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INSTRUCTIONS TO AUTHORS

Any article relevant to orthopaedic surgery, orthopaedic science or the teaching of either will be considered by *The Iowa Orthopaedic Journal* for publication. Articles will be enthusiastically received from alumni, visitors to the department, members of the Iowa Orthopaedic Society, residents and friends of The University of Iowa Department of Orthopaedics. The journal is published annually in May or June. The deadline for receipt of articles for the 1998 journal is February 1, 1998.

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

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

1. The **original manuscript complete with illustrations**. The corresponding author must be clearly identified with address and telephone number. Manuscripts of accepted articles will not be returned.
2. A **bibliography**, alphabetical and double-spaced, or references made in text only. Refer to bibliographies in *The Journal of Bone and Joint Surgery* and follow the style exactly.
3. **Legends** for all illustrations submitted, listed in order and typed double-spaced.

4. Illustrations

- a. *Black-and-white glossy prints* of photographs. Give *magnification* of photographs.
- b. *Original* drawings or charts.
- c. Color illustrations cannot be used unless, in the opinion of the journal, they convey information not available in a black-and-white print. If color is desired, please send both color and black-and-white prints.

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

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

EDITORS' NOTE



It is a privilege to bring you this, the seventeenth edition of *The Iowa Orthopaedic Journal*. Once again, our mission is the education of our readers in hopes of providing better patient care. From its inception, this journal has provided a forum somewhat different from other periodicals; providing a wide variety of articles including those of historical and “philosophical” interest, clinical and basic science studies, general review manuscripts, and case reports. We have worked hard to carry on this exceptional tradition and hope all will find something in the following pages that sparks interest.

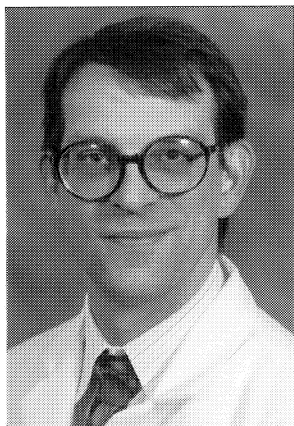
This year, Dr. Stuart Weinstein joins the elite group of physicians to whom this journal has been dedicated. Each edition of *The Journal* has honored one person who has been instrumental in the advancement of orthopaedics at the University of Iowa. Dr. Weinstein continues this tradition and epitomizes what we, as residents, hope to become. He takes a very scientific approach to the practice and teaching of orthopaedics, yet a very personalized approach to the treatment of each of his patients. It is this type of reputation that has brought us here to the University of Iowa to learn the fundamentals of orthopaedics. Dr. Weinstein has not let us down.

The assembly of the “finished product” involves many steps. The solicitation of articles and advertisements, editing of manuscripts, and organization of the distribution is the responsibility of the residents. However, we could not have completed this project without the help of many others. We are indebted to the scientific and financial contributors who have made this publication possible. We thank our faculty advisers, Drs. Reginald Cooper and Joseph Buckwalter, for their guidance. We also acknowledge the administrative assistance of Mr. Paul Etre and the secretarial skills of Ms. Laura Cole.

We hope that you will benefit from our efforts, and we welcome your response and criticism.

James S. Martin
Todd M. Williams

1997 GRADUATING SENIOR RESIDENTS



Robert A. Hart, M.D.

Bob was born and raised in Iowa. He graduated from the University of Iowa in 1984 with a B.A. in Mathematics. He subsequently obtained a M.A. in Biomechanics from the University of California, Berkeley in 1988 and received his M.D. from the University of California, San Diego in 1992. He and his wife Ecco have a daughter Anne, age

seven. Bob will be a fellow in Spine and Spinal Cord Injury at Case-Western University in Cleveland.



Laura J. Prokuski, M.D.

Laura was raised in Iowa, and considers Iowa City her home. She attended Coe College in Cedar Rapids, graduating with a B.A. in chemistry. She returned to Iowa City to attend the University of Iowa College of Medicine. Next year she will be moving to Baltimore, Maryland with her husband Pete, and will be an orthopaedic trauma fel-

low at R. Adams Cowley Shock Trauma Center.



Jon D. Hop, M.D.

Jon was born in Zeeland, Michigan and raised in Midland, Michigan. He attended Hope College in Holland, Michigan where he received a B.S. in biology. After college he went to the University of Michigan where he received his M.D. degree in 1992. Next year, Jon, his wife Char and their daughter Zoë will move to Oxford, England

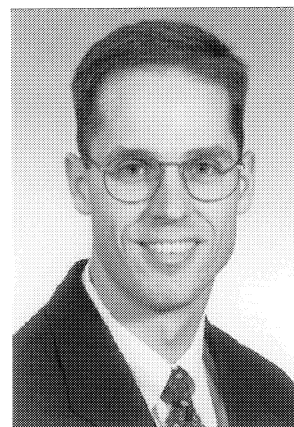
where he will be doing a total joint fellowship.



Andrea M. Saterbak, M.D.

Andrea grew up in Stillwater, Minnesota. As a competitive alpine ski racer, she deferred college for a few years to compete nationally and internationally. She attended the University of Wyoming, where she received a B.S. in biochemistry in 1988. After college, she attended Creighton University and received her M.D. in 1992.

Upon completing her orthopaedic residency, Andrea will return to the Rocky Mountains for a sports medicine fellowship at the Steadman-Hawkins Clinic in Vail, Colorado.



Jay C. Jansen, M.D.

Jay was born in South Bend, Indiana and grew up in Carmel, Indiana with his parents and sister. He attended Indiana University for both his undergraduate and medical education, receiving a B.A. in chemistry in 1988 and a M.D. in 1992. Next year, Jay and his wife Laura will be moving to Birmingham, Alabama to complete a sports medicine fel-

lowship at the American Sports Medicine Institute.

BONFIGLIO EDUCATIONAL ENDOWMENT FUND



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

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

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

Address:

Bonfiglio Educational Endowment Fund
The University of Iowa Hospitals and Clinics
Department of Orthopaedic Surgery, JPP
200 Hawkins Dr.
Iowa City, IA 52242-1088

DEPARTMENT OF ORTHOPAEDIC SURGERY 1997-98 LECTURESHIPS AND CONFERENCES

Senior Residents' and Fellows' Day

May 9-11, 1997

Benjamin E. Bierbaum, M.D.
Tufts University School of Medicine
Boston, MA

Richard H. Gelberman, M.D.
Washington University
St. Louis, MO

Carroll B. Larson Shrine Memorial Lecture

May 22-24, 1997

Neil E. Green, M.D.
Vanderbilt University Medical Center
Nashville, TN

Michael Bonfiglio Iowa Orthopaedic Alumni Meeting

October 9-11, 1997

James Herndon, M.D.
University of Pittsburgh
Pittsburgh, PA

Mark Gebhart, M.D.
Harvard Medical School
Harvard, MA

Bruce Sangeorzan, M.D.
University of Washington
Seattle, WA

Sports Medicine Symposium

December 5-6, 1997

Senior Residents' and Fellows' Day

May 29-30, 1998

Christopher Bulstrode, M.D.
Oxford, England

Randy N. Rosier
University of Rochester
Rochester, NY

Johnston Lectureship in Hip Reconstruction

October 15-17, 1998

Charles A. Engh, M.D.
Arlington, VA

David G. Murray, M.D.
University of Syracuse
Syracuse, NY

STUART WEINSTEIN: ADVANCING THE SPECIALTY OF ORTHOPAEDICS

Joseph A. Buckwalter, M.D.

Orthopaedics began as the medical specialty devoted to the treatment of children suffering from deformities and diseases of the musculoskeletal system. Nicolas Andre (1658—1742), a Professor of Medicine in the Royal College in Paris, derived the term *Orthopaedic* from the Greek words orthon (straight, free from deformity) and paidion (child)⁴. He distinguished orthopaedics from other medical and surgical specialties with his book *L'Orthopedie* published in 1741. The word orthopaedic became part of the English language in 1743 when an English translation of Andre's book appeared with the title *Orthopaedia: or, The Art of Correcting and Preventing Deformities in Children*. Andre was not a surgeon, and he focused his book on the prevention and nonoperative treatment of skeletal deformity. Although the scope of the specialty has expanded considerably since the 1700s, the care of children suffering from deformities, diseases and injuries of the musculoskeletal system remains a central component of orthopaedics. For more than eighty years the Department of Orthopaedics at The University of Iowa has been an internationally recognized center for the treatment of children with developmental and congenital deformities and musculoskeletal diseases. Stuart Weinstein is one of the primary reasons for the continued preeminence of the Department of Orthopaedic Surgery at the University of Iowa. In his nearly twenty-five years as a member of the Department, his commitment to his patients, his students and his profession has led to major advances in the specialty of orthopaedics.

Stuart Weinstein was born and raised in Chicago where he attended Taft High School, an athletic powerhouse. Through hard work and commitment, he became the quarterback of the Taft football team and led them to the city championship played at Soldier Field. Stuart, thus, joined the elite group of individuals who have played quarterback for a championship team at Soldier Field. From Taft he moved on to the University of Illinois where on a blind date he met his future wife Lynn (Figure 1). During college he served as president of a very social fraternity, participated enthusiastically in intramural sports and excelled academically. He graduated Cum Laude from the University of Illinois with a degree in political science and history in 1968.

Following college Stuart chose to attend the University of Iowa medical school where he distinguished himself not only by his academic excellence but by his skill



Figure 1. Stuart and Lynn at the University of Illinois.

as a quarterback for the AKK fraternity intramural team. After his first year in medical school, Stuart and Lynn were married. During his third year, Stuart was selected for the very competitive Oxford clerkship, an honor given to only one student each year. Despite the demands on his time during medical school, Stuart maintained his level of participation in sports and developed a deep and lasting enthusiasm for the Hawkeye athletic teams.

After graduating from medical school in 1972, he completed an internship in Internal Medicine at the University of California, San Francisco. He returned to Iowa to begin his long association with the Department of Orthopaedics as an orthopaedic resident in 1973. During his residency Dr. Weinstein's talent as a clinician



Figure 2. Dr. Ignacio Ponseti.

became obvious to everyone who had the opportunity to work with him. In addition, he conducted a number of studies of orthopaedic conditions including femoral neck fractures¹⁷ and the reconstruction of flexor tendons in severely injured digits²¹. He, also, impressed the faculty and his fellow residents with his interest in teaching medical students and his contributions to departmental educational programs. On one occasion, when he was responsible for preparing slides for the departmental pathology conference, he selected sections of amyloidosis and a hot dog as unknown cases². After a brief discussion, the other residents agreed on the diagnosis of amyloidosis, although some felt that the specimen selected tested their knowledge of histology more than what was necessary. They had more difficulty with the second specimen. After considerable effort, they identified fragments of tissue consistent with necrotic cartilage, bone, dense fibrous tissue, muscle and fat. Suggested diagnoses included necrotic tumors of various types and crushed infarcted limbs.

In 1976 Dr. Weinstein joined the faculty at the University of Iowa as an Assistant Professor of Orthopaedic



Figure 3. Dr. Weinstein with Dr. Dennis Wenger, a former Iowa orthopaedic resident now a well recognized expert in pediatric orthopaedics practicing in San Diego, and Dr. Ignacio Ponseti.

Surgery. In 1980 he was promoted to Associate Professor and then Professor in 1984. In 1987 he was named the Ignacio V. Ponseti (Figure 2) Professor of Orthopaedic Surgery, an honor that recognized Dr. Weinstein's role in continuing the tradition of outstanding pediatric orthopaedics at The University of Iowa: a tradition that began with Dr. Steindler in 1915³, was strengthened by Dr. Ponseti starting in the early 1940s⁴, and followed by Dr. Weinstein and others (Figure 3).

During his career at Iowa, Dr. Weinstein's skillful treatment of children with musculoskeletal disorders has earned him an enviable national and international reputation. He provides compassionate and superb care to patients with complex spinal deformities, developmental dysplasia of the hips, foot deformities, and other musculoskeletal diseases and injuries. Physicians and orthopaedic surgeons throughout the region call on Dr. Weinstein for consultation and refer him their most difficult and challenging pediatric patients. Even his youngest and most anxious patients and their parents instantly recognize his humanity and compassion as well as his exceptional skill.

Dr. Weinstein's thoughtful studies of his clinical experience have helped advance our understanding of musculoskeletal deformities and diseases in children and improve the treatment of spinal deformities, developmental dysplasia of the hip, slipped capital femoral epiphysis, Legg-Calve-Perthes Disease, metatarsus adductus and multiple other conditions^{5,7,8,11-13,18,20,22}. He has reported this work in well over 100 publications and nearly 400 national and international presentations. Understanding the natural history of a disease and the long term results of treatment significantly improve the ability of physicians to develop and select the best treatment or determine when treatment is indicated. Dr.

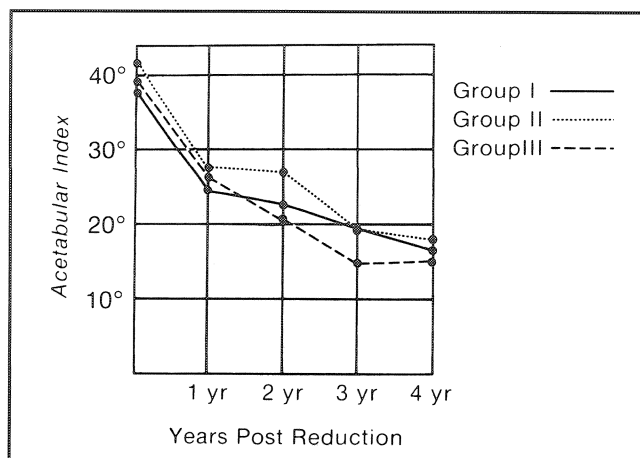


Figure 4. A plot of the change in the acetabular index (a measure of the development of the acetabulum) following reduction of hip dislocations in children. Group I—children one year old or younger at the time of reduction. Group II—children one to two years old at the time of reduction. Group III—children older than two years at the time of reduction. (Reproduced from the *Journal of Bone and Joint Surgery*¹⁹ with permission.)

Weinstein is among the few physicians who have made careful studies of both the natural history of human musculoskeletal diseases and the long term results of operative and nonoperative treatments. He has shown the critical importance of following patients not just for years, but for decades. The selected sample of Dr. Weinstein's work summarized in the following paragraphs illustrates the depth and importance of his contributions^{7,8,11-13,18,20,22}.

His studies of hip dysplasia have helped form the basis for the current treatment of this condition. In 1979 he joined with Dr. Ponseti in reporting the safety and efficacy of their surgical approach to the treatment of dislocated hips in infants¹⁹. In addition to describing their treatment, they studied hip development in their patients and demonstrated how the structure of the acetabulum improved following reduction of the hip (Figure 4). In 1994, along with orthopaedic resident Dr. Thomas Malvitz, Dr. Weinstein described the functional and radiographic results of closed reduction of 152 congenitally dislocated hips in 119 patients at an average of thirty years after treatment¹¹. This unique study showed that the younger the patient at the time of reduction, the better the clinical and radiographic results. Furthermore, patients who did not have a growth disturbance of the proximal end of the femur or evidence of hip subluxation tended to function well for many years despite a radiographically abnormal hip (Figure 5). The results of this study led Dr. Weinstein to institute a treatment protocol designed to establish more normal acetabular structure in patients with dysplastic hips.

In a series of studies reported in 1981 and 1983, Drs.



Figure 5A.

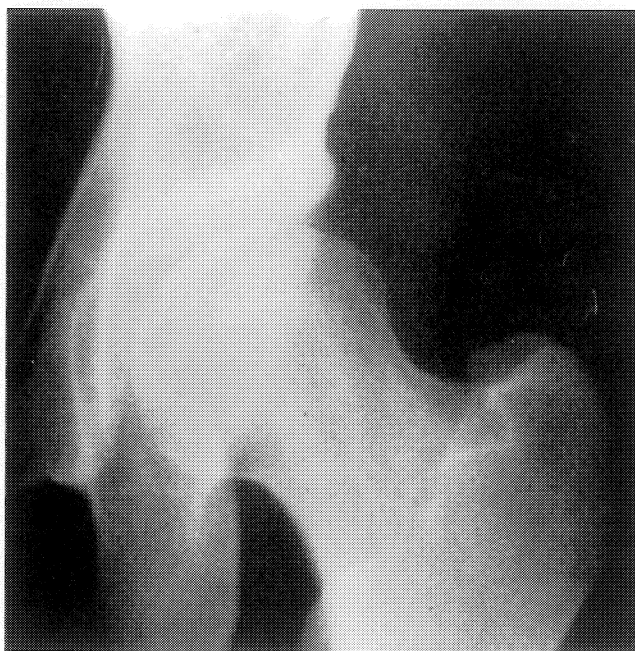


Figure 5B.

Figure 5. Radiographs showing the long term results in a patient with a dislocation of the hip treated by closed reduction at the age of two years and four months. A) Thirty-nine months after reduction accessory centers of ossification are visible in the acetabular cartilage. B) Fifteen years after reduction there is minimal acetabular dysplasia. C) Forty-two years after reduction there is evidence consistent with early joint degeneration. D) Fifty-one years after reduction severe degenerative changes have developed and the hip is subluxated. (Reproduced from the *Journal of Bone and Joint Surgery*¹¹ with permission.)



Figure 5C.



Figure 5D.

Weinstein and Ponseti defined the natural history of spinal curve progression in patients with idiopathic scoliosis^{6,20,22}. This work was the continuation of an earlier investigation by Collis and Ponseti⁶. They evaluated 102 patients with untreated idiopathic scoliosis followed for a average of 40.5 years. Contrary to previous reports indicating that idiopathic scoliotic spinal curves stabilized once patients reached skeletal maturity, Drs. Weinstein and Ponseti showed that idiopathic scoliotic deformities could increase in adult life, especially thoracic curves. In particular they found that spinal curves that measured less than thirty degrees at the time of skeletal maturity tended not to increase during adult life while curves greater than thirty degrees frequently progressed. The progression appeared to be related to the degree of vertebral body rotation, and thoracic curves greater than fifty degrees showed the most rapid progression (Figure 6).

In 1993 Dr. Weinstein and colleagues reported a long term follow-up study of patients with Scheuermann's kyphosis that helped clarify the indications for surgical treatment of this condition. Prior to Dr. Weinstein's report many orthopaedists believed that untreated Scheuermann's kyphosis led to back pain, diminished activity, hamstring muscle tightness, poor social functioning, embarrassment concerning physical appearance, myelopathy, intervertebral disc degeneration, spondylolisthesis and cardiopulmonary failure. Dr.

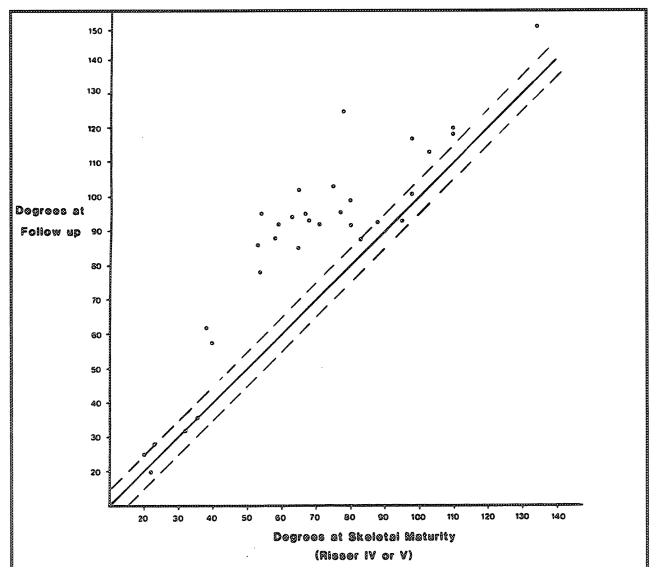


Figure 6. Scatter plot showing the relationship between the degree of spinal curvature at skeletal maturity and the degree of spinal curvature at the time of a follow-up examination for patients with idiopathic thoracic scoliosis. Notice that curves that were less than thirty degrees at the time of skeletal maturity did not progress while curves that were greater than fifty degrees at the time of skeletal maturity did progress. (Reproduced from the *Journal of Bone and Joint Surgery*²⁰ with permission.)

TABLE 1. SURGICAL TREATMENT OF SCHEUERMANN'S KYPHOSIS

Authors	Number of Patients	Mean Degree of Kyphosis	Treatment	Complications of Treatment
Taylor et al. ¹⁶	29	71	Spine Fusion	<ul style="list-style-type: none"> • Tension pneumothorax (1) • Failure of fixation (2) • Persistent kyphosis (4) • Gastrointestinal obstruction (4) • Persistent back pain (9) • Wound hematoma (3)
Speck & Chopin ¹⁵	59		Spine Fusion	<ul style="list-style-type: none"> • Infection (4) • Brown-Sequard syndrome (1) • Failure of fixation (1) • Spinal fracture (7) • Loss of correction (10)
Lowe ¹⁰	24	85	Spine Fusion	<ul style="list-style-type: none"> • Fixation problems (6) • Transient hyperesthesia (4) • Progression of kyphosis (2)
Herndon et al. ⁹	13	72	Spine Fusion	<ul style="list-style-type: none"> • Death (1) • Spinal fracture (1) • Venous thrombosis (1) • Transient motor weakness (1) • Back pain (1) • Psychiatric difficulties (2)
Bradford et al. ¹	24	77	Spine Fusion	<ul style="list-style-type: none"> • Loss of correction (5) • Pulmonary embolus (2) • Infection (1) • Hemothorax (1) • Duodenal obstruction (1) • Pseudarthrosis (1) • Pericardial effusion (1)

Weinstein evaluated sixty-seven patients with Scheuermann's kyphosis with a mean kyphotic angle of seventy-one degrees at an average of thirty-two years after diagnosis and compared them to an age and sex matched control group. The patients with Scheuermann's kyphosis reported more intense back pain and had jobs that tended to have lower physical activity requirements. They had a smaller range of extension and less extension strength of the trunk; but they did not differ from the control group in level of education, number of days absent from work because of back pain, the extent that back pain affected their daily activities, presence of numbness in the lower ex-

tremities, self-consciousness, self-esteem, social limitations, use of medications for pain or level of recreational activities. Dr. Weinstein's review of the surgical treatment of Scheuermann's kyphosis showed that spinal arthrodesis is associated with a number of complications (Table 1). Based on these observations Dr. Weinstein concluded that although patients with untreated Scheuermann's kyphosis have some limitations they can adapt to their condition, and the use of operative treatment of Scheuermann's kyphosis should be carefully reviewed.

Medical students, residents and practicing physicians throughout the world have benefited from Dr.

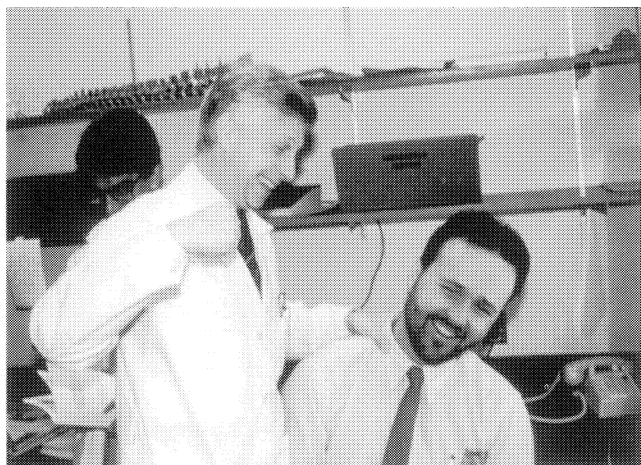


Figure 7. Dr. Weinstein's approaches to resident teaching. A) Dr. Weinstein listening patiently as a resident explains why he has not finished his clinic dictation. B) Dr. Weinstein explaining to the resident the importance of timely completion of clinic dictations.

Weinstein's skill as a teacher (Figures 7 & 8). He is thoughtful and kind, yet provocative and stimulating with a warm sense of humor. He brings out the best in his students and expects that they will follow his example in committing themselves to provide the best possible care for every patient. Institutions throughout the United States, Europe, Asia and South America have benefited from his superb series of lectures on pediatric orthopaedic conditions. No individual who has heard Dr. Weinstein speak doubts his depth of knowledge or critical evaluation of information. He has the ability to present ideas and concepts in ways that are easily understood and help others improve the care of children with orthopaedic conditions.

In addition to his many clinical contributions to orthopaedics at Iowa, Dr. Weinstein has advanced the specialty and brought recognition and honor to the University of Iowa through his service in leadership

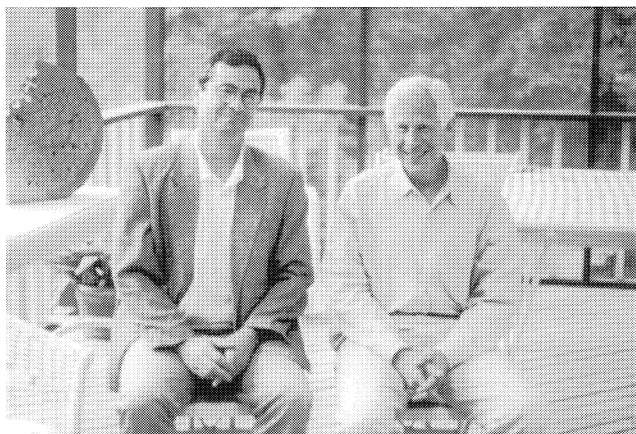


Figure 8. Dr. Weinstein enjoying a recent festive occasion with a former resident.

positions for major orthopaedic organizations. He has served the American Orthopaedic Association in multiple capacities, skillfully promoting one of the major missions of the Association, international orthopaedic education (Figure 9). Among his most important contribution to international orthopaedic education is the development of the International Center of Orthopaedic Education (ICOE), a program that makes it possible for institutions and individuals who offer postgraduate educational experiences to exchange information with individuals interested in these opportunities. In 1996 the ICOE made information concerning more than 1,700 worldwide educational opportunities from 802 institutions and forty-nine countries available to applicants. His contributions to the American Orthopaedic Association will continue as he was elected First President-Elect of the American Orthopaedic Association in 1996 and will serve as President beginning in 1997. He has served as both an oral examiner and member of the Written Examination Committee for the American Board of Orthopaedic Surgery and was elected a Director of the American Board of Orthopaedic Surgery in 1995. He is currently serving as Chairman of the American Board of Orthopaedic Surgery Oral Examination Committee. He has been a major influence in the development of the Pediatric Orthopaedic Society, serving on numerous committees and as President in 1991 and 1992. In addition, he has made many contributions to the American Academy of Orthopaedic Surgeons and the Scoliosis Research Society.

Over the last eighty years, hundreds of thousands of patients, the University of Iowa, the State of Iowa and the specialty of orthopaedics have benefited from the many and diverse strengths of the University of Iowa Department of Orthopaedics. It is not unusual for individuals from other regions of the United States and

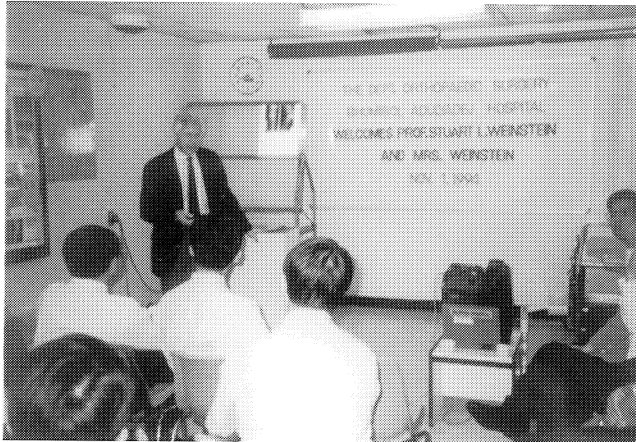


Figure 9. Dr. Weinstein serving as a visiting professor for the Department of Orthopaedic Surgery at Bhumibol Adulyadej Hospital in Thailand. (From top) Dr. Weinstein lecturing; Dr. Weinstein examining a patient; and the members of the orthopaedic department with Dr. Weinstein. Dr. Amnuay Jirasirikul, a former fellow of Dr. Weinstein's, is seated to Dr. Weinstein's left.

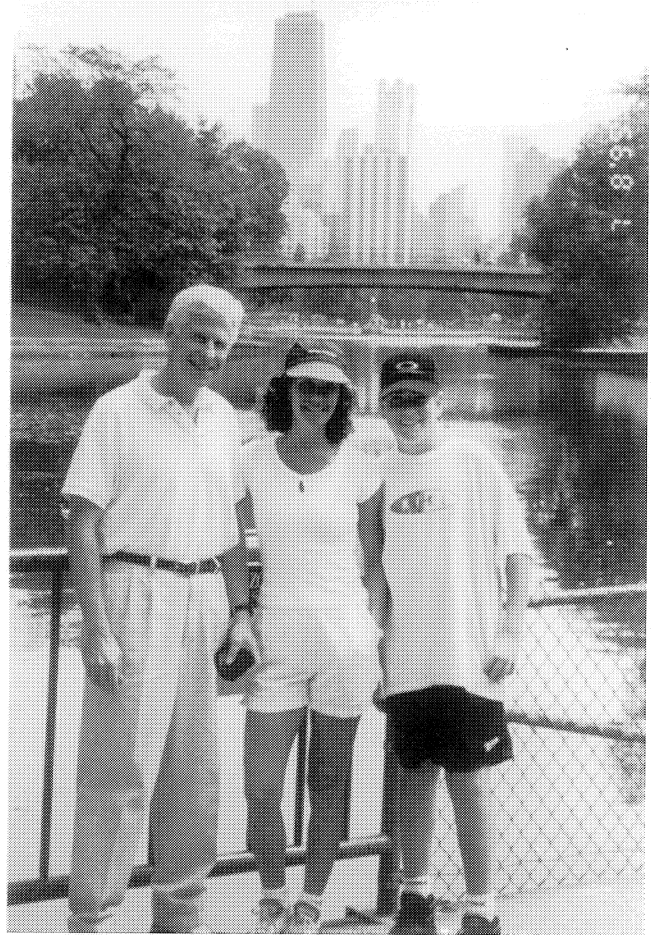


Figure 10. Stuart and his son Willie engaging in a test of skill and strength (top) and the Weinstein family in Chicago (bottom).

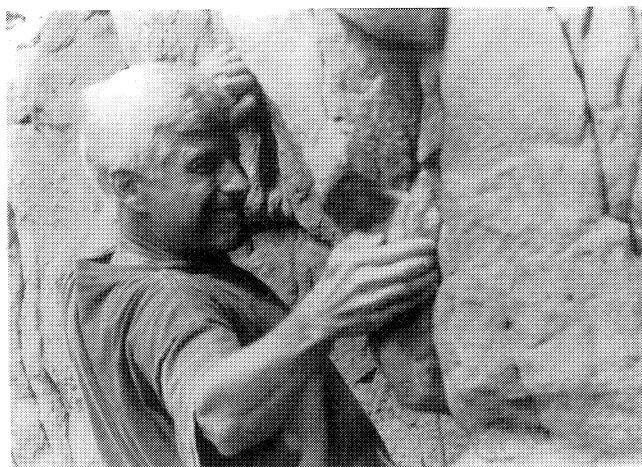


Figure 12. Dr. Weinstein relaxing with his son Willie.

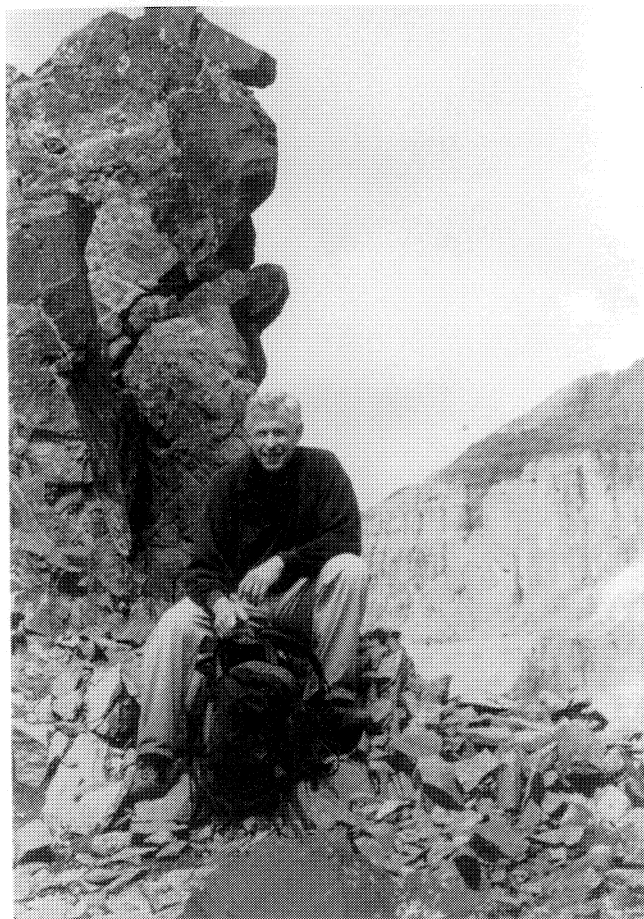


Figure 11. Dr. Weinstein enjoying a challenging climb (top) and a brief rest (bottom).

other countries to ask why the Orthopaedic Department at the University of Iowa has accomplished so much and contributed so greatly to the care of patients with musculoskeletal disorders and to the advancement of the specialty. One critical part of the answer to this question is the dedication, talent and commitment to excellence of Stuart Weinstein. He is an physician of exceptional skill and compassion. He is intensely loyal to his family (Figure 10), his patients, his profession and the University of Iowa. He is a man of unusual determination and commitment who enjoys challenges (Figure 11), but who also enjoys relaxing (Figure 12). Although he has spent his professional life at the University of Iowa, his influence extends far beyond Iowa. His students and residents now help care for patients throughout the United States and many countries abroad. His service in national orthopaedic organizations has improved orthopaedic education and practice, and his critical reviews of the natural history of musculoskeletal deformities and their treatment have not only improved care for patients with these problems, they have raised the standards for clinical research.

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WERE THE HUNTER BROTHERS WRONG? CAN SURGICAL TREATMENT REPAIR ARTICULAR CARTILAGE?

Joseph A. Buckwalter, M.D.



Figure 1. John Hunter (1728-1793)

Few individuals, and no pair of brothers, have had greater influence on the development of surgical practice than William (1718-1783) and John (1728-1793) Hunter. Many authors appropriately credit them with establishing the value of a scientific approach to surgical treatment of injuries, diseases and deformities. William Hunter received a medical degree from the University of Glasgow in 1750 and became a licensed physician in London in 1756. Throughout his life he collected anatomical and pathological human specimens for study and teaching. He is most widely recognized for his role in establishing obstetrics as an accepted

branch of medicine, but he also made significant observations concerning cardiovascular and rheumatic diseases. Most important for orthopaedists, he studied the structure, function and pathology of articular cartilage³⁶. His younger brother, John (Figure 1), dedicated much of his career to the study and healing of musculoskeletal tissues. He never completed a university degree and never attempted to become a doctor of medicine, although after studying and teaching anatomy under his older brother's direction and serving as a surgeon's assistant, he was admitted to membership in a corporation of surgeons and he received a commission as an army surgeon. As an anatomist, physiologist, pathologist and army surgeon he brought an unusual combination of knowledge and experience to his studies. Like his brother he collected thousands, possibly more than 10,000, anatomical and pathologic specimens.

Recently, the influence of the Hunters on orthopaedics has been most apparent in discussions of surgical approaches to restoring damaged or diseased articular surfaces. Scientists and clinicians interested in the restoration of articular cartilage frequently include in their lectures and manuscripts William Hunter's observation: "Ulcerated cartilage is a troublesome thing . . . once destroyed it is not repaired^{25,36}". John Hunter's influence is less obvious, but perhaps as important. He viewed surgical treatment of diseased or injured joints with considerable skepticism. Based on his extensive clinical studies he believed that active motion, muscle strengthening and massage provided the best treatment of injured or diseased joints, and that in general, surgical interventions only interfered with natural healing processes. Thus, the Hunters concluded that damaged or diseased articular cartilage did not restore itself and that surgical treatments would not correct this problem. Generations of surgeons have been directly or indirectly influenced by the Hunters' approach to joint injuries and diseases.

In the last three decades clinical and basic scientific investigations have shown that a variety of methods, including implantation of artificial matrices, growth factors, perichondrium, periosteum and transplanted chondrocytes and mesenchymal stem cells, can stimulate formation of cartilaginous tissue in synovial joint osteochondral and chondral defects^{16,18,21,23}. In addition, a review of several operative procedures used to treat osteoarthritis, (osteotomies, penetration of subchondral

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Figure 2. Light micrographs of rabbit patellar articular cartilage stained with Safranin O to demonstrate the presence of glycosaminoglycans. The joint surface is at the top, the calcified cartilage and subchondral bone at the bottom.

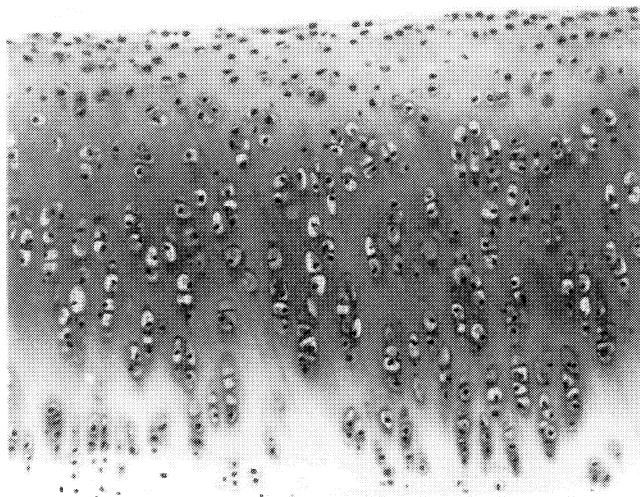


Figure 2A. Normal rabbit articular cartilage. Notice the zonal organization of the matrix: small ellipsoidal chondrocytes in the superficial zone next to the articular surface, isolated and clustered spheroidal chondrocytes in the transitional zone and spheroidal chondrocytes forming columns in the deep zone. The matrix has a homogeneous, hyaline, appearance. The matrix of the transitional and deep zones stains diffusely with Safranin O, i.e., the dark regions of the matrix, indicating a high concentration of glycosaminoglycans.

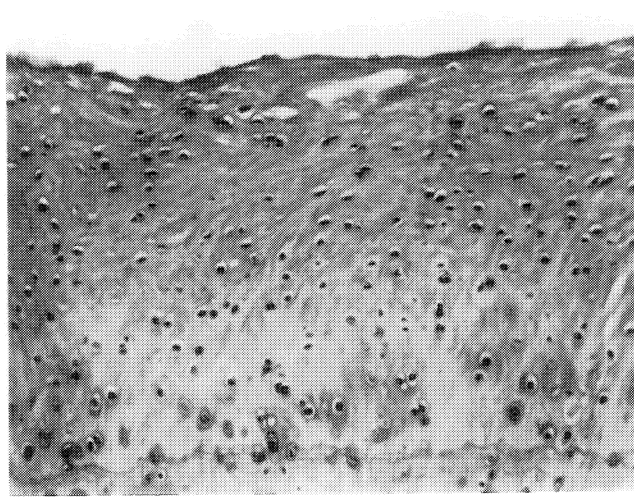


Figure 2B. Well formed repair cartilage six months after creation of an osteochondral defect. Compared with normal articular cartilage the zonal organization is less well defined, the matrix has a fibrous appearance and Safranin O staining concentrates near a few cells. The zone of calcified cartilage has reformed.

bone, joint distraction and motion) has shown that these procedures can stimulate formation of new articular surfaces^{21-23,44,45,57,69}. The apparent potential of these multiple methods for stimulating cartilage formation has created great interest on the part of patients, physicians and scientists, and raised the question: were the Hunter brothers wrong, can surgical interventions repair an articular surface?

WHAT IS ARTICULAR CARTILAGE REPAIR?

To some extent, the answer to this question depends on the definition of articular cartilage repair. The terms repair and regeneration are, at times, used interchangeably in discussions of approaches to restoring lost or damaged articular cartilage. This practice contributes to confusion in evaluating methods of stimulating articular cartilage formation. Repair refers to the replacement of lost or damaged tissue with new tissue that may not duplicate the structure, composition and function of the original tissue. Regeneration refers to the formation of new tissue that duplicates the structure, composition and function of the original tissue^{19,75}. Figure 2 shows the differences between normal articular cartilage (Figure 2A) and well formed repair cartilage (Figure 2B). Although the repair tissue has restored an articular surface, and when examined from the surface may appear nearly normal, the repair tissue does not

duplicate the structure, composition and material properties of normal articular cartilage^{3,22,34}.

The capacity for regeneration of tissue varies among species and tissues, and with age¹⁹. Humans have the capacity to regenerate bone tissue at any age following bone fracture or in some instances, even loss of bone tissue. Osteochondral fracture or degeneration of cartilage extending to bone can stimulate the formation of cartilaginous tissue in humans, but this tissue differs in matrix composition and mechanical properties from normal articular cartilage^{22,24}, and thus is not the result of articular cartilage regeneration in the strictest sense. Infants and possibly even some older children may be an exception to the lack of articular cartilage regeneration in humans¹⁹. In some instances they heal osteochondral injuries with tissue that cannot be distinguished from normal articular cartilage by gross inspection and imaging studies. Although regeneration of normal articular cartilage would be an optimal result for the treatment of cartilage loss or damage, and methods of regenerating human articular cartilage may be discovered, repair tissue can provide excellent function. For example, repair tissue can restore the function of lacerated or ruptured tendons and ligaments¹⁹.

Determining which methods have the greatest promise of restoring articular surface depends on comparing their results. Yet, one of the problems faced by physicians and scientists in comparing the multiple methods of stimulating articular cartilage repair is the variabil-

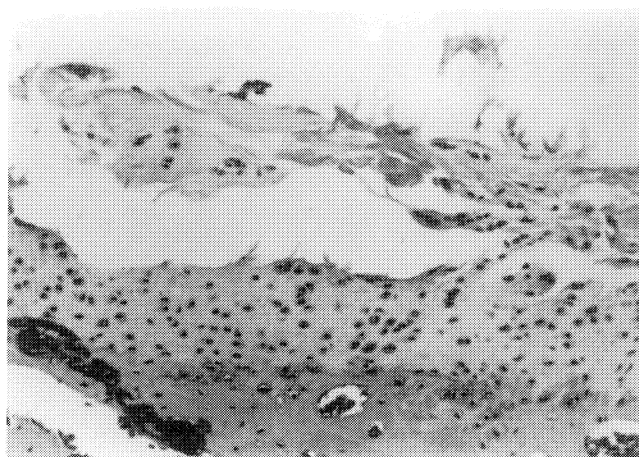


Figure 2C. Degenerating repair cartilage. Notice the fragmentation of the fibrous repair cartilage.

ity in experimental models and clinical applications. For example, species differ in the thickness and mechanical properties of their articular cartilage² and probably the type and quality of natural cartilage repair, or effects of various methods of stimulating repair. Furthermore, methods which stimulate cartilage repair in a normal animal joint will not necessarily lead to similar success in an osteoarthritic human joint. In humans, patient age, condition of the joint (including stability, alignment and extent of degenerative change), loading and motion of the joint following treatment, activity level and body weight may affect results^{15,19,21}.

Considerable variability also exists in methods of measuring the results of attempts to stimulate articular cartilage repair. The ultimate measure of a method of articular cartilage repair or regeneration is the extent to which the method restores the long term function of a synovial joint; that is full painless low friction motion, distribution of loads across the joint and durability. Two years provides a reasonable minimum time necessary to assess restoration of joint function, and five years or more of improved joint function is a reasonable expectation for any procedure that would have significant value for large numbers of people. However, requiring that a method of restoring articular cartilage demonstrate long term restoration of joint function before proceeding with further studies, refinements and clinical trials would considerably delay development and clinical use of promising treatments. For this reason, short term evaluation is also necessary. The available experimental evidence suggests that chondral and osteochondral repair tissue formation is usually complete in six weeks or less following an attempt to stimulate restoration of an articular surface, but that remodeling of the repair tissue may continue for many months and prob-

ably years^{19,21,22}. Degeneration of well formed articular cartilage repair tissue may occur within months, or years after apparent clinically successful improvement in joint function²⁴ (Figure 2C). Thus the minimal time for short term evaluation of cartilage repair for most methods is in the range of four to six weeks, and for those methods that show promise at this earliest time period another evaluation after six months would be desirable. The best short term evaluation of a method of stimulating cartilage repair is the extent to which that method produces new tissue that restores joint function, structure and an articular surface that duplicates the volume, shape, structure, composition and mechanical properties of normal articular cartilage. Table 1 lists measures that can be used to evaluate the short term results of attempts to restore an articular cartilage surface. In addition to these measures, recent work suggests that expression of the proteoglycan epitope 7D4 distinguishes hyaline-like repair cartilage from normal articular cartilage and from fibrous repair cartilage in the early stages of articular cartilage repair, and that with maturation and remodeling of hyaline-like articular cartilage repair tissue, the expression of 7D4 decreases⁴⁶.

It has been generally assumed that cartilaginous repair tissue that more closely resembles normal articular cartilage will also have mechanical properties that more closely resemble normal articular cartilage, and that the volume, morphology and histochemical staining of repair tissue will correlate with the mechanical properties of the tissue. One recent study examined this latter assumption⁶⁶. Spirt and colleagues, using statistical evaluation of inter-rater reliability, progressively refined a semi-quantitative scale for histologic evaluation of cartilage repair. This procedure produced a highly reliable scale for evaluation of cartilage repair consisting of four categories: thickness of the chondral repair tissue, degree of subchondral bone depression, intensity of Safranin O staining in the chondral repair tissue and cell morphology in the chondral repair tissue. Comparison of scores on the refined articular cartilage repair scale with indentation stiffness of the repair tissue showed a strong correlation between better articular cartilage repair as measured by the scale and increased indentation stiffness: the scores on the scale accounted for more than 70 percent of the indentation stiffness of the repair tissue ($r^2 = .71$, $p < .002$). The results of this study suggest that the minimal evaluation of articular cartilage repair tissue should include the volume or thickness of the chondral repair tissue, when appropriate the restoration of the subchondral bone structure, the morphology of the chondral repair tissue and an estimate of the proteoglycan content of the chondral repair tissue.

Table 1. Evaluating Cartilage Repair

Joint Function	<ul style="list-style-type: none"> • Range of Motion • Smoothness of Motion (lack of catching, locking or crepitus) • Pain • Stability
Joint Structure	<ul style="list-style-type: none"> • Articular Surface Integrity, Contour and Congruity • Subchondral Bone Plate Contour, Shape, Thickness and Organization • Synovial Membrane Thickness and Cellularity (Synovitis/Effusion) • Osteophytes • Alignment
Chondral Repair Tissue Structure	<ul style="list-style-type: none"> • Volume (thickness) • Integrity (presence or absence of fibrillation or fissures) • Binding to Adjacent Normal Tissue • Matrix Zonal Organization • Cellularity (Cell Density) • Cell Morphology (Chondrocytic vs. Fibroblastic)
Chondral Repair Tissue Matrix Composition	<ul style="list-style-type: none"> • Type II Collagen Concentration • Type I Collagen Concentration • Water Concentration • Aggrecan Concentration, Degree of Aggregation, Aggrecan Size and Composition • Dermatan Sulfate Proteoglycan Concentration • Non-collagenous Protein Concentrations (Fibronectin, Tenascin)
Repair Tissue Mechanical Properties	<ul style="list-style-type: none"> • Stiffness • Permeability • Strength

PENETRATION OF SUBCHONDRAL BONE

Penetration of subchondral bone was the first method developed to stimulate formation of a new articular surface and is still the most commonly used²¹. In regions with full thickness loss or advanced degeneration of articular cartilage, penetration of the exposed subchondral bone disrupts subchondral blood vessels leading to formation of a fibrin clot over the bone surface^{19,21}. If the surface is protected from excessive loading, undifferentiated mesenchymal cells migrate into the clot, proliferate and differentiate into cells with the morpho-

logic features of chondrocytes⁶⁵. In many instances they form a fibrocartilagenous repair tissue over the bone surface similar to that shown in figure 2B^{44,45}. Experimental studies have shown that the mesenchymal cells in the osseous portion of an osteochondral defect form bone and smaller amounts of cartilage while the cells in the chondral portion of the defect form a cartilaginous tissue that in some instances closely resembles articular cartilage^{19,24,51}. However, the extent and quality of cartilage formed in the chondral portions of the defects varies considerably among defects and rarely fills more than 75 percent of the total volume of the chondral defect²⁴.

Surgeons first debrided degenerated articular cartilage and drilled into the subchondral bone through arthrotomies and found that many patients reported a decrease in symptoms following recovery from the procedure^{5,34,39,48}. One group advocated treating patellar articular surface degeneration by excising damaged cartilage along with underlying subchondral bone, a procedure they referred to as “spongialization.” They found good or excellent results in a high percentage of their patients³¹. Surgeons have developed a variety of other methods of penetrating subchondral bone to stimulate formation of a new cartilaginous surface including arthroscopic abrasion of the articular surface and making multiple small diameter defects or fractures with an awl or similar instrument²¹. A number of surgeons have reported that arthroscopic abrasion of chondral and osteochondral lesions in osteoarthritic joints can decrease symptoms^{4,29,44,45}. Examination of joint surfaces following arthroscopic abrasion has shown that in many individuals it results in formation of a fibrocartilagenous articular surface that varies in composition from dense fibrous tissue with little or no type II collagen to hyaline cartilage-like tissue with predominantly type II collagen^{44,45}. In some patients this tissue persists for years.

Prospective randomized controlled trials of arthroscopic abrasion treatment of osteoarthritic joints have not been reported, but several authors have reviewed series of patients and found that these procedures can decrease the symptoms of osteoarthritis of the knee. One group of investigators treated seventy-three patients with arthroscopic debridement and abrasion of subchondral bone³². At an average of twelve months following the procedure 60 percent of the patients were improved, 34 percent were unchanged and 6 percent were worse. Another investigator found that 73 percent of his patients had relief of pain and stiffness following arthroscopic debridement and abrasion²⁹. He noted that the probability of a satisfactory clinical result decreased with increasing severity of the degen-

erative joint disease. Sprague found 84 percent good results following arthroscopic debridement of seventy-eight knees⁶⁷; and Johnson found that 75 percent of the patients with exposed subchondral bone treated by abrasion arthroplasty had satisfactory results, although only 12 percent of the patients in this series had no symptoms at two years following treatment^{44,45}. Baumgaertner and associates reported less successful results: they found 39 percent early failures in a series of forty-nine knees, and 47 percent failures at final follow up examination⁴. In this same series, excellent results decreased from 41 percent of the patients at the time of maximum improvement to 24 percent at the time of final follow up.

Johnson also found that in many patients with radiographic evidence of cartilage joint space narrowing, or no radiographically demonstrable joint space, the joint space increased following abrasion^{44,45}. Although an increase in radiographic joint space following subchondral abrasion presumably indicates formation of a new articular surface, the development of this new surface does not necessarily result in symptomatic improvement. Bert and Maschka found that 51 percent of fifty-nine patients treated with abrasion arthroplasty had evidence of increased radiographic joint space two years after treatment, but 31 percent of these individuals either had no symptomatic improvement or more severe symptoms.^{8,9}

Some of the variability in the clinical results of attempts to restore an articular surface by penetrating subchondral bone may result from differences in the extent and quality of the repair tissue. However, no studies have documented a relationship between the extent and type of repair tissue and symptomatic or functional results, suggesting that formation of a new articular surface following penetration of subchondral bone does not necessarily relieve pain. The lack of predictable clinical benefit from formation of cartilage repair tissue may result from variability among patients in the severity of the degenerative changes, joint alignment, patterns of joint use, age, perception of pain, pre-operative expectations or other factors. It may also result from the inability of the newly formed tissue to replicate the properties of articular cartilage. Examination of the tissue that forms over the articular surface following penetration of subchondral bone shows that it lacks the structure, composition, mechanical properties and in most instances the durability of articular cartilage^{19,21,22,24,52}. For these reasons, even though it covers the subchondral bone, it may fail to distribute loads across the articular surface in a way that avoids pain with joint loading and further degeneration of the joint.

Currently, it is not clear which method of penetrating subchondral bone produces the best new articular surface, and differences in patient selection and technique among surgeons using the same method may be responsible for variations in results making it difficult to compare techniques. However, comparison of bone abrasion with subchondral drilling for treatment of an experimental chondral defect in rabbits showed that while neither treatment predictably restored the articular surface, drilling appeared to produce better long term results than abrasion³⁰. This observation fits well with previous experimental work showing that chondral repair tissue that grows up through multiple drill holes that pass from the articular surface into vascularized bone will spread over exposed subchondral bone between holes and form a fibrocartilaginous articular surface⁵². It also suggests that small diameter holes that leave the bone intact between defects lead to formation of more stable repair tissue than abraded bone surfaces.³⁰

Despite the evidence that penetration of subchondral bone stimulates formation of fibro-cartilaginous repair tissue, the clinical value of this approach remains uncertain. In contrast with reports of symptomatic improvement in patients with cartilage degeneration treated with penetration of subchondral bone^{29,32,44,45,67}, one investigator has concluded that while joint debridement can improve symptoms in many patients, abrasion or drilling of subchondral bone does not benefit patients with osteoarthritis of the knee and may increase symptoms⁸. In addition, the short periods of follow up, lack of well defined evaluations of outcomes, lack of randomized controlled trials and the possibility for a significant placebo effect⁵³, or an improvement in symptoms due to joint irrigation alone^{33,47}, make it difficult to define the indications for penetration of subchondral bone to stimulate formation of a new articular surface.

OSTEOTOMIES

Osteotomies of the hip and knee are a generally accepted method of treating joints with localized loss or degeneration of the articular surface. Osteotomies have not been commonly used for treatment of articular cartilage loss or degeneration in joints other than the hip and knee, but in one study tibial osteotomies produced good or excellent results in fifteen of eighteen patients with primary ankle osteoarthritis, a rare condition in which osteoarthritis develops in the absence of any history of trauma⁶⁸. In selected patients surgeons perform osteotomies to correct skeletal deformities or joint incongruities that may lead to joint degeneration.

Treatment of an osteoarthritic joint or a joint with a large osteochondral defect with an osteotomy consists of cutting the bone adjacent to the involved joint and then stabilizing the cut bone in a new position, thereby changing the alignment of the joint. Some surgeons have combined joint debridement and penetration of subchondral bone with osteotomy, but this approach is not widely used. In general, surgeons plan osteotomies to decrease loads on the most severely damaged regions of the joint surface, bring regions of the joint surface that have remaining articular cartilage into opposition or correct joint malalignment that may be contributing to symptoms and joint dysfunction. Most hip and knee osteotomies performed to treat osteoarthritis alter joint alignment in the coronal plane (varus and valgus osteotomies). However, surgeons design some hip osteotomies to change joint alignment in the sagittal plane (flexion and extension osteotomies) or alter the relationship of the joint surfaces by rotation of the femoral head relative to the acetabulum (rotational osteotomies).

The optimal planes and degrees of joint realignment for specific osteoarthritic joints have not been defined. Nonetheless, clinical experience shows that osteotomies of the hip and knee can decrease symptoms and stimulate formation of a new articular surface²¹. The decreased pain could result from decreasing stresses on regions of the articular surface with the most advanced cartilage degeneration, decreasing intraosseous pressure or formation of a new articular surface, but the mechanisms of symptomatic improvement and formation of new articular surfaces remain poorly understood²¹.

Most clinical studies have shown that in at least some patients osteotomies lead to improvement in the radiographic signs of joint degeneration including resolution of subchondral cysts or lucencies, decreased subchondral bone density and increased radiographic joint space²¹. This latter change may result either from the altered relationship between the articular surfaces or the formation of a new articular surface. That is, osteotomies may alter joint alignment to separate previously opposed joint surfaces or they may rotate a cartilage covered articular surface into opposition with a surface consisting of exposed bone, thus creating a radiographically visible cartilage space where prior to the osteotomy, bone opposed bone. In one series of 757 intertrochanteric osteotomies performed to treat osteoarthritis of the hip, the radiographic joint space increased immediately following the procedure in approximately one-third of the patients⁷⁴. In these patients the increased joint space presumably resulted from alterations in the relationships between the joint surfaces. In another third of the patients the radiographic joint

space increased during the next eighteen months, and these individuals had better clinical results. This result suggests that over eighteen months these patients developed a new articular surface in some areas of the joint as a result of the altered loading. Evidence that hip osteotomies stimulate formation of fibrocartilaginous tissue over articular surfaces that previously consisted of exposed bone supports this suggestion^{10,43}.

Reports of the treatment of degenerative disease of the knee with osteotomies also describe increased radiographic joint space accompanied by decreased subchondral sclerosis, and in some people formation of a new fibrocartilaginous articular surface²¹. One group of investigators biopsied the articular cartilage of the medial femoral condyle at the time of osteotomy and then again at an average of two years after osteotomy in nineteen patients with degenerative disease of the medial side of the knee joint⁶. The biopsies showed formation of a new fibrocartilaginous articular surface in nine patients, no change in eight patients and deterioration of the articular surface in two patients. Radiographic examination showed that six knees had improved, eleven had remained unchanged and two had deteriorated. There was no correlation among the histologic studies, the radiographic appearance, the postoperative varus-valgus angle or the clinical results. A similar study of fourteen patients found proliferation of a new fibrocartilaginous surface on the tibial condyle in eight patients and on the medial femoral condyle in nine patients two years following osteotomy⁵⁷. This study, also, did not find a correlation between regeneration of an articular surface and clinical outcome.

Long term follow up of patients treated with osteotomies for hip and knee osteoarthritis shows that the clinical results deteriorate with time^{40,62,74}. Reigstad and Gronmark evaluated 103 hips treated by intertrochanteric osteotomy⁶². One year following surgery 70 percent of the hips had a good result, at five years 51 percent had a good result and at ten years only 30 percent of the hips still showed a beneficial effect of the osteotomy. A study of ninety-five knees in eighty-five patients treated with tibial osteotomies showed that the percentage of patients with good or excellent results declined from 97 percent at two year follow up to 85 percent at five years, and that only 15 percent of the knees were pain free nine years or more following surgery⁴⁰. A similar study of thirty-nine knees in thirty-five patients showed that the percentage of patients with good results declined from 87 percent at two years to 57 percent at fifteen years following surgery⁷. Matthews et al. followed forty patients treated with knee osteotomies and found that 86 percent of the patients had useful function of the knee at one year, but this value pro-

gressively declined to 64 percent at three years, 50 percent at five years and 28 percent at nine years⁵⁰. Variables that appear to adversely affect the results of knee osteotomies include advanced patient age, obesity, severe joint degeneration, joint instability, limited joint motion, operative over-correction or under-correction and postoperative loss of correction^{7,27,40,50}. However, even patients who appear to be optimal candidates for osteotomy and who have a good initial surgical outcome tend to develop recurrent pain and evidence of advancing osteoarthritis with time.

Several studies indicate that the results of osteotomies could be improved through advances in technique and patient selection^{56,58,59}. Evaluation of preoperative joint mechanics may also lead to improved results. Surgeons generally use radiographs that demonstrate joint alignment, subchondral bone density and cartilage space to plan osteotomies that will redistribute articular surface loading. They base this practice on the assumption that static joint alignment can be used to predict loading in different regions of a joint. One group of investigators showed that dynamic joint loading also should be considered^{61,73}. They studied patients with knee osteoarthritis and varus deformity using gait analysis and found that the patients could be separated into two groups: those with high adduction moments at the knee and those with low adduction moments. The two groups did not differ in preoperative knee score, initial knee alignment, postoperative knee alignment, age or weight; but those with high preoperative adduction moments had only 50 percent good or excellent results at an average of 3.2 years following osteotomy compared with 100 percent good or excellent results for patients with low preoperative adduction moments⁶¹. With increasing time the results for both groups deteriorated, but the patients with low preoperative adduction moments maintained better clinical results⁷³.

At present the overall clinical results of hip and knee osteotomies vary more than those of joint replacement, and the relationships among the degree of alteration of joint loading, type of osteotomy, quality and extent of articular surface repair, radiographic changes and clinical outcome remain unclear. Given the available information, identifying the patients most likely to benefit from osteotomy, planning the optimal osteotomy for a specific joint and predicting the outcome of the procedure for an individual patient are difficult. A better understanding of the effects of altering joint alignment on the articular surface and possibly combining procedures designed to alter joint alignment with new methods of stimulating cartilage formation could improve the results of these procedures.

JOINT DISTRACTION AND MOTION

Clinical experience with pseudarthroses following fractures, surgical release of muscles that act across osteoarthritic joints and with some resection arthroplasties suggest that decreased contact pressure and motion of bone or articular surfaces promotes cartilage formation over the opposing surfaces^{19,21,22}. Aldegheri and colleagues used joint distraction that allowed motion to treat eighty patients with a variety of hip disorders¹. Twenty-four patients who either had inflammatory joint disease or were older than forty-five years had poor results, and only four patients over forty-five years of age had good results. Forty-two of fifty-nine patients younger than forty-five years with osteoarthritis, hip dysplasia, avascular necrosis and chondrolysis had good results. These results suggest that, at least in people less than forty-five years of age, decreased contact pressure and motion of damaged hip joint surfaces can decrease symptoms. A recent preliminary report describes encouraging results of joint distraction and motion as a treatment for patients with post-traumatic ankle osteoarthritis^{17,69}. van Valburg and colleagues treated advanced post-traumatic osteoarthritis of the ankle with joint distraction in eleven patients⁶⁹. After application of an Ilizarov device, the authors distracted the joints 0.5 mm per day for five days and then maintained the distraction of the articular surfaces throughout the course of treatment. Active joint motion was started between six and twelve weeks after surgery, and after twelve to twenty-two weeks the distraction device was removed. At an average of twenty months after treatment none of the patients had proceeded with an arthrodesis: all eleven patients had less pain, and five were pain free; six had more motion; and three of six that had radiographic studies had increased joint space. The authors concluded that distraction of an osteoarthritic ankle joint delays arthrodesis, and that it may stimulate repair of osteoarthritic cartilage.

SOFT TISSUE GRAFTS

Treatment of osteoarthritic joints by soft tissue grafts usually involves debriding the joint and interposing soft tissue grafts consisting of fascia, muscle, tendon, periosteum or perichondrium between debrided or resected articular surfaces²¹. The potential benefits of soft tissue grafts include introduction of a new cell population along with an organic matrix, a decrease in the probability of ankylosis before a new articular surface can form, and protection of the graft or host cells from excessive loading. The success of soft tissue arthroplasty depends not only on the severity of the joint abnormalities and the type of graft, but on postoperative motion to facilitate generation of a new articular surface.

Animal experiments and clinical experience show that perichondrial and periosteal grafts placed in articular cartilage defects can produce new cartilage²¹. Recently, O'Driscoll has described the use of periosteal grafts for the treatment of isolated chondral and osteochondral defects, and in preliminary evaluation of a small series of patients he has found good or excellent results in more than three quarters of the patients¹⁶. He has stressed the need for further development of this procedure, careful selection of patients and exacting surgical technique. Homminga et al. treated thirty chondral lesions of the knee in twenty patients with rib perichondrial grafts³⁵. The mean score on a scale designed to evaluate knee function improved significantly and arthroscopic examination showed that twenty-eight of the thirty chondral defects had filled almost completely with a tissue resembling articular cartilage. Engkvist and Johannson treated twenty-six patients with painful stiff small joints with rib perichondrial arthroplasty²⁸. Some individuals had improved motion and decreased pain, but a roughly equal number were not improved. Seradge et al. studied the results of rib perichondrial arthroplasties in sixteen metacarpophalangeal joints and twenty proximal interphalangeal joints at a minimum of three years following surgery⁶⁴. Patient age was directly related to the results. One hundred percent of the patients in their twenties and 75 percent of the patients in their thirties had good results following metacarpophalangeal joint arthroplasties. Seventy-five percent of the patients in their teens and 66 percent of the patients in their twenties had good results following proximal interphalangeal joint arthroplasties. None of the patients older than forty years had a good result with either type of arthroplasty. The authors concluded that perichondrial arthroplasty could be used for treatment of post-traumatic osteoarthritis of the metacarpophalangeal joint and proximal interphalangeal joints of the hand in young patients.

The clinical observation that perichondrial grafts produced the best results in younger patients⁶⁴ agrees with the concept that age may adversely affect the ability of undifferentiated cells or chondrocytes to form an articular surface, or that with age the population of cells that can form an articular surface declines²⁶. The age related differences in the ability of cells to form a new articular surface may also help explain some of the variability in the results of other procedures including osteotomies or procedures that penetrate subchondral bone; that is, younger people may have greater potential to produce a more effective articular surface when all other factors are equal.

GROWTH FACTORS

Growth factors influence a variety of cell activities including cell proliferation, migration, matrix synthesis and differentiation. Many of these factors, including the fibroblast growth factors, insulin like growth factors and transforming growth factor betas, have been shown to affect chondrocyte metabolism and chondrogenesis^{19,21}. Bone matrix contains a variety of these molecules including transforming growth factor betas, insulin like growth factors, bone morphogenic proteins, platelet derived growth factors and others^{19,20}. In addition, mesenchymal cells, endothelial cells and platelets produce many of these factors. Thus, osteochondral injuries and exposure of bone due to loss of articular cartilage may release these agents that affect the formation of cartilage repair tissue, and they probably have an important role in the formation of new articular surfaces after currently used surgical procedures, including resection arthroplasty, penetration of subchondral bone, soft tissue grafts and possibly osteotomies.

Local treatment of chondral or osteochondral defects with growth factors has the potential to stimulate restoration of an articular surface superior to that formed after penetration of subchondral bone alone, especially in joints with normal alignment and range of motion and with limited regions of cartilage damage. A recent experimental study of the treatment of partial thickness cartilage defects with enzymatic digestion of proteoglycans that inhibit adhesion of cells to articular cartilage followed by implantation of a fibrin matrix and timed release of TGF-Beta showed that this growth factor can stimulate cartilage repair^{37,38} (Figure 3). The cells that filled the chondral defects migrated into the defects from the synovium and formed a fibrous matrix. Despite the promise of this approach, the wide variety of growth factors, their multiple effects, the interactions among them, the possibility that the responsiveness of cells to growth factors may decline with age^{26,49,60} and the limited understanding of their effects in osteoarthritic joints make it difficult to develop a simple strategy for using these agents to treat patients with osteoarthritis. However, development of growth factor-based treatments for isolated chondral and osteochondral defects and early cartilage degenerative changes in younger people appear promising.

CELL TRANSPLANTATION

The limited ability of host cells to restore articular surfaces^{22,24} has led investigators to seek methods of transplanting cells that can form cartilage into chondral and osteochondral defects. Experimental work has shown that both chondrocytes and undifferentiated mesenchymal cells placed in articular cartilage defects

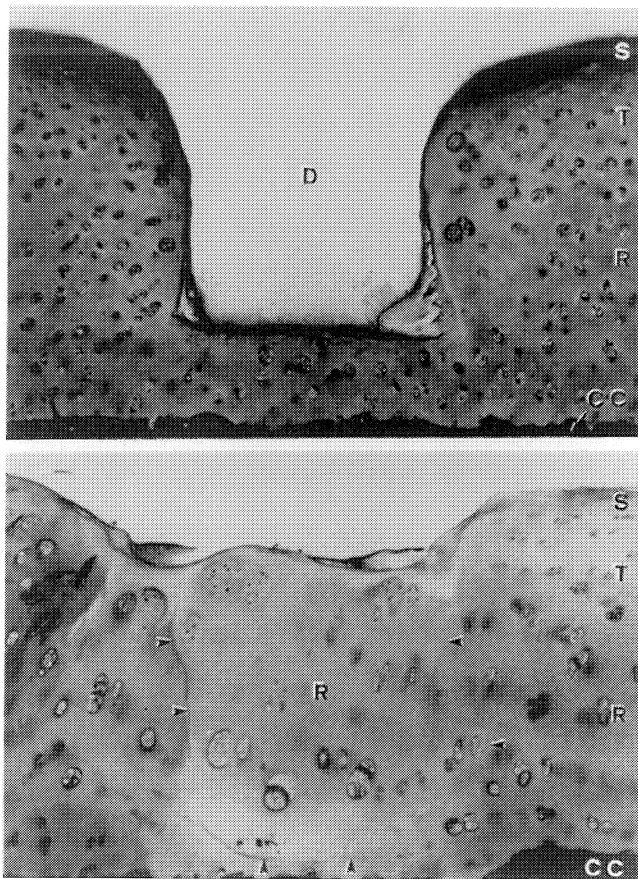


Figure 3. Photomicrographs of chondral defects in minipig articular cartilage. The upper photomicrograph shows an untreated articular surface chondral defect (D) and the lower photomicrograph shows new repair cartilage (R, edges marked by arrows) that formed in a chondral defect treated with free transforming growth factor-beta and liposome-encapsulated transforming growth factor-beta in a fibrin matrix. The letters S, T, R and CC mark the zones of articular cartilage (S = superficial, T = transitional, R = radial and CC = calcified cartilage). Supplied by Ernst Hunziker.

survive and produce a new cartilage matrix²¹. Wakitani and associates found that 80 percent of rabbit osteochondral defects treated with allograft articular chondrocytes embedded in collagen gels healed within twenty-four weeks⁷². Other investigators have reported similar results with chondrocyte transplantation^{41,42,55,63}. Recently, Brittberg and colleagues compared the results of treating chondral defects in rabbit patellar articular surfaces with periosteal grafts alone, carbon fiber scaffolds and periosteum, autologous chondrocytes and periosteum, and carbon fiber scaffolds and periosteum^{11,14}. They found that the addition of autologous chondrocytes improved the histologic quality and amount of repair tissue. Cultured mesenchymal stem cells also can repair large osteochondral defects^{70,71}. Within two weeks of transplantation they differentiate into chondrocytes and begin to produce a new articular surface.

In addition to these animal experiments with cell transplants, a group of investigators has reported using autologous chondrocyte transplants for treatment of localized cartilage defects of the femoral condyle or patella in twenty-three patients¹³. The investigators harvested chondrocytes from the patients, cultured the cells for fourteen to twenty-one days, and then injected them into the area of the defect and covered them with a flap of periosteum. At two or more years following chondrocyte transplantation, fourteen of sixteen patients with condylar defects and two of seven patients with patellar defects had good or excellent clinical results. Biopsies of the defect sites showed hyaline like cartilage in eleven of fifteen femoral and one of seven patellar defects. More recently this group of investigators has reported the results in a larger group of patients¹⁶. They found that at more than two years after treatment for chondral defects of the knee forty-seven of sixty-six patients had improved knee function. These results indicate that chondrocyte transplantation combined with a periosteal graft can promote restoration of an articular surface in humans, but more work is needed to assess the function and durability of the new tissue and determine if it improves joint function and delays or prevents joint degeneration, and if this approach will be beneficial in osteoarthritic joints.

ARTIFICIAL MATRICES

Treatment of chondral defects with growth factors or cell transplants requires a method of delivering, and in most instances at least temporarily stabilizing the growth factors or cells in the defect. For these reasons, the success of these approaches often depends on an artificial matrix. In addition, artificial matrices may allow, and in some instances stimulate ingrowth of host cells, matrix formation and binding of new cells and matrix to host tissue. Investigators have found that implants formed from a variety of biologic and non-biologic materials can facilitate restoration of an articular surface. These materials include: treated cartilage and bone matrices, collagens, collagens combined with hyaluronan, fibrin, carbon fiber, hydroxyapatite, porous polylactic acid, polytetrafluoroethylene, polyester and other synthetic polymers²¹. Lack of studies that directly compare different types of artificial matrices make it difficult to evaluate their relative merits, but the available reports show that this approach can contribute to restoration of an articular surface. For example, in animal experiments collagen gels have proven to be an effective way of implanting chondrocytes and mesenchymal stem cells, and fibrin has been used to implant and allow timed release of a growth factor³⁸. Treatment of osteochondral defects in rats and rabbits with carbon

fiber pads resulted in restoration of a smooth articular surface consisting of firm fibrous tissue that filled the pads⁵⁴. Use of the same approach to treat osteochondral defects of the knee in humans produced a satisfactory result in 77 percent of forty-seven patients evaluated clinically and arthroscopically three years after surgery⁵⁴. Brittberg and colleagues also studied the use of carbon fiber pads for treatment of articular surface defects¹². They found good or excellent results in 83 percent of thirty-six patients at an average of four years after treatment.

CONCLUSIONS

Great progress has been made in the understanding of articular cartilage formation, repair and regeneration since the landmark studies conducted by the Hunter brothers. Penetration of subchondral bone, osteotomy, decreased joint contact pressure combined with motion, periosteal and perichondrial transplants, cell transplants, and implantation of growth factors and artificial matrices all have shown promise for stimulating restoration of a damaged or diseased articular surface. Some of these procedures have been shown to decrease pain and improve joint function in selected patients. However, thus far none of these procedures have been shown to stimulate formation of an articular surface with the composition, structure, mechanical properties and durability of articular cartilage. Instead, the available clinical and experimental evidence indicates that future optimal methods of restoring articular surfaces will begin with a detailed analysis of the structural and functional abnormalities of the involved joint, and the patient's expectations for future joint use. Based on this analysis the surgeon will develop a treatment plan that potentially combines correction of mechanical abnormalities (including malalignment, instability and intra-articular causes of mechanical dysfunction), debridement that may or may not include limited penetration of subchondral bone, and application of growth factors or implants that may consist of a synthetic matrix that incorporates cells or growth factors followed by a post-operative course of controlled loading and motion. Thus far the accumulated evidence suggests that even under optimal conditions this approach will not restore a normal articular surface, but it may produce tissue that performs well enough to decrease symptoms and improve joint function and thus repair the articular cartilage.

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FRACTURES OF THE RADIUS AND ULNA IN ADULTS: AN ANALYSIS OF FACTORS AFFECTING OUTCOME

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ABSTRACT

Concurrent data were collected by the authors for 104 fractures of the shafts of the radius and ulna in 102 adult patients to determine the relationship of subjective, objective, radiographic and economic outcome parameters to the method of treatment, type of fracture (open or closed), degree of comminution, and the presence of other injuries. Patients treated by open reduction and internal fixation (ORIF) had less pain, lost less forearm rotation, and returned to the same work following injury more frequently than those treated by closed reduction and casting (CR) or pins-in-plaster (PIP). The greatest advantages of ORIF over other treatment methods were improved skeletal alignment and forearm rotation, the factors most often associated with return to the same work following injury. Except for a longer time to union and a higher rate of infection, the outcomes of open and closed fractures were very similar. The presence of other injuries was a strong predictor of a compromised end result, primarily because of more pain, greater loss of forearm rotation, and less frequent return to the same work. The inclusion of patient satisfaction and work status in the assessment of outcomes and the concept of "functional malunion", an outcome-based interpretation of a radiographic finding, should help in counselling patients as to the likely economic and functional impacts of these injuries.

INTRODUCTION

Union with restoration of normal anatomy is particularly critical to achieve an optimal outcome for diaphy-

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seal fractures of the shafts of the radius and ulna in adults. These goals have most often been met by open reduction and plate fixation^{1-3,5,8,10,13,14,21,24}. In previous studies, however, outcome measures other than union have received scant attention, and the inclusion of fractures of a single bone with fractures of both bones has made interpretation of results difficult.

The purpose of this study was to determine the relationship of outcome to the method of treatment, type of fracture (open or closed), and presence of associated injuries in adults who sustained fractures of the shafts of both bones of the forearm. The outcome measures investigated were patient satisfaction (amount of pain), forearm rotation, radiographic findings, and work status.

MATERIALS AND METHODS

Criteria for inclusion in this study were skeletally mature patients with fractures of the shafts of both the radius and ulna treated at the University of North Carolina Hospitals. All patients were evaluated by one of the authors (thirty-five patients) or by another attending orthopaedist at the University of North Carolina Hospitals (sixty-seven patients). Complete data were available for 102 patients who had sustained 104 diaphyseal fractures of both the radius and ulna. Data collection and radiographic measurements were standardized for all patients.

All patients were followed at least until bone union occurred or the diagnosis of nonunion was made. The mean follow-up was thirty months (range three to 300 months). Seventy-three patients were male and twenty-nine female, with an average age of twenty-nine years (range fifteen to seventy-nine years). In thirty-seven patients, the fracture involved the dominant limb. Thirty-five of the fractures were open and sixty-nine were closed. The grade of soft tissue injury associated with open fractures was not recorded since many of these injuries preceded the advent of the rating system of Gustillo and Anderson⁸. Forty-six patients had sustained other major musculoskeletal or multi-system injuries.

Three methods of treatment were utilized: open reduction and internal fixation (ORIF), closed reduction

Table 1
Method of Rating End Results

Rating	Subjective	Objective	Radiographic	Economic
4	No pain.	Combined loss of forearm rotation <30°.	Fractures united. Combined malalignment (radius and ulna) <20°.	Same job at pre-injury level of performance.
3	Mild pain, present only with overuse.	Combined loss of forearm rotation 31-60°.	Union, with combined malalignment 21-40°.	Same job, but cannot perform at pre-injury level.
2	Moderate pain, present with routine activities.	Combined loss of forearm rotation 61-90°.	Union, with combined malalignment >40°.	Different job necessitated by fractures.
1	Severe pain, prevents or modifies routine activities.	Combined loss of forearm rotation >90°.	Nonunion, synostosis, or osteomyelitis.	Unable to work because of fractures.

and casting (CR), and external skeletal fixation with pins-in-plaster (PIP). The method of treatment was chosen by the attending surgeon based upon his experience and the type of injury. Minimal displacement of a closed fracture was the most frequent indication for closed reduction, and marked comminution was the primary reason for treatment with pins-in-plaster. All reductions were performed under general anesthesia. The definitive treatment was ORIF in seventy-three forearms, CR in eighteen, and PIP in thirteen. Twenty-one patients in the ORIF group were initially treated unsuccessfully by other methods (eighteen by CR and three by PIP).

Union was defined as the presence of bridging bone or trabeculae spanning the fracture site. Nonunion was identified by the absence of union within twenty-eight weeks following injury. Standards for alignment and measurement of radiographs were based on Sage's study, which defined normal as nine degrees of radial and six degrees of dorsal bowing of the radius and zero degrees in both planes for the ulna¹⁵.

End result ratings were made on a 1-4 point scale in four categories: (a) subjective, according the level of pain in the injured limb; (b) objective, by the range of forearm rotation; (c) radiographic, utilizing the criteria of union, synostosis, and malunion; and (d) economic, as reflected by the impact of the injury on the patient's employment status (Table 1).

Statistical analysis of the data was performed using the two-sided Fishers' exact test to analyze the association of two non-ordinal categorical variables. To analyze the association of a continuous ordinal variable and a categorical variable, the Kruskal-Wallis Test was used.

Statistical significance was defined as $p < 0.05$. Values for p were calculated for each association tested; numerical values of p for associations that did not reach statistical significance were reported only for selected associations.

RESULTS

Subjective Outcomes

Overall, 77 percent of patients reported no pain, with no difference between patients with open and those with closed fractures. While 82 percent of patients treated with ORIF were pain free at their last examination, only 62 percent treated with CR and 54 percent treated with PIP were painless. Patients with isolated fractures were more often pain free than were those with associated injuries (Table 2).

Objective Outcomes

No patient had significant loss of wrist or elbow motion compared to the uninjured side. The average total decrease in forearm rotation, however, was twenty-nine degrees, with loss of slightly more supination than pronation. There was no significant difference in the loss of forearm rotation between closed and open fractures: 63 percent of each group lost less than thirty degrees of forearm rotation.

The method of treatment had a significant effect on the loss of forearm rotation. Seventy-three percent of patients treated with ORIF lost less than thirty degrees of forearm rotation, while only 50 percent treated by CR and 23 percent by PIP lost less than thirty degrees. Patients with multiple injuries lost more forearm rotation than did those with isolated fractures (Table 3).

Table 2
Subjective Outcomes
 (Percent of patients achieving each subjective rating)

Rating	Overall	Open Fractures	Closed Fractures	ORIF	CR	PIP	Multiple Injuries	Isolated Fractures
4	77	77	77	82	62	54	72	83
3	19	20	19	15	32	46	26	14
2	4	3	4	3	6	0	2	3
1	0	0	0	0	0	0	0	0

Table 3
Objective Outcomes
 (Percent of patients achieving each objective rating)

Rating	Overall	Open Fractures	Closed Fractures	ORIF	CR	PIP	Multiple Injuries	Isolated Fractures
4	63	63	62	73	50	23	52	71
3	13	11	13	8	17	31	17	9
2	9	12	9	8	22	0	9	10
1	15	14	16	11	11	46	22	10

Table 4
Effect of Malalignment on
Loss of Forearm Rotation

Radiographic Outcomes

Union occurred in 93 percent of radius fractures and 97 percent of ulna fractures, with an average time to union of 17.7 weeks for the radius and 18.3 weeks for the ulna.

Union was more frequent after closed than after open fractures. This difference was most apparent in radius fractures where 11 percent of open fractures developed nonunions, compared to only 4 percent of closed injuries ($p = 0.171$). Also, the average time to union was 18 percent longer for open than for closed fractures of the radius ($p = 0.027$), and 32 percent longer for open fractures of the ulna ($p = 0.012$). Neither the frequency of nor the time to union varied significantly with the method of treatment.

The amount of forearm rotation lost was directly proportional to the loss of normal alignment, reaching a mean of forty-three degrees when the combined malalignment of the radius and ulna exceeded thirty degrees ($p = 0.06$) (Table 4).

n	Combined Malalignment (radius and ulna)	Mean Loss of Forearm Rotation
43	0-15°	21°
41	16-30°	31°
18	> 30°	43°

Overall, 66 percent of patients had less than twenty degrees combined malalignment of the radius and ulna on the final radiographs, with no difference between those patients with open and those with closed fractures. The method of treatment, however, had a significant effect on the final radiographic alignment: 81 percent of patients treated with ORIF had less than twenty-degrees combined malalignment of the radius and ulna on the final radiographs, a result seen in only 50 percent and 8 percent of patients treated with CR and PIP respectively (Table 5).

Table 5
Radiographic Outcomes
 (Percent of patients achieving each radiographic rating)

Rating	Overall	Open Fractures	Closed Fractures	ORIF	CR	PIP	Multiple Injuries	Isolated Fractures
4	66	66	67	81	50	8	52	79
3	13	14	11	8	17	31	17	7
2	11	9	13	4	11	54	18	7
1	10	11	9	7	22	7	13	7

Table 6
Economic Outcomes
 (Percent of patients achieving each economic rating)

Rating	Overall	Open Fractures	Closed Fractures	ORIF	CR	PIP	Multiple Injuries	Isolated Fractures
4	87	94	83	95	67	69	85	88
3	6	3	7	3	17	8	6	5
2	7	3	10	2	16	23	9	7
1	0	0	0	0	0	0	0	0

Economic Outcomes

Ninety-seven percent of patients with open fractures and 90 percent of those with closed fractures returned to the same work after injury. This difference may be attributed to the fact that nearly all patients with open fractures were treated by ORIF, while many with closed fractures were treated with CR or PIP. Patients treated with ORIF returned to the same work following injury more frequently than those treated with PIP or CR ($p < 0.05$). The presence of other injuries had little effect on the ability of the patients to return to the same work following injury (Table 6).

Pain also appeared to influence the patients' ability to return to the same work following injury; however, the strongest correlation was with the amount of forearm rotation lost. Eighty-eight percent of patients who did not return to the same work lost at least sixty-one degrees of forearm rotation; only 21 percent of patients who returned to the same work lost this much rotation.

COMPLICATIONS

Infection: Four deep infections developed (three following open fractures), for an overall infection rate of 4

percent. The infection rate was 1.5 percent in closed fractures and 9 percent in open fractures. No infections occurred in open fractures treated by immediate ORIF. All infections resolved with surgical debridement and appropriate antibiotic therapy.

Nerve palsy: Twelve patients (11 percent) had nerve palsies; eight radial, three median, and one ulnar. Ten palsies were recognized prior to treatment. The frequency of nerve palsies was similar in patients with open fractures (12 percent) and those with closed fractures (11 percent). All resolved spontaneously within six months of injury.

Synostosis: Radioulnar synostoses occurred in seven patients, all with closed fractures. Five of the patients also had closed head injuries; two had isolated forearm fractures. Three patients had been treated with CR, one with PIP, and three with ORIF.

Loss of reduction: Loss of fracture alignment required conversion to another method of treatment in twenty-one patients after primary treatment by CR or PIP. However, no patient lost reduction after ORIF. Eighteen of thirty-six patients treated initially by CR, and three of sixteen treated by PIP lost reduction and required conversion to ORIF. No nonunions occurred in these patients.

DISCUSSION

Full rotation of the forearm following fractures of the diaphyses of the adult radius and ulna is infrequent because of the difficulty in obtaining and maintaining anatomical reduction. Although numerous methods of treatment have been described for these injuries^{1-4,9-11,15,16,19,20,22}, the results are difficult to analyze because of many fracture and treatment variables, lack of precise definitions, and pooling of results for fractures of both bones with those in which only one bone was fractured.

Other studies have reported rates of nonunion, malunion, and other complications comparable to those in this investigation^{1,3,5,6,13,14,16,18,22,24}. The present study adds outcome measures based on the patients' impressions of their results and their ability to return to work following injury. Hadden et al.⁸ reported on 109 patients with fractures of the forearm, sixty-four of whom had fractures of both bones of the forearm; however, the outcome results were combined for all patients and were not stratified by the bone fractured, whether the fracture was open or closed, or the method of treatment. Fifty-five percent of patients with united fractures were pain free, 91 percent returned to the same occupation, and 3 percent were unable to work because of their forearm fracture. By comparison, 77 percent of patients in this study (82 percent of those treated with ORIF) were pain free at the time of their last evaluation. No patient in this series was unable to work because of his/her forearm fracture, and 93 percent of all patients (98 percent of those treated with ORIF) returned to the same work following injury. The inclusion of patient satisfaction and work status in the assessment of outcomes supplies information about the long term results of these fractures not previously available and permits counseling of patients as to the economic implications of their injuries.

While some authors have stated that closed methods of treatment for displaced diaphyseal fractures of the radius, ulna, or both forearm bones produce unacceptable results^{1,3,13}, Sarmiento et al.¹⁶ reported excellent functional results after closed treatment in forty-three patients. Although ORIF improved the overall outcomes in our study, it is clear that the greatest advantage of ORIF over other methods of treatment was in minimizing malalignment of the forearm and the resulting loss of forearm rotation. The rotation lost following CR and PIP was nearly double that lost following ORIF. Correspondingly, almost 90 percent of patients treated with ORIF had less than forty degrees of angular malalignment, but only 67 percent of patients treated with CR and 39 percent of patients treated with PIP achieved this result. Although alignment of the radius

and ulna has been measured by various methods^{1,11,15,17,18,23}, all studies, including this one, have shown that loss of normal alignment of the radius and ulna closely correlates with loss of pronation and supination^{1,11,17,18,23}. Angular malalignment and the related loss of forearm rotation were the factors in this study most often associated with inability to return to the same work following injury. Although malalignment is measured radiographically, it is a major determinant of function following fractures of the forearm. The term "functional malunion" describes the upper limit of angular malalignment that was associated with return to the same work following injury. Patients in this study who had combined angular malalignment of the radius and ulna of less than forty degrees were limited in forearm rotation by no more than sixty degrees and usually returned to the same occupation. The rationale for defining malunion in terms of function is to provide an outcome-based application of a radiographic finding.

Except for a longer time to union and a higher infection rate, the results of treatment for open and closed fractures were very similar. The infection rate in this study was comparable to that reported by others^{3,6,13,14,19,21}. The incidence of transient nerve palsies was unaffected by the presence of an open injury, although we expected more frequent nerve injury following open fractures because of more extensive soft tissue injuries.

The 44 percent of patients in this series who sustained multiple trauma is similar to the 40 percent incidence reported by Chapman et al.³. Patients in this series with other injuries lost more forearm rotation, and therefore had poorer end result ratings, than patients with isolated forearm fractures. The greater loss of forearm rotation resulted largely from more frequent synostoses in polytraumatized patients—(11.1 percent) compared to those patients with isolated fractures (3.7 percent). Interestingly, all five synostoses in patients with multiple trauma occurred in the setting of closed head injuries. The formation of ectopic bone following forearm fractures in patients with closed head injuries has been well documented^{1,7,12,24,25}.

SUMMARY

For this series of 102 adult patients, the end results following treatment of fractures of the shafts of the radius and ulna were good to excellent regardless of the method of treatment chosen. Except for a longer time to union and a higher infection rate, the outcomes of open and closed fractures were very similar. The presence of associated injuries was a strong predictor of a compromised end result. These patients had more pain, greater loss of forearm rotation, and longer times to

union. Treatment with ORIF resulted in better outcomes than treatment with either CR or PIP, largely because ORIF minimized malalignment and the resulting loss of forearm rotation. These two factors were closely associated with the inability to return to the same work following injury.

The addition of patient satisfaction and work status to the assessment of outcomes following fractures of the shafts of the radius and ulna in adults supplies previously unavailable information about the long term results of these injuries. The concept of "functional malunion" provides an outcome-based interpretation of a radiographic finding that more closely associates the radiographic alignment of the forearm with expected functional limitations.

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COMPARING MERSILENE* TAPE AND STAINLESS STEEL WIRE AS SUBLAMINAR SPINAL FIXATION IN THE CHAGMA BABOON (PAPIO URSINUS)

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ABSTRACT

The development of segmental instrumentation has been a major advancement in the treatment of spinal problems, but the use of sublaminar stainless steel wire (SSW) has not been without untoward effects. This study reports a comparison of Mersilene* tape (MT) and stainless steel wire (SSW) used for sublaminar fixation in the Chagma baboon (*Papio Ursinus*). A similar comparative study has not been reported, although the local effects of sublaminar SSW in the spinal canal have previously been described^{12,17}. The adult Chagma baboon was selected as the experimental animal due to its partial upright posture and spinal anatomy, similar to that of the human. Six levels of the thoracolumbar spine were instrumented with custom designed Harrington hooks and regular one-quarter inch threaded rods used as a distraction system. The four intervening laminae were fixed to the rods using doubled-over, eighteen gauge sublaminar SSW in six cases and five millimeter MT in six cases.

Computed axial tomography used to measure the AP diameter of the bony spinal canal revealed the AP space occupied by the SSW and MT to be

32 percent and 14.8 percent respectively.

In the MT group, the overlying dura mater was found to be totally intact and revealed no signs of abnormal tissue response. A well-formed connective tissue membrane consisting of dense connective tissue surrounded the MT and was found to consist of more mature fibers than that found in the SSW group.

The dura-implant interface was examined histologically and a distinct membrane was identified between the dura and the superficial aspect of the MT's, as well as intervening between the two MT's. Following removal of the MT, in contrast to the SSW, it was apparent that the underlying dura was not injured, most probably due to the soft consistency of the Mersilene* tape and the well-formed overlying membrane.

On clinical grounds the fixation in both groups was adequate but the MT group formed a well-circumscribed membrane that made removal of the MT easier and potentially safer. The AP space occupied by the spinal implant was also found to be less with MT as opposed to SSW.

INTRODUCTION

Segmental sublaminar instrumentation has been a major advancement in the treatment of spinal problems^{10,11}. The use of stainless steel wire (SSW) has been associated with complications including wire breakage and neurological damage ranging from permanent to transient deficits^{1,3,6, 8,9,15,18,20}. Neurological deficits seem to occur more often at the time of insertion and during the period before securing the stainless steel wire-rod construct. The protruding wires (porcupine effect) can inadvertently be jarred by the surgeon or assistant, resulting in spinal cord and/or nerve injury (Figure 1).

Experimental studies in dogs¹⁷ and adult canines⁵ reported the effects of sublaminar wires and contributed to the concern regarding the presence of wires in the spinal canal.

O'Brien reported a preliminary study using nylon straps in sublaminar fixation but recent follow-up on this

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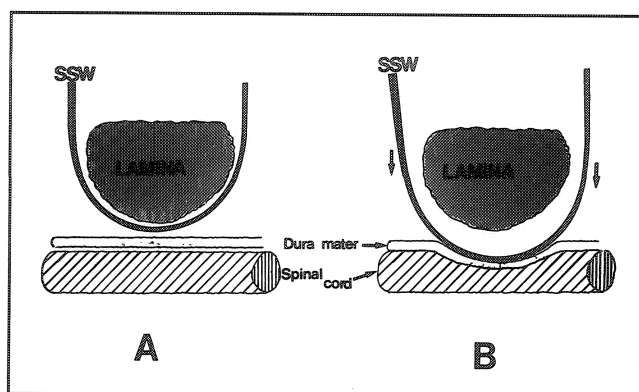


Figure 1. (A) Schematic presentation of sublaminar wires (SSW) in position. (B) Potential for jarring of the spinal contents during the surgical procedure (porcupine effect).

method is unavailable¹³. Gaines reported on the use of Mersilene* tape (MT) in young children with osteopenia to supplement Harrington or Luque rods⁴.

Due to the problems reported with the use of SSW and the subsequent experience gained with MT, we decided to compare and examine the local effects of MT and SSW in the Chagma baboon (*Papio Ursinus*). The adult Chagma baboon was selected as its partial upright posture and spinal anatomy are similar to the human. The local effects of sublaminar wires on the bone and adjoining dura has not been described, and no study to our knowledge has been reported using MT and SSW under similar circumstances in this animal model.

Mersilene* polyester fiber suture (ETHICON INC.) was supplied as five millimeter wide, white tape. Double sublaminar wire fixation (eighteen gauge) was used in the control group.

The goal of this investigation was to:

1. Evaluate MT as a possible substitute for SSW for sublaminar fixation on the basis of an in vivo model, the Chagma baboon.
2. Record the radiological and histological findings of MT and SSW in this experimental animal.
3. Formulate guidelines for suggested clinical use of MT in spinal surgery.
4. To assess the appropriateness of this animal model (*Papio Ursinus*) in evaluating posterior instrumentation procedures.

MATERIALS AND METHODS

Twelve adult animals, average weight 21.8 kg (range 19.0 to 25.0) were anesthetized with sodium pentobarbital (3.0 mg/kg body weight) by IV injection following pre-medication with ketamine (10.0 mg/kg body weight) intramuscularly (Table 1). Through a posterior midline incision, six vertebral levels proximal to the iliac crest were exposed. These levels were instrumented with custom-designed Harrington hooks and regular one-quarter inch threaded rods used as a distraction system. The four intervening laminae were fixed to the rods using either eighteen gauge, double SSW in six cases

TABLE 1
DATA ON THE ANIMALS

Baboon No.	Type of Fixation	Weight (KG) *1	Sacrificed (Weeks) *2
1	MT	24.5	19.4
2	MT	21.5	16.2
3	MT	20.0	22.4
4	MT	22.0	21.8
5	MT	22.2	23.4
6	MT	19.0	22.5
7	SSW	21.5	19.7
8	SSW	22.0	19.2
9	SSW	22.0	20.0
10	SSW	23.5	20.1
11	SSW	25.0	20.5
12	SSW	19.0	21.0

*1 Mersilene Tape (MT) Group 21.5 KG (Range 19.0-24.5) Stainless Steel Wire (SSW) Group 22.2 KG (Range 19.0-25.0)

*2 Mersilene Tape (MT) Group 21 Weeks (Range 16.2-23.4) Stainless Steel Wire (SSW) Group 20.1 Weeks (Range 19.2-21.0)

Figure 2. (A) Model showing placement of the distraction rod system and sublaminar stainless steel wires at the four (4) intervening levels. (B) Identical placement at the four (4) intervening levels using sublaminar Mersilene* tape.

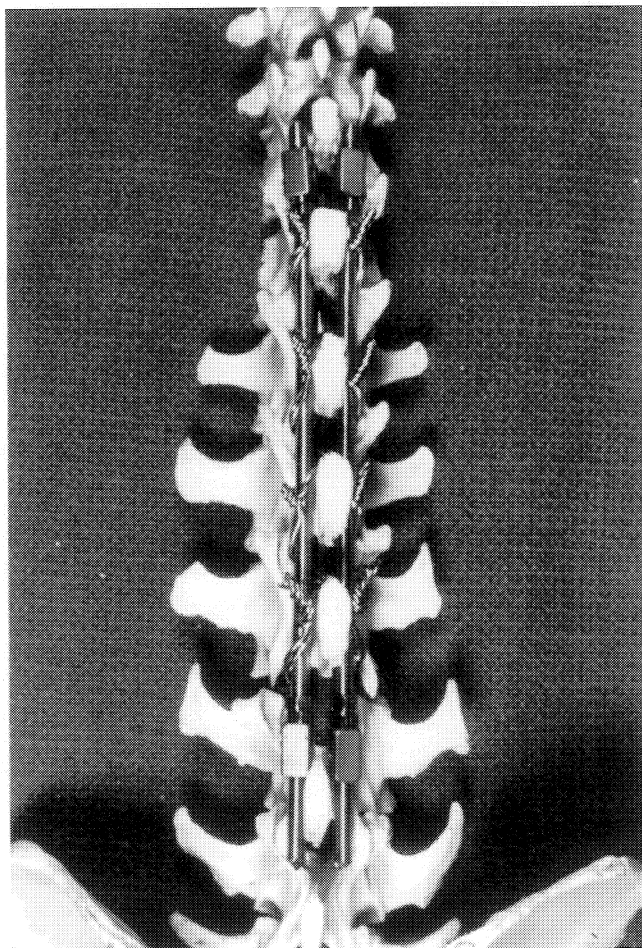


Figure 2A.

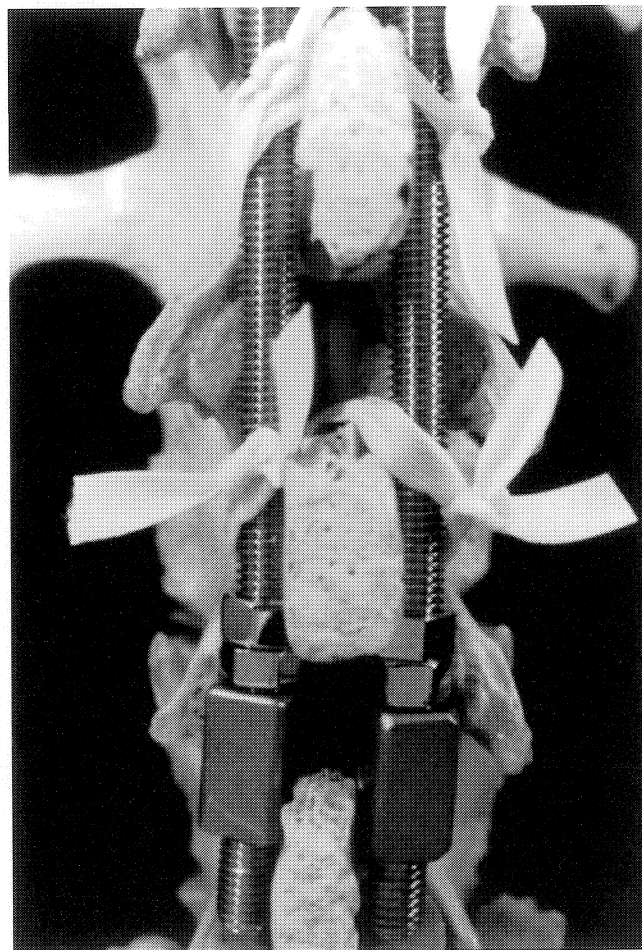


Figure 2B.

or five millimeter MT in the remaining six cases (Figures 2A & B). A customized tension device was used to tighten the tapes.

Prophylactic antibiotics were given in divided doses at the beginning and end of the procedure. Postoperative recovery was uneventful in all animals. No orthosis was used. The animals were housed in cages (size 100 x 95 x 95 cms) for an average of ten days and, thereafter, transferred to larger cages (size 2.5 x 2.5 x 6.5 meters) that allowed unrestricted activity. The animals were sacrificed at an average of 20.5 weeks (range 16.2 to 23.4) postoperatively (Table 1).

After sacrifice, the following assessments were performed:

- A. Gross macroscopic evaluation;
- B. Radiographic evaluation;
- C. Histologic evaluation.

A. Gross Macroscopic Evaluation

Following sacrifice by means of exsanguination under general anesthesia, the spinal columns were carefully removed en-bloc including one vertebra above and one below the instrumented area. The soft tissue was removed and macroscopic assessment of the MT, SSW, and the surrounding tissue was performed. A high speed saw was used to cut the rods and spine at the level of the disk space, keeping the segmental rod, MT and SSW system intact at each level (Figure 3A). The vertebral bodies and spinal cords were then removed exposing the anterior aspects of the laminae while leaving the dura mater intact. The MT and SSW could be visualized clearly and photographs were taken of the intra-spinal appearance of the MT and SSW with the posterior dura mater left intact (Figures 3B, C).

Figure 3. (A) Axial view of a single vertebra shown with rods and sublaminar Mersilene* tapes in position. (B,C) After removal of the vertebral bodies, the anterior aspects of the laminae were assessed. The posterior dura over the Mersilene* tape (B) can clearly be seen to be smooth in contrast to the more prominent SSW underlying the dura (C).

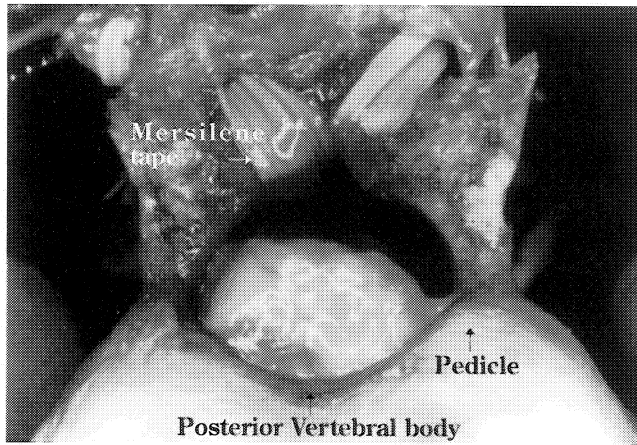


Figure 3A.

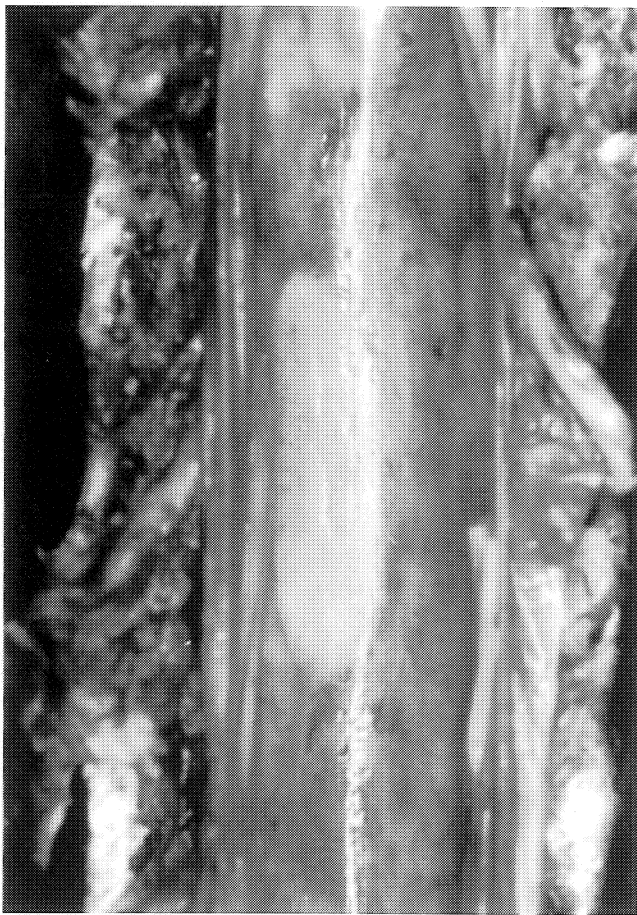


Figure 3B.

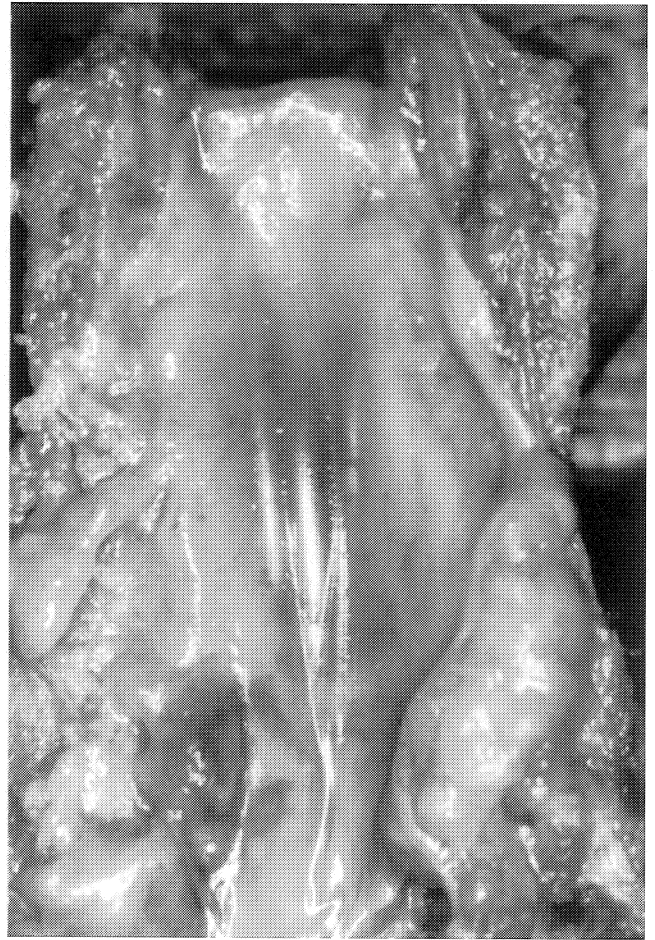


Figure 3C.

B. Radiographic Evaluation

Anteroposterior and lateral radiographs were periodically taken after surgery and at the time of sacrifice (Figures 4A, B). Following removal of the spines, computed tomography (CT) scans were done on eleven cases (Figure 4C). Axial images were obtained of individual vertebrae using a high resolution film. Calculation of the intra-spinal space occupied by the MT and SSW in the anteroposterior plane was also done (Figures 4D, E). The anteroposterior spinal occupancy ratio was calculated. For the first animal sacrificed (case number 2), the vertebral bodies were initially removed in an attempt to obtain a clear view of the intra-spinal area and, therefore, no CT scans or axial radiographs were done for this animal. For ease of identification, the levels of sublaminar fixation were numbered from one to four (cranial to caudal).

C. Histologic Evaluation

The four intervening segments were individually fixed using a "wax cube technique" to ease handling and cutting of the specimens. Using a thin band saw, the vertebrae at levels one and two were cut in the axial plane, and vertebrae three and four in the sagittal plane. Following removal of the wax and decalcification of the specimens, hematoxylin and eosin staining was done. In selected cases, different staining procedures (Masson's trichrome and Picrosirius-polarization technique) were used to assess the fibrous tissue reaction and osteoid reaction in the vicinity of, and below the MT and SSW. The tissue reaction to the presence of MT and SSW was evaluated at the following locations:

- a. posteriorly in the vicinity of the rods, wires and tapes;
- b. adjacent to the dura mater;
- c. around the MT and SSW;
- d. the cortical and sub-cortical bone underneath the MT and SSW wires.

RESULTS

The animals had an uneventful post-operative course, resuming their semi-upright posture within twenty-four hours. Primary healing of all wounds occurred.

Following sacrifice, gross dissection revealed no evidence of local infection. In one case (Number 6 MT), the distal Harrington hook was found to be dislodged on follow-up x-rays and will be more fully discussed.

A. Gross Macroscopic Dissection Studies

1. Posterior aspect:

At the time of initial surgery, the MT was tied using three knots (surgical knots) leaving one centimeter loose ends. The MT knots and length of the loose ends were unchanged, indicating that no slippage occurred. Normal fibrous tissue reaction was found around both the SSW and MT.

2. Intra-spinal canal appearance:

The specimens were cut into four separate vertebral units including the corresponding rod and sublaminar fixation. The spinal cord and vertebral body were removed and the dura left intact. The appearance of the MT and SSW covered by the dura as observed from the spinal surface of the laminae was assessed. Despite sagittal radiographs showing the SSW tight against the inner aspects of the laminae, the prominence and indentation of the dura by the SSW seen on direct vision was disturbing and the dura also appeared very thin and attenuated (Figure 3C). Despite thinning of the dura, at no level did the SSW penetrate the dura. The dura overlying the MT was smooth in appearance, showing no signs of injury, prominence or penetration (Figure 3B).

3. Removal of MT and SSW:

The effects of removal of the MT by a technique similar to that used in the clinical situation was assessed. Sequential photographs were taken while removing the MT, noting the tracks taken by the ends of the MT. It could clearly be seen that there was a distinct membrane between the dura and the superficial aspect of the MT, as was the case between the two MTs. During removal the terminal edge of the MT had a minimal direct influence on the dura mater, and due to its soft consistency, did not seem to produce any significant pressure on the membrane. It was apparent that the underlying dura was not injured, likely due to the soft consistency of the tape and the well-formed membrane around the MT. The same procedure was adopted to remove the SSW, but with less satisfactory results; a distinct indentation and injury to the dura mater was noted.

B. Radiographic Evaluation

Following surgery, anteroposterior and lateral radiographs of the instrumented spines were taken and verified good placement of both hooks and rods. All wires at the four intervening levels in each animal were intact. Following sacrifice, the hook and rod placement was radiologically satisfactory except in the animal number 6 MT in which the distal hook on the left side was found to be dislodged. The dislodgement led to interesting observations on the axial images of the bony canal. The placement and status of the MT's were obviously not visible on the plain radiographs.

The precise anteroposterior diameter of the bony spinal canal and the space occupied by the SSW was assessed by computed axial tomography (CT) in eleven animals. Sections were done at the summit of the wire loop in the SSW group and in line with the laminae in the MT group at all four intervening levels (Figure 4C). In the SSW group, small punched out areas at the wire-bone interfaces were noted. No failure of the SSW-implant construct occurred. In animal number 6 (MT), there was a failure of instrumentation and excessive pressure was, therefore, exerted on the adjacent proximal MT until failure of the tape occurred at level four (Figure 5A). At levels one and two (both tapes intact), minimal bone loss underlying the tape was observed. At level three, the tape was under tremendous tension due to dislodgement of the hook and tape breakage at level four, resulting in increased bone erosion (Figure 5B). At level four, the tape area showed less prominent bone loss, indicating the tape probably broke soon after dislodgement of the hook (Figure 5C).

The intra-spinal anteroposterior diameters of the intervening four vertebrae were calculated using CT scan

Comparing Mersilene Tape and Stainless Steel Wire as Sublaminar Spinal Fixation in the Chagma Baboon*

Figure 4. (A) Anteroposterior and lateral x-ray views of the instrumentation plus stainless steel wire (SSW) in place. (B) Anteroposterior and lateral x-ray views of the instrumentation plus Mersilene* tape (MT) in place. (C) Computed tomography sections were done at the summit of each stainless steel wire placement. (D) High resolution axial view taken to assess and calculate the spinal canal space occupied by SSW (or MT) referred to as "AP spinal canal occupancy ratio". (E) Schematic presentation showing the measurements used to calculate the AP spinal canal occupancy ratio.

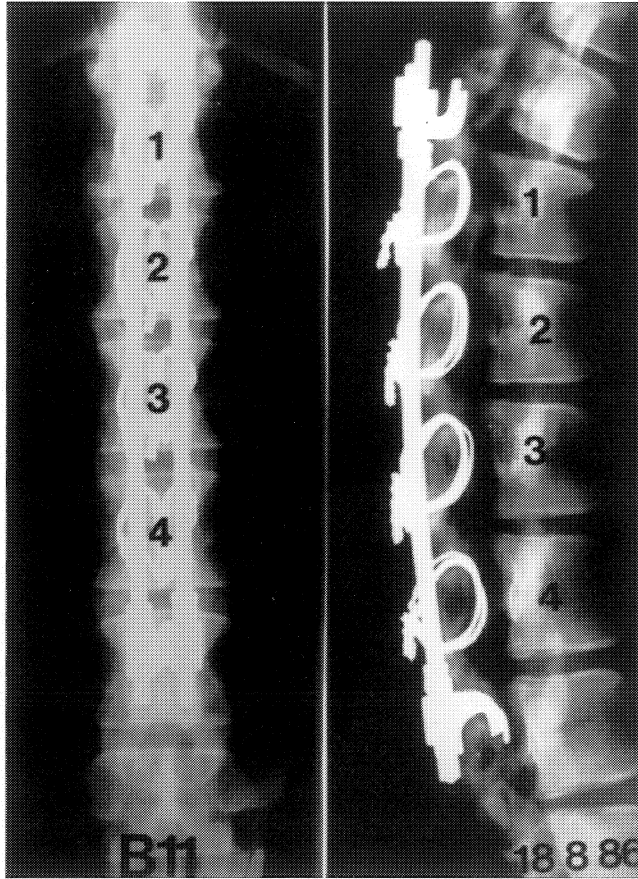


Figure 4A.

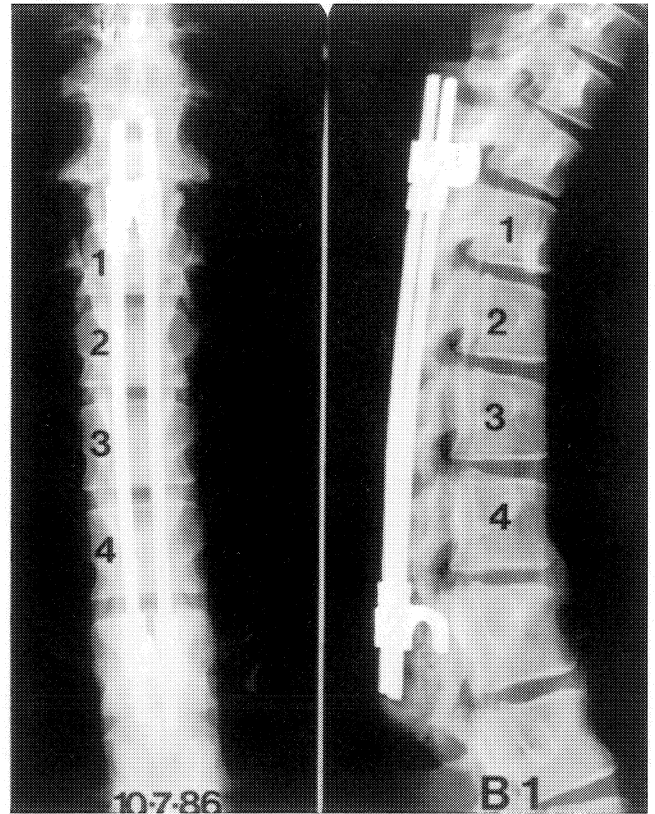


Figure 4B.

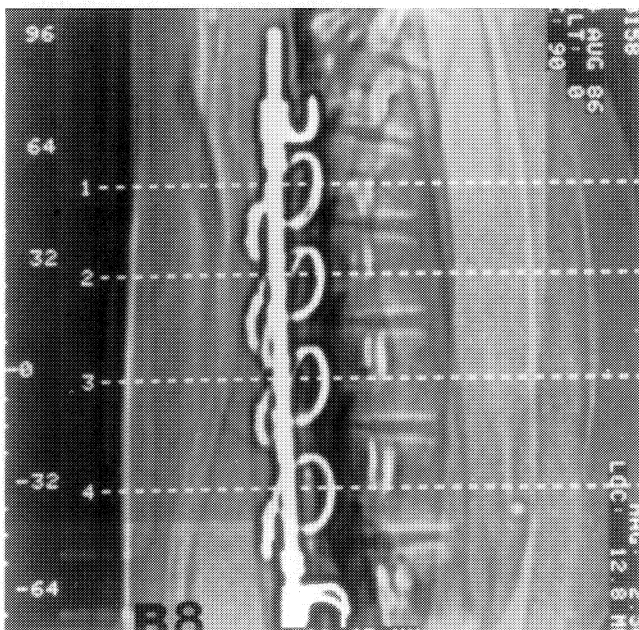


Figure 4C.

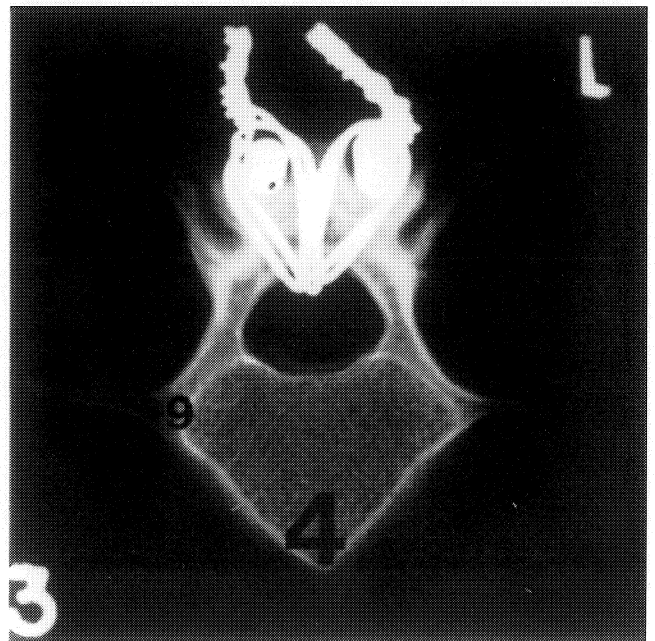


Figure 4D.

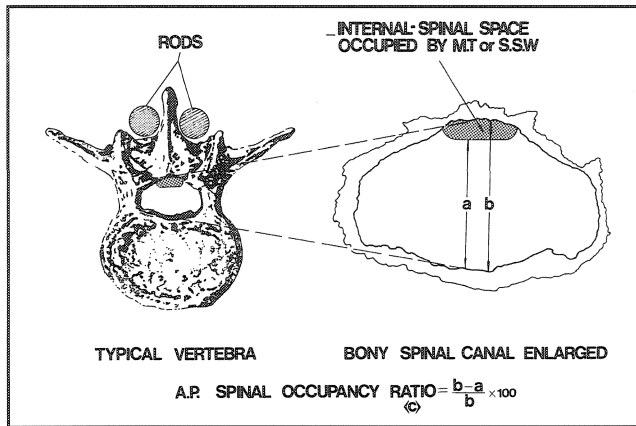


Figure 4E.

measurements or direct measurements on the high resolution views (Table 2). An “anteroposterior (AP) spinal occupancy ratio” was calculated using the measurements of the anteroposterior bony canal and the remaining part of the bony canal not occupied by the SSW or MT (Figure 4E). The percentage occupied by the MT or SSW was obtained at eighteen vertebral levels in the MT group, and twenty-four in the SSW group. The comparative measurements revealed a definite larger space (a factor of 2.1) occupied by the SSW as compared to the MT (Table 3). MT measurements averaged 14.8 percent (Range 11-19 per cent) and SSW averaged 32 percent (Range 21-47 percent).

C. Histologic Evaluation

The axial section taken in both groups revealed the most informative findings and will form the basis for these observations:

1. Dura Mater

In the MT group, the overlying dura mater was totally intact and revealed no signs of disruption or injury (Figures 6A, B). No abnormal tissue response, thinning or abnormal vascularity of the dura was found, and the dural edge in contact with the MT showed minimal or no indentation.

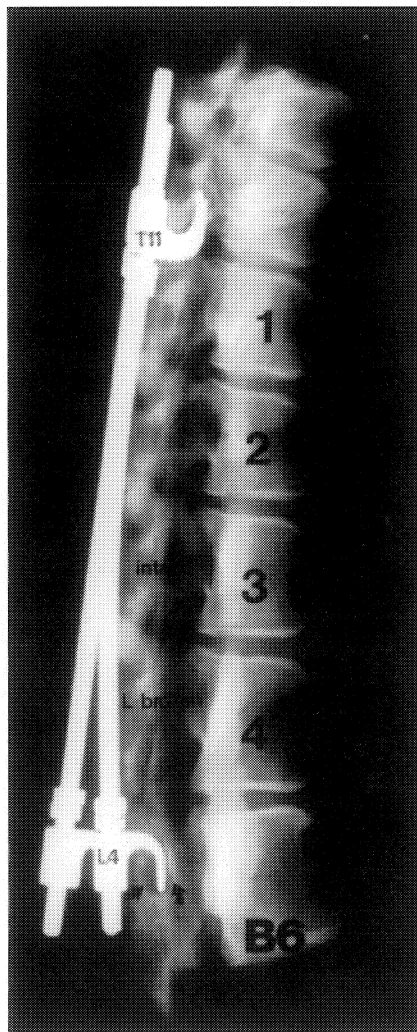


Figure 5A.

Figure 5. (A) Lateral view showing dislodgement of the distal hook. The Mersilene* tape at Level 4 was broken but was found to be intact at Level 3. (B) Due to the intact Mersilene* tape and increased pressure following hook displacement, erosion of the lamina was more prominent at level 3. (C) Less bony resorption was found at Level 4, most probably due to breakage of the Mersilene* tape shortly after hook dislodgement.

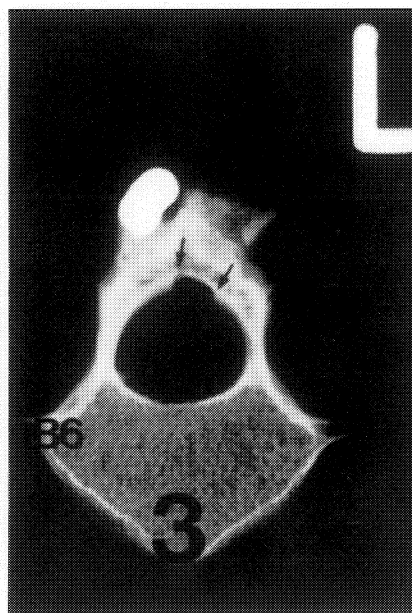


Figure 5B.

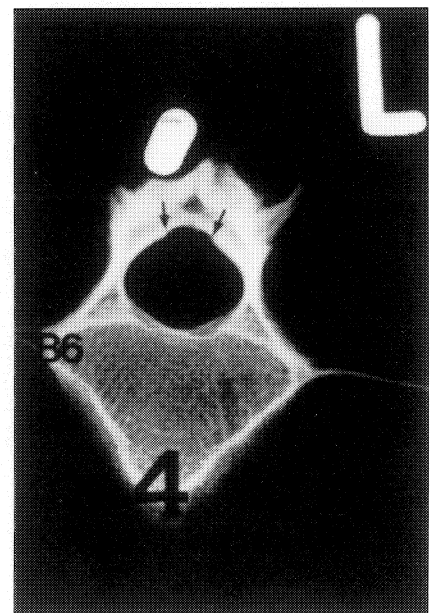


Figure 5C.

2. *Connective Tissue Membrane*

A connective tissue membrane surrounding both the MT and SSW was found, representing a typical physiological response to trauma. No foreign body giant cells or lymphocyte aggregations were seen in the examined specimens. Masson's trichrome stain was used to demonstrate the typical appearance of mature collagen fibers and an absence of abnormal capillary proliferation. The fibrous tissue response surrounding the SSW was of a more criss-cross, reticulated character in contrast to the bland, one-directional appearance found in the MT group. The Masson's trichrome technique further confirmed the impression of a less well-formed fibrous membrane, both around the wire and intervening between the wire and the dura mater (Figures 6C, D).

3. *Bone*

The bony tissue underlying the MTs revealed no osteogenic changes. At the edges of the tape-bone interface, bone remodeling units were seen. The appearance

most probably resulted from minimal, non-progressive reactive tissue formation due to the physiological response to slight movement and the initial trauma of insertion. In the SSW group, due to the loop formed and less bone contact, the underlying bone revealed no abnormal bone lysis. At the edges, some indication of bone remodeling was found.

DISCUSSION

The concern for neurological injury during the use of sublaminar wires relates to insertion, the possible long-term effects on the spinal canal contents and the potential complications related to removal¹⁹.

During the placement of sublaminar SSWs there is a distinct risk for neurologic injury^{1,2,3,6,7,9,11,15,16}. The period after safe passage and before definitive fixation to the rods seems especially hazardous (so called porcupine effect) and can lead to unintentional plunging of the rigid wire construct if the surgical team is not care-

TABLE 2
INTRA-SPINAL MEASUREMENTS

Animal No.	Type of Fixation	Level 1			Level 2			Level 3			Level 4		
		a	b	c	a	b	c	a	b	c	a	b	c
1	MT	0.85	1.05	19.0	*	*	*	1.05	1.20	13	*	*	*
2	MT	*	*	*	*	*	*	*	*	*	*	*	*
3	MT	0.78	0.90	13	0.85	1.00	15	0.90	1.05	14	0.80	0.9	11
4	MT	0.95	1.15	17	1.15	1.30	12	*	*	*	0.70	0.80	13
5	MT	0.95	1.15	17	1.00	1.20	17	1.10	1.25	12	1.10	1.30	15
6	MT	0.70	0.85	18	0.80	0.95	16	0.90	1.05	14	0.95	1.00	15
7	SSW	0.63	.101	38	0.79	1.16	32	0.76	1.26	40	0.57	1.07	47
8	SSW	0.70	0.96	27	0.70	1.06	34	0.88	1.17	25	0.85	1.18	28
9	SSW	0.70	1.10	30	0.75	1.00	25	0.70	1.10	30	0.70	1.20	42
10	SSW	0.75	1.15	35	0.80	1.10	27	0.80	1.25	36	0.85	1.20	29
11	SSW	0.85	1.15	26	0.80	1.15	30	0.70	1.05	33	0.70	1.10	36
12	SSW	0.90	1.20	25	0.95	1.20	21	0.70	1.20	42	0.80	1.15	30

1. MT = Mersilene Tape Group
2. SSW = Stainless Steel Wire Group
3. * = Used for Initial Assessment and no CT Assessment was done
4. a = MT/SSW-Lamina distance
 b = Total AP distance
 c = AP spinal occupancy ratio

ful. We experienced much easier placement of MTs compared to SSWs and the safety of a flexible implant in the canal was obvious; the potential for plunging was nonexistent. This may not be a major point in the hands of experienced spine surgeons, but has definite advantages for those who only occasionally use sublaminar fixation.

The AP occupancy ratio in the spinal canal in this study showed the MT occupied less space (14.8 percent) than the SSW (32 percent). In high risk situations, i.e. grossly deformed spines and in surgery at the cranial-cervical junction, it is an obvious advantage. No laminar thickening or overgrowth of the spinal canal was found¹².

The question of strength, at least in this animal model, revealed no definitive difference. Only in one case was a broken tape found, and this occurred only after the tape was exposed to long-standing tension forces (indicated by the bone erosion) due to hook placement failure.

Olsen found no neurological sequelae after sublaminar wire removal and minimal long-term intracanal changes were confirmed by follow-up MRI studies¹⁴. The easy placement and minimal space occupied by the MT in the spinal canal and added ease of removal of the tapes still makes this an advantageous technique. Schrader et al., using radiographic assessment, described an average indentation of 47 percent of the myelographic column during removal of sublaminar wires in dogs¹⁷. On direct monitoring we found distinct indentation during SSW removal while the MT showed minimal indentation of the dura due to the well-formed fibrous membrane.

Schrader described the chronic effects of sublaminar wires showing epidural, subdural and intradural hemorrhage¹⁷. We also found dural thinning over the SSW as described by Schrader et al., but we were more interested in assessing the possible membrane formation around the implants and its effect on the dura. In retrospect, preservation of the spinal cord and histological assessment of the underlying cord tissue would have increased the strength of our study. This study showed a twofold increase in the AP spinal occupancy ratio comparing SSW and MT, and comparing these two systems (MT and SSW) in the same experimental animal gave a more valid assessment.

Our study revealed formation of a distinct membrane around the MT with minimal encroachment on the spinal cord. Removal of the tapes was aided by this membrane resulting in minimal effort and no apparent injury to the dura. In contrast, the SSW group showed a thinned dura and inadequate membrane around and between the wires. The dura was often penetrated dur-

ing the process of wire removal. It seems, therefore, that both radiologically and histologically the findings favor MT use for sublaminar fixation in selected cases. Comparing the two groups clinically *in vivo* we did not find any indication that the Mersilene* was any weaker than the SSW. The intra-spinal space occupied by the sublaminar wire is distressing (compared to the MT), especially if used in the cervical spine.

SUMMARY

1. Comparing the use of Mersilene* tape (MT) and stainless steel wire (SSW) in this animal model, it appears that fixation in both groups was adequate. Breakage of the MT in animal number 6, likely secondary to hook dislodgement, was seen as a technical failure.
2. The insertion and removal of the MT was found to be less hazardous and may prevent untoward neurologic complications, especially in the presence of a deformed and/or narrow spinal canal.
3. The distinct connective tissue membrane surrounding the MT was more mature and better formed than that surrounding the SSW. This provides a more suitable track for risk-free removal of the MT. Penetration and/or injury of the dura, therefore, is less likely and should be seen as a major clinical advantage.
4. MT should be considered as a method of sublaminar fixation in select cases.
5. The Chagma baboon, used as an experimental animal, appears to be an adequate model to investigate posterior spinal implants to be used in humans.

ACKNOWLEDGMENTS

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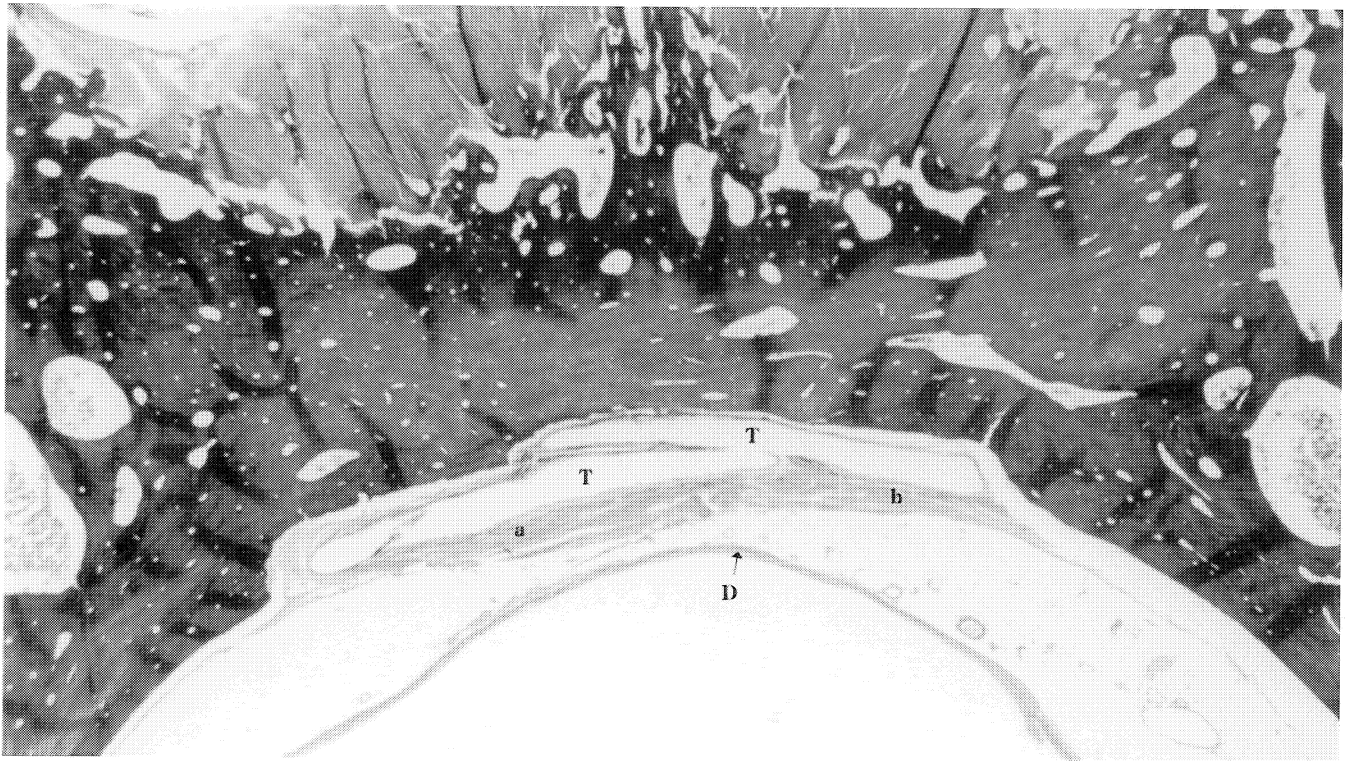


Figure 6A. Microscopic sections (Trichrome-stain) showing distinct membrane formation around the areas occupied by the Mersilene* tapes (a & b). The outline of the Mersilene* tapes are well shown (T). The dura is shown to be smooth and intact (D).

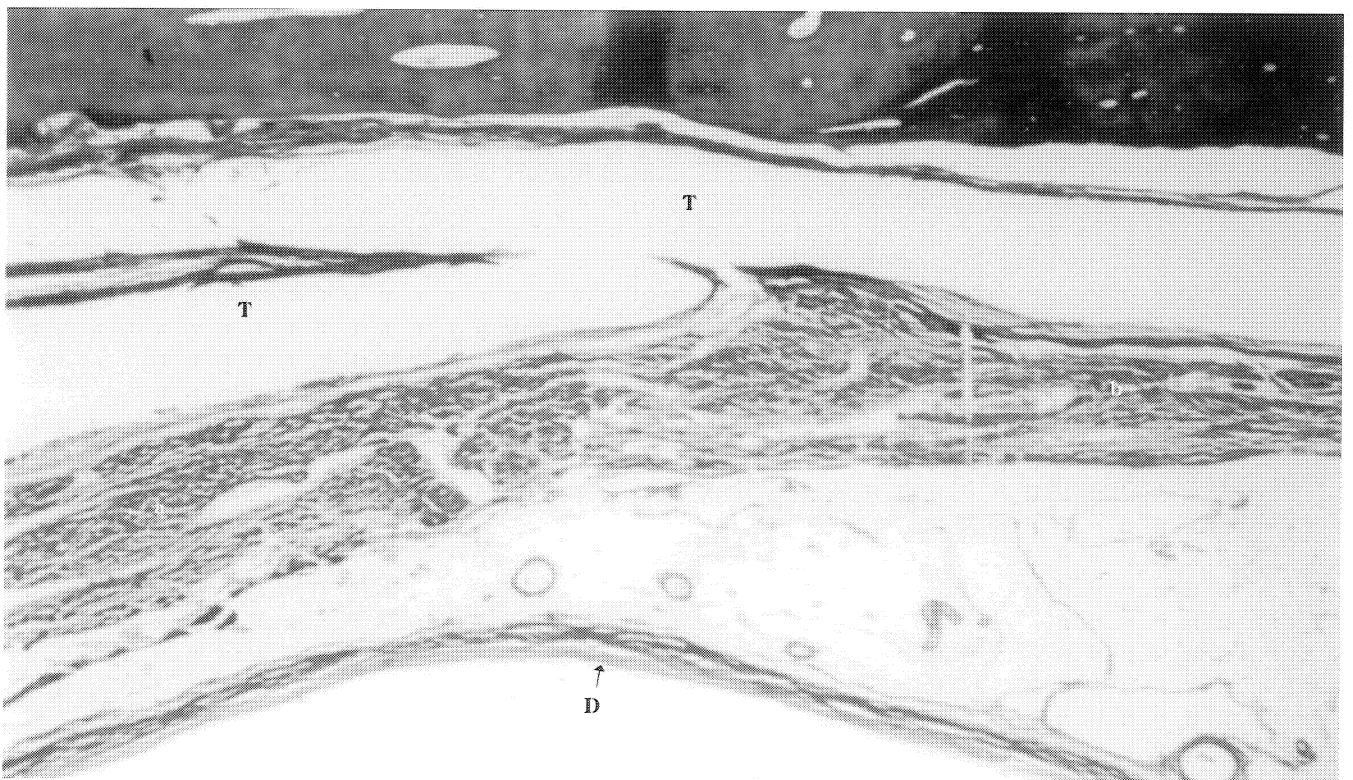


Figure 6B. More detailed histologic view of the area discussed.

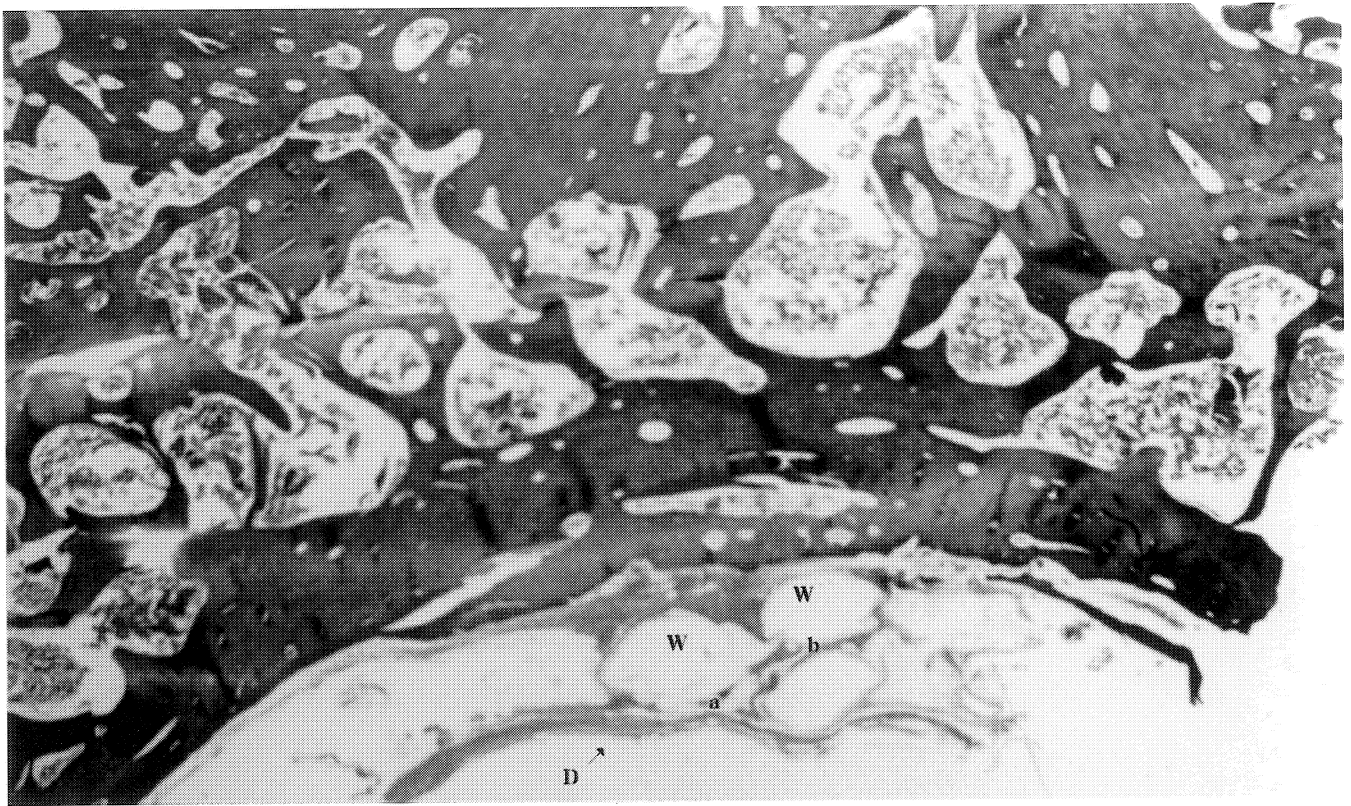


Figure 6C. Microscopic sections showing a less well-formed membrane around the areas occupied by the SSW (a & b). The space after removal of the SSW (W) is shown. Dura is shown to be thin and deformed.

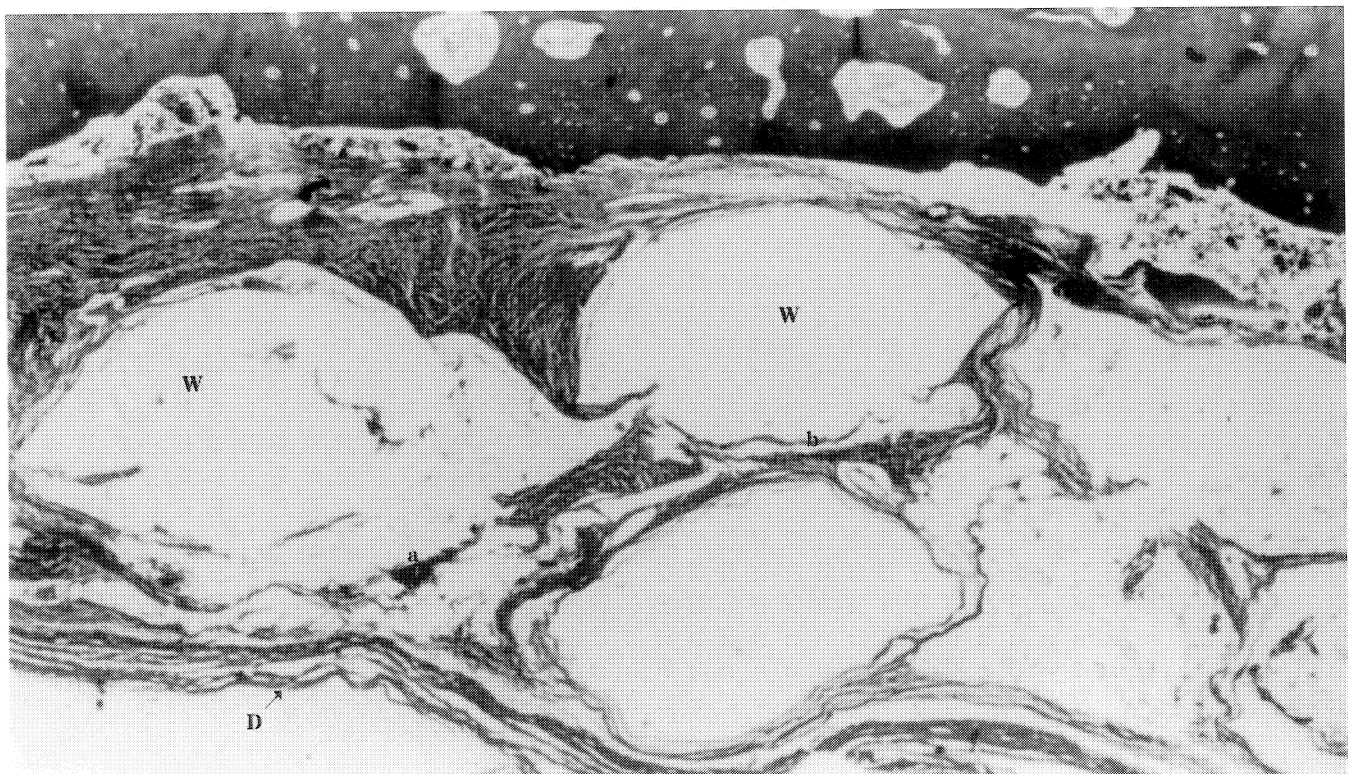


Figure 6D. More detailed histologic view of the area discussed.

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THE DEVELOPMENT OF ORTHOPAEDICS IN 20TH CENTURY WARFARE

James M. Banovetz, M.D., Ph.D.

INTRODUCTION

The disease known as warfare has afflicted humankind since the dawn of human history. The toll it exacts is always high; in the resources consumed, the destruction wrought, and of course, the suffering inflicted on the people involved. As long as soldiers have gone off to battle, they have taken with them those who try to heal the wounds inflicted. For much of recorded history, there was little that could be done, but the barbers, surgeons, and physicians gradually improved the technology, sometimes through brilliance and sometimes by accident, to effectively treat many of the injuries sustained by the combatants. Not all of the progress has been in the twentieth century. Hunter, Paré, and Larrey are a few of the many who advanced the care of combat casualties prior to this century. In all wars, treatment techniques have varied at different times and/or locations, and the writings of no one surgeon or group is necessarily representative of the treatment in an entire conflict. This paper is based on writings in the general literature, but there are many manuals, reports, and studies in government publications not found in a medical library. Brief reviews of military surgery have been done by Cozen¹⁶ and Shepard and Rich⁵⁰, and a wonderful review of the development of orthopaedic surgery in the first half of the twentieth century was done, on a nation by nation basis, by *The Journal of Bone and Joint Surgery* in the thirty-second volume of the British series⁴⁴. Hopefully a similar volume will be forthcoming at the turn of the century.

The evolution of warfare has generally been gradual, and picking out pivotal points is somewhat arbitrary, but a strong case can be made for labeling the American Civil War the first “modern” war, in that it involved the mobilization of the resources of an entire continent. Casualties occurred on a previously unheard of scale. In the two month period from May to June 1864, the Union Army of the Potomac, just one of the armies of one side, sustained 60,188 casualties: 8,503 killed, 43,043 wounded, and 8,642 missing. To deal with this flood of injuries, the Union army developed the first mass casualty system with dedicated stretcher bearers, ambulance wagons, aid stations, field hospitals, and general hospitals. Standardized, professional care was gradually organized, and widespread (although not universal) record keeping was instituted. *The Medical and Surgical His-*

tory of the War of the Rebellion was felt by the European community to be the first significant contribution to the medical literature made by American physicians⁵. An interesting footnote to the war was the discovery, on the Cold Harbor Battlefield, of a fragment of radius that had been internally fixed (and healed) with a brass pin²¹.

WORLD WAR I

When war broke out in Europe in 1914, it developed in an almost farcical way into a conflict no one had foreseen. This was due to a complex set of alliances as most of the war was between countries little concerned with the assassination of the Arch Duke Ferdinand, sole surviving son and heir of the Austro-Hungarian Empire. Austria and France declared war on Germany. Because France and Germany had excellent rail systems and highly developed mobilization plans, they were able to mass troops on their borders faster than the other nations. As a result, the war erupted between these two countries weeks before Austria attacked Serbia.

The expectation of both sides was that this war would develop like previous wars: the armies would meet for a series of sharp engagements with one side developing a clear advantage, and then the weaker party suing for a negotiated peace. Unfortunately, their expectations were based more on previous European and colonial wars than on the American Civil War. The most recent war involving the allies had been the Boer War in South Africa between the British government and Dutch settlers. This had been what we now call a guerrilla (Spanish for “little war”) conflict with small numbers of men engaged over a wide area in semi-arid terrain. Wounds were almost always from relatively low velocity rifle bullets, requiring relatively little in the way of treatment³⁴. World War I surgeons were initially unprepared to face the effects of two devastating weapons: the machine gun and the rapid-firing howitzer (cannon). When the French and German armies met, neither side was able to maneuver effectively, as a single machine gun could destroy entire companies of men or troops of cavalry. Any concentration of men or supplies near the lines for an assault was annihilated by artillery barrage. Both sides were forced to dig into the ground, and the result was the stalemated trench warfare we associate with the First World War. The principle strategy in the war was to break through the enemy’s lines and exploit them

with a rapid advance into their rear areas. The tactic to accomplish this was usually a massive artillery bombardment of the opposing trenches followed by an infantry charge, accompanied later in the war by primitive tanks. These tactics never resulted in the desired decisive breakthrough, but caused astronomical casualties that totally overwhelmed the medical system, and served to establish the basic principles for the care of contaminated, high energy trauma.

The principles of Lister were widely known and practiced in civilian surgery, but for a number of reasons they were insufficient to prevent nearly universal infection of wounds early in the Great War. Neither prior military or civilian experience prepared the surgeon for combat casualties. First, there were the sheer numbers. Wounded in the tens of thousands poured into the aid stations, and there was not time, supplies, or personnel to adequately evaluate and treat them. Few, if any, surgeons had experience with high energy trauma, as high speed vehicles and power machinery were still not in wide spread use by the civilian population. Artillery was the single greatest culprit in the wounding of young soldiers. The static nature of the lines and rapid fire of the weapons allowed a high concentration of destructive power. Tissue damage from a missile wound is a function of the energy imparted. This, in turn, is proportional to the mass of the missile and the square of its velocity. Both the mass and the velocity of shell fragments varied, but they often caused a zone of tissue damage much wider than the obvious laceration. Wounds early in the war were rarely adequately debrided, with the resulting compromised tissue forming a perfect medium for bacterial growth. Shells also contributed to the infection problem by plowing up the environment. Farms, dumps, latrines, and cemeteries were churned up, spreading their waste across the battlefield. The fact that the combatants had to live in damp, underground conditions with few opportunities to bath or change clothes undoubtedly contributed to the high local bacterial counts. Shell fire made approaching or leaving the trenches a dangerous business, and casualties often had to wait hours before evacuation.

Improvements occurred gradually in several areas. One of these was casualty transport. Long trenches snaking back from the front lines allowed safer transport away from the battlefield. The use of trucks for ambulances improved both the speed and the comfort of transport, decreasing the degree of shock upon arrival at field hospitals⁷. The use of battlefield splints had a dramatic effect on the shock and tissue damage caused by transport; decreasing the mortality of open femur fractures from 80 to 20 percent^{6,44}. The static nature of the war allowed the construction of well

equipped and staffed facilities such as "casualty clearing stations" (for triage) and "field hospitals" (for desperate emergencies) right behind the lines. "Evacuation hospitals" (the first hospital for all other cases) were as close as six miles to the front lines, each able to handle 400 to 1200 casualties. The ebb and flow of battle could vary the workload, and staff could be rapidly transferred to reinforce a busy hospital, or patients could be forwarded if one location was overwhelmed⁷. After initial stabilization and wound care, patients were transferred to base hospitals for long-term care. These hospitals were often specialized and had wards that were even more so; some wards being dedicated for femur fracture patients only.

Lewis and Leary described the organization and operation of an evacuation hospital in 1919 (Figure 1). They stated that the ideal hospital should have 500 beds and should use tents for shelter. It should be able to be collapsed or set up in twelve hours and needed a nearby water supply and transportation. Personnel included a surgical chief, assistant chief and fourteen ward surgeons. Each surgeon had two tables and operated on one while his previous patient was splinted, dressed, removed, and replaced by his next patient on the other. Surgeons ideally worked eight hour shifts. Operations were performed on the litter, the skin first dry shaved and cleansed with gasoline followed by iodine. Accurate records were important, but often did not make it to the base hospitals. It was best for new surgeons to first observe, then assist, then lead a team. Experienced surgeons should periodically rotate back to base hospitals to evaluate the results of their procedures, and there should be good communication between the two types of hospitals³⁵.

Because of the overwhelming numbers of casualties, early efforts at wound care focused on finding an ideal "antiseptic." The idea was to find a substance that could simply be poured into a wound, dressed and then intentionally neglected. Some of the substances used are shocking in retrospect, (Table 1). Of these, only alcohol and Dakin's solution are used today. The latter was developed by a British chemist, Dakin, for a French physician, Carrel, who used it in an intermittent irrigation program with serial cultures. Frequent references are found to the solution and the method. Combinations such as salicylic and boric acid in a paste with phenol, cresol or bismuth subnitrate/iodoform/paraffin (B.I.P.) were also used^{7,34}.

The confidence in these antiseptics was high as wounds were often sutured without debridement. Many of these substances caused further tissue death, predisposing the wounds to further infection, sepsis, gangrene, and/or tetanus^{6,7,34}. As the disastrous results of

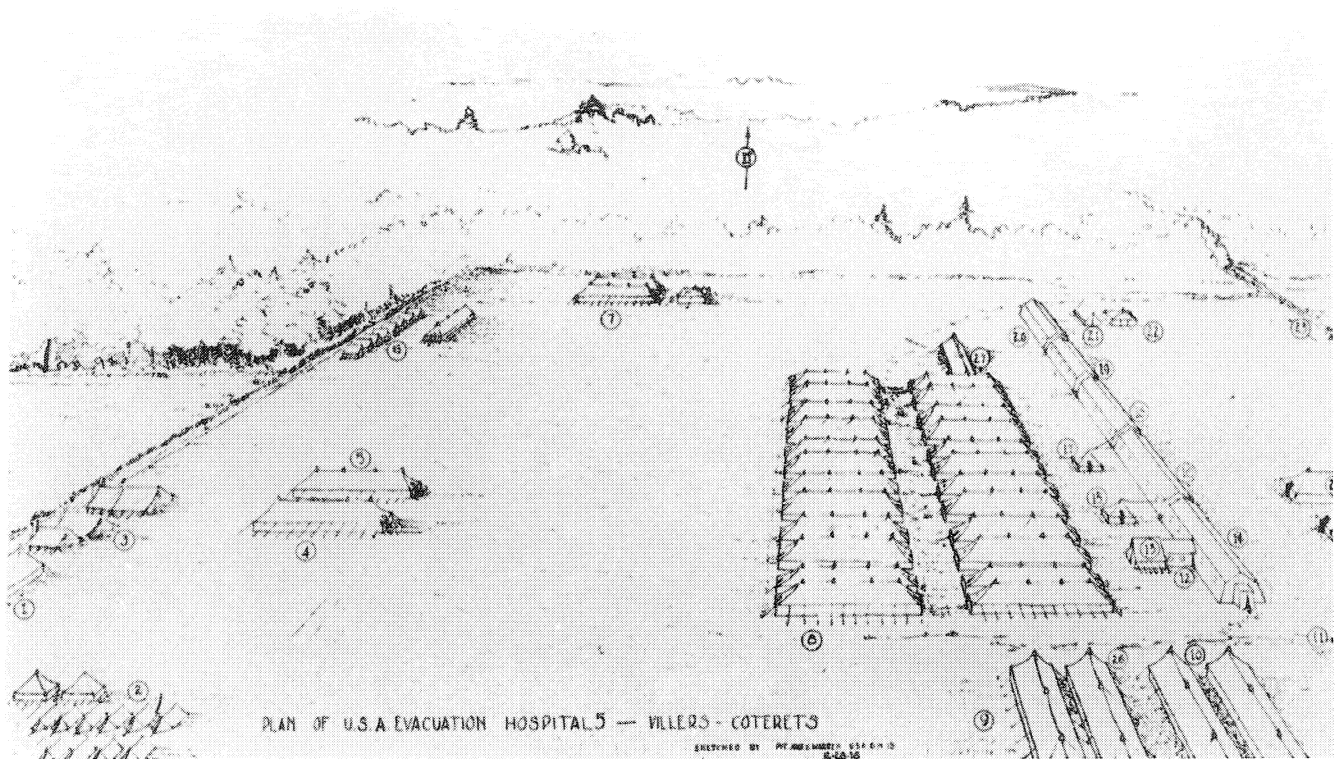


Figure 1. Plan of Evacuation Hospital No. 5, Villers-Cotterets, France, August 29-Sept. 14, 1918. 1, commissary; 2, personnel quarters; 3, kitchens; 4, nurses' mess; 5, officers' mess; 6, officers' quarters; 7, nurses' quarters and recreation tent; 8, surgical wards; 9, non-evacuatable wards; 10 and 26, fracture wards; 11, road to evacuation platform; 12, sterilizing room; 13, dressing tent; 14, operating Bessonneau tent; 15, lighting plant; 16, pre-operative Bessonneau tent; 17, X-ray power plant; 18, X-ray Bessonneau tent; 19, 20, receiving wards; 21, headquarters; 22, Red Cross; 23, main highway; 24, quartermaster; 25, medical supplies; 27, shock ward. (Reproduced with permission from *Annals of Surgery*²⁵)

these methods became evident, the principles currently recognized for the care of traumatic wounds developed: early, careful debridement, serial debridements if needed, adequate drainage, delayed primary closure, secondary closure, or healing by secondary intention as indicated by the state of the wound. The poor results from antiseptics were often ascribed to closure of wounds. Delayed primary closure was sometimes for-

bidden even after there was good data to support it, and articles castigating it can be found right up to the Vietnam war¹⁸.

Only severely mangled limbs were amputated at the evacuation hospitals, and when done at the base hospitals they were usually circular, but with the tissue cut progressively higher from skin down to bone. By the end of the war, below knee amputations were standardized at seven inches and prostheses were ischial weight-bearing³⁴. Initially, evacuation hospitals paid more attention to fracture alignment than wound care, but for most of the war no attempt was made to align fractures until arrival at the base hospital. Long bones were initially treated with immobilization, but results were poor. Only 2 percent of femur fracture patients returned to duty. The results were improved when traction was more widely used, though internal fixation was still thought to be bad practice⁶. The Thomas splint became enormously popular, both for initial immobilization and for definitive treatment of lower extremity fractures^{7,41}. Various methods were used to provide traction, usually through the skin, and pictures of a traction apparatus and a recipe for the glue are shown in Figures 2 and 3¹¹.

Table 1
World War I "Antiseptics"

cyanide	hydrogen peroxide
iodine	mercuric chloride
formalin	carbolic acid
lysol	picric acid
alcohol	potassium permanganate
ether	magnesium sulfate
urea	petrol (gasoline)
camphor	brilliant green
phenol	hypertonic saline
	Dakin's solution

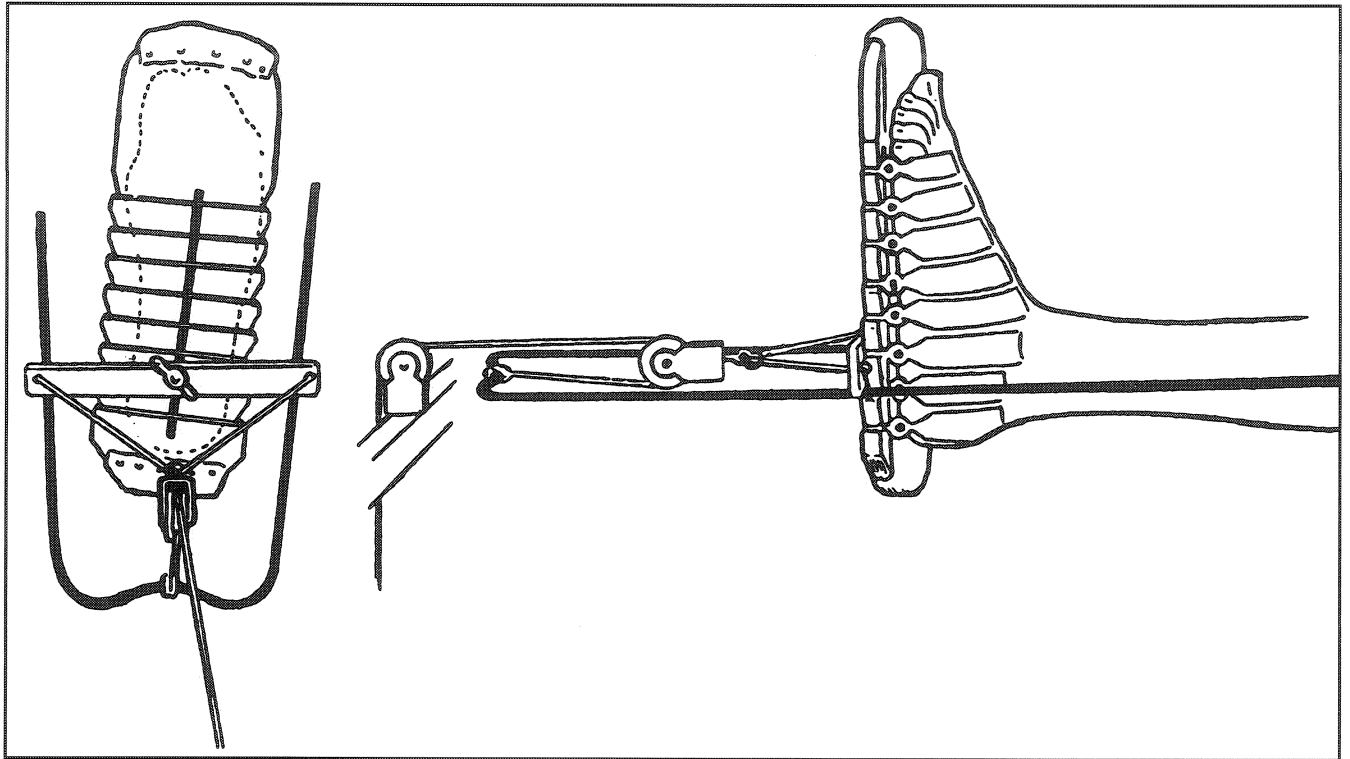


Figure 2. End view of "Sinclair skate" showing manner of securing outward rotation of the foot by adjustment of the transverse bar. Lateral view of "Sinclair skate" and pulley, showing method of securing proper amount of dorsiflexion of the foot by adjusting the transverse bar further up or down the skate as desired, and also showing the method of attaching the pulley. (Reproduced with permission from *Annals of Surgery*¹¹)

A wider variety of equipment was used for the upper extremity, but definitive treatment was still usually with traction, the technique and results of which are shown in a unique way in Figure 4^{7,51}. Joint wounds were serially aspirated or drained early in the war with poor results. Later conversion to open irrigation, debridement and capsular closure provided better results⁷. Dr. Wilms (or Willems) of Brussels introduced early motion of injured joints to prevent ankylosis with astonishing success^{6,18,39,40}. Arterial injuries were treated with ligation, leading to high amputation rates, as attempts at reconstituting arterial flow were dismal failures⁷. Spinal cord injuries had an early mortality rate of 50 percent and a three year mortality rate of 80 percent³⁴.

Later in the war sophisticated studies appeared on wound care. DePage wrote on "General Considerations as to the Treatment of War Wounds" in the *Annals of Surgery* in 1919. He stated that nearly all wounds are contaminated, and debridement is "a formal indication of the first rank." He recommended primary or secondary suture after the wounds were clinically aseptic. Dressings were changed every day and the skin and tissues cleansed with oleate of soda. They followed culture smears starting in 1916, and found that bacterial counts sharply increased on the second day and re-

mained high for some days, and then gradually decreased in an oscillating fashion, approaching to zero in six to eight days. Open fractures and articular wounds took fifteen days to "sterilize." Staphylococcus was the most resistant organism to this treatment, but closure of wounds with remaining clostridium perfringens or streptococcus had the highest risk of infection. Primary suture was only done if the wound smear was negative and the wound was less than eight hours old, had not been previously explored, and was not deep, complex, or soiled²⁰.

McWilliams and Hetzel wrote "Report of 82 cases of knee-joint war injuries from evacuation hospital 1, A.E.F., with remarks on the Willems' treatment by immediate closure and subsequent mobilization, and the management of the subsequent infection by drainage and mobilizations." Joints were carefully debrided, irrigated, cultured, and the capsule closed (unless the joint was frankly infected) with packing of the overlying wounds. Immediately postop the patient began active exercises using his hands to help elevate his thigh and slide his heel along the bed. Aspiration was used to eliminate hemarthroses as often as necessary, and the fluid was cultured. Infected joints were drained, but otherwise treated in the same fashion. Ambulation was begun two

Traction Glue Recipe

Major M. Sinclair, R. A. M. C., has supplied us with the following guide as to the preparation and use of the glue:

Test for the glue:

Place 4 oz. of glue in 4 lbs. of cold water and leave in a cool place for twelve hours.

If dissolved bad
 If coherent and gelatinous weighing 8 oz good
 If coherent and gelatinous weighing 16 oz very good
 If coherent and gelatinous weighing 20 oz excellent

The following is the formula:

Very good glue 50 parts
 Water 50 parts
 Glycerine, or glucose calcium chloride 4 or 6 parts
 Menthol 1 part

Soak for twelve hours and then melt on a water bath. Neutralize with sodium hydrate, as commercial glue at times contains free hydrochloric acid. Add 4 parts in summer, and 6 parts in winter, of glycerine or glucose calcium chloride and 1 part menthol. Frequent heating evaporates the water, which should be added from time to time. When released many times, adhesive power is lost.

Technic:

- (1) The skin is not shaved.
- (2) Wash the skin with soap and hot water, which contains about 4 drams of washing soda to the pint, to convert the oil of the skin into soap, and glue will not adhere to a greasy surface.
- (3) Dry the skin.
- (4) Apply the warm glue evenly, brushing the hairs of the limb in an upward direction.
- (5) Keep a tension on the gauze all the time. Bring it quickly but carefully into contact with the limb (inner and outer surface), and apply a neatly and loosely woven bandage, starting a hands-breadth above the malleoli up, to the knee joint (in case of fracture of the femur).
- (6) When dry apply traction. Note. - The adhesive can be made waterproof with a 2 percent solution of potassium bichromate applied in the dark and then exposed to the light, or by means of formalin.
- (7) The extension must always be very carefully applied, whether with Maw's elastic cotton net or with gauze.
- (8) The extension must be changed at once if the patient complains of tickling or burning sensation under it, but it generally requires changing about the tenth, twentieth, and fortieth days.

Figure 3. (Reproduced with permission from *Annals of Surgery*¹¹)



Figure 4A. Pvt. Semfield. F. C. C. left humerus. Superimposed photograph to show mobilization of elbow.

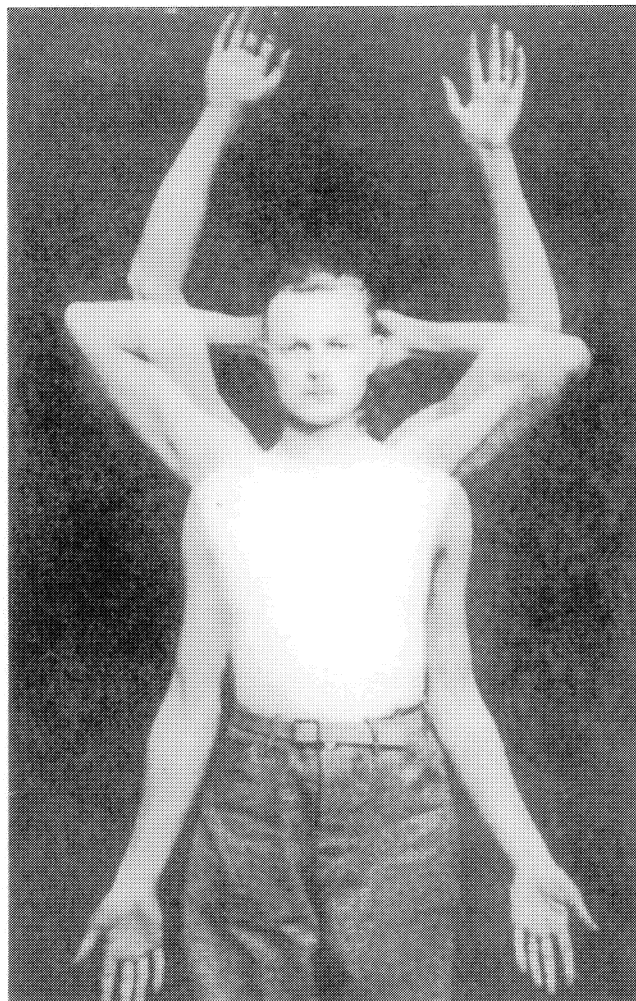


Figure 4B. Superimposed photograph made on the eightieth day. (Reproduced with permission from *Annals of Surgery*⁶¹)

to three days postop for uninfected patients, and on the first day postop for those with infections. Unstable fractures necessitated a delay in the motion program. Of the eighty-two injuries, forty-eight were associated with fractures. Seventy-three were initially free of infection and fifty-seven remained infection free. Lack of supporting staff prevented complete application of the frequent motion program, and the patients had to be relied upon to do their own exercises. Of thirty-six cases followed to final outcome, nine had full motion, twenty-four had some restriction, and three had ankylosis. Twenty-nine of these patients were returned to duty. Of the sixteen who became infected: four ankylosed, four died, three underwent amputation and one underwent resection. They thought mobilization helped to pump pus out of the joint, and stated that immobilization for transport allowed infection to set up, and should be avoided for at least ten days. Finally, they noted that the time from injury to surgery correlated with the result³⁹.

Hinton wrote "Suture of War Wounds" in 1920. He set up a hospital ward specifically for "suture" cases, i.e. primary, delayed, or secondary wound closure. Vitals signs and cultures were monitored, and suspected infections were transferred out. Wounds were treated with methyl alcohol until "charring or drying" occurred, as a "dry" wound did better than a "moist" one. More inflamed wounds were treated with dressings soaked in the hottest water the patient could tolerate and changed every four hours. Wounds were normally closed in three to five days, and the patients were kept at bed rest until suture removal. Contraindications to closure were inflammation, discharge, malodor, fever, tension of the tissue, or systemic illness. Efforts were made to minimize the handling of the tissue and to obliterate dead space. Bones were curetted out and rubbed with B.I.P. Minimal sutures were placed below the skin, as they predisposed to infection. Two hundred and seventy patients were followed and 84 percent healed with no signs of infection, 12 percent required some further drainage, and 5 percent had failure of the closure. Wounds were classified as Type I (skin and subcutaneous fat only), Type II (muscle and fascia involved), and Type III (bone involved). Type III did slightly worse, with 76 percent healing without further problems and 6 percent having a failure of the closure. Secondary closure also did slightly worse. Seventy-five percent of the wounds had positive cultures at the time of closure. The good results go against Carrel's dictum of not suturing culture-positive wounds. Most failures were associated with gram negative bacteria. It is instructive to remember that these results were obtained before the use of antibiotics and still utilized some substances which were probably harmful to tissue; yet the results were not

much worse than what could be obtained today³⁰.

Robert Jones was instrumental in many of the improvements that came during the war, including the development of reconstruction and rehabilitation facilities to maximize the recovery of the wounded and help injured soldiers become functional members of society. He tried to get the wounded involved in regular work tasks as much as possible to help develop a sense of usefulness and self-respect⁴⁴.

Although American involvement in the war was much smaller than that of our European allies, both in time and in the numbers of men involved, the war had a profound impact on American orthopaedics. At the invitation of Robert Jones, a group of American orthopaedists went to England prior to American military involvement to help and learn from the British. Titled the "Goldthwait Unit," after Joel Goldthwait, the leader of the group, they became the core of the orthopaedic division in the American Expeditionary Force. At least fourteen of the men who were a part of this unit went on to serve as president of the American Orthopaedic Association, and twenty-nine went on to careers as orthopaedic teaching faculty^{45,47}.

World War I was a horrible conflict, destroying an entire generation of European youth and devastating a huge area of French and Belgian country. It is to the credit of the surgeons who served that they arose from the quagmire of "antiseptics" to develop the wound care principles that are still in use today. Unfortunately, much of the knowledge developed in World War I was not organized for future military use, and many lessons had to be relearned twenty years later in World War II, although "antiseptics" were replaced by "chemotherapy," the term used at the time for antibiotics. Much to the chagrin of Robert Jones, orthopaedic and rehabilitation centers were closed down and their principles forgotten between the wars. Fortunately, the lessons were relearned more rapidly the second time around. World War I, "The Great War," "The War To End All Wars," unfortunately turned out to be neither the greatest nor the last.

American involvement in World War I was desperately desired by the allies to break the stalemate with Germany, and was finally accomplished when the allies agreed that after the war they would put an end to "power diplomacy," in which each country's foreign policy was determined solely by self interest. This would be replaced by "moral diplomacy," in which policy would be determined on a set of moral principles regardless of national advantage. The moral approach to world affairs fell apart with amazing speed after the war; the United States refused to join Woodrow Wilson's League of Nations and Germany was heavily burdened with war

reparations. The communists butchered millions consolidating their repressive hold on Russia and then began looking avariciously at their neighbors. Political problems were exacerbated by the world economic depression of the 1930's. Hitler in Germany and the military in Japan rose to power on promises of improving their respective countrys' economic state. Their territorial aspirations, together with those of Russia, ignited the second world war. This new world war was far more global than the first, with battles fought on four continents, many different terrains, and on all the world's oceans. The challenges faced by surgeons and their solutions to them varied widely, based on the terrain, weapons, personnel, and supplies.

SPANISH CIVIL WAR

A prelude to the second world war was the Spanish Civil War. Severe political instability and economic depression finally broke out into a bitter military struggle in 1936. Russia and Germany used the conflict as a testbed, sending advisors, pilots, and equipment to the republican and nationalist sides, respectively. The German bombing of Guernica, the first systematic bombing of a city, was widely reviled, and presaged the horrendous area bombing done by both sides (but much more by the allies than the axis powers) in the world war to follow. Both sides were impoverished and medical supplies were few throughout the war, which ended in a nationalist victory in 1939. In this environment, Joseph Trueta popularized the "closed treatment" of open fractures. This method involved a single, careful debridement followed by casting. The cast was not touched for six weeks, no matter how malodorous and suppurative it became. Surprisingly, the fractures had a high rate of healing, although other complications such as stiffness, weakness, and chronic infection were frequent. This method had adherents at least through the Vietnam war, and Trueta himself, who escaped to England after the war, was still writing about the method in 1976. In the absence of adequate supplies, it may still be a reasonable option today⁵⁵.

WORLD WAR II

In 1933 Adolf Hitler rose to power in Germany. Although World War II formally broke out with the German and Russian invasions of Poland in 1939, the first aggressive act in Europe was Hitler's remilitarization of the Rhineland in 1936, followed by occupation of Austria and the Sudetenland of Czechoslovakia in 1938. In March of 1939 Hitler invaded the rest of Czechoslovakia, followed by Poland in September. The War subsequently spread to the Mediterranean, North Africa, and the Atlantic. In the Pacific the war started for

America with the nearly simultaneous Japanese attacks on Pearl Harbor, the Philippines, and Southeast Asia in December of 1941, but for the Chinese it started with the invasion of their country in 1937.

A number of developments promised to improve wound care in this war. Sulfonamide antibiotics were in wide spread use, and penicillin was just becoming available. Unfortunately, these antibiotics, or "chemotherapy agents" as they were frequently called, held the same false promise as antiseptics; that wounds could be sprinkled with sulfa powder and would not require time-consuming debridement. This was no more true in World War II than it had been in World War I; fortunately this was recognized much faster, and the principles of wound debridement evolved as the primary treatment of open wounds. As in the Great War, initial attempts at early wound closure were not successful and the concept abandoned, only to reemerge gradually through the war. For a time, early wound closure was only allowed for airmen, who were presumed to be less susceptible to infection since their wounds occurred thousands of feet above the mud and waste of the battlefields. This philosophy gave too much weight to the environment and not enough to the debridement.

In Germany, Otto Kuntscher developed the intramedullary nail concept for long bone fractures, although his status as a relative outcast prevented its widespread use. It did cause a mystery for allied air forces, who couldn't figure out how German pilots reported to have femur or tibia fractures could be flying again in two weeks, while allied pilots with the same injuries were out for months!

One of the challenges of this war as opposed to the last was the provision of care to the battle front. There was almost no static warfare of the type seen in World War I, and thus no opportunity to set up semi-permanent facilities. The war in the Western Desert of North Africa was particularly mobile, with the front lines being ethereal and shifting by dozens of kilometers in a single day. The pace of European battles was also determined by the tank and airplane, although German supply transport was still almost entirely horse-drawn! In the Pacific, island assaults were the primary type of battle, with initial treatment delivered on ships until a foothold could be obtained. Even when facilities could be set up on shore, they often lacked much in the way of basic supplies such as lumber or fresh water. In some instances, Navy surgeons operated in boxer shorts and masks, as there was insufficient water to launder clothes and gowns! Operations in the China-Burma-India theater took place at the end of a long supply line, with space at a premium on air and water-borne transport. Often the trickle of supplies was so thin as to barely

support operations at all, much less allow for good medical care. Chinese troops in particular were often malnourished, compromising their ability to heal, but even American and British troops could be badly worn down by months of poor food and tropical disease in this theater.

Some of the first literature to come out of the war was from North Africa. The initial war in France was a disorganized rout culminating in the dramatic evacuation of Dunkirk, and there was little opportunity to study casualty management during that time. At the time the United States was plunged into the war, the British Empire was actively fighting on the ground only in the struggle for control of Egypt and Libya, although there was fierce fighting in the skies over Europe and in the Mediterranean and Atlantic oceans. Colonel J. A. MacFarlane of the Canadian military was invited to address the American Orthopaedic Association in 1942 on "wounds in modern war." He described a Canadian innovation for battlefield care in which a small unit consisting of two surgeons, assistants, and supplies for thirty to forty operations were carried in three trucks and could be rapidly deployed to deliver immediate care to wounded soldiers³⁷. Manning described the step by step process of evacuation to a base hospital, which could take two weeks and involve eight different facilities³⁸. MacFarlane advocated closed treatment ala Trueta after initial debridement, and disparaged delayed closure. He found no added benefit from chemotherapy, but did not state whether it was local or systemic, or support his assertion with data. He did recommend dusting wounds with sulfonamide powder prior to evacuation, and although this was widely practiced, it probably did not have much effect on the infection rate. He recommended the use of plaster casts with no padding or splitting, in contrast to the writings of others³⁷. Blackburn wrote of "surgery in the field" in 1944, stating that principles of extremity wound treatment included debridement, drainage, immobilization, chemotherapy, and prophylactic serum. The question of how much skin to debride is always a point of debate, and Blackburn weighed in with an admonition to avoid "extravagant" skin excision. He stated that inadequate fascial incision and incomplete debridement were "surgical crimes," and that fractures did not need to be aligned, but should be immobilized. In contrast with MacFarlane, he recommended that plaster should always be split⁴. Moorhead wrote a similar article on wounds⁴².

The first American writings were anecdotal reports, appearing in 1943. Hayden wrote of his experiences in the naval hospital at Pearl Harbor on December seventh, 1941. They admitted double the hospital's capac-

ity in about three hours that day, many covered with fuel oil, and 47 percent with burns. They used the closed treatment for fractures and felt that sulfa dusting bought time for debridement. He emphasized the need for efficient, streamlined protocols to deal with large numbers of casualties²⁹. Johnston valued sulfa powder, stating that it rendered debridement unnecessary³²! Crile treated patients at a base hospital twelve days post-injury, and noted that primarily closed wounds were nearly always infected, and that amputations without skin traction contracted and therefore had to be revised at a higher level. He decried wide debridement as it left large tissue defects. He again recommended little or no debridement, relying on sulfonamides, but did state later that resistance to sulfonamides was fairly common¹⁷.

Auster wrote up the first large scale American experience. He compiled 2,043 combat injuries, most seen six to forty-eight hours post-injury after aircraft transport. Field treatment included sulfonamide, dressings, plasma, and morphine. Major hemorrhage and severe compound fractures were emergently operated on at aid stations, but most were initially treated by redressing and application of Thomas splints. Clean wounds with small retained metal fragments were usually not explored and rarely became infected. Large fragments were usually removed, and contaminated wounds debrided. Frequently there was bone loss and non-vascularized fragments were excised. Fractures were reduced and splinted, but internal fixation was rarely employed. However hip fractures were usually pinned and casted. Trueta's closed treatment principles were used successfully in most cases. He felt they had a low infection rate in spite of the environment:

"In view of the fact that the combat terrain and the hospital compound were almost constantly muddy and covered with dense vegetation in various stages of decay, mixed with droppings of cattle and other animals, the infrequency of pyogenic, tetanic or gas infections was remarkable. Even in our operating rooms it was impossible to maintain conditions of more than casual sterility, working as we did in sweat-drenched shorts when gowns were unavailable, the mud and coral caked upon our boots mixing with blood and water upon the deck, and the constant movement of necessary personnel about the rooms. The atmosphere was constantly and unavoidably laden with bacteria²."

The first large scale report of casualty management in Europe was done by E. D. Churchill. The now-familiar principles were debridement, stabilization of fractures, avoidance of wound closure, and "chemotherapy." Sulfonamides were used in the field, but he questioned

their value. Penicillin was used perioperatively on a wide scale, but its contribution to success was not clear. The American system had casualty clearing stations to triage patients, with the severely wounded having operations in a nearby field hospital while most were sent to evacuation hospitals. The patients were then sent to base hospitals after initial stabilization and usually had wound closure four days after initial debridement. The decision to close was made based on inspection only, with no attempt to culture the wounds. Of 25,000 soft tissue wounds, 95 percent healed, an apparent improvement over World War I. At the base hospital, definitive alignment of fractures was done with traction, and dependent drainage wounds were made for open fractures. Churchill opined that the "closed treatment" method of Trueta was developed to save lives, but extracted a high cost in limb morbidity and mortality, while the principles used by the American medical system as described above were better for preserving life and limb function¹².

The importance of wound care versus antibiotics was firmly established in the Mediterranean theater of operations (MTO) by Lyons, who did an extensive bacteriological study that proved it was the nutrient value of the wound to bacteria that correlated best with prognosis, rather than the species and susceptibility of the organism. He felt that the role of chemotherapy was to reduce systemic invasion³⁶.

The first mention of internal fixation by the allies was in a paper on reparative surgery of compound battle fractures by Hampton from 1945. He reported that undebrided wounds invariably decomposed, local or systemic "chemotherapy" did not prevent sepsis, and infections tended to spread into surrounding healthy tissue. The injury to surgery interval was often twelve to twenty-four hours. Devitalized bone and soft tissue were removed, and penicillin was used until five to ten days after the last operation. Internal fixation was used if necessary to obtain stabilization, as unstable fractures were more prone to infection. Fixation was often minimal and supplemented by traction or casting²⁵. A diagram of plating technique is shown in Figure 5. He later described expanded indications for internal fixation and reported follow-up results at five to ten months. Of 332 open fractures, 63.3 percent had union and healing of the wound, 18.1 percent required hardware removal after union to obtain wound healing, 6.3 percent had unhealed wounds, 4.2 percent had nonunion but healed wounds, and 7.5 percent had both nonunion and persistent wounds. Fifty-three percent of patients with established sepsis at the time of internal fixation had union and wound healing at follow-up. His worst results were correlated with massive soft tissue loss and plated tibia fractures²⁶.

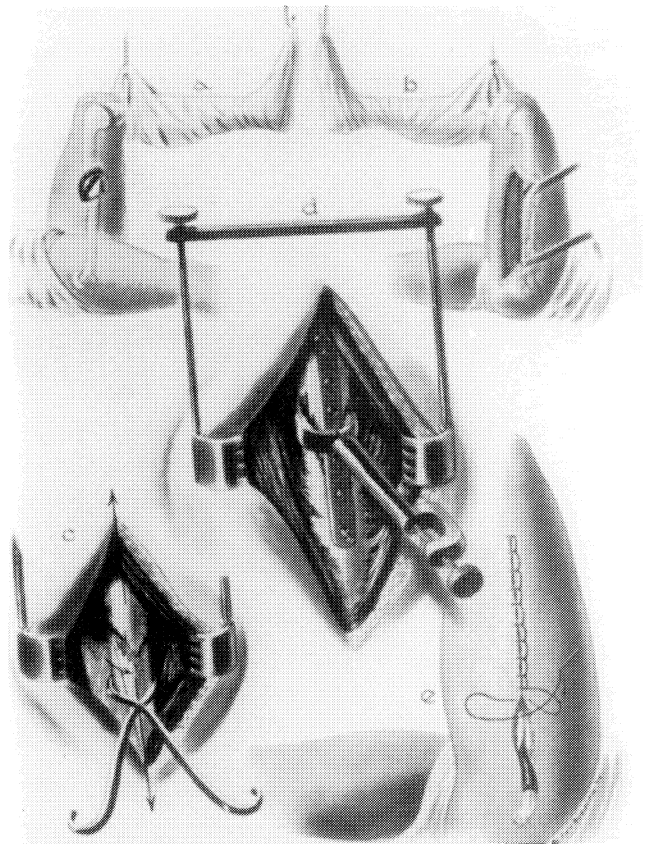


Figure 5. a. Compounding wound. b. Posterolateral approach. c. The fracture reduction. d. Internal fixation. e. Closure of the operative wound, with drainage. The artist failed to depict comminution and an obliquity in the fracture. (Reproduced with permission from *Annals of Surgery*²⁵)

Barbara Stimson, an orthopedist with the British army also wrote about the treatment of compound fractures in the Italian campaign and, although she did not present statistics, she did state that loose bone fragments should be retained and that open reduction and internal fixation could be done in selected cases through a separate incision ten days after wound closure⁵⁴.

The development of orthopaedic care in the European theater of operations (ETO) was very well described in a paper by Colonel Mather Cleveland. He arrived in the theater three weeks before the invasion of Normandy as the Senior Consultant in Orthopaedic Surgery. He noted that of 820 diplomats of the American Board of Orthopaedic Surgery, 301 entered the armed services, but only fifty-seven were available in the ETO, supplemented by ninety-five partially trained orthopedists, eighty-five trauma-general surgeons, and 175 army trained surgeons. The ever increasing cry for orthopaedic specialists was only silenced by the end of hostilities. On the basis of stateside experience with returning casualties, he felt that the closed method of

Trueta left much to be desired, and that a lack of skin traction had left many amputees with exposed bone. He also reported that surgeons returning from the Mediterranean felt that 1 1/2 hip spicas were much superior to the Tobruk plaster method used by the British for transporting femur fractures¹³. The Tobruk method incorporated a Thomas splint and skin traction into a plaster cast⁸. Prior to D-Day, the injuries seen were either in airmen returning from the skies over Europe or as a result of training in England. Cleveland reports an interesting study in which three groups of airmen with wounds were treated with debridement, primary closure, and either penicillin, sulfonamides, or no antibiotics. There was no difference in infection rate or healing between the three groups¹³.

The care of wounded during the invasion began right on the landing ships that evacuated them. Field hospitals were set up in Normandy within a few days, evacuation hospitals within a week, and the first "fixed" or base hospital in two months. Most extremity debridement was done at base hospitals, and the principles were as described above for the MTO, but Cleveland recommended leaving loose bone fragments in place. He suggested that wounds should be dressed, but that packing with Vaseline gauze should be avoided as it prevented drainage. After initial debridement and appropriate dressing at the evacuation hospital, the wounds were left undisturbed until arrival at the base hospital, where delayed primary closure could often be accomplished. He reported that Trueta's closed treatment was initially used, but stated: "This invites infection of bone and soft tissue and healing ensues slowly by second intention with massive granulations which develop into scar tissue." At six months a population of 3190 patients with open fractures had an 81.9 percent union rate¹³.

Amputations were all circular with subsequent skin traction, with a 9 percent mortality rate (25 percent in the Civil War). One and a half percent of all casualties had an amputation. Seventy-six percent were in the lower extremity; 66 percent traumatic and 20 percent vascular. An American soldier in World War II had a 0.175 percent chance of ending up an amputee versus 1.75 percent in the Civil War, a ten-fold improvement due at least in part to the improved medical care¹³.

Cleveland noted that excessive immobilization of hand wounds resulted in fibrosis and lost motion so soft dressings were primarily used. Single digit injuries were amputated much more readily as the war went on, as attempts at salvage often resulted in a less functional hand. Joint capsules were always closed after careful debridement. Penicillin was instilled in the joint post-operatively and repeated as necessary. Associated nerve injuries were explored and repaired three weeks after

wound closure, with bone shortening and fracture fixation as necessary at that time. Colonel Cleveland served in both World Wars and made these comments on the improvements of World War II: more surgeons with better training were available, care was more consistently of a high quality, evacuation was faster, more and better equipment was available, and more blood and plasma were used¹³.

Marshall Urist noted a large number of injuries to the hip joint, often from jeep accidents in the European theater. He tabulated fifty-eight injuries: forty from jeep accidents and the rest from other motor vehicles. Fifteen were hip dislocations, sixteen acetabular fractures, and twenty-seven fracture-dislocations. He published a cartoon showing the presumed mechanism in jeep accidents (Figure 6). Even the dislocations were put in traction for six weeks and kept nonweightbearing for six months! None of the otherwise healthy patients maintained this for more than three months, and results were similar to a group of civilians who had had an even shorter period of non-weightbearing. Although fairly common today, traumatic hip injuries were unusual at that time, and he stated that it would take a major civilian hospital fifteen years to acquire a similar study population. He did not discuss treatment of the fractures in this paper⁵⁶.

Weeden and Stein described their experiences with injuries and diseases of bone while caring for Chinese troops in the war. They treated 1108 fractures, of which 848 were open. Eighty-three percent of their fracture patients returned to active duty, and they had only three deaths in spite of the fact that their patients were malnourished, anemic, malarial, and infested with worms. They primarily used closed treatment but did some internal fixation, including a series of seven femur fractures with no infections. Osteomyelitis was treated with wide excision and delayed bone grafting. They used a three-way splint instead of a hip spica with excellent results⁵⁷.

Amputations were done at the lowest possible level in the field and revised proximally if necessary at the base hospitals. As the war progressed and the infection rate dropped, more definitive amputations were done in the field. Below knee prostheses were tibial and thigh weight-bearing³⁴. Harris wrote on amputations in 1944, recommending preservation of a flap for amputations through fractures and guillotine procedures for vascular or infectious indications. He denounced the use of skin traction in the face of infection. He suggested endbearing stumps, such as Syme or Stokes-Gritti (patella and pre-patellar skin swung under the end of the femur) when possible, and suggested that ideal lengths were 5.5 to six inches below the proximal end of the

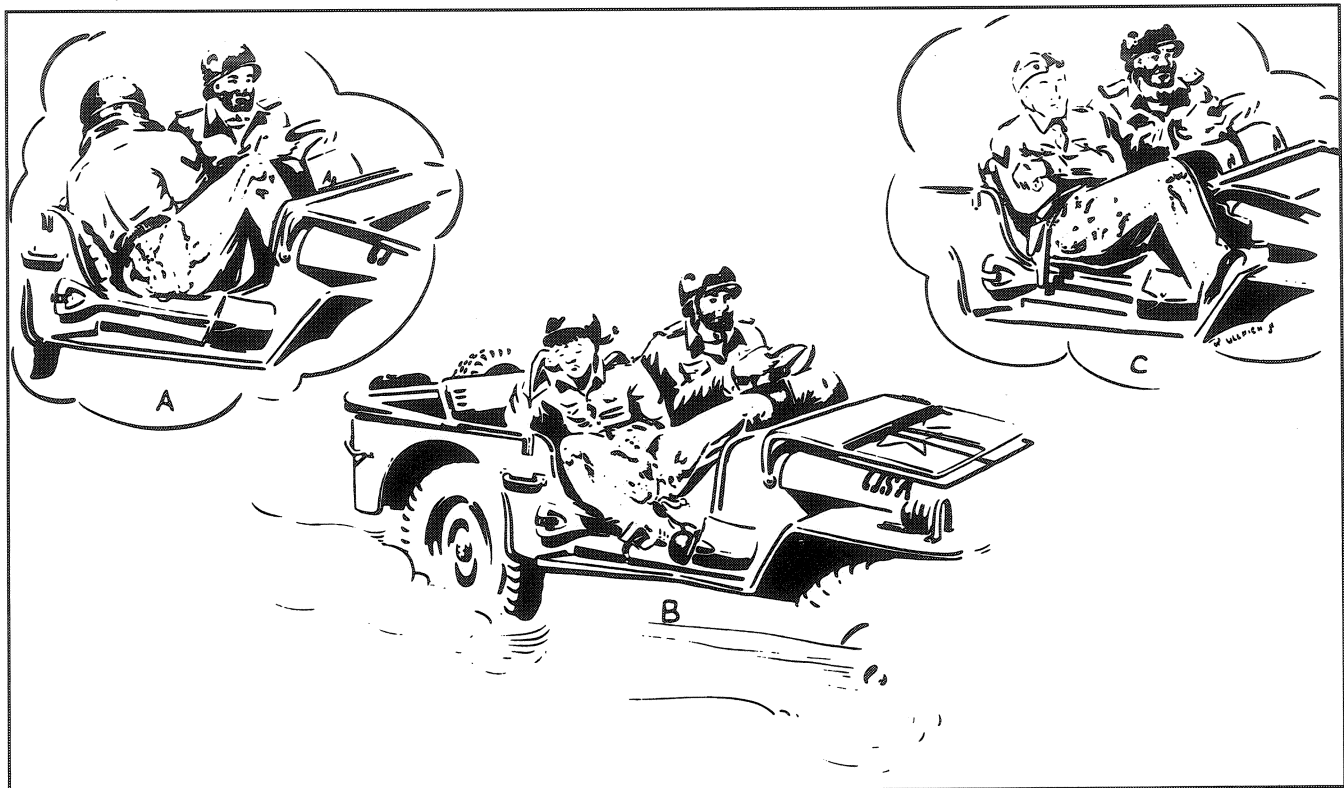


Figure 6. Artist's interpretation of patients' own descriptions of their positions and attitudes at the time of the injury. The accident was usually a head-on collision with resulting dislocations and fractures of the hip joint. A. inside-sitting position, the rider may have fallen out, striking the pelvis and incurring a fracture-dislocation or fracture. B. soldier sitting with legs abducted, in event of collision and dashboard blow on knee, may also have sustained fracture of the acetabulum or a fracture-dislocation. C. if rider was sitting with hips acutely flexed and adducted, the head of the femur may have dislocated with minimum or little damage to bony rim of the acetabulum. (Reproduced with permission from the *American Journal of Surgery*⁵⁶)

tibia for below-knee amputations and eleven inches below the greater trochanter for above-knee amputations²⁸.

LeVay, in his excellent history of orthopaedics, noted several other significant facts about World War II. The British tended to do more closed treatment of fractures while the Americans used more traction. The Americans had more fractures from parachuting than did the British until they abandoned their feet apart landing style for the British feet together technique. Shoulder harnesses decreased the incidence of spine fractures in fliers. Os calcis fractures were a frequent occurrence on torpedoed ships and had a poor prognosis. Femur and tibia fractures frequently led to invaliding. Some reconstructive procedures, such as anterior shoulder repair and meniscectomy were done if there was a fair chance of returning patients to duty. Spinal cord injuries were treated more intensively with frequent turning, passive range of motion, bladder programs, good nutrition, upper body exercise, and functional training. Peripheral nerves were sutured with some success, depending on the nerve, the location, and the gap involved³⁴.

A couple of papers covered the long-term management of the wounded, although this was not nearly as popular a topic as the acute and subacute care. Kelly wrote about the care of unhealed war wounds after the return of patients to the United States and reported successful use of free and pedicle skin grafts for wound coverage³³. Snedecor wrote about reconstructive surgery in patients with war fractures of the ankle and foot, again receiving patients in various stages of healing from all the theaters of war. First, he obtained healing of bone and soft tissues, then rehabilitated the patients with physical therapy and ambulation, and finally performed reconstructive procedures if necessary³².

KOREAN WAR

Of the wars America has fought in the twentieth century, the Korean war is often termed the "forgotten war." The relative paucity of main-stream literature is also reflected in medical journals, in spite of the fact that the war generated 103,284 wounded men. At the end of World War II the Russians and Americans couldn't agree on the mechanism for forming a gov-

ernment in Korea, culminating in a tragic division of the country at the thirty-eighth parallel. On June 25th, 1950, the North Koreans poured across the parallel in a lightening bid to reunite the country by force, and the Truman administration decided America had to intervene. Undertrained and poorly equipped occupation troops from Japan were rushed to Korea and subsequently ground up in the North Korean military machine. Not since Lexington and Concorde had America sent such ill-prepared men into combat. The United Nations forces withdrew into a small pocket near Pusan at the southern tip of the peninsula, and General MacArthur staged a brilliant amphibious assault at Inchon, crushing the North Koreans and advancing all the way to the Chinese Border. The Communist Chinese, fearing the possibility of an invasion, threw millions of their men into the battle, and the war finally stabilized back at the 38th parallel. In 1951, a truce was eventually agreed upon. American servicemen suffered terribly in Korea, often suffering frostbite from having no warm clothing during Korea's fierce winter. In the first year it was a war of rapid maneuver, but for the last two it was trench warfare similar to World War I, and with no peace agreement, the 38th parallel remains a war zone today.

The changes from World War II were primarily in two areas. The first was battlefield evacuation. The tiny Bell H-13 helicopter had a stretcher attached to each skid and was used to transport patients directly from the battlefield to surgical units dramatically improving both the speed and stability of patients in transport. The second improvement was in the area of arterial repairs.

There were a number of studies of vascular repairs. Spencer and Grewe studied ninety-seven arterial injuries, of which eighty-seven were successfully repaired. They still had a 22 percent amputation rate, mostly from femoral or popliteal injuries⁵³. LeVay noted ligature gave an amputation rate of almost 50 percent in both wars, but repair had an amputation rate of 36 percent in WWII and only 13 percent in Korea. This fell further to 8 percent in Vietnam. He noted that internal fixation was associated with reduced infection rates and improved the outcome of infected fractures³⁴.

Adams described eight patients with close range rifle wounds to the hand, resulting in small caliber entrance wounds on the palm and large stellate wounds on the dorsum. Even in cases without metacarpal fracture, extensive soft tissue stripping was found. These injuries were treated with debridement, approximation of tendon and skin edges, and penicillin. A compressive dressing was applied. Elevation was found to be helpful in treating swelling, but sympathetic block was not. Delayed primary closure was performed at about eight

days for the dorsal wounds, but the palmar wounds were allowed to granulate. Motion was encouraged after closure, but patients were transferred elsewhere and no follow-up was possible¹.

VIETNAM

The most recent war in which American forces sustained a substantial number of casualties was the Vietnam war. American involvement gradually escalated over ten years, culminating in a large scale pull-out in 1973 and collapse of the South Vietnamese in 1975. It was a war fought by small infantry units in the jungle, leading to a higher than usual proportion of rifle bullet wounds. Evacuation speed was even higher, with the ubiquitous "Huey" sometimes transporting wounded soldiers from the middle of a firefight directly to a permanent hospital or hospital ship, allowing soldiers who would previously have been "killed in action" to arrive at the hospital alive²³. In spite of this, hospital mortality was only 1.81 percent versus 3.3 percent for WWII and 2.4 percent for the Korean war. Thirty-one percent of wounded soldiers arrived to the hospital within one hour of wounding and 86 percent within four hours, versus an average of four to six hours in Korea and twelve to fifteen hours in the WWII Italian campaign. Twenty-six percent were wounded by small arms, 37 percent by artillery, 10 percent by mines, and most of the rest in accidents. Twenty-five percent had injuries to bone and four percent of these had infections²⁷. The rifles, and especially the M-16 used by American troops, were of a higher velocity than the weapons of previous wars, leaving behind much greater tissue damage in their paths. Studies of these wounds are common in the war-time literature.

DeMuth and Smith noted that the zone of injury of a high velocity missile (greater than 2,000 fps) is much larger than the permanent cavity left by the bullet, and is related to the energy imparted by the round (proportional to bullet weight and to the square of the velocity). They noted that muscle damage may not be apparent at initial debridement, and wounds should be reexplored after several days. Casting, traction, and intramedullary nailing were all acceptable for fixation of long bone fractures¹⁹. The .223 round from the M-16 (5.56 mm) with its 3,250 fps velocity caused severe wounds, surpassing the larger but slower (2,330 fps) 7.62 mm rounds of the AK-47 rifle used by the Viet Cong. Figure 7 is a reprint of pictures comparing low velocity .45 and high velocity .223 wounds of the thigh. The only gunshot wounds worse than those caused by .223 rounds were caused by .50 caliber (12.7 mm) machine gun rounds, the largest solid missile used (velocity of 2,900 fps). Sixty-one percent of the soldiers were



Figure 7A. A low velocity through and through 45 caliber gunshot wound of the posterior thigh.

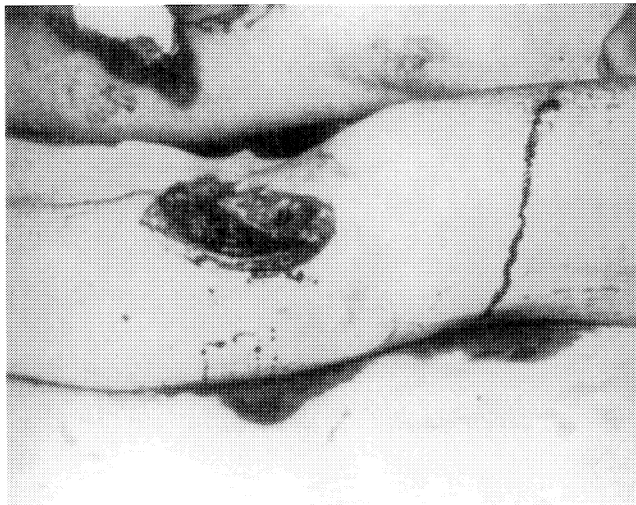


Figure 7B. M-16 (.223 caliber or 5.56 mm) through and through high velocity gunshot wound of the left lower extremity. (Reproduced with permission from *Military Medicine*⁴⁸)

returned to duty with an average hospital stay of sixteen days⁴⁸.

Garrick compared the wounds caused by various types of ordnance. Mines and Booby traps caused an upward blast, resulting in marked contamination and debris much more proximal in the tissue than the entrance wounds. Grenades caused cleaner wounds, and the energy imparted depended on the proximity of the blast. Artillery fragments varied in size and speed, and thus wound potential. All of the above could cause secondary missiles, such as fragments of wood or even bone from another nearby soldier, and could bring pieces of dirt or clothing into the wound. He noted that bullets may cause a wide zone of damage, and may not have straight paths. Punji stake wounds were contaminated and low velocity, but were sometimes combined with blast wounds²⁴. Jabaley and Peterson wrote about

the early treatment of war wounds of the hand and forearm in Vietnam. The patients were observed for several days after delayed primary closure or fourteen days after arterial repair. Irrigation, debridement, fasciotomy, and vascular repair were done initially; reexploration and reparative procedures were done three to five days later, including fixation of fractures. Nerve and tendon repairs were delayed until the wounds healed, therefore after the patients had left the authors' care³¹.

Burkhalter and colleagues looked at 135 patients with delayed primary closure of hand wounds and found three infections after closure. Most were high velocity missile wounds with a large zone of injury. They noted that debris was often sucked in by the missile or entered during evacuation. The delay before treatment for these wounds was usually eight to ten hours. Careful initial debridement was done, as well as carpal tunnel releases or fasciotomies if needed. A list of deficits was made for planning of later repairs. The hand was elevated for three to five days and then reexplored. Internal fixation or joint capsule repair was done, and the wounds closed if they were clinically free of infection⁹. Baksic and colleagues reported on the use of meshed skin grafts for coverage of war wounds and found that the average survival of the graft was 81.6 percent, and that 88 percent of patients had more than 75 percent survival. They also noted that results improved with experience³.

Finally, Rich and colleagues looked at 1000 arterial injuries, of which 459 were repaired with a vein graft, 377 with anastomosis, eighty-seven with lateral suture, fifteen with ligation, four with prosthetic graft, three with autogenous arterial graft, and fifty-five with an unknown type of repair. The amputation rate in their study was 13.5 percent, and popliteal and femoral arteries had the poorest outcomes⁴⁹. Cohen, Baldwin, and Grant reported that the risk of limb loss from vascular injury had dropped from 50 percent in World War II to 13 percent in Korea to 8 percent in Vietnam, and that the main problems contributing to failure and amputation were massive soft tissue or vein loss, repeat surgeries, unstable fractures, inadequate debridement, or unsuspected injuries at lower levels¹⁵.

PERSIAN GULF AND THE FUTURE

The most recent major conflict, the Persian Gulf War, is remarkable for having produced so few casualties, only 148 combat and 145 non-combat injuries. Although several articles have been written by medical personnel involved in covering allied troops, there were not enough injuries in the field of orthopaedics to merit reporting, an unquestionable blessing. The desert terrain of the Gulf War battlefields was primarily respon-

sible for this and the recent, rapid drawdown in U.S. military forces suggests a confidence in American technology regarding their ability to overcome large enemy forces with minimal casualties. A future war in Europe or, more likely, Asia would probably be much more costly. Hopefully, we will be more prepared than we were in Korea, where the price of unpreparedness was paid by civilians and young soldiers. With the large volume of high energy trauma in civilian life today, battle injuries would be more familiar to us than to the surgeons of World War I. The principles of care for high energy, contaminated injuries that military surgeons painfully developed are now universally accepted. The post-Vietnam generations of orthopaedic surgeons have been spared from the specter of battlefield trauma. It is hoped that there are no such experiences in the future, although a study of history should temper one's optimism. If called to service, the legacy of past war surgeons and experience with civilian trauma should provide preparation at least as well, if not better, than those who developed this knowledge in previous conflicts.

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BILATERAL OSTEOFIBROUS DYSPLASIA: A REPORT OF TWO CASES AND REVIEW OF THE LITERATURE

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Osteofibrous dysplasia is a fibro-osseous proliferation which affects the bones of children, almost always before they are ten years old. The distinctive feature of this lesion is its predilection for one site — the anterior cortex of the tibia. Almost always, the disease is unilateral. Osteofibrous dysplasia is not uncommon. Many cases have been reported in the literature. In addition to reports of one or two cases, the nine large studies of this lesion published to date document 219 cases^{2,6,11,15,17,18,20,21,22}. Many cases go unrecognized because they are misdiagnosed as fibrous dysplasia or congenital tibial bowing. Furthermore, because some lesions are asymptomatic, they are not diagnosed at all.

Bilateral osteofibrous dysplasia, by contrast, is extremely rare. We are aware of only five cases that have been documented in the literature. We wish to report our experience with two additional cases and explore the significance of bilaterality to the pathogenesis of this disorder.

Case 1

A four week old female infant, the product of a forty week gestation, was noted to have non-tender bowing of her left tibia at the time of birth. Radiographs of her lower extremities showed metaphyseal-diaphyseal lucent defects in both proximal tibias with anterior bowing and periosteal reaction of the left tibia. The radiographic diagnosis was bilateral osteofibrous dysplasia. On follow-up examination, the patient developed shortening of the right tibia and progressive bowing of the left tibia. At eight months of age, the lucencies in the right tibia had grown (Figure 1) and there was a patho-



Figure 1. Right tibia of patient 1 at age eight months.

logic fracture through the lesion on the left (Figure 2). A resection of the left tibial lesion with allograft replacement was performed. Tissue removed from the lesion was consistent with osteofibrous dysplasia, although cytokeratin stains were negative. The allograft has failed to heal in the two years following surgery. In fact, lytic lesions have appeared extensively throughout the graft. Further surgery is planned. The right lesion continues to grow slowly, although this tibia has remained structurally intact.

Case 2

A three year old boy was first observed to have right anterior tibial swelling at age two. Radiographs of his long bones showed cortex-based lucencies in both the left (Figure 3) and the right (Figure 4) tibias. The radiographic diagnosis was bilateral osteofibrous dysplasia. Family history was remarkable for a mother with spherocytosis and a father with β -thalassemia minor. Hematologic work-up revealed microcytosis without ane-

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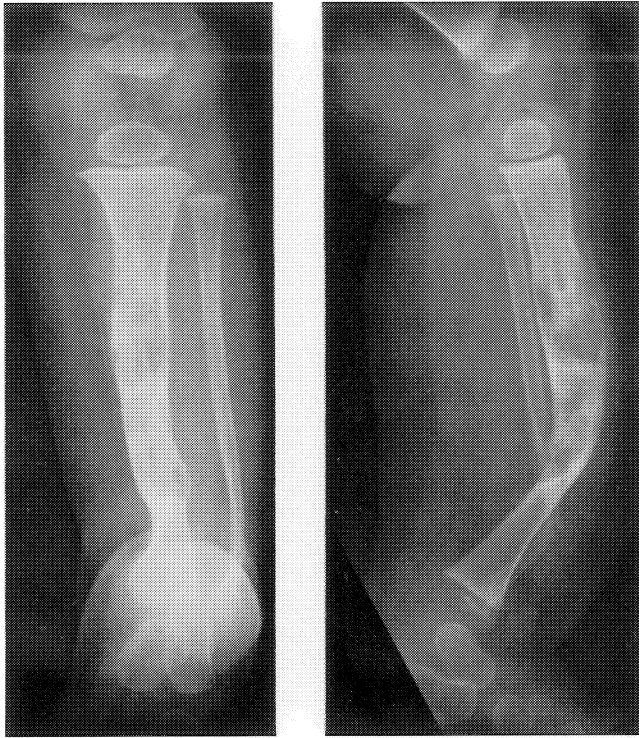


Figure 2. Left tibia of patient 1 at age eight months. A pseudarthrosis is present.

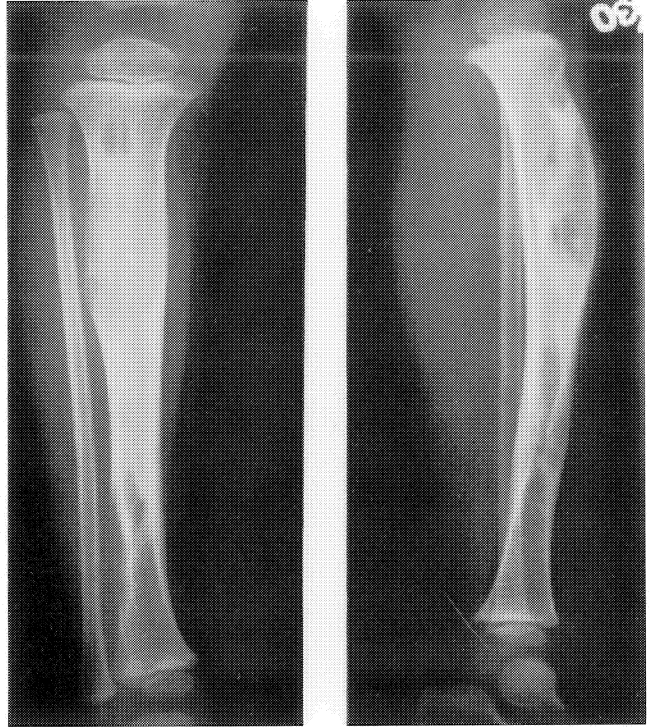


Figure 4. Right tibia of patient 2 at age three years.

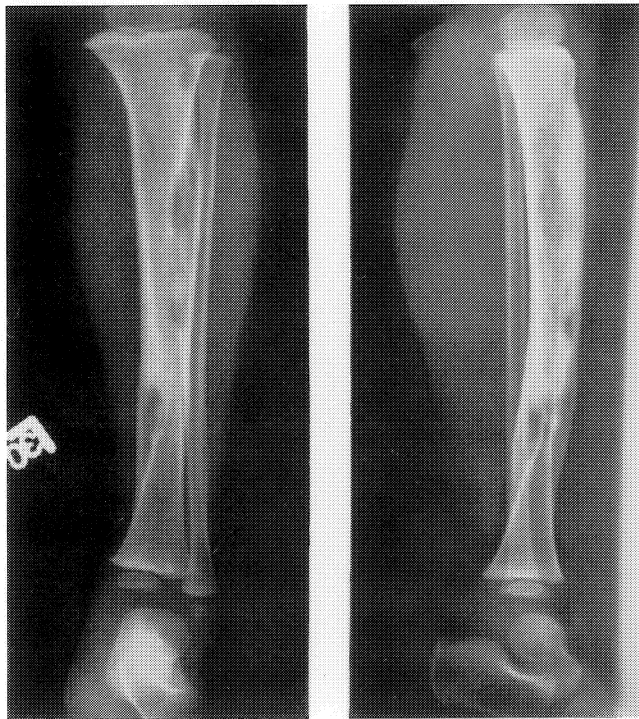


Figure 3. Left tibia of patient 2 at age three years.



Figure 5. Left tibia of patient 2 at age four years. There is a large lytic component.



Figure 6. Right tibia of patient 2 at age four years.

mia consistent with thalassemia trait. By age four, lesions in both tibiae had grown, the left (Figure 5) greater than the right (Figure 6). An expansile lytic component of the left lesion prompted a biopsy. The pathologic diagnosis was osteofibrous dysplasia (Figure 7). Keratin stains were positive. The patient subsequently developed a fracture through the left lesion which is slowly healing with an external brace.

Discussion

Osteofibrous dysplasia was first described by Frangenheim in 1921 as *congenital osteitis fibrosa*¹². In 1936, Compere noted a relationship between this lesion, which he called *localized osteitis fibrosa*, and congenital pseudarthrosis¹⁰. This relationship was further explored by Aegerter in 1950, who believed that this tibial lesion was a form of fibrous dysplasia¹. He postulated that the tibial process was a manifestation of neurofibromatosis, a genetic disease sometimes complicated by congenital pseudarthrosis. In 1966, Kempson's accurate histologic and electron microscopic study distinguished this process from fibrous dysplasia¹⁶. He called this lesion *ossifying fibroma of long bones* because of the histologic similarity to ossifying fibroma of the facial bones. However, it was not until Campanacci's description of thirty-five cases in 1976⁵ that the clinical, radiographic, and histologic spectrum of this disorder was recognized.

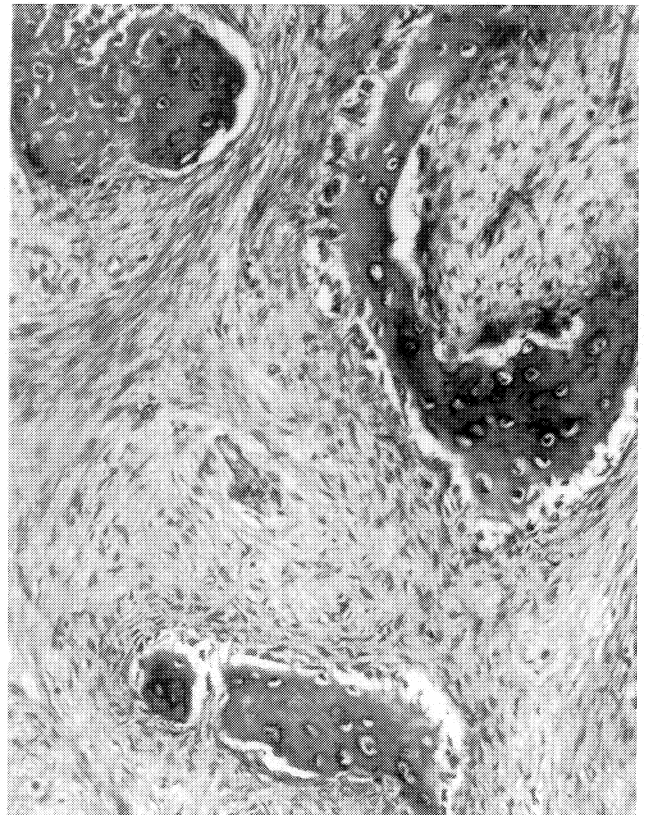


Figure 7. Photomicrograph of tibial biopsy of patient 2. A fibro-osseous lesion with woven bone lined by osteoblasts. (H & E, x320)

Campanacci called this lesion osteofibrous dysplasia to emphasize its non-neoplastic nature.

Osteofibrous dysplasia has a distinct clinical presentation. This disorder is almost exclusively limited to the tibia. Most patients present between five and ten years of age. In rare cases, a lesion may not be recognized until the patient is about age thirty. Also, lesions are sometimes diagnosed in the neonatal period. Patients typically present with painless swelling over the mid-tibia, and anterior bowing is often present.

The distinctive radiographic feature of osteofibrous dysplasia is multiloculated, intracortical lucencies which are almost always centered on the anterior diaphyseal cortex. Varying amounts of sclerotic bone are usually present. Radiographs reveal a spectrum of severity. In some cases, lesions are small (one to two cm) and remain stationary. Other lesions involve most of the tibial shaft. Often, in these severe cases, the lesions expand and coalesce until skeletal growth is complete. In addition, the ipsilateral fibula may be focally involved in as many as 17 percent of cases²². Twelve to 33 percent of patients develop a pseudarthrosis^{6,20}.

Histologically, osteofibrous dysplasia is characterized by irregular new bone trabeculae amidst cellular fibrous tissue, a pattern reminiscent of fibrous dysplasia. How-

ever, unlike the trabeculae of fibrous dysplasia which seem to appear *de novo* in the fibrous stroma, the trabeculae of osteofibrous dysplasia are lined by plump osteoblasts. The trabeculae of osteofibrous dysplasia are arranged in a zonal pattern; the center of the lesion is predominantly fibrous while the periphery contains more new bone. This zonal pattern is not a feature of fibrous dysplasia. The most distinctive histologic feature of osteofibrous dysplasia is keratin positive cells. Ninety-three percent of lesions contain scattered spindle cells which stain immunocytochemically for cytokeratin²². Having no other epithelial features, these cells are not apparent with a routine hematoxylin and eosin (H & E) stain.

The predilection of osteofibrous dysplasia for the tibia and the presence of keratin positive cells has led to considerable speculation about its relationship to adamantinoma^{11,21}. Adamantinomas also occur almost exclusively in the tibia and contain keratin positive cells, although to a much greater degree. Moreover, adamantinomas often contain fibro-osseous tissue identical to the characteristic tissue of osteofibrous dysplasia. The exact nature of this relationship is uncertain. However, despite many similarities, the behavior of these two lesions is quite different. Adamantinomas are malignant neoplasms which occur in patients who are almost always older than age twenty¹¹. Although, they have some radiographic similarities to osteofibrous dysplasia, ar-

reas of cortical destruction and medullary canal involvement are common. In addition, soft tissue masses are often present, and metastases occur in about 30 percent of cases¹⁴.

Osteofibrous dysplasia, by contrast, is a non-neoplastic, self-limited process. There has been no reported case of transformation of this disease into an adamantinoma. Lesions grow slowly until skeletal maturity; thereafter, they stabilize. Small lesions sometimes disappear spontaneously. Large lesions, even those which have resulted in architectural deformity, stop growing. Therefore, if at all possible, surgical removal of these lesions should be avoided⁶. Curettage during the proliferative phase is almost always followed by recurrence¹⁸. Surgery should be reserved for those lesions with severe deformity or pseudarthrosis.

Recently, a new point on the osteofibrous dysplasia—adamantinoma spectrum has been identified. Some lesions, clinical and radiographically identical to severe osteofibrous dysplasia, contain discrete nests of epithelial cells, although they are not as prominent as in adamantinomas. These lesions, called differentiated adamantinomas, may represent burned out cases of severe osteofibrous dysplasia^{11,15}. Alternatively, they may represent an intermediate stage between osteofibrous dysplasia and classic adamantinoma.

In nearly all cases, osteofibrous dysplasia involves only one tibia. The involvement of both tibias has been

TABLE 1
Bilateral Osteofibrous Dysplasia
Summary of Cases

	Sex	Age	Treatment	Follow-up	Status
1 Case 1	F	4 weeks	(L) resection with allograft (R) no treatment	2 years	failed allograft active lesion
2 Case 2	M	3 years	(L) Bracing (R) no treatment	2 years	active lesion active lesion
3 Companacci and Laus (6)			No clinical information		
4 Companacci and Laus (6)			No clinical information		
5 Nakashima et al. (18)	M	2 years 3 years	(L) curettage & bone graft (R) curettage	10 years	healed healed
6 Castellote et al. (9)	F	6 years	(L) Excision (R) No treatment	6 years	healed stable
7 Ozaki et al. (19)	F	18 months	(R) curettage & bone graft (L) no treatment	5 years	healed resolved spontaneously

reported only five times (Table 1). Two of Companacci and Laus' thirty-five patients had bilateral involvement⁶. In one of these patients, both fibulas were also involved. No clinical details were provided. Nakashima et al. reported twelve cases of osteofibrous dysplasia, one of which had bilateral disease¹⁸. The lesion in one tibia was recognized at age two and the other at age three. Both lesions healed after curettage (one with bone grafting). In a report by Castellote et al. of two patients with osteofibrous dysplasia, one had bilateral disease⁹. The patient was a six year old girl with small lesions. One lesion was excised and the other was followed. By age fourteen, both tibias showed only mild anterior bowing with foci of sclerosis; a few minute lytic areas were present on one side. The final report of bilateral osteofibrous dysplasia was by Ozaki et al.¹⁹. These authors described an eighteen month old girl with bilateral tibial lesions as well as lesions in both ulnas and one fibula. Curettage of one tibial lesion resulted in healing. All other lesions disappeared spontaneously.

The two patients we are reporting have severe disease in one tibia and moderate disease in the other. The severely involved tibia in both patients fractured. Patient 1 required an allograft which has not healed. Patient 2 fractured through a lytic focus that was biopsied. This fracture is healing, but the patient requires external bracing. Because our patients are now only age three and five and one-half, we expect continued growth of these lesions. From these two patients and the five previously reported, it appears that lesions in bilateral osteofibrous dysplasia show a spectrum of severity similar to mono-stotic disease.

Bilaterality in osteofibrous dysplasia is further evidence of a close relationship to fibrous dysplasia, a relationship that suggests a similar pathogenesis. Histologic similarities have long been recognized. Both lesions consist of woven bone in a cellular fibrous background. In fact, a few lesions of osteofibrous dysplasia evolve into a radiographic pattern more characteristic of fibrous dysplasia²⁰. Furthermore, similar cytogenetic abnormalities have been demonstrated in lesions of both osteofibrous dysplasia and fibrous dysplasia^{3,4}. These observations, coupled with histologic similarities, have led to the hypothesis that osteofibrous dysplasia is a variant of fibrous dysplasia^{7,20}. Bilaterality in osteofibrous dysplasia, as demonstrated by these seven cases, possibly correlates with the polyostotic variant of fibrous dysplasia. Twenty percent of patients with fibrous dysplasia have involvement of more than one bone. Some of these patients also have endocrine abnormalities and cafe-au-lait spots, a disorder known as the McCune-Albright syndrome. Polyostotic fibrous dysplasia is now known to be a genetic disorder caused by a mosaic state

of an activating mutation in the GNAS 1 gene. This gene codes for an adenosinetriphosphate (ADT)-dependent G protein²³. In addition, over expression of the C-fos proto-oncogene has been noted in the bone of eight patients with fibrous dysplasia⁸. The many similarities of osteofibrous dysplasia and fibrous dysplasia suggests that osteofibrous dysplasia may also have a genetic cause.

Another association which further suggests a genetic origin of osteofibrous dysplasia is an overlapping feature with neurofibromatosis 1—the development of pseudarthrosis. Neurofibromatosis 1, the most common single gene disorder in humans, is due to a mutation in the NF-1 tumor suppressor gene located on chromosome seventeen¹³. At least 50 percent of cases of congenital pseudarthrosis are associated with this genetic disorder. Pseudarthrosis also occurs in as many as one-third of the patients with osteofibrous dysplasia. Furthermore, the spectrum of severity of pseudarthrosis associated with neurofibromatosis parallels, in many ways, the spectrum of bone changes in osteofibrous dysplasia. Some lesions are very mild and present only with anterior tibial bowing. Other lesions are more severe and are fractured at birth. Often, these severe lesions do not heal despite a wide variety of surgical interventions. It is highly probable that many lesions classified as congenital pseudarthrosis, especially those with cystic change, are actually examples of osteofibrous dysplasia.

In summary, we have described two cases of bilateral osteofibrous dysplasia, a presentation that has been noted only five other times. The occurrence of bilaterality suggests a kinship with polyostotic fibrous dysplasia, a known genetic disorder. This kinship, as well as similarities to congenital pseudarthrosis seen in neurofibromatosis 1, suggests a possible genetic origin of osteofibrous dysplasia.

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FATALITY FROM DISSEMINATED INTRAVASCULAR COAGULATION COMPLICATING TOTAL HIP ARTHROPLASTY: A CASE REPORT

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ABSTRACT

Following a routine hybrid total hip arthroplasty in an eighty-two year-old female, profuse bleeding occurred through the suction drain immediately postoperatively. The patient was found to have acute disseminated intravascular coagulation. Despite early recognition and treatment the patient died fourteen days later.

Disseminated intravascular coagulation (DIC) can be a fatal disorder with mortality rates approaching 80 percent in severe disease¹. DIC has been reported to occur as a result of numerous diverse etiologies, but has rarely been reported as a complication of total hip arthroplasty. The following is a case of a healthy, elderly woman who underwent elective total hip arthroplasty and developed DIC postoperatively and subsequently died.

Case Report

An eighty-two year-old white female underwent elective right total hip arthroplasty for increasing pain secondary to degenerative arthritis which was refractory to conservative treatment. She also had radiographic evidence of degenerative changes in her knees and her spine. Her past medical history was remarkable for controlled hypertension, mildly elevated liver enzymes, hematuria with a negative urological work-up, vasovagal syncope, gout and a previous left knee Baker's cyst. She denied a history of cardiovascular, pulmonary or bleeding disorders, and at the time of surgery was without complaints other than right hip pain. Her medications included: Monopril 20 mg daily, enteric coated aspirin 80 mg daily, colchicine 0.6 mg daily, Motrin 400 mg daily, vitamin D 600 mg twice daily, vitamin C 500 mg daily, and a multivitamin. The patient had been under general anesthesia in the past without complications

for an appendectomy and a tonsillectomy/adenoidectomy. She had no known drug allergies.

Laboratory studies at the time of admission revealed a hemoglobin level of 12.7 g/dl, a prothrombin time (PT) of 11.0 seconds, a partial thromboplastin time (PTT) of 25.0 seconds, and a platelet count of 138,000/mm³. Her general screen was remarkable for an increased alkaline phosphatase of 156, lactate dehydrogenase of 239 and AST of forty. The remainder of her presurgical blood work was within normal limits.

The patient was anesthetized under general anesthesia and was monitored intraoperatively via ECG monitoring, indirect blood pressure monitoring, pulse oximetry, and serial arterial blood gas measurements. Her intraoperative course was relatively stable with the exception of several episodes of hypotension to 90/50 mmHg which responded adequately to fluids (one unit packed red blood cells, 500 cc colloid and 500 cc Lactated Ringers) and two 100 mg boluses of phenylephrine. A hybrid total hip replacement was performed, and the patient was taken postoperatively to the Post Anesthesia Critical Care Unit (PACCU) with a hematocrit of 28 percent and an arterial blood gas of 7.43/35/149/0/24. Operative time was 105 minutes and blood loss was estimated at 600 cc. No anticoagulant therapy was administered intraoperatively other than pneumatic compressive stockings.

Upon arrival to the PACCU, her blood pressure remained stable for a few minutes before suddenly dropping to 80/60 mmHg. A normal blood pressure was restored with fluids (two units of packed red blood cells and 1000 cc of Lactated Ringers), two doses of phenylephrine (100 mg) and two doses of ephedrine (five mg). The patient remained stable for approximately forty-five minutes. A chest x-ray was unremarkable, an ECG showed no acute changes, and an arterial blood gas revealed 7.40/26/167/-2/23. Her Constavac^R drain was noted to have significant (700 cc) sanguinous output over the forty-five minutes, urine output was minimal, and her platelets had fallen to 116,000/mm³. Suction to the Constavac^R drain was stopped to avoid further blood loss. The presumptive diagnosis was DIC; there was no suspicion of trauma to a major vessel during the surgery. A right subclavian line was placed which revealed

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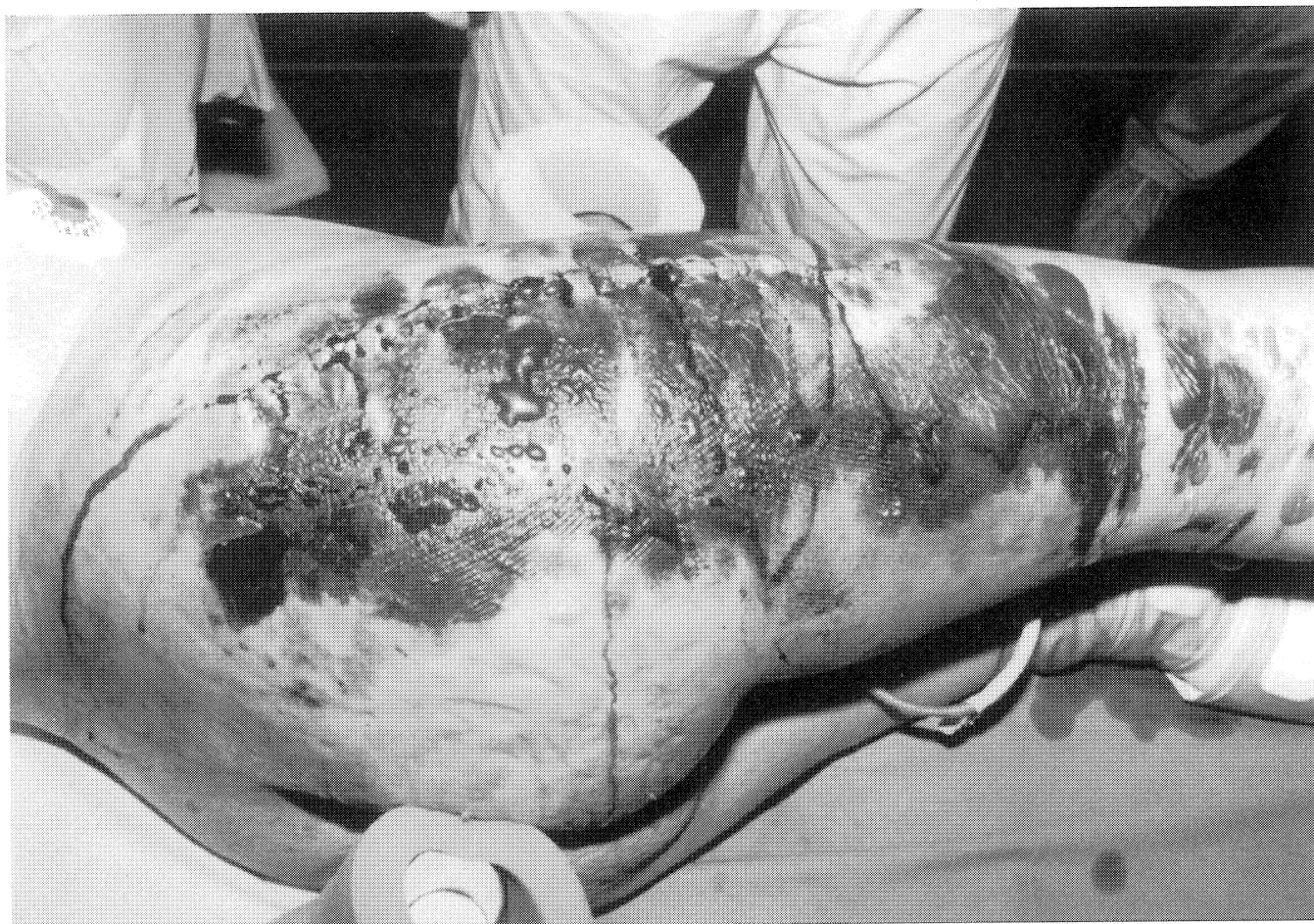


Figure 1. Incision site at the time of hematoma evacuation, post-operative day three. Note the edema and hemorrhagic blistering of the skin over the incision site due to the acute accumulation of blood and expansion of the thigh circumference.

a central venous pressure of negative four centimeters of water. A dopamine drip was started but was quickly discontinued due to severe tachycardia. A phenylephrine drip was then begun at 125 $\mu\text{g}/\text{hr}$ to maintain a systolic blood pressure greater than 90 mmHg.

The patient was transferred to the Surgical Intensive Care Unit (SICU) three hours postoperatively with systolic blood pressures in the sixties and seventies. She had received seven units of packed red blood cells and four units of fresh frozen plasma (FFP) to replace her 2375 cc Constavac[®] drain output. Overnight she developed worsening metabolic acidosis with a blood gas of 7.12/48/207/-12, and her PTT and fibrin degradation products (FDP) remained elevated at sixty-eight seconds and greater than eighty mg/dl respectively. Along with a decreased fibrinogen level of 121 mg/dl (normal 160-340 mg/dl), her hematocrit dropped to 24 percent and her platelets to 33,000/ mm^3 . Heparinization was begun at that time. She continued to require phenylephrine to maintain a blood pressure of 90/60 mm Hg, and

repeated attempts to start a dopamine infusion for renal protection continued to produce excessive tachycardia and atrial fibrillation. Due to the patient's worsening acid-base balance and to protect her airway, she was re-intubated with an oral endotracheal tube. The patient was noted to have a sciatic nerve palsy, most likely from the massive hematoma.

Over the course of the next three days, the patient required nineteen units of packed red blood cells, twenty units of FFP and five packs of platelets in order to maintain a hematocrit greater than 30 percent, PT less than fifteen seconds, and platelets greater than 75,000/ mm^3 . She continued to have significant output in her drains, and ooze from her central and peripheral intravenous line insertion sites. Her renal function remained poor with a urine output of twenty cc/hr under forced diuresis with Lasix, and her BUN and creatine were elevated at twenty-one and 2.1 secondary to myoglobinuria induced acute renal failure. The patient's AST and ALT were also elevated at 3326 and 1527 respec-

TABLE 1
Coagulation Studies and Factor Levels in the Immediate Postoperative Period

	Normal	Preop	Immed. Postop	3 Hrs. Postop	5 Hrs. Postop	24 Hrs. Postop
Hct (%)	35-40	36	28	24	16	23
Platelets (K/mm ³)	151-400	138	116	33	120	73
PT (sec.)	9-13	11	15	16	15	14
PTT (sec.)	22-37	25	—	77	63	39
FDP (µg/ml)	0-9	—	—	>80	>80	>80
D-Dimer (µcg/ml)	0-0.1	—	—	—	—	>4.0

tively, and were believed to be due to “shock liver” from the prior episodes of hypotension. Her diminished renal function, excessive fluid from transfusion, and poor nutritional status presented clinically as anasarca which was treated with continuous veno-venous hemofiltration, central venous nutrition and nasogastric tube placement.

On postoperative day three, she was taken back to the operating room for evacuation of the wound hematoma which had caused the sciatic nerve palsy (Figure 1). The procedure was without complications. Approximately eight liters of consolidated hematoma were evacuated, and her bleeding was adequately controlled. However, upon return to the SICU her blood pressure was 90/50 mmHg. A 500 cc bolus of Plasmanate precipitated a sudden drop in blood pressure to 53/31 mmHg. A normal blood pressure was restored with 500 mg boluses of phenylephrine, and the patient was started on a Levophed drip. Throughout the day, she required six units of FFP, one pack of platelets and four units of packed red blood cells every eight hours in order to maintain adequate hematologic parameters. (Table 1 illustrates the first twenty-four hour coagulation studies.)

By the sixth postoperative day, her PTT had improved to forty-five seconds, PT to thirteen seconds and fibrinogen level to 382 mg/dl. There was no evidence of clinically significant bleeding, and the patient did not require further blood transfusions. However, she had become markedly obtunded, icteric, anuric and remained ventilator and vasopressor dependent. Serial labs showed increasing direct and indirect bilirubin, PT, PTT and ammonia levels which suggested that she was developing worsening hepatic function. Despite aggressive therapy, care was withdrawn on postoperative day fourteen at the request of the family, and the patient died. A postmortem examination was not performed.

DISCUSSION

Deep venous thrombosis and subsequent pulmonary embolism is the most common cause of death in the immediate postoperative period following total hip replacement. Deep vein thrombosis has been reported in up to 70 percent of patients in the absence of anticoagulation therapy². The hypercoagulable state and increased circulating tissue thromboplastins following routine total hip arthroplasty contribute to this occurrence. Although the exact etiology of DIC remains unknown, it is believed that the release of tissue thromboplastins into the systemic circulation or diffuse endothelial damage is the common triggering factor as demonstrated in Figure 2³. However, an autoimmune reaction to the acrylic bone cement has also been hypothesized⁴. The inciting event of this patient's course of DIC can only be postulated. In retrospect, it was suspected that intermittent intraoperative hypotensive episodes combined with the release of tissue thromboplastin during the total hip replacement procedure (i.e. femoral and acetabular reaming) and preexisting mild hepatic dysfunction triggered this event. Her progressive hepatic and renal insufficiency were undoubtedly due to microthrombus-induced, end-organ ischemia.

The diagnosis of DIC can usually be made from the patient's systemic signs and symptoms as well as their abnormal laboratory profile. These patients often have fever, hypotension, acidosis, proteinuria, hypoxia, petechia and purpura, and are bleeding from at least three unrelated sites. Due to systemic microvascular thrombosis, the cardiovascular, pulmonary, renal and central nervous systems are especially prone to ischemic episodes with their resulting sequelae⁵. While the PT, PTT, fibrinogen and FDP levels have traditionally been followed, it is currently recommended that the D-dimer, Antithrombin III (AT-III), Fibrinopeptide A and FDP levels be monitored in order to maximize diagnostic reliability⁶. The D-dimer is specific for fibrin degradation, whereas the FDP is derived from either fibrin or fibrino-

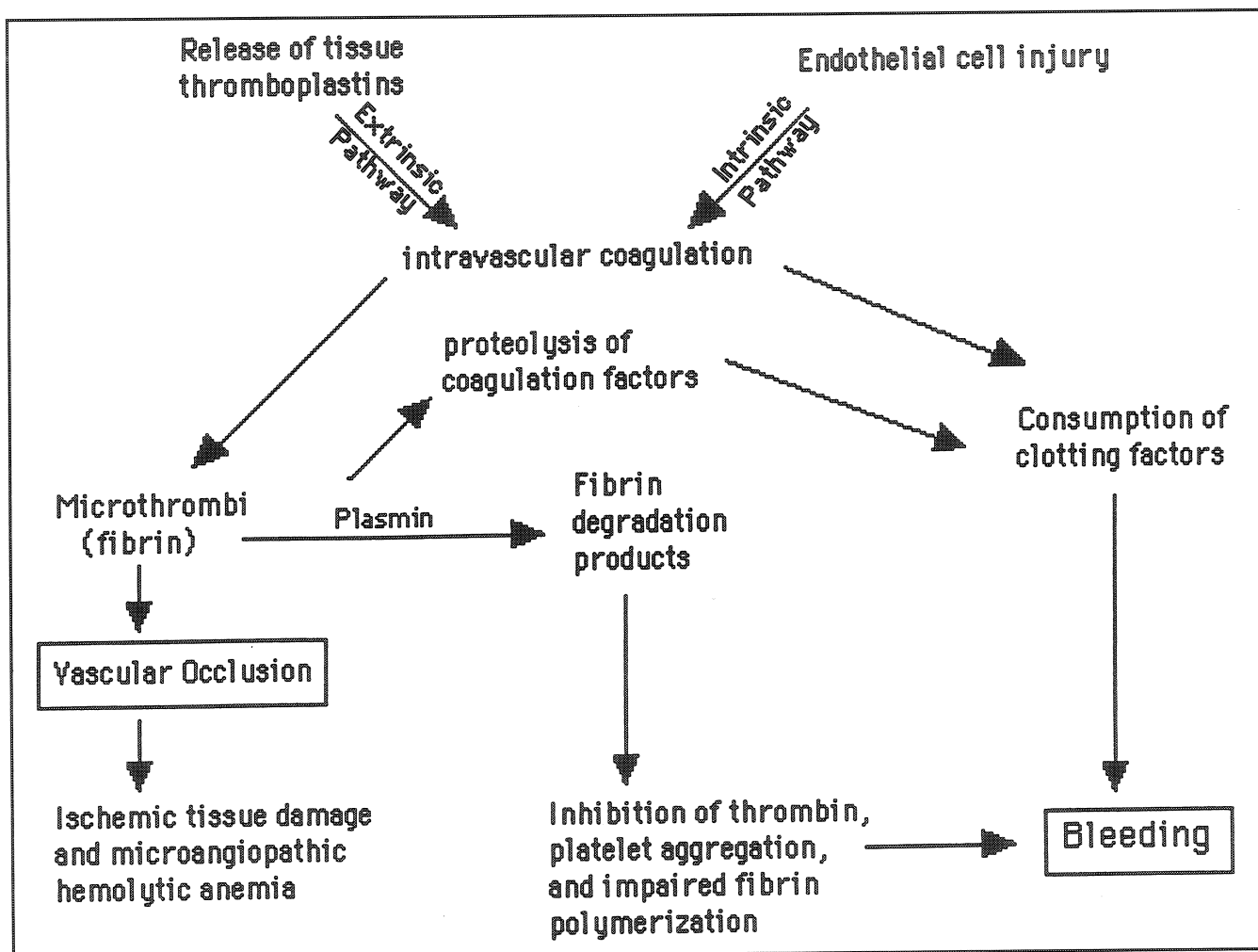


Figure 2. The mechanism of the thrombohemorrhagic state in Disseminated Intravascular Coagulation.

gen. The AT-III concentration will be reduced due to the complexing of activated clotting factors with circulating AT-III, while the Fibrinopeptide A level will be increased due to its liberation during fibrinogen formation⁷.

Although therapeutic options for DIC remain controversial, treatment should initially be targeted at the underlying etiology. If severe hemorrhagic or thrombotic phenomena occur, therapeutic heparin anticoagulation followed by aggressive replacement of specific coagulation factors is indicated. Adequate anticoagulation is crucial since it is the thrombosis of small vessels that has the largest impact on morbidity and mortality. Although clinically alarming, the hemorrhagic effects can generally be controlled⁷.

DIC is rarely encountered by the orthopedic surgeon performing total hip arthroplasty, but it is a potentially life threatening complication which needs to be diagnosed early in order to maximize patient outcome. Two

cases of DIC were described in patients undergoing hip arthroplasty for metastatic carcinoma of the proximal femur. Pugh described a patient who developed DIC following bilateral cemented total hip arthroplasties. A fourth case of DIC after arthroplasty was documented in a healthy patient who had a previous uncomplicated joint replacement of the opposite hip^{8,9}. If aggressive therapy is promptly instituted, small vessel thrombosis can be prevented and the thrombohemorrhagic state reversed. However, in this case the patient was unable to be resuscitated.

If a patient presents in the early postoperative period following total hip replacement with excessive bleeding, the diagnosis of DIC in addition to the more common occurrence of major vessel damage should be considered. Appropriate lab tests should be obtained (D-dimer, Antithrombin III (AT-III), Fibrinopeptide A and Fibrin degradation product) and early therapeutic and resuscitative efforts should be instituted. Although

the condition is usually reversible, this case demonstrates the potential mortality from DIC following total hip replacement in spite of early diagnosis and appropriate treatment.

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ASSESSING THE ACCURACY OF A PROTOTYPE DRILL GUIDE FOR FIBULAR GRAFT PLACEMENT IN FEMORAL HEAD NECROSIS

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ABSTRACT

A prototype drill guide was developed to improve the accuracy of surgical placement of fibular grafts for the treatment of femoral head necrosis. To document performance, two tantalum beads, one placed on the lateral femoral shaft and the other embedded in the superior portion of the head, were used to define the desired graft tract in a series of seven surrogate femurs. Two orthogonal x-rays of the drill guide mounted on each surrogate femur were taken both before and after drilling. After stylus digitization of each x-ray pair, a computer program calculated the achieved accuracy of the drill. The mean of the absolute error between the desired versus obtained position of the drill tip was 3.68 mm (s.d. 1.24 mm), and the random component of the error was 1.98 mm (s.d. 0.89 mm).

INTRODUCTION

Osteonecrotic femoral heads usually progress to collapse if not treated. Total hip replacement is a less than the ideal option for treatment, since its longevity is generally insufficient for the population afflicted with osteonecrosis. As an alternative, structural bone grafting has emerged as one possible means to prevent or postpone collapse of the necrotic femoral head.

Structural bone grafting for femoral head necrosis involves drilling a hole into the proximal lateral femoral cortex, through the neck of the femur, and into the head. A fibular bone graft is then inserted. Clinical results suggest that the success of femoral head struc-

tural grafting is dependent on graft placement^{1,2,5,7}. Also, finite element modeling has shown that the structurally ideal location, that which minimizes stress to strength ratios, involves directing the graft through the main body of the lesion and extending it to just under the subchondral plate³. Grafts which terminate in the necrotic lesion well short of the subchondral plate can actually increase stress to strength ratios, thus creating a situation in which the head is at higher risk of collapse than if no intervention has been performed.

Identifying the structurally optimal graft position is one thing, but physically achieving it is another. Currently, graft tract pilot holes are drilled using freehand manual guidance with fluoroscopic visualization. In an attempt to improve graft placement accuracy, a drill guide prototype has been developed (Figure 1). The objective of this study was to quantify the accuracy of this prototype, and to determine how its design might be improved.

MATERIALS AND METHODS

The ideal line of the axis of the drill tract used to insert the fibular graft can be uniquely described in terms of two points, P_1 and P_2 , representing the lateral cortex entry point and the graft tip point, respectively (Figure 2). These two points can be calculated by a finite element model formulated to minimize the stress to strength ratio in the necrotic femoral head⁶. Assuming the coordinates of P_1 and P_2 were thus specified, two tantalum beads were implanted in each of seven surrogate femurs (left adult composite femur: forty-eight centimeter length, Pacific Research Laboratory, Vashon, WA). The first bead was placed at a plausible graft axis entry point on the lateral femoral cortex and was designated as the entry point for the drill (P_1). The second bead was embedded in the superior portion of the head just below the fiberglass shell, representative of the subchondral plate, and was designated as the desired termination point of the graft (P_2). The desired drill axis trajectory, created by aiming the drill from P_1 to P_2 , passed through the superior portion of the femoral neck.

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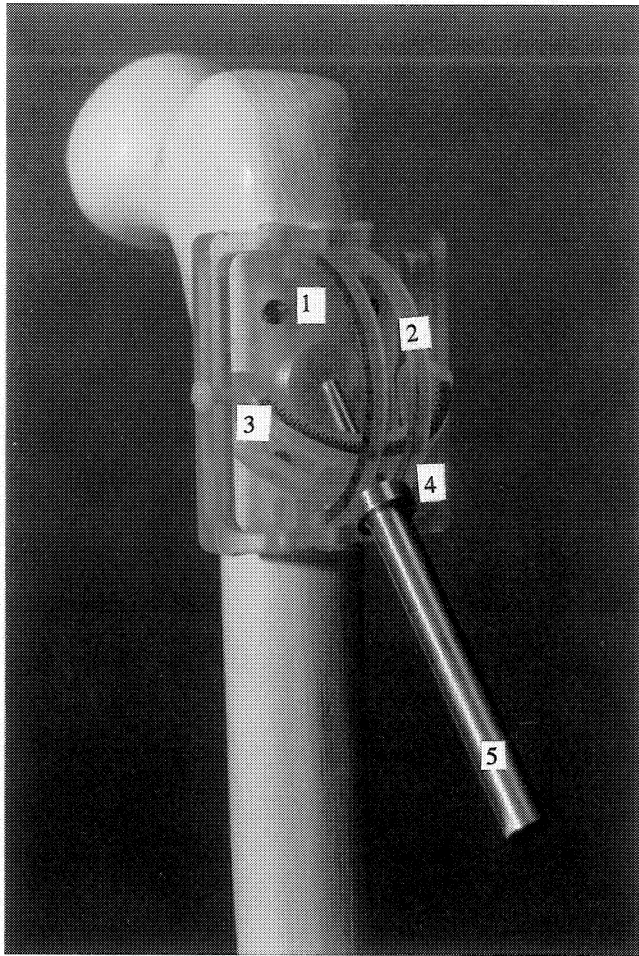


Figure 1. Prototype drill guide mounted on a surrogate proximal femur. Main components of the drill guide are (1) a base plate, screw-affixed to the lateral femoral cortex; concentric calibrated arcs controlling axis orientation vertical tilt (2) and ante/retroversion (3); a threaded locking mechanism (4); and a hollow metal guide tube (5).

The drill guide includes two concentric half-circular slots, which allow direction angles (ϕ , θ) to be set in two perpendicular planes. Regardless of the angular settings, the guide drill bit necessarily passes through the mutual center of the two half-circular slots, a fixed point (Point E in Figure 2). The drill guide was attached to the surrogate femur using four screws.

The mounted drill guide and proximal femur surrogate were positioned on an adjustable stand and placed in a specially configured biplanar radiography cage (Figure 3). Taut marker wires were run vertically and horizontally on each of the cage's vertical sides. Two adjacent sides had slots for holding x-ray cassettes, which allowed perpendicular radiographs to be taken. Using this system, orthogonal views were obtained (Figure 4) which could be combined in a computer program to recreate the actual three-dimensional space. After sty-

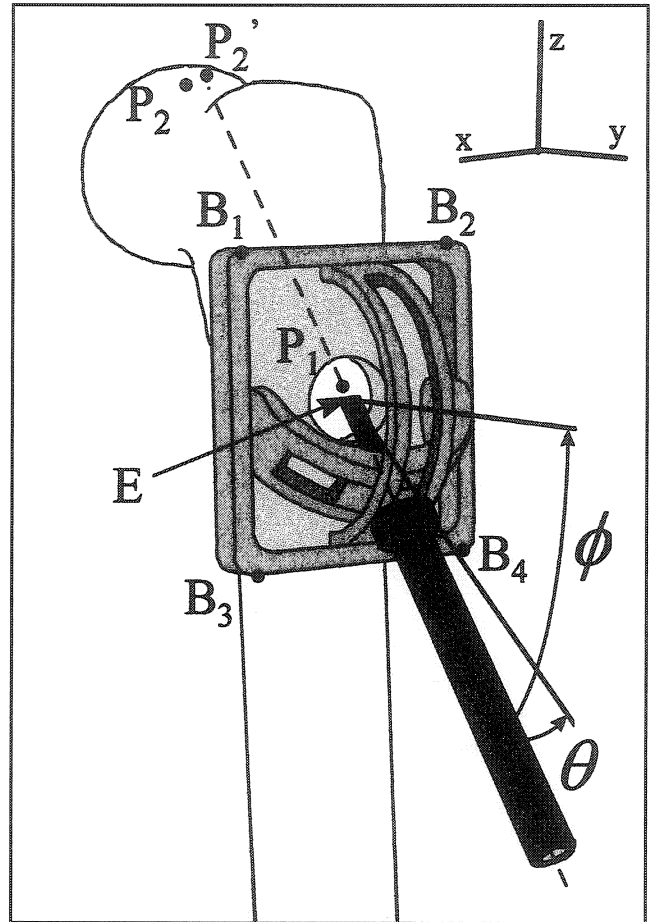


Figure 2. Geometrical definition schematic of the drill guide targeting points.

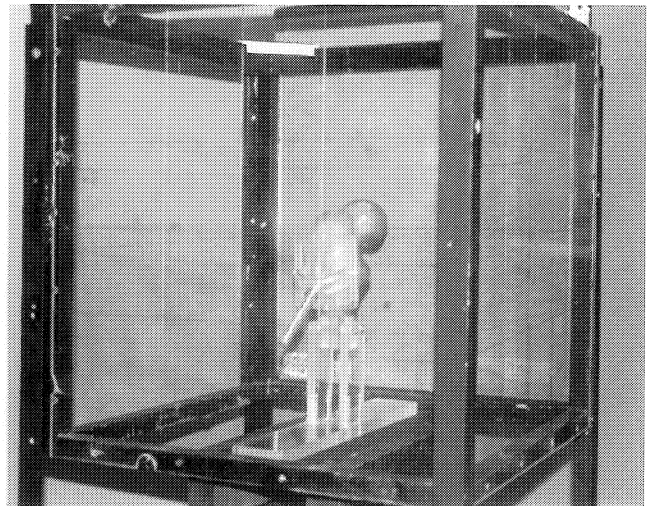


Figure 3. Surrogate proximal femur specimen, with drill guide affixed, positioned in the radiography cage. Film cassette holders at the back two faces of the cage permit orthogonal biplanar radiography. Horizontal and vertical marker wires on the four vertical faces of the frame permit back-projection calculations to determine the position of the x-ray source.

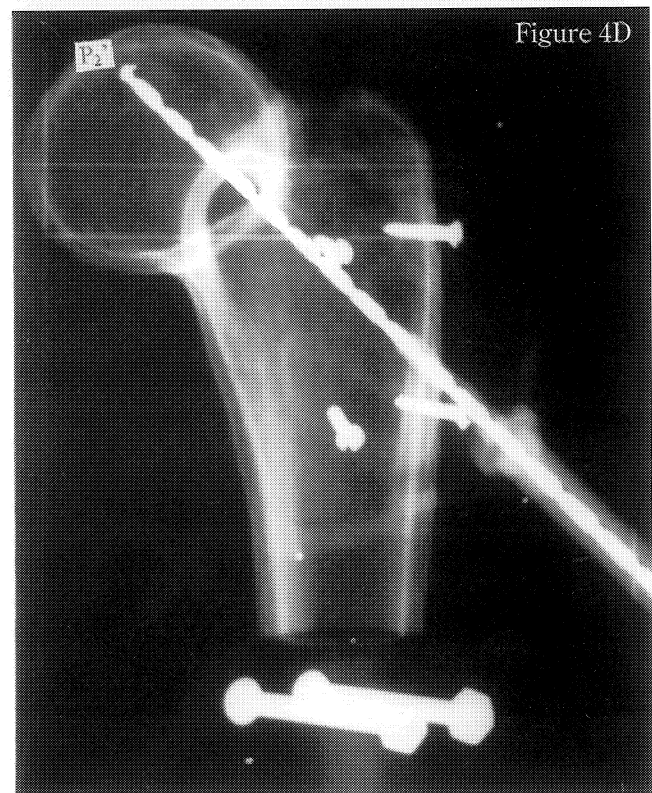
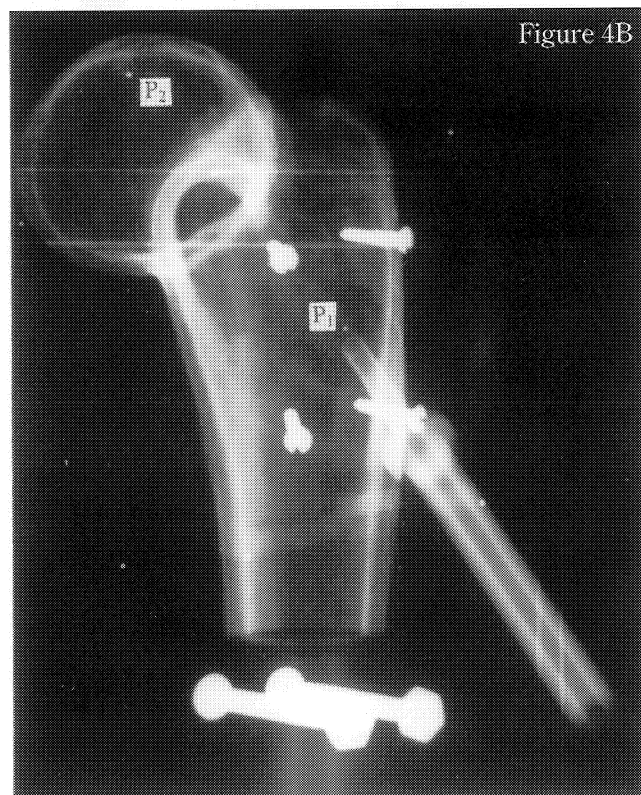
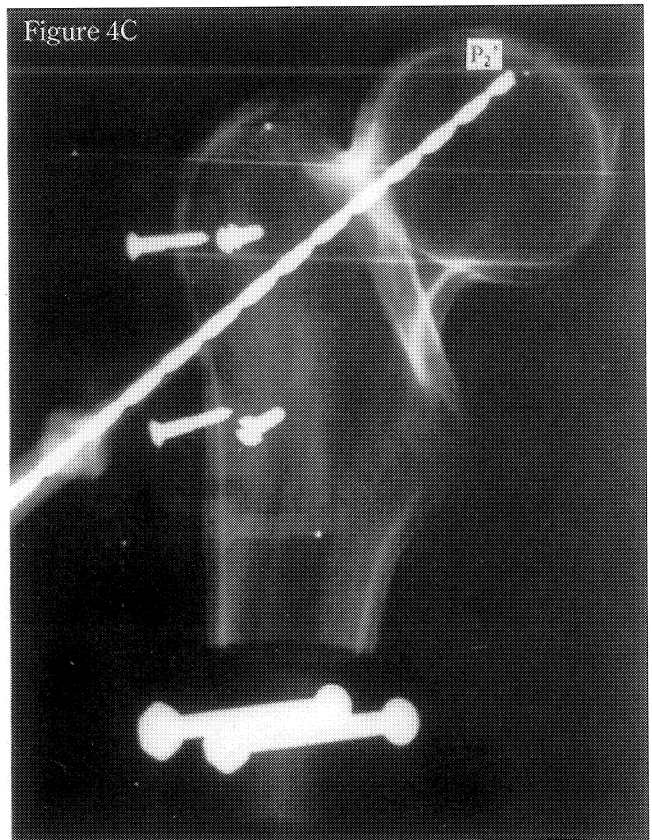
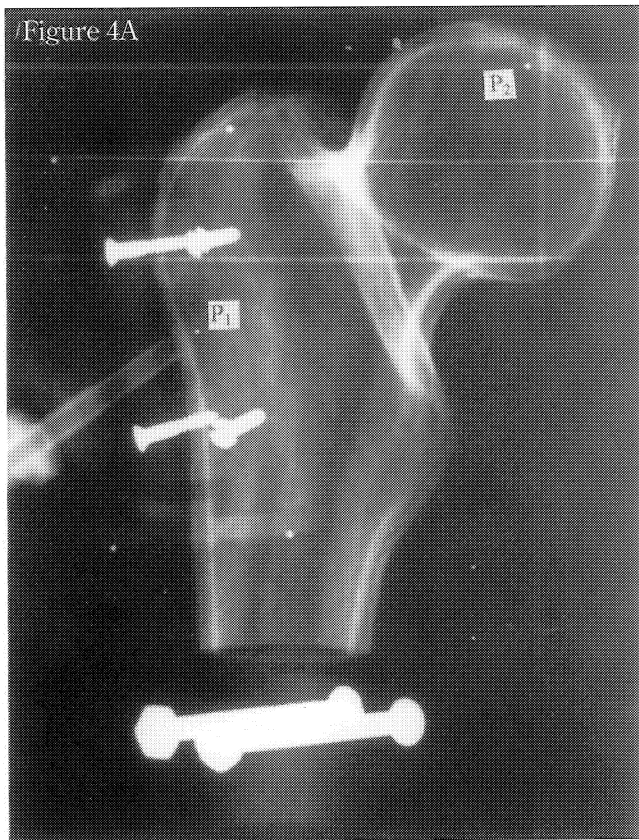


Figure 4. Orthogonal plane x-rays for a typical specimen taken before (A, B) and after (C, D) pilot axis drilling. Point P1 was habitually ablated by the drill bit while point P2 occasionally ablated.

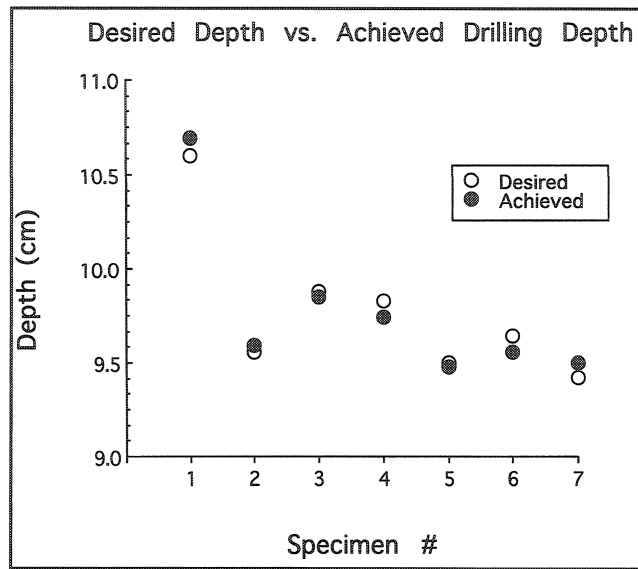


Figure 5A.

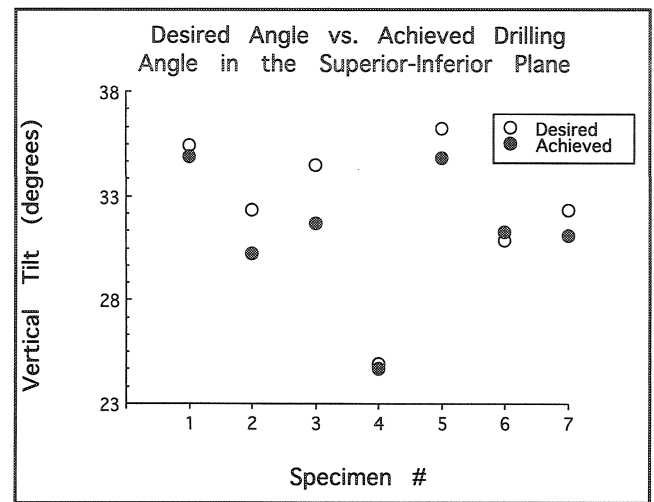


Figure 5B.

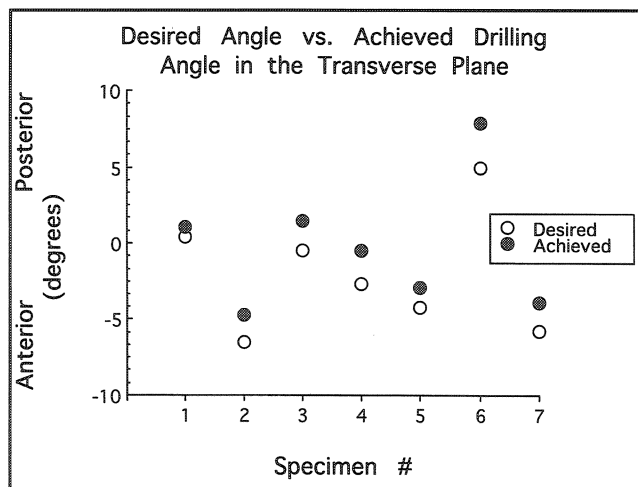


Figure 5C.

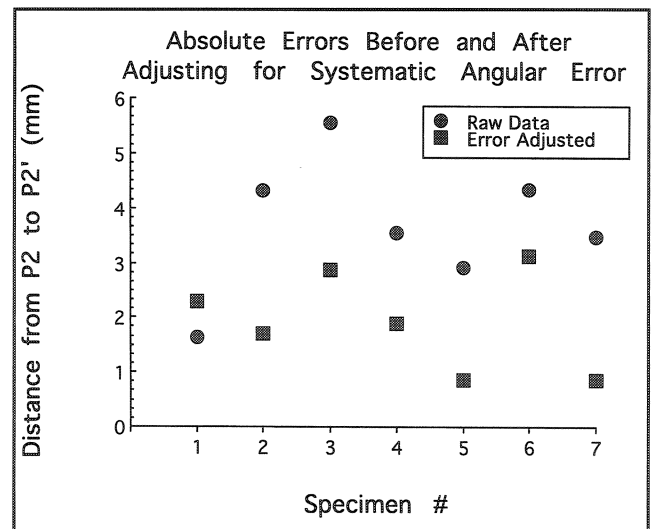


Figure 5D.

Figure 5A. Desired vs. achieved drilling depth. Figure 5B. Desired vs. achieved drilling angle in the superior-inferior plane. Figure 5C. Desired vs. achieved drilling angle in the transverse plane. Figure 5D. Achieved absolute error in the distance from the drill tip to the bead, before and after adjusting for systematic error.

lus digitizing the radiographs, the computer program used the precisely placed perpendicular wires to calculate vectors projecting back to the x-ray source for both radiographs. The program then calculated a set of position vectors from the x-ray source to six points of interest: the two implanted beads in the femoral surrogate (P_1 and P_2), and four beads (B_1, B_2, B_3, B_4) used to designate the plane of the drill guide base plate (Figure 2). Given position vectors computed to connect each interest point with the x-ray source, the location of all six interest points were found in three-dimensional space by finding the intersection of each point's two orthogonal position vectors. If the point's position vectors did

not intersect, the point's location was assumed to be the midpoint of the perpendicular distance between the closest point of mutual approach of the two position vectors.

A local coordinate system was defined relative to the drill guide base plate using the four implanted beads B_1, B_2, B_3 and B_4 . This allowed a position vector to be defined from the end (E) of the drill guide shaft to the termination point of the graft (P_2). This latter position vector was output by the program in a spherical coordinate form, reporting the desired drilling depth and the angles in the superior-inferior, (ϕ) and transverse (θ) planes, which needed to be set on the drill guide.

The specified angles ϕ and θ were locked in place. The depth was set by engaging a metal stop collar on the drill bit. The depth calculation took into account the distance from the target point in the femoral head to the tip of the drill guide, as well as the length of the drill guide shaft itself. The surrogate femur was then drilled and the bit left in place. Another series of x-rays was taken and stylus digitized. This time, however, the tip of the drill was digitized, instead of the embedded bead, to represent the achieved point (P_2') (Figure 2). This second set of x-rays allowed the computer program to calculate the distance the drill had gone into the surrogate femur, as well as the vector its course had taken. The accuracy of the drill tract could then be evaluated by comparing the achieved vector to the desired vector.

RESULTS

Each directional component of the desired versus obtained vectors was compared individually, after which the absolute disparity between the target bead P_2 and the drill bit tip P_2' was reported. The mean error in depth between the target P_2 and the drill bit tip P_2' was 0.6 mm (s.d. = 0.7 mm) (Figure 5a). In the superior-inferior plane (Figure 5b), the drill guide was consistently set superior to the desired position, yielding a mean error of 1.12 degrees (s.d. = 1.11 degrees). In the transverse plane (Figure 5c), the drill guide was consistently set anterior to the desired position, yielding a mean error of 1.81 degrees (s.d. = 0.72 degrees). The disparity in all trials was approximately the same, indicating a systematic error. The mean of the achieved absolute error in the distance from the drill tip to the bead was 3.68 mm (s.d. = 1.24 mm). If the systematic errors were eliminated by subtracting off the mean angular errors, the average random error in the distance between P_2 and P_2' was 1.98 mm (s.d. = 0.89) (Figure 5d).

DISCUSSION

While the present method of assessing the accuracy of the drill guide prototype seemed effective, it should be noted that the present results are not necessarily indicative of what would be achieved during actual surgical performance. First, there was no soft tissue interference when using the surrogate femurs. Second, the drill guide was anchored tightly to the surrogate femurs using four screws, a configuration which might be inappropriate for actual surgery given reports of occasional lateral cortex fracture following core drilling for osteonecrosis⁴. Finally, while the drill bit did pass through the dense fiberglass surrogate bone cortex, it did not encounter the analog of a sclerotic margin of

necrotic zone, which might cause deflection of the drill bit on its path toward P_2 .

The overall design of the current drill guide was effective, but several modifications seem appropriate. First, the graphs of the angular disparities illustrate systematic errors, indicating that the angular markings of the drill guide might be inaccurate or offset. Second, the locking mechanism required that the angles ϕ and θ be simultaneously held in place while tightening the drill guide shaft to mutually lock both concentric arcs. An independent pair of locking mechanisms might be helpful to better control the angular settings. Finally, the fixed point at the mutual center of the drill guide arcs (E) was not coincident with the periosteal surface of the shaft of the femur after attachment. This point lay slightly outside the periosteal entry point specified by P_1 . Therefore, in the present evaluations, the drill guide had to be slightly offset from a position directly centered on P_1 in order to allow a vector to be found which passed through P_1 , P_2 , and the arc center points (E) on the drill guide. Anticipating that the fixed drilling angle in the superior-inferior plane ϕ would be about thirty degrees and that in the transverse plane (θ) would be nearly zero, a reasonable offset was determined for these trials. Ideally, once the drill guide is fixed to the femur and the angles locked in place, a future drill guide design would allow the base to be repositioned with respect to the anchoring platform, thus allowing the drill vector to pass directly through P_1 . This would eliminate the need to exactly position the anchoring platform, a step which might be difficult during surgery.

Several directions are planned to continue drill guide development. One is to redesign the drill guide based on the above suggested changes and repeat the accuracy testing using the protocol described here. Another key step is to compare the drilling accuracy of an experienced surgeon working with versus without the drill guide. This implies modifying the drill guide protocol to more closely mimic what might be feasible in a surgical setting, especially in regard to the method of attaching the drill guide to the bone.

ACKNOWLEDGMENT

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THE EFFECTS OF TIME AND LIGHT EXPOSURE ON CONTACT AND PRESSURE MEASUREMENTS USING FUJI PRESSCALE FILM

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Steven Viegas*

ABSTRACT

The search for methods to demonstrate and accurately measure contact area and pressure within human joints has been and remains an active one. Presensor is a measuring system developed by Fuji Photo Film Co., Ltd. and used in medical applications.

The color intensity fades with time and exposure to light. These changes have not been well documented despite the increasing use of Fuji film in biomedical research. These changes in color intensity provide a source of error in the calculated measurements. The known variability of the color density of film exposed to light and air can be measured and predicted. Methods to control and improve the accuracy and reliability of study data are suggested.

Film was exposed to three different amounts of pressure and placed into one of four subgroups: exposed to light and taped or not taped, and stored in the dark and taped or not taped. Fading of the color density was seen after only a few hours of exposure to light when the film was left untaped. When the film was taped, a slight darkening was seen. For the best accuracy and reliability, the film should be digitized or analyzed within eight hours of exposure to pressure.

INTRODUCTION

Medical researchers have searched for and utilized devices to measure contact area and contact pressure within joints in the human body for decades. Ideally, these devices should not impede or change the measurements which they are supposed to observe. Piezo film, strain gauges, stress paints, and Fuji film are sev-

eral types of measuring devices, or transducers, that have been utilized to obtain a better understanding of the biomechanics of the human body.

During the course of performing biomechanical studies utilizing Fuji film, and in examining old pressure prints, fading and color changes in the Fuji pressure sensitive film were noticed. It was noticed that the color fades with time and exposure to light. Fading was reduced by placing transparent tape over the print after it was exposed to the pressure. However, the rate of fading has not been documented, and there was a concern that the prints obtained at the beginning of a testing period would fade before they could be digitized or compared to later prints. Because any changes that occur in the color of the prints will be a source of error in the calculated pressure measurement, it was decided that a study should be performed to quantify the known variability in the color density of Fuji film that is exposed to light and air.

METHODS AND MATERIALS

Fuji superlow film was cut into strips of rectangular pieces approximately five centimeters by two centimeters. These strips were exposed to pressurized nitrogen for one minute through a chamber with a 1.5 cm diameter hole. Applied pressure was measured through another hole in the chamber with a Transmetrics Inc. (Solon, Ohio) pressure transducer. Through a series of valves, the test pressure inside the chamber was increased. Then, the pressure inside the valve was purged. While the pressure coming from the nitrogen tank was kept constant, the transducer was inserted into the chamber and the valve to the nitrogen tank was reopened to expose the transducer to the desired pressure. Each strip had one of three pressures applied. The three pressures used [51, 130, and 284 psi, (3.6, 9.2, and 19.9 kgf/cm²)] approximate the range of Fuji superlow film (5-25 kgf/cm² or 71-355 psi). Although a pressure of fifty-one psi is below the threshold of the super low film, there was a definite observation of color on the film in this pressure range and this lower application of force was investigated.

After the transducers were exposed to one of three pressures, the "A" film was carefully removed and the resulting "C" film (with the red or orange coloration)

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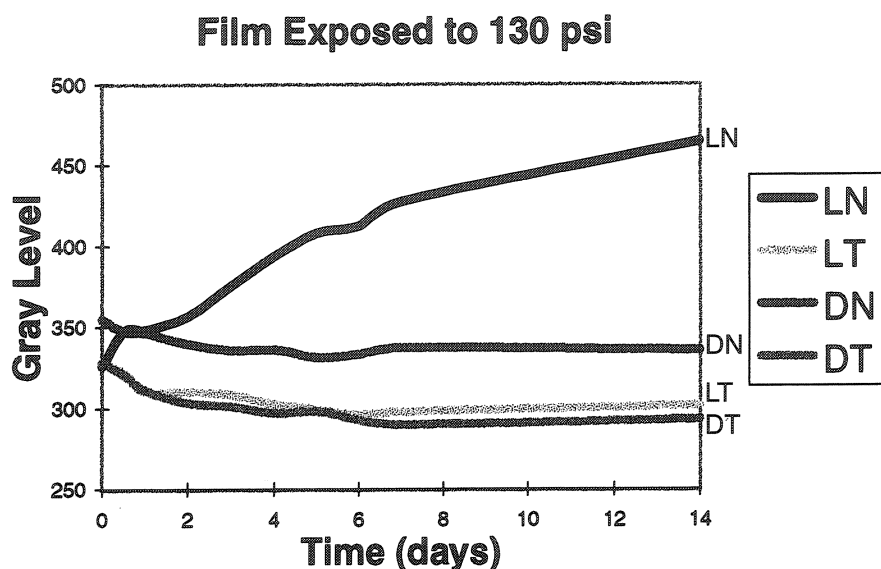


Figure 1. Gray level of Fuji film exposed to 130 psi over time for four conditions: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN).

was placed into one of two main groups. The transducer was either covered with Lepage's invisible tape (Pittsburgh, PA) or left exposed to air. Some pieces were placed in a folder and stored in a closed cabinet (in the dark), while others were placed on top of a cabinet (exposed to fluorescent light for nine hours, an average week day, and then left in the dark).

The pieces of film were placed into one of four subgroups: exposed to light and taped (LT); exposed to light and not taped (LN); stored in the dark and taped (DT); and stored in the dark and not taped (DN). Each subgroup consisted of three pieces of film: one exposed to 51 psi, one exposed to 130 psi, and one exposed to 284 psi. With the use of a video camera, light stand, personal computer, and videodigitizing board, each piece of film was videodigitized and stored as a graphics image. Care was taken to maintain adequate and constant lighting and eliminate shadows on the image.

A Better Basic program was used to analyze the digitized images. The program calculated the average gray level in each of the pieces of film and stored the results for later statistical analysis^{11,12}. In the first part of the experiment, the sets from each group were videodigitized immediately, after three hours, and one, two, three, four, five, six, seven and fourteen days after the film was exposed to the pressures.

There was an identifiable difference in gray levels of the film immediately after exposure compared to one day later. There was no difference in gray level between the film analyzed immediately and those analyzed after three hours. Therefore, in order to gain information after three and before twenty-four hours of exposure, a second experiment was conducted. Twenty pieces of

film were exposed to 150 psi (10.6 kgf/cm²) for one minute and divided into the same four groups: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN). Additionally, a piece of tape over white paper was also included with the film to see if the tape alone darkened over time. The pieces of film and tape over white paper were digitized hourly for nine hours and then daily for the following seven days. Again, the digitized images were analyzed using a Better Basic program.

The data were analyzed using a General Linear Model (GLM) procedure (three way ANOVA) to check for differences among the parameters with gray level as the dependent variable and time as the independent variable. A p value < 0.01 was considered significant. The statistical analysis also included a linear approximation of the gray level for each of the four groups and yielded estimates for the slopes and y intercepts.

RESULTS

During the first part of the experiment, preliminary statistical analysis was performed to determine if there were any significant early changes in the film. The initial results showed significant changes ($p < .01$) in the film exposed to 130 psi when exposed to light (Figure 1). Film that was not taped, faded (the gray level increased) after twenty-four hours of exposure to light and the film covered with tape actually got darker with time (the gray level decreased). This darkening effect was strongest in the 130 psi exposed film. Figures 2 and 3 show light effects on the film exposed to 284 and 51 psi, and confirm the results seen in the film exposed

Film Exposed to 284 psi

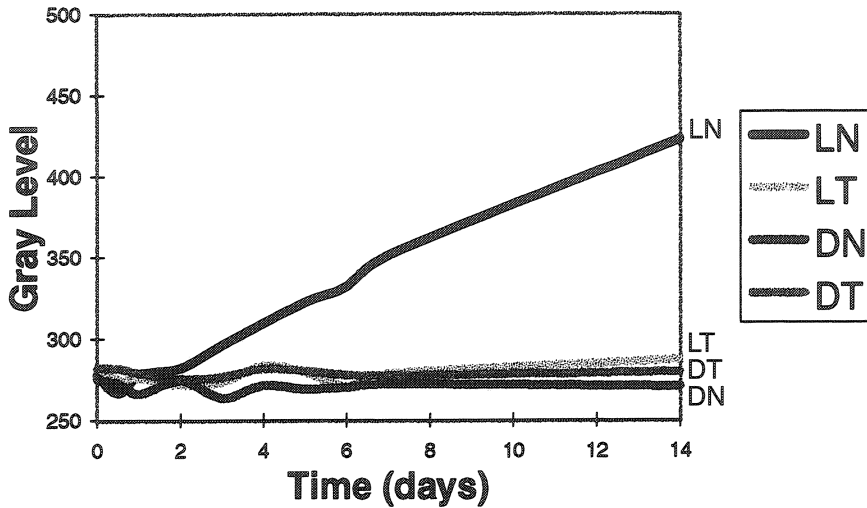


Figure 2. Gray level of Fuji film exposed to 284 psi over time for four conditions: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN).

Film Exposed to 51 psi

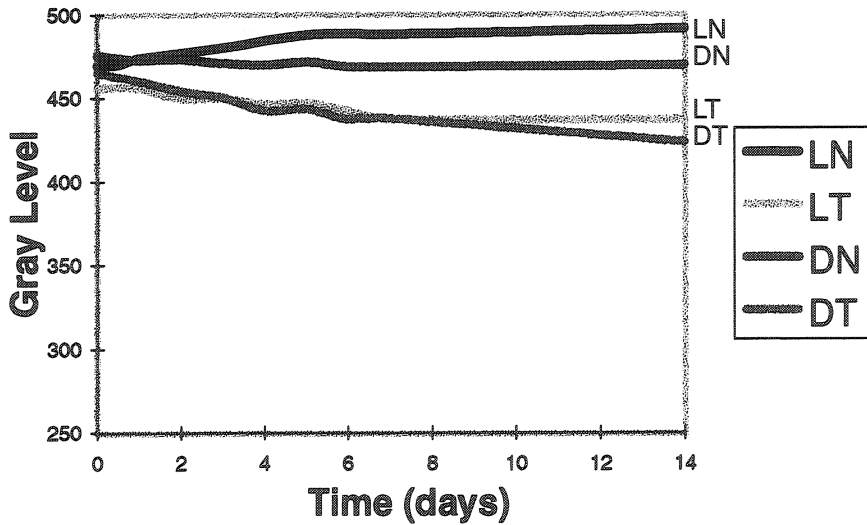


Figure 3. Gray level of Fuji film exposed to 51 psi over time for four conditions: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN).

to 130 psi.

Because the first part of the study showed effects of light on the film after one day, and the greatest change was on the film exposed to 130 psi, an additional study was undertaken to determine how long, in hours, the film could be exposed to light without fading. Figure 4 shows a graph of the average gray level of the pieces of film exposed to 150 psi during the first eight hours of exposure and then daily for seven more days.

A study of the effects of a white piece of paper placed into the same categories as the Fuji film is shown in Figure 5. Although the outlying points are significantly different from the starting point, the maximum change in gray level is less than one percent of the total gray

level number range. Thus, independent of the light factor, all of the taped pieces of film in the low to medium pressure range showed a darkening trend after eight hours of exposure. There was no darkening trend in the tape alone on white paper, implying that the tape did not have a chemical effect on the Fuji film. Tests for the homogeneity of slopes in lines shown in Figures 1 through 3 indicated that the lines did not have similar slopes with the LN slope being more positive and the DN, DT, and LT slopes being more negative (Table 1).

DISCUSSION

Piezo film, strain gauges, and stress paint or

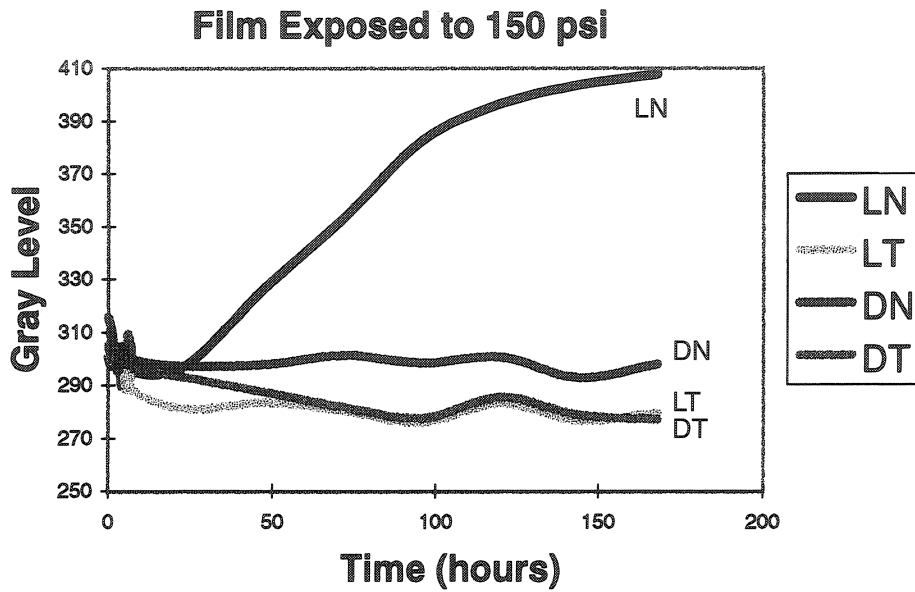


Figure 4. Gray level of Fuji film exposed to 150 psi over time for four conditions: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN).

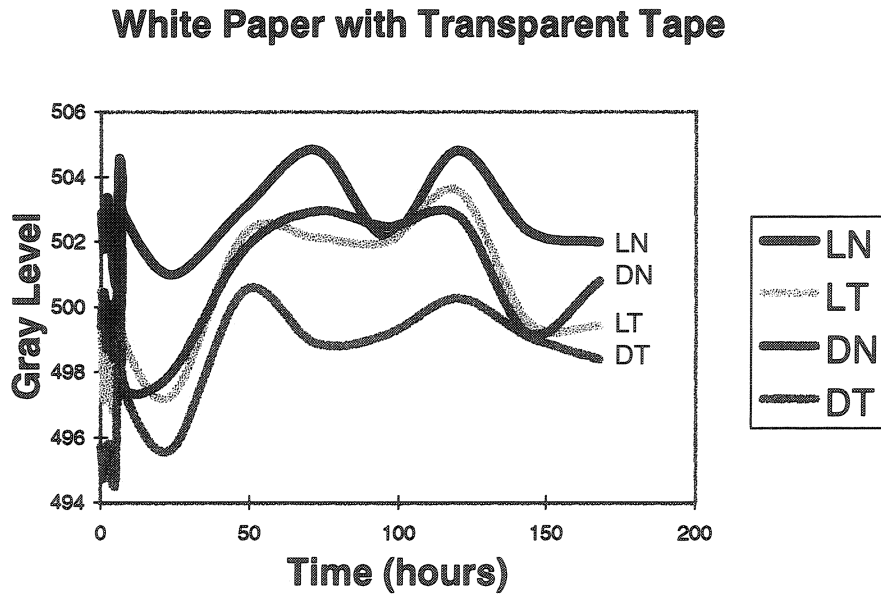


Figure 5. Gray level of white paper covered with transparent tape over time for four conditions: exposed to light and taped (LT), exposed to light and not taped (LN), stored in dark and taped (DT), and stored in dark and not taped (DN).

photoelastic material have been used to measure contact and pressure. These transducers, however, have limitations within the human body. Piezo film will only measure dynamic forces and cannot be used for static load applications. Strain gauges may alter the properties of the bone-gauge interface and will only display stress at one point on the surface studied¹⁷. Stress paints may also alter the properties of the bone-gauge interface and can be associated with expensive equipment.

Presensor is a pressure measuring sheet system which has been developed by Fuji Photo Film Co., Ltd. It has been available since April, 1977 and was devel-

oped in response to industries' need for pressure measurement. It is used in hydraulic presses, in the manufacturing of flanges, and most extensively in the gasket industry to detect leakage of oil^{2,4}. It is also used in industry to measure forces between pressed surfaces⁵ in engines, gear teeth, roll contact areas in paper mills¹⁰ and printers, and various other static and dynamic applications. More recently, Prescale has been used in medical applications^{6,8,9,11,12}.

Prescale, or Fuji pressure sensitive film (C.Itoh, New York, N.Y.), is a type of pressure transducer. It consists of two pieces of film (A and C film) that when placed

TABLE 1

Condition	Slope (Gray level/Time)				
	51 psi	130 psi	284 psi	tape	150 psi
DN	-0.449	-0.092	-0.055	+0.011	-0.053
DT	-3.033	-2.256	-0.069	+0.023	-0.165
LN	+1.661	+9.903	+11.19	+0.003	+0.759
LT	-1.570	-1.726	-0.624	+0.017	+0.122

together can detect pressure. The A film is coated with randomly distributed microspheres that burst under various predetermined pressures. The C film is a developer that turns red when it comes into contact with the dye in the microspheres on the A film. Fuji A film comes in four pressure ranges, super low (5-25 kgf/cm², 71-355 PSI), low (25-100 kgf/cm², 355- 1420 PSI), medium (100-500 kgf/cm², 1420-7102 PSI), and high (500-1300 kgf/cm², 7102-18466 PSI). Fuji C film is compatible with any of the A films.

Fuji film is advantageous because the resulting transducer consisting of A and C film is thin, measuring .2 mm, thus being less invasive within a human joint than other measurement techniques such as strain gauges^{4,7}, rubber transducers³ or miniature load cells . It also has an immediate observable force distribution pattern that is quantifiable. The manufacturer specified limits of accuracy are plus or minus 10 percent, using their densitometer (Fuji Prescale film instruction manual). However, Singerman et al.⁹ was not able to obtain this amount of accuracy . The company also states that the film requires no preparation for use, simply cut the film into the required shape. This process is not so simple when the shapes required are complex, like in a joint of the body. The “cutting” process has become a major portion of the experimental preparation time in the laboratory because the film can only be used once, and a new transducer must be fabricated for each load or test. In addition, the film must remain dry. Any contact with liquids or chemicals will distort the color intensity and measurement of force. Thus, for use in human joints, the film must be sealed.

Experiments in this laboratory, on cadaver specimens, have utilized a custom made die (Kasper Die and Tool, Shiner, TX) to cut the A and C film to match the surface contour of the joint. Each transducer was designed with a “tail” that extended beyond the joint and served as a place to staple the film together, as a

“handle”, and as a place to create a physical reference mark^{11,12} . Then the transducer was taped with a low adhesive type of masking tape to seal the transducer during joint testing. This, along with cutting each side of the film, A and C, is very time consuming.

Fuji film has been determined to be an accurate and reliable method for determining contact areas and stresses within human joints¹⁰. Because the measurement testing in human joints can span several days, it is important to consider the effects of light exposure on the film. The most prominent trend is the progressive fading (increase in gray level or reduction of red coloration on the film) of the dots exposed to light and not taped. This trend is present throughout the pressure range of the film and can, therefore, alter the measurement of pressure. The film must be prepared correctly and several factors including temperature, humidity, rate of loading, curvature of loaded surfaces, and shear stresses must be considered because they may also alter the color intensity measured by the Fuji film.

Fading of the color density was seen after only a few hours of exposure to light when the Fuji film was left untaped. When the film was taped, the tape caused a slight darkening of the color on the film as time passed. Thus, for the best accuracy and reliability, the Fuji film should be digitized or analyzed within 8 hours of exposure to pressure.

One other solution to minimizing error is to create calibration prints along with the pressure measurements. These calibration prints can be analyzed along with the pressure prints because they will fade at the same rate as the pressure prints, and more reliable results can then be recorded.

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DIAGNOSTIC CLARITY IN ORTHOPEDICS DUE TO ADVANCED TECHNOLOGY: THOUGHTS ON THE SURGEON'S ROLE AND RESPONSIBILITIES

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INTRODUCTION

The history of orthopedics is replete with discoveries that have radically changed both the diagnosis and treatment of disease. The first administration of general anesthesia was by Morton in 1846; this has led to modern day anesthesia which allows complex operations to be performed with minimal risk. Prior to the 1860's sepsis following surgery was expected ("laudable pus"). Soon thereafter the contributions of Louis Pasteur and Joseph Lister revolutionized our understanding of asepsis and primary wound healing without infection.

For orthopedic surgery, perhaps the most important development in the last two centuries was the discovery of the x-ray by Wilhelm Conrad Röntgen in 1895 (Figures 1A, 1B). Prior to this, orthopedic decision making relied on a careful history and physical examination (which continue to be of vital importance to orthopedic care today). The discovery of the x-ray radically changed the diagnostic approach to orthopedic problems and provided an entirely new understanding of disease. For example, the ability to differentiate between hip diseases such as developmental dysplasia of the hip, Legg-Calve-Perthes disease, infection and slipped capital femoral epiphysis has greatly improved the treatment and quality of life of the patient. Similarly in the spine, x-ray imaging allows differentiation between acute infection, tuberculosis, tumors and other conditions.

One can only imagine the difficulty in diagnosing and treating hip or spine disease prior to this discovery. Yet, even in that era, most surgeons developed clear and certain opinions about disease, including rigid treatment protocols which they expected all to follow.

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Figure 1A. Wilhelm Conrad Röntgen (1845-1923).

Throughout the twentieth century and even today, plain radiographic methods remain the first tool utilized by an orthopedic surgeon faced with a diagnostic problem. Continued improvements have led to clearer images with less radiation exposure and increased safety for both the patient and the physician. However, it has always been understood that plain radiographs represent only a two dimensional depiction of three dimensional anatomy. As early as the 1920's attempts at three dimensional analysis were made and later stereoscopic methods were developed. However, the sense of three dimensionality was poor and could not be readily appreciated.

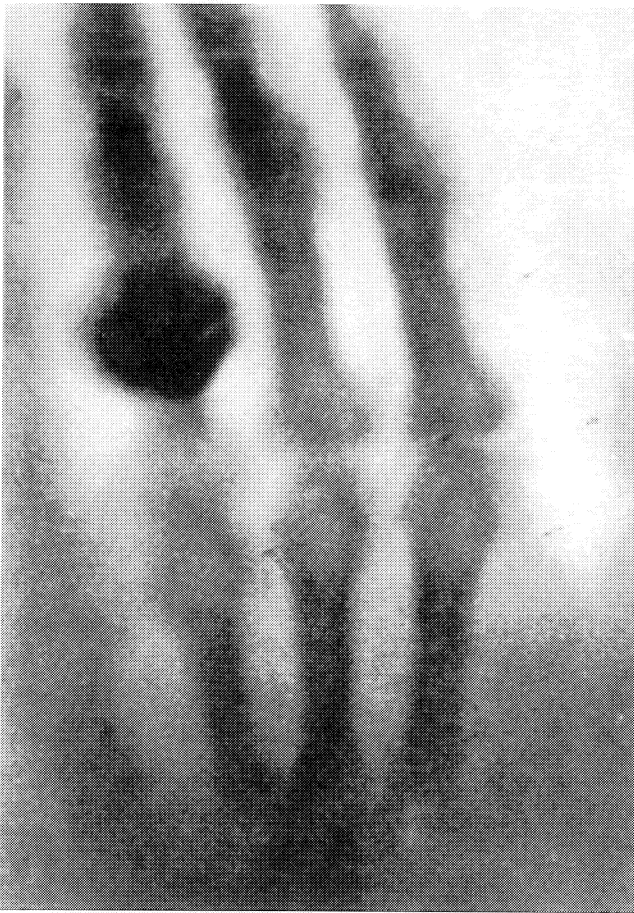


Figure 1B. Radiograph of the hand of anatomist von Koelliker taken as a demonstration at the Wurzburg Physical and Medical Society meeting in 1896. In expressing his thanks, von Koelliker exclaimed that he had never witnessed such a splendid and important presentation and proposed (to a standing ovation) that future x-rays be called Röntgen rays. (Röntgen Rays Centennial Exhibition Catalog, Wurzburg, Germany, 1995).

In the 1960's, research by Hounsfield and Cormack led to the discovery of computerized tomography (CT scan). This technology provided clear cross sectional images of orthopedic pathology that radically changed the understanding of many conditions (spine fractures, bone tumors, skull and brain disorders). In spite of these images being two dimensional, diagnostic value was added because the images were in a plane (transverse) that previously could not be visualized.

Continued advances in computer technology in the 1980's led to the development of three dimensional computerized tomographic scans (3-D CT). This technology reformatted two dimensional computerized tomography (2-D CT) data so that three dimensional images could be visualized. Continuing advances have led to a level of clarity in 3-D CT studies that make availability of this technology vital for the accurate diagnosis and treatment of certain orthopedic conditions.

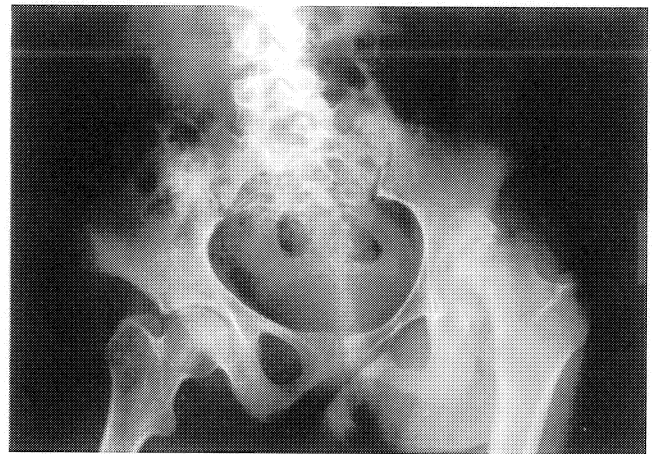


Figure 2A. AP pelvic radiograph demonstrating osteonecrosis of the left hip in a female with idiopathic osteonecrosis (age thirteen).



Figure 2B. AP radiograph of the left hip three years after a shelf procedure and valgus osteotomy of the left hip. Note the apparently satisfactory coverage of the lateral femoral head.

The purpose of this paper is to demonstrate that advanced technology allows for the accurate diagnosis and more specific treatment of many orthopedic conditions, and to speculate as to how the presence or absence of

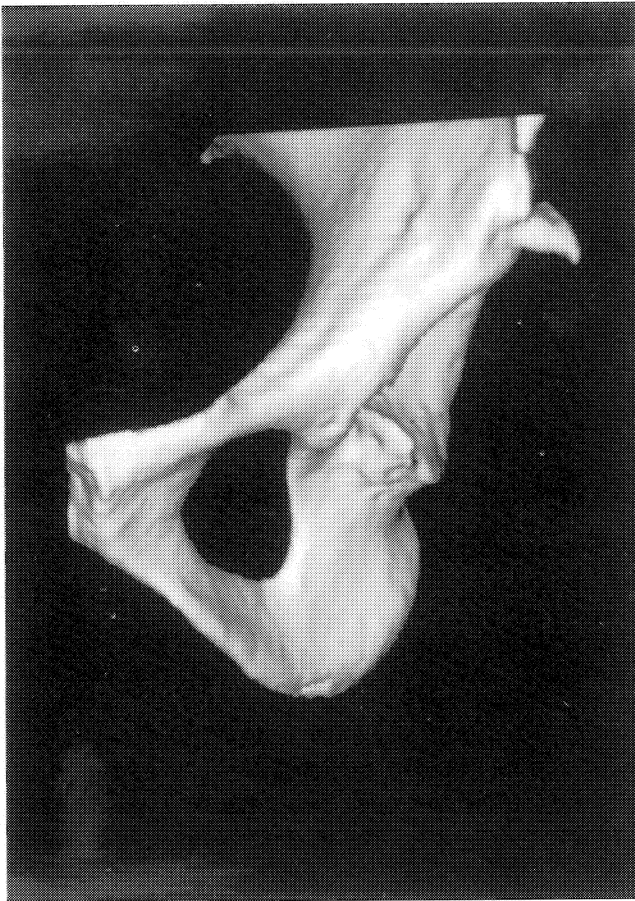


Figure 2C. Three-dimensional CT image of left hip (AP view) demonstrating the bony shelf extruding from the supero-lateral aspect of the acetabulum.



Figure 2D. Three-dimensional CT lateral view of the left hip. Note the minimal amount of anterolateral coverage provided by the bony shelf (→). The superior and posterior areas remain completely uncovered.

such technology may produce ethical dilemmas for the contemporary orthopedic surgeon.

The following case studies (all patients treated at Children's Hospital - San Diego) demonstrate the application of 3-D CT methods for several orthopedic conditions; clarifying that without this technology, the diagnosis, treatment, and functional outcome would be markedly different.

CASE STUDIES

Case 1

A female with the diagnosis of VATER syndrome had undergone prior surgical correction of her radial club hand, Sprengel's deformity of the scapula, and congenital scoliosis. At age thirteen, osteonecrosis of the left hip was diagnosed (Figure 2A). This was treated with a valgus osteotomy of the femur and a shelf procedure of the acetabulum, resulting in initial pain relief. At age sixteen the patient experienced a recurrence of hip pain and loss of motion. A repeat pelvic radiograph (Figure

2B) demonstrated apparent adequate coverage of the femoral head by the shelf of bone graft. A 3-D CT was ordered in an attempt to explain the symptomatology (Figures 2C, 2D). In contrast to the plain radiograph which suggested sufficient coverage, the 3-D CT study showed only minimal anterolateral coverage with no posterolateral coverage. Therefore, a further salvage-type coverage procedure (Chiari) was performed.

Case 2

A female with T12 level spina bifida underwent anterior Dwyer instrumentation and fusion with the addition of Luque-Galveston posterior instrumentation and fusion for a severe neuromuscular kyphoscoliosis at age ten (Figures 3A, 3B). At age seventeen the posterior instrumentation was removed after she developed a local infection. Following removal, increases in kyphosis and scoliosis were noted (Figure 3C). The complexity of the deformity and poor skin coverage led us to advise conservative treatment. In spite of compliant orthotic use, the patient had constant severe pain when

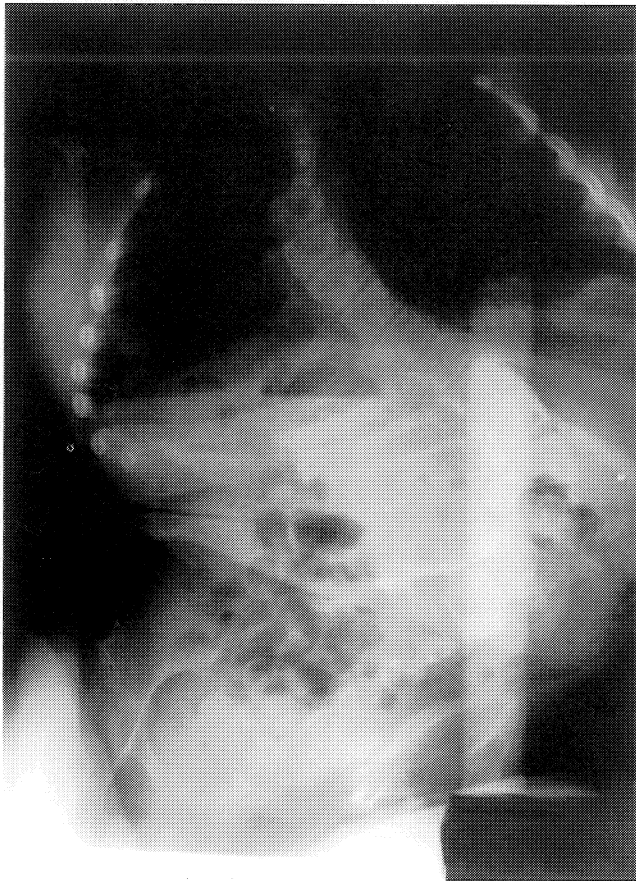


Figure 3A. Female with T12 level spina bifida. AP thoracolumbar spine radiograph demonstrating a 110 degree right thoracolumbar neuromuscular curve (age ten).

sitting. To better understand the problem, and because of the complexity of the previous surgery and poor bone quality, a 3-D CT study was ordered. Figures 3D and 3E demonstrate a massive pseudarthrosis with hypertrophic bone suggesting a Charcot spine. This study allowed planning for complex revision surgery (Figure 3F).

Case 3

A sixteen year old male presented to our clinic two years following percutaneous pinning of a slipped capital femoral epiphysis at another institution (Figure 4A). He stated that his initial limp and pain had actually worsened following fixation. The entry point of the screws in association with the severity of the presenting slip led us to question the exact location of the screw tips (Figures 4B, 4C). Despite multiple x-ray views we could not confirm improper screw placement. A 3-D CT scan demonstrated extra-osseous positioning of the screws with entry into the posterior acetabulum (Figures 4D, 4E). The patient was treated with screw removal followed by a delayed proximal femoral osteotomy.

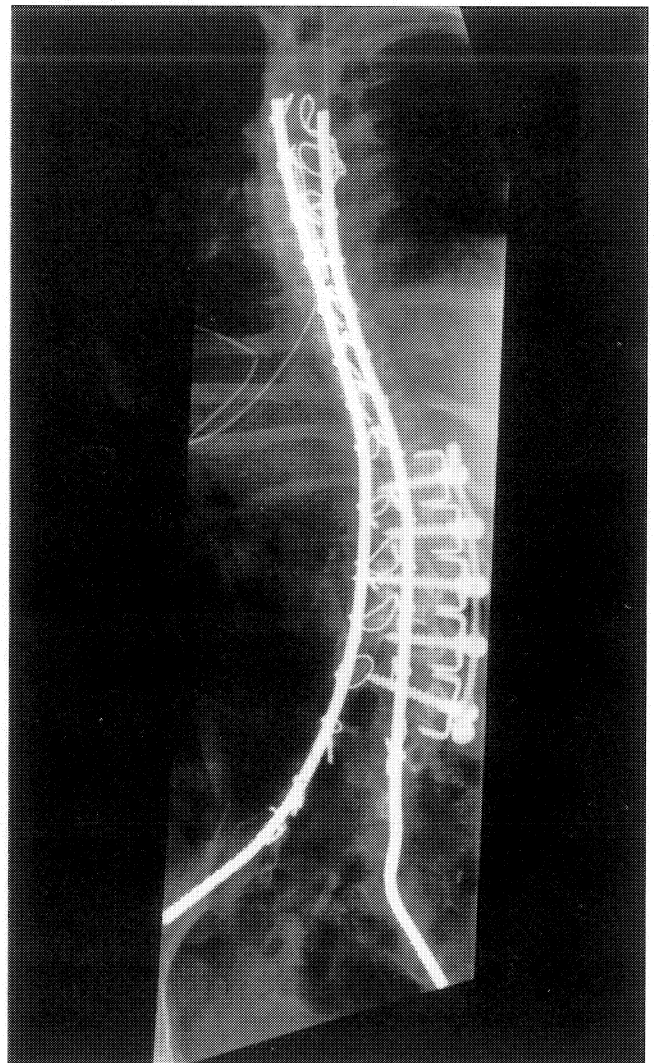


Figure 3B. AP view following surgical correction with anterior Dwyer and posterior Luque-Galveston instrumentation and fusion.

Case 4

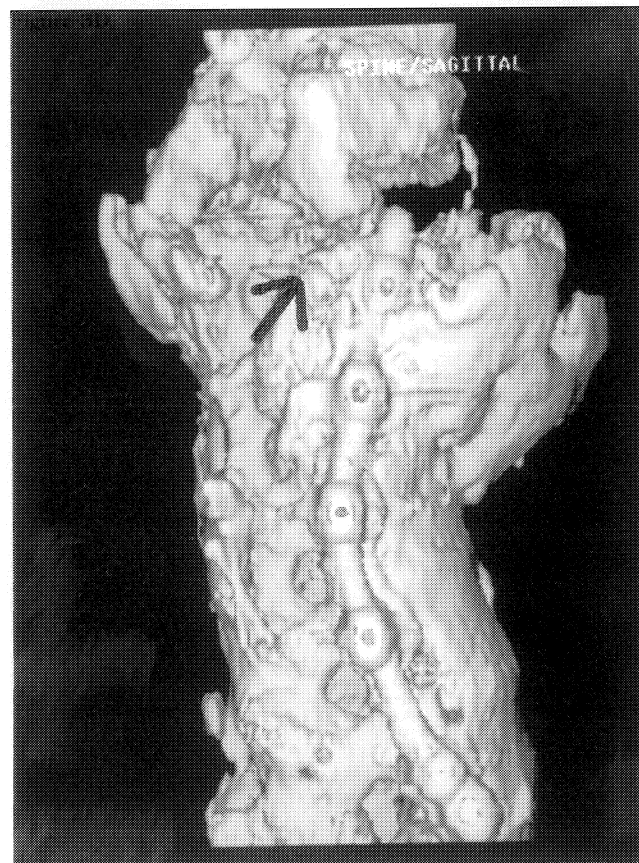
A thirteen year old male presented to the Emergency Room, after a skateboard accident, with left hip pain. An AP pelvic radiograph (Figure 5A) failed to clarify his injury. A 3-D CT study demonstrated a physeal fracture through the triradiate cartilage necessitating open reduction and internal fixation (Figures 5B, 5C).

Case 5

Early plain radiographs (Figure 6A) in this young male with L4 level spina bifida demonstrated mild bilateral acetabular dysplasia which was treated with abduction bracing. Subsequent films at age five years (Figure 6B) showed progressive hip subluxation due to muscle imbalance. In planning for surgical correction, CT studies were ordered. The 2-D CT study (Figure 6C) clarified the marked loss of gluteal and abductor



Figure 3C. Lateral spine radiograph following removal of the posterior instrumentation secondary to late low-grade infection (age seventeen).



Right: (Top) Figure 3D (oblique view) and (Bottom) 3E (lateral view). Three-dimensional CT views demonstrating a massive pseudoarthrosis at the thoracolumbar junction. Note the typical elephant foot appearance of the pseudoarthrosis (→). At surgery a large fluid-filled “false joint” was encountered.

muscle mass that led to the hip subluxation. The AP 3-D CT pelvic view clarified the overall pattern (Figure 6D). However, focal lateral 3-D CT views provided a clearer understanding of the hip subluxation (Figures 6E, 6F). The lateral view of the left hip with the femoral head in position (Figure 6E) demonstrated a hip flexion deformity and the resulting superior and posterior hip dysplasia. The lateral view with the femoral head subtracted (Figure 6F) showed abnormal widening of the posterior wing of the triradiate cartilage which was the result of excessive focal force on this segment of the acetabulum. As a result of this abnormal force, the normal transformation of triradiate cartilage to bone was delayed, which may have led to further deformation. This information provided a clearer understanding of the pathoanatomy of the neuromuscular hip and allowed us to select a combination of muscle transfers and corrective osteotomies (acetabular and femur) that provided optimal hip stability.

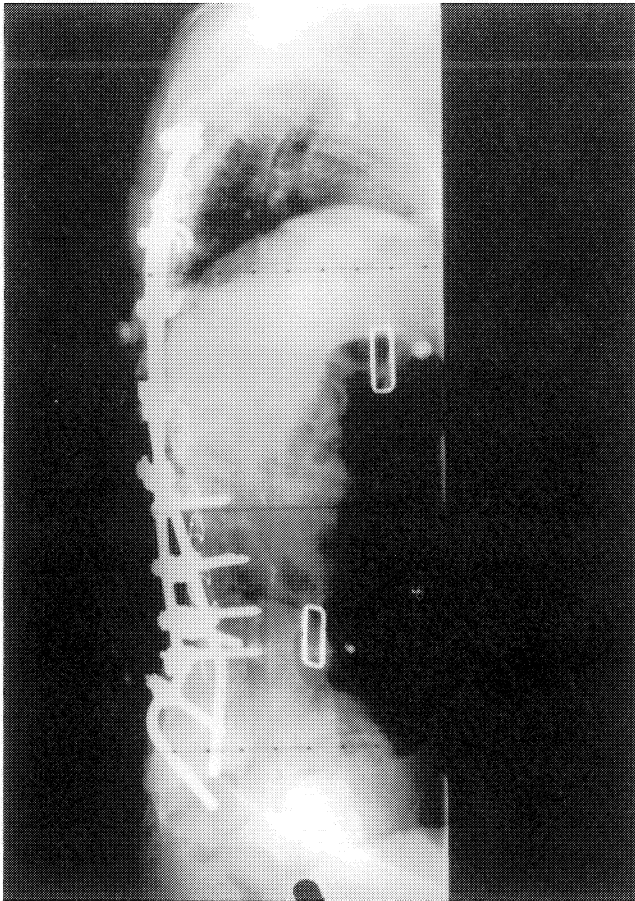


Figure 3F. Lateral view of the thoracolumbar spine following pseudoarthrosis excision and repeat anterior and posterior fusion with instrumentation.

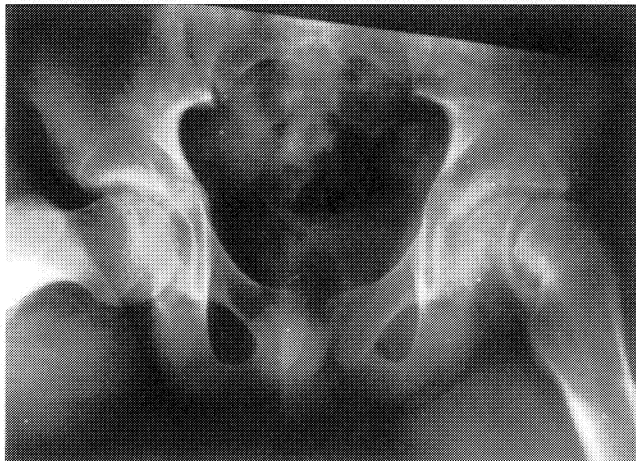


Figure 4A. AP pelvic radiograph demonstrating a left SCFE (male age fourteen).

Figures 4B, 4C. AP and frog lateral pelvic radiographs demonstrating position of the percutaneous screws (age sixteen).



Figure 4B.

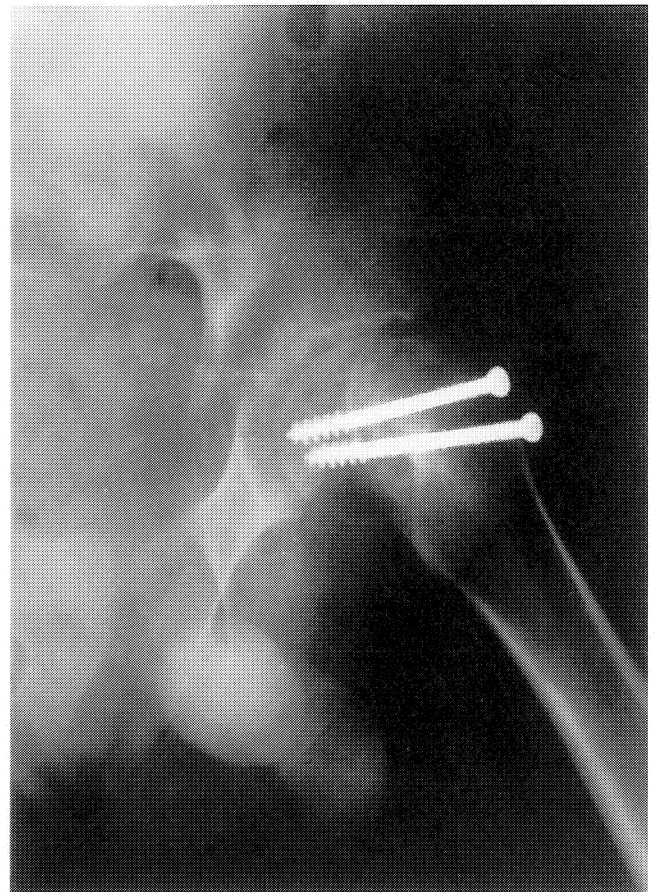


Figure 4C.



Figure 4D. Three-dimensional CT view demonstrating the posterior position of the screws in the femoral head with entry into the posterior acetabulum.

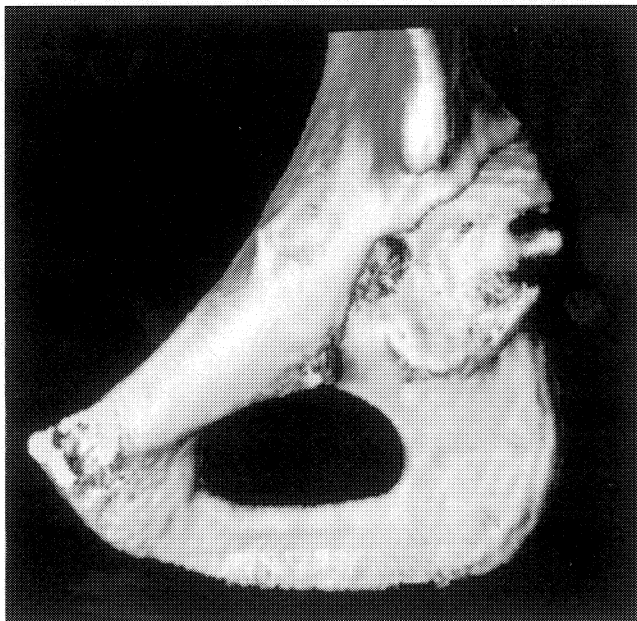


Figure 4E. Three-dimensional CT view demonstrating the posterior acetabular injury created by inappropriate screw placement.



Figure 5A. Male with acetabular fracture. AP radiograph of the left hip without obvious abnormality of the acetabulum.

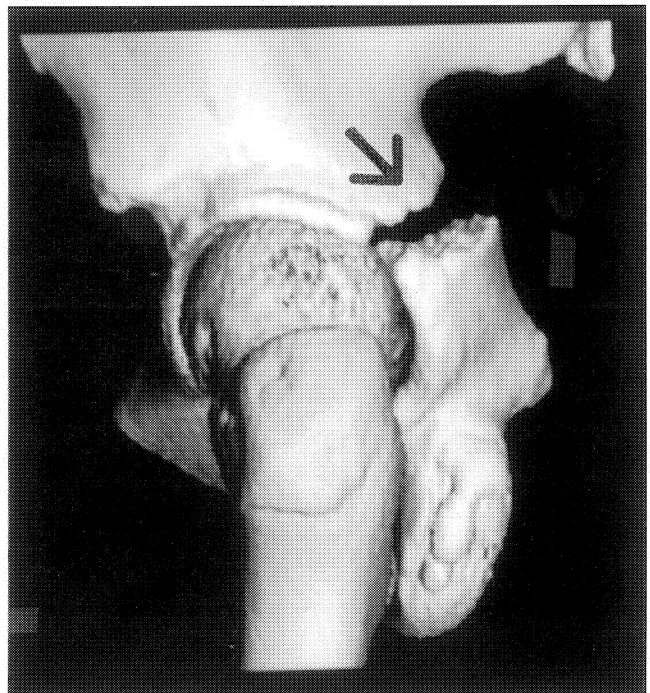


Figure 5B. Lateral three-dimensional CT view of the left hip (femoral head included) suggesting a triradiate cartilage injury (→).

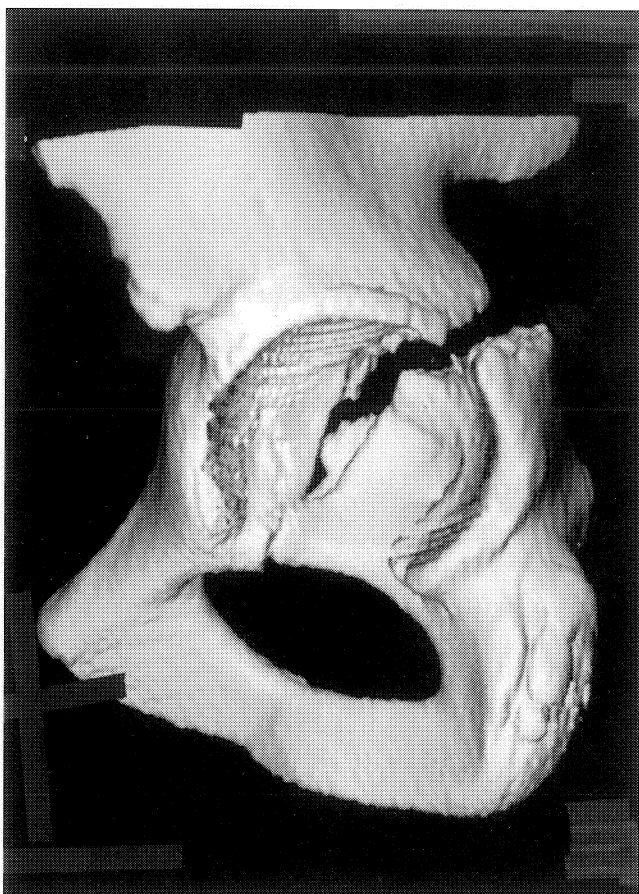


Figure 5C. Same view with the femoral head subtracted. Note the severe degree of injury.

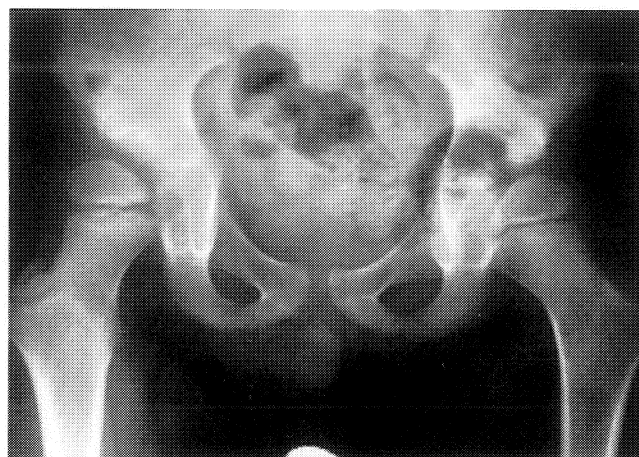


Figure 6B. AP pelvic radiograph at age five years demonstrating progressive dysplasia.

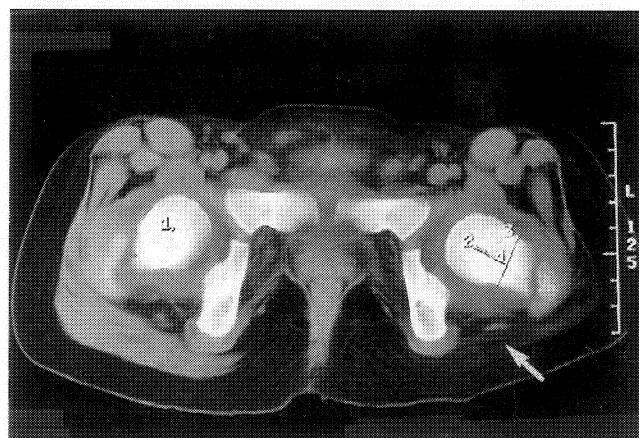


Figure 6C. Two-dimensional CT study demonstrating the marked loss of gluteal and abductor muscle mass that allowed the hips to subluxate (→).



Figure 6A. Male with an L4 spina bifida and acetabular dysplasia. AP pelvic radiograph demonstrating mild bilateral acetabular dysplasia (age two).

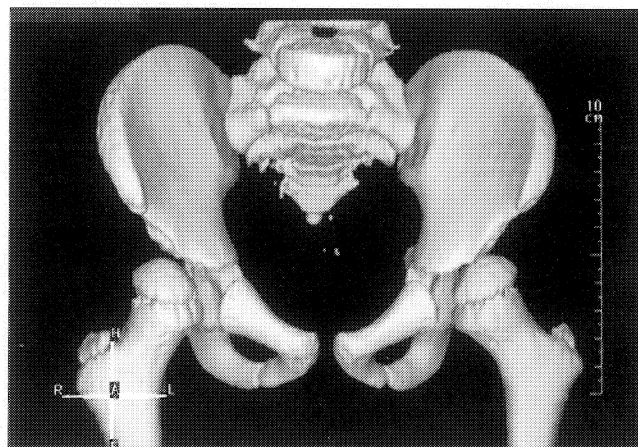


Figure 6D. Three-dimensional CT study of entire pelvis.



Figure 6E. Three-dimensional lateral CT view (femoral head included) demonstrating the hip flexion deformity and superior-posterior acetabular dysplasia.

DISCUSSION

These cases represent only a few of the many cases in which new technology has changed our understanding of orthopedic conditions. The understanding of the etiology and pathogenesis of many pediatric orthopedic diseases has been greatly improved by better visualization of the abnormalities and deformities present. As has been demonstrated, this method is of particular benefit in anatomic locations such as the hip and spine where three dimensional complexities make diagnosis and preoperative planning more difficult. Treatment plans were formulated and executed based on these images. It is interesting to postulate what the diagnosis and recommended treatment would have been without current technology. Several of these patients might have been told that their condition was not serious and did not require treatment. Furthermore, their long term function might be different without accurate diagnosis and treatment.

With the ever-increasing pace of new technology, how should we proceed? Almost certainly, current radiographic clarity of anatomy will be thought of as anti-

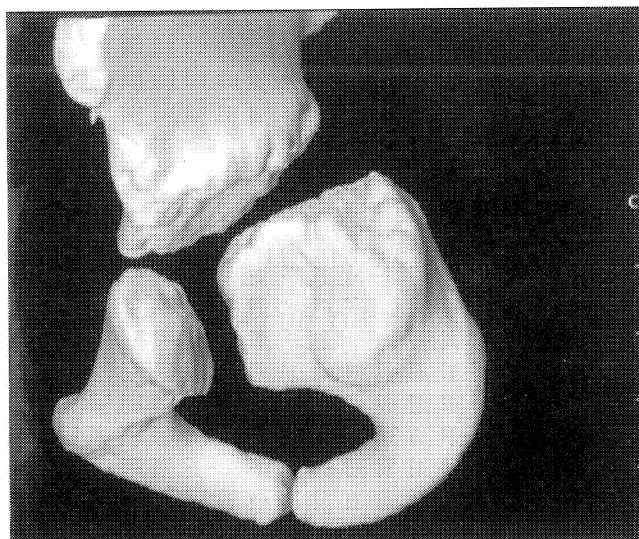


Figure 6F. Three-dimensional lateral CT view with the femoral head subtracted showing abnormal widening of the posterior wing of the triradiate cartilage.

quoted within a few years. One can only imagine the surprise and delight of surgeons in the late nineteenth century when suddenly they were able to peek at the skeleton using the newly discovered x-ray. The x-ray was the pinnacle in musculoskeletal diagnosis at that time. We would be remiss if we accepted current technology as the peak of medical achievement. Computer driven scientific advances will continue to radically change our understanding of the pathoanatomy of disease.

This rapidly changing technology, however, raises many moral and ethical questions. With the ever increasing costs of technology driven medical care, which communities in the world will be able to afford such costly advances? In the simplest form, one could consider third versus first world medicine. Why should these procedures be performed when people are still suffering from preventable diseases such as polio? Clearly, the overall costs of medical care make new technology too prohibitive when basic human needs still need to be met.

Some rural communities do not have medical care at all. These inequities have to be addressed prior to the wholesale use of new and expensive technology. These issues should not stop the progress of medical care; technology can help bridge this gap. Advances in tele-medicine, where physicians in rural communities can obtain new ideas as well as discuss cases with specialized physicians using computer technology, will address some of these imbalances.

The ethical dilemma of technology availability is perhaps most clearly illustrated within our current North

American setting. What is the obligation of the orthopedic surgeon who works in a small community hospital when faced with a condition that could be rapidly clarified with the technology available at a larger institution? In some cases, the treating surgeon may not be aware that better methods available elsewhere would greatly improve the patients outcome. In other instances the surgeon may recognize these deficiencies, but may not be certain of the cost benefit ratio of sending the patient elsewhere for the study. In a more negative scenario, a managed care administrator may deny the advanced study or the surgeon may choose not to order the study for fear of losing the experience of treating the case.

In summary, technological advances in medicine have radically improved a surgeon's approach to complex diseases, yet at the same time these advances have threatened diagnostic confidence. These changes will continue to accelerate, and the orthopedic surgeon of the future will require a clear understanding of how these changes influence the science and ethics of responsible practice in an era of advanced technology, yet finite resources.

DOES PARTICIPATION IN SPORTS CAUSE OSTEOARTHRITIS?

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Nancy E. Lane, M.D.**

Orthopaedic surgeons will be seeing an increasing number of people who want to know if participation in sports increases their risk of developing osteoarthritis. Since it has become clear that regular exercise, including recreational and competitive sports, improves general health and may increase longevity^{4,16,18,34,52}, greater numbers of middle age and older individuals want to participate in sports. Furthermore, the number of people in these age groups is growing and will continue to grow: between 1990 and 2010 the number of people over age forty-five in the United States will increase by forty million, from eighty-two million to 124 million. Helping these people regain or maintain their mobility and quality of life represents a major challenge for the orthopaedic community^{8,10}. Many of them who have normal joints will want to know if participating in sports or exercise programs increases their risk of developing degenerative joint disease, and others with early degenerative joint disease will want to know what types of physical activity accelerate the progression of their joint disease and what types of activity will maintain and possibly improve their joint function and general health.

The answers to these questions are not entirely clear. Studies of the relationships between habitual physical activity and osteoarthritis suggest that at least some types of repetitive joint use contribute to the development of degenerative joint disease. Surveys of individuals with physically demanding occupations including farmers, construction workers, metal workers, miners and pneumatic drill operators suggest that repetitive intense joint loading may lead to the early onset of joint degeneration^{11,35,62}. Specific activities that have been associated with osteoarthritis include repetitively lifting or carrying heavy objects, awkward work posture, vi-

bration, continuously repeated movements and working speed determined by a machine²¹. Other studies have suggested that participation in competitive sports and vigorous exercise increases the risk of osteoarthritis^{11,23,28,30,31,35,64,68}. In contrast to these reports of correlations between habitual increased physical activity and joint degeneration, other studies show that many individuals participate in sports for their entire lifetime without developing osteoarthritis^{6,11,20}. Furthermore, a study of habitually physically active women showed that they had less joint pain and stiffness than less active women⁷⁰; and examination of 589 men and 826 women in the Framingham cohort did not demonstrate an association between level of habitual physical activity in middle age and knee osteoarthritis at a mean age of seventy-three¹⁷. This study did show an association between increased habitual physical activity and asymptomatic osteophytes in men¹⁷. Recent experimental work showing that life long increased joint loading and use did not lead to degeneration of the articular surface in dogs⁴⁶ supports the belief that increased life long physical activity alone does not increase the risk of osteoarthritis in humans. Thus, if participation in sports causes osteoarthritis it does so by a mechanism other than increased frequency of joint motion.

One mechanism by which sports participation could cause joint degeneration is by damaging the articular surface through repetitive intense impact or torsional loading. Age related changes in articular cartilage could increase the probability of tissue damage and decrease the ability of the tissue to repair damage⁴¹. Age related changes in joint proprioception, joint capsules, ligaments and muscle function may also contribute to the probability of articular cartilage damage from impact and torsional loading. This article reviews our current understanding of the effects of impact and torsional loading on articular cartilage, the effects of regular exercise on animal joints, studies of the relationships between sports participation and osteoarthritis in humans and factors that affect the risk of osteoarthritis among people participating in sports.

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EFFECTS OF IMPACT AND TORSIONAL LOADING ON ARTICULAR CARTILAGE

The effects of slowly applied loads and suddenly applied loads on articular cartilage differ considerably. The articular cartilage extracellular matrix consists of water and a macromolecular framework formed primarily by collagens and large aggregating proteoglycans¹². The collagens give the tissue its form and tensile strength, and the interaction of aggregating proteoglycans with water gives the tissue its stiffness to compression, resilience and probably its durability. Loading of articular surfaces causes movement of fluid within the articular cartilage matrix that effectively distributes loads within the cartilage and to the subchondral bone⁴⁵. When this occurs slowly, the fluid movement allows the cartilage to deform and decreases the force applied to the matrix macromolecular framework. When it occurs too rapidly for fluid movement through the matrix and deformation of the tissue to occur, as with sudden impact or torsional joint loading during sports, the matrix macromolecular framework sustains a greater share of the force. Impact and torsional loading of synovial joints occurs frequently and direct blows to synovial joints less frequently during sports. Experimental work shows that acute or repetitive blunt trauma can damage cartilage without first causing visible tissue disruption⁷. The resulting compression or shear forces applied to an articular surface can rupture the cartilage matrix producing chondral fissures, flaps or fractures. Transarticular impact loads can cause fractures in the calcified cartilage zone-subchondral bone region that leave the articular surface intact^{9,12,65}.

In addition to the differences in the effects on articular cartilage, slowly and suddenly applied loads occurring during participation in sports or exercise may differ in the amount of force applied to the joint surface. Contraction of muscles can help protect joint surfaces from impact and torsional loads. When loads are applied slowly, muscle contraction can absorb much of the energy and stabilize joints. When loads are applied suddenly or unexpectedly, muscle contraction may not occur rapidly enough to stabilize joints and decrease the forces on the articular surfaces.

Disrupting normal articular cartilage with a single impact requires substantial force. A study of the response of human articular cartilage to blunt trauma showed that articular cartilage could withstand impact loads of up to twenty-five newtons per square millimeter (twenty-five MPa) without apparent damage. Impact loads exceeding this level caused chondrocyte death and cartilage fissures⁶¹. The authors suggested that reaching a stress level that could cause cartilage damage required a force greater than that necessary to frac-

ture the femur. Another study¹⁹ measured the pressure on human patellofemoral articular cartilage during impact loading and found that impact loads less than the level necessary to fracture bone caused stresses greater than twenty-five MPa in some regions of the articular surface. With the knee flexed ninety degrees, 50 percent of the load necessary to cause a bone fracture produced joint pressures greater than twenty-five MPa for nearly 20 percent of the patellofemoral joint. At 70 percent of the bone fracture load nearly 35 percent of the contact area of the patellofemoral joint pressures exceeded twenty-five MPa. At 100 percent of the bone fracture load, 60 percent of the patellofemoral joint pressures exceeded twenty-five MPa. These latter results show that impact loads can disrupt cartilage without fracturing bone.

Single impact loads may also cause damage in the regions of calcified cartilage and subchondral bone with minimal disruption of the articular surface^{50,65}. A transarticular load of 2170 newtons applied to canine patellofemoral joints consistently caused fractures in the zone of calcified cartilage visible by light microscopy⁶⁵. India ink staining and histologic examination showed articular cartilage fissures that extended from the articular surface to the transitional or superficial radial zone of the articular cartilage in some joints. Six months after impact loading, histologic examination and histochemical staining demonstrated a decrease in proteoglycan concentration and fibrillation of the articular surface in the region of the injury, suggesting that damage to the zone of calcified cartilage and subchondral bone occurring as a result of impact loading of the joint surface may contribute to the development of osteoarthritis.

Other experimental investigations show that repetitive impact loads split articular cartilage matrix and initiate progressive cartilage degeneration^{14,69}. Cyclic loading of human cartilage samples in vitro caused surface fibrillation⁶⁹, and periodic impact loading of bovine metacarpophalangeal joints in vitro combined with joint motion caused degeneration of articular cartilage⁵⁹. Repeated overuse of rabbit joints in vivo combined with peak overloading caused articular cartilage damage including formation of chondrocyte clusters, fibrillation of the matrix, thickening of subchondral bone and penetration of subchondral capillaries into the calcified zone of articular cartilage¹⁴. The extent of cartilage damage appeared to increase with longer periods of repetitive overloading, and deterioration of the cartilage continued following cessation of excessive loading. This latter finding suggests that some cartilage damage is not immediately visible.

An investigation of cartilage plugs also showed that repetitive loading disrupted the tissue and that the severity of the damage increased with increasing load and increasing number of loading cycles⁷³. Two hundred and fifty cycles of a 1000 pound per square inch compression load caused surface abrasions. Five hundred cycles produced primary fissures penetrating to calcified cartilage, and 1000 cycles produced secondary fissures extending from the primary fissures. After 8000 cycles the fissures coalesced and undermined cartilage fragments. Higher loads caused similar changes with fewer cycles. The experiments suggested that repetitive loading can cause propagation of vertical cartilage fissures from the joint surface to calcified cartilage and extension of oblique fissures into areas of intact cartilage, extending the damage and creating cartilage flaps and free fragments.

Clinical studies have identified articular cartilage fissures, flaps, free fragments and changes in subchondral bone similar to those produced experimentally by single and repetitive impact loads^{7,37}. In at least some patients, acute impact loading of the articular surface or twisting movements of the joint apparently caused these injuries. In other patients, the cartilage damage may have resulted from repetitive loading. Magnetic resonance imaging of joints soon after an acute impact or torsional load occasionally shows changes in subchondral bone consistent with damage to the zone of calcified cartilage and subchondral bone even when the articular surface is intact⁶⁶. Many patients with osteoarthritis who participated in sports do not have a clear history of joint injuries, but the diagnosis of injuries limited to articular cartilage or subchondral bone is difficult. Because cartilage lacks innervation, cartilage damage does not cause pain directly; and physical examination and plain radiographs do not show abnormalities. As a result most of these injuries probably are not detected. For these reasons, unrecognized joint injury may be one of the primary risk factors for the development of osteoarthritis associated with participation in sports that expose joints to high levels of impact and torsional loading.

EXPERIMENTAL STUDIES OF THE EFFECTS OF EXERCISE ON ARTICULAR CARTILAGE

Animal experiments have provided considerable information about the effects of repetitive in vivo loading of articular cartilage. In particular they show that increased joint use and impact loading produce different results and that the effects of joint use differ between normal joints, unstable joints and unstable joints with decreased sensory innervation.

Normal Joints

In one series of investigations the effects of regular running on dog joints depended on the distance the animals ran. Moderate running (four kilometers/day five days a week for forty weeks) increased cartilage thickness, proteoglycan content and indentation stiffness^{22,26,27}. A period of more strenuous running (twenty kilometers/day five days a week for fifteen weeks) decreased cartilage thickness and proteoglycan content. Longer term low impact strenuous running (forty kilometers/day for up to one year) decreased cartilage proteoglycan concentration and indentation stiffness and stimulated remodeling of subchondral bone, but these animals did not develop degenerative joint disease^{2,51}. These studies suggest that a limited period of long distance running alters articular cartilage composition and mechanical properties, but it does not accelerate joint degeneration. They also suggest that the reported alterations in the articular cartilage and subchondral bone may reflect adaptation of the joint to repetitive motion and loading rather than early signs of degeneration.

A recent study examined the effects of life long moderate running with added weight in beagles⁴⁶. Animals were divided into two groups: exercised animals (running four kilometers a day with jackets weighing an average of 11.5 Kg, 130 percent of the adult animal's weight, five days a week for 550 weeks) and control animals (cage activity for 550 weeks). At the completion of this study the synovial joints of animals participating in the exercise regimen did not differ grossly or microscopically from the joints of animals limited to cage activity. Mechanical testing of the articular cartilage did not show any differences between exercised and control animals. This investigation indicates that life long moderate exercise, even with more than normal body weight, did not increase the probability of joint degeneration.

In vivo impact loading of normal animal joints produces different results than increased joint use due to running or increased loading⁴⁶. Animal joints subjected to limited periods of forced repetitive impact loading developed degenerative changes similar to those seen in osteoarthritis in humans^{39,57-59}, suggesting that short periods of repetitive impact loading are more likely to cause joint degeneration than life long running.

Abnormal Joints

Although moderate exercise apparently does not adversely affect normal animal joints, it may accelerate degeneration or prevent repair of injured articular cartilage. In one investigation, cast immobilization of dog limbs for six weeks increased cartilage water content and decreased cartilage thickness, proteoglycan synthe-

sis and proteoglycan aggregation. Ad lib activities appeared to slowly reverse these changes, but running six miles a day prevented reversal of the proteoglycan aggregation defect and further decreased cartilage thickness.⁵⁴ Other work shows that forced activity following enzymatically induced articular cartilage injury or anterior cruciate ligament transection^{53,72} can cause joint degeneration, whereas immobilization following injury may prevent degeneration.

Chronic joint instability may also influence the response of a joint to exercise. Experimental models of joint degeneration show that mechanical instability created by transection of the anterior cruciate ligament and meniscectomy can lead to progressive joint degeneration⁴⁴. These studies have not defined the mechanisms responsible for degeneration of the articular surface in unstable joints, but possible adverse effects of joint instability include altering the congruence and regions of contact of the opposing articular surfaces, increasing the number and intensity of impact loadings, and increasing shear and compression forces on some regions of the cartilage. However, instability does not necessarily cause predictable progressive erosion of the articular surface; the tissue can respond to the changes in loading. In one set of experiments transection of dog anterior cruciate ligaments increased cartilage proteoglycan synthesis and concentration⁵. Cartilage thickness, measured by magnetic resonance imaging three years after surgery, also increased. These changes may represent the early stages of osteoarthritis; but clinical experience suggests that in some patients, unstable joints become more stable with increasing patient age, and that even though some of these joints develop osteophytes they do not always develop other symptoms and signs of degeneration.

Joint instability combined with loss of sensory innervation can dramatically increase the susceptibility of joints to degeneration with use. Sensory denervation by dorsal root ganglionectomy did not cause degenerative joint disease in dogs followed for sixteen months, but ganglionectomy followed by anterior cruciate ligament transection led to severe degenerative changes within three weeks of ligament transection⁴⁸. Partial loss of joint innervation also accelerated the development of degenerative joint disease in dogs with anterior cruciate ligament transection⁴⁹. These studies indicate that sensory denervation accelerates the development of degenerative disease in unstable joints. They also suggest that mild neurologic disorders or partial joint denervation at the time of surgery could accelerate the development of osteoarthritis in humans⁴⁹.

CLINICAL STUDIES OF THE RELATIONSHIP BETWEEN SPORTS PARTICIPATION AND OSTEOARTHRITIS

Variability among individuals in predisposition to develop joint degeneration, exercise patterns and joint injuries make studies of the relationship between sports participation and osteoarthritis in humans difficult to perform and evaluate¹¹. People probably vary in their susceptibility to joint degeneration due to differences in joint shape, congruence, stability and innervation, muscle strength, body mass, response of their tissues to exercise and genetically determined composition and metabolism of articular cartilage. Those who participate in sports may differ in their predisposition for the development of osteoarthritis from those who choose not to exercise regularly. For example, people who have the greatest risk for osteoarthritis may develop mild joint symptoms after minimal activity. These symptoms may lead them to avoid participation in sports or vigorous exercise. Therefore, people who choose to participate in sports regularly for many years may be those who are least likely to develop osteoarthritis. Different types of exercise differ in their demands on specific joints and the response to exercise may differ among joints. For example, a sport or recreational activity that causes degenerative changes in the knee may not affect the ankle or hip. Certain sports and recreational activities increase the risk of joint injuries that could lead to joint degeneration^{32,43}. These injuries include not only fractures, dislocations, meniscal tears and ligament injuries, but joint contusions. Therefore, people who choose to exercise regularly and participate in certain sports may have a higher prevalence of osteoarthritis as a result of more frequent joint injuries when compared to less active people.

Since few competitive or recreational long distance runners suffer severe joint injuries and many regular runners can recall how long and how often they have run, studies of these individuals provide one of the best opportunities to examine the relationship between exercise and osteoarthritis^{11,35}. Despite their methodologic problems, including the inability to randomly assign individuals to running and non-running groups and difficulties in reliably diagnosing osteoarthritis, these studies provide insight into the relationship between regular joint use and development of joint degeneration. In one investigation, forty-one long distance runners were compared to matched controls^{33,35,36}. Runners with a mean age of sixty who had run an average of 180 minutes a week for twelve years did not have a greater prevalence of osteoarthritis, although they did have a 40 percent greater density of their vertebral bone. Another investigation compared seventeen people with a

TABLE 1. DIFFERENCES AMONG SPORTS AND EXERCISE ACTIVITIES IN LEVEL OF JOINT IMPACT AND TORSIONAL LOADING

Level of Impact or Torsional Loading of Joints	Examples	
Low	<ul style="list-style-type: none"> • Recreational Swimming • Stationary Rowing or Skiing • Calisthenics 	<ul style="list-style-type: none"> • Golf • Walking • Ballroom Dancing • Water Aerobics
Moderate	<ul style="list-style-type: none"> • Bowling • Fencing • Bicycling • Rowing • Ice Skating • Rock Climbing • Low Impact Aerobics • Doubles Tennis • Jazz Dancing & Ballet • Aerobics 	<ul style="list-style-type: none"> • Weight Lifting • Sailing • Speed Walking • Cross-Country Skiing • Table Tennis • Canoeing • Hiking • Horseback Riding • In-Line Skating • Downhill Skiing
High	<ul style="list-style-type: none"> • Baseball/Softball • Basketball/Volleyball • Football • Handball/Racquetball • Running • Squash 	<ul style="list-style-type: none"> • Lacrosse • Soccer • Rugby • Singles Tennis • Water Skiing • Karate

mean age of fifty-six who had run an average of twenty-eight miles per week for twelve years with eighteen non-runners⁵⁵. Runners had no more complaints of pain and swelling of the hips, knees, ankles and feet than non-runners, and radiographic examination of the joints of the two groups did not show any differences. The hip joints of seventy-four former championship distance runners with a mean age of fifty-five who had competed for an average of twenty-one years showed no greater prevalence of osteoarthritis than controls⁵⁶. However, another study of long distance runners with a mean age of forty-two did show more radiographic evidence of hip degenerative disease in the runners than in bobsled competitors⁴⁰, and increased running pace was associated with increased radiographic evidence of joint degeneration. Furthermore, a retrospective cohort study of eighty-one female ex-elite athletes ranging in age from forty to sixty-five showed that the ex-athletes (runners

and tennis players) had a two to three times greater prevalence of radiographic osteoarthritis, primarily osteophytes⁶⁴. In considering these studies it is important to appreciate that radiographic evidence of osteophyte formation may not necessarily correlate with degeneration of articular cartilage and osteoarthritis.

Individuals who participate in sports that subject joints to more intense impact and torsional loading than running may have an increased prevalence of osteoarthritis. Participants in American football appear to have an increased incidence of degenerative change in multiple joints, possibly due to joint injuries^{11,43,60,67}. One investigator reported that more than 80 percent of American football players with a history of a knee injury had evidence of osteoarthritis ten to thirty years after competing⁶⁰. Baseball pitchers appear to be at increased risk of osteoarthritis of the elbow and shoulder^{1,3} and competitive soccer players have been shown to have an in-

TABLE 2. FACTORS THAT MAY INCREASE THE RISK OF JOINT DEGENERATION WITH PARTICIPATION IN SPORTS

- Previous joint injury or surgery
- Muscle weakness
- Joint incongruity/Joint dysplasia
- Joint instability
- Neurologic deficit (sensory or motor)
- Increased weight

creased prevalence of osteoarthritis in the lower extremity joints compared with age matched controls^{30,31,38}.

Although the available clinical studies have significant limitations, this information combined with our current understanding of the effects of impact and torsional loading on joints allow us to identify sports that may increase the risk of osteoarthritis, especially for older individuals. Table 1 groups sports and other physical activities by estimated intensity, rate and frequency of impact, and torsional loading of joints. Clearly the intensity, rate and frequency of impact loading of joints will vary among individuals participating in the same sport, with different types of equipment including shoes and playing surfaces, and with the level of competition.

FACTORS THAT INFLUENCE THE RISK OF OSTEOARTHRITIS ASSOCIATED WITH PARTICIPATION IN SPORTS

A variety of factors may increase the risk of joint degeneration with participation in sports (Table 2) including genetically determined abnormalities of articular cartilage^{15,71}. Joint instability or disturbances of mechanical function due to loss of normal ligament or meniscal function may accelerate the development of degenerative changes with participation in sports^{13,63}. A study of twenty middle age runners with knee pain for at least three months suggested that joint abnormalities may be related to the development of degenerative disease in association with running⁴². In this investigation, twenty individuals with a mean age of thirty-nine who had run an average of sixty-two miles a week for twenty years were examined clinically and radiographically. Six of the twenty had evidence of degenerative joint disease. All of these individuals had genu varum, and four had a history of previous knee injury. Radiographic degenerative changes were associated with genu varum, a history of previous severe joint injury and more years of long distance running. Studies of people with knee ligament and meniscal injuries indicate that following

TABLE 3. MEASURES THAT MAY DECREASE THE RISK OF JOINT DEGENERATION WITH PARTICIPATION IN SPORTS

- Select sports/exercise programs that subject joints to low levels of impact and torsional loading and injury
- Use equipment and playing/running surfaces that decrease joint impact and torsional loading
- Maintain/improve muscle strength and tone to improve ability of muscles to decrease joint impact loading and protect joints from injury
- Maintain/improve general conditioning to decrease risk of joint injury due to fatigue
- Decrease body weight
- Alternate sports/exercise activities (cross training) to decrease repetition of same patterns of joint loading and motion

these injuries the risk of developing degenerative changes increases^{24,25,47,63}, and the time interval between joint injury and the development of osteoarthritis may be significantly shorter in older people⁶³. Other factors that may increase the risk of joint degeneration include joint incongruity due to injury or joint dysplasia, muscle weakness, neurologic deficits and increased weight^{15,29,31}.

The available evidence suggests that the risk of developing osteoarthritis as a result of participation in sports can be decreased (Table 3). Appropriate diagnosis, treatment and rehabilitation following injury should decrease the risk of subsequent joint injury and osteoarthritis. Other measures that may help preserve normal joint structure and function include selection of sports and exercise programs that avoid high frequency, rate and intensity of joint loading and injury, and use of sports and exercise equipment that minimize joint impact loading. Maintaining or improving muscle strength and tone and general conditioning should help prevent joint injury and minimize articular surface loading due to muscle weakness and fatigue; and alternating sports or exercise activities will decrease the number of repetitions of the same pattern of joint loading and motion.

SUMMARY AND CONCLUSIONS

Many middle age and older people question whether participation in sports or exercise programs increases their risk of developing osteoarthritis or accelerates

degeneration of joints with minimal osteoarthritis. Although articular cartilage undergoes significant age related changes in chondrocyte function and matrix composition, the available evidence shows that moderate habitual exercise by middle age and older people does not increase their risk of developing osteoarthritis. Carefully selected sports and exercise programs improve strength and mobility in older people and people with mild and moderate osteoarthritis. In particular, life long activities such as stationary bicycling, skiing, rowing, swimming, golf, jogging or even moderate low impact running, and racquet sports do not appear to increase the risk for development of osteoarthritis in middle age and older people with normal muscle strength and normal joints (Table 1). However, people who participate in competitive sports as adolescents or young adults, or habitually engage in sports or vigorous exercise throughout life, appear to have an increased risk of developing asymptomatic osteophytes that are not associated with articular cartilage degeneration. Joint injury increases the risk of osteoarthritis, and sports that subject joints to repetitive high levels of impact and torsional loading increase the risk of joint injury and degeneration. It seems likely that the osteoarthritis that develops in some individuals who participated in or continue to participate in competitive sports results from undiagnosed joint injuries. Measures that may decrease the intensity and frequency of impact and torsional loading of joints (Table 3) include use of sports equipment that decrease joint impact loading, like impact absorbing shoes and playing surfaces, and maintaining or improving muscle strength, tone and general conditioning so that muscle contractions help protect joints from injury and high impact. Participation in a variety of sports and exercise activities, rather than limiting physical activities to one sport or type of exercise, should decrease the risk of joint injury due to excessive repetition of the same pattern of joint motion and loading. Individuals with abnormal joint anatomy or alignment, previous significant joint injury or surgery, joint instability, well above average body weight, disturbances of joint or muscle innervation, or inadequate muscle strength probably have increased risk of degenerative joint disease from exercise that subjects their joints to loads greater than those that result from normal activities of daily living, especially activities that involve repetitive impact or torsion (Table 2). These individuals and people with early osteoarthritis can benefit from regular physical activity, but they should have a careful evaluation of their joint structure and function before participating in sports or intense exercise programs. In most instances they would be best advised to select physical activities that maintain joint motion and

muscle strength with minimal impact or torsional loading, thereby gaining improved general health and mobility with minimal increased risk of osteoarthritis.

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SEPTIC ARTHRITIS CAUSED BY CHRONIC OSTEOMYELITIS

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ABSTRACT

We have treated four cases of previously quiescent osteomyelitis which presented as septic arthritis in an adjacent joint. The osteomyelitic focus was in the bone proximal to the involved joints (zero to ten centimeters above the joint line). Based on the presenting history, physical findings, laboratory tests and cultures of joint fluids, the joint sepsis was low grade in all patients which led to delays in diagnosis and treatment. Aggressive surgical debridement of both bone and joint, followed by a prolonged course of antibiotics led to resolution in all patients. A high index of suspicion combined with adequate radiographs of the surrounding bones should lead to the appropriate diagnosis and treatment.

INTRODUCTION

Osteomyelitis can remain dormant and asymptomatic for many years (reports of up to seventy years) before reactivating to cause clinically symptomatic osteomyelitis⁸. More commonly, chronic osteomyelitis produces intermittent bone pain, tenderness, and purulent drainage followed by periods of quiescence. In some cases, the drainage is continuous. Although acute septic arthritis is a well known complication of acute osteomyelitis^{1,9}, septic arthritis has only rarely been recognized as a complication of previously quiescent chronic osteomyelitis⁷. We have treated four cases of acute septic arthritis in a joint adjacent to an unrecognized focus of chronic osteomyelitis. In all cases the complete diagnosis was delayed because of the rare occurrence of this association and the indolent nature of the septic arthritis. In these four cases we have sought features in the presentation and work-up that suggest this unusual association and recommend appropriate treatment based on our experience.

MATERIALS AND METHODS

The charts and radiographs of four patients with septic arthritis associated with a previously quiescent site of osteomyelitis were reviewed to determine the pre-

TABLE I
Clinical, Laboratory, and Radiographic Findings on the Four Patients

History	Sex, Age (years)	Onset of arthropathy	Past History	Temp °C	ESR	WBC	Distance of focus from joint on plain radiographs
1	F, 25	2 weeks pain, swelling Lt knee	Large soft tissue injury to Lt thigh requiring skin grafting, age 7	37.0	22	9,400	Medial femoral condyle adjacent to joint
2	F, 30	2 weeks pain Lt elbow	Supracondylar pin tract infection, age 5	36.9	47	18,000	4 cm above elbow
3	M, 60	5 days swollen, painful Lt knee; unable to bear	Surgical treatment of osteomyelitis Lt	37.4	Not done	12,800	10 cm above knee
4	M, 45	4 week history of pain and swelling Rt ankle	Osteomyelitis Rt distal tibia, age 12	38.4	127	15,300	4 cm above ankle

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Figures 1A-B. AP and lateral radiographs of the left knee from Case 3. There is a 1.0 x 2.5 cm well defined lytic defect in the distal left femur on the AP view (arrows). This lucency is not evident on the lateral view. The lateral view does reveal a suspicious, poorly defined radiolucency in the distal femur. A left knee effusion is present. Initial knee films did not include the distal femur diaphysis and missed the proximal lesion.

senting history, past history, physical examination, and initial laboratory values. For each of these patients, we determined the location and radiographic appearance of the osteomyelitic focus. The cell counts and gram stains of joint fluid aspirates along with specimens for microbiology and pathology were compared. The type of surgical and antimicrobial treatment of each case was recorded. Follow-up data included the presence of pain in the involved joint with weightbearing and range of motion, as well as radiographic evidence of obliteration of the osteomyelitic cavity and joint space narrowing.

RESULTS

The patients' histories, laboratory data and distance of the radiographic focus of osteomyelitis from the involved joint are summarized in Table I. All four patients had a remote history (minimum eighteen years) of infection or trauma in the involved bone that predisposed them to having chronic osteomyelitis. However, none of the patients had symptoms suggestive of, or

had had treatment for, chronic osteomyelitis. Prior to the onset of the arthropathy, all four patients had been asymptomatic.

The onset of the arthropathy was gradual in all patients (range five days to four weeks). At presentation, only one patient had an elevated temperature and none had localized tenderness over the bone or evidence of drainage or sinuses. Three patients had an elevated white blood cell count and erythrocyte sedimentation rate (ESR).

The radiographs of the actual joint were normal in all four patients. However, the bone proximal to the joint demonstrated an osteolytic lesion in all four cases. In one patient (Case 1) this lesion was obvious on the initial radiographs because it abutted the joint. However, in the other three patients (Cases 2, 3 and 4), the lesion was remote from the joint by four or more centimeters. In patient 3, the lesion was not well delineated by the initial knee radiographs which were read as normal (Figure 1). All four joints were evaluated with com-

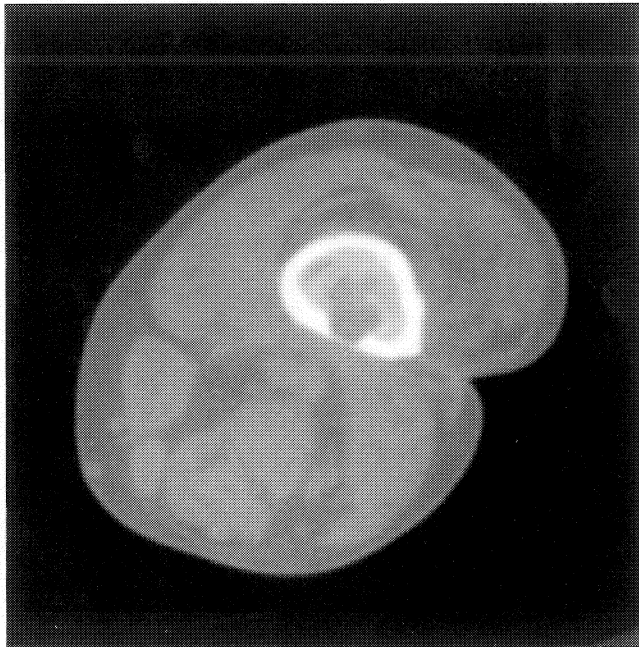


Figure 1C. Transverse CT image through the level of the defect from Case 3 demonstrates the well defined lytic lesion involving both the medullary canal and the adjacent cortex posteriorly.

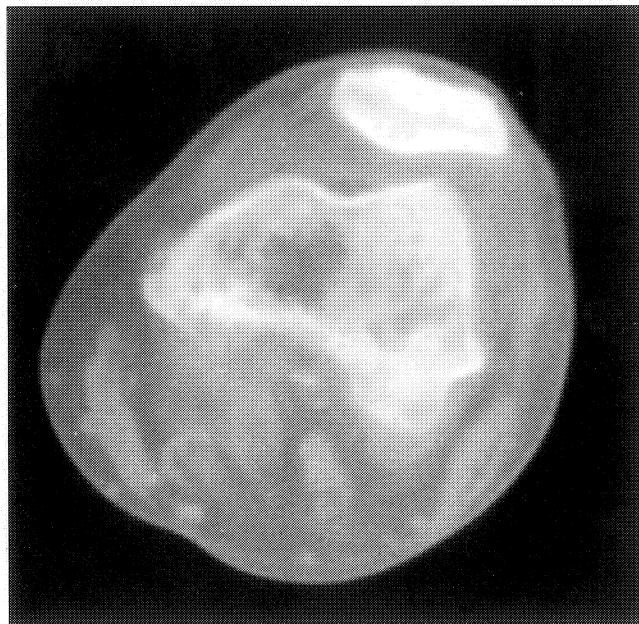


Figure 1D. This more distal transverse CT image at the level of the femoral condyles demonstrates a destructive process in the medial femoral condyle that was not as well visualized on routine X-ray. There is a suggestion of posteromedial intra-articular extension of the osteomyelitic focus.

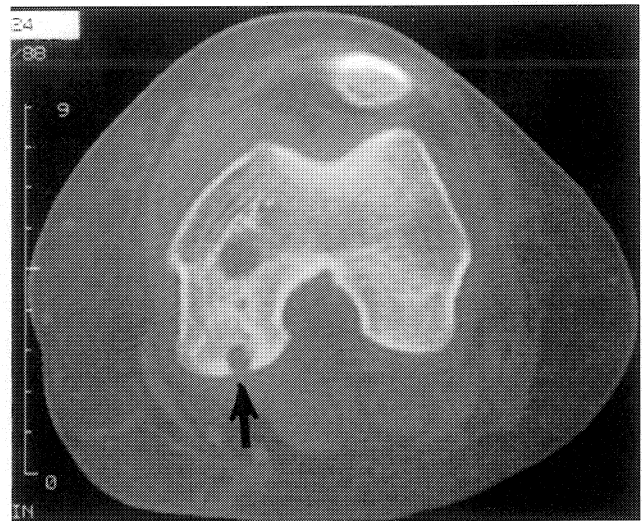


Figure 2. Transverse CT image at the level of the condyles (Case 1) demonstrates destructive lesions in the condyle as well as definite cortical destruction suggesting an osteomyelitic focus with intra-articular communication (arrow).

puted tomography (CT). Direct extension between the lytic lesion and joint space was clearly demonstrated by CT in cases 1, 3 and 4 (Figures 1C, 1D, 2).

Table II shows the results of the joint fluid analysis, cultures, and synovial pathology. The joint fluid cell count was inflammatory in all three joints in which it was tested. Positive cultures were found in three of five initial joint fluid aspirates (one joint [patient 1] was aspirated twice). Gram stains obtained pre-operatively from the joint fluid, and intra-operatively from the joint fluid and lytic bone area were positive in four out of twelve specimens. Intraoperative joint fluid cultures were negative in two out of four joints, but specimens from the osteolytic focus were positive in three of the four. The microscopic examination of the synovial specimens revealed both acute and chronic inflammation in all three joints when it was examined. None of the patients were on antibiotics prior to the initial joint fluid aspirates and only patient 3 received antibiotics (Imipenem, Cefazolin and Nafcillin) prior to intraoperative cultures. This data indicates that these joint infections were low grade and the organism was cultured from joint fluid in approximately one half of specimens tested.

Table III summarizes the treatment and follow-up of these patients (average fourteen months). All patients were treated with surgical irrigation and debridement of both the joint and osteolytic focus, followed by a prolonged course of intravenous antibiotics. In three of the patients we approached the bony lesion through a separate incision to secure drainage and avoid further communication with the joint. In one case (case 2) the

TABLE II
Joint Fluid Analysis, Cultures and Pathology of the Involved Areas

Case	Joint Fluid Aspirate-Preop			Joint Fluid-Intraop		Om Focus-Intraop		Synovial Pathology
	Cell/mm ³	Gm. Stain	Culture	Gm. St.	Culture	Gram Stain	Culture	
1	25,900 85% Neut.	Neg.	First-Neg. Second- Pseudomonas maltophilia	Neg.	Neg.	Neg.	Neg.	Acute and chronic inflammatory cells
2	56,000	Neg.	2 colonies S. aureus	Neg.	Neg.	Neg.	Staph aureus	Acute and chronic inflammatory cells
3	67,000 98% Neut.	Neg.	Neg.	Neg.	one colony S. aureus	gm + cocci	Staph s aureus	Not done
4	not available	gm. + cocci	S. aureus	gm. + cocci	S. aureus	gm + cocci	Staph aureus	Acute and chronic inflammatory cells

osteomyelitic focus was decompressed through the joint by open curettage.

Patients 3 and 4 illustrate how difficulties in identifying the etiology of the septic arthritis led to initially inadequate treatment. In patient 3, the osteolytic focus was not recognized on the initial knee radiographs (Figures 1A, 1B). The patient was treated with joint aspiration and intravenous antibiotics. Further plain radiographs and a CT scan led to joint arthrotomy and curettage of the osteolytic focus, each through a separate incision. In patient 4, the significance of an osteolytic focus above the ankle was not recognized at the time of the initial surgery which consisted only of joint lavage and debridement. Failure of resolution of the joint sepsis postoperatively led to further delineation by CT, three additional irrigations and debridements, and placement and removal of antibiotic impregnated methylmethacrylate beads before the process was clinically resolved.

At the time of the most recent follow-up, three of the four patients had painless, full range of motion of the involved joint (cases 1, 2 and 4). Patient 3 continued to have poor motion and inability to weight-bear secondary to pain. All patients had evidence of a healed osteolytic lesion and a preserved joint space.

DISCUSSION

Chronic osteomyelitis presenting as low grade, indolent septic arthritis has only rarely been described in the literature. LeGoff et al.⁷ reported two cases of acute monoarthritis caused by an adjacent focus of chronic osteomyelitis. The first patient had a seven year

history of multiple episodes of acute monoarthritis treated with anti-inflammatories. In retrospect, the osteomyelitis was evident on the initial presenting radiographs. The patient's only predisposing history was chronic furunculosis and diabetes. Their second patient had a three month history of acute monoarthritis, initially thought to be rheumatologic in origin. An underlying focus of osteomyelitis of unknown age was subsequently found.

Our patients presented with the insidious onset of septic arthritis quite different from the classic presentation of acute septic arthritis which includes a hot, swollen and painful joint with decreased range of motion and systemic symptoms such as fever, chills, skin lesions and sepsis. In contrast, our patients had clinically low grade septic arthritis (i.e. minimal fever, low systemic leukocytosis [except patient 4] and only a moderate inflammatory white blood cell count in the joint fluid) (Table I). Preoperative and intraoperative gram stains were helpful in only one joint (25 percent). In three patients, radiographic evidence of osteomyelitis was subtle or not included on the films (Figure 1A). Due to the unusual presentations, the diagnosis of septic arthritis was delayed in two of the four cases, and osteomyelitis was not initially considered on the differential list.

We suggest that the cause of the septic arthritis was quiescent osteomyelitis that reactivated and seeded the joint. The intraoperative and CT findings of communication between the focus of chronic osteomyelitis and the joint suggest direct inoculation as the cause of the septic arthritis (Figure 2). The indolent focus of osteo-

TABLE III
The Treatment and Follow-up of Four Patients

Case	Treatment			Follow-Up		
	Operations	Approach used	IV Antibiotics	Time	Clinical	X-rays
1	Irrigation; Debridement	Bone window through separate incision	6 weeks Cefazidime	18 months	0-125° flexion, pain-free	Healed lesion; joint space preserved
2	Irrigation; Debridement	Curettage metaphyseal defect through same incision	6 weeks Nafcillin	2 years	0-135° flexion, pain-free	Healed lesion; joint space preserved
3	Irrigation; Debridement	Bone window through separate incision	4 weeks Nafcillin, then 4 weeks Cefazolin	7 months	45-80° flexion, nonambulatory with pain	Healed lesion; joint space preserved
4	Irrigation; Debridement; Placement and removal of antibiotic impregnated cement spacer	Bone window through separate incision and curettage of metaphyseal defect through same incision	8 weeks Nafcillin	1 year	20° dorsi and 30° plantarflexion, pain-free	Large bony defect distal tibia; joint space preserved

myelitis likely decompressed itself into the joint as described by LeGoff et al.⁷ A small bacterial inoculum accounted for the low grade joint sepsis. Once the diagnosis of septic arthritis and osteomyelitis was made, a thorough irrigation and debridement of the entire osteomyelitic focus was necessary for cure. Intraoperative cultures taken from the osteomyelitic focus grew the offending organism more commonly than cultures of the joint fluid. Failure to recognize the associated osteomyelitis can lead to continued joint infection despite the use of appropriate antibiotics (patient 4). Quality radiographs and CT scans of the bone involved facilitate the diagnosis of joint infection secondary to chronic osteomyelitis and aid preoperative planning for adequate surgical treatment. In all four cases, arthrotomy, windowing and curetting the osteolytic lesion, and intravenous antibiotics prevented recurrence of septic arthritis.

In patients presenting with low grade septic arthritis and a remote history of local trauma or infection, the diagnosis of septic arthritis secondary to chronic osteomyelitis should be considered. If any suspicion of osteomyelitis arises, radiographs including the entire bone above and below the joint are indicated. If the extent of the lesions are not clear on the plain radiographs, CT scans are appropriate for preoperative planning for extra-articular debridement of the bone lesion via a cortical window made away from the joint. Aggressive and early surgical treatment with full knowledge of the extent of the osteomyelitis may decrease the need for multiple surgical procedures. Following debridement, long term antibiotic therapy seems necessary for cure.

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LIMB LENGTHENING IN TURNER SYNDROME

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ABSTRACT

We report the results and complications of eight consecutive patients who underwent bilateral tibial lengthenings for dwarfism associated with Turner syndrome. Lengthening was performed via distraction osteogenesis with monolateral external fixation. Tibias were lengthened an average distance of 9.2 centimeters or 33 percent of the original tibial length. The average total treatment time was 268 days. The overall complication rate was 169 percent for each tibia lengthened and each segment required an average of 1.7 additional procedures. Seven cases (44 percent) required Achilles tendon lengthening and nine cases (56 percent) developed angulation before or after fixator removal; six of these segments required corrective osteotomy for axial malalignment. Two cases (12.5 percent) developed distraction site nonunion and required plating and bone grafting. From this series we conclude that tibial lengthening via distraction osteogenesis can be used to treat disproportionate short stature in patients with Turner syndrome. However, the benefit of a cosmetic increase in height may not compensate for the high complication rate. Efforts to determine the psychosocial and functional benefits of limb lengthening in patients with short stature is necessary to determine the true cost-benefit ratio of this procedure.

INTRODUCTION

Limb lengthening via distraction osteogenesis can be utilized for the treatment of limb length discrepancies greater than five centimeters at maturity. In addition, limb lengthening can be useful for patients with short stature due to skeletal dysplasias, endocrine abnormali-

ties and idiopathic short stature. In the latter cases, lengthening for short stature can provide a functional improvement for patients. Greater functional benefit may be achieved by patients who live in societies that do not compensate for the short statured person. Increased stature in affected individuals will facilitate the use of many conveniences such as public restrooms, telephones and automobiles. In addition, limb lengthening may provide a cosmetic benefit in patients with short stature.

Turner syndrome represents a spectrum of physical findings in those female patients with one X chromosome. The incidence of this condition is 1 in 5000 live births²⁰. Phenotypic characteristics include web neck often with a low hairline, coarctation of the aorta, primary amenorrhea, and short stature. A study of Turner dwarfism in ten patients documented a decrease in height of 15.5 percent when compared with normal women³. The documented shortening in stature was not proportional as two-thirds occurred in the extremities as opposed to the trunk. Shortening was also greater in the tibia as compared to the femur³.

For the last seventeen years, the Clinica Universitaria of Navarra (C.U.N.) in Pamplona, Spain has accumulated an extensive experience with limb lengthening via distraction osteogenesis. At this institution, limb lengthening has been utilized for European and South American patients with limb length discrepancies as well as for those patients with short stature. At the C.U.N. several patients have undergone tibial distraction osteogenesis for disproportionate short stature due to Turner syndrome. The purpose of this study is to review the results and complications of tibial lengthening in patients with disproportionate short stature due to Turner syndrome. Comparison of these results and complications with the available literature on tibial distraction osteogenesis for other conditions will be made.

MATERIALS AND METHODS

Retrospective analysis was performed on eight patients who underwent sixteen tibial lengthenings at the C.U.N. from November 1987 until September 1991. For each patient, preoperative data and lengthening parameters were recorded including: age at surgery, diagno-

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sis, waiting period, number of days of distraction, the amount of time needed for callus maturation, total time of fixator use and total treatment time (fixator use and subsequent immobilization). The occurrence of complications was recorded including: neurovascular injury, premature tibial or fibular consolidation, severe pin tract infection requiring intravenous antibiotics or pin removal, and nonunion of the distraction site. Additionally, the incidence of secondary operative procedures was noted including: osteoclasts for premature tibial or fibular consolidation, segment manipulation for angulation or fixator exchange, pin removal for infection, operative stabilization for fracture of the lengthened bone, operative stabilization and bone grafting for distraction site nonunion, Achilles tendon lengthening for equinus contracture, knee manipulation or arthrotomy for contracture or subluxation, and delayed corrective osteotomy for unacceptable residual malalignment. All radiographs used in this study were taken with similar radiographic technique. The films were taken from the same distance using the same X-ray machine. The initial bone length was measured from the preoperative radiograph. The final amount of length gained was measured from the first radiograph taken during the maturation period.

All radiographs were serially examined for coronal malalignment greater than ten degrees prior to fixator removal. Sagittal malalignment was not assessed due to the overlapping projection of the fixator on lateral radiographs. Radiographs were also reviewed to detect the presence of fracture or deformation of the lengthened bone after fixator removal.

For each patient, the percentage of tibia lengthened was determined by dividing the distance gained by the initial tibial length. The rate of distraction in millimeters per day was calculated by dividing the distance gained by the number of days of distraction. The healing index was calculated by dividing the total days of treatment (fixator use and subsequent immobilization) by the total distance gained in centimeters⁸. Indices were only calculated and analyzed for lengthened tibias that consolidated without internal stabilization and bone grafting.

Surgical Procedure and Lengthening Protocol

All cases were managed by members of the Department of Orthopaedics and Traumatology of C.U.N. with the same general protocol. No bifocal lengthening was performed. In each procedure, the fixator was placed on the anteromedial aspect of the tibia prior to osteotomy. In the first twelve cases, the Wagner external fixator was used, and in the later four cases the Monotube external fixator (Howmedica, Rutherford,

New Jersey) was used. In all instances, two pins were placed both proximally and distally. Percutaneous tibial osteotomy was performed as previously described⁴. All osteotomies were performed in the proximal metaphyseal region; no osteotomies were performed in the distal three-fourths of the tibia. The distal fibula was always stabilized either from the tibial side with an external fixator pin or with a screw placed from the lateral aspect of the leg. A one to three centimeter portion of fibula was routinely resected at the junction of the distal and middle third of the fibular diaphysis.

The lengthening protocol closely followed that previously described⁸. After three to five days of hospitalization, patients were discharged and encouraged to ambulate with partial weight bearing when possible. Following the waiting period, distraction was initiated at the rate of one millimeter per day in two 0.5 millimeter increments. Daily pin care was performed with soap and water. Occasionally, inflamed and painful pin sites that were resistant to local care required short courses of oral antibiotics. In some cases, pin site infections necessitated intravenous antibiotics or surgical pin removal and replacement. During the distraction period, patients returned to the clinic every two to three weeks for routine physical and radiographic examination. All patients were instructed to keep their knee extended as much as possible. In addition, patients were uniformly prescribed an ankle foot orthosis to be worn at night. These measures were recommended to decrease the risk of knee subluxation or flexion contracture and Achilles tendon contracture. No attempts were made to promote knee flexion or aggressive ankle range of motion during the distraction period.

Lengthening continued until the desired length was obtained or an additional procedure was required secondary to complications. When the final length was obtained, the fixators were locked and patients were encouraged to increase their activities and weight bearing. At this point, patients were prescribed range of motion exercises to regain knee and ankle motion that was lost during the distraction stage. To improve bone maturation, external fixators were dynamized in those segments treated with Monotube devices. Fixators were removed when definitive cortical continuity was seen in three out of four cortices on anteroposterior and lateral radiographs. The devices were routinely removed under anesthesia and twelve limbs had further immobilization in a cast or splint. The latter limbs were immobilized for an average of sixty-two days (range, thirty to eighty days). Following the lengthening protocol and complete healing of the distraction site, all patients were followed for secondary problems such as residual joint stiffness, malalignment or continued inequality.

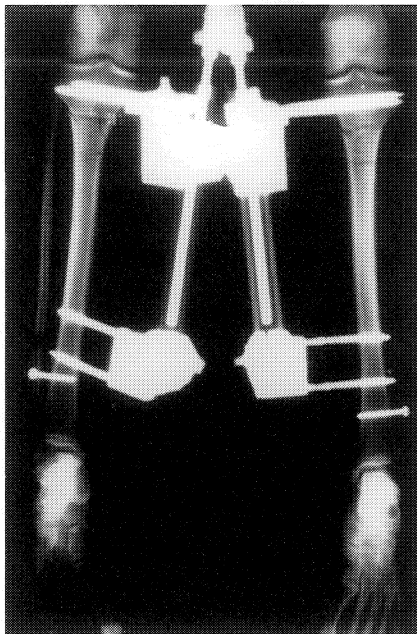


Figure 1a. Patient is an eighteen year old female with Turner dwarfism who underwent bilateral application of external fixators and proximal tibial corticotomy.

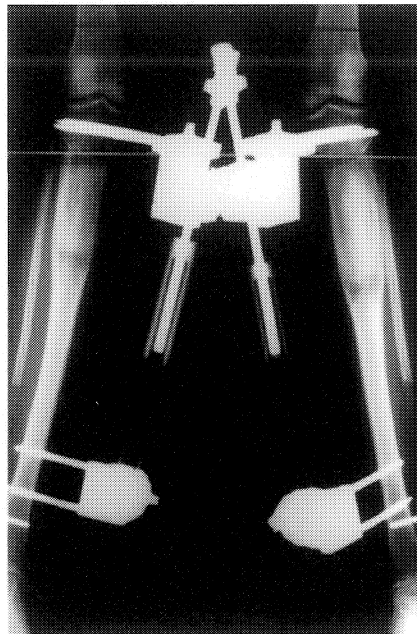


Figure 1b. After six months both the right and left tibiae had been lengthened 11.0 centimeters. At this point she underwent bilateral Achilles tendon lengthenings for ankle contractures.

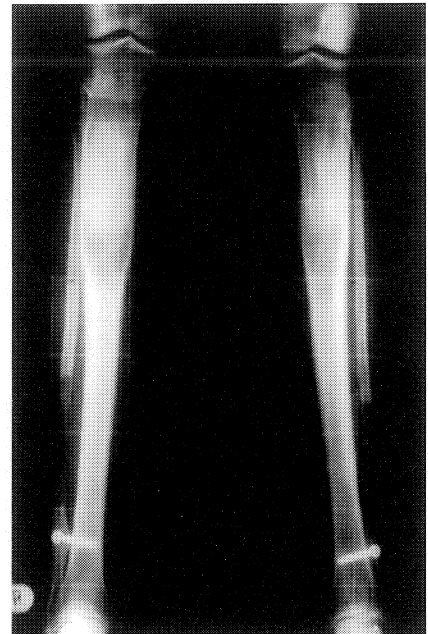


Figure 1c. At fifty-three month follow-up, continued callus maturation was noted.

A representative case example is presented in Figure 1.

RESULTS

Sixteen tibiae from eight female patients were simultaneously lengthened (symmetric lengthening). The mean age of the patients at surgery was 16.1 years (range, thirteen to twenty years). The average waiting period before onset of distraction was eleven days (range, eight to fifteen days). Distraction was performed at an average rate of 1.0 millimeter per day (range, 0.4 to 1.4 millimeters per day) for an average duration of ninety-four days (range, sixty to 146 days). The average total time of fixator use was 215 days (range, 170 to 261 days); and the mean total treatment time was 268 days (range, 227 to 300 days). All patients were followed for an average of 4.3 years (range, 2.7 to 5.8 years).

The mean distance gained was 9.2 centimeters (range, 6.5 to 12.0 centimeters) and the average percentage of length gained was 33 percent of the original tibial length (range, 19.7 to 72.7 percent). The mean healing index was thirty-one days of total treatment time per centimeter of length gained (range, twenty-three to forty days of total treatment time per centimeter of length gained).

Complications

Three segments (18.8 percent) had completion of the lengthening protocol without any complications [coronal malalignment before fixator removal; deformity of the lengthened bone without fracture after fixator removal; fracture of the lengthened bone; severe pin tract infection; nonunion of the distraction site; severe knee joint contracture or subluxation; or equinus contracture]. Two segments (12.5 percent) had one complication, eight segments (50 percent) had two complications, and three segments (18.8 percent) had three complications. The overall rate of complications was 169 percent for each tibia lengthened. The specific frequency for each complication is presented in Table 1. All cases of coronal malalignment produced prior to fixator removal were due to valgus deviation; no cases of varus malalignment were noted. No cases of fibular consolidation or tibial premature consolidation were noted. No cases of neurovascular injury were recorded in these eight patients. All fractures of the lengthened bone and the two cases of callus deformity without fracture after fixator removal were treated with cast immobilization. No significant knee joint contractures were noted after the normal rehabilitation period.

TABLE 1
Total Complications

	Frequency	Percentage
Axial Malalignment * before Fixator Removal	7	43.8
Fracture of Lengthened Bone	2	12.5
Callus Deformation without Fracture after Fixator Removal	2	12.5
Severe Pin Site Infection	7	43.8
Nonunion of Distraction Site	2	12.5
Equinus Contracture	7	43.8
Total Complications	27	169

* All cases of malalignment consisted of valgus angulation; no cases of varus angulation were noted.

Three segments (18.8 percent) had completion of the treatment protocol without any associated operative intervention (excluding fixator removal). Two segments (12.5 percent) required one additional operative procedure, eight cases (50 percent) required two additional procedures, and three segments (18.8 percent) required three additional procedures. The overall rate of associated procedures was 169 percent for each segment lengthened. The rates of each surgical intervention are presented in Table 2. All seven cases which had Achilles tendon lengthening required this during or after the distraction period; no prophylactic tendon lengthenings were performed.

DISCUSSION

In the current study, we review the results, complications and additional procedures from a consecutive series of sixteen tibias from eight patients with Turner syndrome lengthened via distraction osteogenesis. The indications for surgery in these patients reflect cultural and societal differences at odds with the peoples of North America. In European and South American countries, life for the short stature patient is more difficult as society does little to accommodate their special requirements^{15,19}. Previously, Trivella et al. reported the results of lengthenings in patients with Turner dwarfism and concluded that lengthenings in these patients corrected the disproportionate length discrepancies and improved appearance and self-esteem²². In the current study we did not attempt to document any changes in psychosocial development or health. However, we did record a predictable increase in tibial length. In this

TABLE 2
Total Associated Procedures

	Frequency	Percentage
Pin Removal for Infection	7	43.8
Manipulation for Axial Malalignment	5	31.3
Corrective Osteotomy for Residual Malalignment	6	37.5
Achilles Tendon Lengthening	7	43.8
Internal Fixation and Bone Grafting for Nonunion	2	12.5
Total Associated Procedures	27	169

series, tibias were lengthened an average of 9.2 centimeters or 33 percent of the original bone length. This average increase in length is similar to previous reviews of tibial lengthenings in patients with short stature due to achondroplasia which report increases in length of 14.5 to 58 percent^{1,8,16}. Although gains in length are impressive, these results must be balanced by the extended time required for bone consolidation (average treatment time of 268 days) and the presence of complications and associated procedures.

In the current investigation of sixteen tibial lengthenings, we noted twenty-seven complications consisting of malalignment before fixator removal; deformity of the lengthened bone without fracture after fixator removal; fracture of the lengthened bone; severe pin tract infection; nonunion of the distraction site; and equinus contracture. On average, each tibia had a complication 169 percent of the time. Previously published complications rates from papers that include tibial distraction osteogenesis report rates of 10.5 to 250 percent^{2,5-7,9,10,12-14,17,18,21,22}. Variations between the current and previous publications are most likely due to differences in the classification of complications, patient diagnosis, associated limb deformities and the distance that tibias were lengthened. Additional procedures for the treatment of complications or delayed corrective osteotomies for any residual malalignment were documented in twenty-seven instances. On average, each tibia lengthened required 1.7 additional operations to optimize the outcome of the limb.

Published papers have recorded the occurrence of ankle or knee contractures requiring operative intervention in 8.5 to 69 percent of cases^{5-7,9,10,12-14,17,18,21,22}. In this series, we document a high rate of Achilles tendon con-

tracture; approximately 44 percent of tibias lengthened required an Achilles tendon lengthening. Although the previously reported range for these problems is quite large, the current rate is somewhat higher than most reports and is likely due to the relatively longer lengthening performed. Differences in these rates may also reflect different treatment philosophies. At the C.U.N., Achilles lengthening is considered a relatively minor procedure that permits greater tibial lengthening and allows plantigrade foot positioning.

Previously, Trivella et al. reported the results of femoral and tibial lengthenings in sixteen patients with Turner dwarfism due to Turner syndrome, mixed gonadal dysgenesis and Noonan syndrome. They found longer healing times, possibly due to an intrinsic hormonal imbalance. In the current study the healing index was thirty-one days of total treatment time for each centimeter of length gained. On the surface, this value may seem equal to rates of thirty-four to forty-two days per centimeter gained in other studies of tibial distraction osteogenesis^{10,13,17,18,21}. However, we noted problems of bony development with fracture, callus deformation or nonunion at the distraction site in 37.5 percent of tibias. These findings confirm the conclusions reached by Trivella et al. who found a high complication rate in patients with Turner dwarfism undergoing distraction osteogenesis²².

In conclusion, tibial distraction osteogenesis in patients with dwarfism due to Turner syndrome can be used to improve the disproportionate limb length discrepancy. In the current report we lengthened tibias 33 percent of their original length at the expense of high complication rates and associated procedures. Further work needs to be done to reduce the rates of these problems in all patients who undergo limb lengthening. In particular, efforts should be made to determine the psychosocial and functional benefits of limb lengthening in patients with short stature. This will allow a more critical analysis of the benefits of these procedures in light of the cost associated with concurrent complications.

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DECREASE IN FIBRONECTIN OCCURS COINCIDENT WITH THE INCREASED EXPRESSION OF ITS INTEGRIN RECEPTOR $\alpha_5\beta_1$ IN STRESS-DEPRIVED LIGAMENTS

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ABSTRACT

Stress deprivation secondary to immobilization leads to atrophic changes in periarticular soft tissues. The changes in ligaments include a disorganization of collagen and cellular ultrastructure with varied biochemical alterations resulting in a functionally weaker tissue. This study tests the hypothesis that alterations in fibronectin (Fn) and the expression of its integrin receptor $\alpha_5\beta_1$ in ligament fibroblasts accompany the extracellular matrix remodeling which occurs in stress-deprived knee ligaments. The left knees of eighteen New Zealand white rabbits were surgically immobilized in acute flexion. Fibroblasts within three nine week and three twelve week stress-deprived anterior cruciate ligaments (ACLs) and medial collateral ligaments (MCLs) demonstrated markedly increased immunostaining for the β_1 and α_5 integrin subunits, as compared to fibroblasts in the contralateral unoperated control ligaments. The effects of stress deprivation on the concentration of Fn was measured by competitive ELISA on the remaining twelve rabbits. Decreases in Fn of 54.0 percent and 63.7 percent occurred in the ACL after nine and twelve weeks of stress deprivation when compared to contralateral controls. The MCL had less of a decrease, losing 37.7 percent and 41.7 percent at nine and twelve weeks, respectively. These results suggest an important role for the Fn-specific integrin receptor $\alpha_5\beta_1$ in remodeling stress-deprived periarticular ligamentous tissue, and the importance of maintaining normal

stresses on periarticular ligaments to prevent the degradation of extracellular matrix components such as Fn.

INTRODUCTION

The effects of immobilization are well known in bone and muscle and are becoming increasingly evident in soft connective tissues. Reduced stress and motion leads to atrophic changes in periarticular soft tissues including: cartilage¹³, ligaments^{40-42,52,56}, menisci^{30,31} and tendons^{24,33,48}.

The histomorphology³⁹, biomechanical properties^{7,8,56,57}, and biochemical composition^{3-5,7,9,25,26} of the anterior cruciate (ACL) and medial collateral (MCL) ligaments of the rabbit are altered in response to stress deprivation induced by immobilization. The changes are most apparent after prolonged (nine to twelve weeks) immobilization. A model in which rabbit knees are unilaterally immobilized by internal fixation has been described^{3,5,56}. With this model it has been shown that after nine weeks of immobilization the ligaments alter their cellular and extracellular matrix (ECM) organization so that fibroblasts in the ACL, normally ovoid shaped³⁴, become spindle shaped and take on an appearance similar to that of fibroblasts within the MCL³⁹. The femur-ligament-tibia complexes of rabbit knees immobilized for nine weeks have significant reductions in the tensile properties in the substance of the ligaments^{56,57}. Concomitant biochemical changes of chronically stress-deprived ACLs and MCLs occur and include reductions of glycosaminoglycans and water, as well as significant changes in the mass, rate of turnover (both synthesis and degradation), and cross linking of collagen^{3,5,7-9,26}.

Fibronectin (Fn) performs a variety of important roles in ECM remodeling; as seen in the course of tissue inflammation, malignancy, morphogenesis and wound repair. Its functional roles in these processes include serving as: a chemotactic factor, an opsonin, a scaffold for matrix formation and possibly a growth fac-

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tor^{16,47}. Its organizational properties in healing skin have been linked to wound debris clearance and remodeling of the ECM³². Elevated Fn levels have been demonstrated in persistent granulation tissue reactions²⁰, hypertrophic scars²⁹, and Dupuytren's contracture³⁷. Stress deprivation produces marked alterations in the ligament ultrastructure. While it is likely that Fn plays a role in the ECM remodeling which takes place in the contracture process subsequent to joint immobilization, the nature of its involvement and the cellular processes which mediate these alterations are poorly characterized.

Fibronectin has binding sites for collagen and proteoglycans, which are the principal structural components of ligaments¹⁰. Fn also has specific binding sites for members of the integrin family of cell surface adhesion receptors. All integrin receptors are composed of two transmembrane glycoproteins, α and β , each housing large extracellular domains which join together to form binding sites for specific ECM ligands such as the Arginine-Glycine-Aspartic acid (RGD) sequence present in Fn, and small cytoplasmic domains which may have specific binding sites for elements of the cytoskeleton²⁸. Some of the integrins identified to date that have specific binding sites for Fn include $\alpha_5\beta_1$, $\alpha_3\beta_1$, $\alpha_v\beta_1$, $\alpha_v\beta_6$ and $\alpha_{\text{Ib}}\beta_3$ ^{6,15,43,53,55}. We have previously demonstrated that certain integrin forms are present on ligament fibroblasts of the normal rabbit (and human) ACL and MCL.²² Furthermore, we have demonstrated that stress deprivation in the rabbit model is accompanied by increased expression of particular integrins containing the β_1 , α_5 and α_v subunits on the fibroblasts of the ACL and MCL with a time course similar to that previously reported¹. In particular, the $\alpha_5\beta_1$ integrin, which serves exclusively as a Fn receptor in all tissues studied to date, is markedly increased in expression after ligament stress deprivation. This outcome suggests that the $\alpha_5\beta_1$ may be playing an important role in stress-deprivation-induced ECM remodeling. These considerations led us to explore fibronectin's behavior in stress-deprived periarticular ligamentous tissue.

MATERIALS AND METHODS

Animal Model

Eighteen New Zealand white male rabbits, weighing 3.5 ± 0.2 kg, had one of their knees surgically immobilized while the contralateral knee served as a control. The surgical protocol has been previously described⁵⁶. General anesthesia was induced with an intramuscular injection of a ketamine/xylazine mixture (7-10 mg/kg xylazine, 80 mg/kg ketamine) and the rabbit's knee was immobilized in acute flexion by two 2.4 mm diameter

threaded Steinmann pins inserted through drill holes in the mid tibia. The pins were passed posterolateral to the knee joint within the skin folds of the leg. A second incision over the lateral aspect of the femur permitted the pins to be bent over the anterior surface of the proximal femur. A hex nut was then used to secure the knee in flexion. The contralateral knee served as a free moving, unoperated control. After wound closure and routine postoperative observation, the animal was returned to unrestricted cage activity with daily veterinary observation. Euthanasia was performed with an intracardiac injection of Beuthanasia-D (Schering Plough Animal Health Corporation) under appropriate sedation. Nine of the rabbits were immobilized for nine weeks and nine for twelve weeks. The animals were sacrificed at the appropriate time and the tissues harvested from both the experimental and contralateral control limbs.

Immunohistochemistry

The ACLs and MCLs from three rabbits immobilized for nine weeks and three immobilized for twelve weeks were excised, sectioned and immunostained with integrin specific monoclonal antibodies as previously described⁵⁰. Ligaments from both knees were harvested, their insertion sites removed, and midportions longitudinally bisected and mounted in OCT Compound (Miles Inc.). Tissue blocks were then snap frozen in liquid nitrogen cooled 2-methylbutane (Fisher Scientific) and stored at -70°C . Eight micron frozen sections were cut using a Cryocut 1800 cryostat (Reichert Jung, Nussloch, Germany) and mounted on Superfrost/Plus microscope slides (Fisher Scientific). Sections were then fixed in acetone (4°C) for five minutes and, after air drying, hydrated with a solution of 0.15M NaCl, 0.01M Tris, 0.1 percent bovine serum albumin (TBS BSA) for ten minutes.

Sections were then blocked by incubation with either 10 percent horse serum (for mouse monoclonal antibodies) or 10 percent rabbit serum (for rat monoclonal antibodies) in TBS BSA for ten minutes, followed by a second blocking step with 10 percent rabbit serum in TBS-BSA for fifteen more minutes. Sections were then incubated with dilutions of rabbit integrin reactive monoclonal antibodies DH12 (anti β_1 -subunit), BIIG2 (anti α_5 -subunit), or negative control anti-platelet integrin monoclonal antibody 4F10 (anti- $\alpha_{\text{Ib}}\beta_3$) at a final concentration of $2.5 \mu\text{g/ml}$. Slides were then washed three times in TBS BSA and incubated with biotinylated horse anti-mouse or rabbit anti-rat IgG (Vector Laboratories) for thirty minutes.

Sections were washed three times with TBS BSA and then incubated for thirty minutes with a 1:75 dilution of alkaline phosphatase conjugated streptavidin (Vector

Laboratories), then three additional times with TBS BSA and incubated with Vector Red[®] alkaline phosphatase substrate (Vector Laboratories). Sections were then washed with TBS BSA to stop the reaction, counterstained with Harris hematoxylin (Fisher Scientific), and mounted on slides with glass cover slips.

Mounted sections were photographed with bright-field and fluorescent techniques using an inverted transmission-light microscope (Aus JENA, Carl Zeiss Inc., Germany) equipped with a photomicrographic attachment (Canon EOS 630, Canon Inc., Japan). When examined under visible light, the Vector Red[®] precipitate is pink red; when examined under ultraviolet illumination with filters appropriate for the visualization of rhodamine, it fluoresces a brilliant red, thus facilitating photomicroscopy. Fluorescence photomicroscopy was performed utilizing a fifty watt mercury lamp light source with a uniform fixed exposure time used for all images.

Extraction of Tissue Samples

The ACLs and MCLs were excised immediately post-mortem from both hind limbs of six, nine-week immobilized and six, twelve-week immobilized rabbits, washed thoroughly with phosphate buffered saline (PBS) followed by water. The tissues were individually frozen, lyophilized and ground in a Wiley mill.

Fibronectin in the tissue specimens was extracted twice with 4 M urea, PBS, and 10⁻⁴ M phenylmethylsulfonyl fluoride (PMSF). Ten mg of ground tissue (dry weight) was suspended for twenty-four hours at 4°C in one ml of the extraction buffer. The samples were then centrifuged at 12,000 rpm for fifteen minutes and the supernatant was saved for Fn quantitation. The unsolubilized pellet was re-extracted with the same solution, and the supernatant saved. This laboratory has previously reported that greater than 90 percent of Fn was extracted from the ACLs and MCLs using two sequential extractions with 3.5 M urea¹¹. Further urea and collagenase treatment yielded less than 5 percent of the total Fn. The current data is based on a double treatment with urea only.

Purification of Plasma Fibronectin

Standard Fn was purified from the plasma of New Zealand white rabbits by affinity chromatography on gelatin-sepharose as described by Amiel et al.¹¹ based on the original work of Engvall and Ruoslahti¹⁹. Its concentration was determined by using an extinction coefficient of thirteen for a 1 percent Fn solution⁴⁶.

Characterization of Fibronectin

SDS-PAGE and Western blotting were used to characterize Fn. Tissue extracts and purified plasma Fn were subjected to SDS-PAGE on 6 percent polyacrylamide slab gels in the presence of β -mercaptoethanol. The gels were then either stained immediately with Coomassie brilliant blue or transferred onto nitrocellulose for immunostaining as described in detail by Amiel et al.¹¹.

Fibronectin Quantification

Fn content was determined using a competitive enzyme-linked immuno-sorbent assay (ELISA) modified from Rennard et al.⁴⁴, Vuento et al.⁵⁴ and Bray¹⁴, and described in detail by Amiel et al.¹¹. Briefly, peroxidase-linked goat anti-rabbit Fn (Cappel Labs) was mixed with either serially diluted Fn standards or extracts from the tissue samples. The mixtures were then added to flat bottom micro-titer plates coated with 1 μ g/ml Fn dissolved in 0.1 M sodium carbonate, pH=9.6. After the chromogen [1 mg/ml ABTS (2,2-Azinobis <3 Ethyl Benzthiazoline Sulfonic Acid), 0.003 percent H₂O₂ in 0.1 M sodium citrate, pH=4.0] was added, the plates were read at 405 nm on a flow Titertek multiscan spectrophotometer, and a standard curve was generated from the serially diluted Fn standards. Fn concentrations in the tissue extracts were then determined from the standard curve. The values of the total Fn content in the nine and twelve week groups were statistically analyzed by the paired t-test. A significant level of 0.05 was selected.

RESULTS

The gross appearance of the ACLs and MCLs differed between the control and immobilized limbs. The experimental specimens were thinner, had less bulk and lacked the normal glistening reflection. There was no apparent change in the relative cellularity (number of nuclei per microscopic field) of stress-deprived ligaments as compared to their contralateral controls.

Immunostaining of Stress-Deprived Ligaments

A dramatic increase in staining with the β_1 and α_5 integrin-specific monoclonals was observed in ACLs and MCLs that had been stress-deprived for nine and twelve weeks, respectively, while antibody staining was unchanged from normal basal levels in the contralateral control tissues. Figure 1 shows representative results obtained when monoclonal antibodies specific for the rabbit β_1 integrin subunit (DH12) and the α_5 integrin subunit (BIIG2) were used to stain nine week stress-deprived ACLs or contralateral control ACLs. Fibroblasts within the ACL demonstrated cell surface and intracellular staining for the β_1 and α_5 subunits consis-

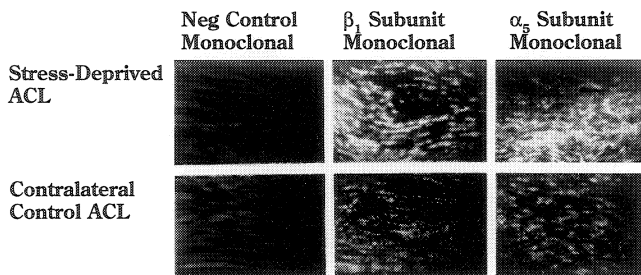


Figure 1. Concerted increased reactivity of monoclonals specific for the β_1 and α_5 integrin subunits with ACL fibroblasts after nine weeks of stress deprivation compared to contralateral controls. Negative control monoclonal 4F10 was unreactive with tissue sections. Deposition of red precipitate which also fluoresces bright red under ultraviolet illumination and visualization appropriate for rhodamine localized the specific binding of monoclonals. Black and white photomicrographs of immunostained sections photographed under fluorescent light are shown.

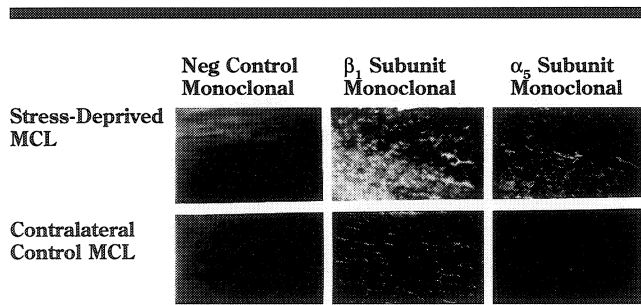


Figure 2. Increased β_1 and α_5 integrin subunit immunoreactivity in twelve week stress-deprived MCL fibroblasts compared to contralateral control MCL fibroblasts.

tent with that observed in previous studies¹. With further stress deprivation (twelve weeks) there was a decrease in the intensity of staining with both the β_1 and α_5 monoclonals when compared to that seen at nine weeks of stress deprivation, but staining intensity remained well above that of controls.

Immunostaining experiments identical to those described above were performed on stress-deprived MCLs simultaneously with the ACL stains. The β_1 and α_5 subunit-specific monoclonals reacted above control basal levels with MCL fibroblasts in ligaments that had been stress-deprived for nine weeks but were not markedly different. However, after twelve weeks of stress deprivation, staining with each of the monoclonals was markedly increased in the rabbits tested (Figure 2).

Extraction and Characterization

SDS-PAGE under reducing conditions of the Fn standard and tissue extracts is shown in Figure 3A. The purity of the standard Fn obtained from plasma is demonstrated in lane 3, where a characteristic monomeric doublet band at 220,000 daltons is observed. Though the amount of Fn extracted from the ACL and MCL is too low to be seen on SDS-PAGE (lanes 1-2), Figure 3B verifies its pres-

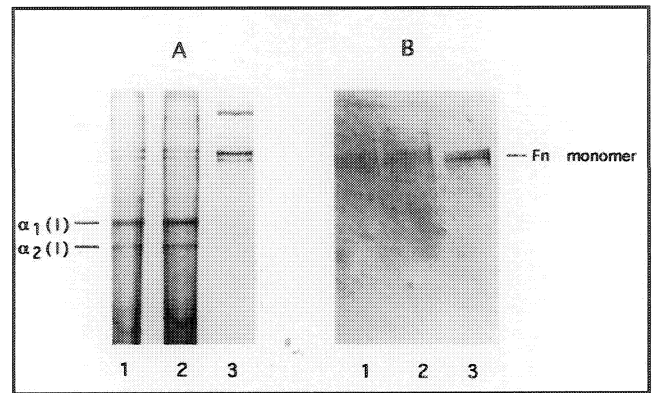


Figure 3. Qualitative characterization of 3.5 M urea extracts of ligaments. A: SDS-PAGE on 5.0 percent slab gels stained with Coomassie brilliant blue. B: Western (immuno) blot of 3.5 M urea extract. 1) Medial collateral ligament. 2) Anterior cruciate ligament. 3) Fibronectin purified from rabbit plasma.

ence by Western immunoblotting. The Fn monomer bands are clearly seen and demonstrate that the anti-Fn antibody is specific to Fn binding: i.e. no non-specific binding of other tissue proteins is observed.

Fibronectin Quantification

Figure 4 shows the results of ELISA quantitation of Fn in the control and experimental ligamentous tissue after nine weeks of immobilization. Significant decreases in the concentrations of Fn were observed in both the ACLs and MCLs when compared to levels in the contralateral control ligaments. Fn concentration in the ACLs decreased 56 percent (from 2.37 to 1.09 (g/mg; $p < .005$), while the decrease in the MCLs was 39 percent (from 0.61 to 0.38 $\mu\text{g/mg}$; $p < .005$).

Figure 5 shows the results after twelve weeks of immobilization. Again both the ACLs and MCLs demonstrated decreases in Fn concentration with immobilization. The respective reductions were 63 percent (from 2.67 to 0.97 ($\mu\text{g/mg}$; $p < .005$) in the ACLs and 40 percent (from 0.62 to 0.37 $\mu\text{g/mg}$; $p < .005$) in the MCLs.

DISCUSSION

This study demonstrates that stress deprivation of the periarticular connective tissue around the knee joint results in significant reduction in Fn levels in the ACL and MCL. This decrease is accompanied by a marked increase in the Fn-specific integrin adhesion receptor $\alpha_5\beta_1$ in ligament fibroblasts stress deprived for nine and twelve weeks.

Increased expression of the Fn integrin receptor $\alpha_5\beta_1$ was observed in stress-deprived ACLs and MCLs and occurred with a time course consistent with that of previously observed histologic, functional and biochemical changes in stress-deprived ligaments¹. Namely, these

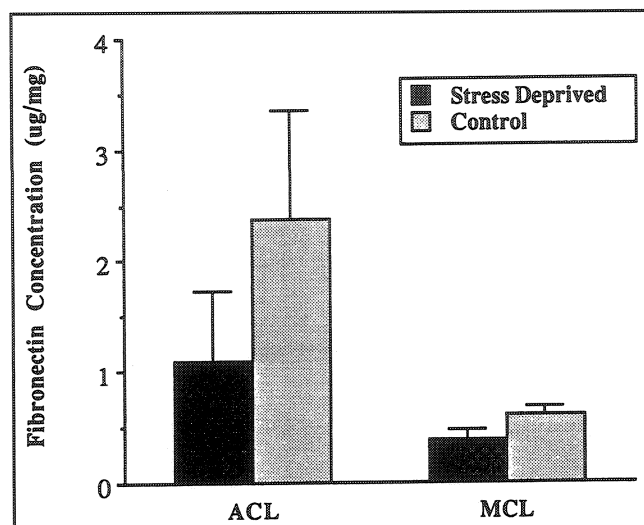


Figure 4. Mean tissue concentrations \pm standard deviation of fibronectin in stress-deprived and contralateral control ACLs and MCLs after nine weeks of immobilization. N = 6 for each tissue type.

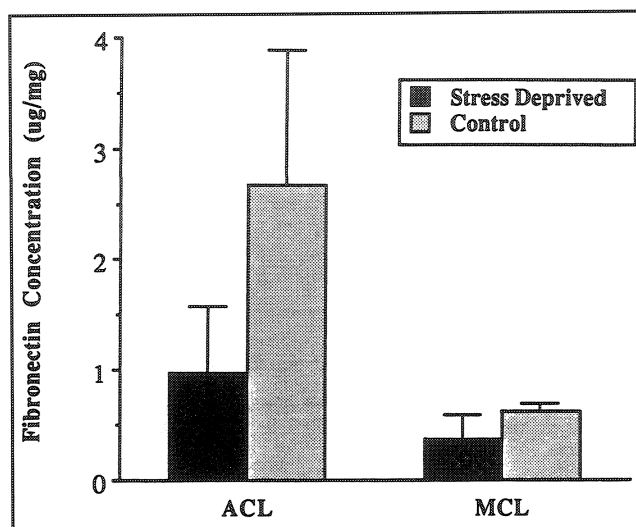


Figure 5. Mean tissue concentrations \pm standard deviation of fibronectin in stress-deprived and contralateral control ACLs and MCLs after twelve weeks of immobilization. N = 6 for each tissue type.

changes in the ligament properties were only observed with prolonged (nine to twelve week) stress deprivation. The decrease in ligament Fn observed at these time points of immobilization suggests that Fn and the Fn-specific integrin receptor $\alpha_5\beta_1$ play a role in ligament tissue remodeling through their interaction with other ECM components such as collagen and proteoglycan.

Periarticular ligament ECM components are known to undergo remodeling and attenuation with stress deprivation induced by prolonged immobilization^{12,26}. Western blot studies on protein extracted from nine and twelve week stress-deprived and contralateral control ACLs and MCLs demonstrated that increased amounts of the β_1 subunit were present in detergent extract prepared from chronically stress-deprived ligaments, as compared to extract prepared from contralateral control ligaments¹. These studies suggest that the increased immunoreactivity observed in the foregoing *in situ* studies were not due to stress deprivation induced increased accessibility of monoclonal antibody probes to cell surface integrins in tissue sections, but due to increased integrin protein expression.

The molecular mechanisms which underly the changes in fibronectin and other matrix changes are not known. Gamble et al.²¹ looked at cellular function during immobilization and found that it caused decreases in the synthetic enzymes and increases in degradative hydrolases. Indeed, the changes seen in periarticular connective tissue with immobilization have been characterized as an accelerated aging response². One of the events suggested to be important in ECM remodeling is the binding of Fn to cell surface receptors such as the $\alpha_5\beta_1$ integrin receptor^{18,36,47}. Moreover,

the various matrix components interact with one another^{45,51}, and such interaction may play some role in the assembly and remodeling of the ECM. Fn has been shown to facilitate wound healing^{17,32,38,49}, and to be required for normal collagen organization and deposition by fibroblasts *in vitro*³⁵. This suggests that changes in matrix Fn may precede and affect changes in matrix collagen deposition during ECM remodeling.

Although it is not possible from the present study to determine if the observed decreases in Fn concentrations are due to decreased synthesis or increased degradation rates, both mechanisms probably contribute. Further studies are needed to elucidate the specific biochemical pathways leading to ECM remodeling subsequent to stress deprivation, and the cause and effect relationship between changes in Fn, its cell surface receptors, and this remodeling process. Further exploration of the biosynthetic and degradative processes involving Fn and its receptors might lead to new therapeutic modalities to alter the effects of stress deprivation on periarticular connective tissue.

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PELVIC FRACTURES AND MORTALITY

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ABSTRACT

A retrospective study of all patients (N= 343) with pelvic fractures admitted to our trauma service was conducted to evaluate the impact of pelvic fractures on mortality. All patients sustained additional injuries with an average Injury Severity Score (ISS) of twenty.

Thirty-six patients died. This group had more severe pelvic fractures as graded by the Tile classification as well as a greater number and severity of associated injuries. Six patients died as a direct result of pelvic hemorrhage. In six other patients, pelvic fractures contributed to their demise. The other twenty-four patients died from brain injury, thoracic hemorrhage, or other non-pelvic causes.

Overall mortality for patients with pelvic fractures was 10.5 percent. This was a 1.4 fold increase in mortality compared to other trauma patients during the same time period without pelvic fractures. Mortality was dramatically increased in patients over sixty years of age (37 percent mortality compared to 8 percent). This greater than four-fold increase in deaths in the elderly appears to be an age related effect because the elderly patients generally had a lower ISS and less severe pelvic trauma than younger patients.

We conclude that sustaining a pelvic fracture places the patient at an increased risk of death. Pelvic fractures contributed directly to death in one-third of the mortalities, one-third died from compli-

cations associated with pelvic fractures, and one-third died from other causes.

INTRODUCTION

Trauma patients with pelvic fractures are known to be at risk of death^{17,23,27}. The most commonly cited cause of death is hemorrhage from posterior pelvic ring disruption^{2,9,14,17,21}. One of the primary goals of the management of pelvic fractures is to minimize the risk of death. However, there is wide variability in the literature on the rate, cause, and risk factors for death in patients with pelvic fractures^{5,14,17,21}. Reported death rates vary from 9 to 50 percent²⁷. Primary causes of death have included posterior pelvic venous hemorrhage, pelvic arterial hemorrhage, extra-pelvic hemorrhage, extra-pelvic trauma, multisystem organ failure (MSOF), adult respiratory distress syndrome (ARDS), and a myriad of other conditions^{1,2,9,14,17,19,21}. Risk factors for chronic pain have been identified as a function of pelvic fracture pattern²⁶, but risk factors for death in patients with pelvic fractures have received less direct attention. We, therefore, undertook a review of patients with pelvic fractures to identify the mortality rate, cause of death and risk factors for patient demise.

MATERIALS AND METHODS

We identified all patients with pelvic fractures admitted to the University of New Mexico trauma service from 1991 to 1995 utilizing our computerized database of 4511 trauma admissions. Database information, injury radiographs, type of pelvic fracture, associated injuries, hospital course, and cause of death were reviewed.

Each trauma patient was initially evaluated and resuscitated by the trauma service utilizing a standard protocol. The treatment protocol instituted for all patients included initial evaluation and volume resuscitation in the trauma room. Volume resuscitation included crystalloid and blood products as required based on hemodynamic stability. Hemodynamic instability was defined as systolic blood pressure less than ninety mm Hg. Standard AP chest, lateral C-spine and AP pelvis radiographs were obtained. Pelvic fractures were categorized according to the Tile classification (Table 1)²⁶. Patients underwent computed tomographic (CT) scan-

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Type A	Stable, minimally displaced
Type B	Rotationally unstable
B1	Open book
B2	Lateral compression (ipsilateral fracture)
B3	Lateral compression (contralateral fracture)
Type C	Rotationally and vertically unstable
C1	Unilateral
C2	Bilateral
C3	Associated acetabular fracture

ning of the head if indicated based on an abnormal Glasgow Coma Score (GCS) or reported loss of consciousness. Intraabdominal injuries were evaluated by peritoneal lavage and/or CT scan. Complex pelvic fractures as determined by the AP radiographs were further evaluated with additional radiographs and/or CT scans. Pelvic stability was clinically assessed utilizing AP and lateral compression stress testing. Patients with intraabdominal or intrathoracic hemorrhage underwent immediate exploratory laparotomy or thoracotomy.

Patients with continued hemodynamic instability, radiographically unstable pelvic fractures and no other source of hemorrhage underwent external fixation of the pelvis. If hemodynamic instability persisted, patients underwent pelvic angiography. Arterial embolization was performed when possible. Internal fixation of the pelvis was utilized to improve long term functional outcome in selected patients but was not used as a means of initial stabilization.

Other patient variables assessed were mechanism of injury, Injury Severity Score (ISS), Revised Trauma Score (RTS), time of death and cause of death. The cause of death was determined from the clinical chart and confirmed by autopsy when performed. In addition,

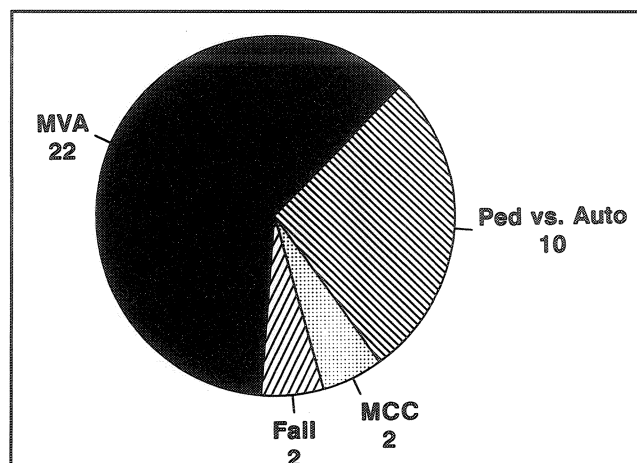


Figure 1. Number of injuries by mechanism in the thirty-six fatal pelvic fractures (MVA= motor vehicle accident; Ped=pedestrian; MCC= motorcycle collision).

patients were designated as young (less than sixty years old) or elderly (sixty years of age or older).

RESULTS

Of 4511 blunt trauma patients evaluated, 343 sustained pelvic fractures. Fifty-eight percent of patients were males, 42 percent were females and the average age was thirty-six years. The majority of these injuries occurred as a result of motor vehicle accidents (MVAs) (Figure 1). Twenty-two of the thirty-six patients who died were involved in MVAs, and motorized vehicles (MVAs, pedestrian vs. auto, and motorcycle accidents) were involved in 89 percent of all mortalities. The average GCS, ISS, and mortality as related to the type of pelvic fracture are shown in Table 2. Type A fractures were the most common (57 percent) followed by type B (27 percent) and type C fractures (16 percent).

A total of thirty-six patients (10.5 percent) with pelvic fractures died, which was 1.4 times the mortality of trauma patients without pelvic fractures (7.5 percent) during the same study period. Figure 2 demonstrates the significant increase in mortality as a function of age. This rate was essentially stable until age sixty when the mortality rate began to rise dramatically. Patient mortality as a function of pelvic fracture severity also showed

	All patients (n= 343)	Type A (n=195)	Type B (n= 92)	Type C (n= 56)	Deceased (n= 36)
GCS	13.5	13.8	13.2	12.1	8.2
ISS	20.5	17.3	22.2	26.8	43.2
Mortality(%)	10.5	4.9	10.3	30.4	100

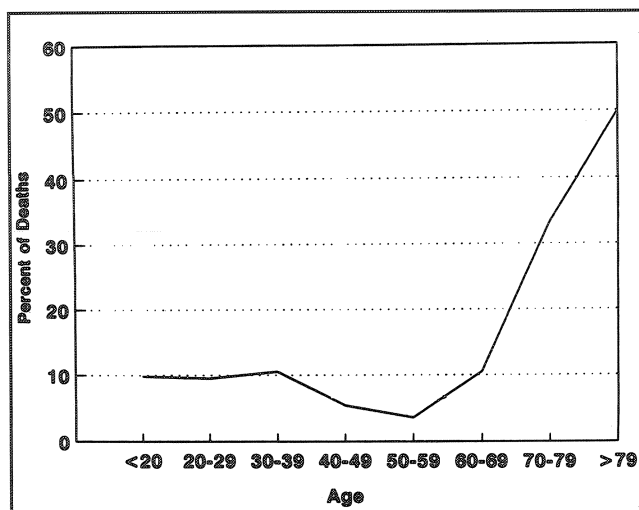


Figure 2. Percentage of patients dying as a function of age.

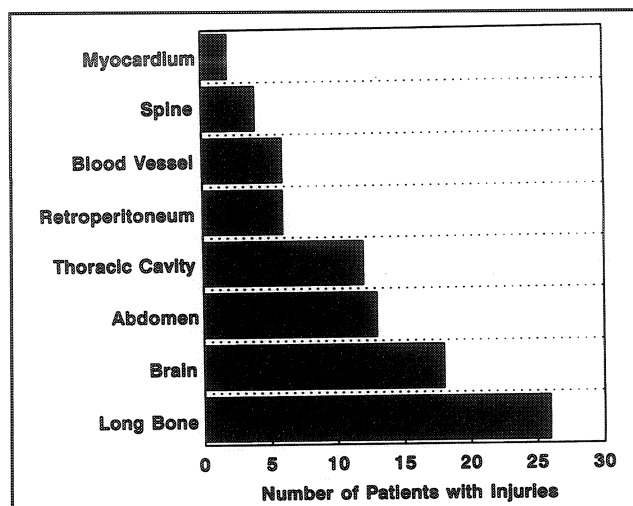


Figure 3. Incidence of common injuries associated with pelvic fractures.

a significant increase in elderly patients as depicted in Table 3. Overall mortality for the entire patient population was also noted to increase with increasing fracture severity.

Fourteen patients died of injuries separate from their pelvic fracture such as brain injury, thoracic or abdominal hemorrhage, and myocardial injuries. Non-survivors had an average of 3.7 associated injuries compared to 2.1 for survivors ($p < 0.01$). Figure 3 depicts the various types of injuries associated with pelvic fractures, long bone fractures being the most common. The frequency of other life threatening injuries was nine times greater in patients with pelvic ring disruptions than in patients without pelvic ring disruptions. Eleven patients died of multisystem organ failure where the presence of the pelvic ring disruption significantly contributed to their demise. Eleven patient deaths were directly attributed to the pelvic ring disruption with either acute hemorrhage or early progressive sepsis from open pelvic wounds.

Twenty-three of the thirty-six patients (64 percent)

died within the first twenty-four hours of admission, and thirty-one of the thirty-six patients (86 percent) died within the first seven days of admission. Cause of death was divided into five main categories: pelvic hemorrhage, non-pelvic injury, brain injury, multisystem organ failure(MSOF) and adult respiratory distress syndrome(ARDS). Time of death was divided into two categories: acute and late (greater than seventy-two hours after injury). Table 4 illustrates that the highest number of deaths occurred as a result of brain injury. Pelvic hemorrhage and non-pelvic injury resulted in approximately the same number of deaths. Late causes of patient death were attributed in part to the pelvic fracture and manifested as MSOF and ARDS between one and three weeks. Seventeen percent of deaths were the result of pelvic hemorrhage and these patients all died within thirty-six hours despite aggressive resuscitation and external fixation. It should be noted that all pelvic fractures that underwent external fixation were classified as either type B or C injuries. A total of sixty-four patients underwent external fixation at the time of

	Mortality (%)			Overall mortality (%)
	Type A	Type B	Type C	
Young (n= 316) (Age < 60)	3	7	26	8
Elderly (n= 27) (Age ≥ 60)	25	37	100	37

TABLE 4. ACUTE AND LATE CAUSES OF DEATH					
Number of Deaths					
	Pelvic hemorrhage	Brain injury	Non-pelvic injury	MSOF	ARDS
Acute (≤ 72 hours)	6	10	5	-	-
Late (> 72 hours)	0	4	1	4	3

injury for radiographically unstable pelvic fractures. Nine of these patients died whereas fifty-five patients survived. Therefore, approximately 14 percent of all patients undergoing external fixation died.

DISCUSSION

The human pelvis is an extremely stable structure requiring a great amount of force to cause disruption. It is also associated with an extensive vascular network. These two factors contribute to the higher mortality associated with pelvic ring disruptions compared to other bony injuries^{2,17,23}. Our experience with pelvic fractures and mortality is similar to other reports in the literature²³. Additionally, we saw a 1.4 fold increase in mortality in patients with pelvic fractures compared to those without pelvic fractures. While not statistically significant, it does suggest that pelvic fractures do contribute to an increase in mortality. Patients with pelvic fractures also had a higher number of associated injuries compared to patients without pelvic fractures. This was statistically significant and is indicative of the significant force required to produce pelvic ring disruption. Pelvic fractures are not only a cause of death, but serve as a marker for other life-threatening injuries.

The severity of the pelvic fracture was associated with the mortality rate. Overall, mortality increased approximately six fold as fracture severity increased (i.e. between type A and type C injuries). Additionally, all patients that died in the acute period sustained type B or C pelvic injuries.

Age was also a prognostic factor regarding survival. The elderly patients had a higher GCS, a lower ISS, and a significantly higher mortality compared to younger individuals. Fox⁵ and Gruen⁸ reported an increased mortality in elderly patients as well. This is attributable to the elderly patients underlying co-morbid conditions.

The role of treatment with regard to its effect on mortality was not directly evaluated in this study. However, several observations can be made. Our study indicated that approximately one-third of deaths in patients with pelvic fractures are directly attributable to the pelvic

fracture, one-third are related to the complications associated with the pelvic fracture and one-third are caused by other injuries. Many authors advocate the use of external fixation for management of pelvic fractures in the trauma patient^{1,10,26,28}. External fixation was selected for many of the most severe injuries and the mortality rate in patients treated with external fixation was 14 percent. This is similar to other reports in the literature^{1,8}. Treatment was not controlled in this study and definitive conclusions regarding surgical indications are beyond the scope of this report. We do feel that surgical stabilization, and external fixation in particular, remains a valuable option for the management of patients with pelvic ring disruptions and helps to minimize their risk of death.

The causes of death in this group of patients can be categorized into five main areas: pelvic hemorrhage, non-pelvic injury, brain injury, multisystem organ failure (MSOF) and adult respiratory distress syndrome (ARDS). Brain injury was the leading cause of death in this series. Acute deaths occurred as a result of pelvic hemorrhage and non-pelvic injuries with equal frequency. It is this population of patients that requires aggressive management to control hemorrhage in order to improve outcomes. Fluid resuscitation in conjunction with external fixation, limited internal fixation and/or angiography when indicated may lead to a decrease in mortality. Additionally, systemic complications that are associated with pelvic fractures, such as MSOF and ARDS, should be anticipated and managed appropriately when identified.

In summary, patients with pelvic ring disruptions are at risk of death (10.5 percent). One-third of these deaths were directly related to the pelvic ring disruption with hemorrhage being a significant factor. In another third the pelvic ring injury contributed to the patient's demise. The remaining third were attributable to other specific injuries where pelvic ring disruption served as a marker for other life threatening injuries commonly associated with high energy trauma. Factors such as greater fracture severity and increased patient age were

associated with higher mortality rates.

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NEW ADVANCES IN THE MOLECULAR BIOLOGY OF MUSCULOSKELETAL NEOPLASMS

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During the past two decades, there has been a revolution in identifying the causes of cancer, with most of this work focused at the molecular level. There is now good reason to believe that cancer is a disease of genes, and most, if not all, causes of cancer act by damaging genes directly or indirectly. The identification and characterization of many of these genes is one of the triumphs of molecular biology. This review is meant to provide an introduction to this field and highlight recent findings relative to the molecular biology of musculoskeletal neoplasms. We believe that this new information should be included in daily orthopaedic clinical practice since it will undoubtedly change the management of patients with these types of neoplasms.

BIOLOGY OF NEOPLASTIC TRANSFORMATION

The human body can be viewed as a dynamic, complex structure integrated by assemblies of individual cells. In each tissue, cells have a well defined shape and fit neatly within the ordered array of cells surrounding them. Such collaboration ensures that each tissue maintains the architecture and function appropriate for the body's needs. Coordination of proliferation and differentiation is, therefore, an essential feature for successful development, growth, tissue renewal, and tissue repair. Unfortunately, the process of DNA replication carries the constant hazard of genetic mutations; random changes that can impair the regulatory circuits of a cell^{10,33-35}. If a single mutation occurs, the newly damaged cell, which may look normal, may undergo unscheduled cell division. Eventually, an accumulation of genetic damage can cause the cell to become unresponsive to external messages and display the signs of neoplasia.

Cancer is characterized by unlimited proliferation of neoplastic cells that fail to respond to physiological control mechanisms, thus destroying surrounding normal tissues, spreading to distant organs, and ultimately kill-

ing the organism. Because all cancers share these characteristics, it has been suspected that there are basic mechanisms applicable to and responsible for the initiation and development of all cancer types.

Modern concepts of neoplasia rely on the evidence that a tumor arises from the clonal progeny of a single transformed cell^{42,56}. This neoplastic cell has multiple sites of DNA damage, and these genetic changes confer selective advantages on the cell clone by disrupting the regulation of the cell proliferation and differentiation processes. Serial mutations are thought to induce the progression from normal unaffected tissue to hyperplasia with a high incidence of proliferative cells; to induction of tumor angiogenesis and tumor dysplasia; to clonally heterogeneous neoplasia; and finally to local invasion and metastasis (Figure 1).

Although a complete understanding of the mechanisms of neoplastic transformation has yet to be developed, the causes are now better understood. Key molecules undergoing alterations during neoplastic transformation are cellular oncogenes, tumor suppressor genes, DNA repair genes, multi-drug resistant

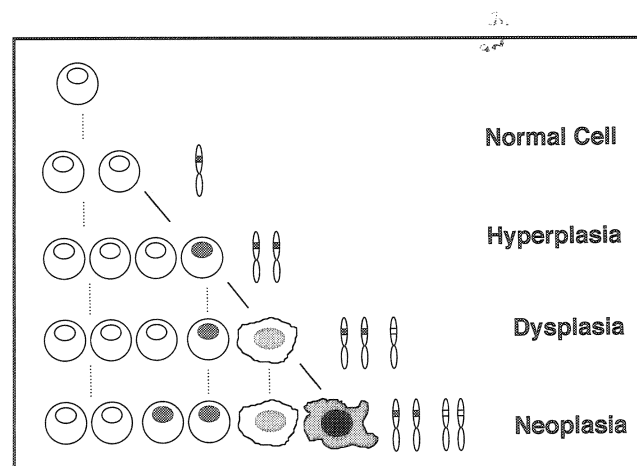


Figure 1. The neoplastic transformation of a cancer cell from a normal one is thought to occur through a process known as clonal evolution. First, a cell inherits or acquires a cancer promoting mutation and passes the defect to all future generations. Cells seem normal but are predisposed to proliferate excessively (hyperplasia). At some point, daughter cells acquire new mutations; they proliferate more rapidly and also undergo structural changes (dysplasia). Finally, some cells accumulate enough mutations to grow uncontrollably, invading surrounding tissue and metastasizing (neoplasia).

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TABLE I
Cytogenetic abnormalities associated with musculoskeletal neoplasms

Histologic Type	Abnormality
Ewing's sarcoma/ PNET	t(11;22) (q24; q12)
Synovial sarcoma	t(X;18) (p11.2; q11.2)
Alveolar rhabdomyosarcoma	t(2;13) (q35-37; q14)
Myxoid liposarcoma	t(12;16) (q13-14; p11)
Neuroblastoma	Loss heterozygosity 1p36.1-1p36.3
Osteosarcoma	Structural rearrangement of 1,2,6,12,17
Chondrosarcoma	Rearrangement of 1p

genes, and multi-drug resistant associated protein genes.

ONCOGENES

The initial search for altered genetic information in neoplasia was through examination of the chromosomes. Chromosomal changes are gross alterations of the genetic information. In some neoplasms there are recognizable chromosomal alterations characteristic of a specific tumor type. For example, Ewing's sarcomas have a translocation between chromosomes 11 and 22. Osteosarcoma, on the other hand, has multiple chromosomal abnormalities but none are characteristic^{13,17,19,22} (Table I). While some of the observed karyotypic changes may be the result of early events in neoplastic transformation, most reflect an accumulation of errors acquired during tumor progression.

In 1970, researchers showed that Rous Sarcoma Virus (RSV), a cause of sarcomas in chickens, can transform a normal cell into a cancerous one. The key advance was the recognition that this supposed viral "oncogene" is present in normal cells, indicating that abnormalities of normal cellular genes may underlie neoplastic transformation^{6,55}. A positive link between genetic mutation and human neoplasia was established when the EJ human bladder carcinoma cell line was shown to contain a point mutation at codon 12 (glycine to valine) in the c-Ha-ras gene^{44,52,53}. Subsequently, about seventy proto-oncogenes have been discovered; each of these can be altered into an oncogene that plays a role in some form of neoplasia^{3-5,8,13,15,17,25,26,39,45,50} (Table II).

The term oncogene refers to genes that encode proteins that contribute to the neoplastic phenotype. As mentioned above, an oncogene evolves from a native gene (proto-oncogene) in the cellular genome that, via mutation, becomes activated. The mutant alleles of proto-oncogenes are defined as dominant because they transform cells despite continued expression of their normal counterparts. There are many conceivable modes of genetic disruption that could convert a proto-oncogene into an oncogene. The gene may be altered

TABLE II
Genetic alterations in bone and soft tissue tumors

Abnormality	Tumor type	
TUMOR SUPPRESSOR GENES		
Nuclear proteins	p53	MFH, liposarcoma, osteosarcoma, chondrosarcoma, fibrosarcoma, Ewing's sarcoma, angiosarcoma, rhabdomyosarcoma
	Rb	Osteosarcoma, liposarcoma, MFH, rhabdomyosarcoma, liposarcoma, leiomyosarcoma, hemangiopericytoma
GTP-ase	NF1	Neurofibromatosis
Other proteins	APS	Desmoid tumor
ONCOGENES		
Protein kinases	MET	Osteosarcoma
Membrane G proteins	H-ras	MFH, embryonal rhabdomyosarcoma
	Ki-ras	Angiosarcoma
Transcription factors	N-myc	Neuroblastoma
	c-myc	Chondrosarcoma, MFH, liposarcoma, fibrosarcoma, osteosarcoma, synovial sarcoma
	c-fos	Fibrous dysplasia
Cyclin-dependent kinase	CDK4	Rhabdomyosarcoma, osteosarcoma
Fusion proteins	TLSCHOP	Myxoid liposarcoma
	TUSCHOP	Myxoid liposarcoma
	EWS-FLI-1	Ewing's sarcoma
	PAX3-FKHR	Alveolar rhabdomyosarcoma
Other proteins	MDM2	Osteosarcoma
	gli	Liposarcoma, osteosarcoma, rhabdomyosarcoma
	CHOP	osteosarcoma, liposarcoma, rhabdomyosarcoma, hemangiopericytoma
	SAS	Hemangiopericytoma, osteosarcoma, rhabdomyosarcoma, MFH, liposarcoma

by point mutations, overexpression or amplification, chromosomal translocations, or insertion of a mobile element resulting in gene fusions. Mutations alter the physiologic roles of these genes, causing them to send continuous signals that result in overexpression, aberrant expression, or inappropriate expression of other growth-related genes. The end result is neoplastic trans-

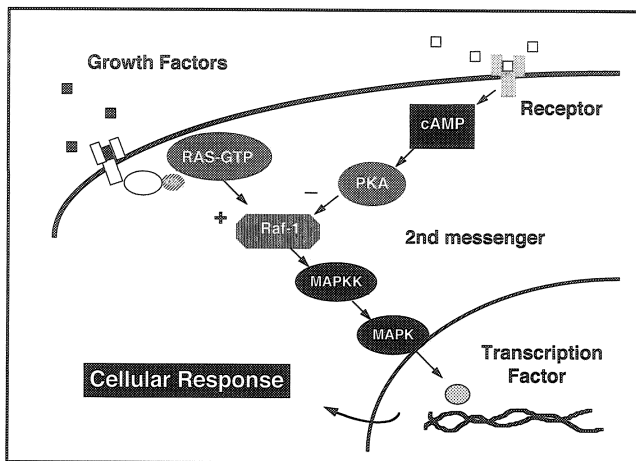


Figure 2. Signal transduction pathways leading to positive or negative regulation of cell proliferation. Signal transduction begins with the binding of a growth factor to its membrane receptor. This event initiates a cascade of intracellular reactions known as second messenger pathways which ultimately activate one or several genes necessary for the appropriate response. Many cancer causing genes are abnormal versions of genes that code for proteins in stimulatory or inhibitory pathways.

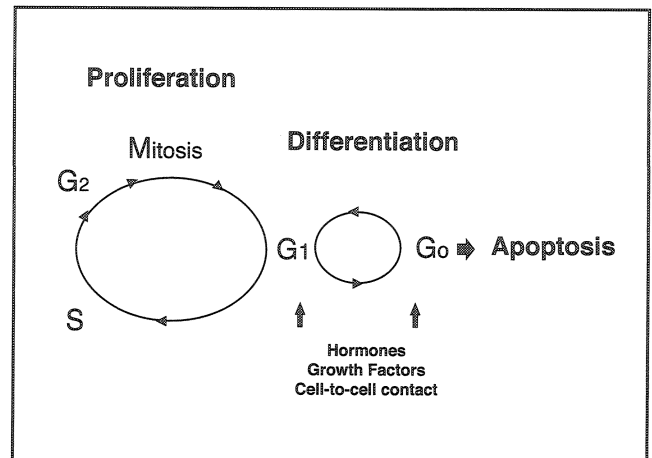


Figure 3. The cell cycle and its interconnection with the differentiation and apoptosis pathways. In general, cells exit the mitotic cycle from G1 in response to extracellular signals and differentiate after passing through an intermediate G0 stage. Many cells can be activated to reenter the cycle during G0. When properly stimulated, cells will undergo apoptosis (programmed cell death). Arrowheads represent checkpoints that regulate the entrance into the next phase of the cycle. G1: preparatory stage; S: DNA synthesis; G2: preparation for mitosis; M: Mitosis; G0: quiescent state.

formation. Together, these quantitative and qualitative changes provide a powerful growth stimulus to the cell.

The molecular functions of these oncogenes are beginning to be elucidated, and an increasing number of them belong to the signal transduction pathway^{28,50}. This pathway is the complex functional entity of molecular events from the first contact of the cell with a signal (growth factor, adhesion molecules, etc.), through the subsequent intracellular second messenger-mediated reactions, resulting ultimately in the activation of one or several genes which encode the product(s) for a cellular response (Figure 2). This system allows the orderly and specific transmission of growth regulatory messages from outside the cell to the machinery controlling replication inside the cell's nucleus, and enables the cell to adapt to changes in the tissue, organ function, or external challenges^{28,43}.

Oncogenes are deregulated intermediates in these signal transduction pathways, and the mechanisms of neoplastic transformation are related to the normal function of these pathway intermediates. Pathway intermediates that can become oncogenic include growth factors that act on the cell surface; growth factor receptors and other G-proteins that convey signals from the receptor to the cytoplasm; and protein kinases, cytoplasmic regulators, and nuclear regulatory proteins that orchestrate differential gene expression. The continued expression of these protein products may result in chronic cell cycle activity, which also may decrease the fidelity of the negative check points leading to other genetic mutations and cell transformation^{1,9,14,25,50}.

TUMOR SUPPRESSOR GENES

Normal cellular homeostasis is regulated through the interplay of two opposing biofeedback systems, one that uses growth promoting genes and another that counterbalances with a series of growth inhibiting genes. For most solid tumors, which represent more than 85 percent of human tumors, it now appears that complete loss of the normal function of a series of cell cycle regulatory genes is the common mechanism of tumorigenesis and tumor progression. These genes are, in a sense, the "opposite" of oncogenes because their normal role is to inhibit, not stimulate, cell growth. Tumor suppressor genes act to slow the progress of the cell cycle, ensuring fidelity of each cell division (Figure 3). If the expression of these genes is inhibited, the cell loses negative growth control and endless cell cycling occurs^{11,12,20,49,50}.

The existence of tumor suppressor genes was suggested by the suppression of the malignant phenotype in hybrid experiments of normal and tumor cells, and by the consideration of the familial rhabdomyosarcoma by Knudson²⁷. Subsequently, the first tumor suppressor gene identified in man was *Rb* on chromosome 13q14. Several studies have confirmed that both copies of the gene are mutated or deleted in those cases of osteosarcoma and soft tissue sarcoma occurring as part of the syndrome of familial retinoblastoma³¹. Other tumor suppressor genes include p53, neurofibromatosis type 1 (NF1), Wilms' tumor gene, APC gene, and DCC gene^{2,16,23,24,30,32,37,38,40,46,48,50,54,57} (Table II).

The precise biochemical mechanism by which any of the products of tumor suppressor genes regulate cell behavior is not completely understood. Major insights into the mechanisms of action of Rb and p53, both of which are nuclear proteins, support a role in the regulation of genes involved in proliferation or differentiation, or in controlling the biochemical mechanisms regulating the initiation of DNA synthesis. For example, several findings suggest that p53 controls a cell cycle checkpoint responsible for maintaining the integrity of the genome. Wild-type p53 mediates arrest of the cell cycle in the G1 phase after sublethal DNA damage (G1 phase of the cell cycle is the quiescent period between mitosis and DNA synthesis). In other cases, p53 expression results in apoptosis (programmed cell death)^{31,50}. If a mutation inactivates this gene, cells with damaged DNA can continue to divide and, in doing so, accumulate other gene mutations that can lead to neoplasia. Furthermore, patients with malfunctioning p53 may be less susceptible to chemotherapy³⁶.

NF1 encodes a GTPase-activating protein which interacts with other proteins through the signal transduction pathway. Loss of NF1 activity favors the active GTP-bound form of Ras and results in removing the negative regulations placed on Ras by GAP-like proteins. DCC tumor suppressor gene encodes a cell surface molecule related to cell adhesion. It is unlikely that DCC mechanically connects the cell to the extracellular matrix, but DCC may be a signal transduction receptor whose loss confers a growth advantage to the neoplastic cell⁵⁰.

As more is learned about cell cycle regulation, it is clear that its control is a complex process with numerous interrelated controlling mechanisms, and many additional genes, yet to be discovered, can lead to neoplastic transformation.

DNA REPAIR GENES

The genetic material is in constant danger of malfunctioning, both because errors introduced during replication and damage caused by environmental agents such as ultraviolet radiation or chemicals. Something on the order of 10^{16} cell divisions take place in the human body in the course of a lifetime. The estimated rate of spontaneous mutations observed in normal human cells is about 1.4×10^{-10} mutations per gene per cell division. Thus, in a life time, every single gene is likely to have undergone mutation on about 10^{10} occasions in any individual^{10,33-35}. If such damage were allowed to persist, cells would cease to work properly, mutations would accumulate, and the chances of neoplasia development would climb.

DNA repair systems protect and maintain the information in the genetic code. There are two major types

of DNA repair pathways, each of which has its own distinct function⁴⁷. Mismatch repair corrects errors such as simple base mismatches while DNA is being copied. The second mechanism, nucleotide excision repair, replaces DNA that was damaged spontaneously or by chemicals and radiation. Strong evidence suggests that defective mismatch repair underlies several types of cancer (i.e. colon cancer and xeroderma pigmentosa), with the resultant genetic destabilization leading to other genetic alterations such as the activation of oncogenes or the inactivation of tumor suppressor genes^{18,41}.

MULTI-DRUG RESISTANT GENES

It is important to recognize that cells find ways to survive the adverse environment created by systemic treatments such as chemotherapy. Studies of osteosarcoma cell lines have shown that one of these mechanisms involves the expression of a cell membrane pump, P-glycoprotein, that is encoded by the MDR-1 gene. This pump actively excretes certain classes of drugs such as adriamycin from the cell, thus preventing the accumulation of the drug in the cytoplasm. The presence of MDR-1 gene expression in human osteosarcoma cells has been associated with a poor prognosis^{7,21}. It is likely that other mechanisms of drug resistance may be present in tumor cells. Identification of these genes will help identify patients at risk for unresponsiveness to antineoplastic drugs.

CLINICAL RELEVANCE

Advances in clinical and surgical oncology in the last two decades have resulted in an improvement in the clinical outcome of patients with musculoskeletal neoplasms. However, the proper diagnosis, management, and overall control of musculoskeletal neoplasms are major challenges in clinical practice. Because the type of treatment depends on the morphological evaluation and staging of the tumor, the diagnosis carries significant consequences. Current tumor classifications are problematic: a single conventional category represents a heterogeneous collection of disorders, each characterized by its own array of lesions. Furthermore, the biologic behavior of a given tumor cannot be fully predicted by its microscopic appearance, and great variations are often observed. If one cannot identify a disease correctly and distinguish it from others, one cannot discover its causes, select the appropriate treatment for a given patient, or predict its prognosis. Molecular biology is beginning to provide tools to identify precisely which genes are amplified, deleted, or mutated in the tumor cells of a given patient. The analysis of tumors using these new markers can provide not only a spe-

cific diagnosis, but a method of following the progression and remission of the disease; detecting presymptomatic lesions in patients who are at risk of inherited cancer, and assessing the biologic behavior of a given tumor in a given patient. Finally, it may also lead to the introduction of new therapeutic modalities targeted specifically to correct the biochemical derangement created by the mutated genes. Such information may prove to be as important for the diagnosis, treatment and prevention of cancer as is the identification of the pathological microorganism in a patient with an infectious disease.

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LONG TERM FOLLOW-UP OF MEDIAL COLUMN FUSION AND TIBIALIS ANTERIOR TRANSPOSITION FOR ADOLESCENT FLATFOOT DEFORMITY

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ABSTRACT

We report the results of three patients (four feet) who had surgical correction of adolescent flatfeet performed over fifty years ago. The surgery involved medial column stabilization with fusion procedures and tibialis anterior transposition into the navicular (Young's tenosuspension procedure). In this small sample, we found a high rate of painful arthrosis that developed over time in the contiguous joints of the foot.

INTRODUCTION

The majority of patients with flexible pes planus are asymptomatic. For the symptomatic, flexible flat foot, numerous authors have described various nonsurgical approaches^{1,2,5,12,24}. In the majority of patients, symptoms are alleviated with nonoperative measures. However, there are occasional patients with persistent symptoms whom are considered for surgical treatment. A wide variety of surgical procedures have been described for the correction of these flatfoot deformities^{1,6,10,11,12,15,16,21,22}.

In 1923 Lowman described his procedure involving talonavicular arthrodesis with transposition and tightening of the tibialis anterior on the navicular with or without tendo Achilles lengthening¹⁸. Four years later, Miller described a technique involving naviculo-medial cuneiform-first metatarsal arthrodesis with distal advancement of an osteoperiosteal flap based on the tibialis posterior tendon at the navicular²⁰. He reported good results in sixteen patients with an average follow-up of 2.5 years with an average age at operation of twelve years. In 1929, Kidner described his procedure for removing the accessory navicular when present, freeing the tibialis posterior, and transplanting the tendon inferiorly on the navicular to pull directly upward, theoretically maintaining the medial longitudinal arch¹⁷. Hoke, in 1931, reported his success in the treatment of the

flexible flatfoot with medial and middle cuneiform-navicular arthrodesis with tendo Achilles lengthening in four patients¹³. Six years later, Butte described his study of 138 patients with flatfoot who underwent naviculocuneiform arthrodesis³. He reported excellent results in thirty feet, good in forty, fair in thirty-four feet, and poor in thirty-four. The length of follow-up was as long as nine years, with the majority between one and five years, and an average age at operation of fourteen years.

In 1939, Young published his results with transposition of the tibialis anterior insertion into the navicular with tendo Achilles lengthening in order to prevent depression of the arch and abduction of the forefoot²⁵. He reported symptomatic relief, correction of the depressed arch, and eversion and abduction of the forefoot in seven patients with pes planus.

From our review of his surgical record cards, it appears that in that same year (1939) Dr. Arthur Steindler incorporated Young's tenosuspension operation with medial column fusion stabilization for the treatment of flatfeet. In this report we describe the long term results of those surgeries.

METHODS

Fifteen surgical procedures were performed by Dr. Steindler on thirteen patients with flatfoot deformities between 1939 and 1943. These procedures included twelve naviculocuneiform arthrodeses with tibialis anterior transposition into the navicular, and three talonavicular arthrodesis with tibialis anterior transposition into the navicular.

In 1995, we attempted to locate these patients for follow-up. We identified three living patients (four cases). Seven patients were deceased and three others were lost to follow-up. Of the three living patients, one patient with bilateral surgery returned for a complete examination and radiographic assessment, and the other two completed a telephone interview and had radiographs taken of both feet. In this study we report the results of these four cases in three patients with greater than fifty years of follow-up.

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CASE REPORTS

Patient 1

B.W. first presented to the University of Iowa Hospitals and Clinics (UIHC) at the age of fourteen with complaints of right foot instability and fatigue. Three years earlier she had jumped over some flowers and severely injured her right foot. Subsequently, she developed a progressive pronation deformity of the midfoot. There was no complaint of pain.

Physical examination revealed bilaterally pronated feet with her right more affected than her left. She was noted to have a tight heelcord on the right. The right peronei were recorded as slightly contracted. Plain radiographs of her right foot revealed an old, healed fracture of the sustentaculum tali. She was diagnosed with a traumatic flatfoot deformity and was treated with a brace. One year later, she underwent naviculocuneiform fusion, tendo Achilles lengthening, and transposition of the tibialis anterior into the navicular of her right foot.

Thirty-five years later, the patient underwent a subtalar fusion for degenerative arthritis. Fifty-five years after the initial operation, we contacted the patient and obtained radiographs (Figure 1). At that time she stated that use of the injured foot caused intermittent pain limiting her daily activities, but also noted that for many years she enjoyed relatively painless function of the foot.



Figure 1A.



Figure 1B.

Figure 1. Standing (A) anteroposterior and (B) lateral radiographs of a patient fifty-five years post-surgery on the right foot. A subtalar arthrodesis was performed for painful arthrosis thirty-five years after the initial procedure. Diffuse degenerative changes in the transverse tarsal and midfoot joints correlate with residual symptoms. Comparative radiographs of the nonoperated foot (C,D) reveal no arthritic changes.



Figure 1C.

Patient 2

J.M. first presented to the UIHC at the age of eleven with complaints of a weakness of his left ankle. His parents reported an incident at age two when he sustained an infra-medial malleolar laceration with resulting hindfoot deformity. Since that time, he had progressive ankle weakness with a noticeable talar head prominence.

Physical examination of the left lower extremity revealed atrophy of the calf musculature with a valgus hindfoot. No palpable activity of the tibialis posterior muscle or tendon was observed. The talus was prominent on the medial aspect of the ankle, and the patient had a tight heelcord.

J.M. subsequently underwent talonavicular fusion and tibialis anterior transposition to the navicular. His postoperative course was complicated by a wound infection and a failure of the talonavicular joint to fuse. Five months following surgery, there was no radiographic evidence of talonavicular fusion, but the patient was ambulating without difficulties and was free of complaints. There was some evidence of hindfoot valgus. At a period of twelve months following surgery the tibialis anterior was noted to actively fire, and there was residual mild hindfoot valgus and forefoot pronation. He was given solid leather insoles to wear and was not contacted for over fifty-six years.

In final follow-up, he had no complaints regarding his foot. He claimed that although he was still flatfooted on both sides, it did not prevent him from walking several miles a day. Plain radiographs obtained of both feet (Figure 2) revealed evidence of the previously attempted talonavicular fusion and mild degenerative changes in the hindfoot.



Figure 1D.

Figure 2. Standing (A) anteroposterior radiograph of a patient fifty-six years after failed fusion of the talonavicular joint and Young's tenosuspension of the left foot. (B) The lateral radiograph of the left foot shows evidence of mild degenerative changes as compared to the nonoperated right foot (C).



Figure 2A.

Patient 3

S.R. first presented to our clinic at the age of eleven with a five to six year history of bilateral, painful, weak feet that tended to go into valgus. She complained of pain in the arch of her feet and fatigue in her calf muscles after any significant period of ambulation. On physical exam, both of her feet were noted to be in valgus with prominent tali medially. She was tender to palpation under the talonavicular joint. Her heel cords were not noticed to be tight. She was given inserts (bilateral inner wedges and anterior heels) to wear in her shoes. She was also given exercises to perform.

The patient's symptoms resolved soon thereafter. She returned six years later complaining of pain and tenderness in both of her feet. Physical examination revealed bilateral, long, narrow feet with tender prominences over the navicular bones. She was also found to be tender over the spring ligaments bilaterally. Her feet were in equinus and valgus. She was again treated with exercise and shoe corrections and started on contrast baths.

Her symptoms persisted and progressed over the following year. At age nineteen, she underwent bilateral accessory navicular excisions, naviculocuneiform fusions and transplantation of the tibialis anterior tendons into the naviculars. Three months after her initial surgery she had bilateral tendo Achilles lengthenings performed. She did well and by age twenty-one stated she was walking as much as she wanted and could play tennis without pain. Her arches were maintained and no tenderness was elicited on examination of both feet. She was able to wear shoes without orthotic support.

S.R. returned fifty-three years after her bilateral naviculocuneiform fusions and tibialis anterior tenosus-



Figure 2B.



Figure 2C.

Figure 3. Standing (A) anteroposterior and (B) lateral radiographs of both feet of a patient fifty-three years after surgery. The patient had diffuse pain throughout both feet around the midfoot and hindfoot regions. The degenerative changes are more striking in the more symptomatic right foot.



Figure 3A.

pensions. She was referred by her primary care physician, who had been treating her bilateral foot pain for eight years with anti-inflammatory medications and shoe inserts. At age seventy-three, she complained of pain around the dorsal and lateral aspects of the hindfoot with ambulation and occasional pain at rest. She had retired from a full career as a nurse and had difficulty remaining active due to difficulties with her feet.

Examination of the right foot revealed tenderness in the subtalar joint region. There was essentially no subtalar motion. She also had some tenderness and pain with motion in the talonavicular and calcaneocuboid joints. On examination of the left foot, she had more subtalar motion (approximately ten degrees) and less pain than the right foot. Her tenderness on the left was mostly in the mid-foot region. Her neurovascular exam was normal.

Plain radiographs revealed her previous fusions with degenerative changes in the subtalar, talonavicular, and calcaneocuboid joints (Figure 3). A bone scan showed increased uptake in the right subtalar, talonavicular and calcaneocuboid joints and the left midfoot (Figure 4). She was prescribed orthotics and shoe modifications.

Despite these measures, she continued to have painful symptoms and underwent a right triple arthrodesis. She gradually increased her ambulation, and by four months after her hindfoot fusion, she complained only of mild anterior ankle pain related to being on her feet for excessive periods of time.

Eight months following her right hindfoot fusion, she reported occasional pain in her right foot only when she descended stairs. Otherwise, her right foot was described as pain free and she was very satisfied with surgery. She desired the same procedure on her left foot, which continued to give her problems.



Figure 3B.

The physical and radiographic examinations of the left foot were not conclusive regarding the source of her pain. We proceeded to sequentially inject lidocaine under fluoroscopic guidance into the subtalar, calcaneocuboid, and talonavicular joints. She experienced substantial pain relief with each of these injections. Therefore, a left foot triple arthrodesis was performed.

Her postoperative course was remarkable for the acute development of a tear in the tibialis anterior tendon after removal of the cast. The tendon was subsequently debrided and repaired. Six months later the patient returned with the new onset of pain in the second and third metatarsal-cuneiform joints. Radiographs revealed degenerative changes at these joints.



Figure 4. The technetium-labelled nuclear bone scan confirms increased bone turnover in the articulations adjacent to the region of previous surgery. On the left side the patient also has increased uptake at the second and third metatarsocuneiform joints.

She was given a full length carbon fiber customized insert and instructed on the use of a cane. Her pain continued and three months later she underwent a left second and third tarsometatarsal joint arthrodesis. Her midfoot pain subsequently resolved.

DISCUSSION

In this extended case series, we report the long term outcomes of three patients of Dr. Steindler who were treated for adolescent flatfoot deformities with the use of a medial stabilization procedure and a Young's tenosuspension. The remaining patients are either deceased (seven) or lost to follow-up (three). From this study we conclude that the results of successful medial column fusions and tibialis anterior transpositions to the navicular are reasonably satisfactory for the major portion of the patient's life, but if the patient lives long enough, he/she will likely develop painful foot arthrosis.

We have no control group for these patients. One patient who had a unilateral procedure stated that the involved foot was never as good as the other, and at follow-up reported that the foot restricted her activities.

We do not know whether this was related to the injury, the deformity or the surgery. In the patient who had a successful tendon transfer but talonavicular pseudarthrosis, the long term outcome was good. In the patient who underwent bilateral fusions and tendon transfers, the ultimate outcome was the development of painful joints in the hindfoot and midfoot. Despite this the patient was pleased with the outcome of her initial surgery because she was able to work unrestricted as a nurse for most of her adult life.

This report presents the longest average follow-up of patients undergoing similar procedures. Others have reported earlier follow-up results for patients with similar conditions. Duncan and Lovell reported a procedure which involved a navicular-medial cuneiform fusion with tibialis anterior and plantar fascia tightening to maintain the arch⁸. They reported an average follow-up of 1.7 years in seventeen feet with all patients experiencing symptomatic relief and an improved medial longitudinal arch. Jack reported his three to six year results with forty-six feet which underwent naviculocuneiform arthrodesis¹⁴. Twenty-five outcomes were deemed ex-

cellent, thirteen were good, and only eight were considered to be unsatisfactory. Caldwell described the Durham flatfoot plasty which involved naviculocuneiform fusion with tightening of a raised osteoperiosteal flap into the sustentacular tunnel, thereby forming a strong, plantar sling to support the medial longitudinal arch of the foot⁴. In seventy-six feet with an average follow-up of six years, the results in 76 percent were considered to be excellent, 18 percent were classified as good, and 5 percent were deemed poor.

Macnicol and Voutsinas reported their results with both: 1) excision of an accessory navicular bone, and 2) the Kidner procedure which involved excision of an accessory navicular bone as well as transplanting the tibialis posterior inferiorly on the navicular¹⁹. With the Kidner procedure, nineteen out of twenty-two patients (86 percent) reported significant symptomatic relief and fourteen out of twenty-two patients (64 percent) had an improved arch. The average follow-up for the Kidner procedure was ten years. With excision of the accessory navicular alone, five out of six patients reported relief of symptoms, and four out of six patients had an improved arch. The average follow-up for this procedure was twelve years. Crego and Ford reported their results of several related procedures⁷. Young's procedure with mobilization of the tibialis anterior was performed in six feet with a 100 percent satisfactory result at an average follow-up of 5.5 years. In the same series, they reported the results of nine naviculocuneiform fusions at an average of 9.5 years. In this group they had seven poor results and two good results.

Fraser et al. reported on thirty-eight naviculocuneiform fusions with advancement of an osteoperiosteal flap beneath the tibialis anterior tendon with an average twelve year follow-up⁹. Thirty-five of the feet underwent tendo Achilles lengthening and seven also had a first metatarsal-cuneiform fusion. Eighty-four percent reported a satisfactory clinical result. They believed that successful results following the Miller and Durham procedures are due, in large part, to the soft tissue suspensions and tightening which appear to protect the tarsal joints from degenerative changes⁹. Interestingly, they reported a 21 percent rate of nonunion which was unrelated to the development of pain⁹. The longest published follow-up of adolescent flatfoot procedures was reported by Seymour. He described thirty-two feet which underwent naviculocuneiform fusion between the ages of eleven to fourteen, with a follow-up of between sixteen and nineteen years following surgery²³. Ten (31 percent) of the cases were deemed excellent, six (19 percent) were deemed good, and sixteen (50 percent) were considered unsatisfactory. He concluded that the early encouraging results of naviculocuneiform fusion

for the mobile flatfoot had not been maintained and this procedure hastens degenerative changes in the other tarsal joints.

The present study documents the eventual demise of contiguous joints after fusion and tenosuspension of the medial longitudinal arch. However, the procedures appeared to restore function and relieve pain for the major portion of the patients' lives. Orthopaedists considering such procedures for the treatment of adolescent flatfeet should realize that if the patient lives long enough, they may develop painful arthrosis of other joints of the hindfoot and midfoot.

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TOTAL JOINT REPLACEMENT: ON INNOVATION, AMBITION, COURAGE, IRONY AND MORSELLIZED BONE, OF COURSE*

Rik Huiskes, Ph.D.**

“Skepticism is the first step on the road to philosophy.” — Denis Diderot

A sensible Russian expression reads “If you’re unhappy, reduce your wishes, and you’re happy again.” Try it; it always works. Unless, of course, ones bread and butter is the innovation of, say, total joint replacement (TJR). There is a revealing anecdote afloat in orthopaedic circles, allegedly created by Shelley Simon (an American orthopaedist) when asking Peter Walker (an English professor of Bioengineering) one day what occupied his mind at the time. Truthfully, Peter answered he was working on a new knee prosthesis. “A new knee prosthesis?” Shelley replied in astonishment, “But why? The problem is solved; you solved it yourself!” This assessment of the state-of-art in total knee replacement (TKR) might have left Peter unhappy for a while, an issue the anecdote does not reveal. In any case, innovators in contrast to the Russian wisdom, tend to increase their wishes in order to find happiness. The question is, is there still room for such ambitions in TJR?

The question of whether the problems of knee and hip replacement are now basically solved is very relevant today. There is a general awareness among orthopaedists that cemented joint replacement provides reproducible results and acceptable failure rates. Despite the interest in noncemented innovations during the last decade, the motion for cement would, if put to a vote, find a large majority of supporters. In fact, the recent decade has witnessed a downfall in the innovative process regarding prosthetic development. There might be a truly perfect jewel along all the junk created, but there is no real opportunity to establish its identity objectively at the present time. Isn’t it a matter of supreme irony that after all the money, time and creative activity spent, the vintage Charnley prosthesis is still documented as the most successful hip replace-

ment, and the Total Condylar as the best knee replacement?

The failure of the innovation process and its consequences for the patients involved also introduced the decay of the ‘cowboy’ era in orthopaedic technology, when patients were considered the experimental animals of choice. We all know that the archaic ‘trial and error’ innovation procedures are extinct; as they are no longer ethically justifiable. Every new prosthetic feature must be pre-clinically tested for safety and efficacy before used in a patient. If its unworthiness is not exposed by stern computer-simulations or laboratory bench tests, it may be verified in limited clinical trials, using roentgen stereophotogrammetry analysis to detect even the smallest motions which, once established, will cause the innovative prosthesis to subside into the drawer; for good. In the unlikely event that it stays put, it’s time to find out whether the orthopaedic community at large is apt to handle it through well-documented prospective, paired, multi-centered clinical trials. The investor is about to receive the first returns on a fortune spent. What follows is post-marketing surveillance in the Swedish Register. The orthopaedic community outside Sweden is recommended not to acquire the new design until it has been proven to endure, relative to the golden Charnley standard, for some ten years. Such is the strategy for a responsible, scientifically accountable, step-wise introductory procedure for an innovation in TJR.

Are you still there, reader? Yes, indeed, my own eyelids were getting heavier, too. Who wants to innovate on these terms? And then, oh irony, the chances of getting better survival rates than the Charnley are so slim that it’ll be nearly impossible to prove with statistic significance within a period of ten years. The best an author can realistically hope for is that his product will not be shown to be worse. It may not be worth our while to improve upon a proven concept.

“The very substance of the ambitious is merely the shadow of a dream.” — Shakespeare

Shouldn’t we, innovators, just quit? Call it a day, turn our collars to the wind and wander out towards new horizons? But that would mean defeat, wouldn’t it? Because, despite the lack of significant clinical improve-

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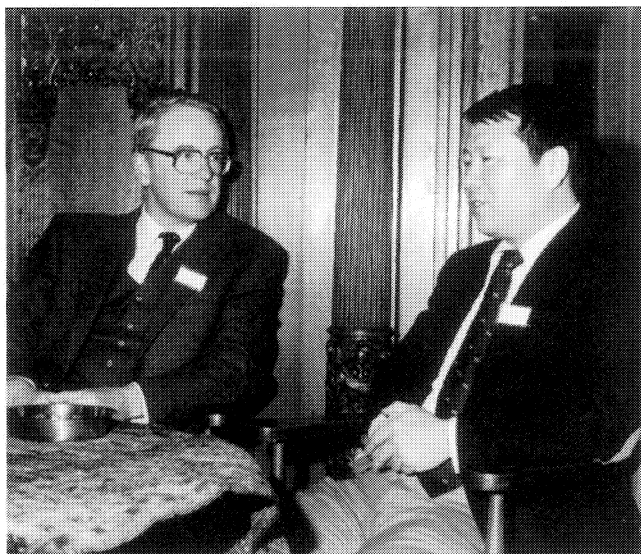


Figure 1. Tom Slooff in 1982, with Ed Chao, during the Conference Banquet, 3rd Congress of the European Society of Biomechanics. First reports on morsellized grafts?

ments, cemented TJR is all but perfect. Think of the archaic process of cementing, the tampering with the slimy stuff in the operating room. Imagine the carpentry of the bone itself, joyfully shaking with the motions of the rasp. Envision the placement and cement-curing phases, the sense of timidity it creates, a feeling not dissimilar to what most golfers experience when approaching the green. Artistic, surely, but perfect? While working on our Acta supplement “Quality Assurance of Joint Replacement”, Lauri Faro once suggested we might rather regulate the surgeons, instead of the prostheses, in order to ensure safety and efficacy of TJR. That should not be necessary. There are no fools in orthopaedics, naturally, but TJR should be fool-proof nonetheless. How to do that? Well, that’s the challenge, isn’t it? Robotic-enhanced surgery? The shadow of a dream.

Think of the cement itself, a material appropriate for filling holes in the walls of a garden shed, but basically too weak to endure the joint forces of daily living for an adequately prolonged period of time. It produces heat, shrinks, debonds, abrades, degrades and accumulates micro-cracks, in that sequence, and finally gives way. Its eventual doom can only be deterred through a timely death of the patient. It should be our ambition to replace it with something better. That this isn’t a trivial matter was illustrated by the many who tried in vain. But still, a goal it must be. Biological coatings morsellized bone? Who knows; but the ultimate future of TJR will be one without cement; it’s inevitable.

Think of the polyethylene; how nicely it shines, how effective it is for making little bags in which to carry



Figure 2. In 1984, Tom Slooff with Bill Harris, two heroic innovators. Massive versus morsellized grafts?

our sandwiches to work. Imagine how convivially they litter the forests after the Sunday picnic; they last forever, indestructible. But for a load carrying joint implant to endure for, say twenty years? Safety belts should be enforced when running on such a material. No, I don’t know anything better, either, but that’s not the issue. The point is that contemporary TJR is far from perfect, despite our gloating over Swedish survival rates.

“Writing free verse is like playing tennis with the net down.” — Robert Frost

On travels I tend to meet orthopaedists and bioengineers, innovators by nature, scheming for hours, in pairs, red ears and excited voices, discussing the details of their new designs. Interesting people; creative, resourceful, responsible, visible, eloquent, socially engaged in the field. I enjoy their company. The questions why is this new prosthesis being developed; what problem it is meant to solve, usually bring nothing but oblivious glassy-eyed stares. Innovators, it seems, are much like mountain climbers; any variety feasible must be tried, any invention applied. Where climbers tend to be restrained in their enterprises by the limited availability of funds, the orthopaedic industry is not reluctant to gamble in the orthopaedic roulette called ‘Chasing the Charnley’.

The truth is that most of the innovations introduced in the last decade are incremental adaptations, variations on a theme. A way to brighten up your day is to

ask a salesperson what are these variations for; they don't know. It's not that they're dense or inarticulate, they just haven't been informed. The problem is, no one knows. In the industrious, convivial, unregulated game of TJR innovation, the players produce one solution after another, but the code which ties these to the problems we all know seems to have gone astray. A game without rules, hence with no winners.

Let's leave this stage for a moment, and sit down to think. If we worship new solutions, the first priority is the identification of problems deserving our attention. I mean, if we want to be heroes, we need heroic goals; saving the ants in the garden may be commendable, but it won't do much for fame. If the Charnley is perceived by the masses as the solution of its typical problems, so be it. Let's leave this alone then, for the time being, and concentrate on alternative issues like, for example, its untypical problems. There are two of those in this field; the very young patient and the revision. That these are indeed problematic is illustrated by the fact that many orthopaedists will simply ignore them, pretending they don't exist, or hope the ill-fated concerned will move elsewhere. But let's forget about the meek and rather talk about heroes for a while.

"There is no darkness, but ignorance." — Shakespeare

Revision surgery is a problem from several perspectives. Many orthopaedists ignore its existence and health insurance organizations refuse to fund it. Which is the cause and which the effect in this matter I can't say, but true it is. So their treatment is left to the altruistic, of which the orthopaedic field probably has few, or to the truly ambitious.

The second problem, partly an effect of the first, naturally, is its common postponement, not in the least because of the hesitation ruling both surgeon and patient. I'm known socially as a lay (hence approachable) person with orthopaedic connections, therefore I'm frequently asked for advice concerning the wisdom of particular treatment modalities suggested to patients by their surgeons. My ground rule for such unbillable consultancies is that the ailed should postpone elective surgery as long as humanly possible. People tend to like that recommendation; its obviously the easiest one to follow, it establishes my independence from the medical community and exposes the shameless advancement of the surgeon's own business interests, completely undeserved of course. Anyway, revision surgery is the exception to my rule. Revision surgery is like the enjoyment of wealth, there's really only one sensible moment to do it: now.

But, like I said, it's usually done too late, such that the bone has withered, introducing the third problem. As any carpenter knows, one can't have one's wood eaten by wood worms and have it, too. In restorations of antique furniture the replacement of bad wood is commendable, be in only to prevent grandma from rolling into the fireplace at the next Christmas reunion. The same principles rule revision surgery. Revision surgery can be considered as the "continuation of joint replacement with other means," paraphrasing von Clausewitz on a statement he never made, or rather never meant, as I heard recently. Anyway, our problem here is "what other means?". Food for real innovators.

"Heroism feels and never reasons and therefore is always right." — Ralph Waldo Emerson

In the late seventies, Tom Slooff, whom you all know, decided to try a solution to this problem. Forcefully, he crumbled excised trabecular bone (the word morsellized had not been coined as yet; in fact it's still not carried by Webster (1996), obtaining a biological analog of 'yogurt with muesli', and used it to repair a leaking acetabular roof. After blending it with cement (as some people foolishly put whipped cream over their 'yogurt'), he pressed the acetabular component in and let the cement cure. Miraculously, the patient survived and marked the onset of an exceptional success story. Conceived some nineteen years ago, revision surgery with cement-blended morsellized bone grafts (impaction grafting) is now the talk of the town in orthopaedic circles. Tom Slooff invented it, and anyone suggesting otherwise may be kicked in the butt on my behalf.

This invention was courageous, certainly. I didn't believe in it at all, all the time. I can bring up my relative youth and lack of biological background in defense, but the truth is that I was notably shortsighted in this matter. I couldn't fathom this muddy substance capable of withstanding the forces. But it did. The clinical results were promising and Tom decided to use it for femoral revisions as well. Sleepless nights is what it gave me. I convinced him to try it out in goats first, poor animals. Unfortunately he consented; good for my faith, valuable scientific data and those sorts of things, but had he not consented, he would have been at it much earlier than the Exeter group, and the method might be known under another name. (Impaction grafting is often referred to as the Ling technique after R.S.M. Ling from Exeter, England who first reported clinical results using impaction grafting for femoral revision surgery). I still blame myself for scorning the enchantment of adventure. But then, of course, Tom's the surgeon, I the scientist; engineers and poets, innovators and contemplators. There's a lesson here, no doubt.

There was another lesson in this experience for me, and I will come back to both below. Let me first say that the behavior of the graft material in all kinds of animal experiments was amazing. Revascularization occurred quickly followed by remodeling and graft incorporation. Although some failures were seen in animal hip replacement, by-and-large secure fixation was obtained. It seemed, however, that the initial stabilization of the morsellized bone substance with cement was important; noncemented prosthetic components tended to loosen at an early stage. So there we were, the biology *can* put it right, if only the proper conditions are created.

Morsellized grafts become a focus of clinical and scientific attention in our department. Its applicability for knee revisions and the restoration of bone loss in avascular necrosis are being investigated, and its behavior under the influence of variable forces and morphogenetic agents are being studied in animal models. The main targets now, of course, are to know how it works, to specify the optimal conditions for the graft, and to unravel the factors that can make it fail. This information could engender better reproducibility and promote applications for other purposes. What a happy ending to an heroic enterprise.

“Rules and models destroy genius and art.” — William Hazlitt

So what about ethical considerations? What about pre-clinical testing and step-wise introduction of TJR innovations? Aren't we doing things in a reversed order? Weren't the patients the first experimental animals? Reluctantly, I can only admit it, but I have two arguments for our defense. First, there was no reliable treatment for revision patients. Just as there was no effective treatment for coxarthrosis and the like in Charnley's time. So experimentation with revision patients was ethically defensible. Second, and I hate to say it, this is probably the only realistic route towards real innovation; I'm talking about significant novelty, of course, not about an alternative bend in a hip stem or a fold in a cup. You see, if it had not been established clinically that this method might work, it would probably never have emerged. In the reversed order, the ethically 'correct' order, we would still be researching the method, forever coming up with more questions. Such is the role of the natural sciences, of course, to unravel the work-

ings of nature in ever refining detail. It is the function of technology to innovate; it takes an engineer, like an orthopaedic surgeon. Science is useful when it comes to explaining why things do or do not work, or for providing the information required to carry a new method towards perfection. Scientists are not inventors by trade, they provide knowledge, useful or not.

I still stand by all I've written about pre-clinical testing and step-wise introduction of innovative TJR, unabridged. It's an excellent framework for protecting patients, and the orthopaedic community at large, against commercialism and its innovative allies. But regulation and innovation are adversaries by nature. To really get ahead in surgery it still takes 'cowboys', despite the change in culture. Whether their actions are justifiable is appraised only by success or failure.

“Science is the knowledge of consequences, and dependence of one fact upon the other.” — Thomas Hobbes

The practice of orthopaedic surgery implies “providing the mechanical and biological conditions to the tissues which allow them to heal, adapt and maintain themselves.” This has become one of my favorite statements. Morsellized bone grafts fit very well in this concept. In fact, the conception of the notion itself was very much inspired by the experience with these grafts in our research program. Biology can put it right, if only provided with the proper stimuli; what a challenge for an orthopaedic scientist. What a contest to unravel these stimuli, define them, measure them, model them, and then apply the information in what is now called the science of 'Tissue Engineering'. This science provides the knowledge that cells produce and maintain musculoskeletal tissues with just the right mechanical properties required to perform their functions. 'Mechanobiology' we've re-baptized our research program, precisely to emphasize that excitement. Who knows, in the future, we may be able to solve the problems of the young patient as well and rid the orthopaedic community of cement.

Tom Slooff will leave us with a method that appears to have revolutionized TJR revision surgery, and may have solved its problems for good. But he, also, leaves interesting questions behind. To a philosopher, this appears even more important.

DETECTION OF WIRE EMG ACTIVITY IN WHIPLASH INJURIES USING WAVELETS

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INTRODUCTION

Whiplash syndrome is one of the most complex and poorly understood neurophysiological disorders of the cervical spine. It is produced when sudden acceleration or deceleration forces are applied to the head and neck.

Up to now, the study and diagnosis of "whiplash" has been based on:

- (1) radiographic appearances, and
- (2) biomechanical studies.

In the latter case, the role of the cervical muscles has not been included. This is due to the fact that the currently used methods of signal analysis (time or frequency) do not provide useful information about muscle activity shortly after collision (125-285ms - according to the study by Tennyson et al.⁸, 1977). However, a newly developed mathematical tool of wavelet transforms (continuous and discrete) can be used to determine localized features of the signal, such as abrupt changes and transients²; this permits evaluation of muscle activity in the cervical muscles (shortly after collision) in a whiplash simulation experiment.

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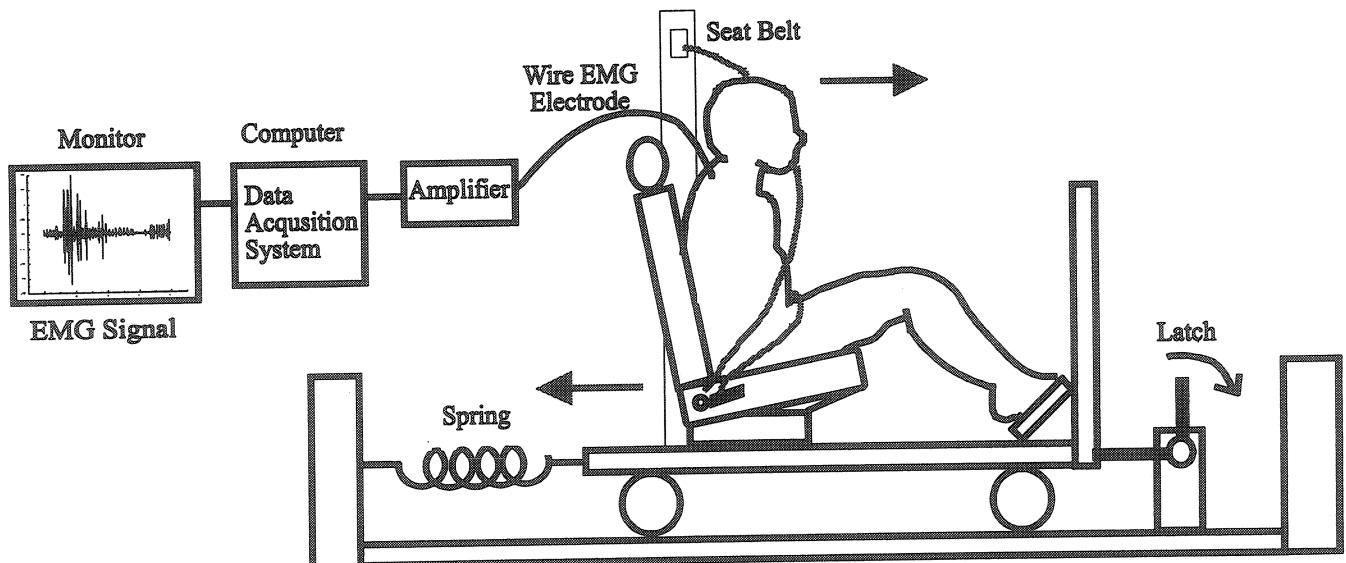


Figure 1. Schematic diagram of whiplash simulation experimental setup. (Arrows represent the direction of movement.)

MATERIALS AND METHODS

A. Wire EMG Experiment

The subject, a male in his early thirties, height: 175 centimeters, weight: seventy kilograms, was seated in a car passenger seat on a cart as illustrated in Figure 1. The cart had a bucket seat and three-point seat belt system from a standard compact automobile and was equipped with an adjustable head rest. The seat was mounted to a wooden cart with four wheels. The wooden cart was placed on a platform, and a spring mechanism was attached to the back of the cart. This mechanism was attached firmly to the back of the platform. To release the cart, a gate latch attached to the front of the platform was used. A spring allowed backward motion of the cart after the release mechanism was triggered.

A wire EMG electrode was applied to the sternocleidomastoid muscle (posterior aspect) at a depth of

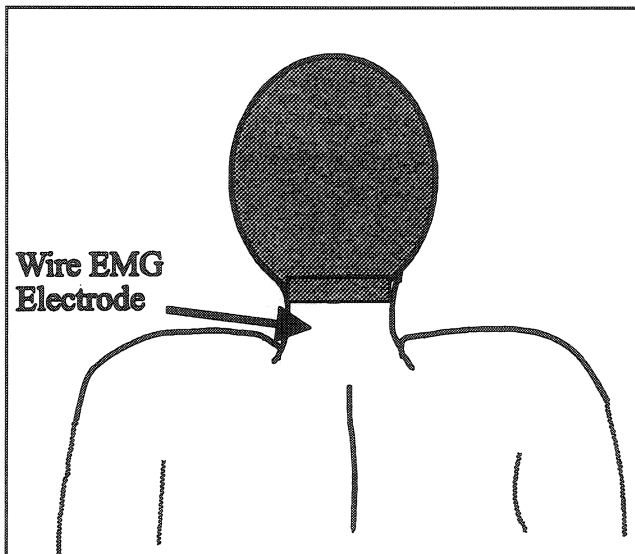


Figure 2. The location of the wire EMG electrode is indicated by the arrow. It was applied in the posterior aspect of the sternocleidomastoid muscle at the C3 level at a depth of 18 mm.

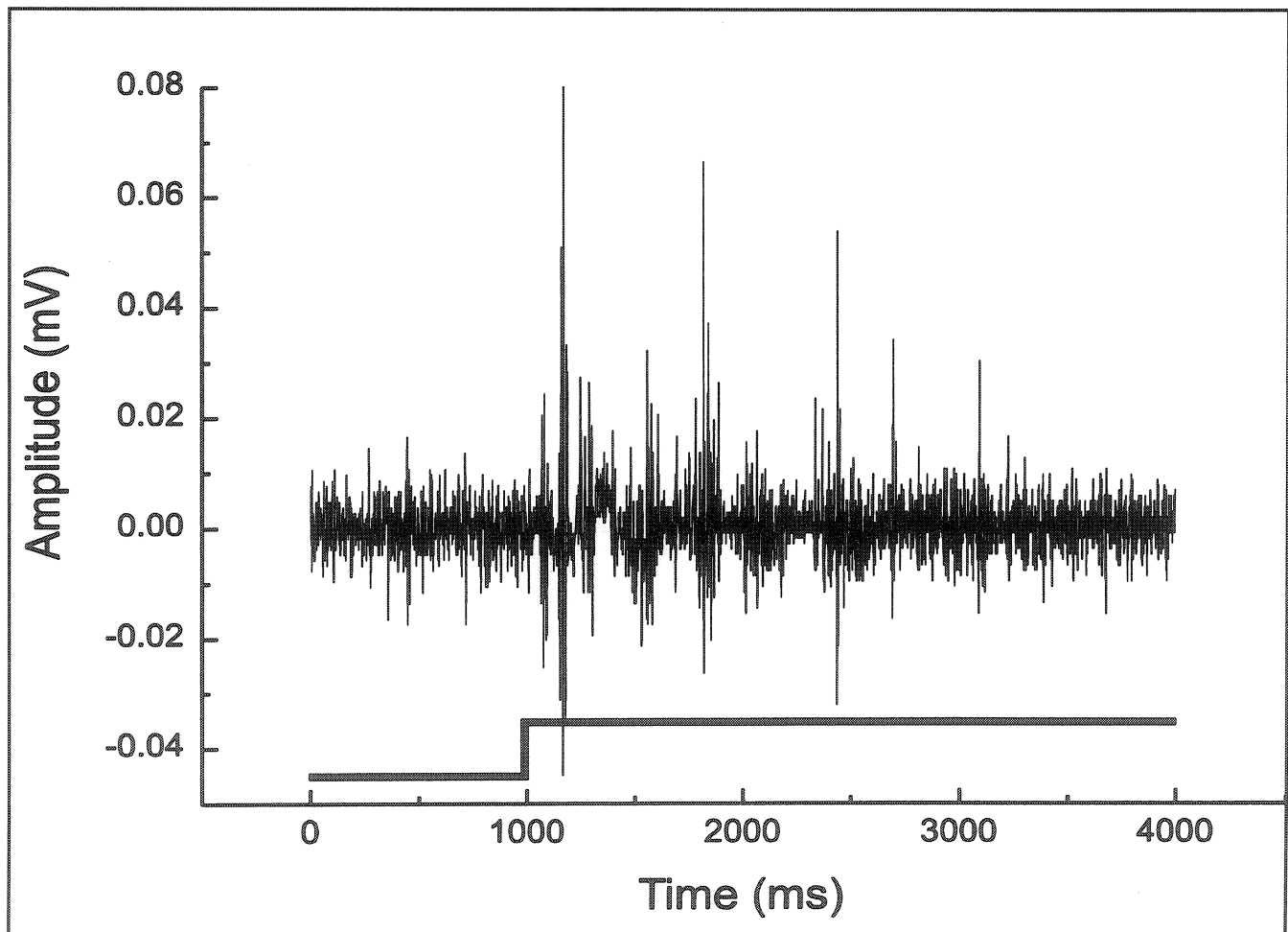


Figure 3. A sample wire EMG signal collected during the unexpected load. The step function at the bottom represents the start time of the motion of the sled.

eighteen millimeters, as shown in Figure 2. A sudden unexpected load was applied from the front by loosening the latch. In this experiment the acceleration was set not to exceed 0.5g (4.9 m/sec²). The EMG activity and other signals were recorded for four seconds at 1000Hz.

The EMG signal plot in Figure 3 shows an example of data collected after unexpected release of the latch.

B. Application of Wavelet Techniques

Traditionally, EMG signal analysis to determine muscle activity during contraction is performed using Fourier Transform based methods - Fast Fourier Transform (FFT) and Short Time Fourier Transform (STFT). Although these techniques help to decompose the signal to its frequency components and help to determine the relative energy of each component, they do not tell when the signal exhibited a particular frequency characteristic. In other words, the FFT has problems in resolving a signal in both time and frequency domains. On the other hand, the STFT positions a time window at any point on the time axis and calculates the FFT of

the signal within the spread of that window. The basic problem with STFT is that once the window duration is chosen (fixed time resolution), the corresponding frequency resolution is also fixed (uncertainty principle). This has the following limitations. If the signal has a transient component which has a duration smaller than the time window used, it is difficult to locate it with precision. In addition, if the signal has important features of different sizes, we cannot find an optimum time window for its analysis. If the window is too wide it takes samples from too many components, and if the window is too narrow it takes fewer samples. The resolution is poor in both cases. Therefore, STFT is suitable only for the analysis of signals when all the features in the signal are of the same scale. In fact, EMG signals obtained during dynamic conditions do not have features of the same scale and, therefore, the STFT is not useful for their processing.

A recently developed mathematical technique, known as wavelet transform (WT), has been successfully applied to the analysis of complex non-stationary signals in other fields. This technique provides the best reso-

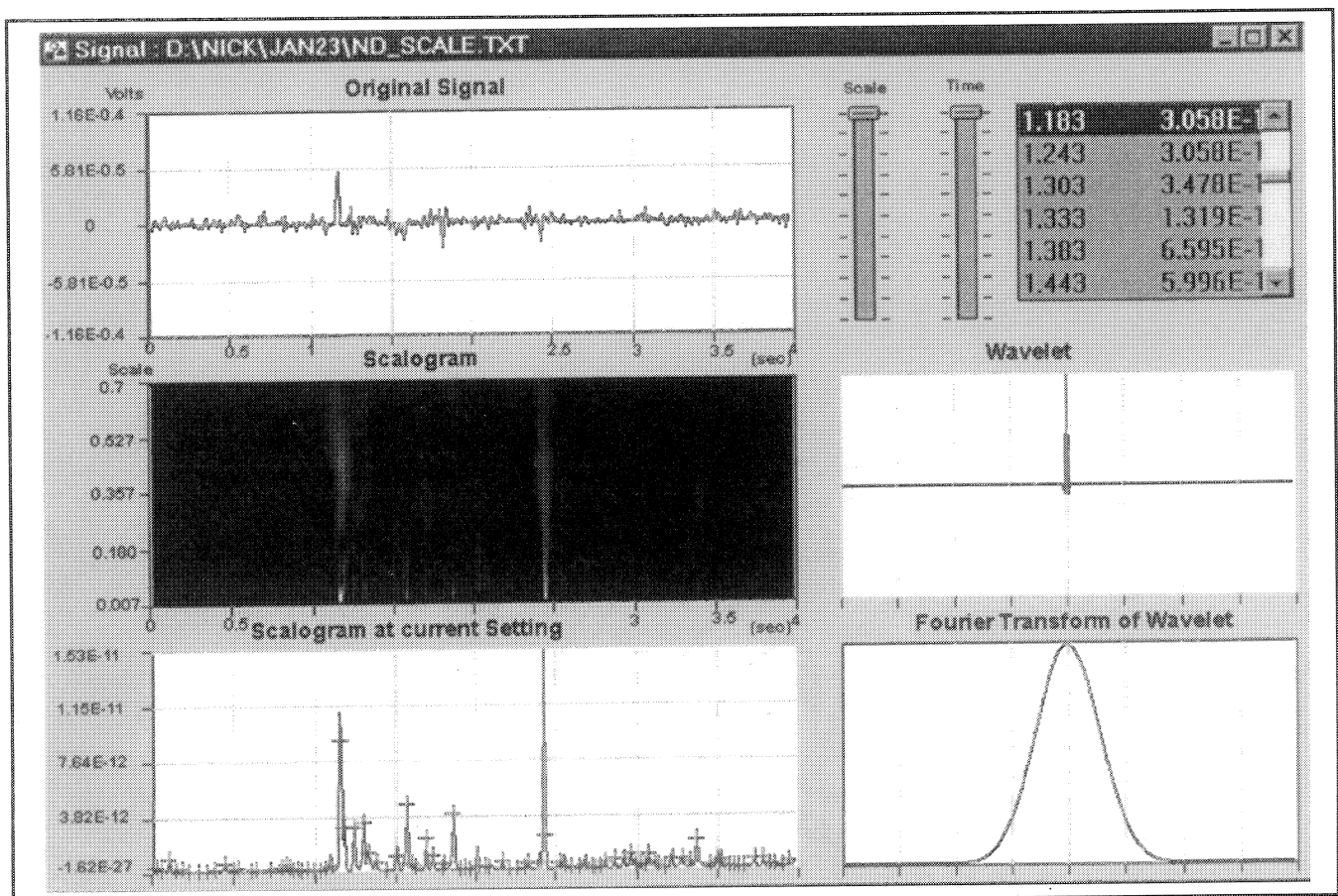


Figure 4. Scalogram obtained by applying Morlet Wavelet Transform to the signal shown in Figure 3.

lution in both time and frequency domains, and it is well suited for most real world signals consisting of high frequency components of short duration and low frequency components of long duration.

(1) The Continuous Wavelet Transform (CWT)

As it is known, Fourier techniques deal with the representation of a signal with a finite sum of sine and cosine terms (called basis functions) multiplied by properly chosen coefficients. These coefficients depend on the signal itself and the basis functions. In the case of the wavelet transform, we replace the sines and cosines with special basis functions formed by scaling and translating a special function known as the “mother wavelet.” These functions must be simultaneously oscillatory and have amplitude which decays quickly to zero in both negative and positive directions. Using these basis functions we can formulate an integral transform known as Continuous Wavelet Transform (CWT). It is this transform that has been applied for the analysis of the wire EMG signal under study. More specifically, the basis function chosen for this study is a Morlet wavelet. Figure 4 displays the results obtained by using the Morlet wavelet transform. In this figure, the scalogram represents the squared modulus of the CWT (CWT^2) versus time for a given scale range. The scale range used for this example was from 0.007 to 0.07. Directly below the scalogram, a representation of a cross-sectional view (scale profile) obtained from the scalogram for a particular scale is presented. On the top right, a table automatically displays the times and the magnitudes of all the local maxima of the energy curve associated with the above mentioned scale profile. The time data shown in this table do not display all the time values of the observed muscle activity. This is due to space limitation. The user can obtain the full time-magnitude information by scrolling the bar on the right side of the table up and down. Directly below the table, the Morlet wavelet, its Fourier transform, and the standard deviations in time and frequency domains are displayed. The latter information constitutes the time-frequency analysis for the given wire EMG signal as the scale changes. Clearly the mother wavelet expands and contracts in time, depending on the scaling parameters causing corresponding contraction or expansion in the frequency domain. We may think of WT as a mathematical microscope with the compressed mother wavelet used to detect singularities or abrupt changes in the signal and the expanded mother wavelet used to detect fine frequency details.

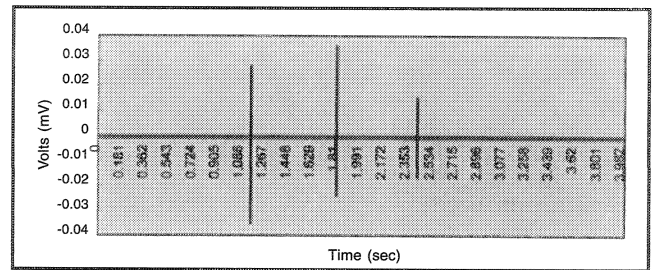


Figure 5. The MRA decomposed filtered signal using scalogram set a threshold of 0.4 percent of total energy in the signal. This signal was decomposed by using Daubechies 16th order wavelet at level eleven resolution.

(2) Multi-Resolution Analysis (MRA)

All finite energy signals are composed of fundamental components generated by functions known as “wavelets.” Wavelets partition the space of finite energy signals into a series of disjointed subspaces known as multi-resolution analysis (MRA). The observed form of the signal is the superimposed combination of all components generated in these wavelet subspaces. The MRA decomposition algorithms separate these fundamental components from the original signal as distinct individual signals. No further decomposition of these component signals into signals with more simple structure is possible. Each of these component signals can then be evaluated according to its contribution to the entire original signal.

The MRA reconstruction algorithms recombine each of these component signals into superposition patterns. If all the fundamental components of the original signal are retained in the recombination operation, then the original signal is reproduced exactly. However, if some of the fundamental components are identified as noise or some other undesirable component, then this component signal is not included in the reconstruction. The resulting signal will then be an improved version of the original signal. Above we display the output of MRA decomposition of the wire EMG signal used in the CWT case discussed earlier by applying Daubechies sixteenth order wavelets as it is shown in Figure 5.

RESULTS

The detection of wire EMG reaction times is obtained by using CWT and MRA techniques. Figures 4 and 5 show clearly that there was muscle activity following the cart motion. More specifically, for the CWT case, the times were 206ms, 827ms and 1496ms for the three first major responses, and 193ms, 827ms and 1445ms for the MRA case. These times are detected automatically and they are in good agreement.

DISCUSSION

For the first time, muscular activity has been observed in an experimental setup of a simulated whiplash experiment. In this study it was demonstrated that cervical muscle activity exists shortly after the initial motion of the cart, and it is detected automatically using two different techniques: continuous wavelet transform and multiresolution analysis. As it is shown, both techniques gave approximately the same results for the times and frequency of occurrences of muscle activity within 125-285ms after the front or back collision occurred.

The significance of this finding lies in that such muscle activity may be a contributing factor in the creation of a more realistic whiplash biomechanical model. However, the main importance of such a whiplash biomechanical model will be in its utilization for the development of preventive whiplash mechanisms. The small difference in the time occurrences can be attributed to the fact that; first, the two algorithms used were different; and, second each algorithm used different wavelets. More specifically, the CWT method used the Morlet wavelet, and the MRA used the Daubechies wavelet.

In this study, we examined the relationship between EMG and the signal processing techniques based on CWT and MRA. The computer algorithm in both cases provided more precise information than human readings of non-processed EMG signals. Up to now, the most commonly used time domain based EMG signal processing techniques are averages or means, rectification, smoothing of the rectified signal, integration and the root-mean-square (RMS) value⁷. There is no specific rule for choosing which processing technique to use for processing the EMG signal. Even the relationship between processed EMG and force is not unique with identical experimental setups and carefully controlled testing conditions⁶.

In addition, the CWT and MRA results provided a quantitative measure of the muscle activity. The relationship between EMG during the resting and contracting periods can be analyzed by counting the number of impulses and the integrated electric activity in EMG⁵. The generally applied quantitative analysis method is the counting of zero-crossings and turns of the motor unit action potential (MUP) signal¹. This manual measurement is very subjective and very time consuming in clinical use³. Therefore, the precise and sensitive time-frequency information of CWT and MRA techniques meets the practical need for the automatization of quantifying EMG.

The cervical spine with its ligaments intact but without muscle activity is a very unstable structure. The muscles and complex neuromuscular control are re-

quired for the stability of the neck during posture and movement. The sternocleidomastoid muscle, the muscle from which EMG signals were obtained, is an important component of the neck musculature. The origin is from the sternum and middle third of the clavicle and it inserts into the mastoid process. It is a powerful flexor of the head and neck during symmetrical, bilateral contraction. This muscle may act to protect the spine during whiplash in which there is time for voluntary control, dissipating kinetic energy by eccentric contraction before impact.

The hypothesis that the cervical muscles cannot react fast enough for protection during sudden acceleration⁴ was not supported from these results. Thus, the assumption that humans are helpless during collision is premature. The muscle reaction appears to be initiated by the initial impact.

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SELECTING TOPICS FOR SCIENTIFIC INVESTIGATION

B. F. Morrey, M.D.

Expertise with basic or clinical research is mandated by the Residency Review Committee as essential for accreditation of orthopedic residency programs. The execution of this mandate varies widely as opportunities differ from program to program based on clinical and financial resources as well as the varied interests among trainee and advisor. However, the one certainty regarding this process is the request (supplication) by the resident in training for assistance in selecting a topic in order to carry out this mandate.

The response from the mentor to this request may vary broadly such as "I have a specific question and group of patient records that are begging to be reviewed" to "I am pretty busy but I will be happy to read your manuscript".

Because the time of a trainee is no less limited and just as valuable as is that of the mentor, some basis or criteria to help select topics and projects would seem to be of value. We have attempted to minimize the process to assure the time invested by the trainee is rewarded according to several measurable outcomes: 1) represents a valid contribution to the orthopedic literature; 2) offers a rewarding experience for the resident that directly translates into practice; 3) provides an opportunity for presentation at a major scientific forum; and 4) provides an enhancement of the educational content and clinical experience of the trainee. Two thoughts should go without saying. First, the intent is to provide a contribution to the most elite of the peer reviewed journals. Second, the advisors must be willing to help design the study, be available for advice and finally offer a timely review of the manuscript once completed. We have found that the attainment of these lofty expectations can be maximized by subjecting the process of topic selection to a few discreet and simple questions or tests.

1) Is the topic clinically relevant; is it important?

It is crucial to realize that a particular question may be of interest to an attending, but it must be questioned whether or not it is of equal or valid interest to the orthopedic community. For example, we may be interested in having the resident look up a discrete series of surgical interventions that we once did in the past and are no longer performing. It may be a procedure that has been supplanted by a subsequent operation. [This is a curiosity search.] Other than our own personal interests, there is really no reason to be investigating such

a topic. One must ask whether the question being addressed is one that has broad clinical relevance? Is it of interest to the surgeon in the trench? Is it relevant to our patients?

2) Has it been adequately addressed; or if there is a supposed answer in the literature, is it correct?

I remember many years ago a resident approached me indicating his interest in reviewing cervical spine films of patients with rheumatoid arthritis since he had just seen a patient with an unstable C1-2 rheumatoid lesion. I was aware that there had been a number of very fine articles on this topic and recommended a detailed review of the literature before he spent his time and effort to repeat what I thought would only be a confirmation of what was already in print. After the review the resident was knowledgeable about this subject and selected another topic to study. A thorough assessment of the literature is, therefore, necessary to answer this question of whether there are issues to be answered. This must be determined before we continue with the process of selecting a topic. Remember, the fastest way to learn the answer to a specific question is to read a book or turn to the literature. Chart review is slow going. Only if the literature is inadequate do we embark on a clinical research project.

3) Do we, at our institution, have sufficient clinical material available to us to definitively answer the question or make a substantive contribution?

A number of years ago I was interested in elbow instability. I was unsure of how to manage the few but increasing number of patients I was seeing with the problem. I reviewed all the records of patients who had surgery for elbow instability at Mayo and found that it was only a handful with a heterogeneous etiology. Shortly thereafter a classic paper by Frank Jobe on medial collateral ligament insufficiency of the athlete gave me most of the information that I needed regarding this topic to treat my patients. I dropped the project.

The completion of items two and three above constitute what I consider the feasibility phase of the project; that is, a review of the literature to determine whether or not the topic has been adequately addressed and a review of our own material to determine whether or not we have sufficient material to make the definitive or a significantly relevant comment or contribution. Next, we enter into the final phase of the analysis, what it takes to bring the project to closure.

4) Completion is solely dependent on the time and effort of the primary investigator.

I tend to avoid any project that has, as an integral portion of the design, a multi-disciplinary contribution. The reason for this is that it requires others to have the same passion or interest in the topic as do I or the trainee. If this interest is not present and if the outside contribution is essential, then the likelihood that the project will come to fruition is lessened. Thus, I try to limit topics to those that are solely within the competence of the resident and the attendee to complete.

5) Do we (the resident) have enough time to complete the project? I feel very strongly that every moment invested in clinical research should be brought to closure. If the above tenants are followed, the project has every opportunity of coming to a satisfactory outcome. However, it is the attendee's responsibility to assure that the value of the project is commensurate with the time available to devote to the effort. Thus, residents in their earlier years are given projects that require greater time for completion than those in the latter years of their training or fellowship. A recent example is a review of over 5,000 cases of patients with elevated acetabular liners inserted at our institution. This was initiated by a first year resident. The article was published in the last year of this devoted resident's training. Similarly, a one-year fellow recently reviewed forty cases of elbow replacement arthroplasty performed for traumatic arthrosis. This hardworking fellow was able to complete this task in the one year avail-

TABLE 1
Criteria for successful clinical research project

• Important topic
• Not answered to date
• Resources definitive
• No outside resources
• Time/commitment present

able to him. Both articles have or will appear in the literature, and the initial manuscript was finished in both instances prior to the individual leaving Rochester.

One may, of course, add or substitute any one of a number of other variables or considerations for those mentioned above. These are the particular criteria that I have used through the years. They are easy to remember and apply, and have been effective for me. With a little practice it is easy to recite to the resident the essential ingredients of successful research: 1) significant or important topic; 2) the answer which is not known; 3) with adequate experience at our institution to answer the question; 4) we need no one else to bring this to closure; and 5) the resident has sufficient time and interest to finish the project in the time allotted (Table 1).

If nothing else, this does provide a framework by which topics might be discussed and selected. Hopefully, this review will stimulate a similar, or modified approach among others.

USE OF ANCHOR SUTURES TO REPAIR LABRAL AVULSIONS OF THE HIP: A BRIEF REPORT

Dennis P. Weigel, M.D.
John J. Callaghan, M.D.



Figure 1. Injury film demonstrating posterior dislocation of the hip with no apparent fractures.

INTRODUCTION

Traumatic dislocation of the hip is uncommon in the pediatric population⁴. Although the association of labral pathology with hip dislocations in both the pediatric and adult population is well documented, the treatment of the labral pathology has received little documentation. We report a case of posterior dislocation of the hip in a child with intra-articular obstruction to reduction caused by labral interposition. The labral pathology was treated using the principles and surgical techniques utilized in the treatment of labral pathology in the shoulder.

CASE REPORT

A nine year old boy suffered a twenty foot fall from a tree landing on his left hip. The patient was initially seen at a local emergency room and was transferred to our hospital later that evening for further evaluation. On exam, the left leg was shortened and internally rotated.

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Palpation of the left groin was painful. Pedal pulses were normal and lower extremity strength was full for all muscle groups except for quadriceps strength which was decreased because of pain. No other injuries were noted on physical examination. Radiographs revealed a posterior dislocation of the left hip without any apparent bony injury (Figure 1). Closed reduction was performed with two mg of IV morphine for pain control. Post-reduction, the hip was stable to ninety degrees of flexion and painful with internal and external rotation.

Radiographs revealed a non-concentric reduction of the hip with medial joint space widening. CT scan demonstrated an intra-articular osseous fragment with a bony defect in the posteroinferior aspect of the acetabulum (Figure 2).

The patient was taken to surgery. A posterolateral approach to the hip was performed by incising the piriformis and conjoint tendons and preserving the medial femoral circumflex vessel. A large defect in the inferior, posterior, medial margin of the joint capsule was identified with the femoral head button-holed through the defect. A horizontal incision was made in the capsule to re-dislocate the hip. On exploration of the joint, a labral avulsion with a small bony fragment attachment

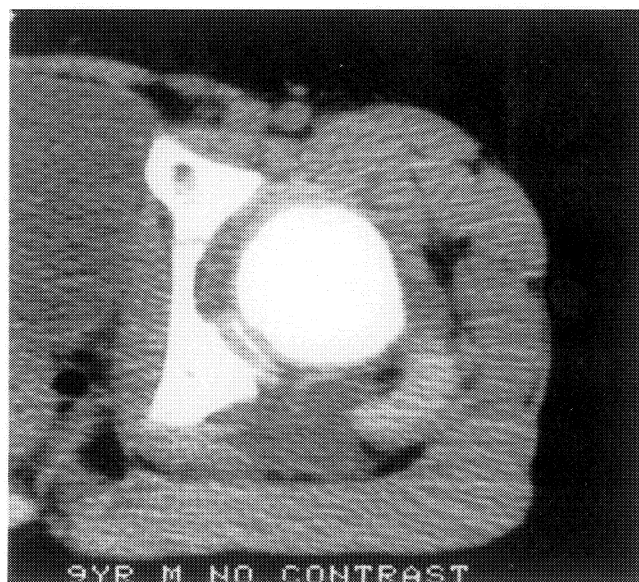


Figure 2. CT scan demonstrating fragment blocking concentric reduction of the femoral head.



Figure 3. Five month postoperative radiograph demonstrating concentric hip reduction and anchor sutures.

was identified. A bony defect in the posteroinferior acetabulum was visualized. The avulsed labrum with its associated bony fragment was reattached at its anatomic location using suture anchors (Mitek Systems, Norwood, Massachusetts) placed in the ischium and posterior column (Figures 3 and 4). The femoral head was reduced. The longitudinal capsular rent and the horizontal capsulotomy were reapproximated with non-absorbable suture. The hip was stable throughout a full range of motion without crepitus. Postoperative radiographs showed a concentric reduction.

That patient ambulated with crutches and touch weight bearing for two months. At two month follow-up, gait was normal and without discomfort. Range of motion was symmetric with the opposite hip and pain-free. The patient continued to be asymptomatic at the five month follow-up with normal gait and range of motion. Radiographs at that time did not show migration of the bone sutures or evidence of osteonecrosis. The patient continues to do well with a normal activity level one year following the injury with no hip symptoms and no evidence of osteonecrosis on radiographs.

DISCUSSION

Reasons for an irreducible posterior hip dislocation include: entrapment of the piriformis muscle, button-hole lesions of the capsule, intra-articular fragments and an inverted labrum¹. Dislocations with associated labral avulsions are rare, but have been reported in the literature⁵.

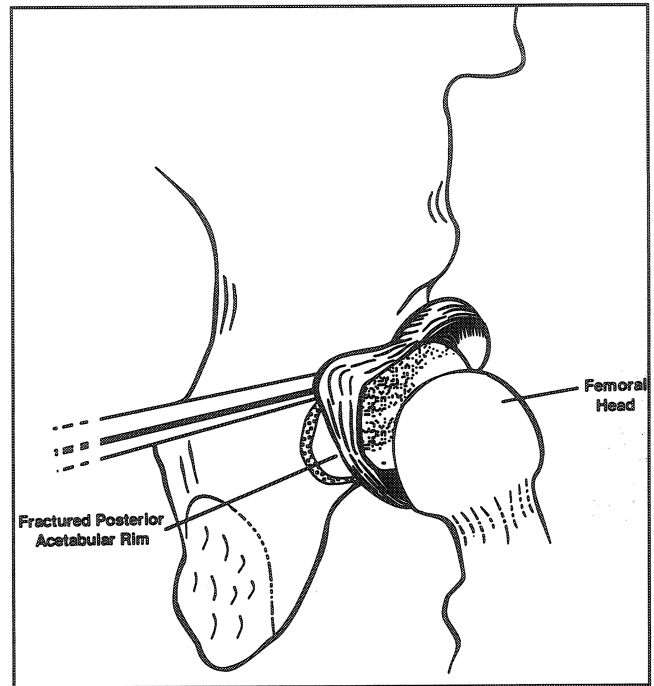


Figure 4A. Schematic diagram of reduced labral avulsion with bony fragment.

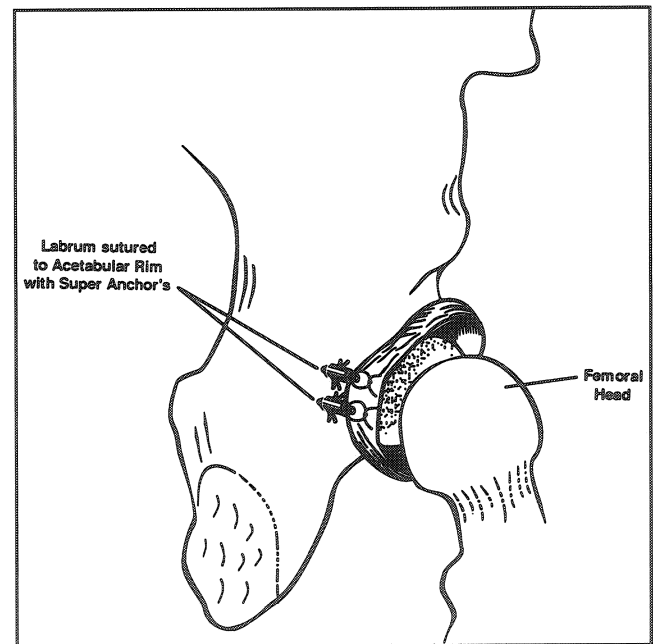


Figure 4B. Schematic diagram of anchor suture repair of the avulsed labrum.

The labrum contributes to hip stability by increasing femoral head coverage and regulating hip joint pressure⁶. Defects in the labrum can predispose to chronic hip instability. Recurrent dislocations in patients who have labral defects associated with a previous traumatic dislocation have been reported^{2,5}.

Previous repairs of labral injuries have involved the use of bone grafts to reinforce the acetabulum in the area of the defect⁵. The repair we report more closely approximates normal hip anatomy than does the use of bone grafts, and it re-establishes hip joint stability with minimal alteration in hip biomechanical loading patterns. This is analogous to the repair of bony labral avulsions in the shoulder⁴.

Suture anchors simplify this potentially difficult procedure. The need for drilling holes into the bone and articular cartilage and the passing of sutures into the deficient acetabular rim is eliminated; thereby reducing the amount of surgical exposure needed as well as any potential for sutures to pull out through the thin bony bridge. Local tissue irritation is also minimized as the suture anchors are buried in the bone³.

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PIGMENTED VILLONODULAR SYNOVITIS ARISING FROM THE SUBTALAR JOINT: A CASE REPORT

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Pigmented villonodular synovitis is a proliferative disorder of the synovium⁴. The knee and upper extremity are the most commonly involved areas. Complete excision is the treatment of choice in most cases⁵. Pigmented villonodular synovitis involving the subtalar joint is extremely uncommon, and has been rarely reported in the English literature^{6,7}.



Figure 1. Coronal fast spin echo proton density image demonstrating the lesion at the subtalar joint.

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DISCLAIMER: The opinions expressed are those of the authors and do not necessarily reflect those of the U.S. Army or the Department of Defense.

CASE REPORT

A sixteen year old male presented with mildly painful swelling over the lateral aspect of his right ankle, which he had been experiencing for several months. The pain was activity related, and improved with rest. He denied blunt or penetrating trauma to this region. His past surgical and medical histories were unremarkable, and the review of systems was negative for fevers, chills, or other systemic symptoms. The family history was noncontributory, and the social history revealed that he had been a smoker for the past two years.

The physical examination revealed a nonmobile mass over the sinus tarsi area, measuring approximately three by four centimeters. The patient did not exhibit any neurologic abnormalities, and there was no adenopathy present. The mass did not transilluminate.

Laboratory tests revealed a normal blood count and erythrocyte sedimentation rate. The plain films were normal, except for soft tissue swelling. Magnetic resonance imaging (MRI) revealed a lobular mass originating from the intracapsular region of the sinus tarsi area. The lesion's signal characteristics were similar to muscle on the T1 images, but slightly higher than muscle and much less than joint fluid on the T2 images (Figures 1, 2 and 3).

Intraoperatively, a lateral incision overlying the sinus tarsi area was made. A multilobular, rubbery, yellowish-gray mass was found arising from the synovium of the subtalar joint. A complete excision and extensive synovectomy was performed. Histology revealed giant cells, foam cells and dispersed hemosiderin deposits, all consistent with pigmented villonodular synovitis (Figure 4).

The extremity was placed in a splint until the sutures were removed. A gradual return to normal activity was allowed over the next three months. At twenty-five months postoperatively, there was no recurrence of the mass, and the patient was asymptomatic.

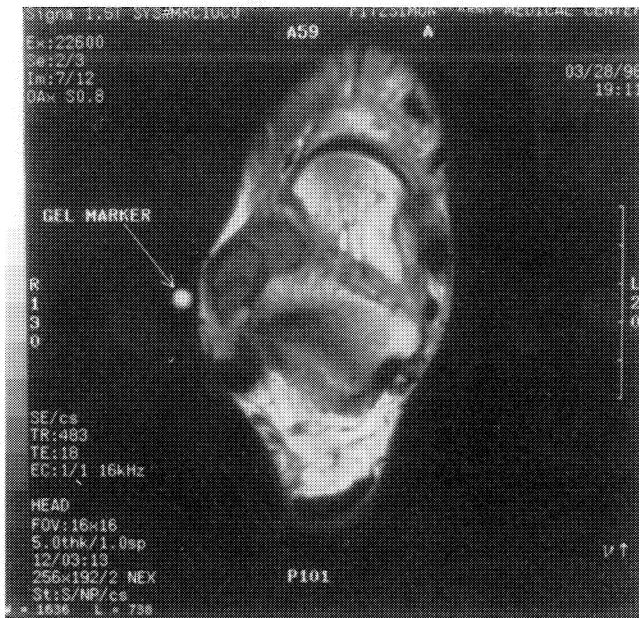


Figure 2. Axial T1 image demonstrating multinodularity.

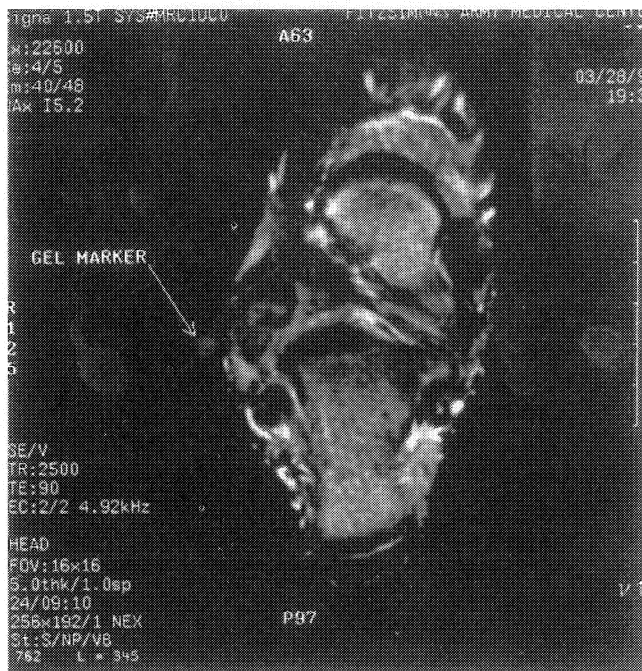


Figure 3. Axial T2 image showing low signal.

DISCUSSION

The etiology of pigmented villonodular synovitis is unclear, although trauma, hemorrhage, metabolic abnormalities, and inflammation have all been examined as potential causes². The diffuse form usually affects the knees or hips, while the localized form most com-

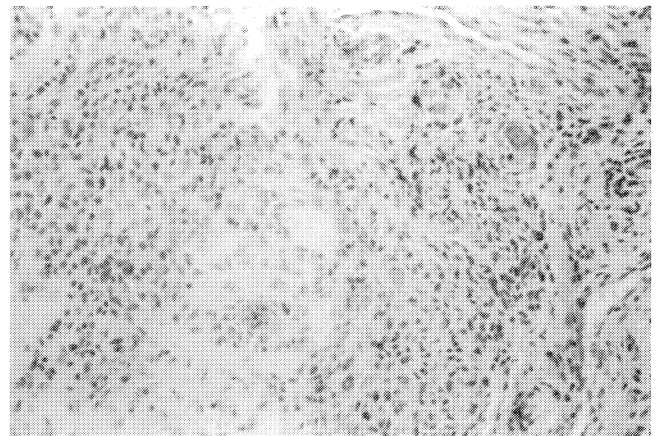


Figure 4. Histology showing giant cells, foam cells, and dispersed hemosiderin deposits.

monly affects the digital tendon sheaths¹. MR images generally demonstrate a somewhat decreased signal intensity on both T1 and T2 weighted images, particularly if there are significant hemosiderin deposits^{3,7}.

Pigmented villonodular synovitis involving the subtalar joint is extremely rare, and only two reported cases exist in the English literature^{6,7}. Rollo et al. described a case in which a patient was seen by five orthopaedic surgeons before proper diagnosis and treatment were initiated. Ugai et al. reported a patient with diffuse involvement of the subtalar joint, both medially and laterally, but the report concentrated on the MR findings. Both of these patients had erosions of the subtalar joint on imaging studies, reflecting the chronicity of the lesion. Both patients experienced symptoms for an average of 3.5 years before appropriate diagnosis and treatment were rendered.

Our patient was unique in that osseous involvement was not found on the radiographic studies or at the time of surgery. This may have been due to the relatively short time between the onset of symptoms and surgical excision.

To our knowledge, this is the first reported case of pigmented villonodular synovitis involving the subtalar joint without bony involvement. We feel that with early diagnosis and treatment, the bony erosion due to pigmented villonodular synovitis at the subtalar joint may be avoided. Although the true incidence is unknown, we believe that pigmented villonodular synovitis should be in the differential diagnosis when evaluating patients with masses in the area of the subtalar joint.

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HEREDITARY DISORDERS WITH MALADIES OF THE WRIST AND ELBOW

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We recently encountered three patients with uncommon heritable disorders who presented with elbow and/or wrist complaints. The purpose of this paper is to review the upper limb manifestations of these unusual disorders and their treatment.

CASE 1

A forty-one year old female presented with bilateral wrist pain and distal radioulnar joint (DRUJ) instability. Prominence and dorsal instability of the distal ulna was noted during forearm rotation. The DRUJ was tender to palpation and the patient's pain was reproduced with ballottement of the distal ulna. A lateral radiograph of the wrist revealed dorsal subluxation of the distal ulna (Figure 1). Treatment consisted of wrist splinting and anti-inflammatory medications.

Ehlers-Danlos syndrome is a heterogeneous group of connective tissue disorders characterized by a defect in the biogenesis of collagen. This results in a number of clinical manifestations including skin fragility and hyperextensibility, soft tissue calcifications or nodules, and joint hypermobility and dislocation^{1,15}.

Ehlers-Danlos syndrome is the most common inherited connective tissue disorder. The condition is typically first noted after one year of age and most commonly affects male Caucasians of European descent. There are numerous genotypic expressions of the disorder and as many as eleven clinical forms have been described^{1,15}.

The diagnosis of Ehlers-Danlos syndrome can be made on the basis of joint hypermobility. The following criteria have been described: passive dorsiflexion of the small finger beyond 90 degrees with the forearm flat on the table, passive opposition of the thumb to the



Figure 1. Lateral radiograph of the wrist demonstrating dorsal subluxation of the distal ulna.

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flexor aspect of the forearm, hyperextension of the elbow beyond 10 degrees, hyperextension of the knee beyond 10 degrees, and forward flexion of the trunk such that the palms of the hands rest on the floor. Patients exhibiting three or more of these findings are considered likely to have Ehlers-Danlos syndrome^{1,2,15}.

The majority of the musculoskeletal manifestations of Ehlers-Danlos syndrome are the result of joint hypermobility which occurs secondarily because of capsular and ligamentous laxity and muscle hypotonia^{2,7}. The hand and wrist are often involved. Common complaints include joint subluxation or dislocation. The most commonly involved joints include the shoulder and elbow along with the trapeziometacarpal, metacarpophalangeal, and interphalangeal joints of the thumb and digits^{2,7,11}. Recurrent dislocations and subluxations can be painful and result in persistent joint effusions and eventually disabling degenerative changes^{2,7,11}. In the digits, joint subluxation can result in a swan-neck deformity⁷.

Less common manifestations include muscle belly and tendon ruptures, prominence of the ulnar styloid, hypoplasia of the small finger proximal phalanx, a shortened fifth metacarpal, and acro-osteolysis of the distal phalanx^{2,11}.

The mainstay of treatment includes splinting and/or orthotics and anti-inflammatory medications. Arthrodesis of affected joints can be performed in severely symptomatic patients who have failed prolonged conservative treatment^{1,7,17}. Soft tissue reconstructive procedures are generally not recommended because of recurrent instability.

CASE 2

A twenty-four year old left hand dominant female presented with intermittent left wrist discomfort and paresthesias of the index and long fingers. The patient had undergone multiple surgical procedures including corrective osteotomies of the radius and ulna and bilateral open carpal tunnel releases. The patient localized her discomfort to the radiocarpal and DRUJ regions.

On physical examination, there was no obvious wrist deformity. Multiple incisions were noted. There was no evidence of thenar atrophy. Phalen's and Tinel's tests did not reproduce paresthesias. The DRUJ was stable but slightly tender to palpation with no swelling or crepitus. There was no effusion in the radiocarpal region and active and passive range of motion of the wrist was painless.

A posteroanterior (PA) radiograph of the wrist revealed premature fusion of the volar ulnar distal radial physis and a healed osteotomy of the distal ulna (Figure 2).



Figure 2. PA radiograph of the wrist demonstrating the typical features of Madelung's deformity including premature closure of the volar ulnar aspect of the distal radius, subluxation of the carpus, and an incongruous DRUJ. Note the previous osteotomy of the distal ulna.

Madelung's deformity is a dyschondroplasia of the distal radius epiphysis. The disorder most commonly affects women and is usually bilateral. Madelung's deformity is believed to result from retarded growth and premature closure of the ulnar aspect of the distal radius physis. Due to the continued growth of the physis, the distal radius articular surface angulates volarly and ulnarly with resultant compensatory wedging and volar subluxation of the proximal carpal bones. The radius appears shortened due to the curvature, resulting in apparent dorsal subluxation of the distal ulna^{4,5}.

Madelung's deformity is usually asymptomatic, although patients may present during early adolescence or adulthood with wrist pain, decreased range of motion or an unsightly appearing wrist. Physical examination reveals limited wrist dorsiflexion and forearm pronation, increased palmar flexion, and normal supination. Patients may present with degenerative changes in the wrist and/or DRUJ secondary to the incongruent articular surfaces. In addition, carpal tunnel

syndrome and secondary extensor tendon rupture are well recognized^{10,13}. Other associated upper extremity features include cubitus valgus, a hypoplastic humeral head, and an irregular, flattened radial head⁵.

Radiologically, the distal articular surface of the radius faces in an ulnar and palmar direction. At the site of the premature fusion in the distal radius, there is a characteristic "beak" with a relative translucent area. The distal ulna may be enlarged and dislocated or subluxated dorsally. The proximal carpal bones appear triangular with the lunate forming the apex as a result of volar subluxation^{4,14}.

Initial treatment of the patient with symptomatic Madelung's deformity is conservative. Splinting or casting may provide symptomatic relief. Surgical treatment is reserved for compliant patients with persistent discomfort who have failed prolonged conservative treatment. A number of surgical procedures have been described including excision of the distal ulna, an ulnar shortening osteotomy, epiphysiodesis of the distal radius and ulna, epiphysiodesis of the ulnar and palmar aspect of the distal radius, corrective osteotomy of the distal radius, and radiocarpal or DRUJ (Sauve-Kapandji procedure) arthrodesis^{10,12,16,18}.

Excision of the distal ulna is considered by many to be the preferred surgical procedure for treatment of a persistently symptomatic Madelung's wrist. While the procedure may restore pronation, it does not prevent radiocarpal articular damage and has been associated with ulnar translocation of the carpus. For severe deformities, distal excision or arthrodesis of the DRUJ combined with a corrective osteotomy of the distal radius can yield a successful outcome¹⁸.

CASE 3

A seventeen year old right hand dominant female presented with increasing right elbow and wrist pain. She noted snapping and popping at both the lateral elbow region and DRUJ. The patient maintained that she had been unable to straighten the elbow since early childhood. Over the last year, daily activities had become difficult due to progressive pain with elbow and forearm motion.

On physical examination, active and passive range of motion of the right elbow was 50 to 135 degrees. Pain and a palpable crepitus was noted at the radiocapitellar joint when the elbow was flexed beyond 90 degrees. Forearm rotation also produced pain and palpable instability of the radial head. Examination of the DRUJ revealed instability on forearm rotation. The DRUJ was tender to palpation and a dorsal prominence representing the ulnar head was noted with the forearm in pronation.



Figure 3. Lateral radiograph of the elbow in a patient with Acrofacial Dysostosis illustrating an anterior dislocation and deformity of the radial head and hypoplasia of the capitellum.

Radiographs of the elbow revealed a dislocated radial head with hypoplasia of the capitellum (Figure 3). Anteroposterior (AP) and lateral radiographs of the wrist demonstrated an incongruous DRUJ with dorsal subluxation of the distal ulna.

The chronic dislocation of the radial head and DRUJ instability were treated with anti-inflammatory medication and splints. Following three months of failed conservative therapy, a radial head resection was performed.

Acrofacial Dysostosis is a subtype of Mandibulofacial Dysostosis which presents with similar facial features to Treacher-Collins syndrome with the addition of skeletal abnormalities. These syndromes have been further categorized by their skeletal lesions as pre- or post-axial. Nager's acrofacial dysostosis typically presents with pre-axial defects while Genee-Wiedemann's or Miller's acrofacial dysostosis is most often associated with post-axial deformities. Both syndromes have well described autosomal dominant and recessive cases with variable expressivity^{6,9,11}.

The cranio-facial features include abnormalities of the external ears, atresia of the external auditory canals with bilateral conductive hearing loss, hypoplasia of the facial bones, downward slanting of the palpebral fissures with colobomata (notching) of the lower eyelids, and cleft palate.

Acrofacial dysostosis is associated with a variety of upper extremity musculoskeletal manifestations. Pre-axial defects include hypoplasia or aplasia of the thumb and/or radius. Post-axial defects include hypoplasia or

aplasia of the small finger and/or ulna. Combinations of both pre- and post-axial defects have also been described. Other upper extremity musculoskeletal conditions reported in patients with acrofacial dysostosis include radioulnar synostosis, subluxation of the elbow joint, hyperextensible joints, syndactyly, polydactyly, and clinodactyly^{6,9}.

Treatment options vary with the type and extent of deformity present. While surgical intervention is rarely necessary for the treatment of congenital radial head dislocation, excision of the radial head can relieve pain and improve function and range of motion in the severely symptomatic patient³. Derotational osteotomy can be performed for radioulnar synostosis. Index finger pollicization is recommended for the treatment of a severely hypoplastic or aplastic thumb⁸. Successful surgical procedures have been described for the treatment of syndactyly, polydactyly and clinodactyly.

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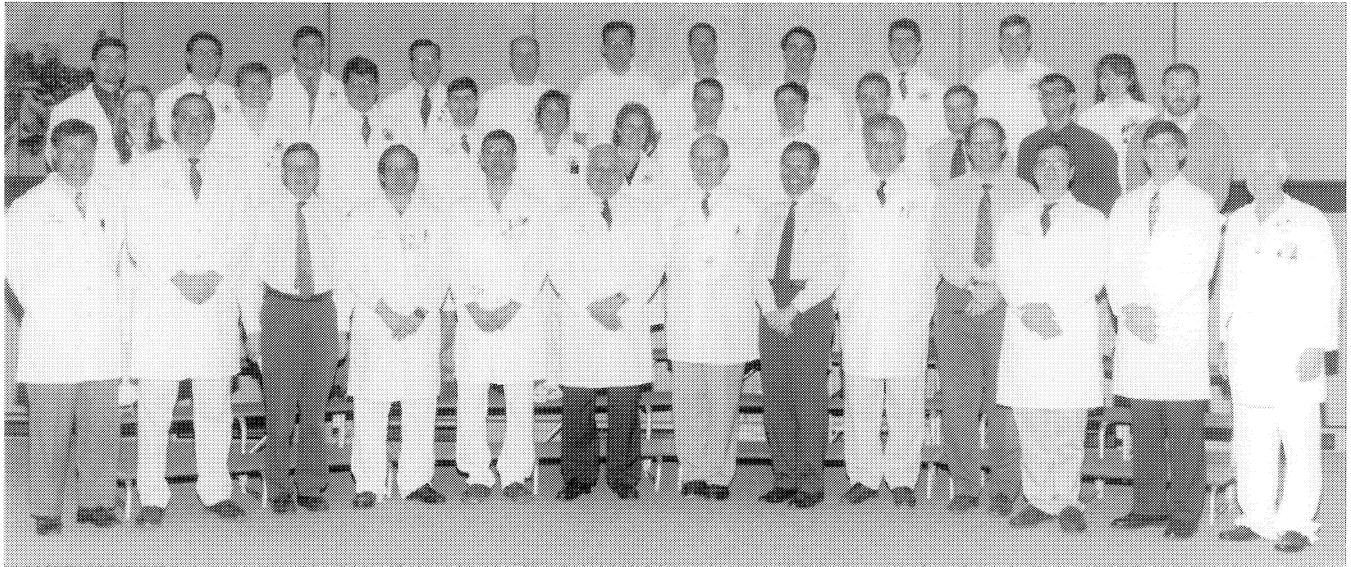
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