to prevent increasing deformity and to improve cosmesis.

**Procedure:** Bilateral triple arthrodeses were performed two years apart (Figures 10C and 10D).

**Follow-up:** Following bilateral triple arthrodeses, the patient developed gradually recurrent unilateral equinovarus deformity of the foot requiring revision five years postoperatively. Over the next four years the patient developed further plastic deformities of both feet, slipping back into an equinovarus position bilaterally. There was severe clawing of the toes of both feet secondary to his neurologic disease. The patient was doing well in terms of function and had no complaints of pain; therefore, nothing more was done. Extra depth shoes and a molded insole were fitted.

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# A HAMARTOMATOUS JOINT MIMICKING DYSPLASIA EPIPHYSEALIS HEMIMELICA OF THE TALUS

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## INTRODUCTION

Dysplasia epiphysealis hemimelica is a developmental disorder of childhood characterized by asymmetrical car-

tilaginous overgrowth of an epiphysis, tarsal bone or carpal bone. This disorder most commonly affects the talus, distal femoral epiphysis and distal tibial epiphysis. Patients seek medical evaluation because of an enlarging mass, pain, deformity, or restricted joint motion. Radiographs reveal an irregular lobulated mass protruding from the affected bone. The ossification center of the lesion is

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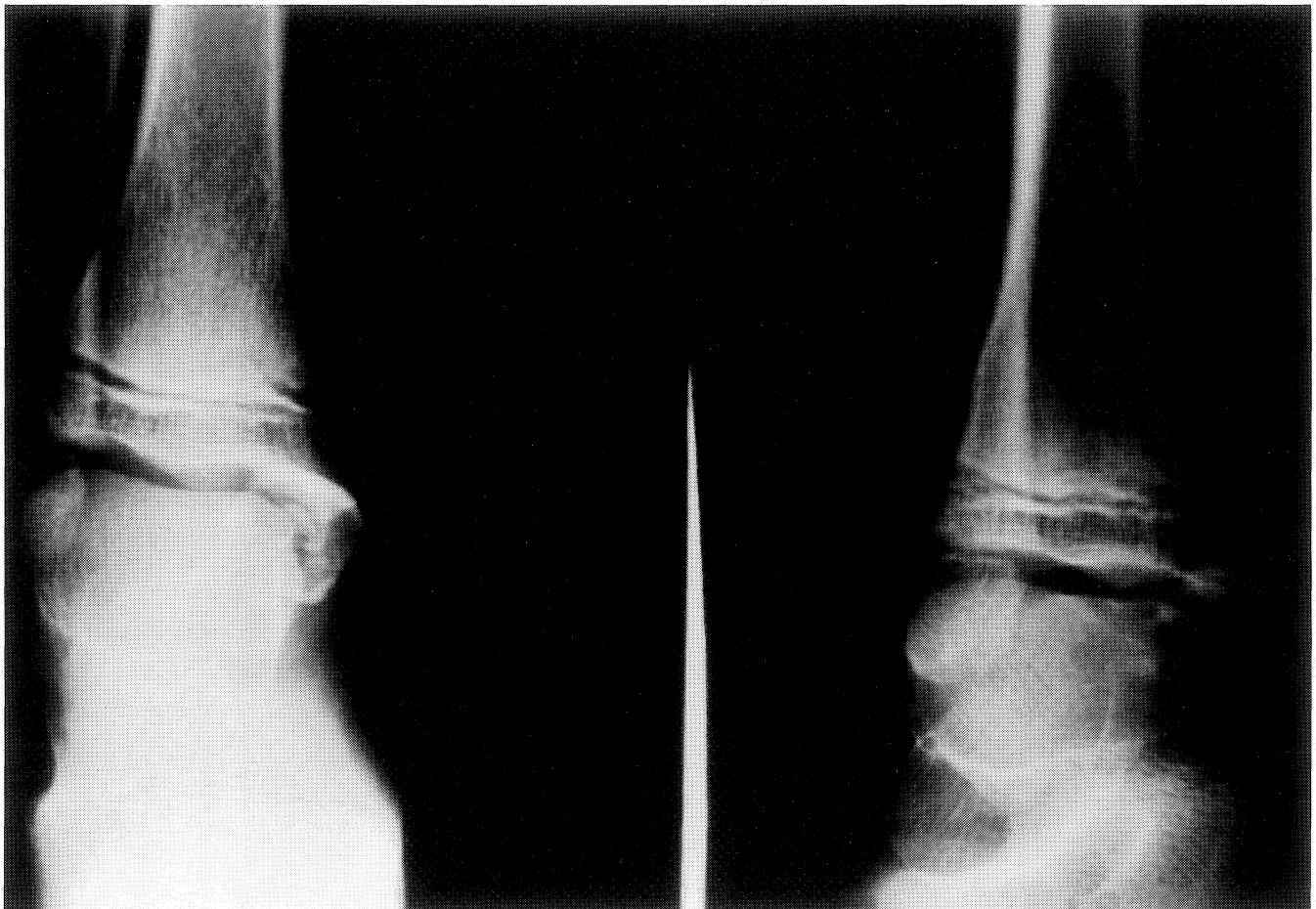


Figure 1a

AP and Mortise of the ankle.



**Figure 1b**  
Lateral of the ankle. The irregular lobulated bony mass was interpreted as a dysplasia epiphysealis hemimelia rising from the talus.

distinct from that of the affected bone in childhood, but becomes confluent with maturity. Pathologically these lesions resemble osteochondromas.

We saw an eleven year old boy who presented with a lesion clinically and radiographically indistinguishable from dysplasia epiphysealis hemimelica of the talus. At surgery the lesion was a hamartomatous joint with an intra-articular ligament connecting the talus to an osteochondroma articulating with the posterior talus.

This finding raises several questions. First, might other lesions previously called dysplasia epiphysealis hemimelica be hamartomatous joints? Second, is this lesion a hamartoma or an atavistic remnant of a joint-ligament-joint complex existing in a lower animal ankle joint? Third, what is the embryology of intra-articular ligament formation? Finally, what is the developmental stimulus and control of intra-articular ligament formation?

#### **Case Report**

A ten year ten month old boy presented for evaluation of a recently noticed posteromedial ankle mass. The patient denied pain except with direct trauma to the mass. Physical examination revealed a 2 x 1 cm apparently fixed,

mildly tender mass between the Achilles tendon and medial malleolus. The affected foot lacked 10° of dorsiflexion compared to the normal foot and the heel was in slightly more valgus than the normal side. Radiographs were interpreted as showing dysplasia epiphysealis hemimelica (Fig. 1).

Over the next year the patient developed increasing discomfort with intermittent local trauma and requested excision. On preoperative examination, the mass was noted to be mobile. It was presumed that a fracture had occurred through the base of the osteochondroma. At surgery, the mass was found to be mobile and it articulated with the posterior talus. When released from surrounding soft tissue, which was hypocellular synovial tissue by light microscopy, the mass was seen to attach to the talus by a 1.5 x 1 x 0.3 cm. ligament. Its origin from the articular surface of the talus can be seen in Figure 3. The ligament was excised from the talus where it was attached to subchondral bone (Fig. 4).

Postoperatively the patient has done well with relief of all symptoms.

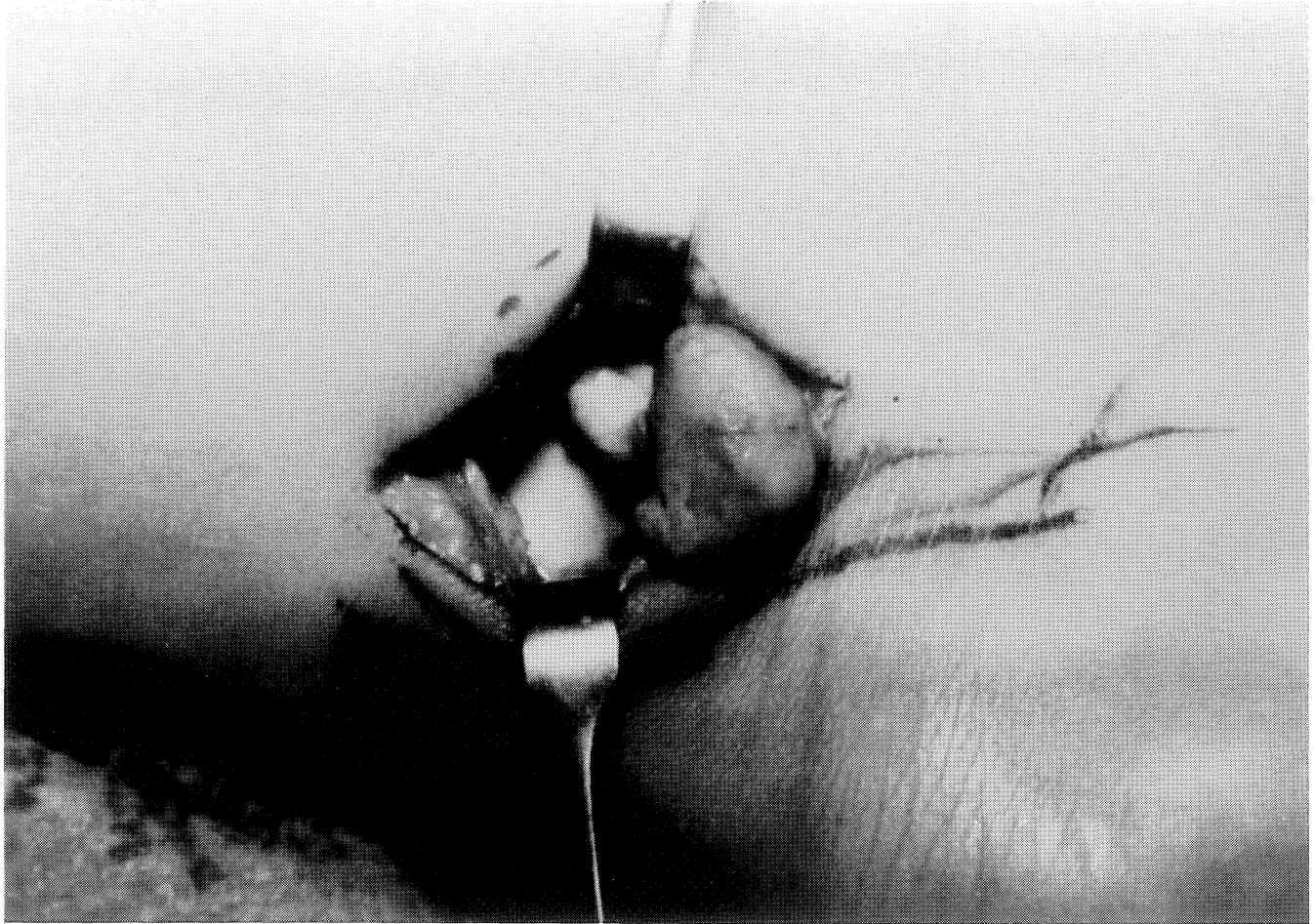


Figure 2

The osteocartilaginous mass is seen *in situ* attached to a stalk of fibrous tissue which inserts on the talus at the base of the wound.

### DISCUSSION

The finding of a hamartomatous anomaly consisting of two joint surfaces connected by an intra-articular ligament raises several questions. Since many lesions carrying the diagnosis of dysplasia epiphysealis hemimelia do not come to surgery, some of these may be similar hamartomas. The occurrence must be rare, however, as no such lesions have been described in the operated cases of dysplasia epiphysealis hemimelia. The only clinical significance in distinguishing these entities is that the prognosis for developing joint deformity or dysfunction is probably better for a hamartomatous joint than a dysplasia epiphysealis hemimelia. However, the presence of this lesion raises questions concerning the normal and abnormal development of intra-articular ligaments.

A reasonable hypothesis for an acquired etiology of this lesion would include an osteochondritic lesion which detached and, rather than healing, became a loose body or imbedded itself within synovium, and then became anchored to the crater via a fibrous stalk. This theory, however, is not consistent with the natural history of

osteochondritis dissecans. Furthermore, a flimsy segment of scar tissue would be expected rather than the well developed ligamentous structure present in our patient.

Recapitulation is a possible etiologic explanation for this abnormality. Differentiation of bones and joints from a generalized blastema is rapid and occurs in a precise sequence. Gardner, in his exhaustive studies on the development and growth of bones and joints, has found no evidence for recapitulation<sup>2</sup>. That is, articular structures characteristic of adult lower forms do not appear during intermediate phases of human development. Also, structures such as ligaments develop *in situ* and undergo no migration of phylogenetic significance<sup>2</sup>. An example is the ligamentum teres of the hip, which earlier was thought to have migrated from its reptilian position outside the joint, where it functioned as a collateral ligament, to its mammalian position inside the joint cavity. However, in rabbits, and most likely in humans, the ligamentum teres arises *in situ* as a condensation<sup>5</sup>. Furthermore, the cruciate ligament of the knee as well as most ligaments of the foot and ankle, also arise *in situ* as cellular condensations<sup>3,4</sup>. Fi-

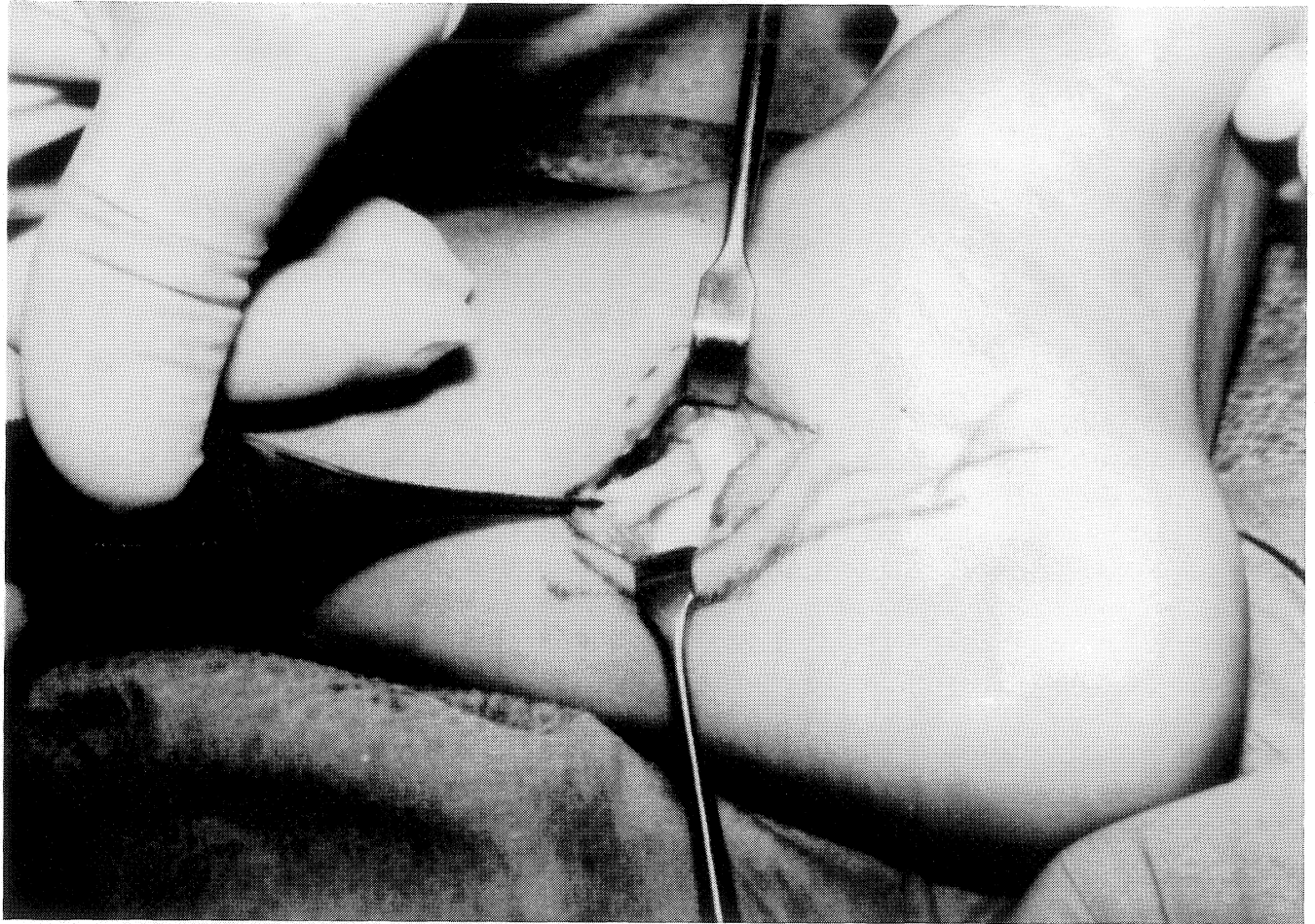


Figure 3

The osteochondral mass has been removed and the ligament-talus complex can be clearly seen.

nally, we were unable to identify any mammals with an intra-articular ankle ligament, although ankle anatomy is rarely presented in books of comparative anatomy.

Errors during embryogenesis may provide an explanation for the development of this anomaly. Whillis found that in relatively late embryos, at stages long after peripheral parts of joint cavities had appeared, adjacent articular cartilages were joined together by a stratum of intervening cells<sup>8</sup>. These cells eventually disappeared, leaving the cartilages continuous with each other<sup>1</sup>. It seems plausible that this stratum of intervening cells could persist, differentiate, and form the above described anomaly.

The well developed structure of the joint-ligament-joint complex found in our patient suggested an alternative hypothesis of faculty embryogenesis. This hypothesis is that the normal signals for pattern formation in the limb were disrupted. Much is known and being learned about the determinants of limb tissue and cell positions during embryogenesis<sup>1,6,7,9</sup>. The proximal-distal positional information is imparted to cells during their time (number of mitoses) under the apical ectodermal ridge<sup>7</sup>. This may be

mediated by variable cell surface adhesion, which is determined by the amount of exposure to integrins stimulated by a transforming growth factor B (TGFB) gradient extending from their apical ectodermal ridge proximally<sup>7</sup>.

The anterior-posterior axis is determined by a zone of polarizing activity from which a retinoic acid gradient diffuses<sup>1,7</sup>. The specific mechanism of action of the gradient is disputed but the retinoic acid gradient may stimulate the activity of a sequence of homeobox genes. These are a class of genes with a highly conserved sequence of amino acids that give morphologic information in every species from insects to man<sup>9</sup>. Bathing amphibian limbs in retinoic acid results in mirror image duplication of digits. Other digital anomalies can be created by transplanting the zone of polarizing activity. Direct evidence for control of morphology by homeobox genes comes from experiments in mice in which alteration of homeobox gene sequence expression can alter vertebral morphology, e.g. transforming a cervical into a thoracic vertebra<sup>6</sup>.

We suspect that an alteration of a normal homeobox sequence or a disruption of a morphogenic gradient re-



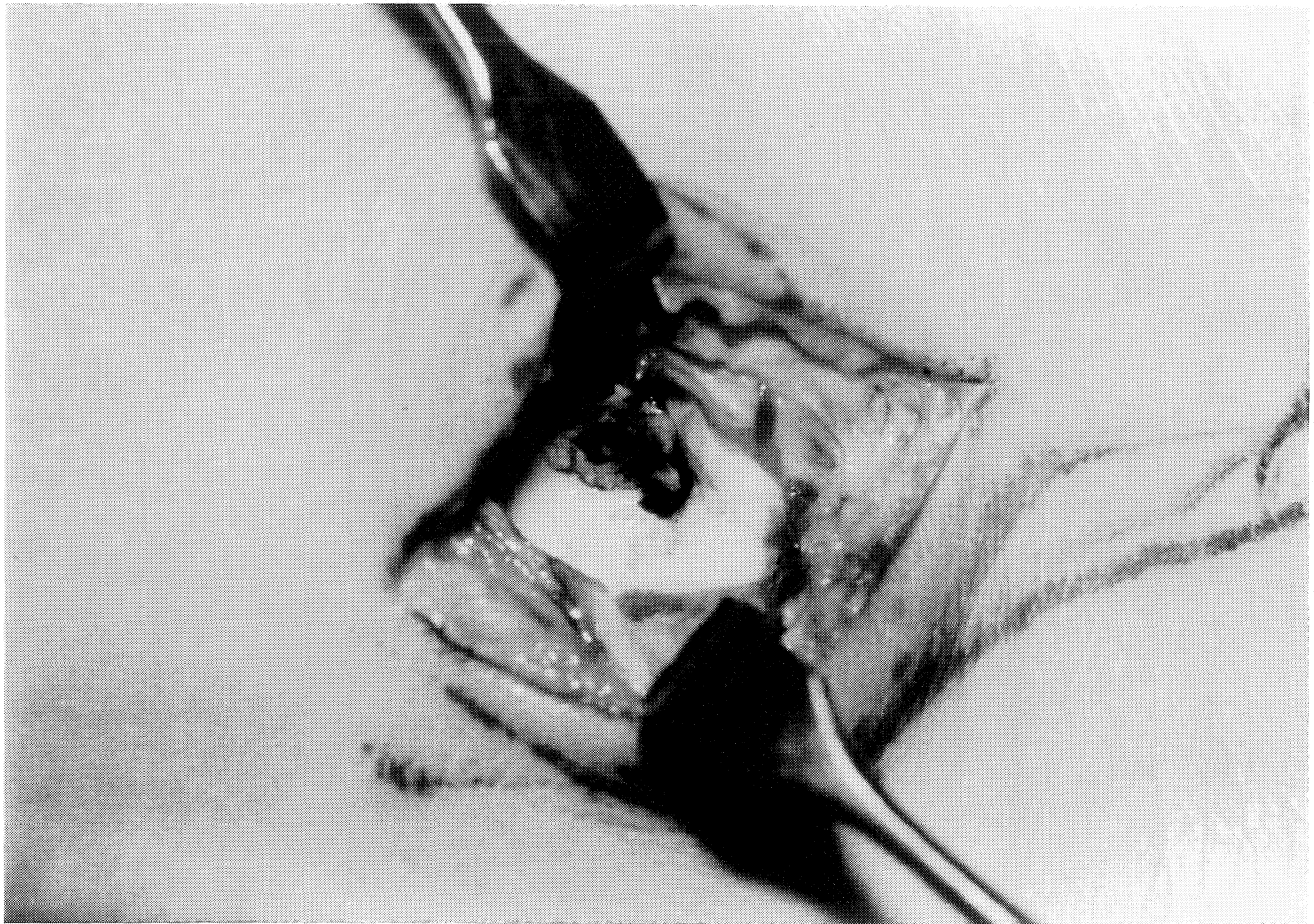


Figure 4

The origin of this ligamentous structure from the talus is seen at the base of the wound.

sulted in this well developed but misplaced joint ligament-joint complex. The ligament appropriately belonged in the hip as a ligamentum teres or in the knee as a cruciate ligament. Further investigation into the determinants of normal limb pattern formation is likely to help explain hamartomas such as described in this report.

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# AN ENVIRONMENTAL HAZARD TO THE DIABETIC FOOT A CASE REPORT

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Reginald R. Cooper, M.D.†

## INTRODUCTION

Diabetes affects an estimated five percent of the U.S. population<sup>1</sup>. The two-thirds of diabetic patients with foot disease<sup>2,3</sup> account for twenty percent of all diabetic hospital admissions<sup>4</sup>. While vascular disease plays an important role<sup>5,6</sup>, decreased protective sensation ranks as the primary risk factor<sup>5,7</sup>. The diabetic patient may remain unaware of a current or developing lesion and therefore not withdraw from the cause. Herein, we discuss an unusual, interesting, and previously unreported example of a potential hazard for the diabetic patient.

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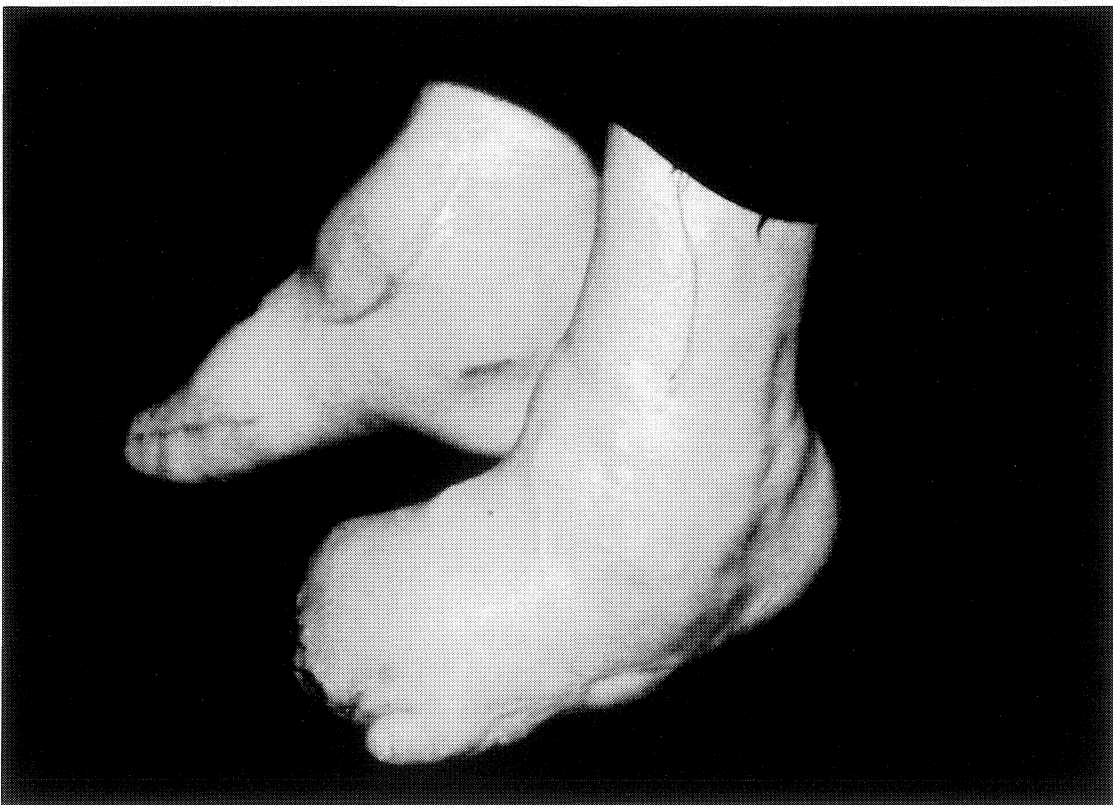
## CASE REPORT

The patient is a thirty-five year old nurse with brittle Type I diabetes mellitus and multiple related medical problems including severe polyneuropathy. She was followed in the Orthopaedic Clinic for Charcot arthropathy of her right ankle. Over an eighteen month period her ankle was managed with a combination of casts and ankle-foot orthoses.

Despite her severe polyneuropathy, her feet remained in relatively good condition. She had had a remote history of osteomyelitis of her right fifth toe which necessitated amputation in 1982. She had no foot ulcerations or infections from 1982 until August of 1991. At that time she presented with traumatic partial amputation of her right great toe.

She suffered the partial amputation during an insulin reaction. Her Lhasa apso (small house dog) ate the otherwise intact portion of her right hallux during a period of unconsciousness. She was treated by revision amputation through the proximal phalanx and after an uneventful





hospitalization she was discharged in good condition.

Three months later, she returned after another insulin reaction during which the dog ate the remainder of her great toe to the level of the metatarsophalangeal joint. Primary ray resection was performed and she was released on oral antibiotics.

Approximately one month later she returned after her dog ate the remaining toes on her right foot during another insulin reaction. This necessitated amputations at the level of the metatarsophalangeal joints. After a course of intravenous antibiotics, she was discharged on oral antibiotics. She returned thirteen days later. This time, the dog had eaten the previously intact first, second, third and fourth toes of her left foot. The amputations were completed at the metatarsophalangeal joints and closed loosely.

At last follow-up, one month after her last amputation, her wounds on both feet were healing with minimal drainage. She developed a superficial ulcer on the dorso-medial aspect of her right foot (Figures). Local authorities have removed the dog from her home.

#### DISCUSSION

As many as seventy percent of all lower extremity amputations are performed on diabetic patients<sup>8</sup>. Fortunately, patient education has a dramatic effect. In one series, patients who participated in regular clinic evaluation and education had two-thirds as many hospital admissions for diabetic foot problems as their less compliant counterparts<sup>9</sup>. Even more diabetic foot disease may be prevented or at least delayed.

This case report illustrates the diversity of environmental hazards which threaten the health of the diabetic

patient's feet. This case will hopefully stimulate other reports of uncommon or seldom suspected hazards in order to prevent further such occurrences.

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## IF THE SHOE FITS, . . .

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Although we tend to glorify the exquisite complexity of the hand, it is the lowly foot that is more advanced and specialized, from an evolutionary standpoint. True, the human foot lost its prehensile abilities to grasp and climb sometime before the Australopithecine stage of evolution - this change was an adaptation to forces that took us down from the trees onto terra firma. The loss of a prehensile foot was a small price to pay for a bipedal existence that freed the upper extremity from locomotive duties. The foot, therefore, became more specialized, adapted to upright weightbearing and balance, providing a stable base for our nomadic ancestors. Our feet, however, evolved on the soft ground of the African savannahs, and have not had time to adapt in an evolutionary sense to the asphalt jungle that is our current habitat. To protect our feet, we wear shoes. Unfortunately, our shoes may contribute to the undoing of our feet.

Bunions (hallux valgus) occur almost exclusively in populations that wear shoes. A study in Hong Kong compared the feet of land dwellers (who wore shoes) with their boat dwelling cousins (who did not wear shoes). The incidence of bunions in this ethnically homogenous group was 33% in shod individuals, and only 2% in the unshod. In Japan, bunions were practically unknown until recent times. This change has been attributed to the decline in popularity of the traditional open, wooden clog (geta), and the concomitant rise in popularity of the standard Western closed, leather shoe.

Although there is undoubtedly a genetic predisposition for bunion formation in certain people, this tendency expresses itself more dramatically when the person wears tight, constricting shoes. Even if a person has a relatively large bunion deformity, it is usually painless as long as the person is barefoot. Narrow shoes cause the great toe to deviate outwards. A painful bump then develops on the inside of the metatarsophalangeal joint (the "bunion") from abnormal shoe pressure and irritation. Loose fitting sandals or sneakers are often quite comfortable, even in the presence of a significantly deformed great toe.

The situation is made worse by the popular styling of the shoe industry. Pointed-toe shoes are a good example of faulty footwear favored by our society. The pointed-toe shoe dates back to medieval times. Originally, the pointed toe piece was added onto the front of a regular shoe, and was not meant to contain the foot. However, this gave the foot an elongated appearance, which was eventually con-

sidered unaesthetic for women. The solution to this problem was to taper the front of the shoe to a point, rather than add a false point to the front of a regular shoe. Squeezing the toes into a tapered shoe resulted in the development of numerous problems, including painful corns, calluses, bunions, bunionettes, hammer toes, and ingrown toenails.

High-heeled shoes are another example of faulty footwear worn in deference to the dictums of style. High heels tip the foot into a semi-vertical position, and don't allow the foot to carry out its normal dynamic movement of "pronation" during the heel strike phase of gait. Pronation is one of the mechanisms that help the foot to absorb and lessen the shock of weight bearing. High heels maintain the feet in an anti-pronated or "supinated" position, and thereby cause more stress to be placed on the knee, hip, and back. The end result is a less efficient gait that can be both tiring and painful. High-heeled shoes also redistribute pressure on the front of the foot, causing callosities and pain under the ball of the foot. Additionally, the angled tilt of the shoe shank in high-heeled shoes stretch the ligaments under the ball of the foot and force the toes to slide forward into the tight toe box. The toes are thus compressed into an unnatural position, leading to hammertoe deformities, bunions, corns, calluses and ingrown toenails. It's no wonder that many professional women now wear comfortable running shoes during the daily commute to and from work, and change into more "acceptable" heeled dress shoes in the office.

Certain shoe styles have (gratefully) fallen out of fashion. Platform shoes made a brief appearance in the 60's. Wearing platform shoes altered the normal gait biomechanics, made balance difficult, and led to many falls, sprains, and fractures. Similarly, the negative heel shoe was a misguided attempt to create a natural, healthy shoe. Scientific testing of the negative heel shoe revealed that natural weight bearing was significantly altered causing the toes to constantly dig into the shoe, as if the person was always climbing uphill. Dr. Nathaniel Gould, former president of the American Orthopaedic Foot and Ankle Society, pointed out that the negative heel shoe caused forefoot soreness, and painful overstretching of the calf muscles. Buyers of these shoes quickly learned that their irksome symptoms disappeared as soon as they changed into shoes of a standard heel height, and the negative heel design was spurned into commercial oblivion.

Women have borne the brunt of the pain inflicted by faulty footwear. Why then do they continue to purchase high-heeled, pointy-toed shoes? Why haven't these shoes been deserted like their cousins, the platform and the negative heel shoe? The answer, in part, is related to the strong symbolism mankind has placed on the foot. According to Drs. Monroe Spero and Earl Schwartz, psychiatrists at New York's Mount Sinai School of Medicine, the important psychosexual aspects of the foot and shoe are deeply rooted in human culture. In some ancient cultures, the foot was a symbol of fertility. Women were thought to derive their procreative powers through foot contact with the life-giving earth. Many cultures have associated the foot with sexual and phallic symbolism. The psychological aberration of foot fetishism is perhaps the most extreme example of foot erotization.

The largest, socially approved experience with foot fetishism was Chinese foot binding. This practice started in the tenth century in the Imperial Court of the Southern T'ang Dynasty and lasted almost one thousand years. This custom of strapping the feet of young girls to stunt growth and create the small, soft, and deeply clefted "lotus feet" climaxed during the Ming Dynasty (14th to 17th century). During that period there was intense interest in these deformed feet including a flurry of literary activity exalting

the erotic beauty of bound feet, the popularity of "tiny feet" beauty contests and ubiquitous advertisements of matchmakers promoting women feet first. The shoes themselves became the object of great attention. Brightly embroidered fabrics lavished shoes in everyday use, whereas scarlet colors were reserved for bedroom activity.

The psychological underpinnings of this national foot fetishism have been explained on several levels. From a purely physical standpoint, binding the feet was thought to result in an attractive gait, fleshy thighs and buttocks as well as increased pelvic muscle tone. From a social standpoint, the bound foot was a hallmark of aristocracy and class distinction as only the affluent could afford to have an intentionally crippled, nonproductive member in the family. On another level, the bound foot exemplified the subordinate position of women in that society (Fig. 1).

Under the current Chinese government, the custom of foot binding is illegal. Fortunately, this practice of intentionally hobbling girls for the sake of some perverse erotic imperative is over. However, in certain ways, the Western practice of forcing women's feet into narrow, high-heeled, pointed-toe shoes is similar to the traditional Chinese practice of foot binding.

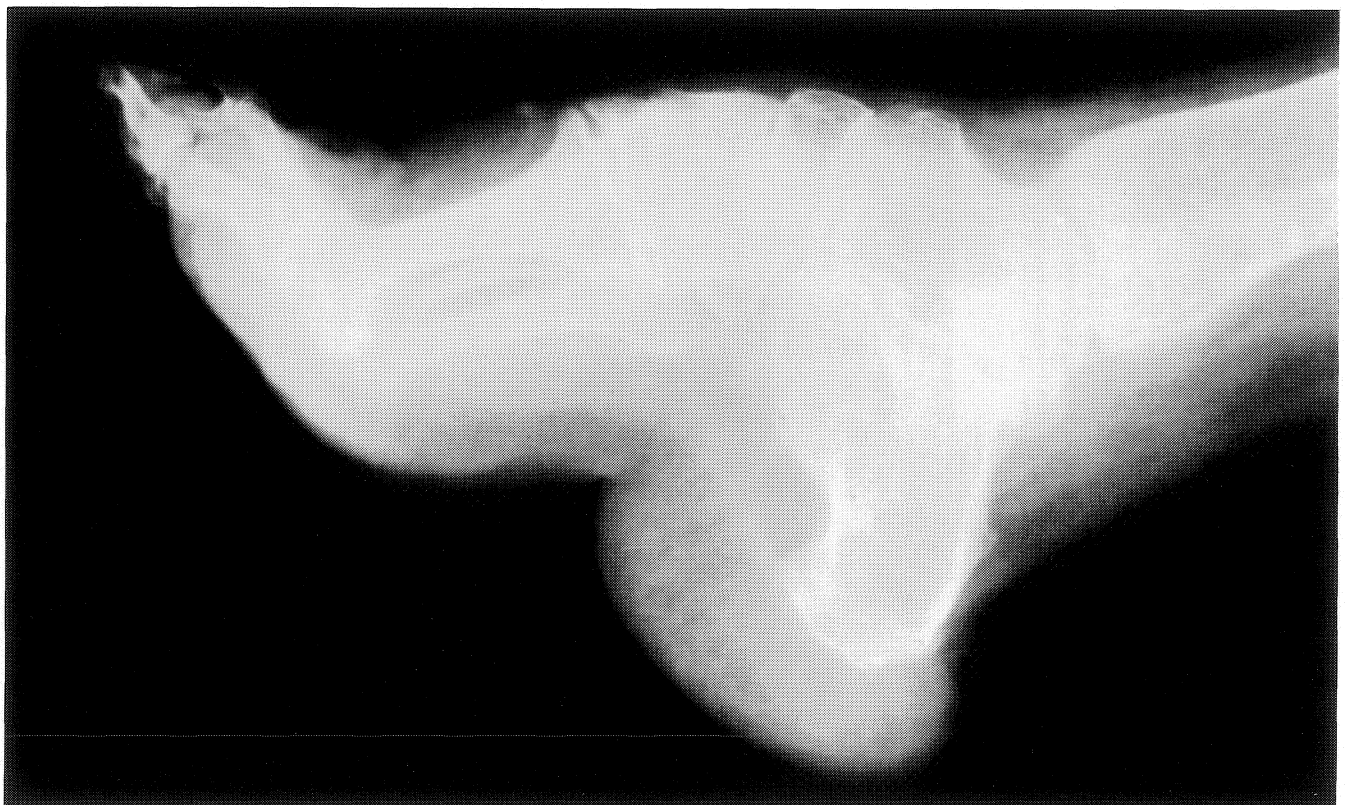


Figure 1.

Lateral foot radiograph of an elderly woman who had her feet bound as a child. This crippling practice has been outlawed in modern Chinese society.

Fashionable footwear for women is seldom functional. Why then do women continue buying heeled or pointed toes? Certainly not for comfort. Moreover, women who force their feet into small and unnatural shoes run the risk of developing painful, and potentially permanent foot deformities. It is indeed unfortunate that so many women feel compelled to sacrifice the health of their feet to be in step with the dictates of the male-dominated fashion industry.

A recent study conducted by the Council on Women's Footwear of the American Orthopaedic Foot and Ankle Society compared shoe and foot width in over 300 women. According to that report, the majority of women . . . "wore shoes that were too small for their feet, had foot pain and deformity, had increased shoe size since age 20 and had not had their feet measured in over 5 years. The women without foot pain or deformity also wore shoes that were smaller than their feet but to a lesser degree." On average, women with foot pain wore shoes that were 1.2 cm (approx. 1/2 inch) narrower than their feet.

Men are also not immune to these problems. Fortunately for men, the styling of their shoes is much less severe than that usually seen in women's shoes. However, the typical businessman's shoe has a tapered forefoot design that pushes the great toe into a bunion producing position, and crimps the lesser toes. Although heavy, hard leather sole wing tips may help the upwardly mobile male executive climb the corporate ladder, they also increase the risk of developing the same forefoot problems epidemic among "civilized" women.

What then, are the attributes of an ideal shoe? First, for the foot's sake, it would be desirable to ignore psychosexual and fashion dicta. The ideal shoe should be made to fit the human foot, rather than forcing the foot to fit the shoe. The ideal shoe should be comfortable, lightweight, and come in a large variety of sizes. The sole should be somewhat flexible and cushioned to absorb shock. The uppers should be made of a soft material that "breathes".

The ideal shoe has laces or straps that allow the shoe to be comfortably attached to the foot. The average foot increases 4% in volume from morning to night; shoe volume thus needs to be somewhat adjustable to accommodate this change.

The American Orthopaedic Foot and Ankle Society, Prescription Footwear Association and National Shoe Retailers Association have developed helpful guidelines for fitting shoes. These include: "1) Sizes vary among shoe brands and styles. Don't select shoes by the size marked inside the shoe. Judge the shoe by how it fits on your foot. 2) Select a shoe that conforms as nearly as possible to the shape of your foot. 3) Have your feet measured regularly. The size of your feet changes as you grow older. 4) Have both feet measured, and fit to the largest foot. 5) Fit at the end of the day when your feet are the largest. 6) Stand during the fitting process and check that there is adequate space between the end of your longest toe and the end of each shoe. 7) Make sure that the ball of your foot fits snugly into the widest part of the shoe. 8) Don't purchase shoes expecting them to "stretch to fit." 9) Your heel should fit comfortably in the shoe with a minimum of slippage. 10) Walk in the shoe to make sure it fits and feels right!"

How can an individual reconcile the perceived need to "look good" (i.e. wearing uncomfortable stylish shoes) with the desire to "feel good" (i.e. wearing comfortable, not-so-stylish shoes). Perhaps certain compromises can be made. For daily footwear, one should strive to wear practical, comfortable shoes that do not constrict the foot. Shoes should be allowed to rest more than just overnight in order to allow the perspiration to evaporate. It is a good idea, therefore, to alternate shoes on a daily basis. For the occasional "night on the town", high heeled shoes probably will not cause permanent deformities or problems. What about commercial availability? It seems that as long as men and women continue to purchase faulty footwear, manufacturers will gladly continue to produce them.

## MEMORIES

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The year is 1993, and I left Iowa City in 1965 to go east of Eden. I'm 62 now, but in my memory it is like yesterday.

Early memories are indelible. My older brother Robert and I had scoliosis. Rob was sent to the great Steindler in about 1939; the whole family went, of course! We stayed in Cedar Rapids with my uncle Bill, the pastor of the Congregational Church. After the initial examination, my folks went home while Rob and I commuted daily to Iowa City on the Crandic—"the vomit comet," for him to receive physical therapy and ultraviolet treatments in the basement of the Children's Hospital. This was supplemented with a handful of calcium tablets and we had to spend time blowing into tubing that transferred water from one gallon jug to another; this was to strengthen his thoracic muscles. I didn't have scoliosis yet, but went along and did everything that he did. Things were a lot less formal in those days! Steindler would visit the gym daily and see everyone. This great man would stop to talk to both of us by name every day. I knew that I had seen God! I'm sure the seed of my future was planted then.

My turn came around 1947, when my family doctor sent me to Dr. Ponseti. I remember a smiling face and a beautiful accent. Years later, I realized that I probably had become the single male with cervicodorsal scoliosis in his series! My dad was a clergyman, wonderful with his flock: he could be a terror when abroad. A Chinese resident was doing my initial workup when he turned to dad and said "You got Ex-Lax?". My dad frowned and said "He's not constipated for God's sake! He's got scoliosis!" After a few more tries, the poor fellow made himself understood and was really asking for X-RAYS!

Back to Steindler. I worshipped the man. When I was in medical school, he gave evening lectures to the orthopaedic residents. I didn't miss these, and still treasure my notes. An even greater treasure is my copy of his book, *Kinesiology*. Annie gave it to me, and he autographed it. Stu McConkey recorded all the lectures on his huge wire recorder. This was in 1955. I had a summer fellowship in the department; I was on cloud nine!

Another Steindler story, from C.N. Hyatt of Corydon, Iowa. He was my mentor for the family practice rotation that summer. When he (Hyatt) was in medical school, circa 1934, Steindler was head of orthopedics and Frank Peterson (Figure 1) was head of general surgery. They used to alternate grand rounds on the two services. One

week Steindler presented a young lady named Sadie and analyzed her orthopaedic problem. He then turned to the group and said "Sooo-vat do ve haf to offer Sadie?" This tickled Peterson, so every time he saw Steindler in the hospital he would say "Sooo-vat do ve haf to offer Sadie?" Steindler would nod, grin, and move on. The day finally came when they saw Sadie's mother on the general surgery grand rounds. Peterson gave a learned discourse on her problems. From then on Steindler would whisper in Peterson's ear "Sooo-vat about Sadie's mother?"

Another Steindler story is second-hand. An acquaintance of mine took his residency at Indiana with Garceau (he of the lateral transfer of the tibialis anticus tendon). Garceau and Steindler were both young academic ortho-



Figure 1  
Frank Peterson—head of General Surgery in Steindler's era.



paedists, a rare commodity in the 1920's. They would visit each other, home and away. Garceau made the comment that Steindler was only a modest technician in the operating room. The secret of his superb results was that he thoroughly studied every case first, and knew exactly what the problem was. The correct operation done with modest skill was better than the most elegant technique performed by uninformed hands!

When I went to Chicago in 1965, I had the privilege of becoming friends with Fred Shapiro, the long-time head of orthopaedics at Cook County Hospital. He was an education in himself, but that is another story! He told me that when Steindler, also Jewish, came to the United States, he weighed a practice in Chicago. He talked to Ridlon, who advised him that "orthopedics in Chicago is not for one of your persuasion". Steindler originally came to Des Moines and then to Iowa City. This particular bit of prejudice enriched us forever!

We lost a treasure with the move out of Children's Hospital. There used to be a letter to Steindler from Walter Jessup, president of the University. Dated 1913, it told Steindler that a new Department of Orthopaedic Surgery had been created at the university. In the letter, Jessup appointed Steindler as acting head until a "qualified individual" could be found. Of course, Steindler turned out to be that "qualified individual" for the next 37 years! Many of us were at his memorial services in 1959, and the sorrow was palpable.

I came to Iowa City in 1949 and got a job as orderly in the University Hospital. The old 40-bed wards with battleship gray paint live on in my memories. I felt I belonged, and this in part was due to the friendly face of Gerhart Hartman, chief administrator of the hospital. He knew everyone, including me, by name. I felt he was a friend. Years later, on his retirement, I wrote and reminded him of this. He responded with a very touching note; the man set the tone for the whole institution and taught me the importance of recognition of others.

I started growing up in my first year at Iowa, working as an orderly on the fourth floor haunts of Reuben Flocks, C-41 and C-42. I worked two full nights a week. Part of my duties included tending the urine jugs under each of the 80 beds. At around 11 P.M., I would empty and wash them, spray each with an incongruous peppermint spray, and replace them. Christmas neared. I had visions of returning to Fort Dodge, my hometown, as "Joe College" (they didn't know I emptied urine jugs!).

The Christmas programs in my dad's church were a part of my life. I mentioned to Barbara Reimer, our charge nurse, that I certainly looked forward to going home. She gave me a blank look and pointed out that I hadn't asked for time off! They were depending upon me! I was crushed, but stayed. On Christmas eve I sat at the nursing

station on the long hall between the wards with Marilyn Gillman, sharing my misery. Then came a sound of voices and running feet from the darkened C-42 ward. I went to the entrance and saw only the lighted Christmas tree at the far end of the ward with vague shapes flitting about the center aisle. Patient rights weren't a big thing in those days, so I just turned on all of the ceiling lights. Jim C. was a tertiary uetetic who just had a TURP. Small little torso, long spindly arms and legs, no teeth and bald, he had pulled out his inflated catheter and was standing atop his bed. He had committed a major social error in his bed, and was throwing pellets of this all over the ward. It was on the floor, walls, ceiling and even in the Christmas tree! The worst was this; he was throwing it at the other patients as well! They were all crouched down beside their beds, like soldiers in foxholes, to avoid his ministrations. First, I had to clean it all up. We had a huge porcelain bathtub in a room at the entrance to the ward, and my final task was to bathe him. I was full of anger, but he was obviously gratified. He splashed, cooed and gave a wide, although toothless, grin. All he needed was a rubber ducky! Suddenly I realized that this was funny as hell, and equally realized that for the first time in my life I had done something worthwhile on Christmas eve! Another of life's lessons well learned . . .

Other fleeting memories of that time; does anyone remember Dr. Yonyama of general surgery? I do, because the good Dr. Y. was once on the operating room schedule to do a "long leg cast." Tell me why I remember that one?

Time finally came for medical school and my introduction to terror. It came in the person of Dr. Jerry Scheldrup of anatomy. I still have my notes from that first day. He had an affected way of speaking, with frequent pauses which were probably designed to conceal a neurologic disorder. His exact first words were "We are going to talk about the axillary pyramid today. You might wonder why it is important to know about this. Well, someday you are going to have a patient with an acute infectious spreading process of the neurovascular lymphatic sheath of the axillary pyramid, and then you will have to know all of this!" This last sentence was rattled off at breakneck speed; before I could write down half of it, he was off and running on the next sentence. I can remember thinking "My God! Maybe I could still be a preacher. . . ."

Another occasion for dismay was an anatomy tag test. The proctor was somewhat befuddled with ethanol and paraldehyde. His main task was to ring a little bell every three or four minutes, telling us to go to the next station. The test was to last around 90 minutes. Well, he rang the bell every 15 to 20 seconds! We were rushing from station to station, grabbing at the next tag to identify the structure. After two or three shifts, most of the tags were torn off, and we finished the test in about 15 minutes. Dr.

Ingram came in and saw what had happened. He graciously told us to go back and look again at anything we had missed. John Fenton was top man in our class. As he retraced his steps, half the class went with him, in desperation. It was like Christ and his disciples; the miracle this time was that they finally discarded the test and gave everyone a "pass"!

Things eventually got better, and I remember some of my patients in the clinical years. One patient with cirrhosis told me he had "the yellow Johnson". Another told me her father died of "sinking chill" and her brother of "creeping palsy".

One of my early victims was a poor fellow named Walter. I was supposed to pass a nasogastric tube to measure free acid. No one supervised us, and I passed most of the long tube into his nose, and drew my samples. When I tried pulling the tube out, it wouldn't come. It had obviously curled upon itself and knotted in his stomach. After repeatedly pulling and passing the tube, it eventually came loose and I was out of trouble! This was the same era that an earnest young surgery resident named Bob Soper told me to quit referring to a patient's "stomach" when I was actually palpating his abdomen. This I remembered, and thanked him for almost forty years later! You probably know that he is the Acting Chairman of General Surgery here at Iowa. This is just another example of the continuity that makes Iowa great.

Another crowd pleaser of those days was Billy Bean, head of the department of Medicine. I'll never forget the agony he suffered. He had several trustee convicts on the metabolic study ward for several months on a rigid diet, supplemented with pantothenic acid. He did daily laboratory studies on each of them. Only when he was done did he find that they had been going out each night to suffer the pleasures of the flesh at the Airliner downtown! At least he got a good story out of it!

Dave Funk ran one of the medical wards at the Veteran's Hospital when I rotated there. He was a superb clinician, but sometimes he outdid himself. One day on rounds, he asked me for some of the symptoms of hypertension. I got it wrong, and mentioned "scintillating scotophobia" when "scintillating scotomata" was the correct response. I actually sort of enjoyed the attention when he corrected me. The denouement was later; we went into an elderly male patient's room. Dave stopped and dramatically pointed at the patient, saying "Just how do I know that this patient has renal failure?" None of us had a clue. Dave said "Look at that granular white ring around his lips! That's uremic frost!" The patient grabbed his sleeve and whispered "Doc, doc- I just had cream of wheat for breakfast!" Dave laughed right along with us, and was a great teacher.

Wilbur Miller was head of Psychiatry, and their hospital was right next to Children's Hospital. One of their stories should live on. One of their patients managed to get an outside line, and called Buildings and Grounds to place an order. Wilbur considered it very thoughtful that they came and placed purple martin houses all around the grounds of the hospital. When he got the bill, he finally figured out what had happened. One of the psych classes of our era had a patient appear before them with the particular disorder of always speaking in verse. She claimed transient fame by telling them "I like doctors, I like M.D.'s, I like to meet them face to face, and knees to knees!" Nowadays she would probably be a featured "rapper"!

John Randall was head of Obstetrics and Gynecology. He once said that medical students were like trained rats; you repeated something to them often enough, and they would remember. He was absolutely right! On one examination, better than half of the class didn't know the length of the average umbilical cord. He spent an entire hour telling us the variations. Forty years later, I can tell you that the average is 55 centimeters. If it is shorter than 35 centimeters, it is short. If it is longer than 75 centimeters, it is long! As Curt Vonnegut would say "And so it goes!"

Ray Bunge (Figure 2) was one of my favorite professors. He had a fantastic sense of humor and could zing you without you ever realizing you'd been had. He wanted to teach us to understand the things we so glibly wrote down in our histories and physicals. In workups, he insisted you write all the specifics, such as blood pressure 125/80 millimeters of mercury at 982 feet above sea level (Iowa City), ward temperature 68 degrees Fahrenheit. Red blood cell count, 4.5 million cells per microliter (again mentioning elevation and temperature). When you got that specific with everything, your handwritten history could run thirty to forty pages long. A typical off the wall example was with Hal Jaffe. He wrote that a urology patient had "average-sized testicles." It came back with the phrase, circled in red, "What is average?" Jaffe rewrote the page, stating that they "were the size of a walnut." Back came the paper, "English or California walnuts?" Jaffe got into the spirit of the thing and wrote, "They compare favorably with my own!" The final response was, "I'll accept that. You have a portable standard of comparison which is always with you!" One of his early research interests was fertility, and he pioneered the preservation of sperm by freezing. He once gave us a lecture on the subject. The introductory slide had pictures of a group of sailors at the rail of their ship, icicles hanging from their beards. The title above it was "Frozen Semen". Dr. Bunge still lives in Iowa City, and I recently visited him. Trust me; he hasn't changed! I treasure people like him.



Figure 2

Raymond Bunge—Urology professor of unlimited imagination!

I also made some new friends. Reg Cooper was the intern and I the senior medical student on the medical ward C-31 in the fall of 1955. Our resident was Bob Sedlacek; I knew his little brother Gordon from college days. Tuberculosis was a big thing in those days; patients took the rest cure at Oakdale sanatorium when diagnosed. I remember from Bacteriology that *Mycobacterium smegmatis* was morphologically indistinguishable from *Mycobacterium tuberculosis*. Reg and I got a sample from one of the many unwashed male patients on our ward and showed the microscopic slide to Bob, telling him this was from a patient on the open ward. Before we told him the truth, I thought he was going to die! After we told him, I thought we were going to die! If you figure out the years, this means that, man and boy, Reggie has been part of the Iowa scene for over thirty-eight years! Not bad!

Now for an Al Meyer story. I was medical student on C-54, and Al was in his general surgery year of orthopaedics. One of his patients was a retarded young man with a fractured femur. As was our wont in those days, he was being treated with bedrest and tibial traction. Al would



Figure 3

Sid Ziffren—Professor of Surgery—a character for all seasons.

approach the bed, and the patient would repeatedly utter his only words, "Mamma, Mamma". This being an early contact with orthopaedics for Al, he adjusted the traction daily (dare I say hourly?) and got daily roentgenograms for position and callus. Then, Sid Ziffren (our beloved "Zippy") (Figure 3) came on rounds. He saw the thick x-ray jacket and said (loudly) "Well, you've probably fried his orchids! What are you going to do next, get a sperm count?" I felt the need to laugh aloud, but didn't. Al was frowning and Al was bigger than I. I'm not stupid. I graduated from medical school!

Jody Buckwalter's dad, Joe (Figure 4) was one of our professors in general surgery. He had four of us in the old emergency room at the east end of the general hospital. Hal Jaffe whispered something to Judy Hopkins as Joe started to talk. The irascible little old lady we were examining said, "He's teaching you, so be quiet and listen!" He showed us how to put an Unna paste boot on her. He would wrap a layer of gauze, and use a paint brush to layer on oleaginous paste from the bucket on the floor. By the time we each rolled a layer, it was pretty thick! As



Figure 4

Joe Buckwalter—Professor of Surgery, mentor, and father of Jody!

we finished, Joe gave us a prefatory smile; at times I couldn't tell if it was really a smile or a pained frown. He left as we were cleaning her up to take back to the floor. The little old lady said "That Dr. Walters is a deal of a nice fellow!" I looked at her blankly and said "Walters?" She said "You know, that Doctor Bud Walters!"

Another Buckwalter story; One of my best friends was John LaMar. He had an innocent quality about him. He could take adversity without complaint, and even laugh about it. This particular time, we had surgery orals in the late winter of our junior year. John was late for his orals. Dressed in his best suit, he ran out of the front door of the Phi Beta house and slipped on some ice on the steep front steps (Figure 5). He landed on his behind on the muddy sidewalk, totally filthy. Without time to change, he went on to the surgery offices. Dirty and abashed, he sat down in the presence of Sid Ziffren, Mike Bonfiglio and Joe Buckwalter. Without any religious prelude, they crucified him. Afterward they closed the door. John being John, he paused to listen through the door. Joe Buckwalter opened the discussion with "I don't think he's stupid!" Mike broke



Figure 5

The infamous front steps of the Phi Beta house; bad news in the winter!

in "Well, he surely isn't bright!" John left. He told me, and we both roared with laughter. He always had the knack of making me feel better, no matter the situation.

Although I knew Joe Buckwalter wasn't God, I knew he was an acceptable substitute. This is because he had spent some time with Sir Reginald Watson-Jones. I pumped him for all of the details, and eventually sort-of felt I knew Sir Reginald as well. I had many idols in those days!

One of my first patients from that era had splenic neutropenia. I was probably the tenth person to do a complete workup on her and I asked her what her problem was. She said "Well, sonny, for one thing I ain't been feeling too swift lately. For another thing, I ain't got enough of them white fighters!"

That was my first contact with public perception and hierarchy. All of us got asked, "Are you one of the regular doctors, or just an intern?" A great symbol of all of this was our garb. As freshmen and sophomores, we got to wear white lab jackets. In the junior year, we felt more important immediately as we added white trousers! The absolute top of the ladder was for interns and residents, who got to wear a beautiful white tunic, with the buttons on the side of the neck. I went through all of these steps at Iowa, and then went to the University of Illinois as an attending. I looked forward to wearing the treasured long white coat of the attending man. Their hierarchy was different. My first day on the job, an old guy came to sweep up the clinic. He was wearing the long white coat! I went to Marshall Fields and bought a new suit.

I can remember another thing from my medical school days. The orthopedic staff would often come in the rear door of the Children's Hospital at the same time; Carroll B., Pons, Mike and Bob Newman. Mike always used to wear a slouch hat, ala Indiana Jones. They were an impressive crew, and another set of my idols! Later, Adrian Flatt came and won my allegiance forever. He had

an elfin sense of humor. Years after my residency when I was in town I would leave notes signed "The Shadow". Just as promptly, I would get a note in the mail signed "The Green Hornet"! During our osteology course he told us wild stories from his medical school day; my favorite was having a mouthful of carpal bones, and naming them as he spit them out. I'm not sure if it was true, but of course it has to be!

Reg will remember some of the following stories. Carrol B. (Figure 6) used to get problem hip cases from all over the nation. On one occasion, we operated on a lady from Alaska with achondroplasia who had severe arthritis of the hip. This was complicated by ochronosis. The cartilage of her hip joint was black as coal when he did her cup arthroplasty. On another occasion, a wealthy lady from the east coast came to Iowa City for hip surgery. She complained of feeling "bloated" on the preoperative night, so they contacted the resident on call. He probably had been a farm boy in his own country, because he knew just what to do. He went to operating room, got a biopsy

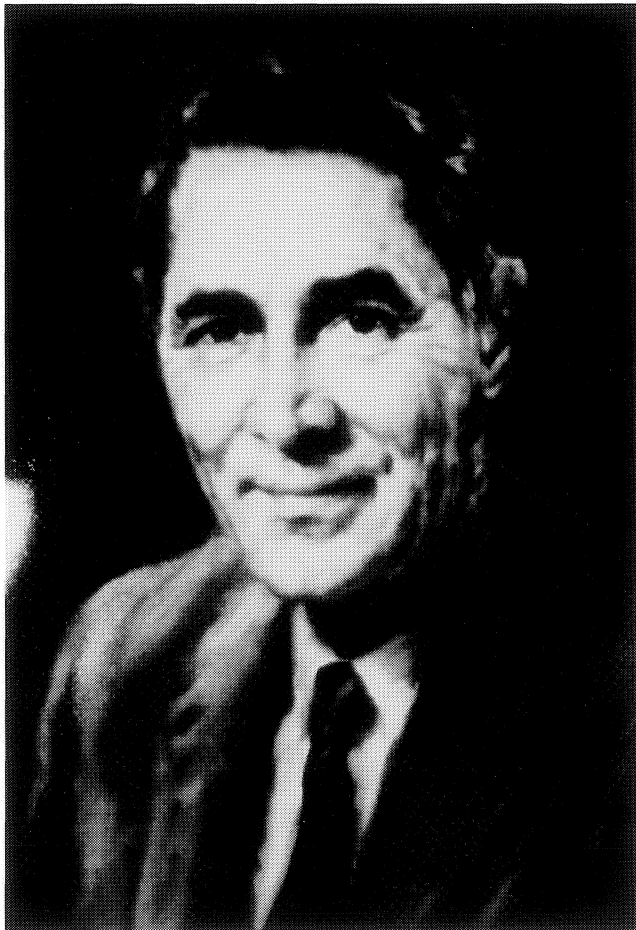


Figure 6

Carroll B.—head of Orthopedic Surgery—our icon.

trephine, and trochared her! I doubt she had the fortitude to complain about anything else that night. She did well after her surgery.

You older types will recall the venue of our endeavors; the Children's Hospital, and that little stage where we were occasionally known to suffer! Real old-time stalwarts will recall some of our euphemisms. We often had overweight patients, and our buzz-word was "The Iowa Unit", which was 100 pounds. We would casually refer to a patient as being three Iowa units, and so forth. I was about one and one-half Iowa units in that era, so it worked for me. With the completion of my residency, I climbed to two and one-half units at one time. That is when I conveniently forgot the unit of measure (until now, in this time of remembrance!). It is alright, because one of the famous blonde playboy pinups of an earlier era called the drastic expansion of her pulchritude in middle age "development!" I now use a treadmill to control my development.

Carl Gillies, our orthopedic radiologist, became an old friend during our resident years. He interviewed me for medical school, and I was surprised to meet him again 10 years later as I began residency. Our orthopedic radiologist, he used to give us covert grades on our care of patients with hip fractures. An "A" was "examination shows excellent reduction of the fracture and placement of the Smith-Petersen nail" Lower on the feeding chain was "examination shows a fracture of the femoral neck; position is somewhat improved and a nail has been placed." On one occasion, the resident thought he had an intertrochanteric fracture and placed a nail and sideplate. The report read, "The greater trochanter has been avulsed. The femoral neck and shaft are intact. A nail and sideplate have been placed." He gave us excellent lectures on the classification and radiographic analysis of ankle fractures. In later years during a visit to Iowa City I brought him the latest edition of Bonnin's "Injuries to the Ankle", which he subsequently placed in the Radiology Department library, where a picture of him hangs. He was a gentleman, and I still smile when I think of him.

Gordon McCreedy was usually unflappable during residency. I'll always remember him telling me to keep my elbows at my side while working in the operating room unless I was getting ready to take off a liaison with the flying nun! Well, even he had his breaking point. This was in the early 1960's. The Academy, in conjunction with the Office of Research in Medical Education at the University of Illinois, gave all orthopedic residents a little quiz to decide what the determinants of an orthopedic surgeon really were; what "made them tick". It was called the Runner test. Gordie came to one of the questions, choked and broke up. He literally couldn't stifle himself for the rest of the test. He gave everyone the number of the question, which innocently read, "Do you like the feel of a good tool

in your hand?" I reminded him of this about five years ago, and he broke up all over again!

Do you remember the great Christmas parties that Carroll B. used to give? At least a couple of times, Adrian Flatt brought along Sir Reginald Watson-Jones as a guest. Sir Reginald would partake of the liquid refreshment. When his inhibitions were sufficiently suppressed, he would insist on playing the piano, with we colonials singing Christmas carols. His playing was atrocious, but that made it all the better! My wife give me his fracture books for Christmas when I was interning, and he later autographed them for me. Go back a little deeper in your memory and recall the fireplace in the front room. Carroll B. had a cubenstuhl sitting here. It is a piece of furniture peculiar to Norsemen. They still make them in Decorah, Iowa. They square up the proximal three feet or so of the large tree trunk and hollow a seat out of it. They often shave off the bark and do elaborate carving on the sides; this is the type he had. He inherited it from the famous Marius Nygard Smith-Petersen of Boston (you can still see a painting of "Smith-Pete" in the orthopedic conference room at M.G.H.) I sat on that darned stool, and hope you did also. It makes for a great story!

Another resident, who shall remain nameless, was an early Carroll B. resident. Carroll B. encouraged appreciation of the humanities and craftsmanship in his residents. This orthopedist was pursuing the muse in the art department, and inadvertently sliced off one of his distal phalanges. Mike Bonfiglio repaired the damage, leaving him a painless and functional finger. He avows that this blunted finger is great for percussion of the chest, and even better to show patients with similar injuries. He shall remain nameless because he is a lot bigger than I am. He lives in Des Moines, and you can surely figure it out from here!

For a while, Carroll B. also drove a huge, forest green, four-door Mercedes Benz convertible. This was big-time for Iowa City! Of course, we all knew that our chief was big time. When he later became president of the Academy, I knew this with a certainty.

Many of us will remember the little old lady who took care of the rabbits in the experimental pathology laboratories. If she knew the rabbits were to be used the next day, she would often hide them in other rooms. Her little idiosyncrasy was transportation. She hated walking up Newton Road to the path labs. If you were stopped at the bottom of the hill with your turning signal blinking, she would bashfully slip into the passenger seat and say, "Thanks!" We all came to look for her after the surprise of the first few times. Gordon McCreedy, usually rather taciturn, loved to tell people about this (almost more than I did!).

This was about the time Bob Hickey (from General Surgery) addressed a continuing education seminar on

G.I. bleeding that was being held for general practitioners. He concluded that care of G.I. bleeding was a team effort involving the general surgeon, the internist and a good nursing staff!

I remember clearly my time at Massachusetts General Hospital in 1961 and 1962. I had been there about six weeks and Dr. Ponseti came to town. It was in the late summer, and many of the staff were on vacation at the Cape. I took him on a grand tour, and then noon came. We all sat down to lunch around the table with Joe Barr, the head of orthopedics then. He, with Mixter, wrote the first major paper on the herniated lumbar disc. Also there were Bill Harris and Mel Glimcher. We exchanged a few pleasantries, and then Pons leaned forward and earnestly said "I've looked around. My goodness, when are you going to start doing some work?" Three heads instantly snapped in my direction. A nice smile was my only defense!

Everyone of my era remembers Shorty Paul and his research on Bufferin. They wrote this up recently in the alumni journal. I still have an antecubital fossa riddled with needle marks from that project. Ten dollars a time did help, and it bought the battery charger I used to keep my ancient car going! Even more memorable were his secretaries, Gretch and Dianne. Now there were two to remember! Some of you will recall that they were the stars of my home movies on human walking in 1962. Dianne wore green silk stockings and Gretch was a typical Grinnell graduate in black. I got shots from angles, as they walked as suggestively as was possible in the 1960's. As I gave that seminar, Carroll B. gave me a "What is this?" glance which I well remember. What is life if you can't astound someone? I didn't try it again!

The more ancient types among us will recall Shorty's clinical prowess. If it was there, he could percuss or palpate it! Some flaky types once invited him to the anatomy labs to percuss the chest of a cadaver who "had a major cardiac abnormality". What they didn't tell him was that the abnormality was that they had exercised the heart through the hole in the posterior chest wall! Their victory was somewhat hollow; they forgot they weren't graduates yet. Shorty had ways of making people regret transgressions.

Do you remember the basement ramp in Children's Hospital? What did you do on late evenings when you were bored? I tried this several times. You could get a caster-wheeled office chair from the recovery room, which was at the top of the stairs. You could then launch yourself down the ramp and use your arms to carom off the wall at the bend in the ramp. It was exhilarating and I never did break anything.

Some memories are so delicious that I grin from ear-to-ear recalling them. Three memories from my med-

ical school days are about the same orthopedic resident, who shall remain nameless. You all remember the old brace shop in the basement of the Children's Hospital. For your own education, they would personally fit you with any of their devices so that you could have a better appreciation of their value; I got a pair of insoles myself. This resident also availed himself of their services. Later, he was handling the slide projector for Mike Bonfiglio's lecture on low back pain to our junior class. Mike pulled out a table in the front of the room and motioned for the resident to come down, so that low back examination could be demonstrated upon him. The resident demurred. Mike frowned and motioned again, whereupon the resident reclined on the table. Mike was a little angry with him, and abruptly demonstrated straight-leg raising. The resident's body elevated as a unit, and he was literally resting on the back of his neck. A look of consternation crossed Mike's face as the resident hissed into his ear, "I've-got-on-a-low-back-brace! That's what I was trying to tell you!" Mike said, "Do you have a backache?" The resident answered,

"Well, no." Almost silently Mike said, "Well, for Christ-sakes!" and went on with the lecture.

Dr. Ponseti was usually unflappable, but this same resident touched even him one day. He had read of a new anterior approach to the knee. You made a midline anterior approach to the knee, and then split the patella. It was a simple, yet elegant approach guaranteed to give access to anything and everything! Pons said "Oh my goodness, no! Don't you do it!" Well, the resident thought Pons was out of town, so he went ahead and did it. The silence was deafening and absolute when Pons, who was in town, came into the room and saw what had happened.

A final vignette on the same individual. Many of you will remember our joint venture from medical school days. Published yearly, it was an underground newspaper called "Biopsy." It could be clever, but mostly it wasn't!. Its anonymous specialty was not in awarding Golden Apples to professors, but rather in awarding hypothetical buckets of merde to unpopular professor! While still a student, the



Figure 7

Robert Bonfiglio hits the front page (The New Orleans Times Picayune, I think).

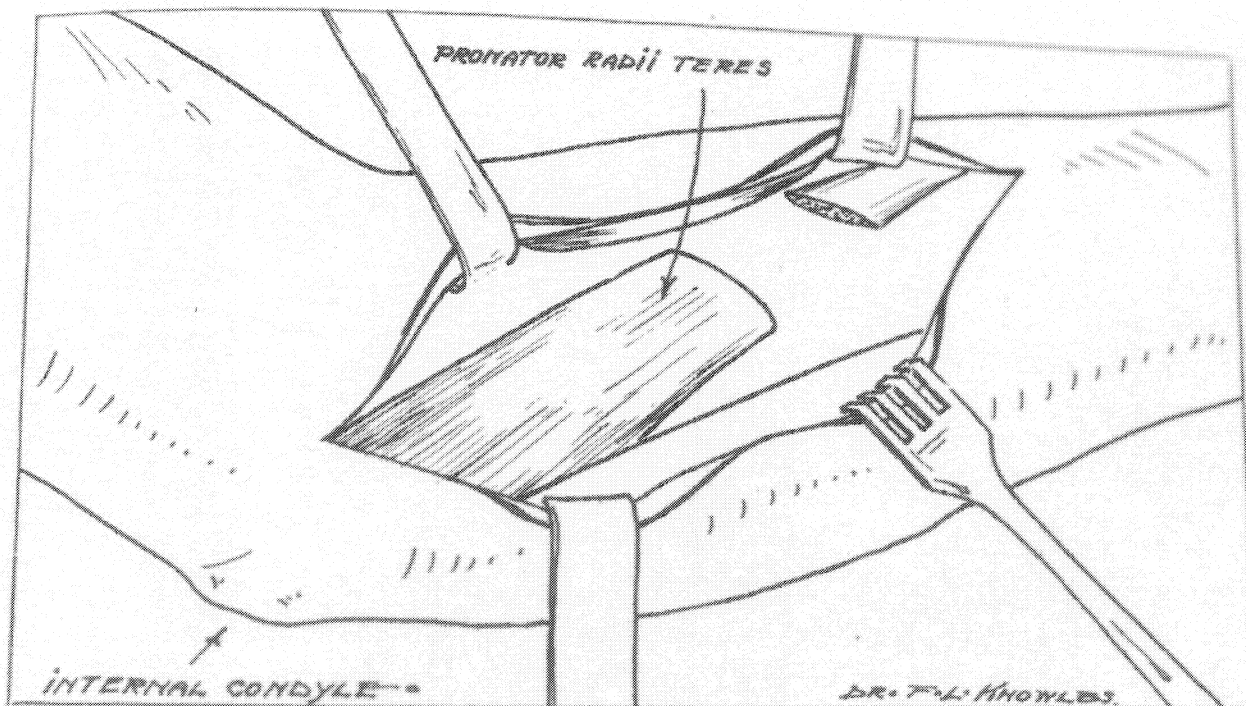
future orthopedist went into the Iowa Hall of Fame as he unanimously won the bucket of the year from this classmates!

Another story from this era tickles me to the marrow (a good orthopedic allusion). Mike Bonfiglio's son Robert was a little boy, and he felt they had disciplined him unfairly. He and a friend ran away from home. They found the two boys hours later sitting in a field next to a campfire. They had taken a supply of hot dogs and marshmallows with them, and brought a toy bow and arrow, so that they might live off the land! I remember all this vividly years later when Annie and I were having supper with the Bonfiglios during an annual academy meeting in New Orleans. The local newspaper (Figure 7) headlined a story about the coming concert by the famous harmonica virtuoso, Robert Bonfiglio! A year or so later, a network special on television featured Robert. I called Mike long distance afterwards, just as excited as he was!

Life can be dull at times, so I often like to incite people. In 1962, Howard Hogshead and I were on the same service. We had a lady patient, Lupe, for shoulder surgery. On rounds one day, I asked Lupe how you said "Hogshead" in Spanish. She brightened, and eager to help said "Cabeza de puerco!" I call him that for the rest of the rotation. She liked him, and affectionately referred to him as "Cabeza".

Another story is too good not to tell you, but not so good that I'll tell you who it is about! During medical school days, one future orthopedist was an avid fan of Dr. Alvarez of the Mayo Clinic. Alvarez wrote a folksy column about common sense in everyday medicine. He wrote one column about peristalsis, Valsalva's maneuver, and hemorrhoids. He claimed that hemorrhoids were caused by people in a rush performing Valsalva's maneuver, which increased intraabdominal pressure and dilated the hemorrhoidal veins. He recommended taking a small glass of warm water to the throne, taking small sips of water and awaiting the imperative of nature. The water would stimulate waves of peristalsis the length of the gastrointestinal tract, and preserve the integrity of one's natural attributes. I told him that if he was a perfect—as we grew older, it would be proof that it worked. I doubt he will ever tell me, one way or the other because I might write about it!

I've always enjoyed teaching, and have graduated about 90 orthopedists from our program in Albany. We had about another 40 during my days at the University of Illinois in Chicago. We've always had fun bugging each other about minutae. Remember, I grew up in Fort Dodge, and here is one of my favorites. Fred Knowles was one of Steindler's early residents. He did many of the drawings in Steindler's early books. This (Figure 8) is an example of his artwork



2

Figure 8

Fred Knowles included medical illustration as one of his duties as a Steindler resident.



from "Reconstructive Surgery of the Upper Extremity" which Steindler published in 1923. I am sad to report that I was the first to check this book out of the Albany Medical College library. It was seventy years late, but has now been done! I still often use Knowles pins for undisplaced fractures of the neck of the femur. When I do use Knowles pins, the following dialogue is standard with my residents.

Q. "Who was Knowles?"

A. "Fred Knowles, inventor of this pin."

Q. "Where did he practice?"

A. "Fort Dodge, Iowa."

Q. "He had two beautiful daughters. What were their names?"

A. "Nancy and Joanne."

Q. "Where did Nancy go for Easter vacation in 1947?"

A. "New Orleans"

Q. "At what famous restaurant did she eat?"

A. "Antoines."

Q. "What did she have for dessert?"

A. "Baked Alaska."

I know all of this stuff because Nancy reported her adventure to our French class in high school. As you can see, I do remember things! I always figure by the time we finish they will remember Knowles, and they do, all of them! It may not be fair, but I'm not the resident anymore. It is my turn now. They all claim they understand, and I'm afraid they just might.

Many wonderful people have been with our group at Iowa. We really ought to try to remember them. A few

years ago, we wrote a paper for the Iowa Orthopaedic Journal about Fred Hark, whom I came to know well in Chicago. He was one of Steindler's first residents. The Hark procedure is still used. My best description of him is that he was like an old Iowa farmer; pragmatic and did what was necessary to do the job without a lot of frills and fanfare. He was really a lovely man, and made me proud to have come from the same place. Many others come to mind. We really shouldn't allow precious memories to fade.

When I look at the size and depth of the department that Reg and his staff have developed, I'm simply staggered. When I was here, there were five attending staff and twelve residents. One of the highlights of his career was in his presidency of the Academy, but some other sidelights really tickle me as well. Before you leave, go and look on the walls of his office. His history is there, from grade school triumphs on. As I said earlier, I most enjoy knowing people who are originals; he's definitely been that for all of the thirty-eight years I have known him.

Memories can live on, and they enrich us all. Each of us lived through a different era at Iowa. The present one is right there at the top. To steal from Voltaire, "This is the best of all possible worlds!"

Editors' Note: The above is a presentation given by Dr. Jacobs at the 1993 University of Iowa Orthopaedics Alumni Dinner. Dr. Jacobs is an alumnus and frequent guest of the Department.

## ALUMNI AND FACULTY LIST

The following list was generated from responses to a recent mailing, the Iowa Foundation database, and a review of our current Alumni list. In an effort to keep this list up to date and complete, we would appreciate notification of needed changes and additions.

Dr. John F. Abele  
1614 Northeast Hogan Drive  
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Dr. R. Marshall Ackerman  
9715 Medical Center Drive #415  
Rockville, MD 20850

Resident: 1966 - 1969

Dr. Brian D. Adams  
Univ. of Iowa Hospitals & Clinics  
Dept. of Orthopaedic Surgery  
Iowa City, IA 52242

Resident: 1984 - 1987

Staff: 1993 - Present

Dr. Cemil M. Adli  
13108 Sherwood Dr.  
Shawnee Mission, KS 66209-3428

Dr. Seymour M. Albert  
5401 Old York Road #200  
Philadelphia, PA 19141

Resident: 1940 - 1943

Dr. John P. Albright  
Univ of Iowa Hospitals & Clinics  
Dept. of Orthopaedic Surgery  
Iowa City, IA 52242

Staff: 1971 - Present

Dr. Fernando P. Aleu  
70 East 55 Street  
New York, NY 10022

Fellow: 1954 - 1955

Dr. James D. Alway  
Orthopaedic Surgeons  
1415 N 7th Ave  
Phoenix, AZ 85007

Resident: 1954-1957

Dr. Leon J. Armalavage (Retired)  
509 Neopolitan Way  
Naples, FL 33940

Fellow: 1945 - 1946

Dr. J. Garcia Arosemena  
Clinica San Fernando  
P.O. Box 461  
Panama, 9A  
Panama, CENTRAL AMERICA

Resident: 1953 - 1955

Dr. Dennis R. Assenmacher  
1050 Isaac St Dr. #122  
Oregon, OH 43616-3207

Resident: 1970 - 1974

Dr. William A. Baird (Retired)  
3911 Stone Brooke Circle  
Ames, IA 50010

Resident: 1948 - 1951

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(Retired)  
6420 Hills Drive  
Bloomfield Hills, MI 48301

Resident: 1954 - 1957

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Resident: 1953 - 1956

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La Jolla, CA 92037-7821

Dr. Jorge M. Basora  
Orthopaedic Surgical Ctr. Hosp.  
Universite De Sherbrooke  
Sherbrooke, Quebec  
CANADA

Dr. Robert L. Bass  
Cincinnati Hand Surgery  
Consultants  
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Cincinnati, OH 45208

Resident: 1988 - 1993

Dr. George S. Bassett  
Childrens Hosp. of Los Angeles  
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Resident: 1976 - 1981

Dr. Jerry R. Becker  
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Resident: 1962 - 1966

Dr. Lawrence Bell  
173 Evergreen Tr  
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Fellow: 1983 - 1984

Dr. Richard A. Berger  
Mayo Clinic, Orthopaedic Surgery  
200 First Street SW  
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Resident: 1984 - 1989

Dr. Earl Y. Bickel  
Iowa Medical Clinic  
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Cedar Rapids IA 52401-2112

Resident: 1958 - 1961

Dr. J. Sybil Biermann  
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Ann Arbor, MI 48109-0328

Resident: 1987 - 1992

*Alumni and Faculty List*

Dr. John E. Bishop  
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Resident: 1969 - 1972

Dr. Arcelino C.M. Bitar  
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Rio de Janeiro  
RJ 22260-090, BRAZIL  
Fellow: 1944 - 1945

Dr. Donald W. Blair  
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Resident: 1948 - 1951

Dr. William F. Blair  
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Staff: 1980 - Present

Dr. Jamie Isidoro Blumenfeld  
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Buenos Aires, ARGENTINA 1425

Dr. William G. Boettcher  
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Resident: 1975 - 1980

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Mr. Gabriel Reyes Zumeta  
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## Department of Orthopaedics 1993-94

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- Second Row (L to R): Kenneth J. Noonan, Kathi F. Teixeira, J. Lawrence Marsh, L. Gene Thornburg, James C. Krieg, W. Tim Ballard, Robert A. Hart, Gregory P. Guyton, Steven D. Meletiou, Thomas Fyda, Laura J. Prokuski, Ernest M. Found, Jr.
- Third Row (L to R): John J. Callaghan, Kary R. Schulte, Jay C. Jansen, Todd M. Williams, Jon D. Hop, James N. Weinstein, Randy G. Delcore, Margaret Fehrle, Joseph A. Buckwalter, Kristy L. Weber, David S. Tearse, Curtis M. Steyers, Jr., Frederick R. Dietz, George J. Emodi
- Not Pictured: Susan K. Bonar, Mark G. Creighton, Steven Madey, James S. Martin, Michael P. Pyevich, Richard A. Brand, Ignacio V. Ponseti, Lenita R. Williamson