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Department of Orthopaedics

Arthur Steindler
1912-1949

Charles Saltzman
1991-present

John Callaghan
1990-present

David Tearse
1989-1991

Ernest Found
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Lawrence Marsh
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Curtis Steyers
1985-present

James Nepola
1984-present

Fred Dietz
1984-present

James Weinstein
1983-present

Barbara Campbell
1982-1984

Charles Clark
1980-present

William Blair
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William Pontarelli
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Joseph Buckwalter
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Stuart Weinstein
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Mike Mickelson
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Richard Brand
1974-present

Bruce Sprague
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Theodore Willis
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Adrian Flatt
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INSTRUCTIONS FOR AUTHORS

Any article relevant to orthopaedic surgery, orthopaedic science and the teaching of either will be considered by *The Iowa Orthopaedic Journal* for publication. Articles will be enthusiastically received from alumni, visitors to the department, members of the Iowa Orthopaedic Society, residents and friends of the University of Iowa Department of Orthopaedics. The journal will be published annually in May or June. The deadline for receipt of articles for the 1993 journal is February 1, 1992.

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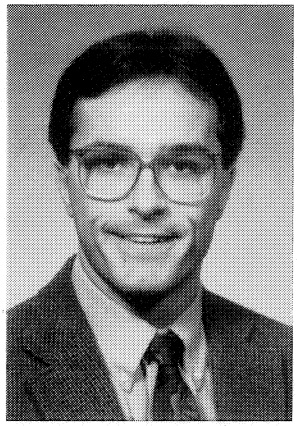
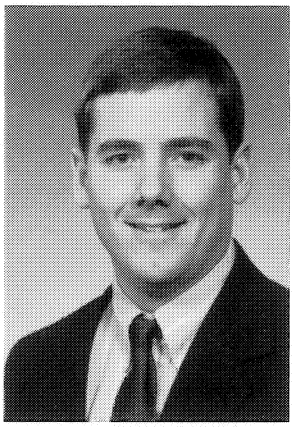
1993 GRADUATING SENIOR RESIDENTS



Brian D. Mulliken, M.D.

Brian was born and grew up in Elgin, Illinois. He received his B.S. in Biochemistry/Chemistry from the University of Illinois in 1984, and his M.D. from the Johns Hopkins University School of Medicine in 1988. His wife Debbie and he will spend a year in London, Ontario, Canada. There he will participate in a Joint Reconstructive Surgery Fellowship with Doctors Cecil Rorabeck and Robert Bouvene.

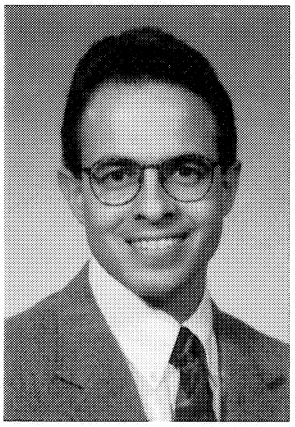


Douglas M. Cooper, M.D.

Doug was born in Pensacola, Florida on February 15, 1961. He has lived in Iowa City, Iowa since the age of four. Doug attended The University of Iowa where he received his B.S. in 1983 in General Science. He received his M.D. from The University of Iowa College of Medicine in 1988. His plans for next year include finding a general orthopaedic practice.

Joseph O. Martin, M.D.

Joe Martin was born in February, 1963, in Chicago, Illinois. Raised in Cedar Rapids, Iowa, he is the fifth of eight siblings. He attended the University of Notre Dame and received a B.S. degree in microbiology. Returning to Iowa, he graduate from the University of Iowa College of Medicine in 1988. His wife Carolyn is an associate in the Department of Obstetrics and Gynecology. They have a baby daughter, Anne, born in October of 1992. Next year he plans a year of study at the Nuffield Orthopaedic Centre in Oxford, England.



Robert L. Bass, M.D.

Rob was born in Hartford, CT on October 1, 1962. His father was a career Army Officer and therefore the family traveled extensively during his youth. Rob is a temporarily displaced Texan and considers Austin, TX his hometown. He attended the University of Texas, at Austin, and received his B.A. in 1984 in Biology. Subsequently Rob attended Southwestern Medical School at the University of Texas Medical Center, Dallas, TX, receiving his M.D. in 1988. Also, to be congratulated is Rob's wife, Sally H. Bass, M.D., and their new daughter Melanie Kate. Next year, Rob will be a Hand and Microsurgery fellow under the direction of Peter Stern, M.D., in Cincinnati, OH. Sally will be an Assistant Professor of Radiology at the University of Cincinnati Medical Center.

EDITORS' NOTE

In the past editions of the Iowa Orthopaedic Journal the editors have made a tradition of dedicating its publication to a member of our department who has greatly influenced our education. As we produce the thirteenth edition we wish to call attention to the outstanding biomechanical research and researchers at Iowa who have influenced the current practice of Orthopaedics. The ongoing legacy of the biomechanics laboratory has a daily impact on our education, as we are taught to think critically and base clinical decisions on scientific evidence.

In this year's publication we have included a short history and synopsis of the work produced by the biomechanics laboratory. It is important for the reader to realize that many other advances have been made by other members of the Department. Our attempt to focus on this particular area of orthopaedic research should in no way lessen the importance of other studies in biochemistry, retrospective analysis, morphology and other clinical research.

We wish to thank Drs. Cooper, Johnston, Brown and especially Dr. Brand for their assistance.

Kenneth J. Noonan
Lacy E. Thornburg

BIOMECHANICAL RESEARCH AT IOWA

Richard A. Brand, M.D.
Kenneth J. Noonan, M.D.
Lacy E. Thornburg, M.D.

BIOMECHANICAL RESEARCH AT IOWA

Biomechanics as a critical aspect of orthopaedic research began concurrently with the arrival of Dr. Arthur Steindler and the inception of the department in 1915. As early as 1909 Dr. Steindler had written of the potential role of muscle forces in the correction of scoliosis[1]. He brought from Vienna a tradition of mechanical treatment of disease, and a tradition of explaining disease or response to treatment by the methods of mechanics. In the early years of the department, with polio victims constituting a substantial portion of the patient population, Dr. Steindler used engineering and mathematical principles to develop surgical treatments of paralytic conditions with and without deformities[2,3].

During the 1930's, Dr. Steindler worked and studied with faculty in the College of Engineering in many areas. Most important was the application of kinematic principles to the study of normal and pathologic gait[6,7,8]. Dr. Steindler's landmark book *Kinesiology of the Human Body* was first published in 1935. Later work[12,13] led to a substantially expanded volume in 1955[14]. In this work Dr. Steindler comprehensively compiled a wealth of information from various sources largely unknown and unavailable to American readers. This text was the only reference on gait at the time, and remained so for years to come. Both editions remain treasures of historical references. Perhaps as importantly, he transmitted his enthusiasm for biomechanical studies to many of his students and staff. This resulted in studies of the patella[4], muscle transfers[5,8,10,11], and even the development of the first sterilizable electric bone saw[9].

After Dr. Steindler's retirement in 1949, enthusiasm for biomechanical studies continued under the leadership of Dr. Carroll Larson, who received his appointment as Chairman of the department in 1950. Dr. Larson began to work quite closely with Professor Royce Beckett, as well as other faculty in the College of Engineering. Dr. Larson advised several physical therapy master candidates on projects related to posture and centers of gravity[55,56]. He also worked with Dr. Imig on a paper entitled "Measurements of Peripheral Blood Flow under Conditions of Physiologic Stress." This study was the first to utilize space in Children's Hospital specifically designated for biomechanical investigations. Importantly, the collaborations established by orthopaedic faculty stimulated independent, if related, biomechanical investigations by engineering faculty[19,24,35,46]. This predated the establishment of an independent Department of Biomedical Engineering in the mid-1980's.

Dr. Adrian Flatt, owing in part to his interest in finger joint prostheses, similarly recognized the value of kinematic approaches. In 1960 he (with a grant from the Iowa Arthritis Foundation - perhaps the first external funding for biomechanical studies at the University of Iowa) established a small laboratory for studying hand function[15,22]. He analyzed metacarpophalangeal joint motion and the pathomechanics of ulnar drift[17]. Dr. Flatt, having formed a section of Hand Surgery in 1966, further developed the upper extremity lab and during the ensuing years worked with a number of residents, fellows, and graduate students on methods to analyze finger motion and loading[25,26,28]. In 1969 he finished studies which were published as a major work entitled "The Pathomechanics of Ulnar Drift"[37]. This work led to a Kappa Delta Award for outstanding research and enabled Dr. Flatt to obtain major grants from the NIH as well as the National Institute of Arthritis, Metabolism, and Digestive Disease.

Knee kinematics and the relationship of ligament injury to abnormal motion additionally interested Dr. Flatt. He published some of the earliest objective kinematic studies of knee motion[18,20,21,35]. Recognizing the limitation of kinematic analysis alone, in 1963 Dr. Flatt began to collaborate with Gary Fisher and later Don Bartel (both students in the College of Engineering) to consider not only joint motion, but the muscle forces required to produce deformity[22-26,28,29,39]. Realizing the "overdetermined" nature of the problem of calculating muscle forces*, this group was among the first to provide a solution using the engineering principles of

*Braune and Fischer, working in the late 1800's and early 1900's first recognized the "overdetermined" (i.e., mathematically indeterminate or redundant) nature of the problem of calculating joint contact and muscle forces. In this class of problems, there are more unknowns than available equations, in which case an infinite number of solutions exist. This situation applies to all joints: there are more unknown muscle, ligament, and joint contact forces than available equations of motion. Thus, a unique solution cannot be found without either simplifying assumptions or additional approaches, such as optimization. Braune and Fischer, not having optimization as a tool, provided no solution to the problem. Subsequent investigators (even to this day) most often make unrealistically simplifying assumptions, usually without justification, to obtain a solution.

optimization+[57]. These pioneering studies were first applied to the hand[25], and later applied by other departmental investigators to gait[43,46,58]. Although Dr. Steindler had a primary interest in gait during the latter part of his career, laboratory space for this study did not develop until the mid-1960's. With the encouragement of Dr. Larson, Dr. Richard Johnston began a series of investigations on gait, mostly as related to the hip[30]. Dr. Johnston did most of his work in collaboration with Gary Smidt (then a graduate student and later Professor and Past-Director of the Physical Therapy Graduate Program), Don Bartel (an engineering graduate student and now a Professor of Biomechanics at Cornell University), Edmond Chao (an engineering graduate student and currently Professor of Orthopaedics at Johns Hopkins University), and Dwight Davy (and engineering student and currently Professor of Orthopaedics and Biomechanics at Case-Western Reserve University). Space was allocated for these studies in 1966. Dr. Don Kettelkamp joined the faculty about that time and became involved in a number of projects primarily involving kinematics of the knee joint[34,43,44,47]. These projects involved a number of faculty from both engineering and physical therapy. The earliest gait studies concentrated on kinematics, and led to clinically useful information on hip and knee motion[32,33,34,36,38,40,41,42, 44,45,48]; these fundamental investigations remain to date the principal sources of information on normal hip and knee motion. Dr. Bryon Marsolais, an orthopaedic resident who also obtained a Ph.D. in mechanics, wrote his thesis on an innovative method to calculate the kinematics of the skeleton[31]. Dr. Marsolais has continued his work on gait, and currently pioneers computer-controlled muscle stimulation to induce walking patterns in paraplegics.

About the same time that Dr. Flatt and his colleagues introduced the notion of using optimization principles to calculate joint forces, Dr. Johnston and his collaborators pioneered similar approaches to calculate hip forces during gait[43,46]. These early studies on hip forces led to funding from the Veteran's Administration Hospital in 1970; major funding was obtained from NIH in 1973 and continued until 1989. Although Dr. Johnston left the faculty for private practice in Des Moines in 1970, he continued his work in the gait laboratory through the mid-1970's, visiting the laboratory every Friday. Dr. Richard Brand, current co-director of the Biomechanics Laboratory, joined the faculty in 1974. Dr. Roy Crowninshield (now President, Zimmer USA) also joined the faculty in 1975. They along with Dr. Johnston further developed techniques to calculate hip muscle and joint forces[58].

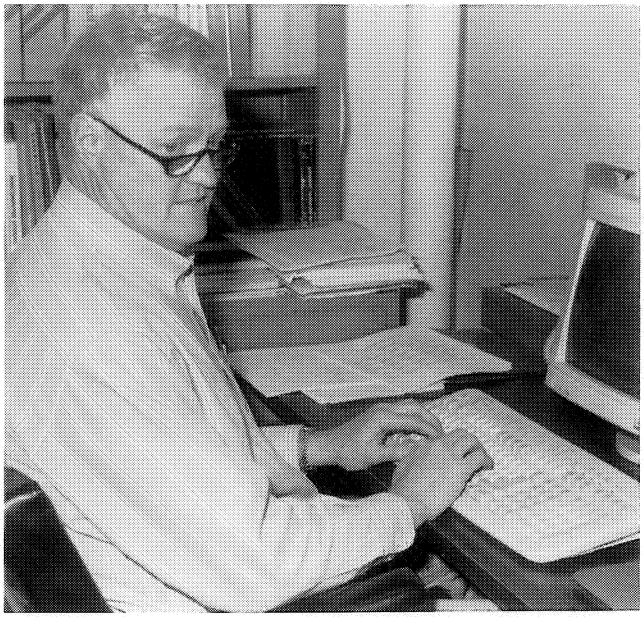


Figure 1 Ron Gabel (staff engineer) writing software for analysis of EMG recordings.

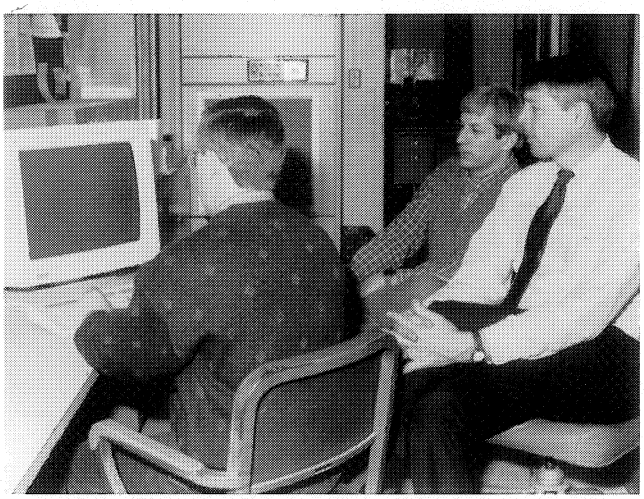


Figure 2 Reviewing finite element results for stress analysis of femoral head osteonecrosis. Left to right: Doug Pedersen (staff engineer, at keyboard), Jim Rudert (graduate student), Dr. Tom Brown.

Dr. Johnston importantly recognized that optimization principles could also be used to geometrically optimize placement of hip prostheses. Working with Don Bartel, he initially applied static optimization approaches to determine optimal cup arthroplasty placement[27,30]. These studies represent the very first attempts to improve surgery using mathematical principles. Drs. Crowninshield, Johnston, and Brand then combined gait analysis with optimization to determine ideal total hip component

+A unique solution can be obtained to an indeterminate problem by requiring the solution not only to satisfy the equations of motion, but also some more or less arbitrary "optimization criteria" or "cost function" that is an additional equation. While one more equation per se does not provide a solution, if one requires that the answer occur only when the result is at a minimum or maximum of the cost function, then a single answer can be found. Naturally, the cost function should be physiologically justified, and the answer validated.

placement[50]. This work was recognized in 1982 by a second Kappa Delta Award to the Orthopaedic Biomechanics Laboratories; it remains a seminal and often cited work regarding hip reconstruction. Dr. Brand and his colleague Doug Pedersen similarly explored the mechanical implications of hip osteotomy, determining that contrary to the results of simplified planar models, hip osteotomy does not substantially affect hip joint forces[49]. The importance of this work is reflected by an Otto Aufranc Award from the Hip Society in 1984. Since that time, Doug Pedersen has contributed invaluably to a broad spectrum of studies with many faculty members and students. Work has continued with funding from NIH toward a collaborative grant with Dr. Dwight Davy to validate the hip joint forces. Another collaborative grant with Dr. George Rab, University of California-Davis, involved hip joint forces in patients using braces for treatment of Legg-Calvé-Perthes Disease.

During the late 1970's Drs. Crowninshield, Johnston, and Brand directed attention toward the biomechanical implications of implant design. This group was among the first to pioneer the use of finite element analysis to compute stresses in biological systems. A number of studies demonstrated a major sensitivity of bone and cement stress levels to implant design. Research pointed out previously marketed devices associated with deleteriously high stress levels, and suggested approaches to minimize bone and/or cement stresses[51-54]. These studies led to scientific recognition by a second Otto Aufranc Award from the Hip Society. The very popular and successful Iowa Total Hip System was further developed from this work. Other finite element analysis led the laboratory to be one of the first to identify the stress distribution benefits of metal-backed acetabular components[55,56].

In 1983, current laboratory co-director, Dr. Tom Brown Ph.D., joined the faculty. He has many interests, but is particularly interested in experimental and theoretical stress analysis, including that in osteonecrosis of the hip and in articular cartilage. This work has attracted major NIH funding to explore stress analysis as a means of determining whether a patient with early necrosis needs surgery, and if so, how to surgically optimize treatment (coring, grafting, osteotomies etc)[68].

Dr. Brown has worked closely with several members of the department since his arrival. He collaborated with Dr. William Blair to develop new techniques in the 1980's for noninvasively determining intra-arterial pressures in small vessels using the principles of Doppler ultrasound analysis[59-61]. These approaches have been applied to determine the optimum suture methods in vessels for maintaining blood flow[62,63]. This work has been recognized by a third Kappa Delta Award for the Orthopaedic

Figure 3 Discussing MTS set-up for torsional micro-motion testing of a new cementless femoral component design. Left to right: Dr. Tom Brown; Doug Adams (graduate student); Doug Pedersen (staff engineer); Jim Rudert; Erika Skaro; Dan Fitzpatrick (graduate students).

Figure 4 Reviewing bi-planar x-rays of a cadaver preparation for kinematic evaluation of external fixator dynamization. Dr. Tom Brown, with graduate students Erika Skaro and Dan Fitzpatrick.

Biomechanics Laboratory. Working with Drs. Marsh and Nepola, Dr. Brown has pioneered and validated new approaches to estimate joint contact stresses, and then used those to determine optimal treatments of intra-articular fractures. Contrary to general wisdom, several studies have suggested that displaced intra-articular fractures do not lead to deleteriously high pressures, although they may have high pressure gradients. Drs. Brown, Nepola, and Marsh have further studied the biomechanical effects of external fixation for the treatment of long bone, intra-articular, and pelvic fractures. Along with Drs.

Nancy Hadley and Stuart Weinstein, Dr. Brown has confirmed that time is a critical factor in determining whether high pressures have a bearing on the development of osteoarthrosis[64].

Dr. Nepola and Dr. Joy Baker are currently exploring the biomechanics of fracture healing at the new Oakdale research facility. Pulsed electromagnetic field stimulation is used in a acute fracture model. External fixators are used for fracture stabilization and distraction osteogenesis. Collagen prostheses used in segmental fracture defects and vertebral disk replacements are subjected to mechanical testing after remodeling by host bone.

Since his faculty appointment in 1990, Dr. John Callaghan has brought his talent and energy to the biomechanics laboratory. His interest and outside experience have stimulated work in multiple areas of biomechanics and orthopaedics. In the experimental study of hip reconstruction, he and Dr. Brown have explored micromotion in non-cemented implants[65]. They have recently received a Frank E. Stinchfield Award from The Hip Society (1993) for their innovative work determining interfacial pressures with under-reaming uncemented acetabular components[66].

The recent addition of Dr. Charles Saltzman has stimulated the development of biomechanical studies of the foot. In some of the earliest studies, Drs. Saltzman and Brown with their students have studied the mechanical implications of sesamoid bone removal[67]. This work has recently been supported by a grant from OREF to include the problem of hallux valgus deformities.

In the mid 1980's, Dr. Brand began exploring the functional implications of neural sensors in ligaments. In collaboration with Dr. Kelly Cole (Department of Exercise Science), and with major funding from OREF and NIH, they are studying innervation and functional significance of mechanoreceptors in the cat anterior cruciate ligament (ACL)[69]. They demonstrated for the first time the presence of electrically active receptors in the ACL. Working with Dr. David Tearse (a recent faculty member with a primary interest in sports medicine) and Ron Gabel in the gait laboratory, work is being directed toward determining the functional effects of ACL deficiency in patients. This is done using innovative approaches to analyze EMG data.

Currently Drs. Brand and Brown, in collaboration with Dr. Clinton Rubin at the State University of New York, Stony Brook, are exploring Wolff's Law using complementary experimental and mathematical approaches. This work, supported by major NIH funding, intends to determine which features of stress initiate bone remodeling.

Dr. James Weinstein, in collaboration with Dr. V.J. Goel, has made many contributions to our biomechanical knowledge of the spine. They have explored load displacement behavior and stress distribution in the spine following various stabilization procedures. A finite element model of the lumbar motion segment has been developed to aid these investigations. Metal beads implanted in the spine have been used in conjunction with bi-planar roentgenograms and digitization technology by Drs. Goel, Charles Clark, Weinstein, and others to study in vivo motion.

For more than 75 years, faculty in the Department of Orthopaedic Surgery have provided enlightening investigations regarding disease origins and the implications of treatment using the science of mechanics. These contributions are from many people in addition to those mentioned above. We regret that we are unable to mention the names of the many residents, students, and post-doctoral fellows whose work is so important. The Orthopaedic Biomechanics Laboratory has provided a fruitful resource for the training of students, many of whom now occupy prominent positions in the "world" of biomechanics. Hopefully the laboratory will continue to provide rational scientific basis for the treatment of difficult musculoskeletal problems.

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NOTE OF APPRECIATION, CONNIE V. PIERSON

Connie V. Pierson

The authors of this years Journal would like to recognize Connie V. Pierson prior to his retirement from the V.A. Medical Center.

Mr. Pierson has a long history of government service dating back to his induction into the Army in 1957. Following tours in the United States, Japan, and during the Vietnam War, Mr. Pierson began work at the Iowa City V.A. Medical Center in 1971. For over twenty years Mr. Pierson has overseen the daily function of the orthopaedic clinic and service. These duties have involved organization of clinic patients and appointments, maintenance of the cast room, cast and splint application, surgical assistance, and general patient care.

To the V.A. Medical Center Mr. Pierson will always be remembered for his organizational skills, efficiency, empathy for patients, and his willingness to perform any duties.

To the two decades of orthopaedic residents past and present, Mr. Pierson will be remembered for his effort, his jokes, his cookie cupboard, and his supreme ability to keep the V.A. administrative red-tape at bay. Only after his departure will his skills and contributions be truly appreciated. Yet most of all, the residents will remember Connie for his friendship; it will be missed.

Kenneth J. Noonan
Lacy E. Thornburg

WHY HAVE WE LEFT CHARNLEY LOW FRICTION ARTHROPLASTY?

Devon D. Goetz, M.D.*
William H. Harris, M.D.**

In November of 1962, Sir John Charnley initiated a prospective study to establish the results of low friction arthroplasty of the hip using a high molecular weight polyethylene acetabular component. Although the sepsis rate in the early days of the operation was high (8.9 per cent), the clinical results in the remaining patients after ten years of follow-up and longer were reported as excellent. These procedures were performed through a lateral approach using a trochanteric osteotomy and contemporary standard Charnley prosthetic components: a high molecular weight polyethylene (HMWPE) acetabular component, a stainless-steel femoral component with a twenty-two millimeter diameter head, and polymethylmethacrylate (PMMA) cement inserted with first generation cementing techniques (see Table 1)[20]. Wroblewski has maintained follow-up of these patients, and has reported continued excellent results[105,106].

Table 1
Evolution of Modern Cemented Total Hip Arthroplasty

1 a)	First Generation Cement Technique: Hand-packed (No plug, no gun)	Prior to 1976
b)	Prostheses with sharp corners made of cast metals. (No super-alloy prostheses)	
2 a)	Second Generation Cement Technique: Canal plug, Cement gun	1976-1983
b)	Stem with broad medial border and no sharp corners, frequently collared, made of super-alloy metals.	
3 a)	Third Generation Cement Technique: Porosity reduction with centrifuge or vacuum mixing Pulsatile lavage Cement pressurizer Precoated prosthesis with PMMA	since 1983
b)	Rough surfaced femoral component.	
c)	Centralizer.	
d)	Adrenaline-soaked sponges for femoral canal.	

Since the time of Charnley's initial reports, interest in improving his results has been enormous, and literally hundreds of modifications in the original procedure have been attempted. A review of the advertisement section of any orthopaedic journal will reveal that the number and variety of prosthetic components currently available is enormous as well.

Recently, minimum twenty-year follow-up results from a single surgeon using the Charnley prosthesis inserted with first generation cementing techniques have become available. In a series of 330 primary total hip arthroplasties (THA) performed by Richard Johnston in Iowa, Schulte et al. reported a 5.7 per cent total incidence of acetabular revision for aseptic loosening, and a 2.5 per cent total incidence of femoral revision for aseptic loosening. One femoral component had fractured and the incidence of osteolysis was very low. Only 3.3 per cent of cases showed acetabular lysis and another 3.3 per cent showed femoral lysis[94].

A number of recent studies reporting results of THA using newer prosthetic designs inserted with newer surgical techniques document unquestionably *worse* results at *shorter* follow-up than those noted above. This discrepancy raises an important question: why have we left Charnley low friction arthroplasty?

To answer this question, it is important to realize that the majority of surgeons have *not* been able to achieve these excellent results using the Charnley prosthesis with first generation techniques. Thus, Dr. Johnston's results merely demonstrate what *can* be achieved using early techniques. Although initial inspection of Charnley's own results suggests that they were equally good, more critical analysis of the data reveals that there were clearly problems. In the ten year prospective study, Charnley reported a 2 per cent incidence of acetabular loosening and no cases of femoral loosening. At an average 13.2 year follow-up, the results were again reported as excellent, with 1.5 per cent of cases denoted clinical failures, due to loosening of cement. In greater than 10,000 operations performed subsequent to the prospective series, the total revision rate for loose prostheses was reported as only 0.21 per cent at two to twelve years[20]. It is dangerous to compare these data to current reports for several reasons. First of all, it is important to realize that in the years since Charnley evaluated his first series of patients, investigators have enhanced our understanding of femoral component loosening. Prosthesis-cement debonding is commonly accepted as evidence of femoral loosening today, yet there is no mention of this phenomenon in Charnley's early reports. We now know that debonding is common

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even after short-term follow-up of Charnley low friction arthroplasty; for example, 4.5 per cent of cases debonded at the one-year follow-up in the Mayo clinic series[11]. In fact, neither of two initial follow-up studies by Eftaker (1971) and Charnley and Cupic (1973) mention any definition of radiographic loosening[21,39]. Also of concern in a subsequent follow-up reported by DeLee and Charnley in 1976, nearly 10 per cent of acetabular components had migrated, while another 60 per cent had radiographic demarcation at the bone-cement interface[35]. This increasing acetabular loosening rate was probably largely related to improved radiographic interpretation, rather than just an acceleration in the acetabular loosening rate. Although Charnley knew of only twenty-three revisions after 10,913 operations performed at Wrightington over a ten-year period, it is not clear what portion of cases in this large group were actually seen in follow-up. In other words, only direct information regarding the fate of a patient's operation should be used in calculating the revision rate. Finally, the long-term results reported by Wroblewski in Charnley's patients noted that 53.5 per cent of sockets and 29 per cent of the stems were radiographically loose at an average 16.6 years. In addition, there was osteolysis around 20 per cent of the femoral components[105]. Unfortunately only 2 per cent of the original cases were available in the final follow-up study[106]. Thus it appears that many surgeons using the Charnley system were unable to obtain the excellent long term results that Dr. Johnston has been able to obtain.

Review of the literature suggests that there are three phenomena that stimulated total hip surgeons to make a move away from Charnley low friction arthroplasty. The first was increasing rates of femoral and acetabular loosening in series of Charnley and Mueller cemented prostheses inserted with first generation cementing techniques[96,98]. The second was the recognition of periprosthetic bone destruction, known as osteolysis[50,56,101]. This process was attributed to the presence of cement, and was later incorrectly called "cement disease"[60]. The third and possibly most prominent phenomenon was the high long term failure rate reported with the use of cemented fixation in young patients[19,24,37,38]. These concerns led investigators to seek various avenues of change. Conceptually, progress in total hip arthroplasty was three-fold: while some chose to "improve upon" the techniques of Charnley by improving cementing techniques (and modifying cemented prostheses), others chose to "move away from" his techniques completely by eliminating cement or attempting surface replacement. While surface replacement has been largely abandoned, modern surgeons are still faced with the dilemma of choosing cemented versus cementless fixation for their patients.

Given the excellent long-term results reported by Dr. Johnston using the Charnley prosthesis inserted with first generation cement techniques, each of us must examine our own techniques to discern whether the procedures we currently perform are superior to those of the past. The authors are convinced that the progress in THA over the past thirty years has been both substantial and beneficial. While there has been important progress in many areas of THA including prevention of sepsis and thromboembolism, this discussion will deal only with general issues regarding choice of component design and fixation. We will substantiate our current recommendation for both revision and primary THA; that is, hybrid total hip arthroplasty using a cemented femoral component and a cementless acetabular component.

Femoral Component Fixation

Over the last thirteen years femoral component fixation in THA has largely evolved into two separate and popular forms—cemented versus uncemented fixation. Those surgeons who chose uncemented fixation hope that this leads to lower femoral osteolysis rates and improved component longevity, especially in the young patient.

The concern for compromised longevity with cemented THA arose with the well-known reports from the Mayo Clinic and Cleveland Clinic in 1982. Stauffer, reporting on a multi-surgeon series of 231 Charnley hips with first generation cement techniques from the Mayo Clinic, noted that 29.9 per cent of the femoral components were radiographically loose at ten years follow-up. In addition, over 6 per cent of femoral components were revised for aseptic loosening[96]. Sutherland's report on one hundred cases using the Mueller prosthesis caused even more concern, with a total of 40 per cent of the femoral components considered loose (either revised or migrated)[98].

Since the time that these procedures were performed, there have been major improvements in femoral cementing techniques. At an average eleven year follow-up reported from the Massachusetts General Hospital using second generation cementing techniques, the total femoral loosening rate (definitely loose plus revision for loosening) was only 3 per cent, while the femoral revision rate for aseptic loosening was only 2 per cent[73]. Compared to the series of Stauffer and Sutherland, which reported results using first generation techniques with similar follow-up durations, these results were statistically significantly improved. These findings were corroborated by two other institutions that reported improved results with improved cementing techniques *within the same* institution. At the Mayo Clinic the five year definite femoral loosening rate was 24 per cent using first generation techniques and 1.2 per cent using second generation techniques. The femoral revision rate for aseptic loosening decreased as well, from

2 per cent using first generation techniques, to 0 per cent using second generation techniques[11,90]. In a retrospective matched pair study at the Brigham and Women's Hospital, the femoral loosening rates were significantly improved with second generation cementing as well, with 21 per cent of femoral components considered definitely loose in the early cementing group, and none considered definitely loose in the late cementing group[89].

The subsequent fifteen year follow-up results of second generation femoral cementing techniques at the Massachusetts General Hospital are now available. In 161 hips undergoing primary cemented total hip arthroplasty, the revision rate for aseptic femoral loosening was only 1.2 per cent, and in the 102 hips in patients still surviving at an average 15.2 years, there were no femoral revisions for aseptic loosening. The average age in the entire population was 61 years at the time of surgery, while the average age in the surviving patients was only 57 years at the time of surgery[74]. Given the extremely low revision rates reported at this long term follow-up, is there any reason to improve upon these results with improved cementing techniques? The answer to this question lies in the details. The average age in the Iowa series at the time of surgery was approximately 67, and only 30 per cent of patients were still alive at the time of twenty year follow-up[94]. While the noted results remain superb for these survivors, increased life expectancy is an important reason for attempting to *improve upon* the first generation Charnley techniques. Currently, the life expectancy of a healthy 75 year old person is approximately 15 years, and 60 per cent of 65 year olds will live more than 20 years. Clearly, the trend in THA is to perform these procedures in younger and younger patients. Thus, we must attempt to improve upon these excellent results.

Another pertinent detail is in the interpretation of radiographic information when evaluating the results of THA. When analyzing the Mayo Clinic ten-year results reported by Stauffer, the radiographic failure rate is in large part based on the finding of radiolucencies at the bone-cement interface[96]. This finding is no longer of primary importance, as the initiation of failure in cemented femoral components has been shown to be debonding at the prosthesis-cement interface[58,59,66]. In the Iowa study, almost 40 per cent of femoral components had a radiolucent line at the prosthesis-cement interface[94]. These debonded prostheses would be considered loose by our current criteria. This compared to a 7 per cent incidence of debonding in the fifteen-year follow-up series of second generation techniques from the Massachusetts General Hospital[73]. We agree with Charnley, who maintained that radiographs could be used to predict clinical failures[20]. While the natural history of femoral component debonding has not yet been elucidated in clinical follow-up studies, the available basic science studies suggest that the longevity of a debonded femoral component is compromised[1,5,29,34,48,49,51,84,85,100].

The benefits of contemporary femoral cementing techniques are even more dramatic in the young patient. In a long term follow-up study of 50 THA inserted with second generation cementing techniques in patients aged 50 years and under, the femoral revision rate for aseptic loosening was zero, and only 2 per cent of femoral components were considered radiographically loose[6]. This is compared to femoral revision rates of 17 to 27 per cent reported after long term follow-up of cemented THA in young patients in other studies[19,24,37].

The benefits of contemporary femoral cementing techniques in revision surgery are equally dramatic. Femoral loosening rates of 30 to 40 per cent and femoral rerevision rates of around 10 per cent have been reported after four to eight year follow-up of revision THA performed with first generation femoral cementing techniques[61,81,82]. In contrast, a recent twelve year follow-up of femoral revisions performed with second generation techniques noted only a 20 per cent incidence of femoral loosening, and a similar 10 per cent rerevision rate[42].

Using contemporary cementing techniques, the surgeon now has the ability to positively influence the outcome of THA. Using a medullary cement plug, cement gun, and a cement pressurizer, the distribution of cement into the periprosthetic bone is substantially improved[8,13,42,73,75,76,77,89,90]. Porosity reduction with a centrifuge or vacuum-mixing has been shown to increase the fatigue life of bone cement by at least a factor of five[14,33,102]. Femoral canal preparation has been improved by the use of pulsatile lavage and adrenaline soaked sponges, and centralizers enhance the surgeon's ability to obtain a uniform cement mantel with a minimum 2 millimeter thickness. Finally, the strength of the cement-prosthesis interface can be substantially improved by the use of roughened metal surfaces and PMMA precoating[1,5,29,34,48,49,51,84,85,100]. Given the unpredictable natural history of the debonded femoral component and the increasing use of THA in young patients, we believe that it is imperative to use these second and third generation cementing techniques.

Rather than pursuing cemented femoral fixation with improved techniques, a large population of surgeons have chosen to move away from the principles of Charnley and use uncemented femoral fixation. The longevity of uncemented femoral fixation remains unknown. Short and intermediate-term follow-up results of uncemented designs from United States centers are now becoming available[15]. In a prospective series reporting six year follow-up of 100 consecutive hips treated with PCA femoral component, the femoral revision rate was 1 per cent,

Table 2
LYSIS AROUND UNCEMENTED FEMORAL COMPONENTS

PROSTHESIS	*AUTHOR/SOURCE*		*LYSIS INCIDENCE*	*AVERAGE FOLLOW-UP*
AML	OH et al (78)	1992 AAOS	56%	6.5 YRS
	BEAUCHESNE et al (10)	1992 AAOS	22%	6.0 YRS
APR	COX et al (27)	1992 AAOS	16%	5.0 YRS
HGP	SMITH & HARRIS (95)		31%	4.5 YRS
	MARTEL et al (70)	1991 AAOS	7.5% stable implants	5.5 YRS
PCA	OH et al (78)	1992 AAOS	37%	5.5 YRS
	CRUTCHER et al (31)	1991 AAOS	26%	5.9 YRS
	STULBERG et al (97)	1992 AAOS	19%	5-7 YRS
	HEEKIN et al (55)	1993 JBJS	18%	6.0 YRS
			7% stable implants	

and the femoral subsidence rate was 5 per cent[55]. In another series reporting at a minimum two year follow-up of 118 hips using the same prosthesis, the femoral revision rate was 8 per cent and the femoral subsidence rate was 8 per cent[54]. Many others have reported concerning results with the PCA femoral component[16,37,67]. Likewise, a series of 132 hips reporting the use of the HGP system after a minimum three year follow-up noted that 4 per cent of the hips had been revised for femoral loosening, and 9 per cent of the hips had radiographic signs of femoral loosening[69]. One must realize when interpreting these data that while poor results after short-term follow-up are clearly an ominous sign, good results in series with less than a five year follow-up are completely nondiscriminatory. Almost all prostheses appear to be functioning well after two years; it is only with long term follow-up that the results of THA become discriminatory. Currently there are no published minimum ten-year follow-up studies of uncemented total hip arthroplasty from U.S. centers. Likewise, even though uncemented femoral fixation is widely recommended for revision surgery and for young patients, there are no series available that report comparable results to those noted previously using contemporary cemented femoral fixation.

Of additional importance when comparing cemented and uncemented femoral fixation is that almost all series reporting the results of uncemented femoral fixation have noted an average twenty percent incidence of thigh pain[8,16,32,36,40,47,54,55,62,65,67,80,103].

While the long term stability of uncemented femoral fixation remains unknown, recent five to seven year follow-up reports have highlighted an issue of even greater concern. Several recent series reporting the results of uncemented fixation using a variety of prostheses have reported alarming rates of femoral osteolysis (see Table 2)[10,27,31,55,70,78,95,97]. At five to seven years follow-up the incidence of osteolysis around uncemented femoral components is from 7 to 56 per cent. This is in marked contrast to the reported incidence around cemented femoral components inserted with contemporary techniques. In an eleven year follow-up study from our institution using second generation cementing techniques, the incidence of major femoral osteolysis was zero, and the incidence of focal femoral lysis around stable implants was 7 per cent[73]. These data suggest that the incidence of femoral lysis is significantly greater in uncemented fixation when compared to cemented fixation using contemporary techniques. However, such a comparison could be biased by any of multiple variables. We have therefore recently performed a retrospective matched-pair study comparing the incidence of femoral lysis associated with uncemented versus cemented femoral fixation. All cases were matched for age, gender, weight, diagnosis, and duration of follow-up. All procedures were performed by the same surgeon using the same uncemented acetabular component and the same chrome-cobalt femoral head. At an average six year follow-up, the incidence of femoral osteolysis in the forty-one hips that received an uncemented femoral component (29 per cent) was greater than the incidence in the forty-one hips that received a cemented femoral component (zero) ($p < .0002$)[46]. This is in contradistinction to earlier concerns for osteolysis around cemented femoral components, which ironically led many to chose uncemented femoral fixation.

Femoral Component Material

The original Charnley femoral prosthesis was manufactured by forgings of 316 or 316L-type stainless steel, followed by annealing. Subsequent biomechanical testing has shown that the yield strengths of fully annealed 316L stainless steel and cast stainless steel are very low, and are even within the expected range of stresses that are applied to an implanted stem. Femoral component fracture was a significant concern following reported rates of 0.23 to 11 per cent in series evaluating the Charnley and Charnley-Mueller prostheses. There are many variables

that might contribute to an increased incidence of femoral component fracture, including increased patient weight, increased patient activity, femoral component loosening, lack of support at the calcar femorale, varus stem position, and defects in the component itself. However, despite the continued presence of these variables, the incidence of fracture with newer components is extremely low. The newer biocompatible materials, including cold-worked 316L stainless steel, forged cobalt-chromium, and Ti6Al4V alloys, have substantially improved fatigue strengths[44]. Superalloys provide an additional advantage in contemporary femoral cementing; they can be made with smaller component diameters which allows a minimum two millimeter cement mantle around the femoral component in essentially all patients.

Femoral Component Shape

The early Charnley femoral components were available in three different shapes: the first generation or flat-back, the second generation or round-back, and the third generation or flanged design (Cobra). By the time his book Low Friction Arthroplasty of the Hip was published in 1979, Charnley was using predominately the largest Cobra designs, and less often the largest round-back designs. The flat-back prosthesis was no longer used[20]. Those who chose the popular Mueller prosthesis were faced with inferior clinical results. The curved or banana-shaped component was diamond-shaped in cross-section, and has been associated with high loosening rates[25,83,98] as well as a high incidence of femoral component fractures[23,45,91]. It was thus empirically learned that sharp corners, especially along the proximal-medial and distal-lateral surfaces of the prosthesis, were to be avoided. Three-dimensional finite element analysis of a variety of stem shapes demonstrated a two-fold difference in peak tensile cement stress when comparing a modeled component with sharp medial and lateral borders to one with rounded borders[30]. Modern cemented femoral component designs tend to reflect the general shape of the Charnley round-back prosthesis.

Femoral Component Modularity

The Charnley femoral component was nonmodular or monobloc, as were all femoral components of that era. Modular head and neck assemblies became popular in the 1980s, and now dominate the field of available femoral prosthesis designs. This option offers advantages in terms of inventory reduction and fine tuning of soft tissue tensioning and leg length at the time of surgery. However, a number of real concerns regarding femoral component modularity have arisen. Fatigue failure at the head-neck junction has been reported[72], and metal-metal fretting and corrosion at the Morse taper interface is a potential source of intraarticular debris[22]. In addition, the enlarged metal "skirts" associated with long-neck components have been shown to reduce maximum range of motion, increasing the potential for impingement[7]. Recently, investigators have documented actual migration of metallic debris from the Morse taper to the metal-polyethylene articulation, suggesting that this interface is a potential source of particles which could contribute to free body wear of the polyethylene liner[57]. Finally, there have been documented complications relating to the use of modular implants such as the insertion of trial components, mismatching of the modular components, postoperative detachment of modular heads, and dislodging of modular liners[7]. At this time it remains unclear whether or not femoral component modularity is a real improvement upon the original Charnley prosthesis.

Femoral Component Head Size

Charnley chose a small head size (22.25 millimeters) for his femoral component because he felt that the combination of a small prosthetic femoral head with a large socket would lower the torque on the socket and its associated interfaces[20]. Those who chose to move away from Charnley's principles recommended larger head sizes because of theoretical advantages of increased range of motion and lower dislocation rates. A number of popular implants used a 32 millimeter femoral head, including the cemented Mueller, the uncemented Anatomic Medullary Locking, and Porous Coated Anatomic prostheses. However, clinical studies have shown that the postoperative range of motion is essentially the same after arthroplasties using 22, 28, and 32 millimeter head diameters[2,11,21,26,86,87,98]. In addition, an analysis of over 10,000 THA at the Mayo Clinic revealed no clinical evidence that larger femoral heads were more stable than smaller ones; other investigators have confirmed this experience[17,39,63,86,87,104]. Furthermore, the larger 32 millimeter head is typically associated with a thinner polyethylene implant, which has theoretical disadvantages in terms of force distribution and polyethylene wear. Currently, mid-size femoral heads (26 or 28 millimeter) are recommended; the 22 millimeter head size has been associated with greater linear wear and the 32 millimeter head has been associated with higher volumetric wear[64].

Acetabular Component Fixation

The Charnley acetabular socket was a hemispherical component made entirely of HMWPE. Initially, there was only one design and two sizes; subsequently, multiple sizes and designs became available. By 1978 he was using primarily a long-posterior-wall (LPW) configuration that included a special pressure injection (PIJ) flange around its periphery; this was designed to aid in pressurization of the acetabular cement. All of his sockets had a semicircular

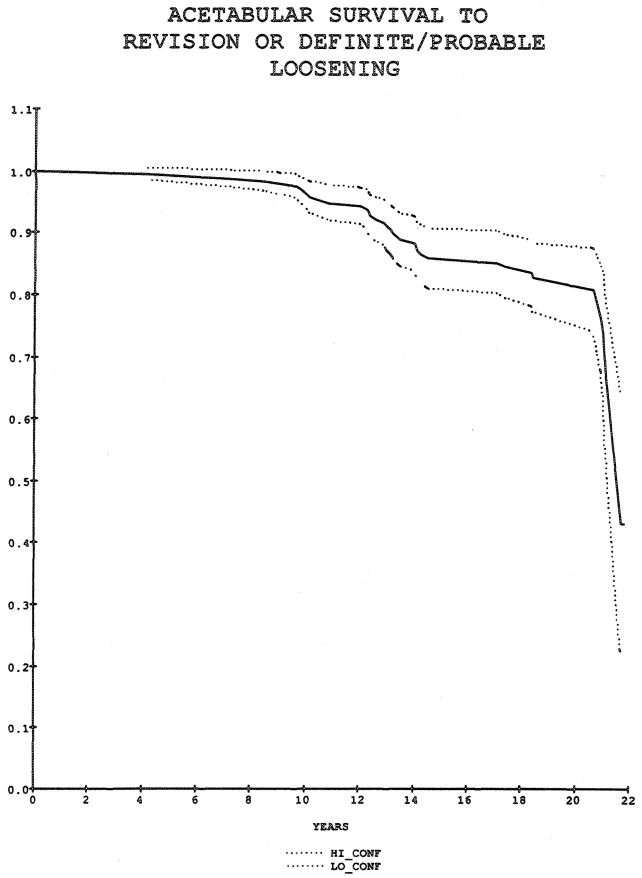

Figure 1 Twenty year follow-up of Charnley THA. Graph demonstrates survivorship of acetabular components. (Reprinted with permission, American Journal of Bone and Joint Surgery).

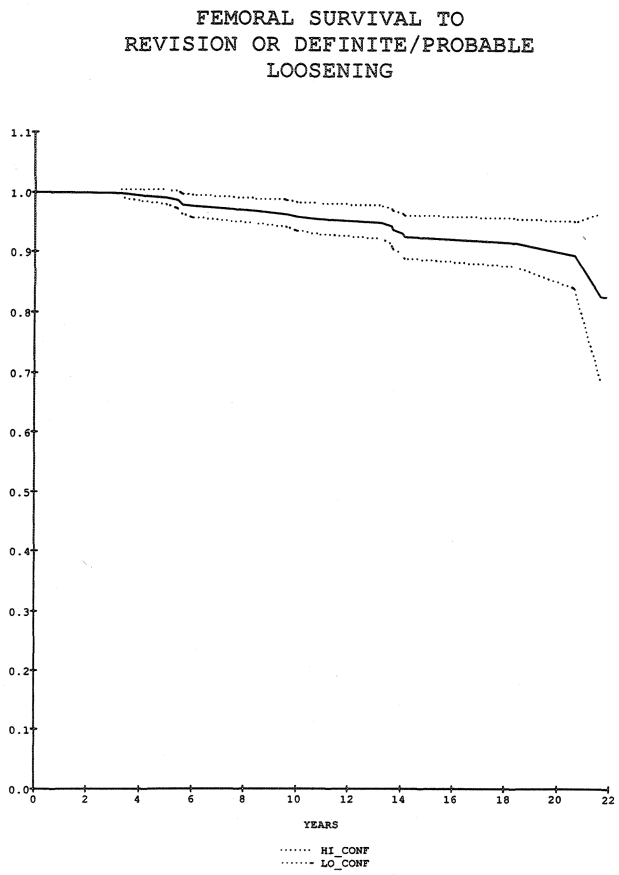

Figure 2 Twenty year follow-up of Charnley THA. Graph demonstrates survivorship of femoral components. (Reprinted with permission, American Journal of Bone and Joint Surgery).

wire marker that was to be oriented in the coronal plane on insertion; follow-up radiographs could then be analyzed to measure wear[20]. Initial results using this component with early cementing techniques were reported as excellent, with 1.4% of component migration at both 7-8 years and 9-10 years respectively[21,39]. However, with follow-up of greater than ten years, Charnley noted that the incidence of acetabular migration rose to 9.2% after ten years[35] and to 12.5% after fourteen years[20]. Likewise, in the ten year follow-up reported by DeLee, it was noted that nearly 70 percent of acetabular components were problematic (60.3 per cent demarcated, 9.2 per cent migrated)[35].

Other authors have reiterated Charnley's concern for an increasing acetabular failure rate with long-term follow-up. In the twenty year follow-up from Iowa, using early cementing techniques, the acetabular loosening rate (radiographic loosening plus revision) was 23.4 per cent in those patients living twenty years. This is in contrast to a femoral loosening rate in the same patients of 7.5 per cent (See Figures 1 and 2)[94]. Unfortunately, "improved" acetabular cementing techniques have not proven to be a solution to this problem. In an eleven year follow-up from the Massachusetts General Hospital, the incidence of radiographic loosening on the acetabular side was 42 per cent, while only 3 per cent of femoral components were noted to be definitely loose. This represented a twenty-fold increase in the incidence of acetabular loosening between five and eleven years[73]. Subsequent follow-up of these patients at an average of fifteen years revealed that the acetabular loosening rate was over seven times the femoral loosening and debonding rate[74]. The results in young patients from the same institution were similar: at an average twelve year follow-up in patients 50 years of age or younger, the acetabular loosening rate (revision for aseptic loosening plus radiographic loosening) was 44 per cent, compared to only 2 per cent on the femoral side[6]. Thus, long-term follow-up studies suggest that modifications in acetabular cementing techniques have not improved upon the results of early Charnley socket fixation.

Another attempt to improve upon the results of cemented socket fixation was with metal-backing of the acetabular component. A number of biomechanical studies noted lower cement stresses with the addition of metal-backing[9,18,79], and the early clinical results of this modifi-

cation were also promising[51,71]. However, more recent long term follow-up studies have demonstrated no obvious advantage with the use of metal-backed acetabular components[50,73,88].

Currently, most surgeons are utilizing uncemented fixation on the acetabular side for the majority of their patients. Early attempts at uncemented acetabular fixation were distinct failures. Charnley himself tried a nonporous press-fit socket in almost 300 patients between 1963 and 1965. In his 12 to 15 year follow-up study, greater than 35 per cent (three times the rate in his cemented sockets) of these sockets had migrated[20]. The results of acetabular reconstruction using smooth threaded acetabular components have been poor as well; Engh et al. reported that at a mean 3.9 year follow-up, 21 per cent of patients with this component had radiographic signs of instability, and 25 per cent were symptomatic[4,41]. On the other hand, the short-term results of reconstruction using porous acetabular components are more promising. A recent series reporting results of the HG porous-coated acetabular component with screws noted no revisions for component loosening and no evidence of radiographic loosening at an average of 5.7 years follow-up[92]. The clinical and radiographic results using the same component inserted press-fit without screws were also excellent after an average five years follow-up[93]. Acetabular revision has been noted to be technically easier using uncemented components, and the short-term results have proven superior to those of cemented acetabular revision[99].

Conclusion

A recent single-surgeon series has established excellent clinical results at twenty-years using cemented Charnley low friction arthroplasty. Despite this, many total hip surgeons have chosen to move away from Charnley's principles in favor of more modern designs and techniques. Currently, there are growing concerns regarding many features of modern total hip implants, including component modularity, large femoral head sizes, and osteolysis around uncemented femoral components. Those who have chosen to follow these more recent trends have frequently met with failure. On the other hand, there have been substantial advances in THA introduced by Charnley, especially in the areas of component materials, cementing techniques, and infection prophylaxis.

More than thirty years have passed since the introduction of THA, and there are literally hundreds of different prosthetic designs available, several options for prosthetic fixation, and several ways to perform the procedure. Now that the long-term results of cemented Charnley low friction arthroplasty are available, each surgeon must critically analyze the procedures that they are choosing for their patients. Using the Charnley procedure as the standard, surgeons must be able to justify those portions of their techniques that differ greatly from Charnley's. Given the excellent long-term results of cemented femoral fixation combined with the increasing incidence of osteolysis around cementless femoral components, the current procedure of choice on the femoral side is third-generation cement fixation. In contrast, the long-term results of cemented acetabular fixation have proven to be suboptimal, while the short-term results of cementless acetabular fixation are very promising. Based on these observations, we currently recommend hybrid arthroplasty for both primary and revision cases. It is our belief that long-term follow-up will demonstrate this to be the appropriate choice for our patients.

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DRUG TREATMENT OF SOFT TISSUE INJURIES EFFICACY AND TISSUE EFFECTS

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INTRODUCTION

Acute and chronic soft tissue injuries occur far more commonly than fractures. Accidents, physical labor, and sports frequently cause sprains, strains, contusions, and overuse syndromes of tendons, muscles, musculotendonous junctions, ligaments, and synovial joints. Although the soft tissue injuries that result from demanding labor and sports receive the most attention, the same types of problems can result from participation in recreation and fitness programs, or even more modest physical activity.

Few soft tissue injuries resulting from minor accidents, sports, or physical labor require surgical intervention. Ideally, partial suppression of inflammation caused by injury reduces secondary tissue damage due to release of degradative enzymes and other events during inflammation. Suppression might also limit disuse changes in the tissues, allow earlier rehabilitation by decreasing pain and swelling, and accelerate healing by shortening the duration of acute inflammation. Therefore, when patients seek treatment for the pain and impaired function of soft tissue injuries, physicians commonly prescribe modification of activity and anti-inflammatory medications; primarily oral non-steroidal anti-inflammatory drugs (NSAID's) and injectable corticosteroids. Physicians use these drugs with the intent of decreasing pain and promoting recovery[1,21,34,49]. Some physicians also use short courses of oral corticosteroids for the same reasons[13,31]. Besides these medically accepted anti-inflammatory medications, patients may choose other drugs to treat their injuries. These medically unaccepted treatments of soft tissue injuries include anabolic steroids and dimethyl sulfoxide (DMSO)[2,6,37,38,45,50,59,73,85,90].

This article reviews the efficacy of medications commonly used to treat soft tissue injuries and the effects of these drugs on the tissues. The first section surveys the drugs commonly used in attempts to decrease pain and promote recovery from injury. The next section reviews the effects of these drugs in soft tissue injuries, and the last section discusses the specific effects of these drugs on dense fibrous tissues and cartilage.

DRUGS USED TO TREAT SOFT TISSUE INJURIES

Non-steroidal Anti-inflammatory Drugs

The group of commonly used NSAID's includes aspirin, diflunisal, fenoprofen calcium, ibuprofen, indomethacin, naproxen, piroxicam, phenylbutazone, sulindac, and tolmetin sodium. These chemically heterogeneous drugs have important differences in their activities, but they share certain clinical and tissue effects[1,21,89]. They all have some analgesic, antipyretic, and anti-inflammatory activity. They also share side effects such as variable potential for gastric or intestinal ulceration and interference with platelet function. They all inhibit the synthesis and release of prostaglandins. Damaged cells release prostaglandins and the available evidence shows that prostaglandins act as mediators of inflammation. Presumably NSAID's suppress inflammation primarily by inhibition of prostaglandin synthesis, although they may also affect inflammation by other mechanisms.

Corticosteroids

Corticosteroids used for their anti-inflammatory activity include cortisone, hydrocortisone, prednisone, methylprednisolone, triamcinalone, and dexamethasone. They suppress or prevent the initial events in inflammation by inhibiting capillary dilation, migration of inflammatory cells, and tissue edema. Once the inflammatory process starts they inhibit capillary and fibroblast proliferation and collagen synthesis. These later effects can compromise healing[9,13,22,39,88].

These medications also influence metabolism, fluid and electrolyte balance, and the function of cells in multiple organs and organ systems including the kidney, liver, skeletal muscle, bone, cartilage, cardiovascular system, immune system, and nervous system[13]. Individual drugs vary considerably in their spectrum of activity. For example, at equivalent dose levels, triamcinolone has five times the anti-inflammatory activity of hydrocortisone, and dexamethasone has twenty-five times the anti-inflammatory activity of hydrocortisone. On the other hand, hydrocortisone causes sodium retention while triamcinalone and dexamethasone have little or no effect on sodium retention.

Along with the intended therapeutic results, administration of corticosteroids can cause tissue damage and disturb the function of a variety of tissues and organ systems. Despite multiple reports of the deleterious consequences of oral or parenteral corticosteroids[9,13,50], the relationships between corticosteroid dose levels and specific complications remain unclear. A safe dose of corticosteroids has not been clearly established.[5] Short term moderate or low dose oral corticosteroid therapy has not been shown to cause significant complications in normal people,

but multiple case reports describe bone necrosis associated with short term high dose corticosteroid therapy[5,84]. Reported complications of prolonged or repeated use of oral corticosteroids include disturbances of fluid and electrolyte balance, glucose metabolism, hypertension, increased susceptibility to infections, impaired wound healing, bone necrosis, tendon ruptures, gastrointestinal ulceration, behavioral disturbances, osteoporosis, myopathy, and in children, inhibition of growth[9,13,39].

Few systemic complications of local corticosteroid injections have been described[20,48,49]; one author reported a patient who developed bone necrosis following multiple corticosteroid injections[77]. Studies discussing the problems associated with corticosteroid injections show that subcutaneous fat necrosis and loss of skin pigmentation are the most common adverse effects; tendon ruptures and accelerated joint destruction have occurred less frequently[20,48,49].

Anabolic Steroids

Examples of anabolic steroids include methyltestosterone, testosterone propionate, methandrostenolone, oxandrolone, and stanozolol. They all have androgenic and anabolic activity, but they vary in the ratio of anabolic to androgenic activity. For example, testosterone propionate has an anabolic-androgenic ratio of 1:1, but stanozolol has a ratio of 100:1. These drugs have two generally accepted medical uses: treatment of selected types of anemia and treatment of hypogondal males[37,38]. Although the predominant activities of these medications are anabolic and androgenic, like other steroids, they influence cell function in multiple tissues and organ systems including muscle, liver, reproductive organs, the immune system, the central nervous system, and the hematopoetic system.

Some athletes use oral and injectable anabolic steroids with the intent of improving performance through gains in strength, ability to endure increased training, and accelerated recovery from soft tissue injury[6,37,38,46]. Despite extensive anecdotal evidence[6], the efficacy of anabolic steroids for these purposes remains questionable. Published results of anabolic steroid use by athletes show that these drugs do not predictably improve physical performance or aerobic capacity, and they have inconsistent effects on strength[37,38,46]. They may help increase strength, as measured by a single repetition maximum weight lift. This increase in power is seen in athletes who have been training intensively in a weight lifting program before the start of steroid use, and who also continue intensive training and maintain a high protein diet[37,38]. With other measures of strength, and in athletes that do not meet these criteria, anabolic steroids have not been shown to predictably increase strength or improve performance in specific sports.

Complications of anabolic steroid use occur frequently. More than 30% of athletes taking anabolic steroids reported subjective side effects including changes in libido, aggressiveness, and muscle spasm[37,38]. Use of these drugs also causes abnormalities of liver function tests, decreased serum testosterone levels, and decreased spermatogenisis. In addition, benign and malignant liver tumors have been reported in association with anabolic steroid use[37,38].

Dimethyl Sulfoxide

The physical and chemical characteristics of DMSO, a clear colorless liquid, make it an exceptional solvent, better than water for many substances. It lowers the freezing point of fluids and protects cells against damage due to freezing, and therefore has an important role in preserving tissues and cells like erthrocytes, platelets, and bone marrow elements[73,85]. When applied topically, it easily penetrates the skin and appears in the blood within minutes. It probably has local anesthetic activity and may have a central analgesic effect[85]. In some experiments it appears to have anti-inflammatory activity and several studies suggest that it may reduce collagen synthesis or enhance collagen degradation[85]. Currently accepted uses include preservation of cells and treatment of intersitial cystitis of the bladder, gastrointestinal amyloidosis, and dermatologic lesions of scleroderma[73].

EFFECTS OF DRUGS IN SOFT TISSUE INJURIES

Non-steroidal Anti-inflammatory Drugs

NSAID's have established roles in the treatment of chronic inflammatory diseases involving the musculoskeletal system, including rheumatoid arthritis and other rheumatologic disorders. They also form the primary medical therapy for osteoarthritis. The efficacy of NSAID's in providing symptomatic improvement and their tissue effects have been examined in patients with these chronic conditions and in animal experiments designed to assess the effects of repeated use of these medications on normal tissues.

Despite their widespread use for treatment of acute soft tissue injuries such as ligament and joint capsule sprains, and chronic injuries such as patellar or achilles tendonitis, the efficacy of NSAID's has not been clearly demonstrated[1,21,89]. Non-steroidal anti-inflammatory drugs decrease acute soft tissue inflammation, and clinical experience suggests that they decrease the pain associated with tissue injury and joint stiffness[1,21,33,34]. There is less evidence that NSAID's can promote restoration of normal tissue function following injury. A study of ligament repair in rats showed that piroxicam increased the strength of

healing rat ligaments fourteen days after injury if the drug was administered for the first six days after injury[32]. It did not affect the ultimate strength of healed ligaments or the strength of normal ligaments. An experimental study suggested that a NSAID promoted return of function following muscle strain[3], but experimental and clinical studies have not clearly shown that NSAID's promote a more rapid return to full function or improve performance following injury[89].

Corticosteroids

The anti-inflammatory potency of corticosteroids far exceeds that of the available non-steroidal medications, but the frequency of serious complications is also much higher. Like the NSAID's, corticosteroids have a generally accepted role in the treatment of chronic inflammatory diseases of the musculoskeletal system including rheumatoid arthritis. The role of these medications in the treatment of acute and chronic soft tissue injuries is less clear.

Corticosteroid Injections

Despite the limited documented evidence of efficacy in treatment of soft tissue injuries, many physicians use corticosteroid injections based on clinical experience[20,49,50]. One investigator reported that the symptoms of bursitis and tendonitis responded more frequently to corticosteroid injections than other conditions, including knee synovitis associated intra-articular derrangements and acromioclavicular joint arthiritis. However, many patients had symptoms return following injection[49]. Experimental studies show that corticosteroids decrease scar tissue adhesions following tendon injuries[41] and decrease joint stiffness following fractures[36]. They have not shown that corticosteroids accelerate healing or return to function[49].

Recent reviews of corticosteroid injections for treatment of acute and chronic sports injuries stress that injections should be used with caution[48,49]. The author advised physicians to consider corticosteroid injection only after other non-surgical treatments have failed. Injections are most efficacious when the physician can identify a discrete, palpable source of the patient's symptoms. It is recommended that no more than three injections spaced weeks apart should be given. A second or third injection should be given only if the first injection decreased symptoms [48,49]. Following injection, the patient should have a period of rest or protection from further injury. Corticosteroid injections should not be used immediately after acute tendon, ligament, or joint injury; immediately before competition; or in the presence of infection[48,49]. Corticosteroids should not be injected into tendons or ligaments.

Oral Corticosteroids

Although oral corticosteroids have potent anti-inflammatory effects, few physicians use them for the treatment of soft tissue injuries[13]. Lack of studies on the use of these drugs for treatment of soft tissue injuries in the last ten years[13,50] makes it difficult to assess their efficacy in improving function or accelerating return to activity following injury. Because of the difficulty in documenting the efficacy of oral corticosteroids and their potential complications, some authors recommend against use of these medications for the routine treatment of soft tissue injuries[13,50].

Anabolic Steroids

Among some groups of athletes, anabolic steroids have a reputation of expediting recovery from injury[6,50]; however, available objective evidence does not confirm this effect[37,38,46]. No reported studies have shown accelerated healing of ligament, tendon, or joint injuries due to anabolic steroids[37,38]; in fact, several reports suggest that these drugs may increase the probability of certain injuries[37,45,59,90].

Dimethyl Sulfoxide

Some patients report excellent results of topical DMSO treatment of musculoskeletal soft tissue injuries, but attempts to prove the efficacy of this treatment in controlled trials have produced conflicting results[18,19,56,73,74,78,79,85]. Application of 60% to 95% DMSO reportedly relieved the symptoms of acute bursitis within thirty minutes in about 90% of patients[78,79]. A study of 80% DMSO treatment of acute sprains, strains, bursitis and tendonitis found significantly better results with DMSO than with placebo[18,29], but another study found thirteen treatment failures in twenty patients with acute bursitis or tendonitis[56]. A double blind trial of DMSO treatment of rotator cuff tendonitis and tennis elbow did not find any significant benefit of the drug[74].

EFFECTS OF ANTI-INFLAMMATORY DRUGS ON DENSE FIBROUS TISSUES

Nonsteroidal Anti-inflammatory Drugs

Despite the extensive use of NSAID's, no evidence of damage to normal dense fibrous tissue has been reported[32]. The anti-inflammatory activity of NSAID's may have an effect on the early stages of dense fibrous tissue repair; but clinically significant inhibition of healing has not been documented. One study showed that they may temporarily increase the strength of healing dense fibrous tissues[32].

Corticosteroids

In contrast to the NSAID's, corticosteroids have been reported to cause harmful effects in normal and injured

dense fibrous tissues[49,64]. They alter the metabolism of normal tissues and multiple authors have reported spontaneous tendon and plantar fascia ruptures following corticosteroid injection or systemic use[10,20,30,35,40,44,47,51,58,80]. It is controversial whether inflammation or injury weakened the tissues before steroid use in these patients. However, animal experiments show that steroids inhibit matrix synthesis by normal mesenchymal cells[4]. Clinical experience also shows that multiple steroid injections may cause tissue atrophy[49].

The mechanism of apparent spontaneous rupture of tendons following corticosteroid use remains uncertain. Normal composition, structure, and mechanical properties of these tissues depend on matrix turnover. Conceivably, corticosteroids suppress synthesis of the matrix macromolecules, thereby preventing replacement of degraded matrix due to normal turnover. With time, this negative balance would weaken the tissue. It is also possible that corticosteroid injection might directly disrupt matrix organization[42,86]. Damage associated with steroid injection may be more severe than that associated with saline injection[86]. Some authors have found hyaline material in the region of corticosteroid injection into dense fibrous tissue, suggesting necrosis following injection[7,42,86].

Studies that examined the strength of normal tendons after steroid injections have yielded variable results. Some of the inconsistency may result from differences in doses of corticosteroids, location of the injection (injection into the tissues surrounding the tendon versus injection into the tendon), time of testing after injection, and methods of measuring tendon strength. Two groups of investigators found that corticosteroid injections did not weaken normal rabbit tendons[52,57,75]. However, repeated intra-articular injections of large doses of corticosteroids decreased the strength and stiffness of monkey anterior cruciate ligament bone-ligament-bone units[64]. Two other studies have shown decreases in tendon strength following injections directly into the tendon substance[42,86]. In one study, the ultimate strength of normal rabbit Achilles tendon decreased 35% within forty-eight hours after intra-tendinous injection of corticosteroid.[42]. Microscopic examination of the injected tendons showed disruption of the normal collagen fibril arrangement and clefts within the matrix. Two weeks after injection the failure strength of the injected tendons had improved to near normal. An amorphous eosinophilic staining material had appeared within the substance of the matrix, and the collagen fibril arrangement appeared near normal. These results showed that injection of corticosteroids directly into dense fibrous tissue weakens the tissue, but following a single injection the cells can restore the matrix toward normal. Because of the potential increased risk of tendon rupture, several authors have advised physicians to use extreme caution in selecting corticosteroid injections for treatment of tenosynovitis, or to avoid using this treatment altogether[49,50,83,86].

Corticosteroids also alter the healing of dense fibrous tissues. Steroid mediated inhibition of fibroblast proliferation and synthesis of new matrix has the benefit of decreasing adhesions between injured dense fibrous tissues and the surrounding tissues[43,91]; however, it delays development of wound strength[39,41]. In experimental studies corticosteroid injections of transected tendons decreased tendon weight, load to failure, and energy to failure[41,91]. Presumably, these consequences of corticosteroid injections result from inhibition of cell synthetic function. They may prolong healing and increase the probability of complications such as failure of healing and wound disruption[9,39,49,88].

Anabolic Steroids

Anabolic steroids may also weaken normal dense fibrous tissues. Several reports describe tendon ruptures or tears associated with anabolic steroid use[37,45]. Experimental studies also suggest that these drugs damage dense fibrous tissues[59,90]. Administration of an anabolic steroid to mice subjected to an endurance training program caused degenerative changes in musculo-tendonous junctions including increased variability in collagen fibril diameter, organization, disruption of collagen fibrils, and calcification[59]. A study of rat tendons showed that exercise and anabolic steroids caused tendons to reach breaking strains earlier and supported the argument that anabolic steroids may predispose dense fibrous tissues to injury[90].

Dimethyl Sulfoxide

Like corticosteroids and anabolic steroids, DMSO may weaken dense fibrous tissues. In tissue cultures it inhibits fibroblast proliferation[15] and decreases collagen synthesis in at least one cell line[8]. If the drug has the same consequences in vivo, it could increase the probability of tendon, ligament, and joint injury. In one experimental study the investigators washed the skin over mice Achilles tendons with a 70% solution of DMSO and then measured the strength of the achilles tendons[2]. They found a variable effect on the force required to separate the tendons. In the first week of treatment it decreased 20.2%; however, over the next two weeks it increased and then decreased again. The investigators concluded that the decreased separation force due to DMSO treatment made the tendons more susceptible to injury[2].

EFFECTS OF ANTI-INFLAMMATORY DRUGS ON CARTILAGE

Nonsteroidal Anti-inflammatory Drugs

Selected NSAID's alter the synthetic activity of normal chondrocytes, and thereby change the composition and possibly the mechanical properties of the cartilage matrix. A series of studies shows that prolonged administration of salicylates and several other NSAID's suppresses proteoglycan synthesis in normal cartilage and sometimes alters the organization of the cartilage matrix by interfering with proteoglycan aggregate formation[16,17,67-69,71,72]. However, one study found that a different NSAID decreased cartilage proteoglycan turnover and increased the stiffness of normal cartilage[76].

The clinical significance of the effects of NSAID's on normal cartilage in vivo remains uncertain, but a significant decrease in cartilage proteoglycan concentration decreases cartilage stiffness and increases cartilage permeability[23,26,27,62,63]. These changes might theoretically make the tissue more vulnerable to injury[22], but none of the reported studies show that NSAID's cause progressive degeneration of normal cartilage.

NSAID's also affect chondrocyte function in injured or degenerating cartilage[16,17,66,70,72]; several investigations have shown that aspirin suppresses proteoglycan synthesis more severely in osteoarthritic cartilage than in normal cartilage[70,72]. Prolonged oral administration of aspirin aggravated the degeneration of canine articular cartilage caused by immobilization[66] and exacerbated the degeneration of articular cartilage in unstable joints[70]. In dogs with knee instability due to transection of the anterior cruciate ligament, prolonged administration of aspirin decreased articular cartilage thickness, cartilage proteoglycan content, and proteoglycan synthesis compared with the unstable knees of dogs that did not receive aspirin[70]. Although these studies show that NSAID's, and in particular aspirin, alter chondrocyte synthetic function, the clinical significance of these observations has not been demonstrated.

Corticosteroids

Multiple experimental studies show that repeated intra-articular injections of corticosteroids cause progressive deterioration of normal articular cartilage, and that increasing amounts of corticosteroids increase the severity of cartilage damage[11,53,54,61]. Following intra-articular corticosteroid injections, chondrocyte synthesis of collagen and proteoglycans rapidly and profoundly decreases [11,53,54,65]. Then, matrix proteoglycan concentration drops, decreasing cartilage stiffness and increasing permeability[63]. Acute or repetitive loading of the damaged articular cartilage may cause mechanical disruption of the weakened matrix and cause progressive loss of the articular cartilage[11,12,22,25].

Systemic corticosteroid administration also depresses chondrocyte synthetic activity[55]. In otherwise normal joints, if the articular cartilage damage due to corticosteroids leaves the tissue physically intact with enough viable chondrocytes, the cells will attempt to repair the damage. Following cessation of intra-articular steroid injections, chondrocytes increase their rate of proteoglycan and collagen synthesis up to 900%[12]. The increase results from accelerated activity by existing cells and an apparent increase in the number of cells due to cell proliferation. Under favorable circumstances, the increased matrix synthesis will return the matrix proteoglycan concentration toward normal[12].

Corticosteroid injection of synovial joints damaged by rheumatoid or osteoarthritis often gives patients rapid relief of pain[29]. In advanced joint disease, where synovial inflammation contributes to the patient's symptoms and to the progression of the disease, suppression of inflammation by corticosteroids may help maintain joint function. Unfortunately, other effects of the corticosteroids may more than offset these potential benefits. Corticosteroid injections presumably suppress chondrocyte synthetic activity in injured or degenerated cartilage at least as effectively as they do in normal cartilage. Suppression of chondrocyte synthetic activity may prevent the cells from repairing matrix defects due to injury or disease and thereby hasten the deterioration of the articular cartilage. Multiple clinical reports describe rapid joint disintegration in patients with rheumatoid arthritis and osteoarthritis following intra-articular steroid injections[14,28,60,81-83,92]. Although the steroid induced inhibition of chondrocyte synthetic activity may have accelerated the joint destruction in these patients, decreased pain may have also had a role. Relief of pain following the injections allows the patients to increase their activity, and the increased loading may contribute to loss of the damaged articular cartilage[28]. The apparent frequency of this problem and the results of the clinical and experimental studies have led some physicians to recommend stopping the practice of multiple joint injections with corticosteroids, and that there should be strong justification for single joint injections[83].

Conclusions

Drug treatment of acute and chronic soft tissue injuries is a common practice. Physicians frequently recommend NSAID's or local corticosteroid injections to decrease pain, and in some instances accelerate restoration of function. Oral corticosteroids have also been used, and some patients elect to use drugs like anabolic steroids or dimethyl sulfoxide. Yet the efficacy and effects of many of these drugs in the treatment of soft tissue injuries have

not been clearly established.

Non-steroidal anti-inflammatory drugs suppress inflammation and provide analgesia, but their capacity to minimize tissue damage and accelerate return to normal function after injury have not been clearly proven. Although some NSAID's alter chondrocyte synthetic function, the clinical significance of these observations has not been demonstrated. No evidence of damage to normal dense fibrous tissues caused by these drugs has been reported. Thus, when treatment of an injury requires analgesia and supression of inflammation, short duration treatment with NSAID's may be helpful and is not likely to cause significant complications. If there is no need for an anti-inflammatory effect, acetaminophen offers a reasonable alternative.

Corticosteroids have much greater anti-inflammatory potency than NSAID's, but they also have not been shown to accelerate restoration of function. Most importantly, their use is associated with more serious complications. Several reports describe tendon ruptures following use of oral corticosteroids and corticosteroid injections for treatment of tenosynovitis. Multiple studies show that repeated intra-articular injections of corticosteroids are associated with progressive deterioration of articular cartilage. Therefore, corticosteroid injections of tendons or ligaments should be avoided and repeated corticosteroid injection of the same site should be performed with caution. In most patients corticosteroid injections should be used only after other non-surgical treatments have failed or can reasonably be expected to fail based on previous clinical experience, and when the physician can identify a discrete localized source of the patient's symptoms. Following injection, the patient's activity should be restricted. Because of the lack of controlled studies showing the efficacy of oral corticosteroids, as well as their potential complications, physicians should not recommend these medications for the routine treatment of soft tissue injuries. If oral corticosteroids are used, doses should be carefully monitored. No reported studies have shown accelerated healing of ligament, tendon or joint injuries due to anabolic steroids; several reports suggest that these drugs may increase the probability of certain injuries as well as cause other complications. Therefore, physicians should discourage use of these drugs.

Some patients report good results of topical DMSO for treatment of soft tissue injuries, but attempts to prove the efficacy of this approach in controlled trials has produced conflicting results.

A clear need exists for more and better clinical and experimental studies of the efficacy and tissue effects of drug treatment of soft tissue injuries. Currently, physicians must base drug treatment of soft tissue injuries on clinical experience, knowledge of selected experimental and clinical studies, as well as understanding of the general analgesic and anti-inflammatory activities of these commonly used medications.

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THE COLLAGEN IN NORMAL LIGAMENTS

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INTRODUCTION

Collagen is the single most abundant animal protein in mammals, accounting for up to 30% of all proteins. The collagen molecules, after being secreted by the cells, assemble into characteristic fibers responsible for the functional integrity of tissues such as bone, cartilage, skin, and tendon[1]. They contribute a structural framework to other tissues such as blood vessels and most organs. Crosslinks between adjacent molecules are a prerequisite for the collagen fibers to withstand the physical stresses to which they are exposed. Significant progress has been made towards understanding the functional groups on the molecules that are involved in the formation of such crosslinks, their nature, and location. A variety of human conditions, normal and pathologic, involve the ability of tissues to repair and regenerate their collagenous framework. Some of these conditions are characterized by excessive deposition of collagen (e.g., cirrhosis, scleroderma, keloid, pulmonary fibrosis, diabetes, etc.). After trauma or surgery, abnormal deposition of collagen may impair function (adhesions following repair of long tendons, scar formation during healing, etc.). In addition, many disabling conditions result from changes in the nature and organization of collagen (heart-valve lesions, osteoarthritis, rheumatoid arthritis, and congenital collagen diseases such as Marfan's and Ehlers-Danlos syndromes, osteogenesis imperfecta, etc.).

In human tissue there are twelve different collagen types that have been well characterized. Many others are currently being studied.

This paper will address the various fundamental steps in the anabolism and catabolism of collagen, as well as the unique types of collagen present in tendon and ligaments, and will focus on the accumulation of collagen which follows injury and accompanies the repair process.

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I. THE COLLAGEN MOLECULE

The arrangement of amino acids in the collagen molecule is shown schematically in Fig. 1. Every third amino acid is glycine. Proline and hydroxy-proline follow each other relatively frequently, and the (gly, pro, hyp) sequence makes up about 10% of the molecule. This triple helical structure generates a symmetrical pattern of three left-handed helical chains that are, in turn, slightly displaced to the right, superimposing an additional "supercoil" with a pitch of approximately 8.6 nanometers (nm). These chains, known as α-chains, have a molecular weight of around 100 k Daltons and contain approximately 1000 amino acids for the interstitial collagen Types I, II and III (Fig. 2). The amino acids within each chain are displaced by a distance h = 0.201 nm with a relative twist of 100 degrees, making the number of residues per turn 3.27 and the distance between each third glycine 0.87 nm. The individual residues are nearly fully extended in the collagen structure, since the maximum displacement within a fully stretched chain would be approximately 0.36 nm. This separation is such that it will not allow *intrachain* bonds to form (as does occur in the alpha helix), and only interchain hydrogen bonds are possible. The exact number of hydrogen bonds that stabilize the triple helical structure has not been determined. One model describes two hydrogen bonds for every three amino acids whereas another assumes one.

In addition to these intramolecular conformational patterns, there seems to exist a supermolecular coiling. Microfibrils, possibly representing intermediate stages of packing, have been described.

A process of *self-assembly* causes the collagen molecules to organize into fibers. The thermodynamics of such a

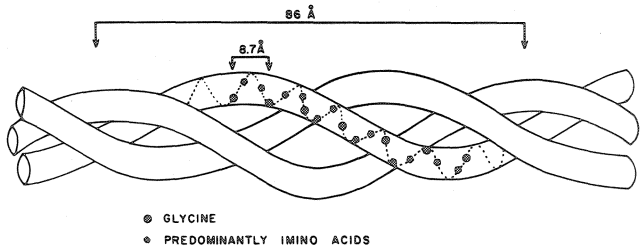

FIGURE 1: The collagen triple helix. The individual α chains are left-handed helices with approximately three residues per turn. The chains are in turn coiled around each other following a right-handed twist. The hydrogen bonds which stabilize the triple helix (not shown) form between opposing residues in different chains (interpeptide hydrogen bonding) and are therefore quite different from α helices which occur between amino acids located within the same polypeptide. O, Glycine; [], predominantly amino acids.

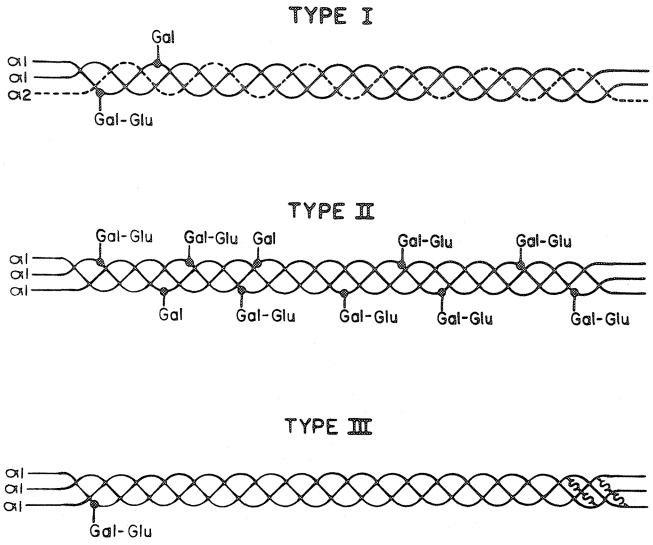

FIGURE 2: Diagram of the three interstitial types of collagen. Type I is present in skin, bone, tendon, etc.; Type II is present in cartilage; and Type III is present in blood vessels and developing tissues and as a minor component in skin and other tissues. There are differences in the chain composition and degrees of glycosylation. Disulfide crosslinks are only seen in Type III collagen.

system involve changes in the state of the water molecules, many of which are associated with nonpolar regions of the collagen molecule[2].

II. BIOSYNTHESIS: PROCOLLAGEN

In order for the organism to develop an extracellular network of collagen fibers, the cells involved in the biosynthetic process must first synthesize a precursor known as procollagen. This molecule is later enzymatically trimmed of its nonhelical ends, giving rise to a collagen molecule that spontaneously assembles into fibers in the extracellular space. Procollagen molecules have been identified as precursors of the three interstitial collagens (Types I, II, and III). Several of the N- and C-terminal peptides (propeptides) have been characterized and the primary sequences determined.

The carboxyterminal propeptides of both pro α1 and pro α2 chains have molecular weights of 30,000-35,000 daltons and globular conformations without any collagen-like domain. These peptides contain asparagine-linked oligosaccharide units composed of N-acetylglucosamine and mannose. Once the molecule is completed and translocated to the cell surface, the extensions are enzymatically removed from those collagens, which then form fibrils. Enzymes that selectively remove these extensions can be found in a variety of connective tissues, and in the culture media derived from collagen-secreting cells.

A. Gene Expression

Since the discovery about twenty years ago of a distinct form of collagen in cartilage, now known as Type II

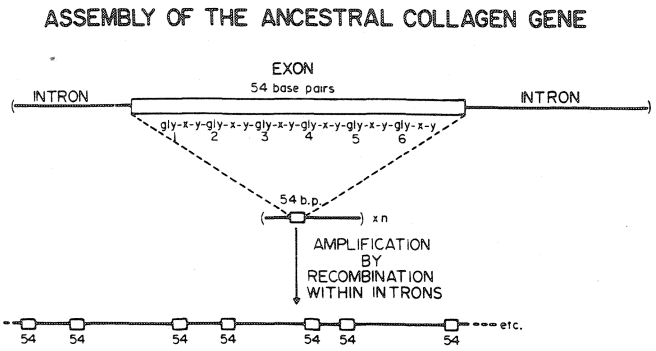

FIGURE 3: The collagen gene is made up of multiple units containing 54 base pairs, each of which corresponds to sequences of 18 amino acids. The conservation of this minimum sequence and the fact that it is repeated in such an exacting fashion provide valuable information to investigators interested in the process of evolution of proteins. (Redrawn from Dr. DeCromburgghe.)

collagen, many other unique molecular species have been observed. Types I, II, III, V and XI collagen are categorized as *fiber forming collagens*. They all exhibit lengthy, uninterrupted collagenous domains and are first synthesized as biosynthetic precursors (procollagens). Gene cloning experiments have demonstrated that the Group I collagen genes are evolutionarily related, for they share a common ancestral gene structure. Human chromosome number 17 contains the coding information for the α1 chain of Type I collagen while chromosome 7 codes for its complementary α2 chain. A comparison of the five fibrillar collagens described shows that with one exception [Types III and α2(V) are located on chromosome 2] all other genes are located on different chromosomes.

The genes coding for fiber forming collagens are large, about 10 times the size of the functional mRNA. Many of the exons (coding sequences) are 54 base pairs (bp) in length and are separated from each other by large intervening sequences (introns) that range in size from about 80 to 2,000 bp's. The gene itself contains 38,000 bp's and is very complex. The finding that most exons of these genes have identical lengths suggests that the ancestral gene for collagen was assembled by multiple duplications of single genetic units containing an exon of 54 bp (Fig. 3). It is likely that a primordial exon this size could have encoded for a gly-pro-pro tripeptide repeated six times (3 x 3 x 6). Such a polypeptide of 18 amino acids probably had the minimum length needed to form a stable triple helical structure.

B. Translational, Cotranslational, and Early Post-translational Events

After the gene is transcribed, it is spliced to remove introns and to yield a functional mRNA that contains about 3,000 bases. Specific mRNAs for each chain and collagen type are translocated to the cytoplasm and translated into proteins in the rough endoplasmic reticulum on membrane

bound polysomes. As the collagen polypeptide is synthesized in the rough ER, it is modified in important ways. Two major constituents of collagen are the modified amino acids hydroxyproline and hydroxylysine but neither of these can be directly incorporated into proteins. Instead, proline and lysine are incorporated and then modified by two hydroxylating enzymes, prolyl and lysyl hydroxylases. These enzymes require ferrous iron, ascorbate, and α-ketoglutarate for their activity. The degree of hydroxylation differs from tissue to tissue and depends on availability of substrate, rate of synthesis, turnover, and the time during which the molecule remains in the presence of the hydroxylating enzymes. The time required for the synthesis of a complete pro α chain is about 6.7 min.

As lysyl residues in the newly synthesized pro α chains are hydroxylated, sugar residues are added to the resulting hydroxylysyl groups. Glycosylation is catalyzed by two specific enzymes, a galactosyltransferase and glucosyltransferase. Once the translation, modifications, and additions are completed, the individual pro α chains become properly aligned for the triple-helix to form.

C. Intracellular Translocation of Procollagen and Extrusion into the Extracellular Space

The procollagen molecules, now detached from the ribosome, emerge from the endoplasmic reticulum and move towards the Golgi apparatus through the microsomal lumen. In the Golgi, the C-terminal mannose-rich carbohydrate extensions are remodeled, the molecules are packaged in vesicles, and subsequently carried towards the cellular membrane (Fig. 4).

The small aggregates of oriented procollagen molecules are probably trimmed of their nonhelical amino and carboxyl extensions by specific peptidases when they reach the extracellular space. In the case of Type I collagen, the first peptidase to act seems to be the amino protease; this is followed by a carboxyprotease. In Type III collagen the sequence of removal may be reversed.

D. Lysyl Oxidase

Recently formed microfibrils seem to be recognized by the enzyme lysyl oxidase, which converts certain peptide-bound lysines and hydroxylysines to aldehydes (Fig. 5). The enzyme is an extracelluar amine oxidase, which has been purified from a variety of connective tissues. It requires Cu^{2+} and probably pyridaxal as cofactors; molecular oxygen seems to be the cosubstrate and hydrogen acceptor. It is irreversibly inhibited by the lathyrogen BAPN (β-amino pro-pronitrile, a substance found in the flowering sweet-pea, lathyrus odoratus). This enzyme exhibits maximal activity when acting on collagen fibrils rather than upon monomeric collagen.

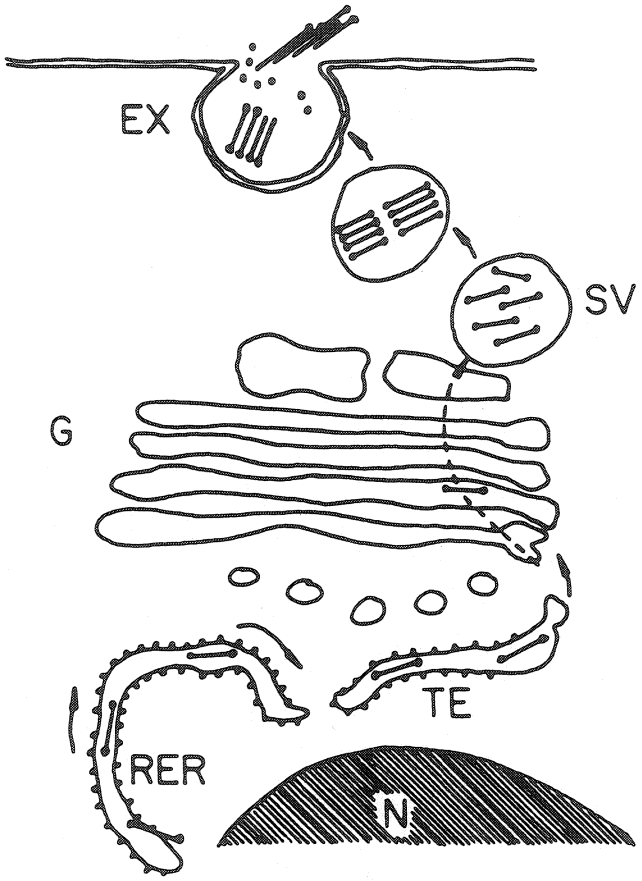

FIGURE 4: Movement of procollagen through the cisternae of the rough endoplasmic reticulum (RER) and through a transitional endoplasm (TE) to the Golgi apparatus (G), where it is packaged into secretory vesicle (SV) prior to extrusion (EX) by exocytosis. N, Nucleus.

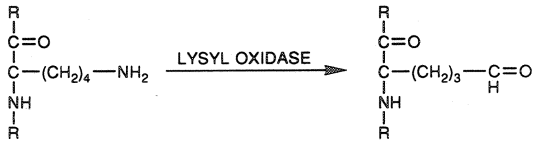

FIGURE 5: The oxidative deamination of peptide-bound lysine by the enzyme lysyl oxidase generates the aldehydes associated with the collagen molecule.

E. Fibrillogenesis

The tendency of collagen molecules to form macromolecular aggregates is well known. This tendency is common with most fibrous proteins that form filaments with helical symmetry and which occupy equivalent or quasiequivalent positions.

The exact mode in which the collagen molecules pack into microfibrils (precursors of the larger fibrils) still remains a subject for speculation. A five-stranded microfibril was first suggested to account for such a sub-

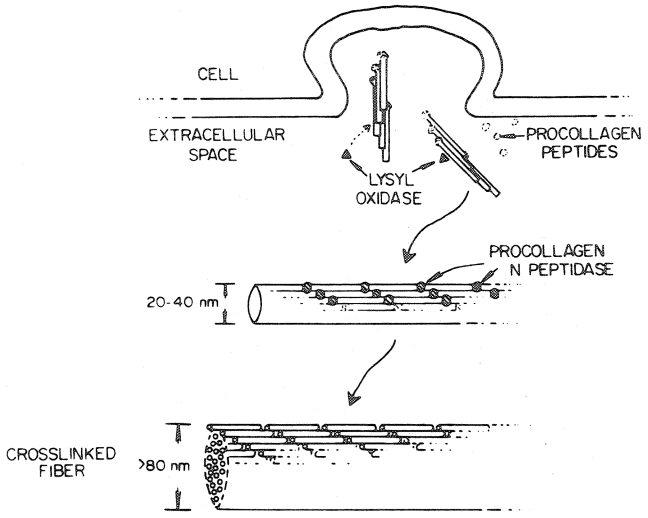

FIGURE 6: Fibrillogenesis: Microfibrils in a quarter-staggered configuration have lost their carboxy-terminal nonhelical extensions, part of their amino (N)-terminal nonhelical extensions, and part of their amino (N)-terminal extensions. In this form they seem to organize readily into small-diameter fibrils which retain part of the amino-terminal nonhelical extensions. After being relieved of these peptides by a procollagen peptidase, fibrils are able to grow in diameter by apposition of microfibrils or by merger with other small-diameter fibrils.

structure, one that would satisfy the condition that adjacent molecules were equivalently related by a quarterstagger (Fig. 6).

When monomeric collagen is heated to 37°C it progressively polymerizes, generating a turbidity curve that reflects the presence of intermediate aggregates. The lag phase (persistence of monomers), the nucleation and appearance of turbidity (microfibrils), and the rapid increase in turbidity (fiber formation) have been equated to how the cell may handle this process.

III. DIFFERENT TYPES OF COLLAGEN

Almost two decades have passed since we first realized that all collagen fibers within a particular organism are not made up of identical molecules. The different collagen types are usually identified using Roman numerals, assigned as they are purified and characterized.

Since this paper focuses on ligaments, we shall only describe the major characteristics of the two collagen types present in the anterior cruciate ligament, namely I and III.

A. Type I Collagen

Before 1969, Type I collagen was the only mammalian collagen known. It is composed of three chains - two identical, termed $\alpha 1$ chains, and one different from the other two, called $\alpha 2$. Type I collagen is most abundant in skin, tendon, ligament, bone, cornea, etc.; it comprises between 80% and 99% of the body's total collagen. Bone matrix is essentially all Type I collagen. The most common technique used to isolate this molecule, and distinguish it both qualitatively and quantitatively from the other collagens, involves the use of solvents of different ionic strength and pH followed by differential salt precipitation.

The amino acid composition of some of the better characterized and more abundant human collagen chains are shown in Table 1. The differences in amino acid composition of human collagen chains specific to collagen types are noted in columns, with the type in parentheses. On many occasions these differences have been used to identify collagen types or their mixtures and to suspect the presence of new or abnormal collagen species.

B. Type III Collagen

When human dermis is digested with pepsin under conditions in which the collagen molecules retain their helical conformation, Type I molecules can be separated from Type III by differential salt precipitation at pH 7.5. The Type III molecules are composed of three identical chains. Characteristic of this collagen is the presence of intramolecular disulfide bonds involving two cysteine residues close to the C-terminal region of the triple helix. Because the ratio of Type I and Type III collagen changes with age, Type III being predominant in fetal skin, this type of collagen is many times referred to as *fetal* or *embryonic collagen.*

Formation of intermolecular disulfide bridges by Type III collagen could be of great advantage during early development and wound healing, where collagen is deposited at a rapid rate in order to fill a gap.

Normal bone matrix may be the only tissue containing Type I collagen that lacks Type III collagen. Blood vessels are particularly rich in Type III collagen.

IV. COLLAGEN METABOLISM

Collagen is the most abundant of all body proteins. Tissues such as bone, which are involved in active remodeling, are responsible for the major turnover. Other less dynamic tissues in the full grown individual, such as skin and tendons, may exhibit slow and almost negligible turnover. The collagen synthesizing activity of cells is usually assessed by their ability to synthesize hydroxyproline or by the activities of specific enzymes such as the proline and lysyl hydroxylases.

A. Degradation: Collagenases, Bacterial and Mammalian

Because of its triple helical structure stabilized by hydrogen bonds, the collagen molecules are quite resistant to enzymatic degradation in their native configuration. They can be degraded by collagenases. The first of these to be isolated were of bacterial origin, specifically,

Table 1. Amino Acid Composition of the Human Collagen Chains (Residues/1000 Total Residues)

Amino Acid	α1(I)	α2(I)	α1(II)	α1(III)	α1(IV)	α2(V)	α1(V)	α3(V)	1α	2α
3-Hydroxyproline	1	1	2	0	7	3	5	1	...	...
4-Hydroxyproline	108	93	97	125	**133**	106	110	91	98	93
Aspartic Acid	42	44	43	42	51	50	49	42	46	50
Threonine	16	19	23	13	20	29	21	19	17	25
Serine	34	30	25	39	37	34	23	34	25	28
Glutamic Acid	73	68	89	71	79	89	100	97	107	98
Proline	124	113	120	107	**65**	107	130	98	109	119
Glycine	333	338	333	350	328	331	332	330	334	327
Alanine	115	102	103	96	**37**	**54**	**39**	**49**	**54**	**49**
Half-Cystine	0	0	0	**2**	0-1	0	0	1	0	0
Valine	21	35	18	**14**	28	27	17	29	28	18
Methionine	7	5	10	8	13	11	9	8	10	9
Isoleucine	6	14	9	13	**29**	15	17	20	15	16
Leucine	19	30	26	22	**52**	37	36	**56**	35	39
Tyrosine	1	4	2	3	2	2	4	2	2	3
Phenylalanine	12	12	13	8	**29**	11	12	9	11	11
Hydroxylysine	9	12	**20**	5	**49**	**23**	**36**	**43**	**38**	**40**
Lysine	26	18	15	30	9	13	14	15	19	15
Histidine	3	12	2	6	6	10	6	14	6	11
Arginine	50	50	50	46	**26**	48	40	42	45	48
Gal-Hydroxylysine	1	1	4	...	2	3	5	7	...	...
Glc-Gal-Hydroxylysine	1	2	12	...	30	5	29	17	28	34

Clostridium Histolyticum. These enzymes are quite specific for collagen but will also degrade gelatin, which is denatured collagen. They are inhibited by cysteine and other SH-compounds and by EDTA, a chelator for divalent cations. These enzymes are specific for peptide bonds involving glycine in a collagen helix conformation. Because of the abundance of this amino acid in collagen (every 3rd residue) this enzyme generates a large number of small peptides.

The first enzyme derived from animal tissue capable of degrading collagen at neutral pH was isolated from the culture fluid of tadpole tissue. It cleaves the native molecule into two fragments in a highly specific fashion at a temperature below that of substrate denaturation. Since then collagenolytic enzymes have been obtained from a wide range of animal tissues. In general, these enzymes have fundamental properties in common; they all have a neutral pH optima and are not stored within the cell, but rather are secreted either in an inactive form or bound to inhibitors. Figure 7 summarizes schematically the fundamental aspects of these enzymes and their mode of action. They appear to be zinc metalloenzymes requiring calcium, and are not inhibited by agents that block serine or sulphydryl type proteinases. Nearly all the collagenases studied so far have a molecular mass that ranges from 25,000 to 60,000 daltons. Mammalian collagenases display a great deal of specificity, cleaving bands between Gly-Leu or Gly-Ile. There are slight differences in the amino acid sequences surrounding the scission site; these may account for the differences in the rates at which various collagens are degraded.

The enzymes interact tightly with the collagen fibers and appear to remain bound to the macromolecular aggregate during the degradation process. Approximately 10% of the collagen molecules in reconstituted collagen fibrils appear accessible for binding, in close agreement with the theoretical number of molecules estimated to be present near the surface of the fiber. The in vitro data obtained seem to indicate that digestion proceeds to completion by hopping from one molecule to another without returning to the solution. Collagen from older individuals is more resistant to enzymatic digestion.

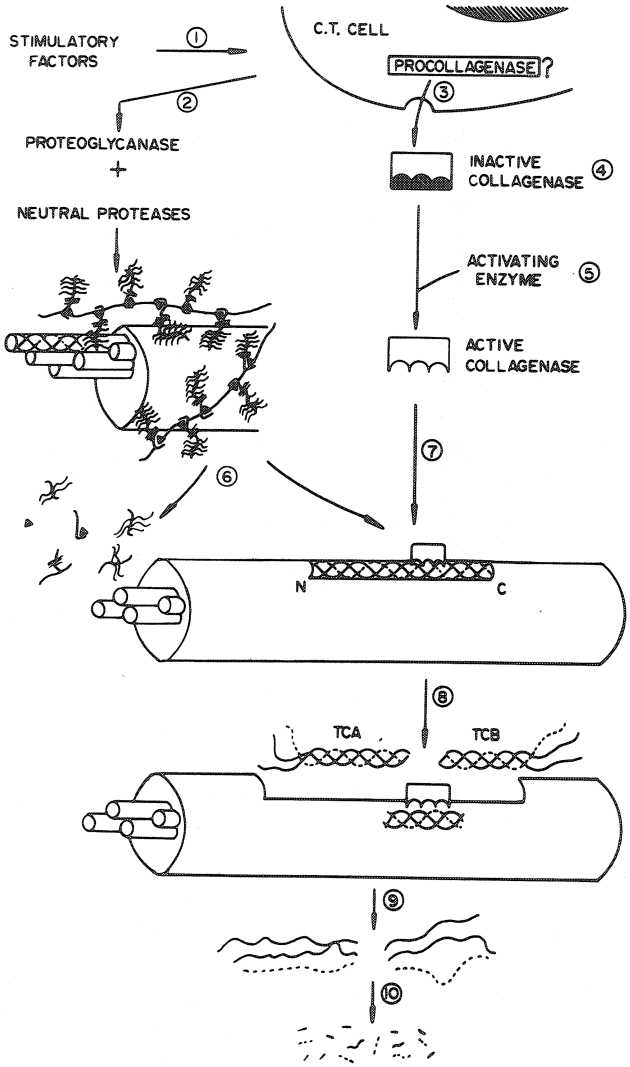

FIGURE 7: Sequence of events leading to the degradation of collagen fibers by the enzyme collagenase. (1) A variety of factors stimulate connective tissue (CT) cells to synthesize collagenase, glycosidases, and neutral proteases. (2) The proteoglycan-degrading enzymes remove the mucopolysaccharides which surround collagen fibers and expose them to collagenase. (3) Inactive collagenase is secreted. (4) The enzyme is usually found in the extracellular space bound to an inhibitor. (5) An activating enzyme removes the inhibitor. (6) Glycosidases complete the degradation of the proteoglycans. (7) The active collagenase binds to fibrillar collagen. (8) Collagenase splits the first collagen molecule into two fragments (TCA and TCB), which denature and begin to unfold at body temperature. The enzyme now moves on to an adjacent molecule. (9) The denatured collagen fragments are now susceptible to other proteases. (10) Nonspecific neutral proteases degrade the collagen polypeptides.

V. CROSSLINKING

A. Intramolecular and Intermolecular Crosslinks

Crosslinking renders the collagen fibers stable, and provides them with a degree of tensile strength and visco-elasticity adequate to perform their structural role. The degree of crosslinking, the number and density of the fibers in a particular tissue, and the orientation and

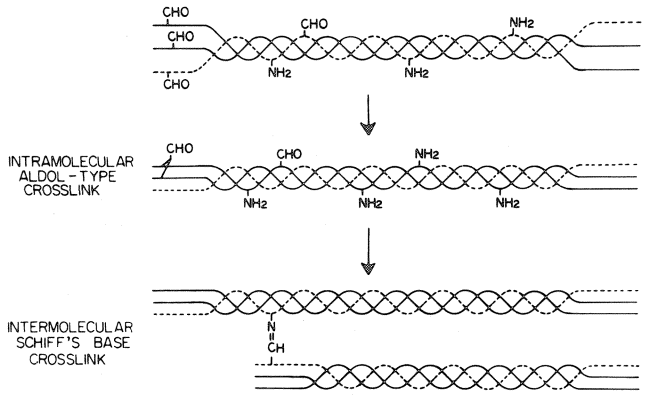

FIGURE 8: Formation of intramolecular and intermolecular crosslinks in type I collagen. Intramolecular crosslinks occur in the nonhelical regions and involve a condensation reaction between lysine- or hydroxylysine-derived aldehydes within a single molecule. Intermolecular crosslinks, on the other hand, involve aldehydes and ε-amino groups of lysine present in different molecules.

diameter combine to provide this function. Crosslinking begins with the oxidative deamination of the ε-carbon of lysine or hydroxylysine to yield the corresponding semi-aldehydes and is mediated by the enzyme lysyloxidase (Fig. 8). Enzymatic activity is inhibited by β-aminoproprionitrile, chelating agents, isonicotinic acid hydrazide, and other carbonyl reagents. Lysyl oxidase exhibits particular affinity for the lysines and hydroxylysines present in the nonhelical extensions of collagen, but can at a slower pace also alter residues located in the helical region of the molecule.

In general, lysine-derived crosslinks seem to predominate in soft connective tissues such as skin and tendon, whereas hydroxylysine-derived crosslinks are prevalent in the harder connective tissues such as bone, cartilage, and dentine, which are less prone to yield soluble collagens.

Several other crosslinks have been identified and their location established. These more complex polyfunctional crosslinks can contain histidine, or can result in the formation of naturally fluorescent pyridinium ring structures (Fig. 9).

The study of collagen crosslinking has advanced steadily, even though hindered by the difficulty in dealing with an insoluble three-dimensional matrix composed of quarter staggered molecules. Many crosslinking regions, primarily those involving the nonhelical extension peptides, have been identified (Fig. 10). Interestingly, covalent crosslinks between Type I and Type III molecules have been recently described.

B. Inhibition of Collagen Crosslinking: Amino nitriles and D-penicillamine

Lathyrism is a connective tissue disorder associated with the ingestion or injection of BAPN (β-amino propionitrile and its chemical analogues), extracts of the sweet

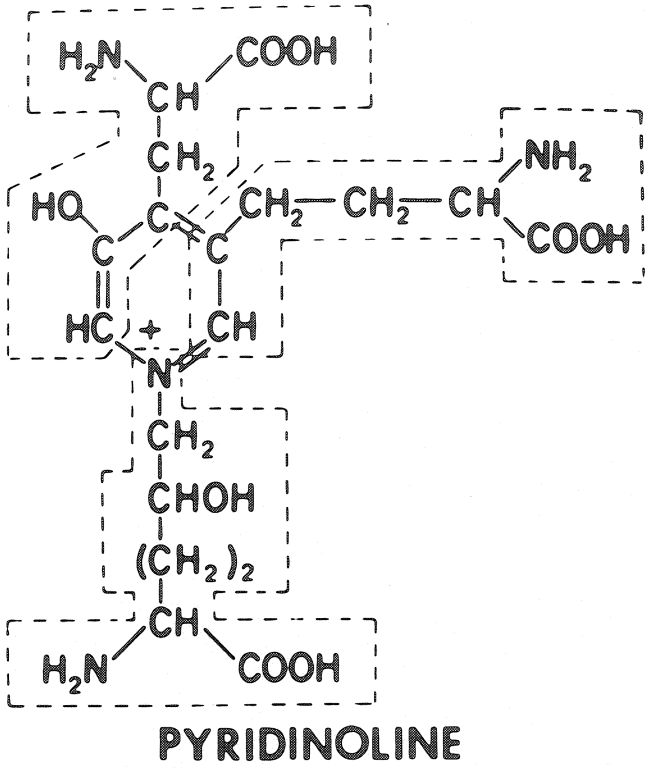

FIGURE 9: Pyridinoline: This trifunctional pyridinium crosslink, which joins three adjacent collagen molecules, can be generated by one hydroxylysine residue and two hydroxylysine-derived aldehydes or by spontaneous interaction of two hydroxylysine-5-keto-norleucine residues formed from a hydroxylysine and a hydroxylysine aldehyde.

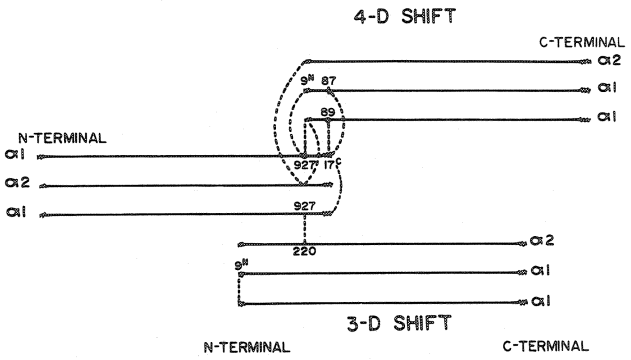

FIGURE 10: Schematic representation of Type I collagen molecules aligned in three-dimensional (3-D) and four-dimensional (4-D) staggered positions. The known crosslinking sites are indicated by dashed lines. The intermolecular crosslink formed between hydroxylysine residues 9^N from the amino (N)-terminal region of α1 chains is among the first crosslinking sites to be recognized. The carboxy (C)-terminal hydroxylysyl residue 927 of α1 chain can crosslink to one or two α1 chains through residue 9^N. Hydroxylysyl residue 927 crosslinks to 9^N of an α1 chain or to 9^N of an α2 chain. The carboxy-terminal 17^c hydroxylysyl residue of the α1 chain crosslinks to hydroxylysyl residue 87 from an α1 chain of another molecule. The residue 17^c might also form an aldo-type intramolecular crosslink with a similar residue of the other α1 chain. Histidine 89 from the carboxy-terminal region of an adjacent molecule adds to this crosslink via a Michael addition. Recently an intermolecular crosslink between hydroxylysyl residues 927 and 220 in the helical region was found in dentin and bone, supporting the observation that aldehydes form in the collagen helix.

pea, or others members of the lathyrus family usually consumed during periods of great famine. The skeletal changes observed differ among species and vary with age, being much more pronounced in younger animals. The epiphyseal plate is a prime target.

The connective tissue abnormalities are associated with crosslinking defects in collagen and elastin. They are revealed by an increased solubility in hypertonic neutral salt solutions, due to an inhibition of lysyl oxidase activity. Since Cu^{2+} deficiency also inhibits the enzymatic activity, the similarities of the defects induced by these two mechanisms are readily explainable.

Administration of penicillamine to animals and humans also causes an accumulation of neutral salt soluble collagen in skin and various soft tissues. Two of the more characteristic properties of penicillamine, namely the ability to trap carbonyl compounds and to chelate heavy metals, are of primary significance in impairing collagen crosslinking. The former property manifests itself in all effective dose ranges, whereas the latter occurs only at dosages far higher than those administered to humans.

The collagen extracted from tissues of animals treated with D-penicillamine is able to form stable fibers in vitro and is not deficient in aldehydes, as is that from BAP-treated animals. In fact, its aldehyde content is even higher than normal, suggesting that the mechanisms of action of BAPN and penicillamine are different.

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THE FATE OF AUTOGENOUS TENDON GRAFTS

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INTRODUCTION

The traditional method of tendon grafting employing autogenous extrasynovial tendon grafts to replace injured Zone II flexor tendons has met with limited success[2,7,12,13,15,22,25,26]. Experimental studies have shown that a necessary, if undesirable, component of the repair of extrasynovial tendon graft is the formation of mesotenon like connections from the synovial sheath[8,16,19,23]. Tendon grafts which incorporate by this method must undergo significant surface remodeling to gain complete tendon excursion and full digital function.

Recently it has been shown that the intrasynovial flexor tendon has structural characteristics which may facilitate survival within the digital sheath[9,17,18,24]. Intrasynovial flexor tendons posses a series of canaliculae, connecting the tendon interior with the surface, which may facilitate synovial fluid diffusion into the tendon providing nutritional support[24].

It was hypothesized that these structural differences between the two types of donor tendons may indicate different metabolic needs and allow fundamentally different methods of incorporation after flexor tendon grafting. Over the past several years we have performed a series of experiments to characterize the effect of the donor tendon, the repair response, and method of tendon graft incorporation of both intrasynovial and extrasynovial donor tendons.

MATERIALS AND METHODS

A canine model using the peroneus longus tendon as an extrasynovial donor tendon and the flexor digitorum profundus tendon from the hindpaw as an intrasynovial donor tendon was developed (Figure 1)[10,21]. The paratenon covered peroneus longus tendon was selected because of its similarity in caliber to the forepaw flexor digitorum profundus tendon. The hindpaw flexor digitorum profundus tendon was used because of its anatomical homology to the forepaw flexor digitorum profundus tendon.

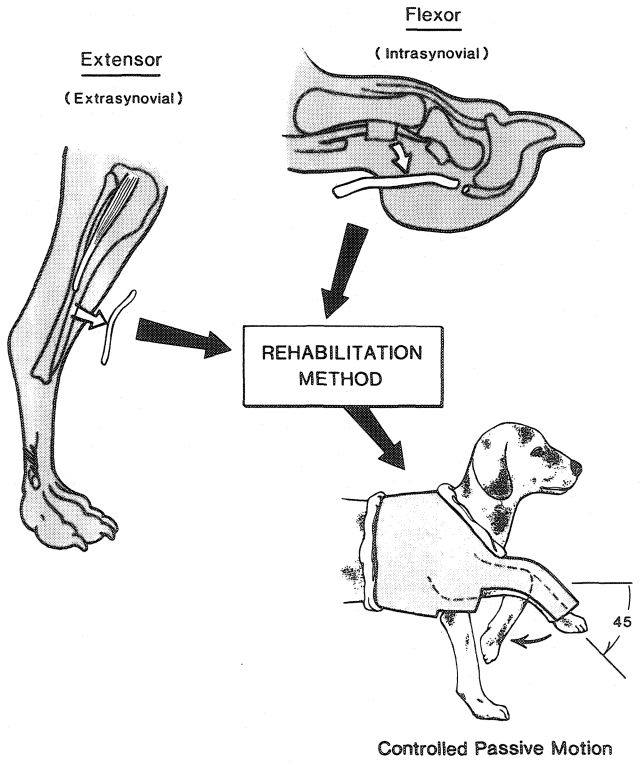

Figure 1
Schematic drawing depicting the use of extrasynovial and intrasynovial autogenous tendon grafts. Illustration reproduced with permission from the Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery.

A total of 156 tendon grafts were performed. The morphologic changes, revascularization, structural and functional properties, and cell viability of grafted tendons was studied at regular intervals from ten days to six weeks following surgery.

Animal Model

Initially we assessed the repair responses of intercalary short segment intrasynovial and extrasynovial tendon grafts when the repair was done in an intrasynovial location[10]. Animals were divided into two groups with Group I animals undergoing flexor tendon grafting with intrasynovial donor tendons from the ipsilateral hindpaw while Group II animals received tendon grafting with hindlimb extrasynovial donor tendons. The flexor appara-

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tus of the left second and fifth forepaw toes were exposed through separate midline incisions. The flexor tendon sheaths were incised between the A-2 and A-4 pulleys. The flexor digitorum profundus tendon was transected distally at the level of the proximal interphalangeal joint and proximally in the region of the mesotenon attachment to excise a two and one-half centimeter segment. The incised tendon was replaced by a graft sutured distally as described by Kessler et. al. with 4-0 braided dacron suture. A 6-0 nylon epitendinous suture was subsequently placed invaginating each repair site.

For all other projects, the model was revised to focus on the process of tendon graft incorporation within Zone II[21]. The flexor digitorum profundus of the left second and fifth forepaw toes were approached through one centimeter incisions made in the tendon sheath at the level of the distal interphalangeal joint and at the level of the proximal mesotenon attachment. The flexor digitorum profundus was then divided distally, two centimeters proximal to the distal osseous insertion and proximally in the region of the mesotenon attachment. In each animal's forepaw, an extrasynovial tendon graft from the ipsilateral hindlimb was placed in the synovial sheath of the second toe while a hindpaw intrasynovial tendon graft was placed in the fifth toe. The distal repair was completed using a modified grasping 3-0 nylon suture placed in the distal tendon graft and passed through drill holes in the distal phalanx. The sutures were secured over dorsally placed padded buttons. Proximally, the tendons were repaired using a tendon weave technique secured with two horizontal mattress sutures of 4-0 braided dacron (Figure 2)[20].

Postoperatively, animals were placed in polyurethane shoulder spica casts with elbows at 90 degrees and wrists at 45 degrees of flexion. All animals underwent postoperative rehabilitation with early controlled protected passive mobilization.

Assessment Of The Repair Response

The repair response of intracalary grafts of intrasynovial and extrasynovial origin was studied by light and transmission electron microscopy at ten and twenty-one days. At the time of sacrifice, the flexor apparatus was excised en-bloc from the musculotendinous junction to the base of the distal phalanx. Tendons were preserved immediately in neutral buffered formalin for standard histologic examination and in a solution of 4% periformaldehyde and 2% glutaraldehyde for electron microscopy. For light microscopy, the tendons were bisected longitudinally to maximize visualization of the healing response. One-half was then processed routinely into five micron sections stained with hematoxylin and eosin. Samples 0.25 x 1.0 x 4.0 mms were harvested from the repair site and at 1-2 mm intervals along the length of tendon grafts prepared for electron microscopy.

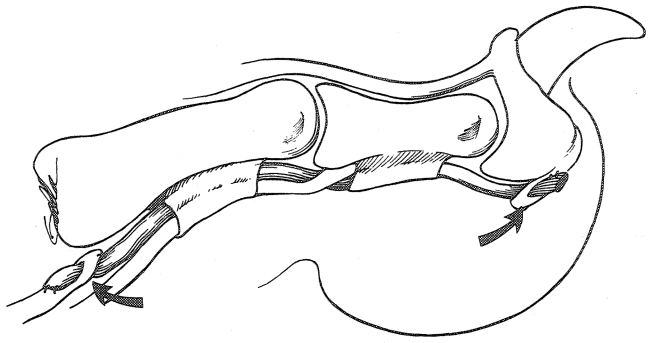

Figure 2
Schematic drawing depicting the method of intrasynovial and extrasynovial tendon grafting used to study tendon graft incorporation. Solid arrows point to the proximal and distal repair sites.

Comparison Of Tendon Graft Incorporation

To focus on the process of tendon graft incorporation, we modified our experimental model to graft the entire portion of the canine flexor digitorum profundus tendon in clinical Zone II and to place one intrasynovial and one extrasynovial tendon graft in each left forepaw (Figure 2). Specific parameters in comparing the process of tendon graft incorporation between the two types of flexor tendon grafts included light microscopy and electron microscopy. Specimens were harvested at three and six weeks.

Microangiography

To correlate these findings with the revascularization of the tendon grafts, we performed a vascular injection study designed to determine the sources, patterns and extent of neovascularization in intrasynovial and extrasynovial tendon grafts[11]. Experimental animals were sacrificed at two, four, and six week intervals. The left forepaw median artery was cannulated and injected using firm manual pressure with 200 milliliters of filtered India ink (diluted 9:1 with normal saline). The flexor apparatus was excised en bloc, preserved in formalin and clarified by modified Spalteholz technique. Intact transilluminated tendons were observed through a stereodissection microscope equipped with a calibrated grid.

Biochemistry

The viability of the cells within the grafts was assessed by measurement of the DNA content and by measuring the ability to synthesize DNA in vitro. Comparable segments of intrasynovial and extrasynovial tendon grafts and control tendons were harvested and divided for examination by the two methods at ten days, three weeks, and six weeks following tendon grafting. The total DNA content was determined[6]. In vitro DNA synthesis was measured following harvest of tendon graft samples at each time interval as an indication of cell proliferation[1]. Tendon segments were incubated with DMEM medium at 37°C in a water saturated atmosphere containing 5% carbon diox-

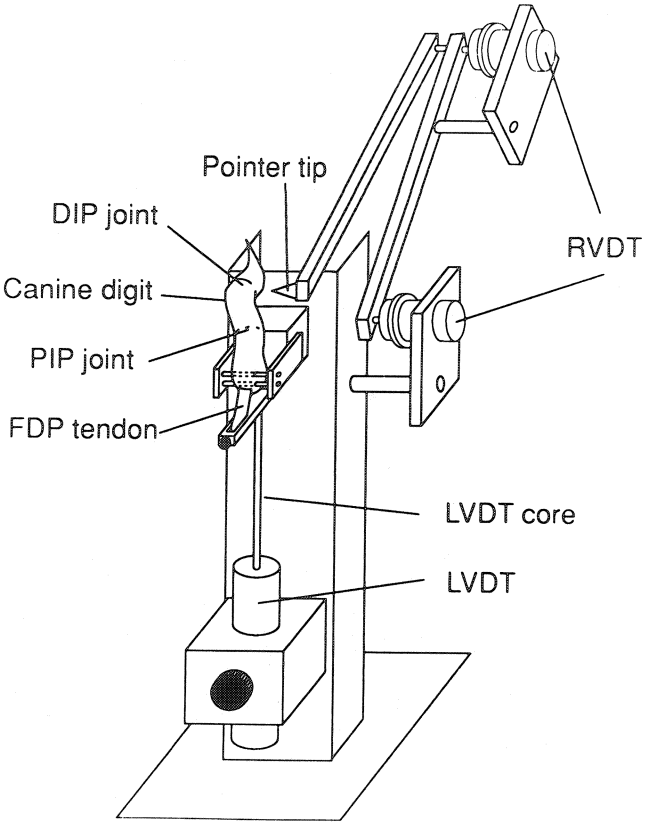

Figure 3
Schematic drawing depicting the apparatus used for biomechanical testing.

ide. After 24 hours the medium was replaced and supplemented with fetal calf serum and thymidine (1.2μg/ml). On the third day, the procedure was repeated and the tendons were labeled with 3H-thymidine (10 μci/ml). After rinse, chase incubation, and lyophilization the tendon dry weight was determined. Following recovery of the DNA, radioactive counts corresponding to the 3H-thymidine were measured in scintillation counter[1].

Biomechanical Testing

Biomechanical characteristics were evaluated at three and six weeks following tendon grafting[21]. Specimens were harvested by excising the digit through the metatarsophalangeal joint, preserving the synovial sheath and a proximal stump of the flexor digitorum longus tendon. The excursion (gliding function) of the tendon grafts within the sheath was evaluated with a device designed to measure angular rotation of the proximal interphalangeal joint with a 1.5N load applied to the proximal tendon end (Figure 3).

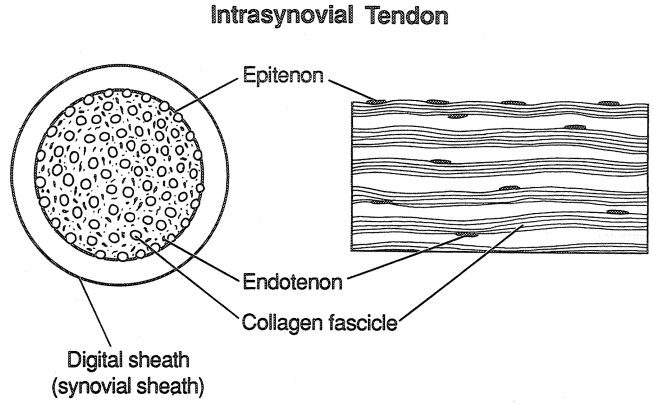

Figure 4
Schematic drawing of a normal intrasynovial tendon.

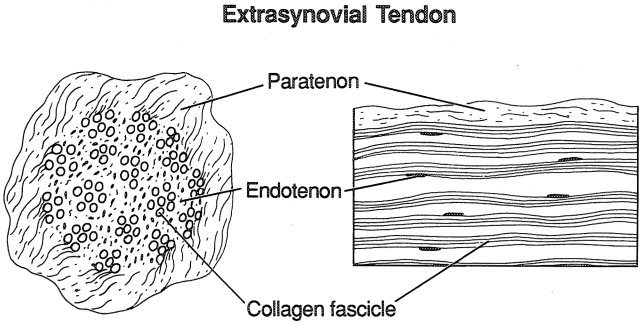

Figure 5
Schematic drawing of a normal extrasynovial tendon.

RESULTS

Normal Tendons

Histology And Ultrastructure

Normal intrasynovial tendons were covered by a single layer of flattened epitenon cells (Figure 4). The underlying collagen bundles were uniform in diameter and parallel in arrangement. Interior fibroblasts were uniformly distributed and blood vessels were few in number. While the extrasynovial tendons differed from the intrasynovial tendons in that their surfaces were covered by thin layers of loose fibrous tissue without lining epitenon cells, the interior structure was similar for both tendons (Figure 5).

The flexor digitorum profundus tendons taken from the hindpaws were similar in size, configuration, and vascularity to flexor digitorum profundus tendons obtained from the forepaw. The proximal portion of the tendons had a well vascularized mesotenon. Ten millimeters from the synovial reflection, in an area that demarcated the transition between the mesotenon proximally and the intrasynovial portion of the tendon distally, intratendinous vessels terminated in distinct microvascular loops. The vessels formed a net-like pattern within the dorsal portion of the tendon. The palmar aspect had fewer vessels and these were oriented in a longitudinal pattern around the midline

vessel. There was a thirty millimeter avascular zone which corresponded to that portion of the normal flexor digitorum profundus tendon located between the second and fourth annular pulleys. Distally, sparse, fine caliber intratendinous vessels originating from the vinculum brevis adjacent to the distal osseous insertion extended proximally for fifteen millimeters to supply the dorsal portion of the tendon. There was an absence of vessels in the palmar aspect of the tendon distally.

The extrasynovial portion of the peroneus longus tendon was vascularized from two sources: intratendinous large caliber longitudinal vessels originating from the musculotendinous junction and an extratendinous system of longitudinally oriented vessels which formed frequent transverse anastomoses with intratendinous vessels. Small transverse branches extended from longitudinal intratendinous vessels at one millimeter intervals. Terminal loops were not seen.

Repair Response

Early repair in the extrasynovial tendons occurred by an ingrowth of connective tissue from the digital sheath. Adhesions, which were circumferential and most prominent around suture sites, extended for the length of the graft proximally, obliterating the gliding surfaces and occupying the spaces between the tendons' gliding surfaces and surrounding tissues. Most of the extrasynovial tendon grafts surface layers could not be distinguished from the inflammatory scar. Adhesions containing a mixture of capillaries and haphazardly arranged fibroblasts appeared to enter the reparative site. While fibroblasts within the repair site demonstrated considerable new collagen fibril formation at the ultrastructural level, there was a lack of longitudinal remodeling.

In contrast, the intrasynovial tendon grafts showed early healing by an orderly proliferation and migration of cells from the epitenon. The surface layer, thickened by proliferating fibroblasts and newly formed vessels, extended symmetrically along the surfaces of both the host tendon and intrasynovial grafts for five to ten millimeters from the repair site. Greater than ten millimeters from the repair site, the epitenon thinned to a more normal appearing single cell layer covering both the graft and host tendon circumferentially. The intrasynovial tendon grafts had smooth surfaces with either no adhesions or slight filmy adhesions attached directly to the repair site. On electron microscopy, the epitenon appeared active and showed characteristics of migrating cells. By twenty-one days, new collagen fibrils were noted to be tightly packed in longitudinally oriented arrays between cuboidal shaped fibroblasts.

The repair response of the tendon grafts in this study appeared to be donor tissue specific. The healing of intrasynovial tendon grafts originated primarily by epitenon cell proliferation and migration to the repair site. This mechanism is similar to that of primarily repaired flexor tendons treated with early postoperative controlled passive motion. Evidence of new collagen fibril formation by ten days, and longitudinal remodeling of collagen fibers by three weeks in the intrasynovial tendon grafts suggested that these grafts were metabolically active, contributing to the repair process.

The extrasynovial tendon grafts demonstrated three consistent morphological differences from the intrasynovial donors. There was extensive adhesion formation to the repair sites and to the surfaces of the grafts. There was no evidence of surface cell proliferation or migration to the repair sites, and finally, the collagen that was formed within the repair site contained a haphazard orientation with respect to the longitudinal axis of the tendon at both ten and five weeks, despite the institution of early controlled mobilization. The findings of this study indicated a capacity for intrinsic repair in the intrasynovial tendon grafts that was not seen in the extrasynovial tendon grafts.

Tendon Graft Incorporation

The incorporation of extrasynovial tendon grafts was characterized by the early formation of circumferential adhesions which ultimately obliterated the gliding surface of the tendon graft (Figure 6). Early observations showed significant cellular dropout from the endotenon area of the graft. Following the ingrowth of adhesions, the grafts were seen to repopulate with pleomorphic fibroblasts and become hypercellular as compared with normal tendons. Electron microscopic examination confirmed the observations made on light microscopy. Degenerating fibroblasts were seen at ten days and three weeks. By six weeks the formation of haphazard new collagen was seen.

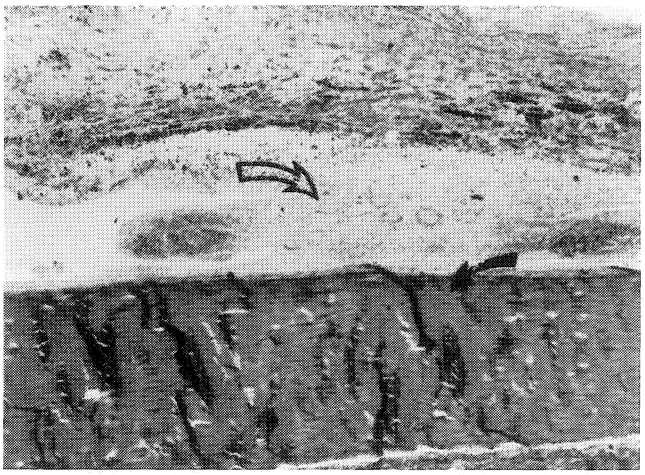

Figure 6
The extrasynovial tendon graft (closed arrow) has a broad fibrovascular adhesion (open arrow). (x231). Illustration reproduced with permission from the Journal of Bone and Joint Surgery.

Intrasynovial tendon grafts incorporated in a different manner. The endotenon cellularity and the gliding surfaces of these grafts were preserved throughout the period of study (Figure 7). Observations at six weeks consistently showed the ingrowth of longitudinal vessels from the area of tendon suture. Electron microscopic examination showed viable internal fibroblasts at each interval of study. Minimal new collagen formation was noted in the intrasynovial tendon graft specimens at six weeks.

Microangiography

The extrasynovial tendon grafts were completely revascularized at two weeks through vascular adhesions arising from the surfaces of the flexor digitorum superficialis and the tendon sheath. These adhesions became progressively more plexiform. By six weeks, the vascularity of the extrasynovial tendon grafts appeared completely integrated with that of the surrounding tissues.

In contrast, the intrasynovial flexor tendon grafts healed without vascular adhesion ingrowth. Primary intrinsic neovascularization originated from both proximal and distal repair sites. The proximal angiogenic response predominated but was limited in linear progression. A profuse surface neovascularization was noted initially, and although intratendinous vessels increased in number during succeeding time intervals, surface vessels continued to predominate. While much less prolific and characterized by finer caliber vessels, the distal neovascularization response showed proximal progress throughout the period of study.

The adhesion-free intrinsic neovascularization of the intrasynovial tendon grafts contrasted sharply with the adhesion dependent process observed following extrasynovial tendon grafting and was consistent with morphological findings indicating increased viability of the intrasynovial tendon grafts.

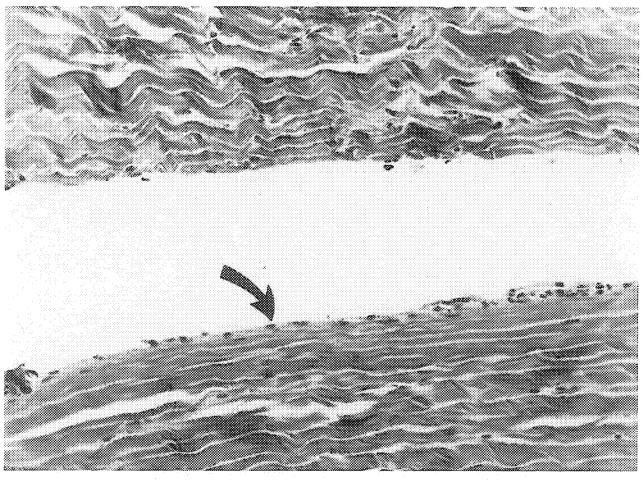

Figure 7
Intrasynovial tendon grafts showed consistent preservation of the gliding surface, which was lined by flattened epitenon cells (closed arrow). (x200). Illustration reproduced with permission from the Journal of Bone and Joint Surgery.

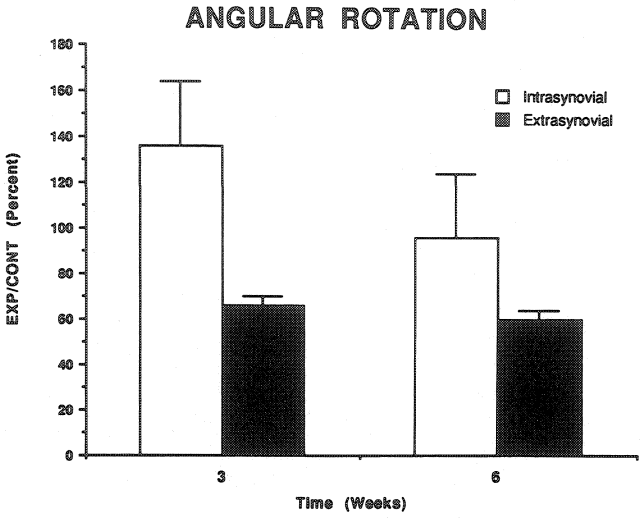

Figure 8
Digital Angular Rotation Following Intrasynovial and Extrasynovial Tendon Grafting. Illustration reproduced with permission from the Journal of Bone and Joint Surgery.

Biochemistry

The concentration of DNA in control segments of intrasynovial and extrasynovial tendon grafts were normalized to their respective controls. At ten days, the DNA content was 1.60 ± 0.15 and 1.08 ± 0.08 in the extrasynovial and intrasynovial tendon grafts respectively. At three weeks the values were 1.7 ± 0.11 and 1.26 ± 0.17. At six weeks the values were 1.61 ± 0.14 and 1.17 ± 0.07. Two way analysis of variance showed a significantly higher ($p < 0.05$) DNA content in the extrasynovial tendon grafts. No statistically significant difference for the effect of time could be determined ($p < 0.05$).

The average incorporation of 3H-thymidine in DNA for extrasynovial and intrasynovial tendon grafts were also normalized to their respective controls. Ten days following tendon grafting, the radioactive uptake was 6.53 ± 1.17 and 4.54 ± 2.20 in the extrasynovial and intrasynovial tendon grafts respectively. At three weeks the values were 6.34 ± 1.25 and 4.68 ± 1.53. At six weeks the values were 7.02 ± 2.07 and 4.79 ± 1.79. Two way analysis of variance showed a statistically significant differences in ($p < 0.05$) DNA content comparing extrasynovial and intrasynovial tendon grafts. No statistically significant difference for the effect of time could be determined ($p < 0.05$).

Biomechanical Testing

The results for angular rotation of the proximal interphalangeal joint are depicted graphically as experimental/control (E/C) in Figure 8. Normalizing the experimental

values to those of the contralateral controls minimized the inter-animal variation of this measurement. At three weeks, the respective values for intrasynovial tendon grafts and extrasynovial tendon grafts were 136% +/- 50% and 66% +/- 17%. At six weeks, the values were 96% +/- 13% and 60% +/- 12%. Two-way analysis of variance revealed that the differences between the intrasynovial and extrasynovial specimens were statistically significant ($p < 0.05$) at both three and six weeks. No significant effects were found for time of healing ($p < 0.05$).

DISCUSSION

The cumulative results of this series of experiments showed significantly different methods of flexor tendon graft repair and incorporation for extrasynovial and intrasynovial donor tendons.

Extrasynovial tendon grafts appear to function primarily as a scaffolding for the early ingrowth of new vessels and cells. These grafts were observed to pass through stages of graft remodeling similar to that described for tendon grafts used to reconstruct the anterior cruciate ligament[3,4,5,14]. Extrasynovial tendon grafts rapidly lost significant interior cellularity. By ten days postoperatively, filmy adhesions had formed along the surfaces of the grafts. With time, the adhesions thickened, increased in number and appeared to be contributing new cells to the grafts. These grafts neovascularized within six weeks, largely through the early ingrowth of new vessels extending into the graft from the synovial sheath. The large increases in DNA synthesis and in DNA content, at all intervals studied, reflected the early and thorough process of cellular substitution during tendon graft incorporation. The dense adhesion formation decreased tendon excursion and limited proximal interphalangeal joint rotation.

In contrast, intrasynovial tendon grafts appeared to remain viable when transferred to the synovial space. Intercalary short segment grafts appeared able to mount a repair response similar to that described for the optimal repair of transected Zone II flexor tendons. The interiors of intrasynovial tendon grafts were histologically similar to control specimens, showing maintenance of cellularity and longitudinal orientation. Intrasynovial tendon grafts revascularized at a slower rate than did extrasynovial tendon grafts and with a distinctly different pattern. New vessels derived from proximal and distal repair sites revascularized the grafts by six weeks. The increases in DNA synthesis and content seen for intrasynovial tendon grafts were consistent with the early thickening of the superficial layer followed subsequently by central revasularization and remodeling.

In contrast to extrasynovial tendon grafts, intrasynovial tendon grafts appeared specially adapted to survive and repair in the environment of synovial sheath. These tissue specific effects explain in part the variable repair responses seen clinically following traditional methods of digital flexor tendon grafting. The incorporation process of intrasynovial tendon grafts allows the gliding surface of the tendon to be preserved, which optimizes tendon excursion and digital flexion. The tissue specific effect of the donor tendon may improve results following staged flexor tendon grafting.

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NON-OSSEOUS COMPLICATIONS FOLLOWING DISTAL RADIUS FRACTURES

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INTRODUCTION

Colles' fractures are often considered lightly and treated inadequately[6,12,13,24]. A poor outcome can result in permanent loss of mobility, weakness, and pain[12]. Bacorn and Kurtzke[6] stated that Colles' fractures result in some residual impairment in hand and wrist function even under the most favorable circumstances, and only 3% of patients are free of disability following this fracture.

Complications following Colles' fracture may be classified as osteoarticular, soft tissue, or both[13]. In a review of 565 patients, Cooney et al.[13] reported a 31% overall complication rate. They noted a positive correlation between fracture severity and the incidence of complications. Zemel also correlated an increased complication rate with inadequate reduction[61].

This paper reviews soft tissue complications following Colles' fractures. Complication categories include:

1. Nerve injury
2. Post-traumatic sympathetic dystrophy
3. Tendon injury
4. Compartment syndrome
5. DeQuervain's tenosynovitis
6. Dupuytren's disease/Palmar fasciitis
7. Shoulder-hand syndrome
8. Hand stiffness

NERVE INJURY

Median Nerve

Median nerve injury is well recognized following Colles' fractures and is the most frequent complication[13,18]. Causes of nerve dysfunction include hematoma within the carpal tunnel or beneath the deep forearm fascia at the fracture site, wrist hyperextension at the time of injury causing nerve stretch, injection of local anesthetic into the fracture hematoma, immobilization in wrist flexion, and excess callus from healing or malunion[20].

The incidence of median nerve injury is variably reported. In 1984, McCarroll[34] reviewed the literature and reported an incidence from 0.2-3.2% depending on the degree of symptoms and the duration of follow-up. Both Wainapel et al.[57] and Stewart[55] noted a 12% incidence; Aro et al.[2], 8%; Cooney et al.[13], 7%; Lynch and Lipscomb[30], 3%; and Stark[53], 5%. Wainapel et al.[57] suggested that electromyography (EMG) was not to be considered more sensitive than the history and physical exam in detecting median nerve dysfunction. It should only be utilized to "confirm" the diagnosis.

Hematoma and swelling following Colles' fracture can cause acute onset of median nerve dysfunction[18,28]. Compression can be located within the carpal tunnel or deep to the forearm fascia near the fracture site[28]. Hematoma in the carpal tunnel causing median nerve compression has been reported due to anticoagulant therapy[7,18,28]. Kongsholm and Olerud[25] stated that hematoma formation can be related to the degree of trauma at the time of fracture, and they related increased tunnel pressures to increased Frykman classification. Lewis[28] drew attention to formation of hematoma and subsequent fibrosis beneath the deep fascia at the level of the fracture site. He presented four cases, stating that this can be an isolated finding or occur in association with compression within the carpal canal. If symptoms of carpal tunnel syndrome are severe enough to warrant decompression and findings in the carpal tunnel are normal, the incision should be extended proximally to include division of the antebrachial fascia over the fracture.

Clinically, it may be impossible to distinguish between direct median nerve contusion or stretch, and nerve compression due to increased carpal tunnel pressure[7,19]. Direct nerve injury is reported, but is felt to be rare due to soft tissue protection offered by the pronator quadratus and the flexor tendons[34]. Acute onset of symptoms can follow reduction maneuvers. To aid in the diagnosis, a good neurologic exam before and after reduction is mandatory[34]. The ability to directly measure increased pressure within the canal and to compare this to established pressure thresholds has increased our recognition of nerve compression following wrist trauma[18].

Post-reduction radiographs are also needed to look for a bony fragment (spike) which may be tenting the nerve[37,42]. Wong and Pho[59] and Kumar[26] noted median nerve impingement at the fracture site due to displaced volar fragments with no actual contact of bone and nerve. Goldie and Powell[20] reported the only case of transfixation of the median nerve by a volar bony spike. All resolved following decompression.

Hematoma block anesthesia is also a potential cause of median nerve compression[25]. Kongsholm and Olerud[25] evaluated thirteen patients with Colles' fractures and ten

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matched controls, measuring carpal tunnel pressures by the wick catheter technique. Measurements prior to manipulation were compared with those following injection of ten milliliters of 1% lidocaine. An average pressure increase of 9.7 +/- 7.4 mmHg was recorded. Increased pressures correlated with fracture severity.

Position of wrist immobilization plays an important role in median nerve dysfunction. Specifically, the Cotton-Loder position (acute wrist flexion, ulnar deviation and forearm pronation) has been implicated by several authors and is strongly discouraged[30,34,35,45,53]. The carpal tunnel pressure is increased with the wrist in neutral position following Colles' fracture and strongly increased in positions of palmar flexion or extension[18,19,25]. Kongsholm and Olerud[25] noted a 0.8 mm Hg linear pressure increase for each degree of volar flexion.

Finally, radial malunion may be a contributing factor[28,45]. Aro et al[2] observed that 85% of patients with late median nerve compression had radial collapse[2]. Wong and Pho[59] suggested an above elbow cast to maintain radial length may be worthwhile in preventing carpal tunnel syndrome.

It is generally agreed that early recognition and treatment of median nerve dysfunction is important in prevention of long term disability[6,17,28,34]. Treatment varies with severity of symptoms. Mild symptoms can be monitored, or the wrist can be brought into a more neutral position, and tight bandages loosened. When concerned, electrodiagnostic studies can be obtained to confirm the diagnosis[57]. Immediate decompression should be considered in the following circumstances:

1. Volar fracture spike fragments are noted on post-reduction films in association with symptoms.
2. Acute unresolving symptoms occur following reduction maneuvers.
3. Significant symptoms persist following adjustment of wrist position from a flexed to a more neutral position[34].
4. Finally, if an open reduction of the radial fracture is being carried out, concomitant median nerve decompression is recommended[34].

Ulnar Nerve

Ulnar nerve dysfunction is much less common than median nerve dysfunction[34]. Bacorn and Kurtzke[6] noted one case of isolated ulnar nerve palsy out of more than two thousand workmen's compensation cases in New York State. Frykman[17] reported a 0.9% incidence. Zoega[62], and Vance and Gelberman[56] each reported three cases of acute isolated ulnar neuropathies. These were noted in younger patients with higher injury traumas and with significant displacement of the distal fracture fragment. Aro et al.[2] reported a 4% late ulnar nerve palsy associated with either Colles' fracture or volar subluxation of the ulnar head.

Several factors make the ulnar nerve less vulnerable to injury. Vance and Gelberman[56] noted that the relationship of the ulnar and median nerves to the bones of the forearm are very nearly identical from the mid-forearm to the wrist area. Both nerves are protected from bone by the pronator quadratus muscle and the digital flexor tendons. In addition, the ulnar nerve has more excursion across the wrist than the median nerve[34], this is because Guyon's canal is shorter in length and more removed from the fracture site. Also, the volar carpal ligament is less substantial than the transverse carpal ligament.

Treatment for these injuries varies and depends on presentation. Acute neuropathies generally require monitoring with good return of function anticipated in four to five months. Robinson et al.[46] recommended aggressive treatment for severe dysfunction and found that electrodiagnostic testing was a helpful predictor of the benefit of surgical decompression with profound changes having the worst results. We recommend decompression for those cases presenting or worsening with closed reduction, as well as late presentations.

Combined median and ulnar nerve injury is exceedingly rare[17,47,51,61]. Siegel and Weiden[51] reported three cases, both in comminuted fractures with persistent edema. No fracture fragments were compressing the nerves in either case. Recovery with conservative treatment was complete in two of the three cases with one patient lost to follow-up.

Radial Nerve

Injury to the superficial branch of the radial nerve following Colles' fractures is extremely rare and is usually of iatrogenic origin. Cooney et al.[13] noted five of 565 Colles' patients with radial nerve injuries, all acute presentations following treatment. Three were caused by improper immobilization, and two by external pin fixation of the fracture. All resolved with removal of the offending compressing or irritating agent. No late radial nerve injuries were noted. McCarroll[34] noted that the radial nerve is uncommonly injured, unless via a direct laceration.

POST-TRAUMATIC SYMPATHETIC DYSTROPHY

Post-traumatic sympathetic dystrophy (PTSD) or reflex sympathetic dystrophy (RSD) is an ill defined syndrome[3,5]. It is characterized by tenderness and pain, burning or aching in nature, and disproportionate to the underlying injury[8,54]. In addition there is vasomotor instability, swelling, and stiffness[5,8,43]. Its etiology is unknown, but it is commonly triggered by minor injury[5,8,27,54].

The incidence of PTSD following Colles' fractures in the literature is controversial, ranging from 2 to 37%[4,5]. Retrospective series[6,17,21,29,43,44] report a lower inci-

dence, while prospective studies[3-5,55] which follow the patient sooner after injury state a higher incidence.

Atkins and co-workers[3-5], suspecting a more common occurrence than realized, studied this extensively. In a prospective study following patients from the time of fracture, they noted PTSD in twenty-seven of 109 (25%)[5] patients at nine weeks after fracture. A second follow-up at six months noted a decreased incidence to 17% with eight patients lost to follow-up. A repeat study utilizing dolorimetry[4], an objective instrument that quantitates tenderness, noted an incidence of tenderness in twenty-three of sixty (37%) patients at two to six weeks after cast removal. There appears to be no correlation of occurrence of PTSD with patient's age, severity of fracture, number of reductions, or adequacy of reduction[4].

Plewes[43] noted the plain radiographic appearance of PTSD to be a spotty rarefaction (Sudeck's atrophy). This is noted mostly in the phalanges, metacarpals, carpus, and distal radius and ulna. The shafts of the bones are only slightly affected. These changes develop six to eight weeks following injury. This in contrast to the generalized ground glass appearance seen with disuse osteoporosis which requires longer to develop.

Tests available to substantiate this syndrome include bone scan, thermography, and sympathetic blockade[1,8]. Bone scans will show diffusely increased uptake on the delayed phase[31]. Thermography will display abnormal heat patterns not conforming to typical neurological topography. Sympathetic blockade is useful as both a diagnostic and treatment adjunct.

The key to treatment is early recognition. Plewes[43] felt that if the diagnosis was made within six weeks of onset, the hand responded well to conservative measures. These can include heat (105-110°), elevation, graded function, sympathetic blockade, or vigorous desensitization. Stein reported five cases of Sudeck's Syndrome following Colles' fracture, all responding to median nerve decompression at the wrist[54]. Surgical sympathectomy is indicated only when all other modalities have failed and there has been a positive response to sympathetic blocks. Chronic disability is often related to treatment delay[8].

TENDON INJURY

Extensor Tendons

Extensor tendon ruptures as a complication of Colles' fractures occur three times more commonly than flexor tendons[9]. The extensor pollicis longus (EPL) is the most common[11,61]. The site of EPL rupture following Colles' fractures is proximal to the extensor hood. The time interval between fracture and rupture is usually one to twelve months[32]. Engknist and Lundborg[16] did microangiographic studies in cadavers which showed that the EPL in the region of Lister's tubercle was poorly vascularized. They postulated that a hematoma within the third dorsal compartment could interfere with tendon nutrition and result in delayed rupture. McMaster[36] concluded that a partial severance of the tendon at the time of fracture resulted in incomplete healing and ultimate rupture. He placed less emphasis on the theory of disruptive tendon blood supply leading to local tendon necrosis. Sadr[49] attributed EPL rupture to crush ischemia, attrition over a bony spur or callus, or adherence to callus. Helal et al.[23] in sixteen cases noted a higher incidence of EPL ruptures with undisplaced Colles' fractures. At surgical exploration, the extensor retinaculum was still intact and held the tendons tightly against the dorsal radius. Disruption was felt to be attritional and occurred at the distal edge of the retinaculum. In displaced fractures the extensor retinaculum was torn loose from the radius, which allowed the tendons to be separated from the fracture site.

Flexor Tendons

Ruptures of flexor tendons occur much less often than ruptures of extensor tendons[9,10,48,52,60]. Etiologies include rheumatoid arthritis, forearm fracture, wrist dislocation, contusion, athletic injury, carpal bone anomaly, or tenosynovitis[41]. Anatomically, the flexor tendons are separated from the distal radius by the pronator quadratus, which tends to protect the tendons from insult and subsequent rupture[52,60]. The high tensile strength and flexibility of these tendons also protects them. Normal tendons have a built-in reserve of strength and excessive force will not cause rupture, the bony insertion or the musculo-tendonous junction will give way instead. For the tendon to become the "weak link" at least 50% of its fibers must be cut[22]. Profundus ruptures occur five times more frequently than sublimis ruptures, with the ring and middle fingers being most common[9].

Boyes et al.[9] in a review of eighty flexor tendon ruptures documented only two (3%) flexor tendon ruptures following a wrist fracture. Southmayd et al.[52] reported one case of immediate flexor digitorum profundus (FDP) and flexor digitorum superficialis (FDS) rupture to the index finger. This was thought to be secondary to a volar bony spike which had penetrated the pronator quadratus muscle. Younger and DeFiore[60] in 1977 noted that only three cases of flexor tendon rupture associated with Colles' fracture had been reported in the literature prior to their report of one case. They noted rupture of the FDP and FDS to the small finger along with FDS of the ring finger secondary to attrition over the ulnar head. Diamond and Newman[14] in 1987 reported a case of multiple flexor tendon ruptures thought to be due to compromised tendon blood supply and eventual rupture due to prolonged pressure over a malunited fracture.

Rymaszewski and Walker[48] also reported a case of attritional rupture associated with incomplete healing of a partial tendon laceration.

Treatment of these injuries is beyond the scope of this paper. Secondary tenorrhaphy is usually not feasible, making tendon transfer or intercalated graft the preferred treatment.

COMPARTMENT SYNDROME

An acute compartment syndrome along with its possible sequelae of Volkmann's ischemic contracture is uncommon following Colles' fracture[13,33,40,50]. Cooney et al.[13] noted that three of their four patients with an established Volkmann's contracture following a Colles' fracture had constricting casts retained despite persistent complaints of pain. Continuous use of analgesics had masked the pain in two of the patients. Patients should be monitored closely, with frequent neurovascular checks and cautious use of analgesic medication. Compartment pressures should be measured and treated promptly with fasciotomy.

DEQUERVAIN'S TENOSYNOVITIS

Stenosing tenosynovitis following a Colles' fracture is extremely rare. Only one study mentioned this as a possible complication[61]. Due to the proximity of the first dorsal compartment pulley to the fracture site, edema and hematoma can lead to thickening of the pulley, synovitis, and scar. The tendons of the abductor pollicis longus and extensor pollicis brevis can become constricted by surrounding synovitis and scar. As with DeQuervain's disease due to other etiologies, cortisone injection and/or splinting are first-line treatments. If results are less than adequate, division of the thickened pulley is suggested.

DUPUYTREN'S DISEASE/PALMAR FASCIITIS

The literature regarding Dupuytren's disease in association with Colles' fracture is sparse. Bacorn and Kurtzke[6] noted a 0.2% incidence. Stewart et al.[55] reported an incidence of 4% at three months after fracture and 11% at six months. All cases were mild and mostly nodular in variety, with little progression as late as twenty-seven months following fracture. This association was noted most frequently among older female patients. There was no correlation with fracture severity. The nodules are usually *painful* in contrast to palmar fibromatosis unassociated with Colles' fractures. Treatment is decidedly nonoperative, as the nodules usually resolve or become asymptomatic over time.

SHOULDER-HAND-FINGER SYNDROME

This condition is highly complex and a concise definition is difficult to find. Moberg[38] describes it as having two components, a shoulder component and a hand-finger component. The shoulder is affected primarily by either a traumatic event to the upper extremity or by a visceral disease such as a heart condition[21]. Hand and finger involvement follows the shoulder symptoms and the elbow is never affected. Some feel it is a clinical form of RSD; however, the possible visceral etiology, less hand pain, and fingers stiff in extension rather than flexion help distinguish this syndrome from RSD. Others attribute it to a decrease in the venous and lymphatic pumping mechanism of the upper extremity brought on by limitation of active shoulder motion[17,38]. Causes following Colles' fracture include excessive wrist flexion with median nerve dysfunction, radial nerve irritation due to pin irritation, severely displaced and unreduced fractures, or prolonged shoulder immobilization[13].

The incidence ranges from 0.1 to 10%[6,13,17]. Frykman[17] had the best controlled study and noted a 2.1% incidence. He noted that the condition never occurred in patients less than forty years and was most common after age fifty. He also noted a higher incidence (50%) in patients requiring repeated fracture reduction compared with only 4% in those requiring a single reduction. Emotional instability may also play an important role. Along with inactivity, Frykman included this as one of the two components necessary for development of this syndrome. One-third of the patients with this complication were emotionally unstable.

The clinical course is in three stages[17]. The first stage begins less than twenty days following trauma. It involves burning shoulder pain followed a short time later by hand and finger pain. There is also a loss of passive finger motion and flattening of the digital flexor creases. Stage two is characterized by the absence of shoulder pain, decreased hand and finger pain, and changes in the hand similar to palmar fibromatosis. Stage three has no pain but "frozen" hand and finger motion due to fibrosis and contractures. Each of the first two stages can last three to six months, with the last stage possibly leaving irreversible changes. Resolution can also occur spontaneously or with treatment in each stage.

Treatment is difficult and depends on what one considers the cause[17]. If it is felt that the etiology is mechanical, treatment is mainly preventative. Frykman stated that he kept the incidence very low "simply by instituting measures to promote the circulation right from the start". Use of a sling is strongly discouraged because it limits shoulder motion[38,58]. Early active motion of the shoulder and hand are encouraged[15]. Fingers should remain free from immobilization in the cast or splint[58]. If patients are reluctant to begin early motion, monitored physical therapy sessions are initiated[17]. After onset of the syndrome, treatment is generally geared toward vigorous, progressive functional

motion of the involved upper extremity[58]. If one feels that this is a variant of RSD, treatment should be directed toward decreasing the sympathetic output to the upper extremity via chemical sympathetic blockades or sympathectomy.

HAND STIFFNESS

Hand stiffness following Colles' fracture, although not considered a major complication, can cause significant morbidity[6,13,21,39]. It may be due to edema, immobility, or pain[39]. The incidence is unclear. Green and Gray[21] noted "finger stiffness" in only three of seventy-five private practice patients. Morey[39], on the other hand, stated that hand stiffness following distal upper extremity fractures is a common problem. Bacorn and Kurtzke[6] noted a 48% incidence in more than two thousand Worker's Compensation cases of "restricted" motion of digits, although they did not suggest a cause for the stiffness. Flexion defects were noted twice as often as extension. The distal interphalangeal joints were the most commonly affected with the index finger being the most and the thumb being the least common.

Digital immobility may be due to fracture pain or poor casting techniques[13,39]. Pain usually resolves fairly soon with proper fracture immobilization and early digit motion. Casts which block metacarpophalangeal (MP) flexion or push the thumb metacarpal into an adducted position may result in an MP extension contracture or a thumb adduction contracture respectively. Most patients tolerate the blocked motion and do not complain to their physician.

Aggressive range of motion exercise and the use of elastic garments to control edema are important measures in preventing digital stiffness[39]. In the Bacorn and Kurtzke[6] review, physical therapy averaged four months. It did not affect the final loss of function. Rather, they felt that the willingness of the patient to perform early, frequent wrist and finger exercises played a greater role in reducing final disability. They also noted agreement from others on this opinion[21]. In contrast, Morey[39] felt that a team approach of physical and occupational therapy lessened the time needed for full recovery.

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MRI IN THE DIAGNOSIS OF KNEE INJURIES

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INTRODUCTION

Trauma to the knee may result in injury to the menisci, cartilage, ligaments or bone. Physical examination of the painful knee in the acute phase may be difficult and frequently imaging studies are required to aid in the assessment of these injuries. Plain radiography is the most readily available and least expensive imaging modality but it lacks sensitivity for the detection of meniscus, cartilage, bone marrow and ligamentous injuries. MR imaging is now accepted as the modality of choice for the diagnosis of internal knee derangements, and in most centers in the U.S. it has replaced arthrography and diagnostic arthroscopy[11,22]. MR has been shown to be particularly useful in assessing meniscal abnormalities,[3,4,14,21,26] however its role in the evaluation of cartilage and ligament injuries is still evolving. MR imaging does not utilize ionizing radiation and therefore it is entirely safe; it is also noninvasive, painless, and allows acquisition of images in multiple planes without repositioning the patient. In addition, MR provides excellent spatial and contrast resolution of both intra- and extra-articular structures.

TECHNICAL ASPECTS

A magnet with high field strength (1.5 T) is preferable for the study of joints. Currently, the spin-echo pulse (SE) sequence is the most commonly used pulse sequence in MR imaging of the knee. To examine the knee with MR, the knee is placed comfortably in an extremity coil in about 15 degrees of external rotation. Our protocol includes an angled sagittal proton density and T2- weighted SE images with repetition time of 2000 msec and echo time of 20 and 80 msec respectively. Coronal T1-weighted SE images are also obtained with a repetition time (TR) of 600 msec and echo time (TE) of 20 msec. A 16 cm field of view is used with an acquisition matrix of 256 X 192 and 2 NEX. Section thickness is 3 mm with 1 mm interslice gap. Laser printed images are obtained with regular windows. The magnification factors for image printing are between 1.25 and 2.0. In certain difficult cases the images are viewed on a TV monitor at a different window level.

T1-weighted and proton density images are better than T2-weighted images for detecting meniscal tears. The sagittal images demonstrate both menisci, articular cartilage, both cruciate ligaments, the quadriceps mechanism, popliteal fossa, infrapatellar fat pad, suprapatellar pouch and patella (Figure 1). The coronal images are particularly useful in the assessment of the medial and lateral collateral ligaments, articular cartilage and bone marrow. The sagittal images provide the most information when assessing meniscal injuries; coronal images serve to confirm the findings seen on the sagittal images. The axial plane is used occasionally to visualize abnormalities in the articular cartilage of the patella and patellar ligament.

As alternatives to spin-echo pulse sequences, the gradient refocused echo (GRE) sequences have achieved wide spread acceptance because they can provide effective T2-weighted images more rapidly than the conventional spin-echo sequences. These fast sequences appear in the literature under different acronyms, such as GRASS (gradient refocused acquisition in a steady state), FLASH (fast low-angle shot) and FISP (fast imaging with steady procession). The tissue contrast in these fast scanning techniques is dependant on parameters such as the TE and to a lesser extent the TR, but the most important variable is the flip angle. Obtaining images of complex structures such as the meniscus or the anterior cruciate ligament in nonorthogonal planes is sometime advantageous. Fast scan imaging has allowed the meniscus to be viewed in radial cross-section with images obtained perpendicular to the long axis of the meniscus. Theoretically, radial images are thought to better depict the far posterior and anterior zones of the meniscus. Tears near the apex of the meniscus (parrot beak tears) are also believed to be easier to visualize with radial images. In a recent study, Quinn et al.[24] compared radial multiplanar gradient recalled-echo (MPGRE) imaging with sagittal spin-echo imaging. They found no statistical difference in the accuracy of interpretations using radial versus sagittal images in assessing meniscal tears. They also noted that sagittal images were more helpful in the evaluation of meniscocapsular attachment. Overall, the radial images increased the conspicuity of meniscal tears but at the expense of anatomic detail.

Volume acquisition or three-dimensional Fourier transform (3DFT) imaging is another application of fast imaging which has attracted great interest. With this technique

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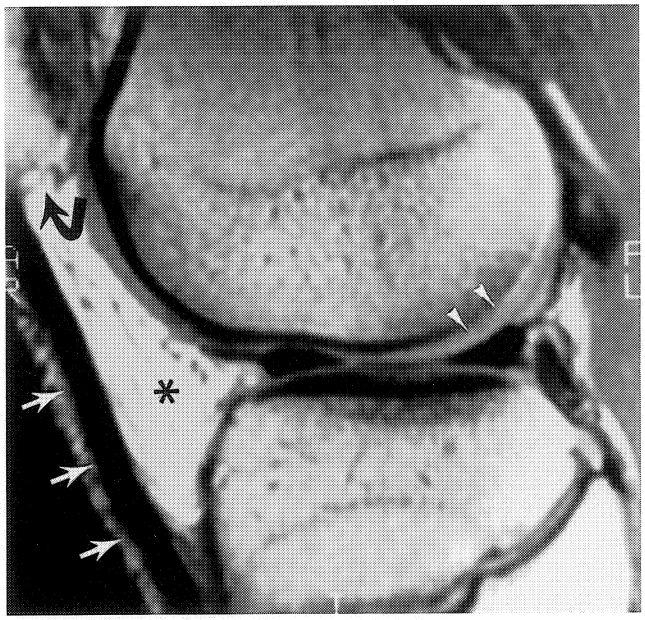
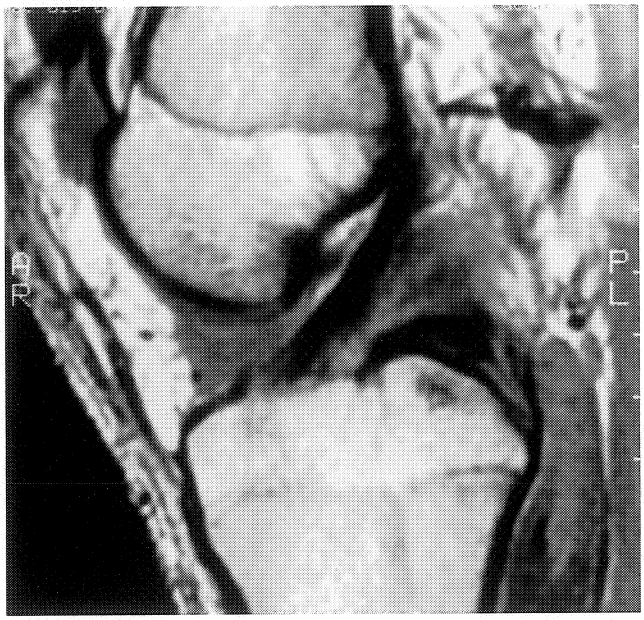
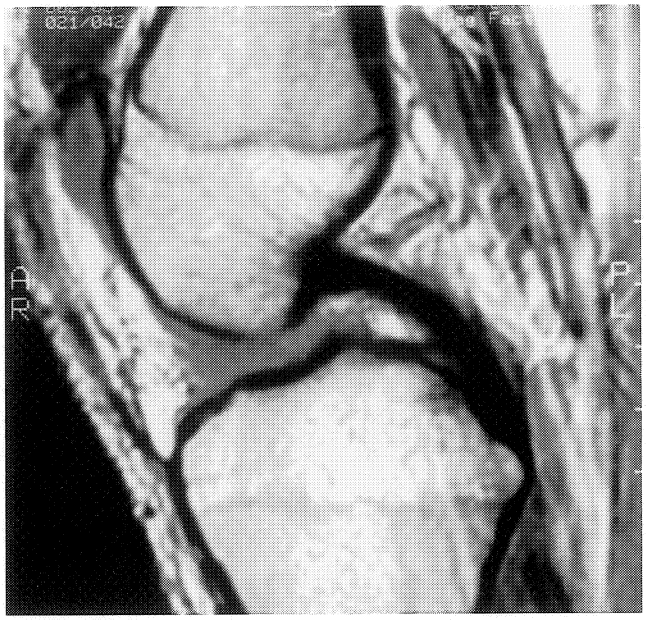


1A



1B



1C

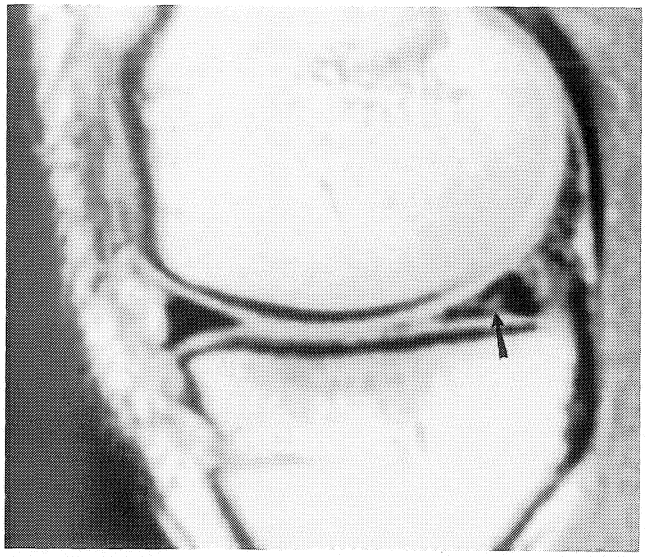
Figure 1 (A-C).
Normal knee anatomy. Sagittal proton density images (TR = 2000 msec, TE = 20 msec). (A) Normal signal void within the menisci. The adjacent articular cartillage (arrowheads) is higher in signal intensity than the menisci, but less than fat in the intrapatellar fat pad (asterisk). The patellar tendon is also seen as a line of signal void (white arrows) inferior to the patella (curved arrow). (B) Normal linear appearance of the anterior cruciate ligament. (C) Normal curved appearance of the posterior cruciate.

slices 1 mm or less in thickness may be acquired, in any plane, and without an interslice gap[11,14,25,27,28,31]. Several studies comparing the conventional two-dimensional Fourier transform (2DFT) spin echo (SE) sequences with three-dimensional Fourier transform (3DFT) sequences failed to show any convincing evidence of the superiority of one technique over the other[11,25,28]. Araki et al.[1] used axial images obtained by 3DFT GRASS pulse sequence to evaluate meniscal tears. He then compared them with images obtained by 2DFT spin echo pulse sequence and found no statistically significant difference in accuracy between the two techniques.

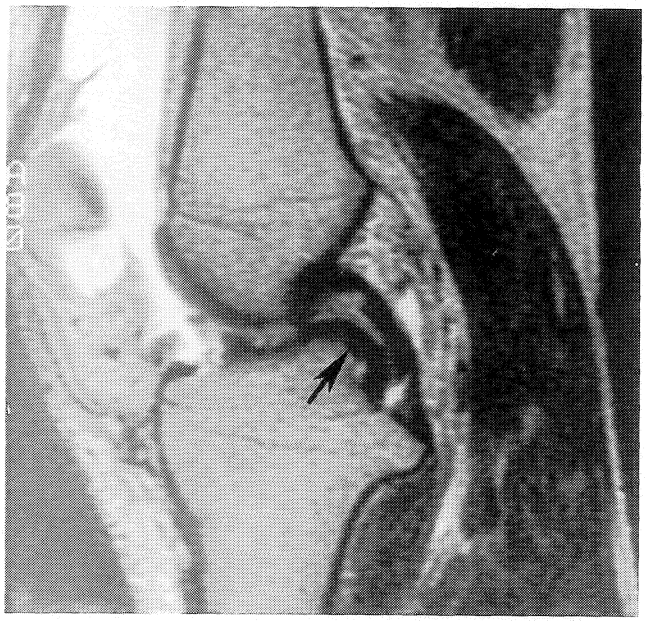
MENISCI

The menisci are seen on MRI as wedge-shaped structures which are of low signal intensity (black) in nearly all pulse sequences[2]. This is in contradistinction with the relatively high signal intensity of the surrounding articular cartilage and the periarticular fat (Figure 1). Li et al. explained this difference in MR appearance of the menisci and articular cartilage to be due to differences in their collagen and water content. Articular (hyaline) cartilage contains approximately 75 to 80% water and about half of its dry weight is collagen type II. Menisci are mainly fibrocartilage and contain type I collagen which is also present in ligaments and tendons. Type II collagen contains higher levels of hydroxylysine which increases the hydrophilia of its matrix . The high water content of the articular cartilage probably accounts for their fairly high signal intensity on MRI.

The MR diagnosis of meniscal tears hinges on the presence of specific patterns of high signal intensities within the meniscus or abnormalities in meniscal contour. After the age of twenty, areas of increased signal intensity frequently appear within the substance of the meniscus.



2A



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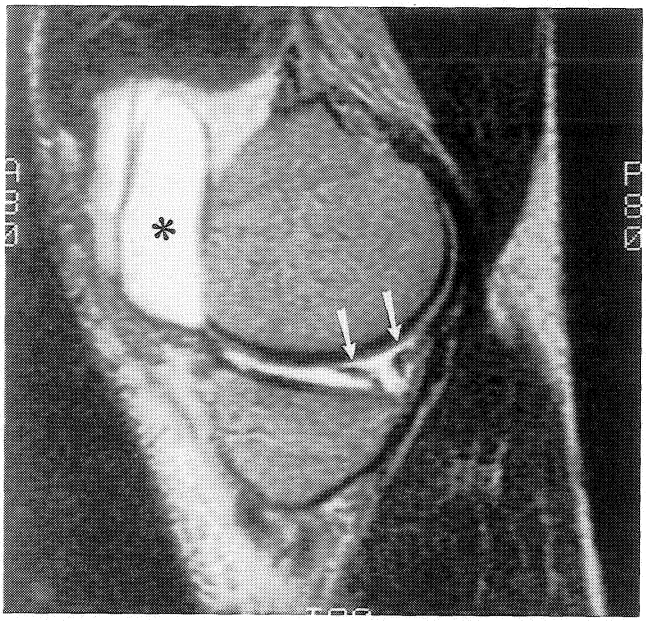


2C

Figure 2 (A-C).
(A) Sagittal proton density image (TR = 2000 msec, TE = 20 msec) showing a grade 3 tear of the posterior horn of the medial menisus (arrow). Note how the abnormally increased signal extends to the articular surface. Midline (B) and far medial (C) sagittal T2-weighted images (TR = 2000 msec, TE = 80 msec) of a displaced bucket handle meniscal tear. On the midline image, the meniscal fragment (black arrow) lies underneath the posterior cruciate ligament. On the far medial section (C) the meniscus is seen to be separated into two parts (white arrows). An effusion is present within the knee (asterisk).

These areas have been shown histologically to represent intrameniscal mucoid degeneration without tear[15,29]. Since not all signals within a meniscus represent clinical tears, grading systems were developed to improve the specificity of MRI and in order to predict surgically significant lesions[18]. A widely accepted grading system, both clinically and also as a research tool, is the one proposed by Lotysch et al. in 1986. Several modifications of this scheme have been developed since then, but for the most part the basic grades are essentially similar[4,6,10,18,21]. Grade 0 indicates a meniscus that has uniformly low signal intensity (Figure 1A). Grade 1 represents a globular signal intensity within the meniscus which is not contiguous with an articular surface; grade 2 represents a linear increased signal intensity which does not extend to the articular surface; grade 3 is given when the increased signal reaches the articular surface of the meniscus (Figure 2A). Grade 4 indicates deformity in the contour of the meniscus such as amputation or blunting of the free edge (Figure 2B and C)[10]. Grades 1 and 2 signals are rarely associated with surgically detectable tears, whereas grade 3 signals are associated with tears in over 90% of cases[4,12,21,26]. Grade 4 is typically seen with acute bucket handle tears (Figure 2B and C).

There are normal structures within the knee joint which can masquerade as meniscus tears. The transverse ligament (Figure 3) and lateral inferior geniculate artery can present as pseudotears in the region of the anterior horn of the lateral meniscus, whereas the popliteus tendon and meniscofemoral ligament of Wrisberg can present as pseudotears in the region of the posterior horn of the lateral meniscus[13,32,33,34] (Figure 8B).

LIGAMENTS

Most researchers believe the anterior cruciate ligament (ACL) consists of two or three separate fiber bundles which often have different signal intensities on MRI. The

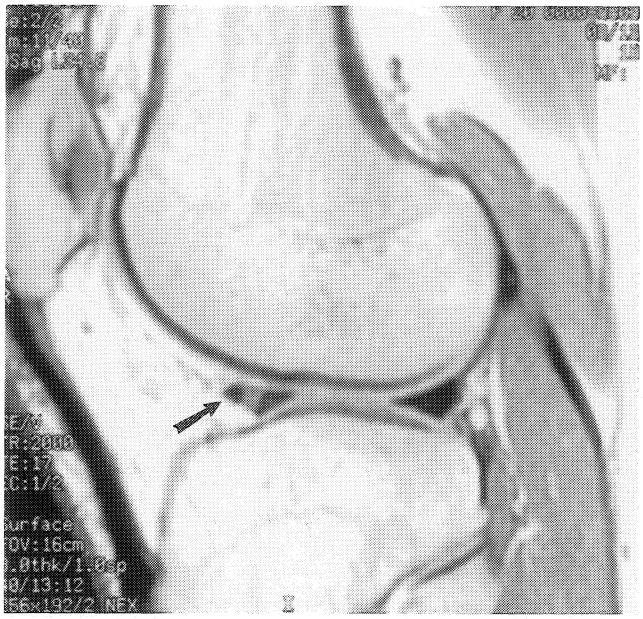
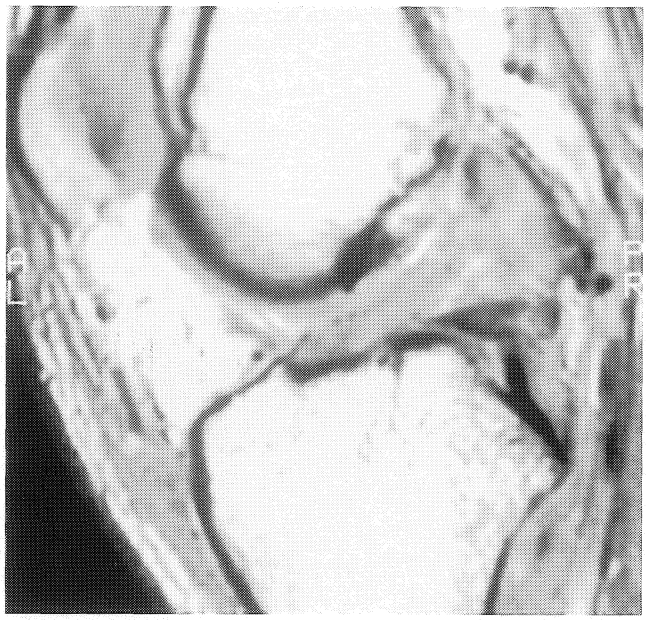
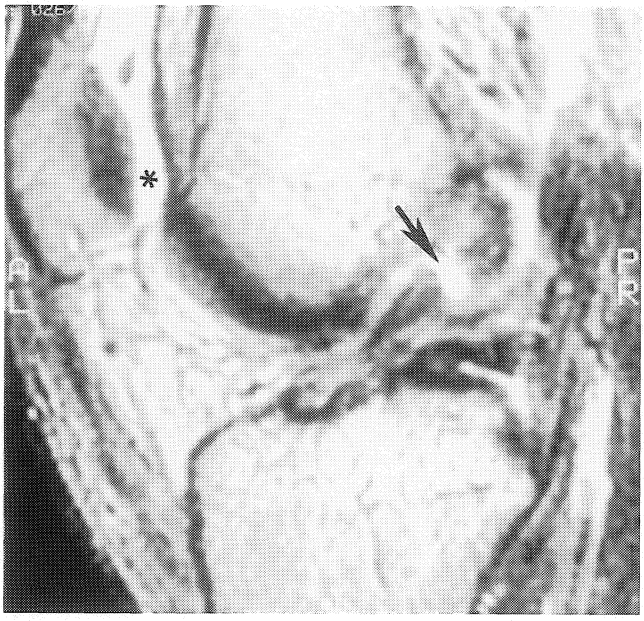


Figure 3.
Transverse meniscal ligament. Proton density (TR = 2000 msec, TE = 20 msec) sagittal image. The transverse meniscal ligament (arrow) may mimick a tear of the anterior horn of the lateral meniscus.

ACL consistently has higher signal intensity than the posterior cruciate ligament (PCL) on MR images. Some of the differences in signal intensity between the ACL and PCL may be explained by the oblique course of the ACL relative to the sagittal plane and also by differences in ligament architecture[15]. Vahey et al.[32] defined an intact ACL on MRI as being a continuous linear band with low signal intensity that demonstrates normal orientation. Forceful valgus-external rotation injury is the most common mechanism of injury to the ACL. In a large series, 79% of tears of the medial meniscus were accompanied by tears of the ACL, as were 69% of all cases of knee injury that were brought to surgery[30]. Tears of the ACL typically occur in the middle portion rather than at the points of ligamentous attachment to the bone[16]. On MRI an acute ACL tear is suggested if the ligament is focally or diffusely disrupted and soft tissue edema is present[32] (Figure 4). Bone impaction sites are also noted in the majority of acute complete ACL tears. These are located on the posterolateral portion of the lateral tibial plateau, usually extending under the meniscus, as well as on the lateral femoral condyle between its middle and anterior portions[21-23]. These bone lesions show regions of low signal intensity in the subchondral medullary bone on T1-weighted images and have increased signal intensity on T2-weighted images (Figure 5). The precise pathologic correlate for the alterations in the marrow signal is unknown; however, it could represent edema, blood hyperemia of the marrow, or



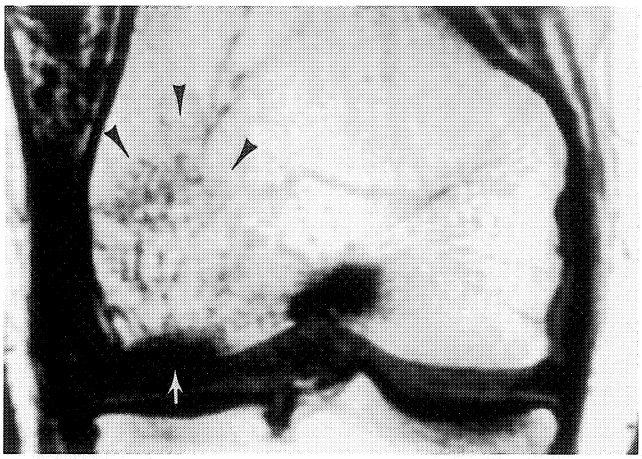
4A



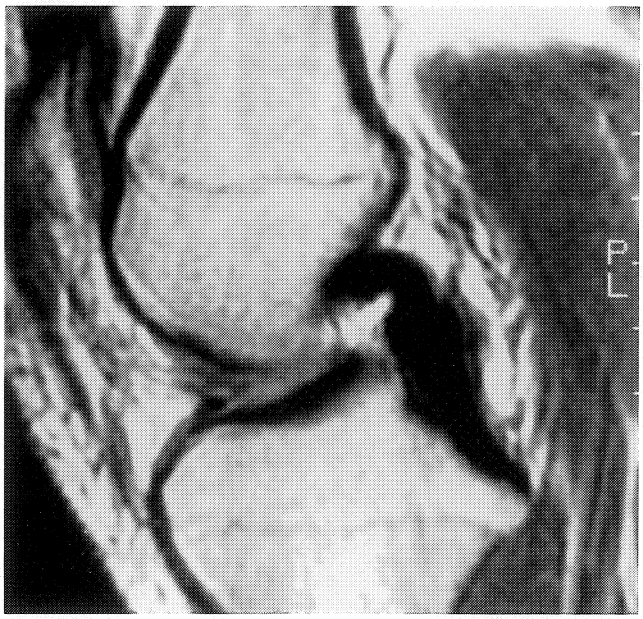
4B

Figure 4 (A-B).
Acute anterior cruciate ligament tear. (A) Sagittal proton density (TR = 2000 msec, TE = 20 msec) shows a lack of normal signal void within the ACL and a cloud-like area of medium signal intensity within the femoral notch representing edema. (B) The T2-weighted image (TR = 2000 msec, TE = 20 msec) shows fluid intensity signal at the femoral site of attachment of the ACL (arrow). There is also fluid within the suprapatellar bursa (asterisk).

microfractures in the trabecular bone. A chronic ACL tear is suggested if the ligament is focally or diffusely disrupted without evidence of significant edema (Figure 6). The ligament is also considered chronically torn even if it



5A



5B

Figure 5 (A-B).
Bone impaction site. (A) Coronal proton-density (TR = 2000, TE = 20) image showing decreased signal subchondrally in the lateral femoral condyle (arrow). An area of less dramatic decreased marrow signal more proximally (black arrowheads) indicates marrow edema. (B) T2-weighted sagittal image shows deformation of the cortical surface (white arrow) with increased signal in the region of marrow edema (white arrowheads).

appears as a continuous band but demonstrates significant angulation[32]. Lee et al. and Boeree and Ackroyd[3,17] have demonstrated that the sigmoid bowing or curling up of the PCL is a sensitive secondary sign of ACL tear (Figure 7). Murphy et al.[23] proposed MRI signs for the diagnosis of partial ACL tears but the reliability and validity of these signs have never been tested. Glashow et al.[8] stated that they were unable to differentiate by MRI between a partial and complete tear of the anterior cruciate ligament. The real challenge for investigators in the future will be to outline sound MR criteria which can differentiate complete from partial ACL tears both acutely and in the

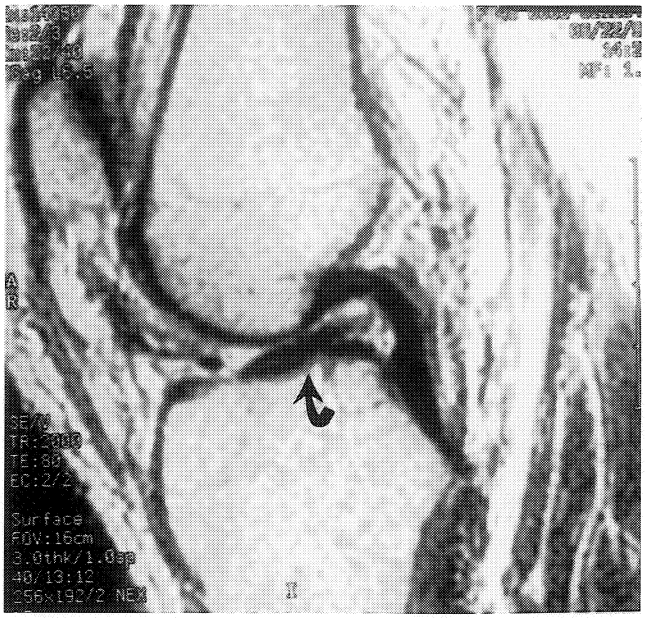


Figure 6.
Chronic anterior cruciate ligament tear. Proton density (TR = 2000 msec, TE = 20 msec) sagittal image showing an abnormal, more horizontal course of the ACL (curved arrow). Note also the lack of joint effusion or increased soft-tissue signal surrounding the ligament.

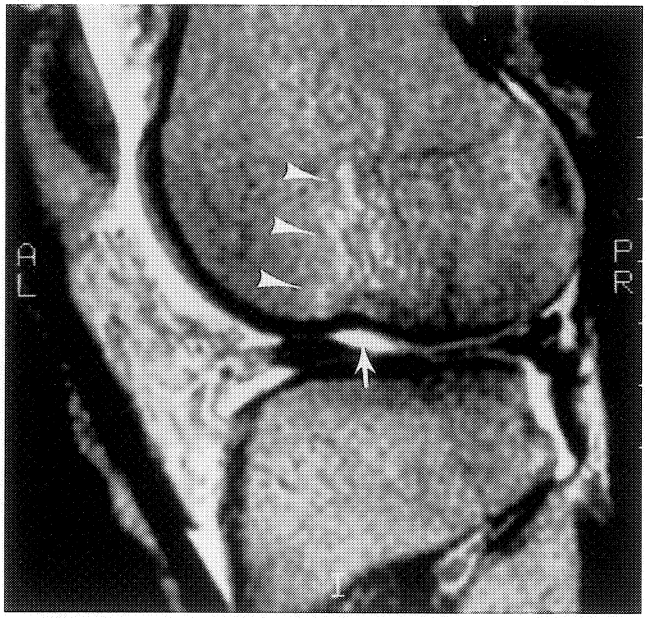
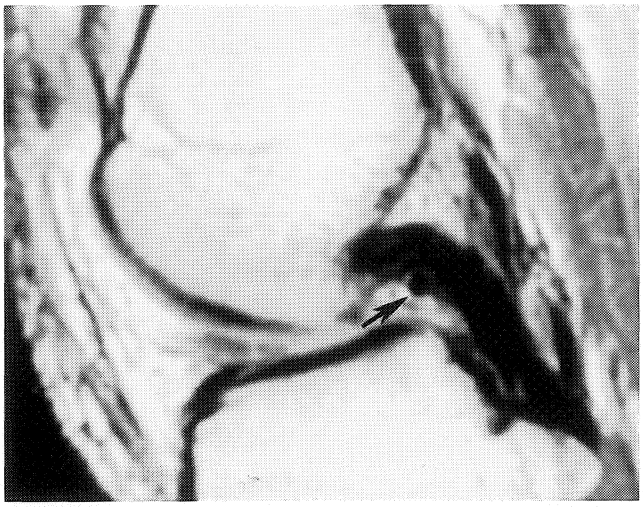


Figure 7.
Anterior cruciate ligament disruption with laxity of the posterior cruciate ligament. Sagittal proton density (TR = 2000 msec, TE = 20 msec) image showing an abnormal "sigmoid" shape of the PCL secondary to disruption of the ACL.



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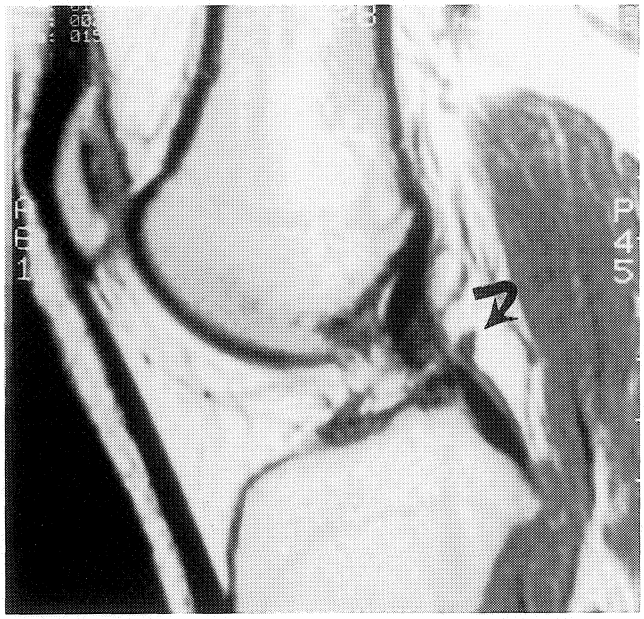
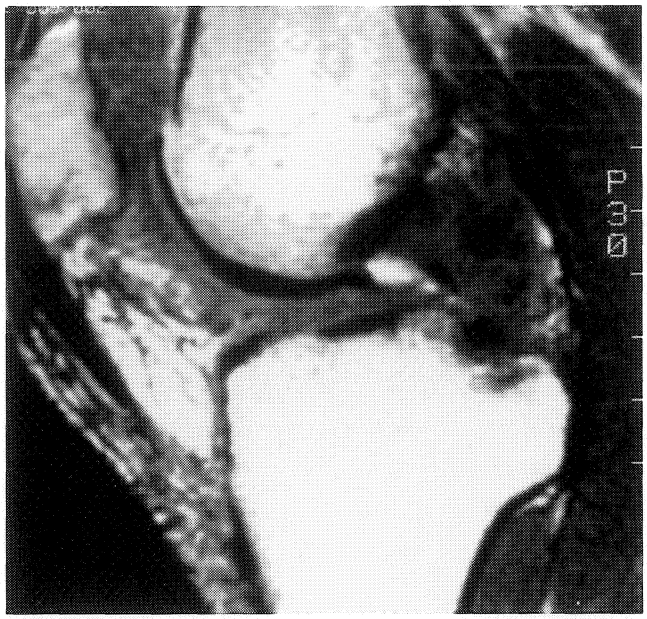
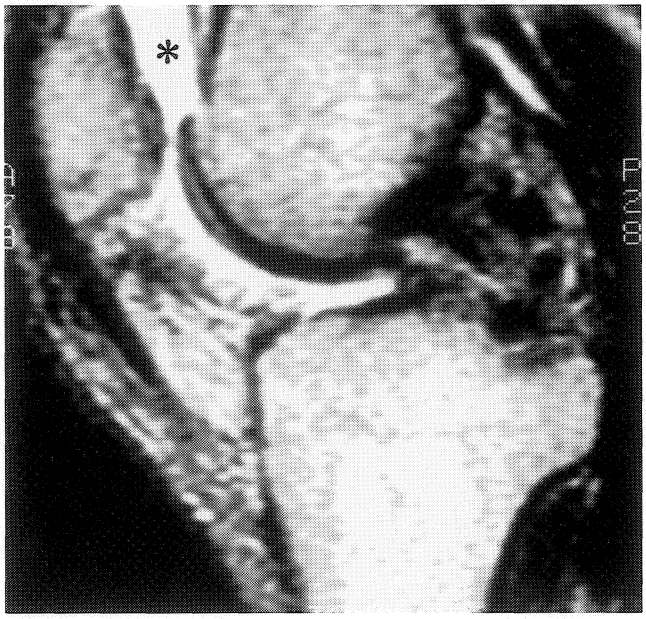


8B

Figure 8 (A-B).
The menisco-femoral ligaments. Sagittal proton density (TR = 2000 msec, TE = 20 msec) images showing (A) the anterior meniscofemoral ligament of Humphrey (arrow) and (B) the posterior meniscofemoral ligament of Wrisberg (curved arrow).



9A



9B

Figure 9 (A-B).
Acute posterior cruciate ligament tear. (A) Sagittal proton density (TR = 2000 msec, TE = 20 msec) image showing discontinuity and increased size and signal of the PCL. (B) T2-weighted (TR = 2000 msec, TE = 80 msec) image demonstrating fragmentation of the PCL and a joint effusion (asterisk).

chronic phase. Vahey et al.[32] have reported the sensitivity, specificity, and accuracy of MRI in the diagnosis of ACL tears to exceed 90%.

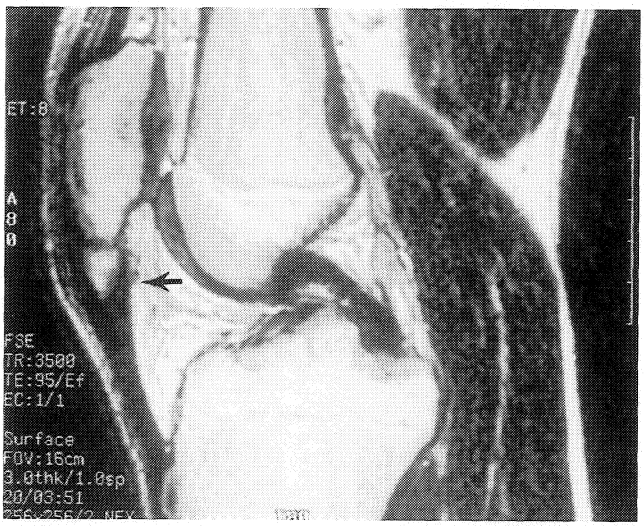
The PCL is larger and twice as strong as the ACL or medial collateral ligament[16]. Disruption of the PCL implies a severe injury, usually a part of multiligamentous knee trauma such as combined ACL, MCL, and meniscal injuries[9]. The PCL is best evaluated on sagittal images and a normal ligament has a very low signal intensity on all sequences. An anterior meniscofemoral ligament (Humphrey's ligament) (Figure 8A) is seen in 24% of of cases, posterior mensicofemoral ligament (Wrisberg's ligament) (Figure 8B) in 23%, and both in 11%. The PCL has an arcuate shape. PCL insufficiency may result from either a tear within the substance of the ligament or an avulsion from its bony insertion at the tibia[20]. The MR diagnosis of a PCL tear is made when anatomic disruption of the

ligament is associated with changes in signal intensity within the ligament (Figure 9).

It is also possible to evaluate the patellar tendon on standard MR images of the knee. The normal patellar tendon has uniformly low signal intensity on T1-, T2, and proton density- weighted images; its AP diameter increases slightly from proximal to distal and it displays distinct margins. The AP diameter of a normal tendon, in its proximal portion, should not exceed 7 mm[7]. In patellar tendonitis, the tendon shows increased signal intensity on T1-, T2-, and proton density-weighted images; it also shows increased AP diameter proximally. The margins of the affected tendon are indistinct, especially posterior to the thickened segment (Figure 10). Females generally have thicker proximal tendons than males[7].

Very little has been written about MRI of the collateral ligaments in the knee. Dungan et al.[5] studied the accuracy of some MRI criteria in predicting medial collateral ligament (MCL) tears and compared these results with the clinical findings. They found focal disruption of the MCL, increased signal intensity on T2-weighted images, trabecular injury, and subcutaneous edema to be predictive of a tear (Figure 11). MRI however, was shown to be of limited value in determining the grade of the MCL injury.

In conclusion, MRI is an effective noninvasive method for the detection of multiple soft tissue injuries within the knee which are undetectable by other modalities. MR has practically replaced knee arthrography in the evaluation of internal knee derangements. Future investigation will likely focus on newer pulse sequences for increased speed and the evaluation of the articular cartilage, as well as more fully defining the reliablity and validity of this technique.



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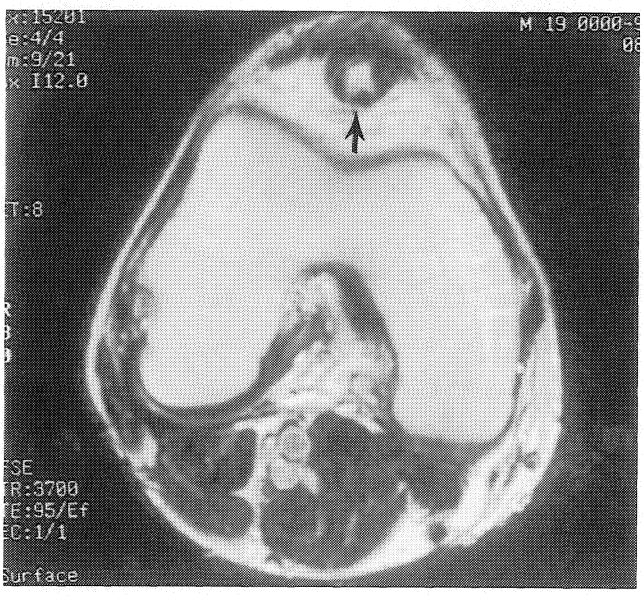

10B

Figure 10 (A-B).
Patellar tendinitis. (A) T2-weighted sagittal image showing increased thickness of the proximal patellar tendon with increased signal. The posterior margin of the tendon is indistinct (arrow). (B) T2-weighted axial image showing focal enlargement and increased signal of the tendon.

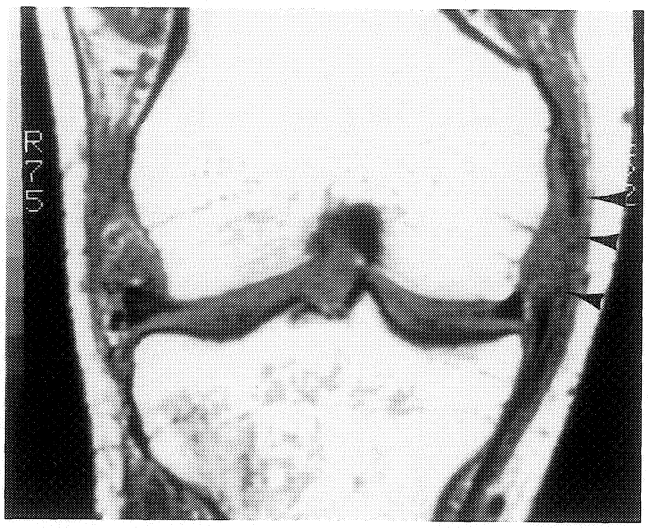

Figure 11.
Medial collateral ligament tear. Coronal T1-weighted (TR = 500 msec, TE = 12 msec) image showing discontinuity, enlargement, and increased signal in the medial collateral ligament.

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MAGNETIC RESONANCE IMAGING OF THE EPIPHYSIS: AN EXPERIMENTAL STUDY

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ABSTRACT

The purpose of this study was to determine if metabolically altered cartilage could be distinguished from normal cartilage in the epiphysis by magnetic resonance imaging. Epiphysis from ten New Zealand White rabbits and five newborn lambs were imaged before and after heat-killing. The physiological change produced was documented with tritiated thymidine and radioactive sulfate autoradiography. Magnetic resonance imaging could detect differences in the tissues produced by heat-killing. The changes were most pronounced in the marrow, less pronounced in the epiphyseal cartilage and least pronounced but still detectable in the growth plate.

INTRODUCTION

Part of the challenge of pediatric orthopaedics results from analyzing and altering the skeletal system at a time when the osseous tissues are still forming from the fibrocartilaginous tissues. While the former are radiopaque and the latter radiolucent; it is frequently the latter which are the more important physiologically. Hence, conventional radiography, while important for the pediatric orthopaedist, has obvious limitations. The newer modalities for imaging fibrocartilaginous tissue change this situation dramatically. Magnetic resonance imaging is capable of providing both anatomic and physiologic information[6]. The implications of this for cartilage imaging are exciting and still evolving.

One implication is that terminology becomes increasingly important. Cartilage for an adult orthopaedic surgeon has largely meant articular cartilage. Cartilage for a pediatric orthopaedic surgeon has meant a physiologically heterogeneous tissue[4] employed not only in articulation but also in the development and growth of the skeletal system[10,12,17]. The growing end of a long bone has several cell populations devoted to different functions to which varying nomenclature has been applied. One possibility is to term the entire growing end of a long bone the epiphysis[15]. A secondary center of ossification may or may not be present. If present, the cartilage surrounding it may be considered to include a "spherical growth plate"[18] and the remaining epiphyseal cartilage. Alternatively the secondary center of ossification may be called the epiphysis[2] and the surrounding cartilage the chondroepiphysis[3] including in it the "spherical growth plate." Growth of the secondary center of ossification or epiphysis is initially spherical, thus the terminology for the cartilage dedicated to its growth. As the secondary center approaches the growth plate, that part of the epiphyseal cartilage adjacent to the growth plate is replaced by bone and the secondary center assumes a hemispherical configuration[2]. The epiphyseal cartilage therefore is also hemispherical and continues to contribute to its own growth and that of the secondary center of ossification[11] until maturity. The growth plate or physis is responsible for the longitudinal growth of the bone surrounded at its periphery by the groove of Ranvier and the perichondrial ring of LaCroix[2,16].

These cartilaginous cell populations at the end of a long bone are crucial for co-ordinated growth of the epiphysis and longitudinal growth of the bone. In mammals, the evolutionary advance of a secondary center of ossification provides support to and also anatomically separates the cartilaginous cell populations[7,8].

There is a tendency to view the cartilaginous cell populations as the radiolucent areas that define the osseous structures when physiologically it is the bone that follows the cartilage. Even on magnetic resonance imaging, it is the marrow which tends to attract attention. Yet, perhaps even more important for pediatric orthopaedics, cartilage may now be imaged with magnetic resonance providing both anatomic and physiologic data.

The purpose of the present study is to demonstrate that magnetic resonance imaging can detect distinct cell populations within epiphyseal cartilage and changes in these populations induced by heat-killing.

METHODS

The study was performed with ten New Zealand White rabbits, one to four days of age, and five newborn lambs with normal epiphyses which had been sacrificed in the course of a non-orthopaedic experiment. All animals were used in accordance with The Guide for the Care and Use of Laboratory Animals. The rationale for using the rabbit,

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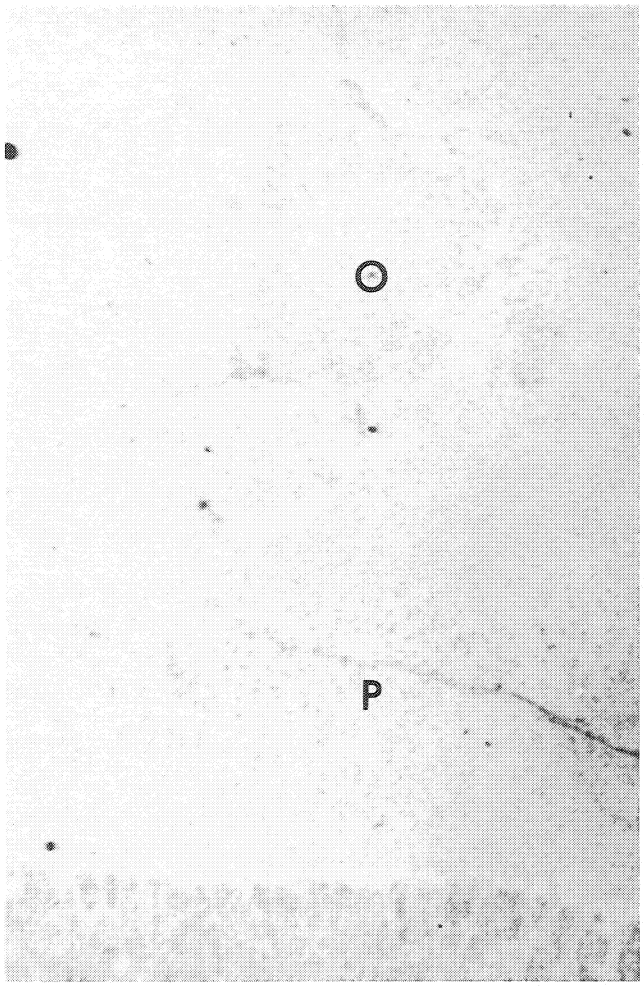


Figure 1
Radioactive sulfate autoradiograph of the proximal tibial epiphysis of the rabbit following heating. There has been no incorporation of nuclide throughout the epiphysis. (P = physis, 0 = ossification center, hematoxylin and eosin, 10X)

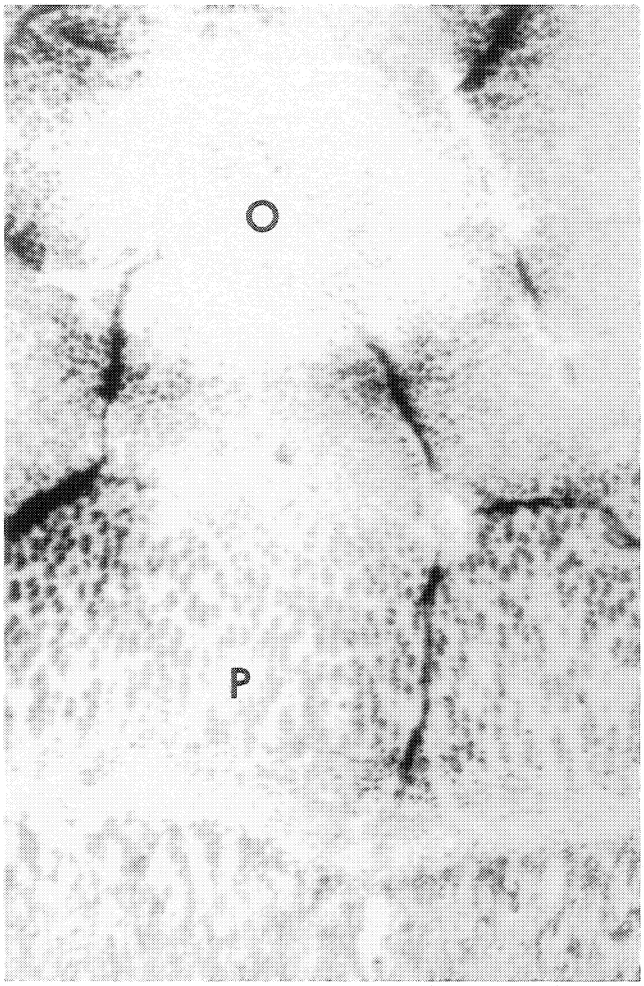


Figure 2
Radioactive sulfate autoradiograph of the proximal tibial epiphysis of a rabbit without heating. High labeling is noted in the physis (P) and spherical growth plate, surrounding the secondary center of ossification (O) (10X)

a small species, was that pre- and post-mortem imaging would be available. The rationale for using the lamb was its larger epiphyseal size.

Rabbit Protocol

For imaging, the rabbits were sedated with ketamine (Ketalar, Parke-Davis, Morris-Plains, NJ) 40 mg/kg IM and xylazine (Rompun, Mobay Corporation, Shawnee, KA) 5 mg/kg. The proximal femoral, distal femoral and proximal tibial epiphyses of the rabbits were imaged in vivo with magnetic resonance using a 1.5 Tesla system (GE Signa, Milwaukee, WI) with a 7.5 cm surface coil. A typical examination included the following pulse sequences: spin echo (SE)- axial T1-weighted 300/20 (repetition time [TR] = 300 msec/echo time [TE] = 20 msec), sagittal T1-weighted 400/20 and T2-weighted 2000/20,80; gradient echo (GRE) - sagittal 60/15/30° (TR/TE/flip angle). Fat and water bound to protein are of high signal intensity (bright) on T1-weighted images, whereas free water has a high signal intensity on T2-weighted images. Images were obtained using three millimeter (mm) slices at one mm intervals with four excitations. Immediately after imaging the rabbits were sacrificed with an overdose of sodium pentobarbital. Both femora and tibiae were then harvested. The left femora and tibiae were placed immediately in a culture tube with Dulbecco's Modified Eagle Medium (DMEM). The right femora and tibiae were heat killed in saline at 60°C for fifteen minutes and then placed in a separate culture tube with DMEM. The proximal femoral, distal femoral and proximal tibial epiphyses were re-imaged in vitro as above. Conventional radiographs were obtained.

The right and left distal femoral epiphyses were then divided longitudinally. One half of each was cultured for one hour in twenty milliliters of DMEM containing either 200 microcuries of tritiated thymidine (specific activity 80

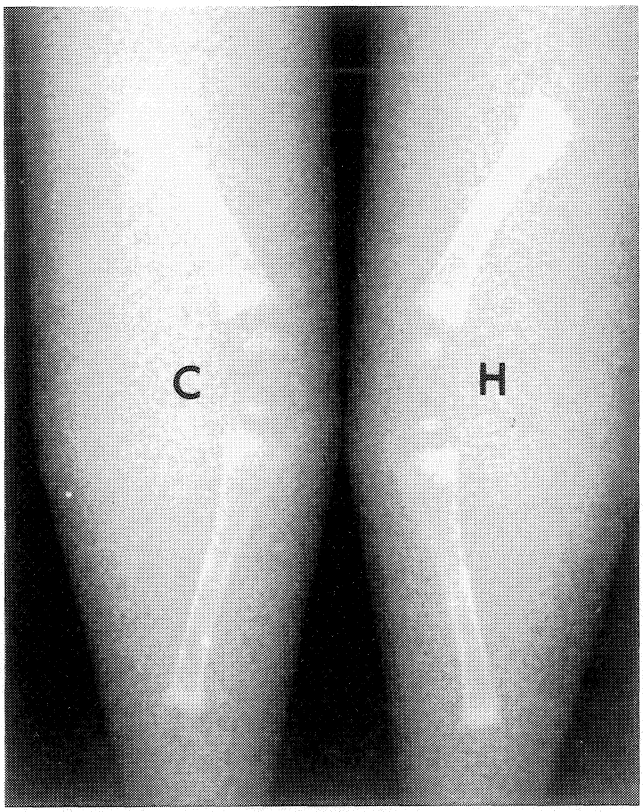


Figure 3
Conventional radiograph of right and left rabbit femora and tibiae in vitro (culture tube with DMEM) following heat killing (H) of one side and control culturing (C) of the other. There is no radiographic difference between the two sides.

μCi/mM, New England Nuclear, Boston, MA) or 200 microcuries of radiosulfate (specific activity 400 μCi/mM). The specimens were subsequently prepared for autoradiography.[19] The right and left proximal tibial epiphyses were prepared for histology using hematoxylin and eosin and Safranin 0 stains.

Lamb Protocol

The preliminary data from the rabbit protocol indicated that the magnetic resonance imaging of the live epiphyses, whether in vivo or in vitro, was identical and that the protocol for heat-killing was effective as documented by autoradiography. More anatomic detail could be obtained by imaging a larger epiphysis. Lamb epiphyses were available from a non-orthopaedic experiment but could only be imaged after sacrifice. After euthanasia, lamb tibiae were imaged in a manner identical to that in the rabbit protocol except that imaging was performed predominately in the coronal plane, since this plane allowed for the best resolution.

Data Analysis

The autoradiography and histology were analyzed blindly by the orthopaedists (MM,DZ). The images were analyzed blindly by the radiologist (DJ). A correlation was then drawn between the data.

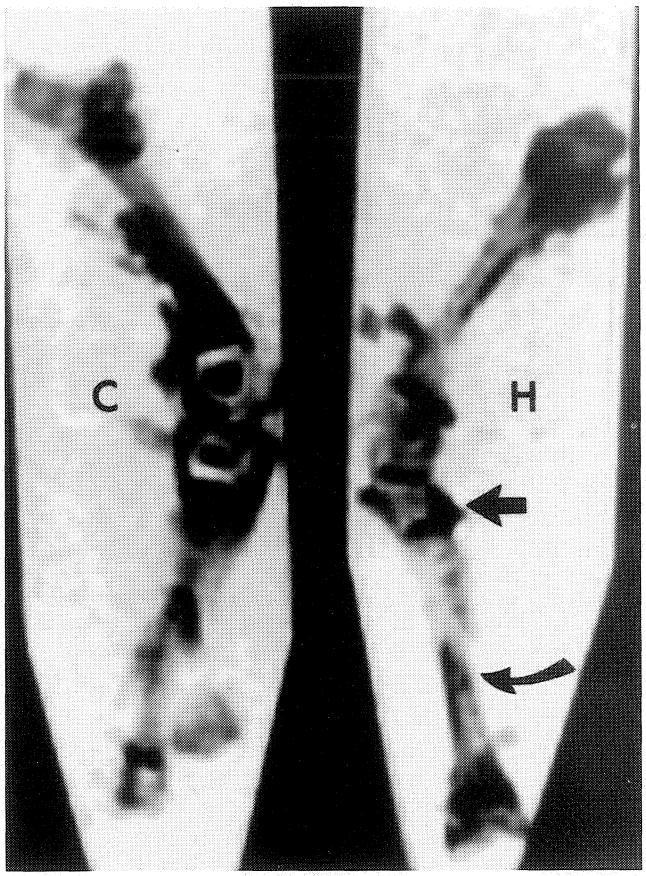


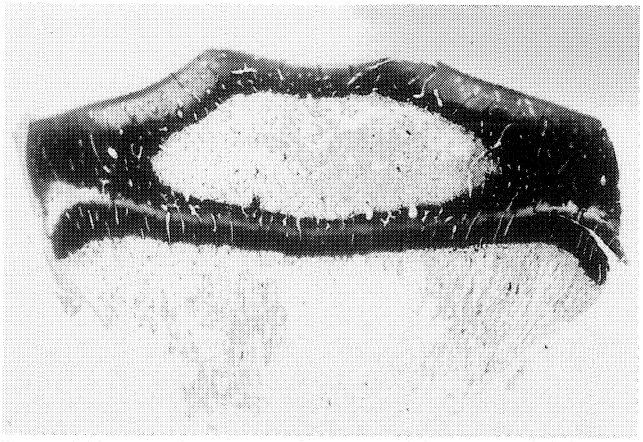
Figure 4
T2-weighted (SE 2000/80) magnetic resonance image of right and left rabbit femora and tibiae in vitro (culture tube with DMEM) following heat killing (H) of one side and control culturing of the other (C). The specimen altered by heating shows increase in signal intensity of the marrow (curved arrow) and decrease in signal intensity of the epiphyseal cartilage (straight arrow). The demarcation between the ossification center and the cartilage has been obscured.

RESULTS

Rabbit Protocol

Secondary centers of ossification were present in all the distal femoral and proximal tibial epiphyses. Four of the older rabbits had early formation of a proximal femoral secondary center of ossification on conventional radiography; six did not. On T1- and T2- weighted images, the live secondary center of ossification was hypointense (dark) consistent with hematopoetic marrow similar to that of the metaphysis. The T2-weighted image of an epiphysis without a secondary center of ossification was uniformly bright unless hypertrophic chondrocytes were present (noted on the histologic correlation). In this case a halo of decreased signal intensity appeared within the epiphysis on T2-weighted image.

Figure 5
Coronal histological section of the lamb proximal tibial epiphysis (unmagnified, Safranin O/fast green). The various regions (articular cartilage, epiphyseal cartilage, "spherical growth plate" surrounding the secondary center of ossification, physis and metaphysis) are seen in the tissue correlating with the magnetic resonance images which follow.



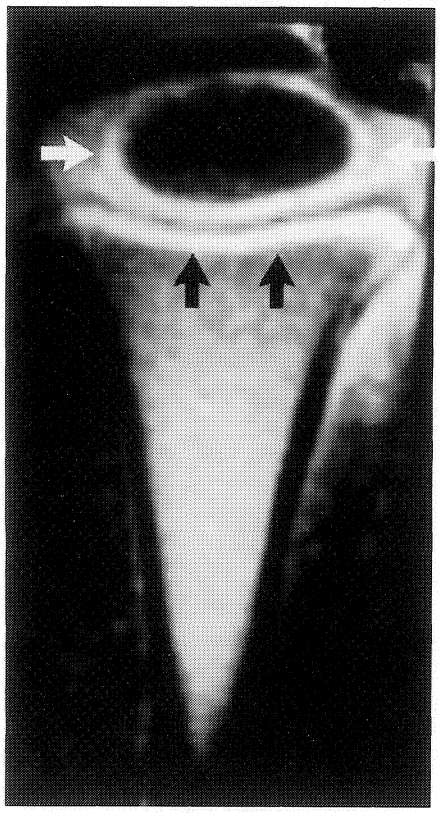


Figure 6
T1-weighted (SE 400/20) coronal magnetic resonance image of a lamb proximal tibial epiphysis in vitro. There is a rim of high signal intensity surrounding the secondary center of ossification with a fainter dark halo around it (white arrows) and a dark zone on the proliferative side of the physis. The signal intensity of the spherical growth plate resembles that of the physis (dark arrows).

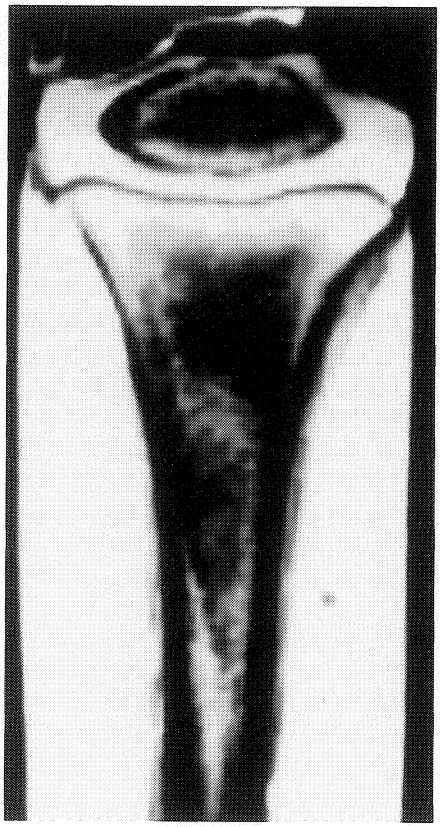


Figure 7
T2-weighted (SE 2000/80) coronal magnetic resonance image of a lamb proximal tibial epiphysis in vitro. As compared to Figure 6, the dark zones surrounding the "spherical growth plate" and on the proliferative side of the physis are no longer apparent. There is instead a dark zone at the physeal-metaphyseal junction.

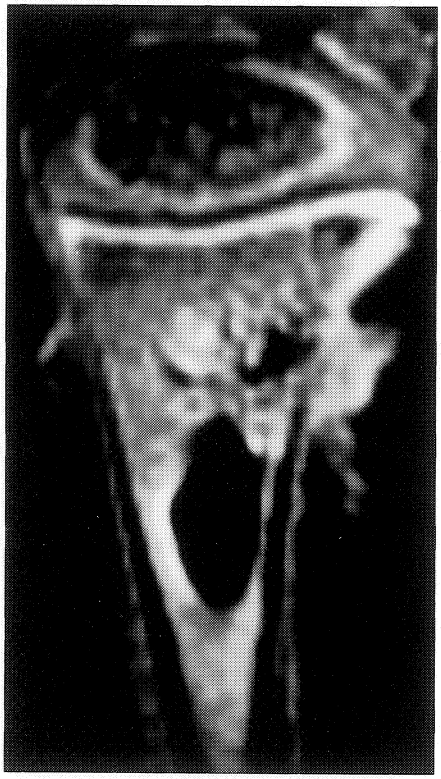


Figure 8
T1-weighted (SE 400/20) coronal magnetic resonance image of a lamb proximal tibial epiphysis in vitro after heat-killing. When compared to Figure 6, the signal from the marrow has become heterogeneous. The signal from the "spherical growth plate" and the epiphyseal cartilage has decreased. There is some but less striking decrease in the signal intensity from the physis. The dark region in the metaphyseal-diaphyseal junction is an artifact due to air.

No changes were detected in the magnetic resonance images between the in vivo images and the in vitro images in DMEM. The heat killing did not dramatically change the microscopic anatomy of the cartilage. While some heat-killed specimens had more chondrocytes with pyknotic nuclei, this did not prove to be a consistent finding on blinded examination. The marrow in the heat-killed specimens did contain some fibrin clumping and pyknotic nuclei, but even these changes could be missed with cursory inspection. The autoradiographic studies corre-

lated completely with the metabolic status of the tissue. No radioactive sulfate or tritiated thymidine incorporation was seen in the heat-killed cartilage (Figure 1), in contrast to controls containing live cartilage cells (Figure 2). Thus, while the microscopic anatomy did not dramatically change with heat-killing, the physiology did.

Conventional radiography was not altered by the heat-killing (Figure 3). The magnetic resonance appearance was altered by heat-killing (Figure 4), although this was detected on blinded examination in the specimens from only six of the ten animals. The alteration was seen most clearly on T2-weighted images. The marrow changes were most striking, with the signal becoming brighter and inhomogeneous. The signal from the cartilage generally decreased. The changes in the various cell populations of the epiphysis were better seen with the lamb epiphyses and will be presented in that section.

Lamb Protocol

The larger size of the proximal tibial epiphysis of the lamb afforded the opportunity for greater resolution of its cell populations (Figure 5).

On T1-weighted images (Figure 6), four cartilaginous zones surrounded the dark marrow of the secondary growth center: a bright zone isointense with the physis which appeared to correspond histologically to the "spherical growth plate"; a thin, dark zone surrounding the spherical growth plate corresponding to the junction of the "spherical growth plate" and epiphyseal cartilage; a broad zone of intermediate signal intensity corresponding to the epiphyseal cartilage; and the articular cartilage which was slightly brighter than the epiphyseal cartilage. At the interface between the epiphyseal cartilage and the physis, there was a darker zone similar in intensity to the zone surrounding the spherical growth plate. Thus, in both growth plates the bright cartilage was between darker cartilage and even darker bone. The findings remained unchanged following switching of phase and frequency encoding directions, indicating that they did not represent chemical shift artifact. In two lambs, fat suppression T1-weighted imaging revealed no change in the high signal of the spherical plate, indicating a lack of significant contribution of fat to the high signal intensity of this region. The marrow of the secondary center of ossification had a low intensity as did the metaphyseal marrow. The diaphyseal marrow was of relatively higher intensity.

On the T2-weighted images (Figure 7), the dark zones surrounding the cartilage were no longer apparent. A dark band was present separating the physis from the metaphyseal marrow. The marrow of the secondary center of ossification and the diaphysis had a very low signal intensity, whereas the metaphyseal marrow had a slightly higher signal.

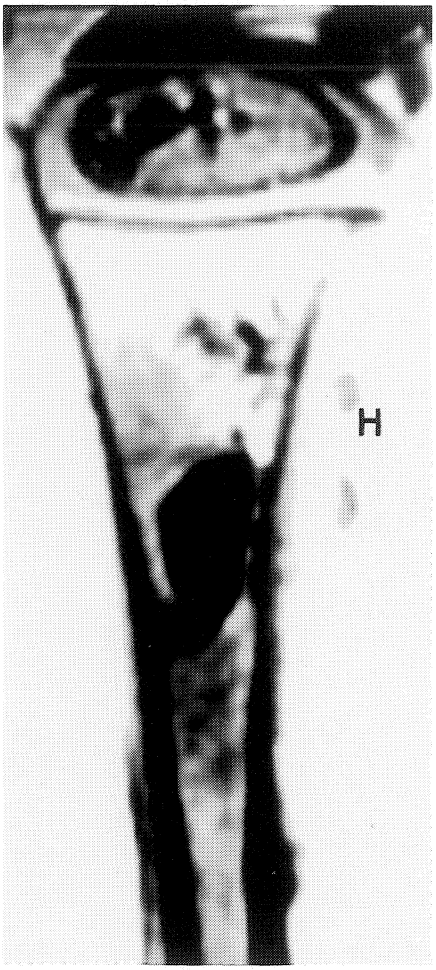


Figure 9.
T2-weighted (SE 2000/80) coronal magnetic resonance image of a lamb proximal tibial epiphysis in vitro after heat-killing. Changes are again greatest in the marrow which shows increased signal intensity when compared to the image obtained before heating. There is less change in the epiphyseal cartilage and least izn the physis when compared to Figure 7. The dark region in the metaphyseal-diaphyseal junction is again an artifact due to air.

After heat-killing, all five lamb epiphyses (sensitivity 100%) demonstrated the same changes that the six rabbit epiphyses had (sensitivity 60%), but in greater detail.

On T1-weighted images, the heat-killed lamb epiphysis (Figure 8) demonstrated striking changes in the marrow. The secondary center of ossification and metaphysis became heterogeneous or granular in appearance. While less striking than the marrow changes, the cartilage also revealed changes following heating. The epiphyseal cartilage and "spherical growth plate" produced less signal. The boundary between the "spherical growth plate" and the secondary center of ossification became less distinct. The heat-killed physeal cartilage also lost signal relative to controls, but this was the least striking difference when comparing experimental and control specimens. On T2-weighted images, similar changes were seen (Figure 9). The marrow of the secondary center of ossification and the metaphysis became relatively brighter and granular. The "spherical growth plate" and the epiphyseal cartilage became darker. The physis again changed the least relative to controls.

DISCUSSION

Magnetic resonance imaging is capable of detecting heterogeneity in living cartilage and changes in metabolically altered (heat-killed) cartilage in epiphyses from rabbits and lambs. Mammalian epiphyses share a similar structure[7] so it is reasonable to search for similar findings in human epiphyses with magnetic resonance imaging. Heat-killing is a drastic alteration in metabolism but as a model system produces the desired change in physiology without a change in anatomy. It is not clear that the same changes produced by heat-killing would be seen in the cell populations of cartilage in clinical entities. Similar marrow changes have been described in Perthes' Disease[1,13,14], however, although not necessarily with complete sensitivity at early stages[5].

The alteration of physiology in cartilage detectable by magnetic resonance imaging probably reflects a change in the water binding characteristics of the proteoglycan in the cartilage matrix[9]. The resolution obtained in the imaging for this study is difficult to obtain in the clinical setting. The imaging was produced with long acquisition times (twenty-four minutes for T1-weighted images (typical clinical acquisition times one to three minutes, and thirty-four minutes for T2-weighted images compared with typical clinical acquisition times of five to ten minutes.

A technology does exist which seems to detect subtle changes in physiology before changes in anatomy (growth). Whether this will actually be the case will require much further research. Further investigation is required to fully utilize this technology in clinical practice, but the possibilities are exciting.

ACKNOWLEDGMENT

This work was supported in part by the Peabody Foundation, Boston.

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VERTEBRAL COMPRESSION FRACTURES: DIFFERENTIATION BETWEEN BENIGN AND MALIGNANT CAUSES

Kevin D. Harrington, M.D.

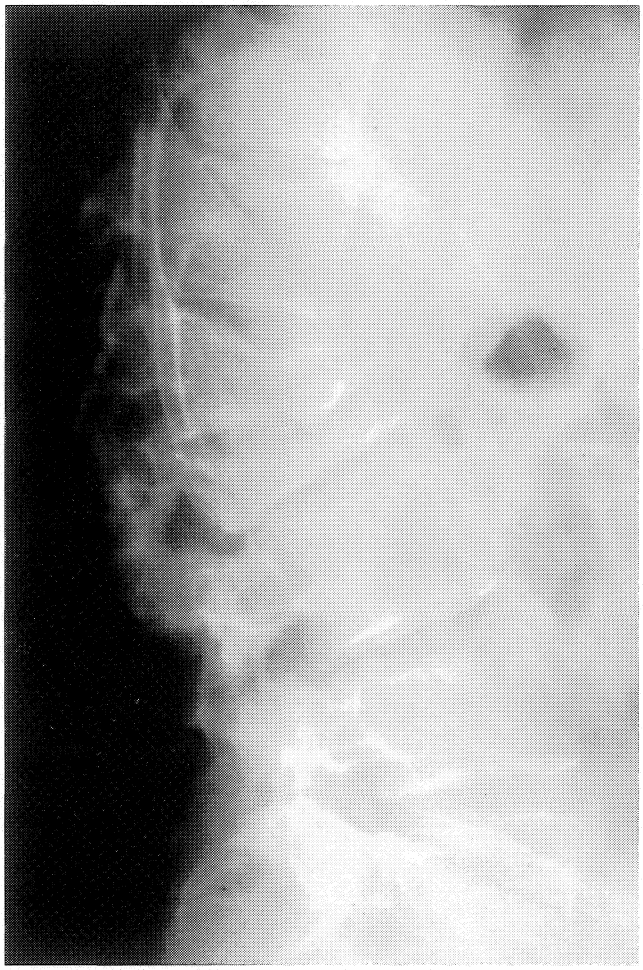

Figure 1
Lateral radiograph of a sixty-six year old woman with known breast cancer and scintigraphically demonstrable metastases to T11 and T12. Although the wedge compression fractures are secondary to metastases, the appearance on plain radiographs is indistinguishable from benign pathological fractures secondary to osteoporosis.

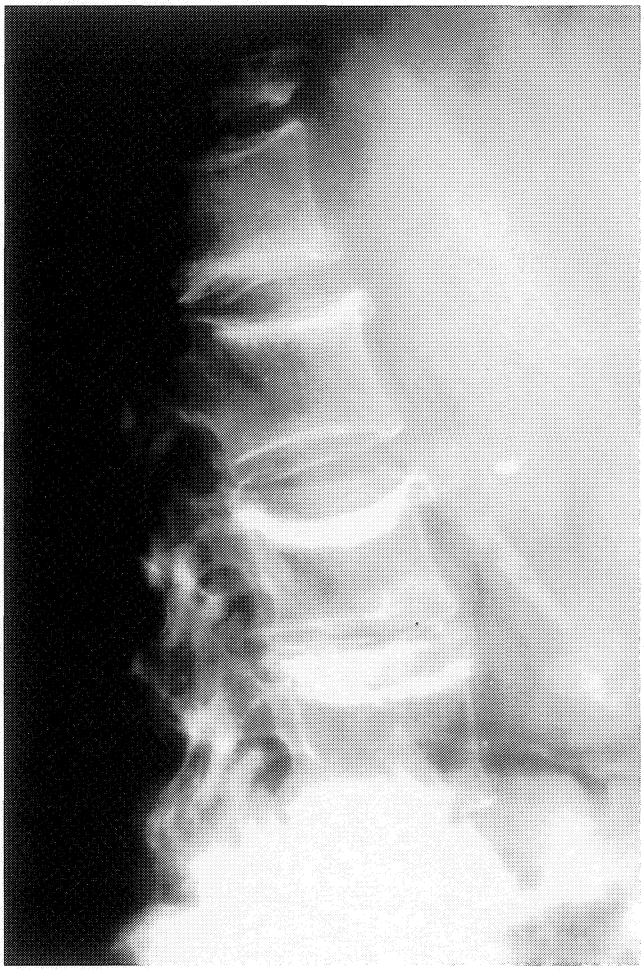

Figure 2
Lateral radiograph of a thirty-five year old female undergoing chemotherapy and systemic steroid therapy for metastatic breast cancer with hypercalcemia. There is diffuse osteoporosis with multiple compression fractures of the superior vertebral endplates. Magnetic resonance scanning showed no evidence of bony metastases.

Attempting to ascertain the underlying cause of a vertebral compression deformity may present a difficult diagnostic dilemma (Figure 1). The spine is a common site of metastatic disease accounting for thirty to fifty-five percent of all bony metastases. If there is sufficient tumor osteolysis, a pathological fracture will occur. However, progressive softening of the vertebrae in post-menopausal women is also very common. Spontaneous compression fractures secondary to osteoporosis often result and may be indistinguishable from those of malignant origin. Benign metabolic processes such as hypothyroidism, hyperparathyroidism, Paget's disease, and others may result in fractures that are also difficult to differentiate both clinically and radiographically from metastases or osteoporosis. Benign or malignant primary neoplasms of vertebrae, including hemangioma and myeloma, may progress to cause compression fractures as well. Finally, silent and indolent osteomyelitis, typically tubercular or secondary to hematogenous spread of gram-negative sepsis, may result in vertebral collapse which can closely mimic any of the benign or malignant processes already mentioned.

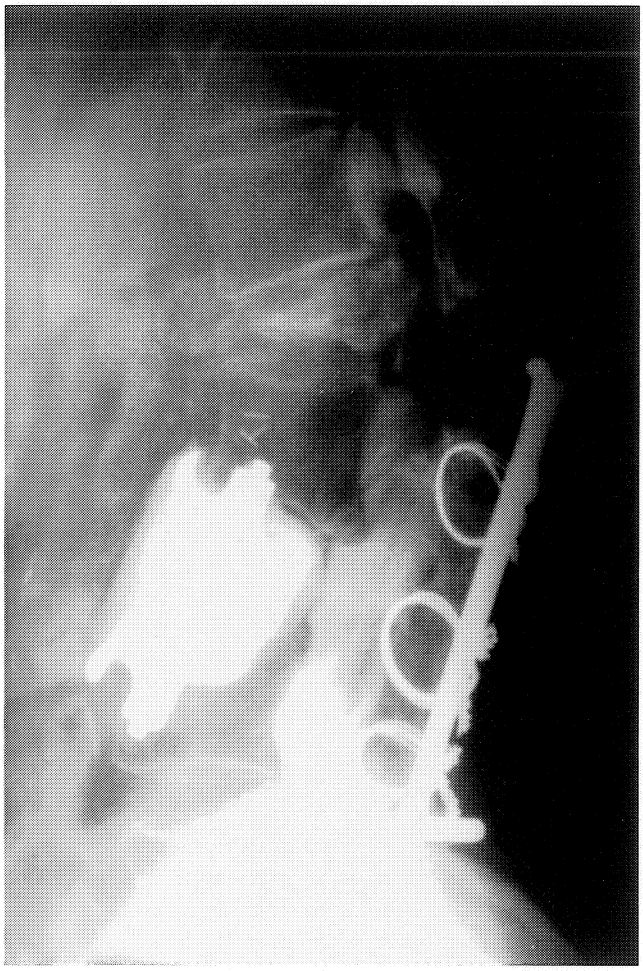

Figure 3
Lateral radiograph of a seventy-one year old female with known multiple myeloma, six years following vertebrectomy and replacement stabilization at L2-4 followed by irradiation. A new compression fracture has appeared at L1. At operation radiation osteitis without tumor was apparent.

Figure 4
Sagittal MRI of the lumbar spine of a sixty-six year old male on chemotherapy for metastatic prostatic carcinoma. Patient developed spontaneous hematogenous osteomyelitis at L4-5.

Obviously, clinical data concerning preexisting disease processes may be very helpful in differentiating between the various potential causes of vertebral collapse in any given patient. A young patient with known bony metastases from breast carcinoma presumably develops her compression fracture because of spinal metastases, but even such obvious conclusions may prove erroneous. Steroids or chemotherapy, given as a part of treatment, may result in marked osteoporosis (Figure 2). Insufficiency fractures of the spine from local irradiation may appear years after such treatment has been completed (Figure 3). Debilitated cancer patients on chemotherapy typically become chronically pancytopenic and at risk for hematogenous osteomyelitis involving the spine (Figure 4). Patients with debilitating diseases may also be unlucky enough to suffer direct trauma to the spine.

All of these processes initially present as back pain, but this pain may be of sudden or insidious onset and with or without neurological compromise. A history of progressive quadriparesis or radiculopathy may be of minimal benefit in differentiation between the various potential causes of spinal deformity. The oft quoted maxim that sudden fracture myelopathy is invariably the result of acute trauma has been proven invalid (Figure 5), just as has the concept that acute trauma never results in gradual or progressive neurological compromise (Figure 6).

Fortunately, the current availability of many different radiological modalities has enhanced our ability to establish criteria for differentiating between the various types of vertebral fractures by using a combination of imaging techniques. These include plain roentgenography, bone and soft tissue scintigraphy, computerized tomography (CT), and magnetic resonance imaging (MRI). The following is an analysis of the imaging characteristics for verte-

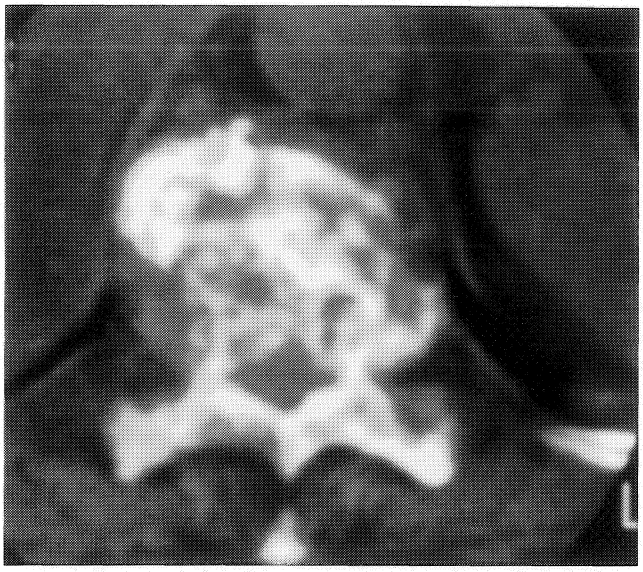

Figure 5
A transverse CT scan of the L3 vertebral body of a man with an acute and progressive cauda equina syndrome secondary to metastatic prostate carcinoma. Fragmentation of the vertebral bodies and marked spinal canal compromise are apparent.

bral fractures caused by infection, trauma, osteoporosis, and benign or malignant tumor processes.

A. Infection

Early in the course of vertebral osteomyelitis, particularly when secondary to a discitis, plain roentenograms may be deceptively normal. As the infection progresses, the disc space often becomes narrowed, the vertebral end plates show irregular destruction, and a paravertebral soft tissue mass may appear. However, the patterns of vertebral collapse and even the configuration of the paravertebral mass seen on plain films does not differentiate the infectious process from an aggressive and infiltrative malignancy such as a myeloma, Ewing's or osteosarcoma, or from many metastatic lesions. Technetium scanning has a high sensitivity in demonstrating the infectious process, but its specificity is only seventy-five percent at best. The radionuclide may not be taken up preferentially by an affected vertebral body if the infection has resulted in ischemic changes, or if there is little reactive osteoblastic response. SPECT imaging is usually more accurate in demonstrating disc abnormalities, and may exceed magnetic resonance in demonstrating small paravertebral masses. When gallium scanning is combined with technetium, both the accuracy and specificity improve to better than ninety percent, but false negative gallium scans may occur when activity within the bowel effectively obscures vertebral uptake.

Computerized tomography and MRI are equally effective in showing typical mixed erosive and sclerotic changes within the vertebral body (Figure 7), but MRI better

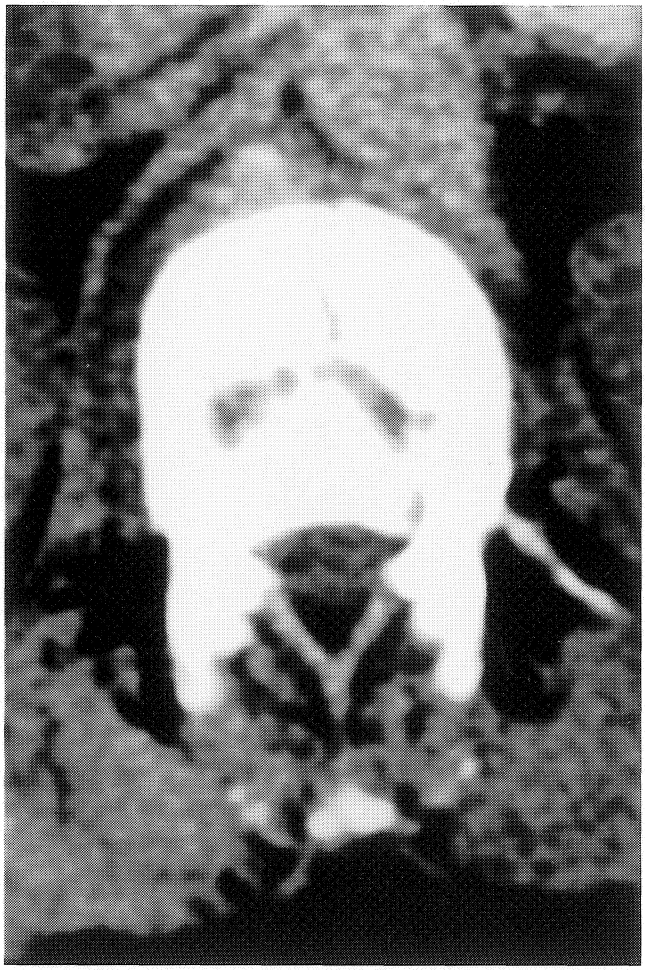

Figure 6
A transverse CT scan of an acute traumatic burst fracture of the L1 vertebral body has resulted in moderate extrusion of bone into the spinal canal. Neurological compromise progressed slowly, and the patient did not seek medical attention for her neurological deficit until five months after the injury.

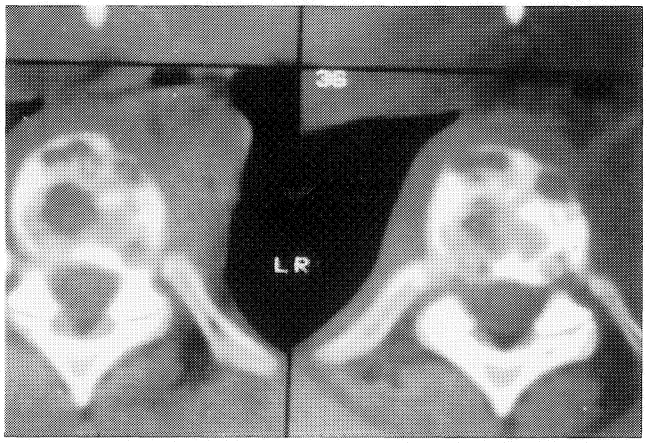

Figure 7
A transverse CT scan of a patient with salmonella osteomyelitis and discitis. The T10 vertebral body shows erosion of trabecular bone with secondary sclerotic changes. The bony changes are also compatible with low grade metastases and secondary blastic repair.

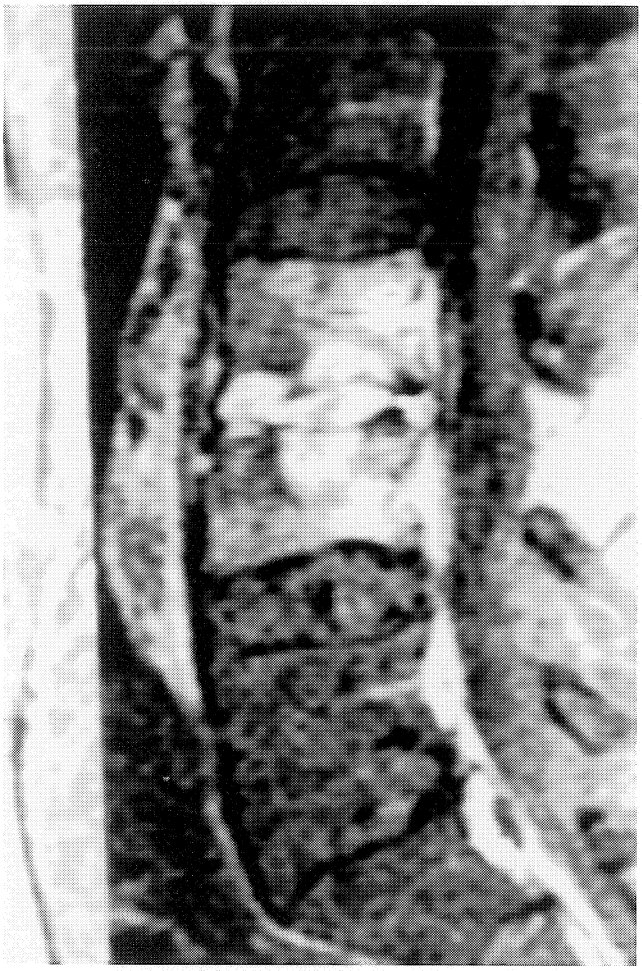

Figure 8a
Sagittal MRI of the lumbar spine of a forty-three year old male with post-laminectomy discitis and osteomyelitis at L3 and L5. This T2-weighted image shows a markedly increased signal in both vertebral bodies and in the intervertebral disc which is typical of infection. The increased signal is reflective of hydrogen ion excitation in edematous but relatively avascular tissue with purulent infiltration.

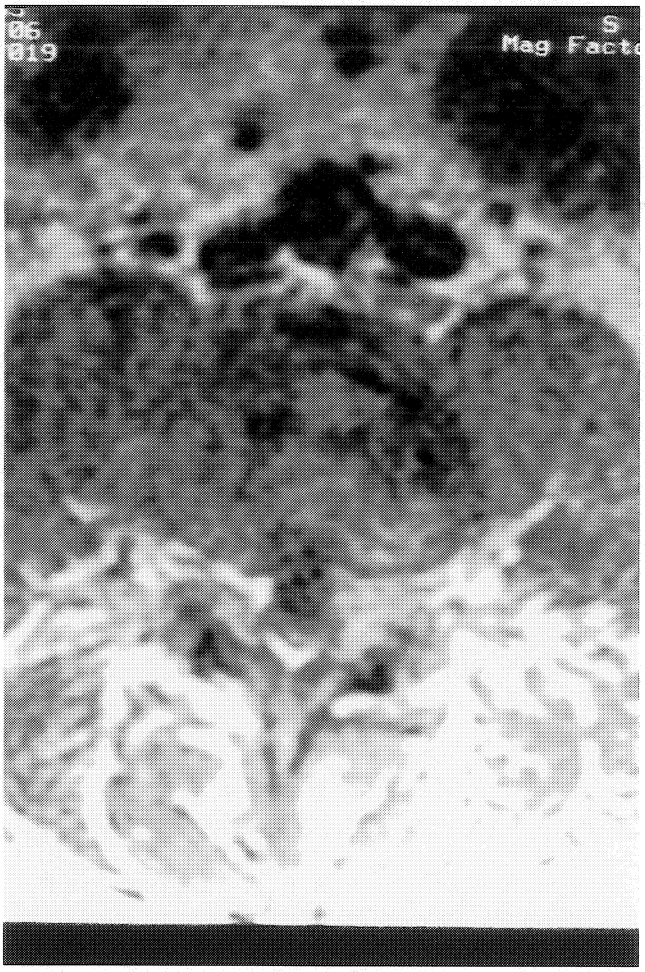

Figure 8b
A cross-section MRI scan of the same patient demonstrates both paravertebral and epidural soft tissue masses.

demonstrates the extent of disc and adjacent vertebral end plate destruction and the size of a paravertebral mass (Figures 8a and 8b). T1-weighted images commonly demonstrate a confluent diminution in signal intensity because of an increase in water content of the marrow spaces and a decrease in vascularity of the bone. It is often impossible to separate the involved disc space from adjacent bone (Figure 4). T2-weighted images often show increased activity in adjacent vertebral bodies. In early osteomyelitis, where disc space involvement is minimal, the diffuse irregular loss of marrow signal may be highly suggestive of focal metastatic disease.

B. Trauma

An acute compression or burst fracture of a vertebral body usually reveals sharp delineation of the bony fragments on plain roentgenograms with acute invagination of the anterior bony cortex (Figure 9a). Increase in technetium uptake by the vertebral body occurs promptly after fractures, but bone scanning rarely is helpful in separating benign from malignant vertebral collapse (9b) unless multiple metastatic foci are demonstrable (Figure 10). Increased uptake may be apparent scintigraphically in the affected vertebral body for an indefinite period as bone remodeling occurs. A CT scan is effective in demonstrating sharp delineation of fracture fragments typical of an acute fracture in previously normal bone (Figure 11). Computerized tomography is also the most accurate method of imaging the extent of impingement of bony fragments into the spinal canal. The accuracy of CT scanning exceeds MRI in demonstrating details of bony derangements and canal compromise. However, as described in the next paragraph, MRI can be much more helpful in differentiating between fractures of previously normal bone and those occurring in bone weakened by osteoporosis or tumor infiltration.

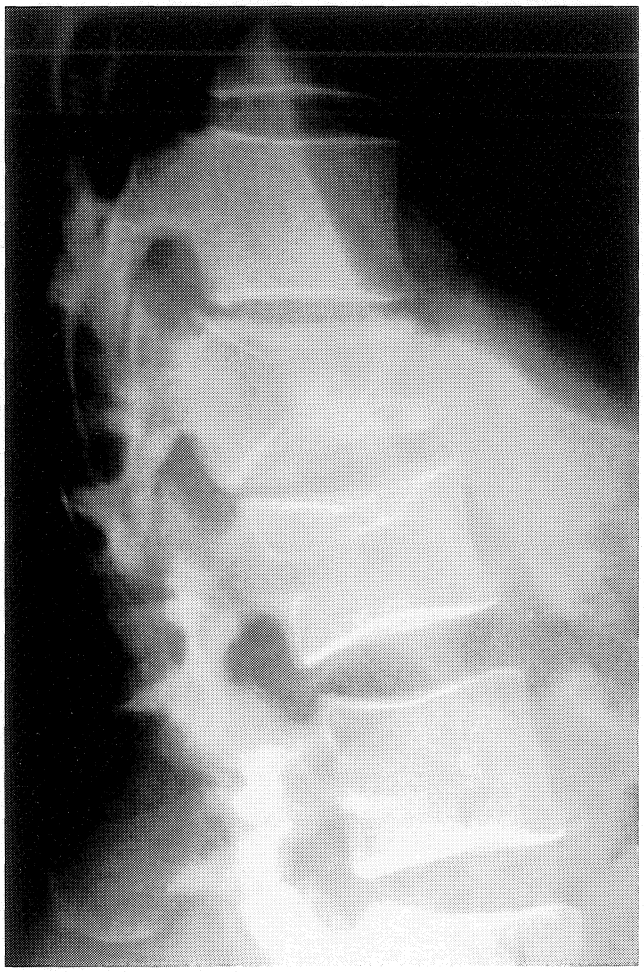

Figure 9a
Lateral roentgenogram of a benign traumatic T12 compression fracture. Invagination of the anterior cortex is typical of compression through previously normal bone.

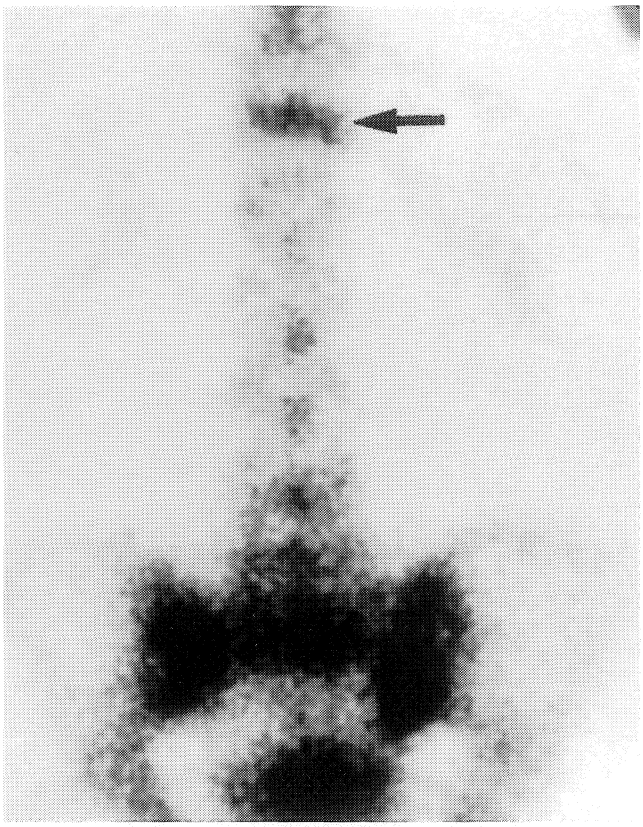

Figure 9b
The posterior planar scintigram of this patient shows abnormal tracer labeling (arrow) at T12, consistent with a compression fracture. Although biopsy demonstrated that this patient had lymphomatous destruction of the T12 vertebral body leading to the fracture, neither study was helpful in differentiating the condition from a benign osteoporotic compression.

C. Osteoporosis Versus Metastatic Disease

The universal phenomenon of vertebral osteoporosis in the post-menopausal woman, and to a lesser extent in the elderly male, results in varying degrees of cortical and trabecular bone thinning. The relative percentage of bone marrow remains fairly constant. As the bone mass decreases, vertebral fat content increases. With softening of bone, nonmalignant pathological fractures can occur which may be indistinguishable by plain radiographs or scintigraphy from fractures secondary to malignancy, unless widespread metastatic disease is obvious. Spontaneous fractures of osteoporotic vertebrae often cause bulging of soft bone posteriorly into the spinal canal, and occasionally result in neurological compromise. Moreover, the pattern on plain radiographs may be virtually indistinguishable from compression fractures secondary to metastatic malignancy. However, spinal instability rarely occurs in benign compression fractures as it occasionally does in metastatic disease (Figure 12a). The structural integrity of the posterior elements ordinarily is maintained in the more benign process (Figure 12b). Technetium scintigraphy may show multiple skeletal foci as demonstrated in Figure 10, but the origin of a solitary and isolated vertebral compression fracture in an elderly patient rarely is clarified by bone scanning techniques.

After completing plain radiographs, bone scans, and perhaps CT, one often is left with the continuing dilemma of whether the fracture represents the sequela of osteoporosis, a metastatic lesion, a solitary myeloma, or some other primary bone neoplasm. Magnetic resonance imaging has helped to resolve this dilemma. Characteristically, the malignant pathological fracture occurs when the vertebral body has been infiltrated by tumor spreading initially through hematopoietic tissue and then gradually destroying bone. Benign compression fractures occur because the bone substance itself has been lost or weakened, but here the hematopoietic tissue remains virtually intact. In both instances, the disc remains unaffected, thus helping to differentiate either lesion from osteomyelitis.

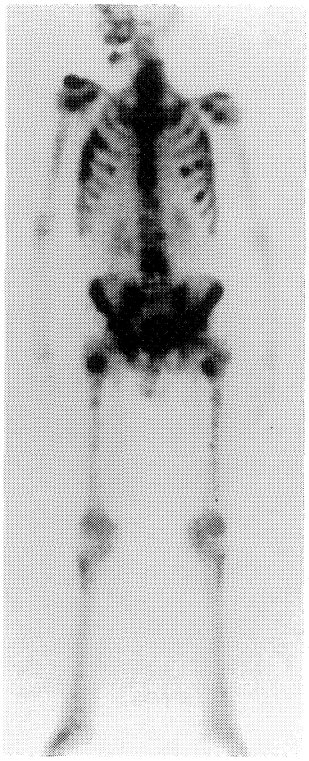

Figure 10
Anterior whole body radionuclide image reveals multiple focal areas of increased tracer deposition, virtually diagnostic of widely metastatic prostate carcinoma.

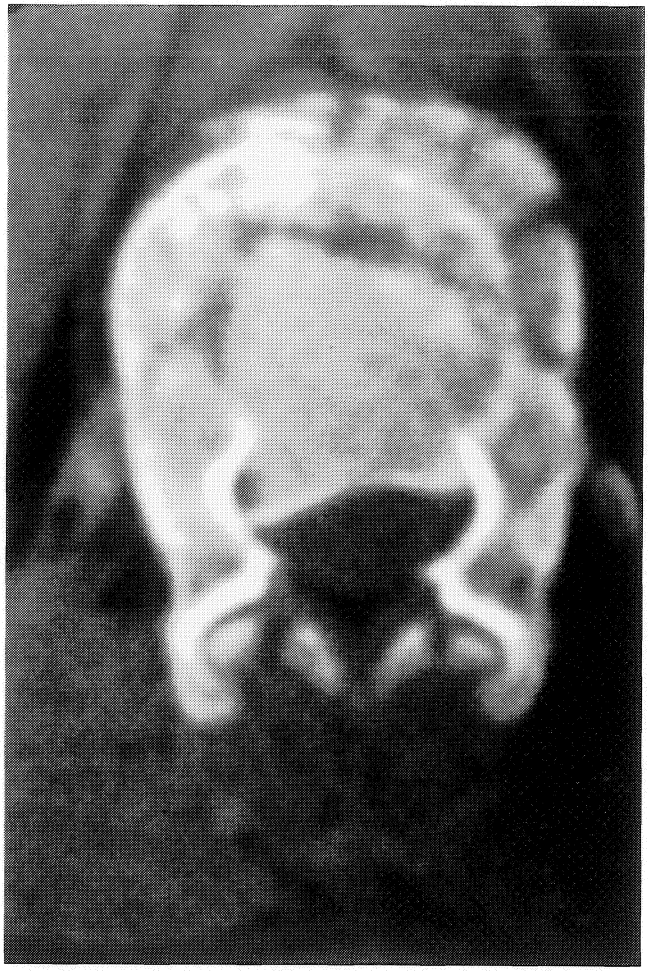

Figure 11
A transverse CT view of an acute burst fracture of the L1 vertebral body. The marrow pattern remains intact, and the fracture fragments are sharply delineated.

On MRI, a benign compression fracture usually preserves the normal marrow signal because the marrow has not been replaced. However, there is often displacement of the marrow along the vectors created by the compression deformity (Figure 13). This phenomenon is particularly apparent in T1-weighted images where a combination of impaction of the hematopoietic tissue, edema, and bleeding all result in an increase in focal water content which in turn increases the intensity of that signal (Figure 14). However, the overall marrow signal ordinarily remains intact. Acute benign compression fractures of the superior end plates typically cause temporary linear striation of the marrow distribution into the remaining vertebra, particularly on T1 imaging (Figure 14), but again this usually occurs in a uniform pattern and is reversible as fracture healing occurs. Rarely, dramatic spinal instability may become associated with benign compression fractures (Figure 15), but even here the marrow signal usually will remain normal on MRI, and the disc signal will be maintained.

In contrast, the MRI of a compression fracture secondary to metastatic malignancy reveals total or subtotal replacement of the normal bone marrow by tumor. This is reflected by a decreased signal (darker) image on T1-weighted images (Figure 16a), and increased intensity (white) on T2-weighted images (Figure 16b). There may be incomplete replacement of marrow, but its pattern will be irregular, reflecting focal destruction rather than uniform compression of hematopoietic tissue and fat.

On occasion the specificity of MRI may become blurred when an acute benign fracture is associated with marked edema and bleeding into the marrow space. In this case T1- and T2-weighted signals may mimic the typical tumor pattern. Diffuse marrow signal changes may even extend into the pedicles (Figure 17a and 17b), becoming strongly suggestive of tumor infiltration. Occasionally, bulging of the partially collapsed vertebral body may further increase this suspicion. Gadolinium enhancement of T1-weighted images may further the suggestion of a malignant process (Figure 18a and 18b), or may help clarify the essentially benign nature of the fracture (Figure 17c). In any event, it is extremely rare to see acute endplate destruction with a

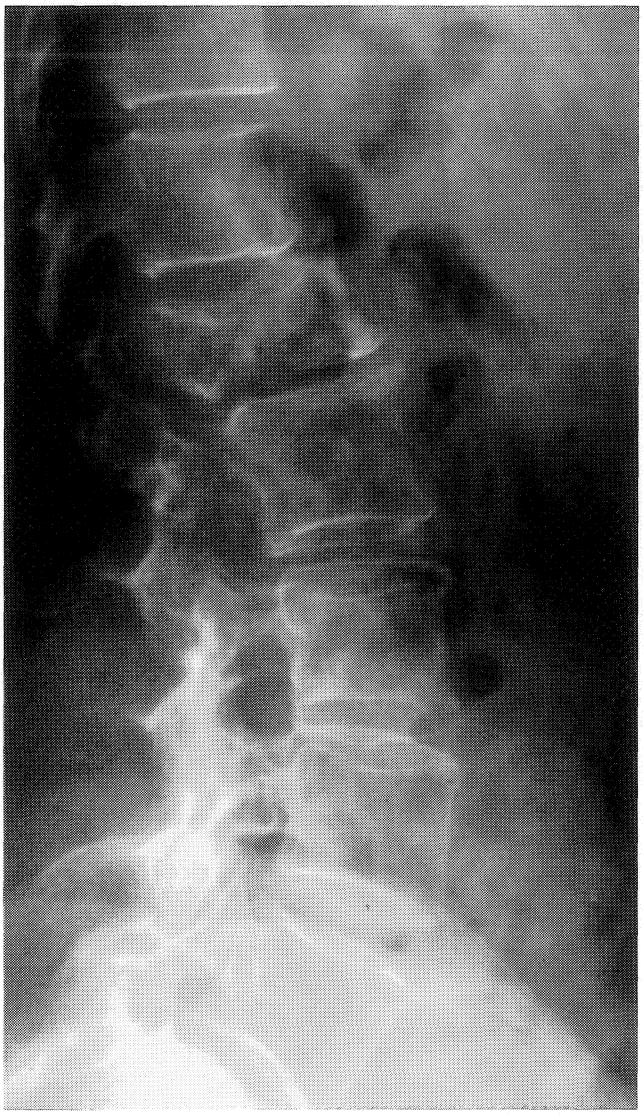

Figure 12a
Lateral radiograph of a spontaneous fracture of L1 from known metastatic breast cancer. The patient had received 5500 cGy local irradiation two years earlier.

benign osteoporotic fracture, and, of course, a large paravertebral mass is almost pathognomonic of a malignant process.

Solitary primary malignancy such as plasmacytoma will have an MRI pattern reflective of marrow replacement which is indistinguishable from a solitary metastasis. Solitary benign tumors tend to be more circumscribed with a regular marrow pattern preserved through the unaffected portion of the vertebra (Figure 19), or characteristic reactive bony changes on cross sectional imaging studies (Figure 20).

In summary, magnetic imaging is the single most helpful study in attempting to clarify the cause of a vertebral compression fracture secondary to infection, benign collapse from trauma, osteoporosis, or vertebral destruction and collapse from malignancy. The hallmark of vertebral deformity secondary to infection is the characteristic avascular destruction of the intervertebral disc. This is reflected by decreased signal across the disc, particularly on the T1-weighted image. These changes are not seen in the other types of fractures. Benign fractures are not associated with marrow replacement, although acutely the marrow may be compressed posteriorly along the vectors of bony extrusion or into a linear striated pattern after end plate collapse. With malignant pathological fractures, caused either by metastatic disease or the much less common primary vertebral malignancy, the hematopoietic tissue and accompanying fat have been replaced either entirely or in an irregular and infiltrative pattern.

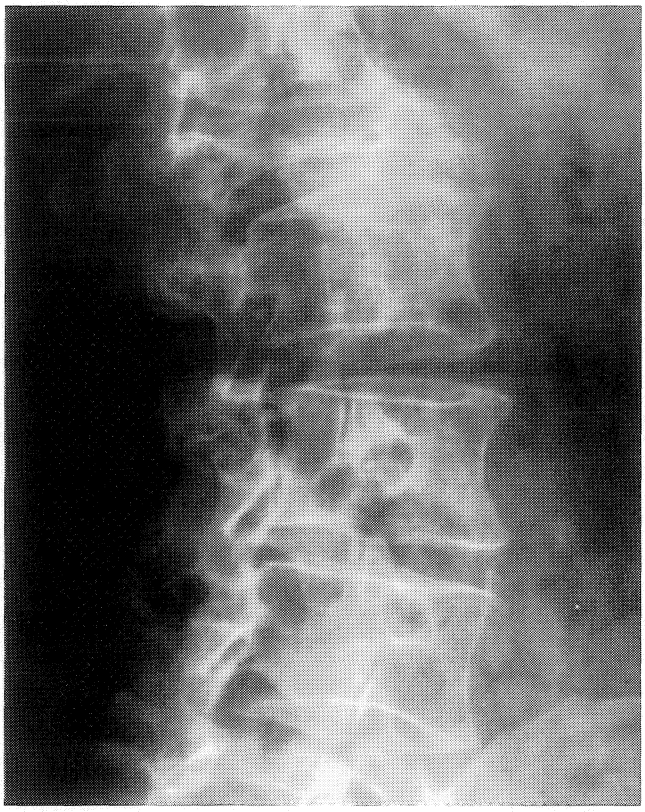

Figure 12b
Within six weeks the vertebral body had collapsed completely, and a severe cauda equina syndrome had developed. At operation, there was no evidence of viable tumor, but bony destruction extending into both pedicles appeared secondary to a combination of previous tumor osteolysis and radiation osteitis.

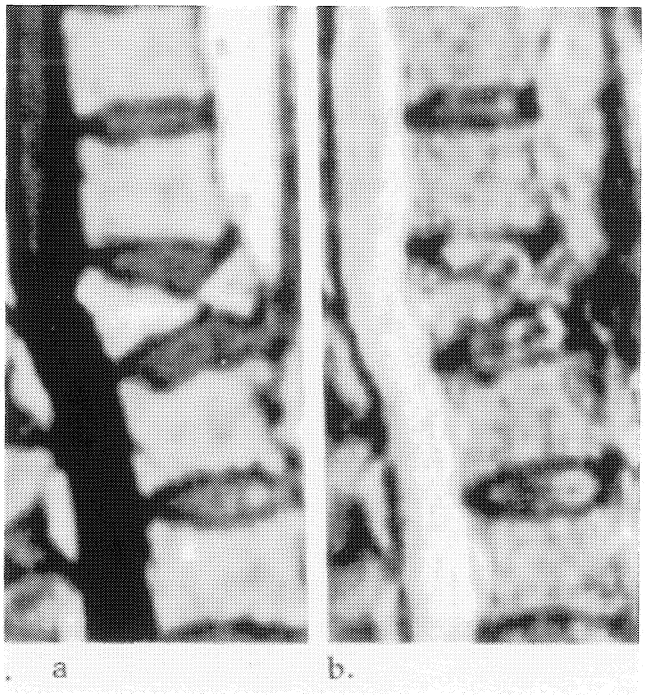

Figure 13
An MRI of benign compression fracture with intact residual bone marrow in a patient with no history or trauma. (a) The sagittal T1-weighted image shows complete preservation of normal bone marrow with smooth borders of the fractured bone. The configuration follows the direction of trauma force vectors. (b) Sagittal T2-weighted image shows bone marrow signal intensity in the fractured bone similar to that in the rest of the vertebral bodies.

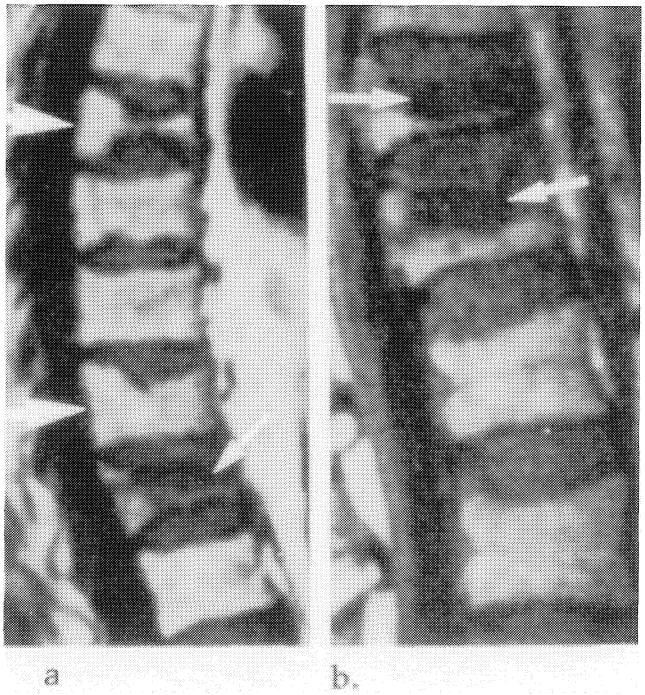

Figure 14
An MRI of benign compression fractures. (a) Sagittal T1-weighted image shows three benign fractures. Two vertebra show complete preservation of bone marrow (large arrows), whereas the third has incomplete and irregular replacement (small arrow) which could be confused with a metastatic fracture. (b) Sagittal T1-weighted image of two benign compression fractures shows typical linear incomplete bone marrow replacement with a peripheral low signal intensity band (arrow).

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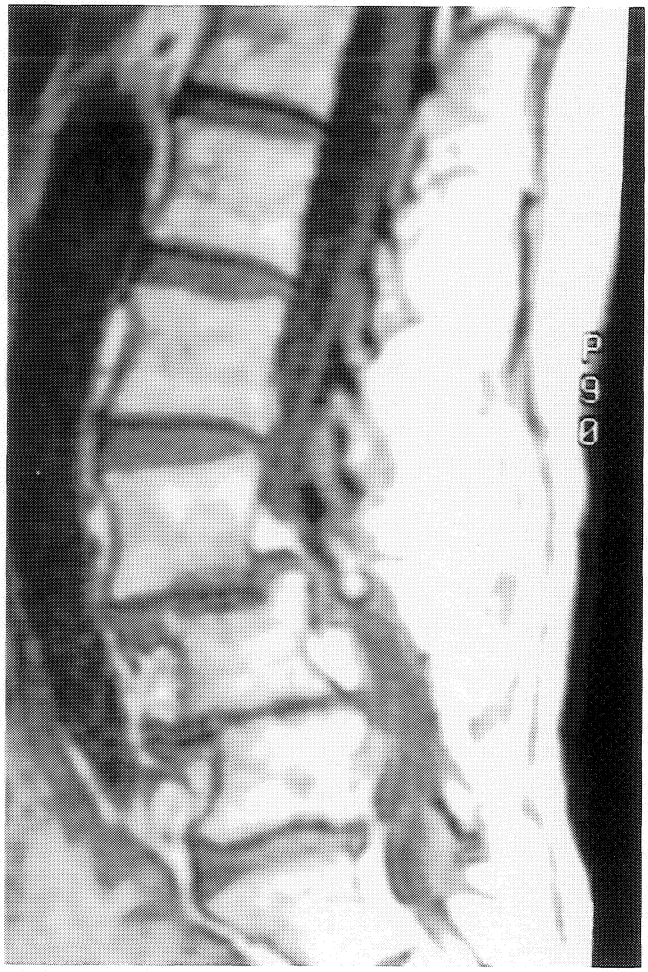


Figure 15
Sagittal T1-weighted MRI of a sixty year old woman with severe lumbar disc degeneration, degenerative cysts in the vertebral endplates and focal instability at L3-4-5. The marrow and disc signals are well maintained.

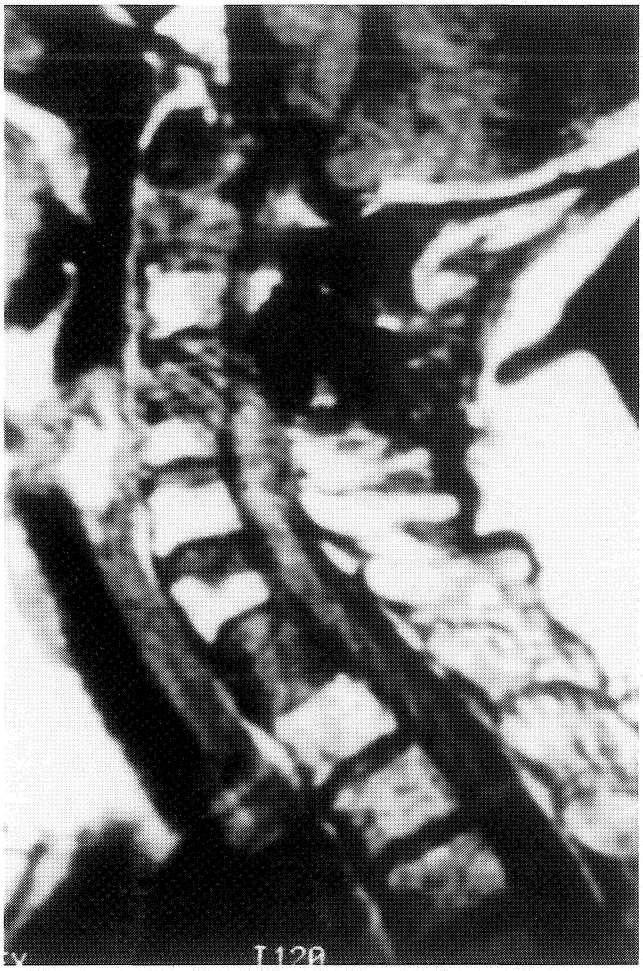


Figure 16a
Sagittal T1-weighted MRI of the cervical spine of a sixty-nine year old woman with widely metastatic breast carcinoma. There are multiple foci of abnormal replacement of the marrow signal particularly apparent in the C1, 2, 4, and T1 vertebral bodies.

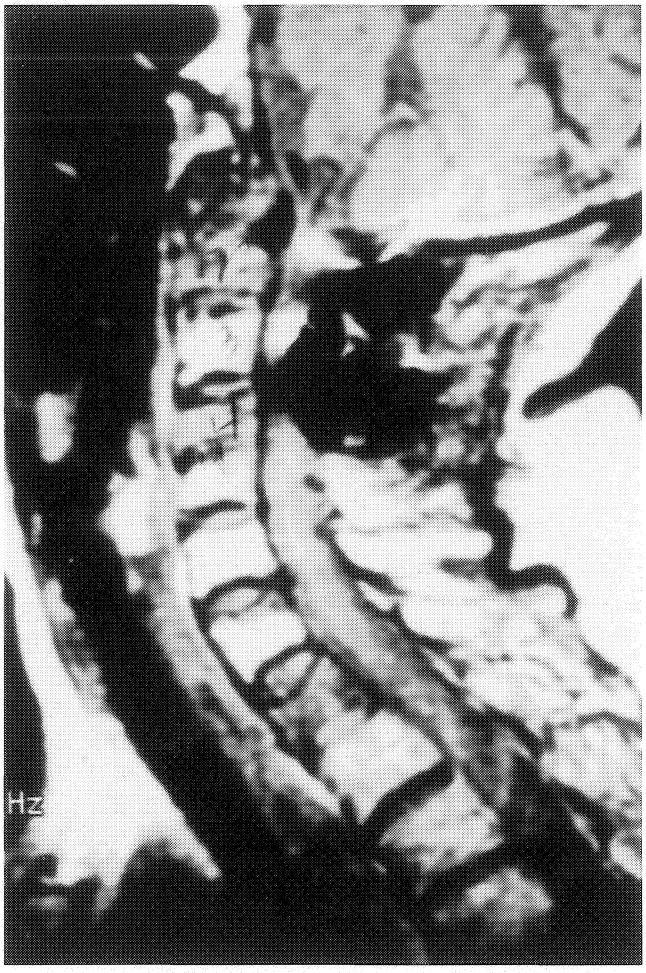
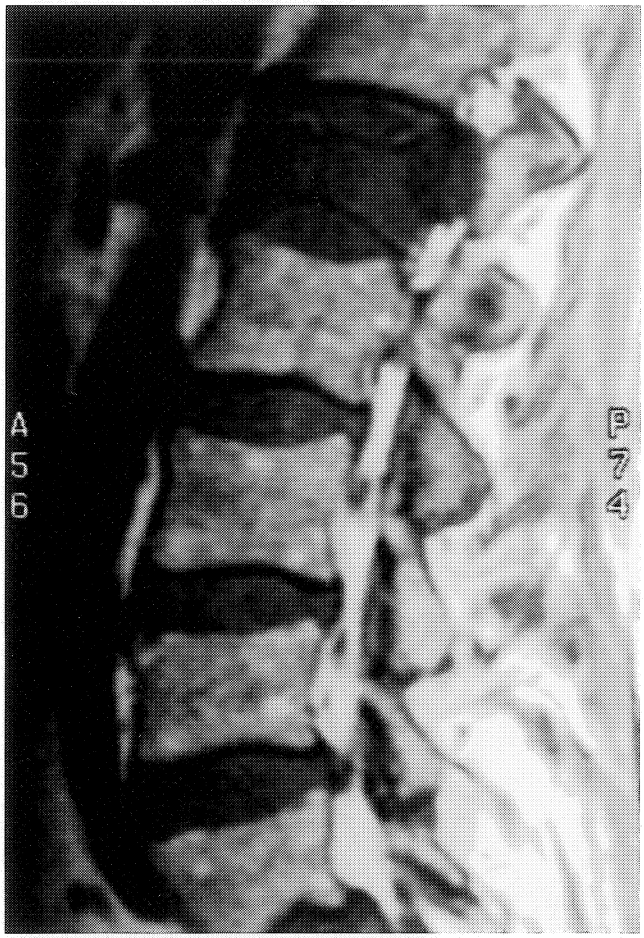


Figure 16b
The T2-weighted MRI demonstrated enhanced uptake in the C3, 5, and 6 vertebral bodies secondary to tumor invasion of the medullary bone and increased fluid secondary to edema.



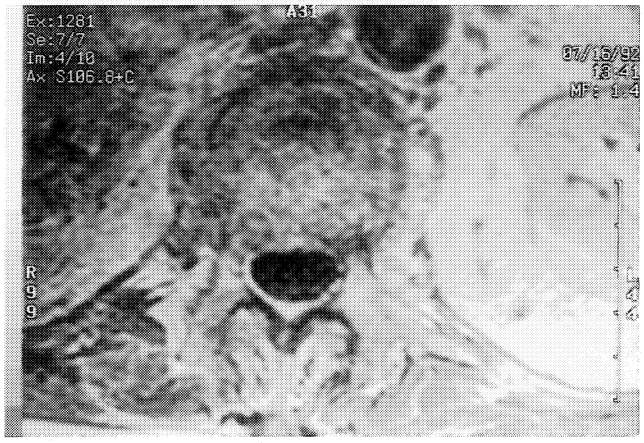


Figure 17a-b
An MRI of a seventy-two year old male with sudden onset of severe thoracolumbar pain without trauma. (a) Sagittal T1-weighted image shows marked and homogeneously decreased signal intensity with some posterior bulging of the vertebral cortex into the canal. (b) The T1-weighted axial image shows that these abnormal signal changes extend into both pedicles. Both T2-weighted images were interpreted as suggestive of tumor infiltration of the vertebral body.

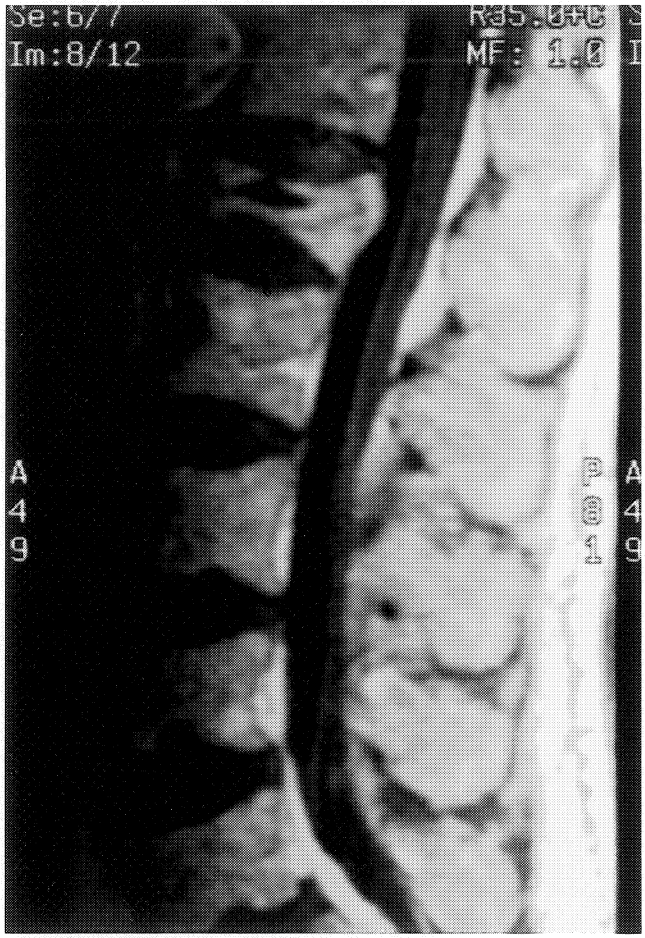


Figure 17c
However, gadolinium enhanced T2-weighted sagittal images show a normal enhancement pattern for the marrow except for a central cleft. This pattern is suggestive of benign compression fracture. A CT controlled trocar biopsy revealed only benign histologic changes and a follow-up MRI in eight months revealed no residual suggestion of a malignant process.

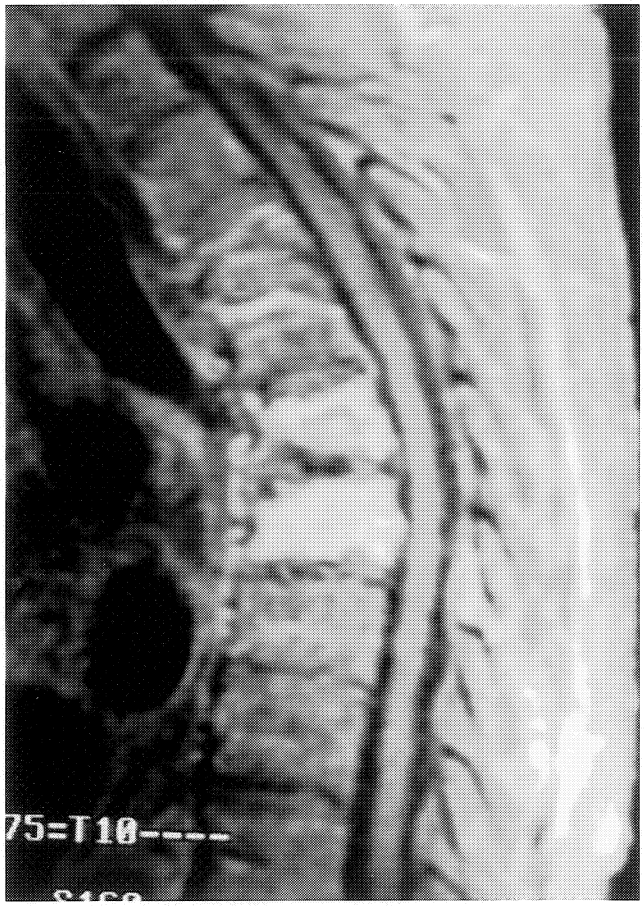


Figure 18a
Sagittal T1-weighted MRI with gadolinium of the thoracic spine of a seventy-eight year old female on chronic steroid therapy for Lupus and also eight years following simple mastectomy for solitary breast malignancy without nodal involvement. She has suffered numerous spontaneous thoracic and lumbar compression fractures. There is moderate loss of height of the T6 and T7 vertebral bodies with minimal cortical expansion and with the endplates intact. These findings are suggestive of a benign compression fracture. However, diffuse marrow enhancement by gadolinium is suggestive of metastasis.

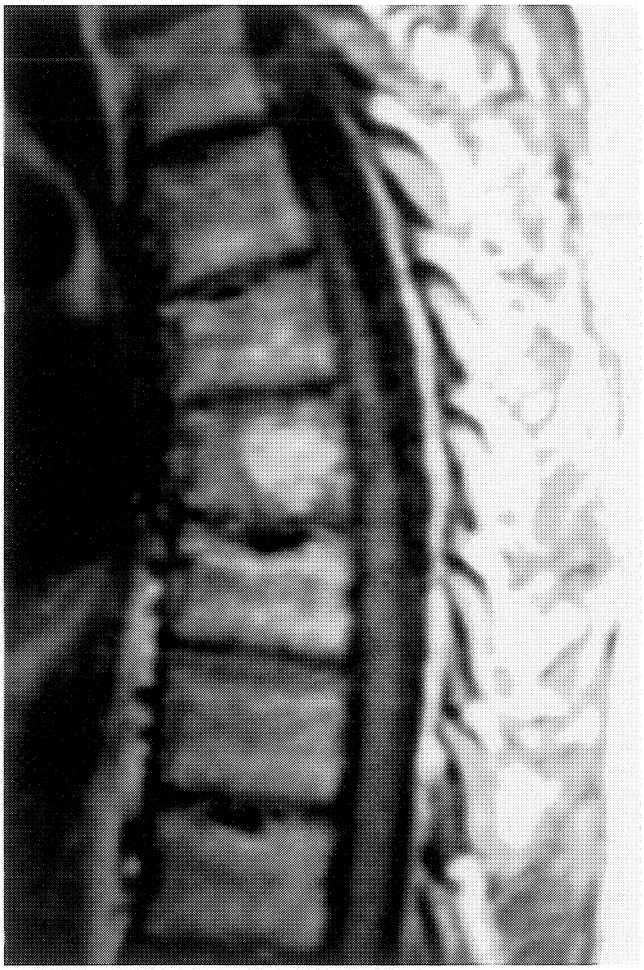


Figure 19
Sagittal T1-weighted MRI of the thoracic spine in a twenty-seven year old male with compression fractures of T6 and T7 after mild trauma. A discreet circumscribed lesion is apparent in the posterior body of T7. However, in this patient the marrow signal is well maintained in the remainder of that vertebra and in all others. This proved to be a benign hemangioma.

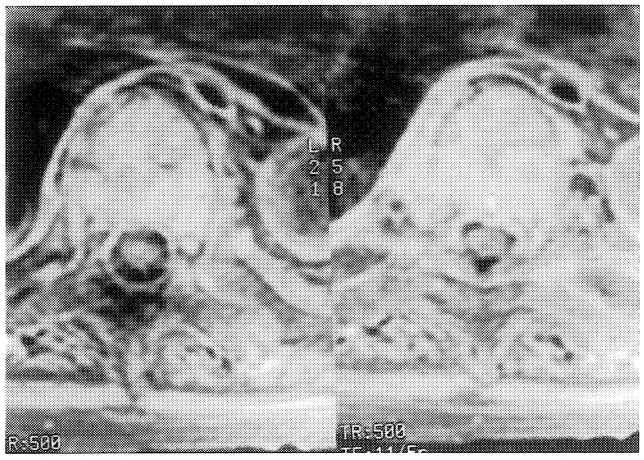


Figure 18b
On axial T1-weighted MRI with contrast, this marrow signal abnormality can be seen to extend posteriorly into the pedicles and perhaps into the lateral masses, suggestive of possible malignant tumor infiltration. This is also compatible with reactive changes occasionally seen with benign fractures. A follow-up MRI after six months showed resolution of these worrisome changes and evidence of progressive healing of these benign fractures.

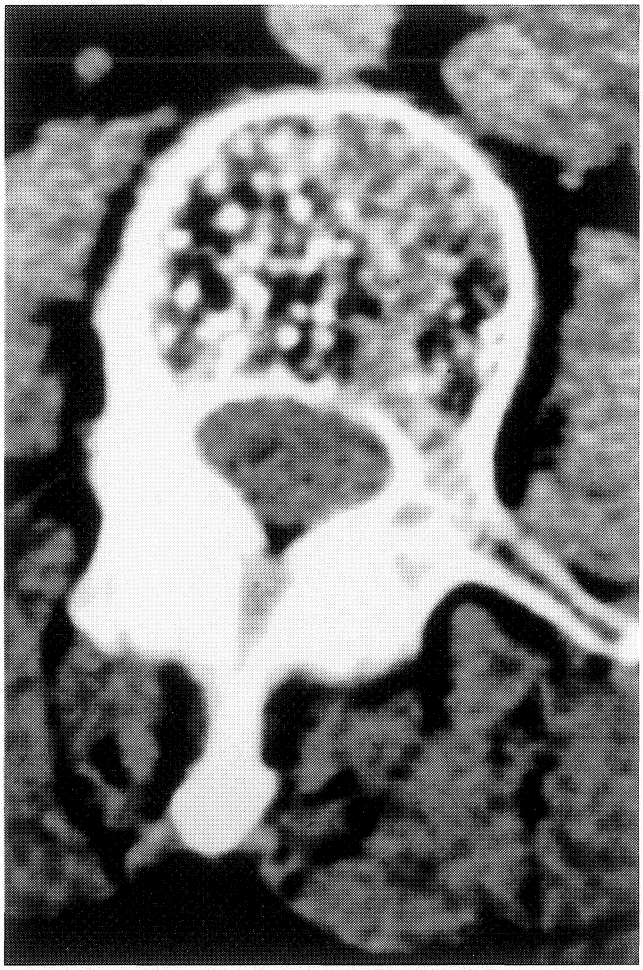


Figure 20
Cross-sectional CT scan of L3 in a patient with chronic low back pain. Apparent is the characteristic pattern of a benign hemangioma with areas of low density separated by remaining high density bony trabeculae. Histologically, the lesion consists of many vascular channels resulting in bone resorption with the remaining trabeculae becoming more prominent. Paget's disease of bone and occasionally osteoporosis may have a similar appearance.

RECURRENT SHOULDER DISLOCATION

James V. Nepola, M.D.
Kenneth E. Newhouse, M.D.

INTRODUCTION

Dislocation of the shoulder is a common orthopaedic injury. It is usually the result of trauma, although "atraumatic" and even voluntary dislocation of the shoulder may occur.

Special attention should be given to trauma patients complaining of shoulder pain in which an AP radiograph is "normal", since posterior dislocations may be easily overlooked on this view. In addition, posterior dislocations are often associated with seizures, electrocution, and voluntary "dislocation". It is especially important to recognize the latter group of patients, as surgical correction of the pathology is doomed to failure.

Rowe, in his study of 500 shoulder dislocations seen over a twenty year period, showed that this injury occurred with equal frequency in persons under and over 40 years of age[1]. However, persons in the younger group were much more likely to redislocate than were their older counterparts. This has been corroborated by others. Rowe suggests that this may be due to increased demands placed on the shoulder, or the longer period of time over which they have a chance to dislocate. In addition, it may have to do with the reduced elasticity of connective tissue in the older patient.

Recurrent shoulder dislocations are more commonly a problem seen in young adults. The initial dislocation is most often secondary to trauma such as that sustained in a motor vehicle accident or as a result of a sporting injury.

Patients may complain of weakness, a perception of instability, or "apprehension" when using the shoulder in certain positions at risk. Pain may be present but is rarely the primary complaint.

Anatomy

The scapula is oriented approximately 30 degrees anterior to the coronal plane of the body, thus orienting the glenohumeral joint with the humerus anterior to the glenoid. This accounts for the vast majority of dislocations being anterior. It also has important implications for imaging this joint; a true AP radiograph is therefore not in the coronal plane of the body.

The anatomy of the glenohumeral joint can be divided into four aspects: (1) the bony anatomy comprised of the scapula, glenoid, and humeral head (2) a labrum or rim of tissue surrounding the glenoid (3) the capsule with incorporated ligamentous structures, and (4) the muscles of the rotator cuff that surround the joint.

Although the glenohumeral joint is a modified ball and socket, it can be contrasted with the hip. The hip has a deep stable articulation with relatively confined motion and is designed for weight bearing. The glenohumeral articulation is comprised of the spherical humeral head and slightly concave glenoid which is shaped like an inverted comma. This joint is quite shallow and allows for a tremendous range of motion, but has much less stability than the hip.

Surrounding the glenoid is a fibrocartilaginous structure attached to its edge, the labrum. This structure serves to deepen the glenoid socket and contain the humeral head. It also serves as an attachment for the capsule and the ligamentous structures about the shoulder. The discontinuity of the anterior structures may predispose a patient to persistent instability unless surgically corrected. Repair of the detached of the labrum in recurrent shoulder dislocations was described by Bankart in 1923[2]; however the procedure was first performed by Perthes in 1906[3].

The shoulder capsule is a relatively thin structure with condensation in its substance constituting the superior, middle, and inferior glenohumeral ligaments. These structures offer added stability to the shoulder joint. The inferior glenohumeral ligament in particular can serve as a restraint to both anterior and posterior translation of the humeral head.

The muscles of the rotator cuff (supraspinatus, infraspinatus, subscapularis, and teres minor) play a major role in the *dynamic* stability of the glenohumeral joint. This group of muscles acts to anchor the humeral head against the glenoid. Individually, each muscle helps prevent specific directional translation based on its position. A deficiency in any one muscle group can result in instability due to the unopposed action of the remaining musculature. The long head of the biceps is an intraarticular structure which also serves as a head depressor and prevents anterior translation of the humeral head.

Physical Examination

Proper examination of the patient requires that the examiner observe both shoulders from the front and back. Notation is made of any surgical scar, atrophy, or asymmetric posturing of the shoulders. Active and passive forward flexion (elevation), abduction, internal and external rotation should be assessed. The approximate degree

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of active range may be obtained with the patient upright, but accurate measurements of elevation and external rotation are more reproducible with the patient supine. While assessing passive motion, a hand placed over the shoulder can discern any crepitance. Classically, the patient with recurrent dislocation has normal range of motion although guarding or apprehension may be seen at an *"at risk"* position.

Muscular strength should be assessed by resisting internal rotation, external rotation, and abduction. This generally tests all components of the rotator cuff and deltoid. Axillary nerve lesions with deltoid dysfunction and atony are common after dislocation. Marked weakness of the rotator cuff may also indicate rupture of the cuff tendon, especially in the older patient. Comparison with the contralateral side is crucial when assessing weakness.

Testing for instability can be divided into maneuvers which cause apprehension and those which demonstrate abnormal translation of the humeral head. There is considerable variability in the "normal" amount of laxity in the shoulder joint. Therefore, it is very important to examine each patient's unaffected shoulder first.

With the patient seated, relaxed, and his/her arm at the side, the examiner stabilizes the glenoid side of the shoulder joint and translates the humeral head anteriorly, posteriorly, and inferiorly. The amount of translation which can be obtained in each plane can be graded and compared to the normal side. This translation can range from a minimal movement between the glenoid and the humeral head to actual dislocation.

Judging abnormal translation associated with posterior subluxation can be subtle. In this case a subluxation-relocation maneuver can be extremely helpful. With the patient supine, the humerus is forward flexed to 90 degrees and internally rotated. The elbow is then gently forced backwards axially. If painful and resisted by the patient, this constitutes posterior apprehension. Posterior subluxation of the humeral head may or may not be recognized by the patient or examiner. While in this position the scapula is stabilized and the elbow is moved into abduction; a posteriorly subluxated humeral head may reduce with a palpable or audible clunk. This procedure is very similar to the Barlow maneuver used in the diagnosis of congenital hip dislocation.

Testing for anterior apprehension is performed with the arm abducted and externally rotated 90 degrees. While in this position, the humeral shaft is gently levered anteriorly. Resistance and pain are indicative of impending shoulder dislocation and constitute a positive test.

Imaging

Routine AP and axillary views of the glenohumeral joint are to be done in patients with shoulder instability. Occasionally "notch views" may show a significant impression fracture of the posterosuperior humeral head (Hill-Sach's lesion). In the absence of significant abnormality on plain radiographs[21], sophisticated imaging studies are not indicated for the evaluation of recurrent dislocation. Arthrogram-CT scan or MRI may reveal the presence of Bankart lesions (labral detachment). However, if surgery is indicated for treatment of recurrent shoulder dislocation, this lesion will be discovered and appropriate reconstruction may be performed. Costly studies are therefore not required.

Indications

The incidence of recurrent dislocation after initial anterior dislocation ranges between 10 and 95%[1,4,5,6]. Incidence varies with age[1], with the adolescent patient more prone to recurrence[7,8]. Although some clinicians feel muscular rehabilitation to be the appropriate treatment for patients with recurrence, after a second shoulder dislocation the likelihood of recurrence is high. In the patient who experiences a repeat episode of dislocation within a year, it is reasonable to advise surgical reconstruction. Contraindications remain in patients with uncontrolled seizure disorders, voluntary dislocations, or associated severe neuromuscular deficits. Patients should be advised preoperatively that compliance with postoperative immobilization and the rehabilitation regimen is necessary for success. Postoperative spica casting may be necessary in patients who are unable to cooperate, such as in a patient with mental retardation.

Authors Preferred Method

Our approach is based on anatomic restoration of shoulder stability. Other procedures have been described which achieve stability through limitation of range of motion, such as the Putti-Platt[9] or Magnuson-Stack[10] operations. Other strategies involve the use of a bony block to dislocation, such as the Bristow procedure[11]. These operations, while successful, are not based on restoring normal anatomy, and may not address the causative factors associated with the patient's instability.

Anatomic deficiencies which may be corrected surgically are commonly osseous, such as a glenoid rim fracture or humeral head impression fracture. Soft tissue abnormalities include Bankart lesions, capsular redundancies, and less commonly tendon disruptions such as avulsion of the subscapularis.

The following procedures for anterior and posterior reconstruction attempt to correct the anatomic deficiency while allowing restoration of near normal range of motion. In the majority of cases, instability is secondary to detachment of labral tissue or capsular redundancy (Figure 1). Occasionally one needs to address glenoid insufficiency. When greater than one-third of the surface of the glenoid has

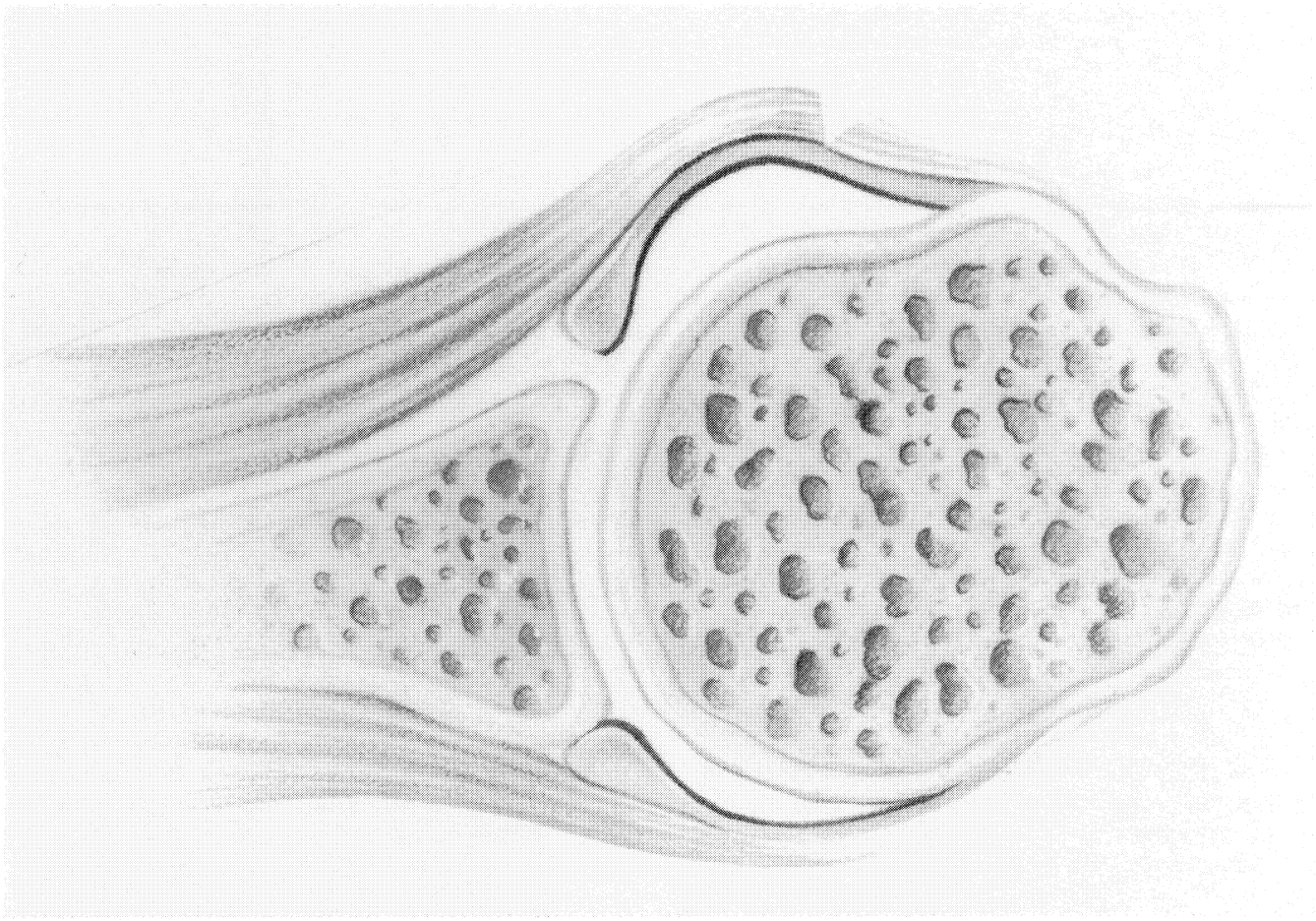

Figure 1 Cross-section of the glenohumeral joint. Note avulsion of anterior labrum and intact posterior labrum with capsule.

been displaced, consideration should be given to iliac crest grafting to restore bony integrity of the corresponding anterior or posterior articular surface. Likewise, a significant humeral head impaction (Hill-Sachs or reverse Hill-Sachs lesion) would influence one toward transplantation of either the subscapularis or infraspinatus tendon into the respective deficits. Fortunately, bony deficiency is a rare problem. In seven years of orthopaedic practice specializing in shoulder surgery, the author has yet to encounter a case requiring such bony reconstruction for cases of recurrent dislocation.

Preoperative Examination Under Anesthesia

Prior to embarking on any surgical repair, the surgeon should carefully examine the patient under anesthesia with paralytic agents to assess the instability pattern and make final plans for the appropriate procedure. A patient thought to have routine anterior recurrent dislocation may demonstrate significant posterior laxity. The rare patient who describes multiple episodes of dislocation without radiographic documentation may rarely be manifesting a psychological disorder rather than an orthopaedic problem.

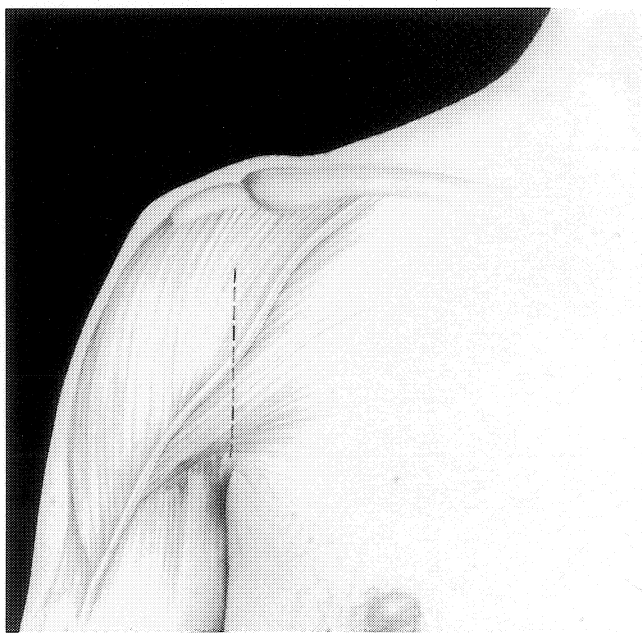

Figure 2 Planned incision for anterior shoulder reconstruction.

The examiner should confirm the preoperative diagnosis by actually dislocating the shoulder either anteriorly or posteriorly.

Anterior Shoulder Reconstruction

The patient is placed in a modified beach chair position in 20 degrees of flexion at the waist. A short arm board may be used beneath the elbow to support the arm during the procedure. The patient is moved to the side of the operating room table, placing the axillary line over the edge. The axilla is wet-shaved using a mild antiseptic soap. The shoulder is prepped from neck to nipple to hand. The posterior aspect of the shoulder should be prepped and accessible.

An incision is made in the anterior axillary line approximately 8-9 centimeters (cm) in length (Figure 2). It may be positioned so that when standing with the arm internally rotated, the scar will lay within the fold of the skin. In some cases, cosmesis dictates the use of the axillary approach. Though exposure is adequate with this approach, the relative cosmetic improvement should be weighed against possible increased risk of infection.

The incision is deepened and the skin and subcutaneous tissues are elevated from the underlying muscle fascia, allowing adequate exposure of the deltopectoral groove. The deltopectoral interval is defined by the cephalic vein. This interval is bluntly dissected. Using the fingers, the vein is retracted laterally with the deltoid. Branches of the vein are coagulated. The plane between the deltoid, the pectoralis major, and the clavipectoral fascia is bluntly dissected. The clavipectoral fascia is incised along the lateral edge of the conjoined tendon, which is defined and retracted medially to reveal the subscapularis muscle and tendon (Figure 3). The coracoid process need not be osteotomized. The anterior circumflex humeral veins can be seen marking the inferior border of the subscapularis. These are cauterized laterally.

With rotation of the humerus, the bicipital groove between the lesser and greater tuberosity is palpated. The subscapularis tendon is incised 1.5 to 2 cm from the bicipital groove. This leaves ample tendon for reapproximation. Separation of the subscapularis tendon from the underlying capsule may be facilitated by passing a blunt curved clamp (Shallcross) between the subscapularis tendon and the capsule from the inferior border of the subscapularis to the rotator interval. The tendon is incised over this clamp, avoiding the capsule. In cases of reoperation, scarring will necessitate a more careful dissection of the tendon from the capsule, erring on the side of leaving tendon fibers on the capsule. The tendon and muscle of the subscapularis are then dissected medially off the capsule with the use of a Darrach or Cobb elevator and retracted. The rotator interval is closed with one or two nonabsorable sutures, taking care to avoid the long head of the biceps tendon (Figure 4).

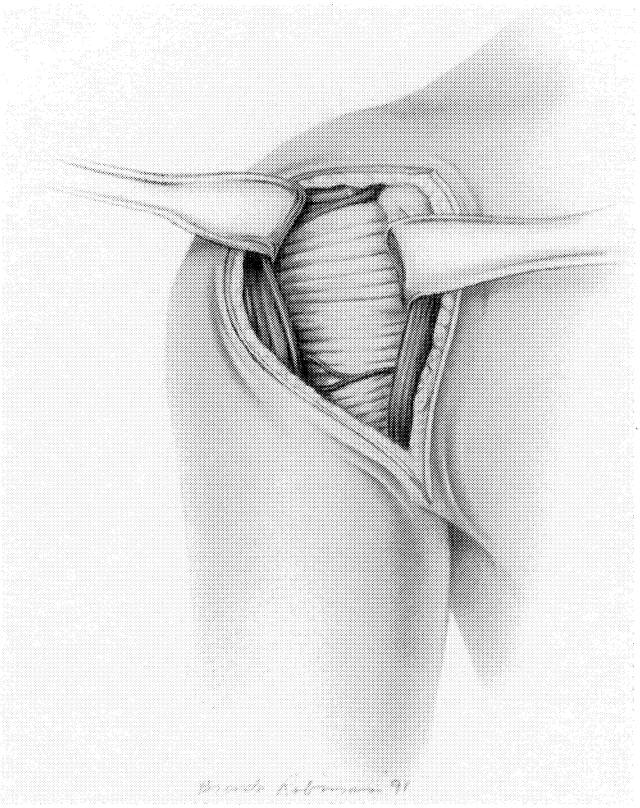

Figure 3 After incising the medial border of the clavipectoral fascia, the subscapularis muscle and tendon are readily visualized.

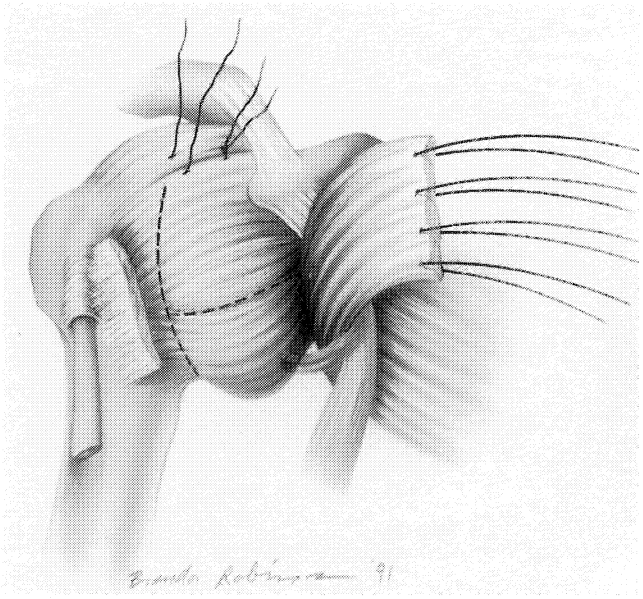

Figure 4 Closure of rotator interval.

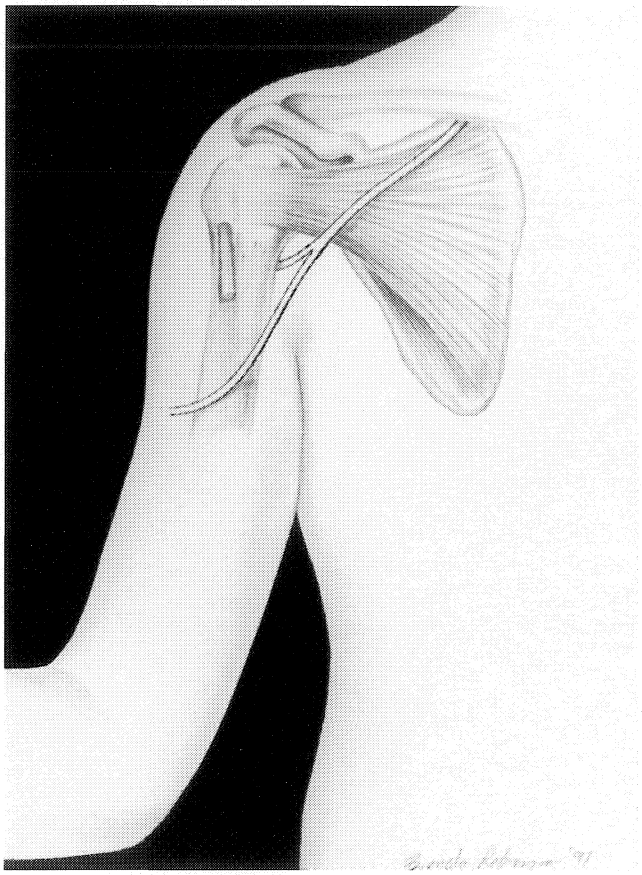

Figure 5 Proper positioning of the arm prior to capsule incision. With the arm adducted and externally rotated the axillary nerve retracts away from the humeral neck.

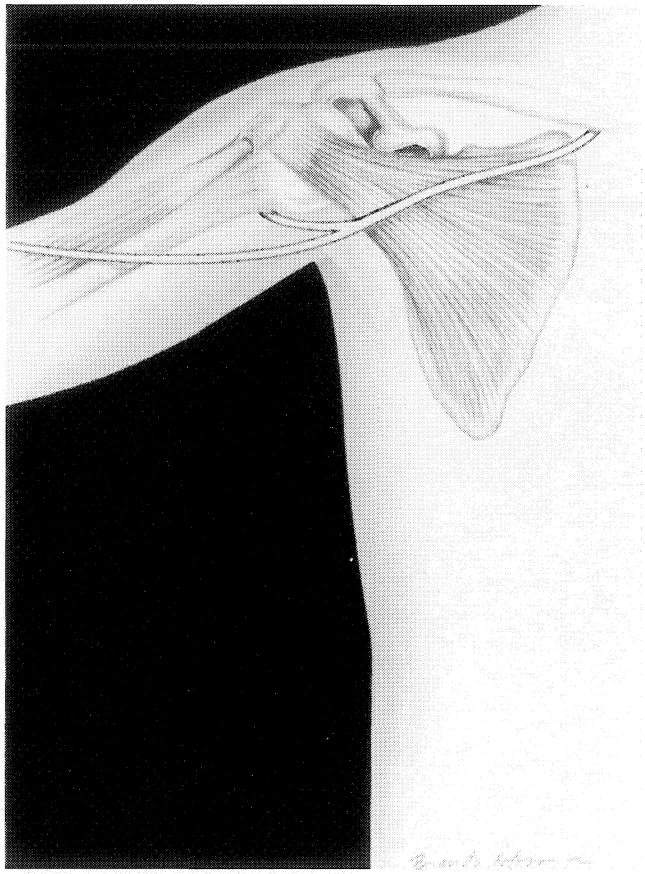

Figure 6 Improper positioning (abduction), forces the axillary nerve in proximity to the humeral neck. This increases risk of iatrogenic injury.

The capsule is now incised approximately 5 mm medial to the subscapularis tenotomy along the anatomic neck of the humerus. The arm is kept at the side in 40 degrees of external rotation to avoid forcing the axillary nerve into proximity with the inferior humeral neck (Figures 5 and 6). At the juncture of the upper two-thirds and inferior one-third of the capsular incision, two tagging sutures are placed in the edge of the capsule. The capsule is then "T'd" toward the "four o'clock" position on the glenoid, or toward the center of a Bankart lesion. The joint is examined and any loose debris removed.

Superior and lateral traction is placed on the inferior capsular flap. Using a Cobb elevator, the inferior aspect of the capsule is released from the anatomic neck of the humerus as necessary to eliminate any further inferior redundancy (Figure 7). This is generally minimal in cases of routine anterior recurrent dislocation.

A ringed humeral head retractor (Fukuda) is carefully inserted to facilitate visualization of the anterior glenoid (Figure 8). In cases without detachment of the labrum, attention is turned to the capsulorrhaphy. However, when a Bankart lesion exists, this is to be repaired. The extra-articular edge of the glenoid rim is roughened using

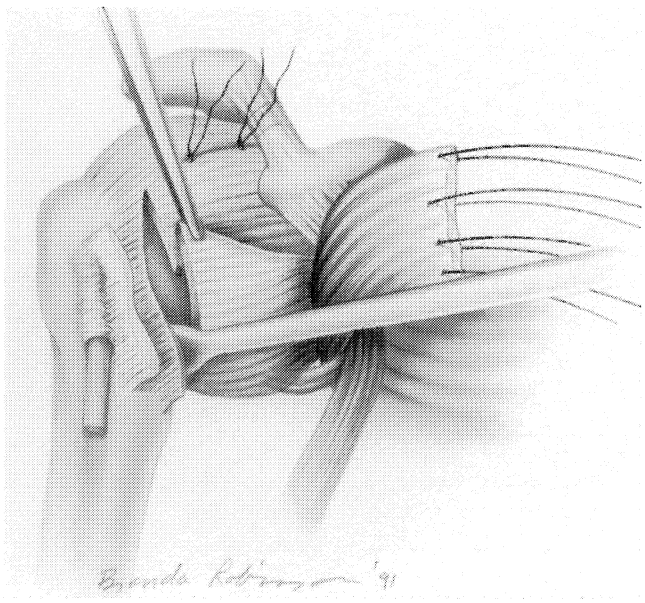

Figure 7 Releasing capsular adhesions allows reduction of capsular redundancy prior to final repair.

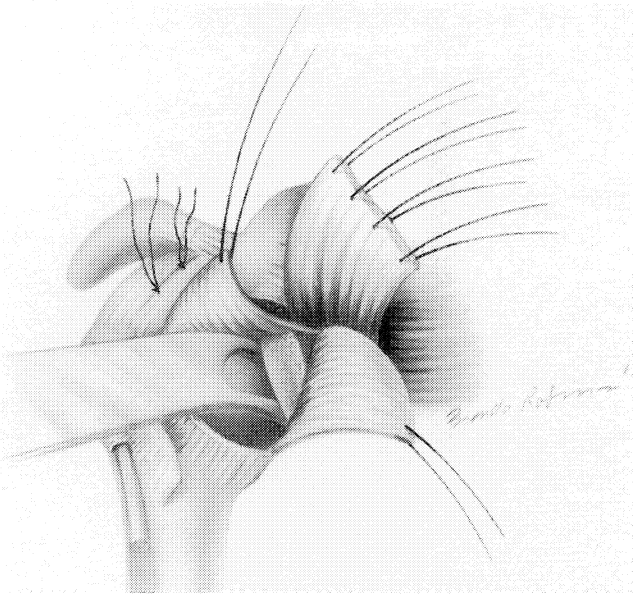

Figure 8 Using the Fukuda retractor, the humeral head is retracted posteriorly; the anterior glenoid is then visualized.

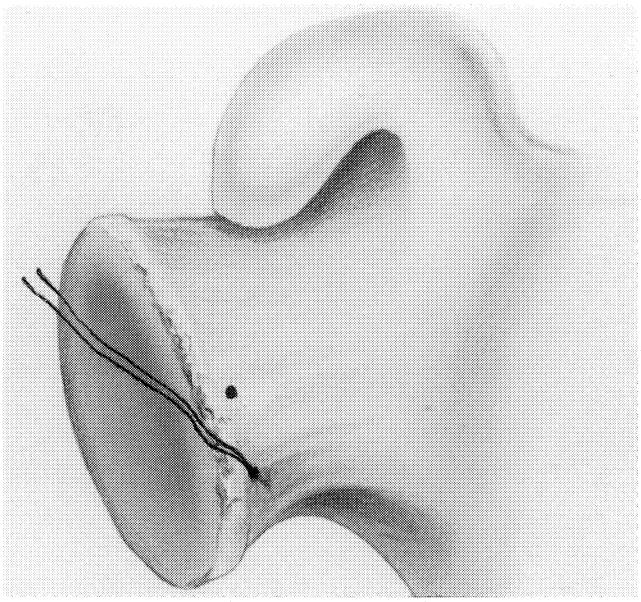

Figure 10. Recently the development of interosseous anchors has eased reattachment to the labrum.

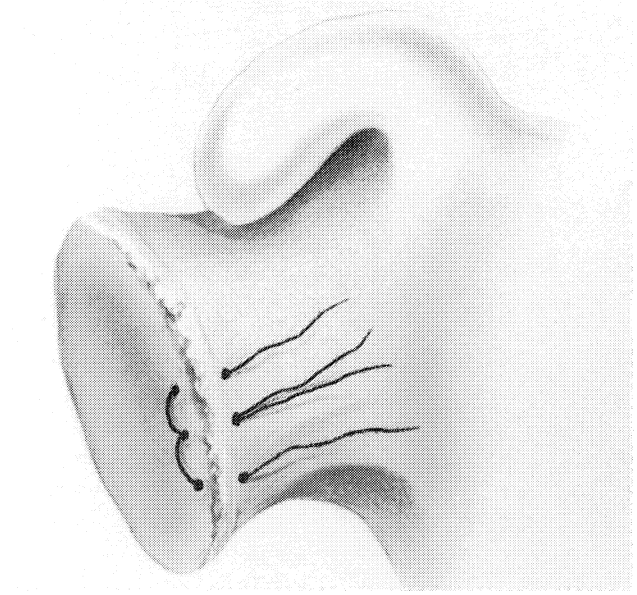

Figure 9 Traditional technique of suture anchoring for Bankart repair.

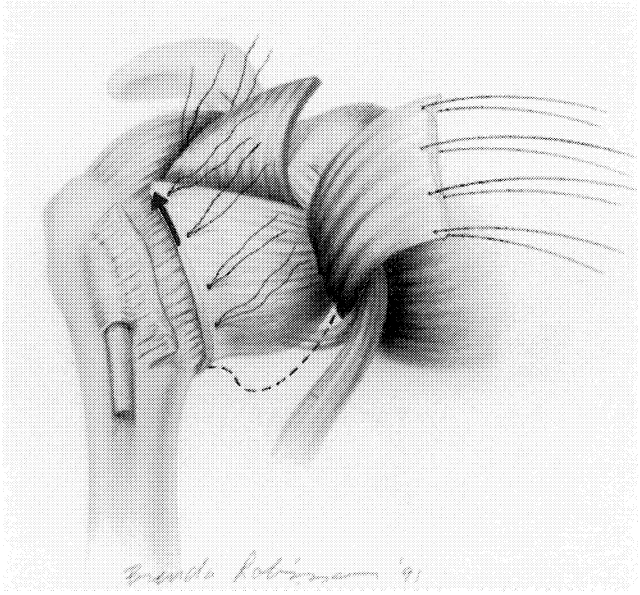

Figure 11 Stage I of the capsulorrhaphy with the inferior capsule sutured superolaterally.

a surgical burr or curette. Anchoring sutures are then placed into the rim of the glenoid. Formerly, these were passed through drill holes traversing the glenoid rim (Figure 9). Recently, the use of interosseous suture anchors has facilitated the procedure (Figure 10).

The capsule is retracted laterally and the anchored sutures are passed through and tied firmly, reaffixing the labrum to the edge of the glenoid. This is tested with the surgeon's finger. It should be noted that an inadvertent capsulorrhaphy may be performed at this stage if the Bankart repair sutures are placed too far laterally through the capsule.

With the capsule reattached, attention is turned to the capsulorrhaphy. This is performed in the style of Neer, Mendoza, and others[12,13,14]. With the arm in 40 degrees of external rotation and adducted at the side, the inferior capsular flap is advanced superiorly and laterally and sutured to the capsular remnant with nonabsorable sutures (Figure 11). This eliminates the inferior and anterior redundancy. In revision surgery, inadequate capsule sub-

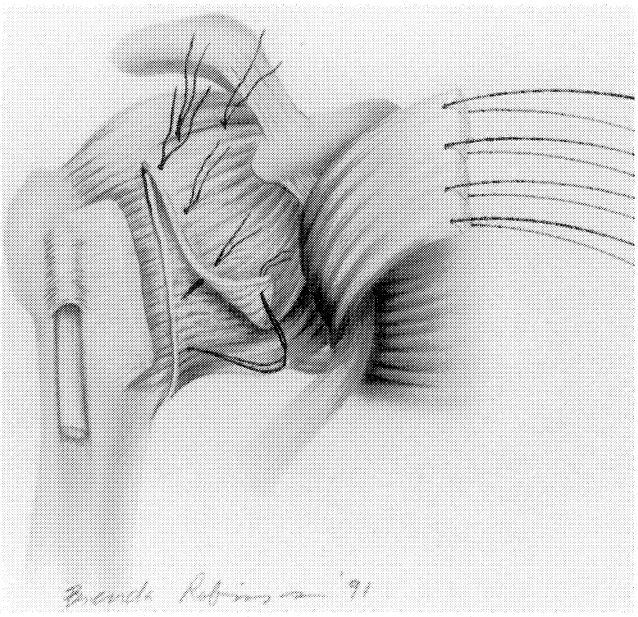

Figure 12 In the final stage of the "Pants over vest" capsulorrhaphy the superior capsule is reattached inferolaterally.

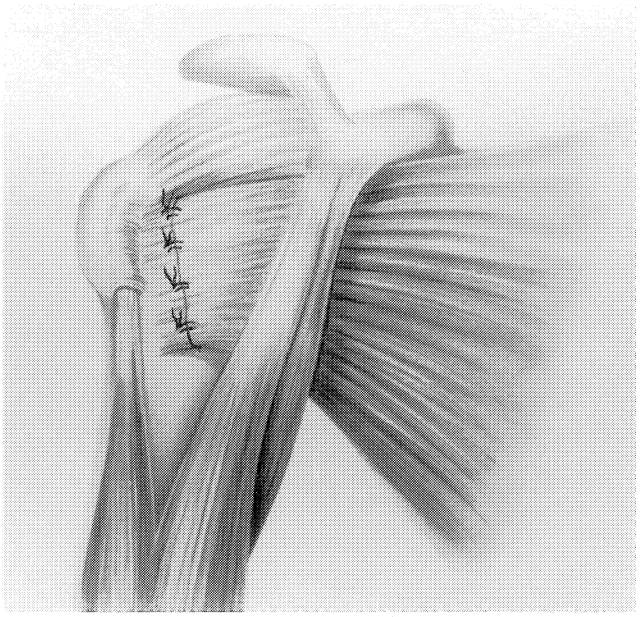

Figure 13 Anatomic repair of the subscapularis tendon.

stance may occasionally require the passage of anchoring sutures through a bony trough created in the anatomic neck. Sutures reattaching the lateral aspect of the capsule are not cut. These are left long. They will be utilized to imbricate the upper capsular flap over the inferior, when closing the capsulorrhaphy. This completes the anterior capsular repair (Figure 12).

With the capsulorrhaphy complete, the tendon of subscapularis is reattached in its anatomic position using absorbable sutures (Figure 13). The wound is closed with

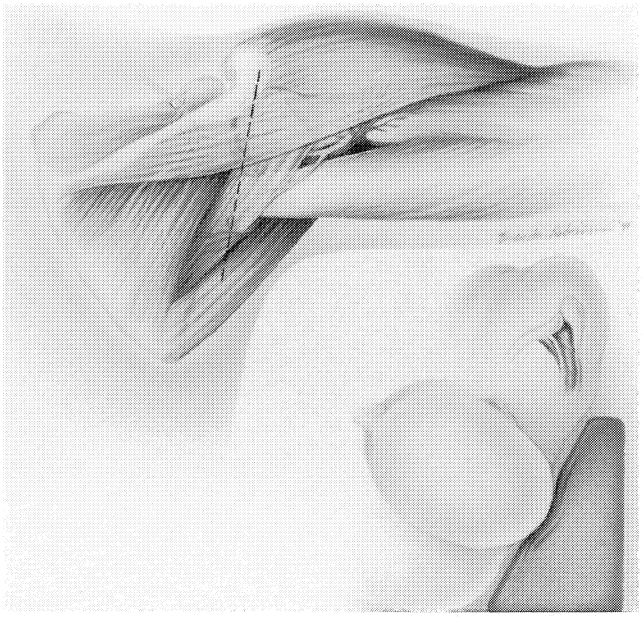

Figure 14 Positioning and incision for posterior shoulder reconstruction.

subcutaneous sutures followed by a subcuticular skin closure, utilizing a monofilament absorbable suture. The arm is placed in a shoulder immobilizer in the internally rotated or "Velpeau" position.

Postoperatively, the patient is examined for neurovascular function. Of special importance is the exam for integrity of musculocutaneous and axillary nerves. The patient may be requested to isometrically abduct the arm against the hand of the examiner while still in the sling. Activity of the deltoid muscle can be palpated. Sensory exam alone for axillary nerve function is inadequate to assess function. Ability to flex the elbow with firing of the biceps musculature confirms function of the musculocutaneous nerve. Postoperative immobilization using the shoulder immobilizer is maintained for three or four weeks in most cases. This time may be extended in younger patients where concern for the integrity of the repair outweighs the fear of stiffness. The patient is to perform isometric muscle strengthening exercises involving the deltoid and shoulder *external* rotators while immobilized. Range of motion exercises are performed involving the elbow. In older patients, gentle pendulum exercises with the arm in internal rotation may be begun under the supervision of the knowledgeable therapist after the second week. No heavy lifting is permitted for six months after repair. In the competitive athlete, sporting activity is curtailed for a similar period of time.

Repair of Recurrent Posterior Dislocation

The patient is placed in the lateral decubitus position and inclined slightly posteriorly for easy assess to the poste-

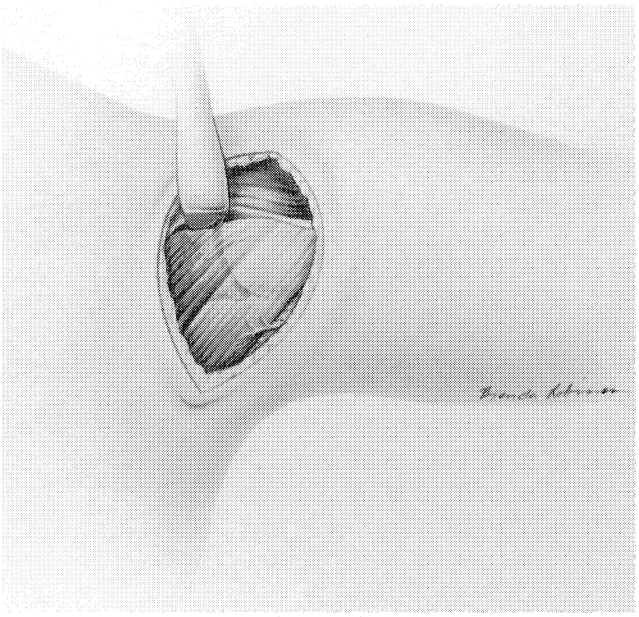

Figure 15 Lateral retraction of the deltoid muscle exposes the rotator cuff.

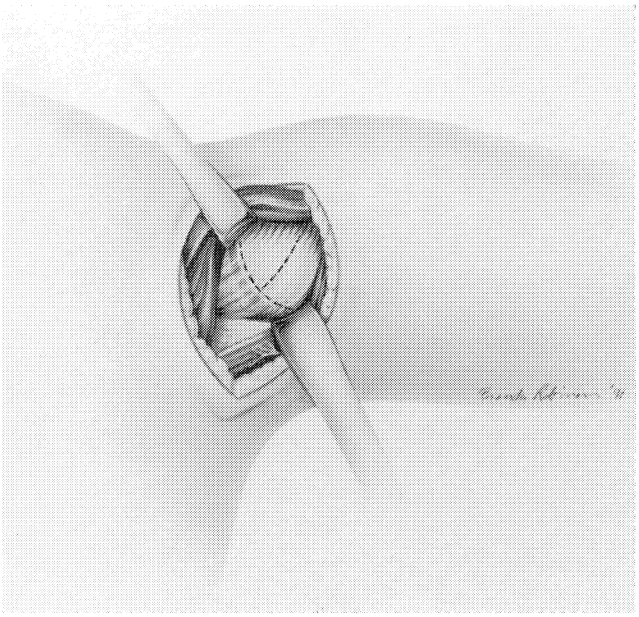

Figure 16 The interval between the teres minor and infraspinatus is developed.

rior shoulder. The axilla is wet shaved. The patient is prepped from the shoulder including the neck and angle of the scapula to the hand. The surgical approach used is similar to that suggested by Brodsky, et. al[15].

With the arm at 90 degrees of abduction, a vertical incision 8-9 cm in length is made in the posterior axillary line from the acromion distally (Figure 14). The medial edge of the deltoid is mobilized and retracted laterally, exposing the underlying infraspinatus and teres minor musculature (Figure 15). Occasionally, the inferior most

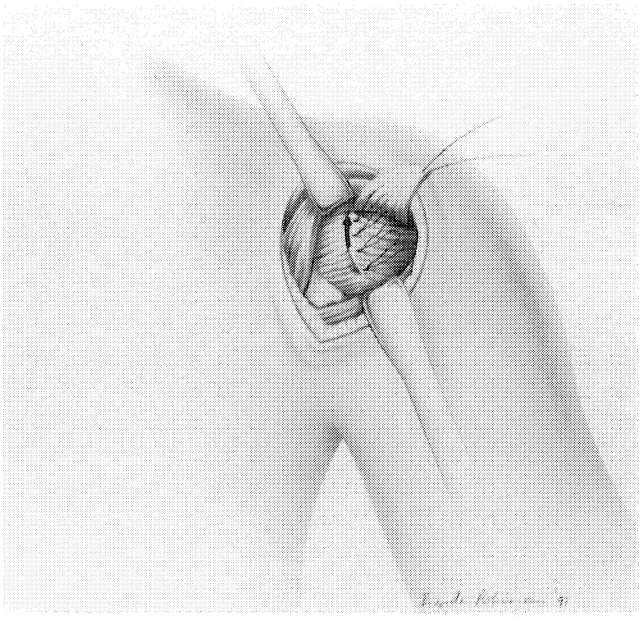

Figure 17 Retraction of the inferior capsular flap and suture is performed first.

insertion of the deltoid may be released to ease exposure. The interval between infraspinatus and teres minor is dissected bluntly and the rotator cuff musculature is retracted, exposing the capsule (Figure 16). The capsule is incised along the joint line. The surgeon is careful to palpate and protect the axillary nerve exiting the quadrangular space inferiorly. With the arm in abduction, the nerve may be pulled close to the capsule at the humeral neck and is vulnerable. For this reason, the inferior extent of the capsular incision is best made with the arm in adduction and external rotation.

As in the anterior procedure, the capsule is "T'd" and the flaps imbricated. With the arm at the side and in neutral rotation, the lower capsular flap is advanced cephalomedial and sutured to the capsule remnant and labrum with nonabsorbable braided suture material (Figure 17). The sutures are tied, but not cut. The tails are then passed through the upper flap which is imbricated over the lower flap completing the repair (Figure 18). The wound is then irrigated and subcutaneous tissue approximated with absorbable suture. The skin is closed with a subcuticular absorbable monofilament suture. The patient's shoulder is maintained in neutral rotation throughout the capsular repair and closure. After bandaging, it should be splinted in that position to avoid tension on the repair. The arm is maintained in this position for six weeks. During this time, isometric strengthening of the deltoid and the rotator cuff musculature may be performed. After removal of the splint, range of motion exercises are begun. Activity restrictions are maintained for six months as in patients with anterior repair.

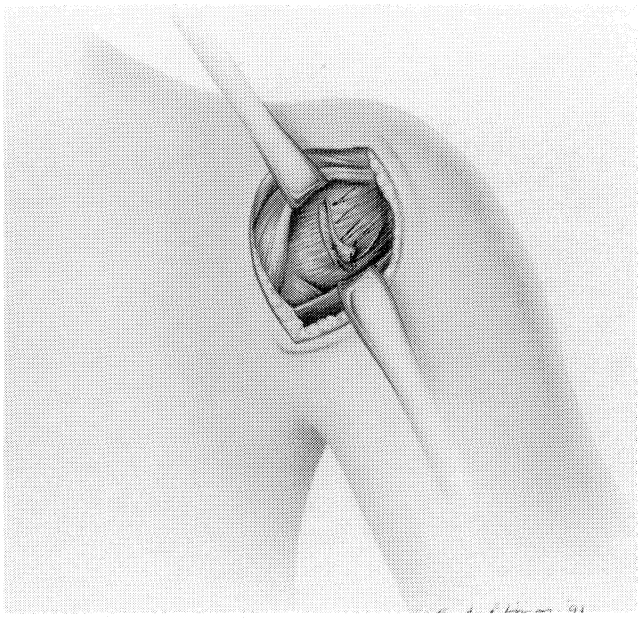

Figure 18. Final "Pants over vest" repair of the posterior glenohumeral joint.

Complications

Complications associated with operative treatment of recurrent dislocation involve infection, redislocation, neurovascular injury, joint contracture, and arthrosis. Infection rates should be similar to those of other procedures in the given institution, usually less than 1-2%.

Redislocation is an uncommon complication which has been reported to range between 0 and 18%[16,17,18]. Rockwood has reviewed over 3,000 operative cases in the literature and noted a redislocation incidence of 3%[17]. Interestingly, it has been noted that redislocation may often occur two or more years after surgery[16]. The most commonly cited reason for recurrent instability has been unrepaired Bankart lesions with associated capsular laxity[17].

Neurologic injury may occur during reconstruction, but it is uncommon. This usually involves either the axillary or musculocutaneous nerve. The proximity of the axillary nerve during dissection of the inferior capsule along the neck of the humerus places it at risk. It should be identified and protected during capsular release, and care should be taken that sutures placed in the capsule do not inadvertently compromise the nerve. the musculocutaneous nerve may be placed under tension by retraction of the conjoined tendon. This can be compounded by coracoid osteotomy. Care should be taken to identify and preserve the musculocutaneous nerve in cases where coracoid transfer is anticipated. The authors believe that adequate exposure is obtained without osteotomy, and advise against such procedures for routine anterior repair. Injury to the axillary artery is rare and usually involves hardware complications[19].

Many procedures have been designed to achieve glenohumeral stability through restriction of range of motion and by avoiding so called positions "at risk". Most often, these precautions are used for anterior instability and will restrict external rotation[8,9]. Overtightening of the anterior capsule and musculature can unfortunately result in extrusion of the humeral head posteriorly, and is cited as one of the problems associated with unrecognized multidirectional laxity. Arthrosis associated with recurrent dislocation has been reported, although it is infrequent[20].

The overall incidence of complications associated with surgery for reconstruction of the glenohumeral joint in recurrent dislocation is low, and attention to detail will assure that these problems are minimized.

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ASSESSMENT OF DONOR CELL SURVIVAL IN FRESH ALLOGRAFTS (LIGAMENT, TENDON, AND MENISCUS) USING DNA PROBE ANALYSIS IN A GOAT MODEL

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Timothy Simon, M.D.†

INTRODUCTION

There has been increasing interest in replacing irreparably damaged ligaments and menisci with allografts. Conceptually, replacing the abnormal structure with an identical biologic replacement seems ideal if the material and mechanical properties of the replacement structure are maintained. Our initial work in this area began using freeze dried anterior cruciate ligament (ACL) allografts to reconstruct the ACL in a goat model. These grafts were repopulated with host cells that histologically appeared similar to the oriented fibroblasts seen in the control ACLs. Revascularization occurred in all the grafts, and histologically the ACL allografts showed encouraging signs of incorporation. Their ultimate strength to failure at one year was diminished by 75% compared with their strength at the time of implantation and with control ACLs[7]. Augmentation of the ACL allografts improved ultimate strength to failure to 50% of the control ACL[8]. As a result of these observations, we became concerned that the host cells and the associated changes of revascularization would not maintain the initial mechanical properties of any biologic graft used to reconstruct the ACL.

To test this hypothesis we devised a study in which we killed the cells and devascularized an ACL in situ using alternate freezing and thawing[9]. This in situ freeze technique controlled many of the technical variables involving use of allografts for ACL reconstruction. These included several factors that could potentially compromise the end result such as graft sizing, positioning, orientation, tensioning and fixation. This model simulated a perfectly placed ACL collagen graft and allowed evaluation of the changes following cellular repopulation and revascularization. The ultimate strength to failure in these devitalized ACLs initially dropped but returned to normal values by six months. Transmission electron microscopy revealed a proliferation of small diameter collagen fibrils. This increase was associated with an increased cross sectional area in these previously devitalized ACLs[9]. This suggested that the cells that repopulated the ACL were unable to maintain or replace the larger diameter native ACL collagen fibrils. The changes were similar to the altered ACL collagen fibril pattern seen in ACL autografts and allografts. These observations raised the question of transplanting living mature fibroblasts in an ACL allograft in order to maintain the extracellular matrix in a more normal pattern[9]. The existing literature suggests that successful transplantation of living cells is important in the long term survival of both articular cartilage[3,4,17,18] and heart valve transplants[15,19,20,21,22].

The concept of transplanting mature living cells that will maintain the pre-implantation structural and mechanical properties of the allograft is appealing. Meniscal fibrochondrocytes are isolated in an avascular environment, surrounded by the extracellular matrix they produce. The immunoprivileged nature of fibrochondrocytes within the meniscus has stimulated interest in transplanting these tissues with viable cells. We have performed transplantation of fresh and cryopreserved meniscal allografts and documented an increase in water and decrease in uronic acid in their ground substance after six months in vivo[10]. These additional observations made us question further the ability of native cells to survive transplantation.

With the advent of DNA probe analysis it became possible to identify whether cells present in a transplanted fresh allograft were of donor or host origin. Our preliminary studies demonstrated two DNA probes that were capable of distinguishing individual members of the Spanish goat species. Using these probes we documented that the donor DNA rapidly disappeared within four weeks from fresh patellar tendon, anterior cruciate ligament, and meniscal allografts implanted into the stifle joint of the Spanish goat. The donor DNA in these three structures was replaced by host DNA in a simultaneous rapid manner.

Simultaneous full thickness skin transplants in the same animal were not rejected during the interval of rapid loss of donor DNA from the allografts. The absence of rejection of the corresponding skin graft during this period of rapid loss of cellular DNA suggests that no pre-existing antibody associated with an immune reaction was responsible for loss of DNA in the allografts[13].

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MATERIALS AND METHODS

Pilot studies confirmed the accuracy, reproducibility, and general time framework for the DNA replacement in tendon, ligament, and semilunar cartilage. The final experimental design was implemented using a total of thirty-six additional animals which underwent allograft transplant and subsequent DNA probe analysis. Subgroups utilizing patellar tendon (ten goats), anterior cruciate ligament (ten goats), and medial meniscus (sixteen goats) allografts were studied (Table 1).

Table 1
Experimental Study Design

# of Recipient Goats	Allograft Type	Evaluation Interval Post Transplant 1 Wks	2 Wks	4 Wks	6 Mths
10**	bone-patellar tendon-bone (fresh)		5 goats	5 goats	
10**	bone-ACL-bone (fresh)		5 goats	5 goats	
16*	meniscus & bony inserts (fresh)	8 goats			8 goats

*Evaluation at each time interval consist of: DNA analysis - 6 goats; histological evaluation - 2 goats

**All animals used for DNA analysis.

Surgical Procedure. After premedication with an intramuscular dose of xylazine hydrochloride, the hair of the goat's stifle joint was closely cropped. Intramuscular administration of ketamine hydrochloride was titrated to an appropriate level of general anesthesia. The knees of all goats in the study were examined preoperatively. Results were recorded with reference to instability, crepitance, range of motion, patellar tracking, varus and valgus stability, and McMurray testing. The lower extremity was prepped and draped in a sterile manner. An anterior midline incision was made and the patella was dislocated laterally. Visualization of the entire joint allowed the documentation and grading of any degenerative changes or other abnormalities.

Allograft Harvesting. Once the knee joint (stifle joint) was exposed, a six millimeter (mm) bone-patellar tendon-bone, bone-ACL-bone, or medial meniscus with bone plugs (Figure 1a, b, c) was procured. The allografts were harvested, washed, and held in buffered saline during the preparation of corresponding recipient animals. The allografts were maintained in room-temperature phosphate-buffered saline solution for thirty minutes to four hours, until transplantation. The viability of the cells from the

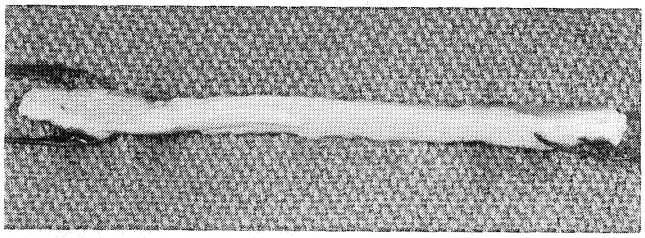

Figure 1a

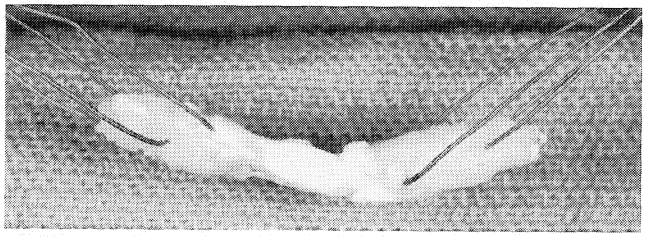

Figure 1b

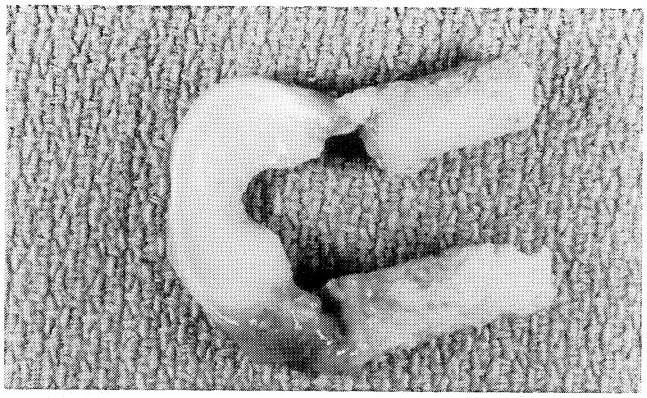

Figure 1c

Figure 1
Typical appearance of harvested allografts from a) bone-patellar tendon-bone, b) bone-anterior cruciate ligament-bone, c) medial meniscus with bone plugs incorporating horn attachments.

patellar and anterior cruciate ligaments, assessed by trypan-blue exclusion, ranged from 98 percent at thirty minutes to 95 percent at four hours. Corresponding structures were removed from the contralateral stifle joint for later comparison as the donor DNA.

ALLOGRAFT TRANSPLANTATION

Ligament Tendon Transplants. Each of the four grafts from the single donor were then implanted into one knee of four separate goats in which the anterior cruciate ligament had been excised. The sites of insertion of the anterior cruciate ligament were drilled to produce osseous tunnels for the allograft, as described previously. Number-2 Tevdek sutures (Deknatel, Fall River, Massachusetts), passed through drill-holes in the osseous ends of the graft, were used to pull and tension the grafts into snugly fitting femoral and tibial tunnels. The graft was tensioned at five pounds (2.3 kilograms) in 30 degrees of flexion, which

eliminated the increased anterior-posterior translation. The sutures were then tied over a screw-post and washer for fixation. The animals were not immobilized and were allowed to bear weight immediately postoperatively.

Meniscal Transplants. After the knee joint was visualized, the medial collateral ligament's femoral attachment was clearly identified. A trapezoidal block of bone incorporating the femoral medial collateral ligament attachment was osteotomized and mobilized distally for exposure. The bone block-medial collateral ligament complex was prepared so that screw fixation could later be used for re-establishing stability during closure. The medial meniscus was exposed and removed by sharp dissection from the capsular coronary ligaments and with its anterior and posterior horn bony attachments. Two osseous tunnels for the allograft bone plugs were prepared using a two mm diameter K-wire and a cannulated seven mm drill bit. The traction sutures were passed through the osseous tunnels and used to pull the bone plugs into their respective osseous tunnels. The sutures were tied across a bone bridge between the anterior and posterior tibial tunnels. The periphery of the meniscus was sutured to the capsule with interrupted 3/0 nylon sutures. The femoral attachment of the medial collateral ligament was then fixed using a single 4 x 22 mm cancellous half-threaded A-O screw. After knee joint irrigation the capsule and skin were closed in separate layers with interrupted sutures of #2 chromic. The wound was dressed with a spray-on moisture barrier dressing.

Skin Transplants. Recipients of tendon, ligament, and meniscal allografts also received a full thickness skin graft taken from the corresponding donor.

Evaluation Interval. The recipient animals were sacrificed at two and four weeks for the ligaments and tendons, and at one and six weeks for meniscal transplants (Table 1). The skin grafts were removed for histological sectioning.

DNA Procedure. The details of organic DNA extraction, DNA cleavage, molecular weight sizing, transfer from gel to nylon membrane, hybridization, and method of distinguishing banding patterns have been described[13]. This method isolates DNA from the tissue of the allograft with use of an organic extraction technique. The tissue was minced with a razor blade and was incubated overnight at 37 degrees Celsius in three milliliters of ten-millimolar Tris (ph 7.4), ten-millimolar ethylene-diaminetetraacetate, ten-millimolar sodium chloride, one percent sodium dodecyl sulphate, and 500 micrograms per milliliter of proteinase K. The solution was extracted one time each with an equal volume of phenol, phenol chloroform, and chloroform. The DNA solution was dialyzed overnight with three changes against ten-millimolar Tris (ph 8.0) and one-millimolar ethylene-diaminetetraacetate at four degrees Celsius. The yield of DNA from the samples was determined by comparison with that from control samples on an agarose gel.

The DNA was cleaved under the conditions specified by the supplier, with twenty units of restriction endonuclease HaeIII (BRL, Bethesda, Maryland) per microgram of DNA. The completeness of digestion was assayed by comparison with that of control samples on an agarose gel. Two micrograms of digested DNA from each goat were loaded into a one percent analytical agarose gel. Molecular weight sizing standards also were loaded, so that each set of samples was bracketed by these standards. This set of standard DNA fragments comprise forty-eight bands, ranging in size from 0.504 to 34.679 kilobase-pairs. The gel was run in forty-millimolar Tris (ph 7.8), and one-millimolar ethylene-diaminetetraacetate for a total of 1200 volt-hours.

The DNA was transferred from the gel to a nylon membrane (Biodyne B; Pall Biosupport, East Hills, New York) with use of an alkaline transfer technique. The gel was soaked in 0.4 sodium hydroxide and 1.0 molar sodium chloride for twenty minutes. The membrane was then placed on top of the gel, and a stack of paper towels was placed on top of the membrane to draw the solution through and into the paper towels.

The transfer was allowed to proceed for three hours, after which the membrane was removed and was soaked for twenty minutes in 2X SSC (300-millimolar sodium chloride and thirty-millimolar sodium citrate). The membrane was stored at room temperature until it was used for hybridization.

The membrane was hybridized with two oligonucleotide probes, derived from complementary human repeat sequences: OPT-02 (probe 1) and OPT-05 (probe 2) (Therion, Troy, New York)[14]. In a pilot study, these DNA probes distinguished among individual members of the goat species and were useful in assessment of relative amounts of two specimens in a mixture. The probes were labeled with ^{32}p (New England Nuclear, Boston, Massachusetts) by primer extension, and unincorporated nucleotide was removed on a NUC-Trap column (Stratagene, La Jolla, California). Hybridization was carried out as has been described previously[14].

Banding patterns of the DNA profiles were used to identify samples from the donor, recipient, and graft samples in each set. Characteristic DNA bands from the samples of donor tissue that did not appear in DNA profiles from the samples of recipient tissue were used to identify and compare the relative amounts of donor DNA in the samples taken from the grafts. Similarly, unique recipient bands from the host tissue were used to assess recipient DNA in the grafts. The relative position and density of the

DNA bands was determined with use of an image-processor (BiogImage, Ann Arbor, Michigan).

By comparison of the band-patterns in the graft with those in the pure donor and recipient, this technique allows specific identification and a semiquantitative analysis of transplanted specimens (determination of the percentage of DNA attributable to donor or recipient cell populations).

Statistical Analysis. Calculations of the sizes of the samples were made on the assumption that the graft contained 100 percent donor cells at the time of the operation and 0 percent at four weeks (tendon and ligament) or six weeks (meniscus) after implantation of the graft. The data were analyzed with use of the Wilcoxon signed-rank test. An alpha level of 0.05 or less was considered significant.

Table 2
Molecular Weight Sizing Standard

Band No.	Size in Kilobase Pairs	Band No.	Size in Kilobase Pairs
1	34.679	26	4.324
2	31.039	27	4.126
3	23.892	28	3.887
4	20.916	29	3.675
5	10.020	30	3.461
6	17.234	31	3.222
7	16.044	32	3.034
8	14.403	33	2.863
9	13.823	34	2.693
10	12.436	35	2.523
11	11.515	36	2.323
12	10.219	37	2.164
13	9.489	38	1.925
14	8.894	39	1.789
15	8.453	40	1.668
16	7.885	41	1.499
17	7.243	42	1.353
18	6.750	gap (no band)	
19	6.369	44	1.078
gap (no band)		45	0.974
21	5.687	46	0.872
22	5.386	47 0.763	
23	5.095	48	0.684
24	4.822	49	0.603
25	4.573	50	0.504

RESULTS

DNA Anlaysis. In pilot studies the two specific complimentary DNA probes, OPT-02 (probe 1) and OPT-05 (probe 2) (Therion Corp, Troy, New York), that best differentiated the DNA of individual goats were established using mixtures of minced ligament tissue. Each individual goat had a different and characteristic DNA pattern depending on whether probe 1 or 2 was used. The more related (inbred) the goats are, the greater the number of DNA bands they have in common. Common and unique DNA bands from the mixture of two tissues can be seen in Figure 2. Characteristic DNA bands from goat A, "pure A" (marked with closed arrows and an "A") are shown with probe 1 at or between band numbers 13, 17-18, 23-24, 29-30, and probe 2 at 13-14, 23-24, and 31 (see Table 2 for conversion of band numbers to kilobase pairs). Likewise, unique DNA bands from goat B, "pure B" (open arrows with a "B"), are shown with probe I between 18-19, 23-24, and probe 2 between 23-24. The density of the unique DNA bands in the mixture lanes

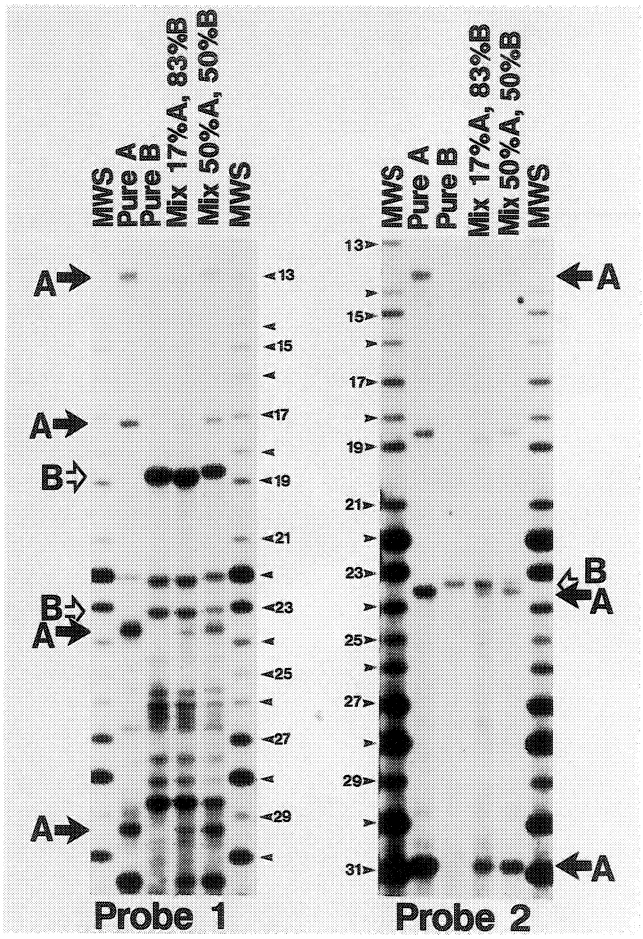

Figure 2
DNA band patterns obtained from mixtures of tissues from two different goats using DNA probes 1 and 2. Lanes marked Pure A and Pure B are samples from goats A and B. Lanes marked with Mix indicate percentage of A and B in sample. Closed arrows marked with A indicate position of unique Pure A DNA bands (goat A) that are not common with goat B and can also be identified in lanes with mixtures of A and B. Open arrows marked with B indicate position of Pure B bands that are not found in goat A and are identified in lanes with mixtures of A and B. MWS = lane containing molecular weight sizing standard. The small arrowhead markers and numbers indicate the relative band position and band numbers of the molecular weight sizing standard. The conversion of band numbers to molecular weight given in kilobase pairs is given in Table 2.

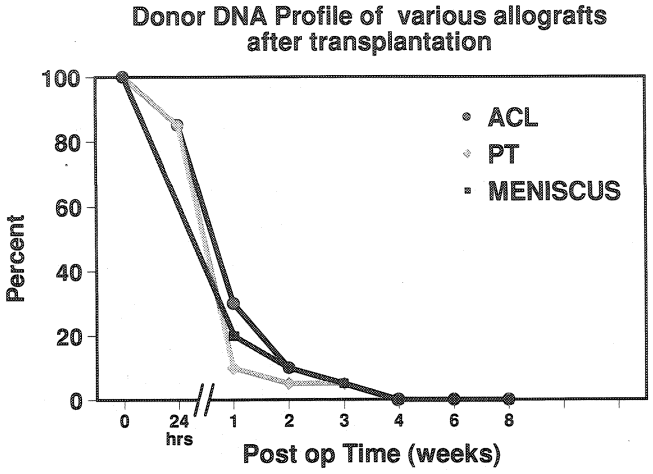

Figure 3
This graph illustrates the relative rates of donor cell DNA loss from patellar tendon, ACL, and meniscal allografts.

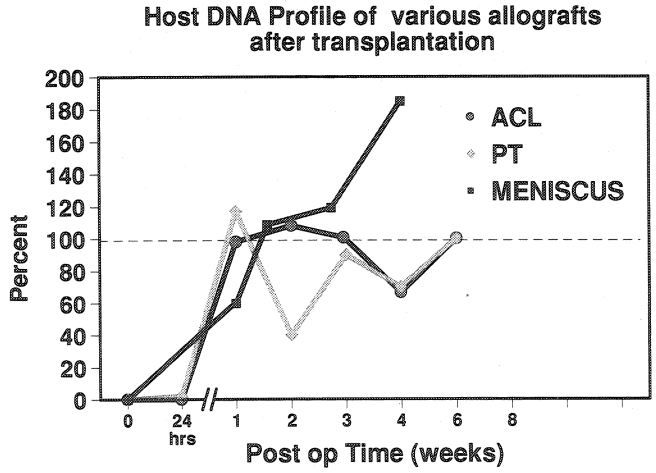

Figure 4
This graph illustrates the relative rates of repopulation with host DNA in patellar tendon, ACL, and meniscus allografts.

when compared to the corresponding bands in the "pure" A or B lanes reflect relative concentrations of the A and B tissue present.

A comparison of the loss of donor DNA in patellar tendon, ligament, and meniscal allografts is shown in Figure 3. A comparison of host DNA detected in tendon, ACL, and meniscal allografts is shown in Figure 4 and demonstrates that the allografts are repopulated with host cells relatively quickly. However, the DNA analysis does not discriminate cell types present in the tissue, i.e., fibrochondrocytes, chondrocytes, mesenchymal, monocytes, lymphocytes, etc.

Histology. A consistent observation throughout all of these studies has been the presence of "normal" appearing cells within one to two weeks in the allografts prior to revascularization. The fibroblasts appear somewhat plumper but oriented within the tendon and ACL (Figures 5a, b, 6a, b). The fibrochondrocytes are seen within the lacunae in the meniscus (Figure 7a, b). These cells tend to be larger and occasionally more than one cell occupies a lacuna.

The histologic appearance of the skin in all studies over the evaluation interval was that of a normal rejection. There was no indication of the presence of a rapid rejection reaction (humoral immune response) that would suggest the existence of a performed antibody to the donor tissue.

DISCUSSION

The fate of transplanted viable cells in tendon, ligament, and meniscal allografts prior to these studies had not been documented. The loss of donor DNA was consistent in the transplanted tendon, ligament, and menisci[12,13]. To date,

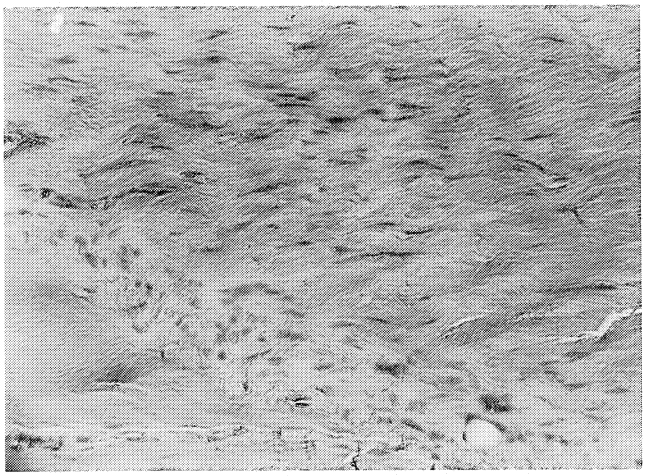

Figure 5a

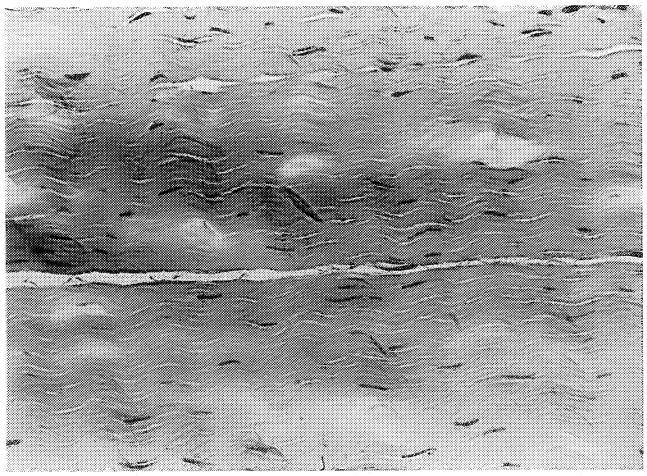

Figure 5b

Figure 5
a) Histologic appearance of patellar tendon allograft used for ACL reconstruction 4 weeks post transplantation. Note presence of fibroblasts which are of host origin. b) Normal patellar tendon. Hematoxylin and eosin, original magnification x 52.5.

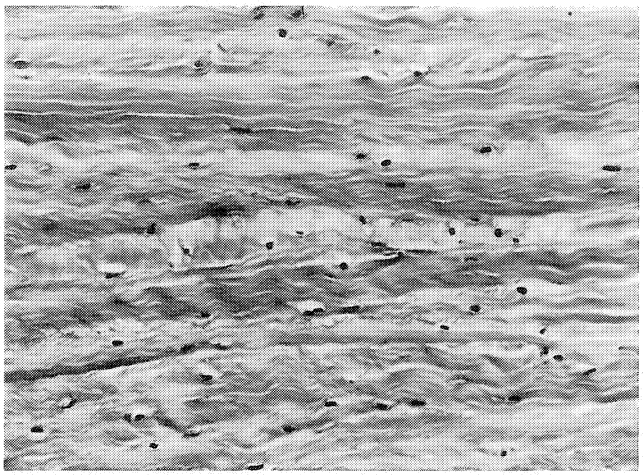

Figure 6a

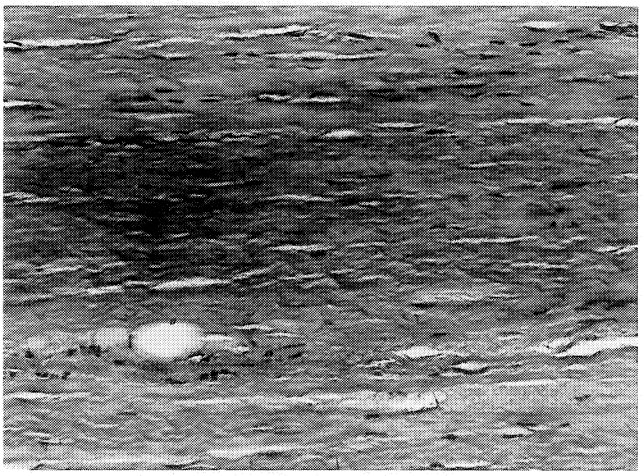

Figure 6b

Figure 6
a) Histologic appearance of ACL allograft used to reconstruct the ACL 4 weeks post transplantation. Normal appearing fibroblasts of host origin are present. b) Contralateral control ACL. Hematoxylin and eosin, original magnification x 62.5.

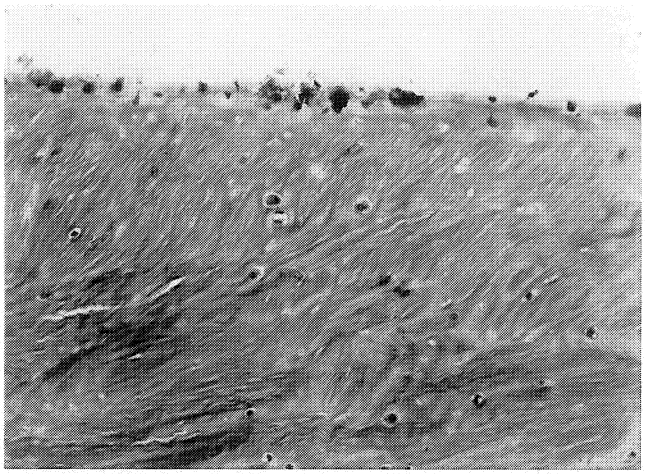

Figure 7a

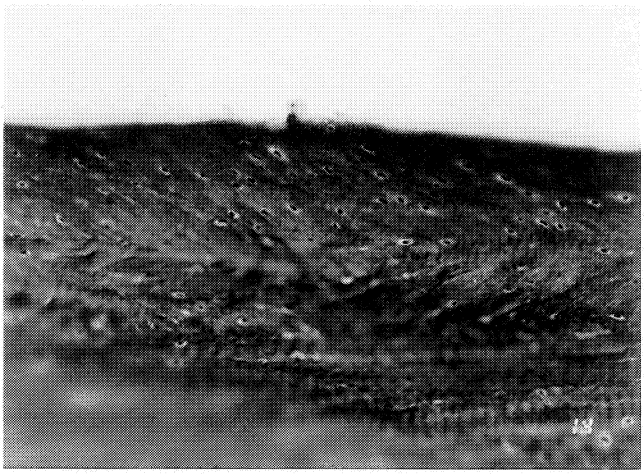

Figure 7b

Figure 7
a) Histologic appearance of meniscal allograft 4 weeks after transplantation surgery. Fibrochondrocytes appear in substance of allograft and are of host origin. b) Contralateral control meniscus. hematoxylin and eosin, original magnification x 62.5.

there is no evidence that viable cells in these types of allografts will survive in the intra-articular environment of the recipient's knee.

The high sensitivity and specificity[14,16] of the hybridized human probes used were applicable in our goat model because of the shared common structure among species that is characteristic of all DNA. This technique requires the amount of DNA present in approximately three thousand cells. From our extraction studies in the Spanish goat, it is estimated that there are approximately 40 million cells in a normal ACL, 35-40 million cells in a six mm wide patellar tendon allograft used for an ACL reconstruction (intra-articular portion), and 2.8-3.0 million cells in a normal medial meniscus.

Documentation that these allografts became rapidly repopulated with host cells that start migrating into their substance within a few days explained some of the confusion in the existing literature. The histologic presence of living cells has been misinterpreted as being surviving and living cells of donor origin. These cells that migrate into the allografts differentiate into cells resembling fibroblasts, fibrochondrocytes and chondrocytes. They appear in the body of the ligament, tendon, and meniscus prior to revascularization. In addition, there was a peripheral vascular response and the presence of inflammatory cells associated with the ligament, tendon, and meniscal allografts[2,10,11,12]. The presence of these various cellular components in and around the surface of transplanted allografts is consistent with our quantitative findings of increased replacement of recipient DNA. The probes do not distinguish these inflammatory cells from the cells that repopulate and function within the graft.

Our findings do not refute that the meniscus may be immunologically privileged. The rapid loss of donor DNA in the ligament, tendon, and meniscal allografts and the absence of simultaneous skin graft rejection suggests there were no preformed antibodies and that the cells in the allografts die prior to a traditional immunological reaction. The exact mechanism resulting in this precipitous loss of DNA needs to be defined. This may result from a specific cell necrosis mechanism and/or a failure of the transplanted allograft cells to survive the trauma of transplantation into a new avascular intraarticular environment. If viable and functioning fibroblasts and fibrochondrocytes in the transplant remains a goal, new methods to maintain the viability of the donor cells are necessary. Transplantation of allografts with viable cells increases the expense, surgical logistics, and potentially the risk of disease transmission[4,5,6]. The results of this study demonstrate that transplantation of viable cells in the ligament, tendon, and meniscal allografts do not survive in the recipient goat.

Another approach to obtain the desired cell function in the transplanted allograft is to alter the phenotypic expression of the multipotential host cells that migrate into the graft. The enhancement of their differentiation into a mature population of fibroblasts or fibrochondrocytes that function as the native cell population is a challenge. Advances in molecular biology may offer approaches to alter or stimulate the cells repopulating these allografts. Allografts are a limited resource; if host cells can be stimulated and manipulated, then a synthesized, molded, or reconstituted biologic ligament, tendon, or meniscus substrate becomes more of a possibility. A ligament, tendon, or meniscal cartilage scaffold or prosthesis has the potential of improving availability, minimizing sizing problems, reducing the risks of disease transmission, and reducing potential immunologic considerations.

CLINICAL RELEVANCE

The long term survival of allograft transplants of ligaments, tendons, and menisci is dependent on the cells that maintain their unique structural and material properties. Using a DNA probe technique that clearly distinguishes donor cells from host cells, the authors have demonstrated that intra-articular transplantation of viable allograft cells in ligament, tendon, and fibrocartilage do not survive in the Spanish goat model. These donor cells are removed and replaced by host cells in a rapid manner. The host cells that repopulate the allografts assume the histologic appearance of the fibroblasts and fibrochondrocytes they replace. The clinical basis for utilizing intra-articular allografts with living cells needs further justification to account for their increased expense, complicated surgical logistics, and potential risk of disease transmission.

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TOTAL QUADRICEPS SPARING, ENDOSCOPIC SINGLE-INCISION ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION USING FRESH FROZEN ALLOGRAFT TISSUE: SURGICAL TECHNIQUE AND POTENTIAL PITFALLS

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INTRODUCTION

Reconstruction is now the procedure of choice in symptomatic patients who are physically active following rupture of the anterior cruciate ligament (ACL). Previous reported treatment of ACL tears include: primary repair, repair with extra-articular augmentation, isolated extra-articular augmentation, intra-articular reconstruction through mini-arthrotomy, and arthroscopic-assisted intra-articular reconstruction. Graft material has varied and has included the iliotibial band, hamstring tendons, patellar ligament, allograft tissue, xenograft tissue, and prosthetic devices. The advent of arthroscopically-assisted reconstruction techniques has lessened tissue damage and enhanced early rehabilitation. In addition, the current trend towards early aggressive physical therapy, consisting of immediate motion, immediate weight bearing, and an early return to functional activities, has shortened the previously prolonged rehabilitation process[14]. Based on these considerations, ACL reconstructive surgery performed arthroscopically has become more attractive to both the surgeon and patient.

Fresh frozen allografts have been used at the senior author's institution over the last six years. Our results compare favorably with published results using autograft tissue[12]. Advantages of allograft tissue include less donor site morbidity, improved cosmesis, shortened operative time, and possibly a decreased incidence of stiff knee following reconstruction[4]. Potential disadvantages of using bone-patellar tendon-bone autograft tissue include harvest morbidity, donor site pain, quadriceps weakness, patellar tendon weakness/rupture, and patellar fracture. In selected cases of multiple ligament surgery and revision ligament surgery, allograft tissue is preferred over autograft. Our technique of using fresh frozen allograft tissue allows ACL reconstruction through two standard arthroscopic portals, and a single 3 centimeter (cm) tibial incision. This avoids a lateral incision altogether, thus completely sparing the quadriceps mechanism.

Our endoscopic technique has the advantages of arthroscopic-assisted ACL reconstruction and offers several advantages over mini-arthrotomy techniques. These include improved visualization to allow for more accurate graft placement, improved cosmesis, decreased post-operative pain and hospital stay, and simpler wound care[13]. We believe this technique may allow for future surgery on an out-patient basis.

While this technique has many advantages, we have found it to be a technically demanding procedure with a slow learning curve[9]. The purpose of this article is to describe the senior author's surgical technique and potential pitfalls of endoscopic ACL reconstruction using fresh frozen allograft bone-patellar tendon-bone.

Patient Positioning and Preparation

After satisfactory general or regional anesthesia, an examination under anesthesia is performed. Examination of both knees for physiologic anterior laxity and anterolateral instability must be included. Associated secondary instabilities must be diagnosed and managed appropriately; failure to do so will result in persistent instability and surgical failure. The uninvolved leg is flexed and abducted at the hip and flexed at the knee while positioned in a well-padded leg holder. A tourniquet is placed high on the involved extremity, which is placed in an arthroscopic leg holder. The bottom portion of the table is then flexed to allow a minimum of 120° of knee flexion.

Diagnostic Arthroscopy

Diagnostic arthroscopy is performed to confirm rupture of the ACL, while noting any associated abnormalities. The medial inflow portal is made with the knee in full extension while retracting the vastus medialis proximally to permit easy insertion of the 5 millimeter (mm) inflow catheter. Failure to retract the vastus medialis with resultant puncture can lead to persistent post-operative swelling and prolonged rehabilitation. A single anterolateral portal 1-2 mm lateral to the patellar tendon is made for arthroscope insertion. A second anteromedial portal is made 5-8 mm medial to the patellar tendon and slightly inferior (5-10 mm) to the lateral portal. This is important

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for later interference screw insertion. Meniscal disease is first addressed with either repair or excision as appropriate. If meniscal repair is performed, suture tying is delayed until after ACL fixation.

Notch Preparation

Pre-operative radiographs should be assessed for notch architecture, stenosis, and intercondylar osteophytes[5,15]. Remnant ACL tissue is removed with a full radius resector. A small footprint of the native ACL at the insertion site is left to guide in the placement of the tibial bone tunnel as well as provide a vascularized cuff of soft tissue for the graft. This also acts as a one-way valve to decrease water leakage after creation of the tibial tunnel. Inadequate soft tissue debridement can lead to difficult graft passage and the development of a "cyclops" lesion[7]. On the femoral side, complete removal of soft tissue is needed to visualize the over-the-top region and allow confirmation with an arthroscopic probe.

The notchplasty is performed with two goals in mind: to prevent graft impingement superiorly and laterally, and to insure adequate visualization and exposure of the posterior margin of the notch. A notchplasty is undertaken with an arthroscopic osteotome, full radius resector, and burr. Minimal bone is removed from the anatomic attachment site near the over-the-top region to avoid graft malpositioning. Fine-tuning of the notchplasty can be performed after the femoral drill hole has been made.

Tibial Bone Tunnel

Placement of the tibial bone tunnel is extremely important when performing the endoscopic technique. A poorly placed tibial tunnel may block endoscopic drill access to the correct femoral tunnel starting point[11]. This may lead to graft impingement and/or excessive graft length changes with resultant failure[6,16]. A tibial drill guide is inserted through the anteromedial portal and seated in the posteromedial aspect of the ACL "footprint". The ideal exterior starting point on the tibia is about halfway between the apex of the tibial tubercle and the posteromedial border of the tibia. Too lateral a starting point causes lateral femoral condylar impingement, a tendency for the guide wire to skive off the medial wall of the lateral femoral condyle, and a femoral tunnel which exits too proximally. Too medial a starting point causes impingement against the medial femoral condyle and posterior cruciate ligament (PCL), and difficulty reaching posteriorly within the intercondylar notch for the femoral starting point. A three cm longitudinal incision is made at the exterior starting point. The incision is carried down to the periosteum and periosteal flaps are created for later closure. A tibial guide wire is drilled through the tibial jig under direct arthroscopic visualization. It must exit in the posteromedial "footprint" of the ACL so that it is five to eight mm posterior to the anterior intercondylar outlet with the knee in full extension. The tibial guide pin is then over-drilled with a ten mm cannulated reamer while protecting the posterior cruciate ligament with a small curette placed in the anteromedial portal. This tunnel can easily be expanded to match larger grafts if required. The tibial tunnel is then chamfered and smoothed with an arthroscopic rasp to prevent graft abrasion.

Femoral Tunnel

The goal of femoral tunnel placement is to avoid placing the tunnel too far anteriorly, yet avoid posterior cortical breakout. This can be accomplished easily by thoroughly removing the remnant of the ACL and clearly visualizing the posterior cortical margin. Do not proceed with femoral tunnel placement until an adequate notchplasty allows complete visualization of the posterior notch. Through the tibial tunnel, a free K-wire is inserted either free-hand or using the Arthrex "over-the-top" guide (Portsmouth, New Hamsphire). The K-wire is then impacted or drilled into place at approximately the two or ten o'clock position (depending on right or left knee) and within six to seven mm of the posterior cortical margin. A minimum of 90 degrees of knee flexion must be kept to prevent fracture of the femoral tunnel out the posterior femoral cortex. Guide pin placement should be reassessed with an arthroscopic probe after partial insertion to verify approximately five to seven mm of posterior bone. If a non-anatomic tibial tunnel has been made, the surgeon will experience technical difficulty in placement of this guide pin. It is then better to abandon the endoscopic technique and proceed with a lateral incision and an over-the-top position. Once the guide pin is in place, avoid flexion/extension of the knee so that the guide pin does not bend. Bending can lead to eccentric drilling and guide wire breakage.

A cannulated arthroscopic drill the identical size of the femoral bone plug (ten mm) is inserted over the guide wire. The lateral femoral condyle is drilled up to, but not through, the posterior cortex. These drills are labeled at five mm increments, which allows direct visualization of the length of the femoral tunnel (Figure 1). The average femoral tunnel measures thirty to forty mm. During drilling, a motorized shaver is inserted through the anteromedial portal to improve visualization by removing debris using suction and shaving. Insufficient knee flexion (less than sixty degrees) during femoral tunnel drilling increases the risk of a posterior femoral "blow-out" fracture and superior lateral geniculate artery injury[11]. If a posterior "blow-out" fracture occurs or anterior tunnel placement has occurred, a lateral incision can be made and an over-the-top position can be used with an AO-screw and washer as fixation. Chamfering the tunnel surface with a rasp is done to prevent graft wear or impingement. A

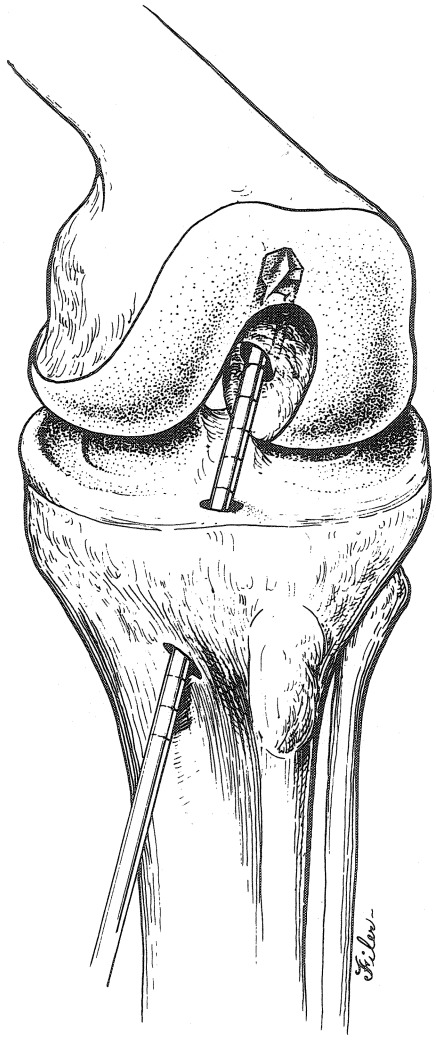

Figure 1
Endoscopic drill creating femoral bone tunnel through tibial bone tunnel.

small indentation at the superior lateral outlet of the tunnel is made with an arthroscopic curette to allow a starting point for the cannulated interference screw[8]. The posterior cortex of the lateral femoral condyle tunnel is then perforated with a long 4.5 mm AO drill passed through the tibial tunnel. Perforation should be done in the superior lateral quadrant to facilitate needle placement and pick-up.

Impingement Test

An impingement test is now performed to verify that there is no superior or lateral graft impingement. It is much easier to enlarge the notchplasty before the graft is inserted and secured. A ten mm (same size as graft) bone tunnel expander (Instrument Makar) is placed into the notch through the tibial tunnel (Figure 2). The knee is then placed through a complete range of motion while observing under arthroscopic visualization for any impingement upon the device. An arthroscopic burr may be used to fine-tune

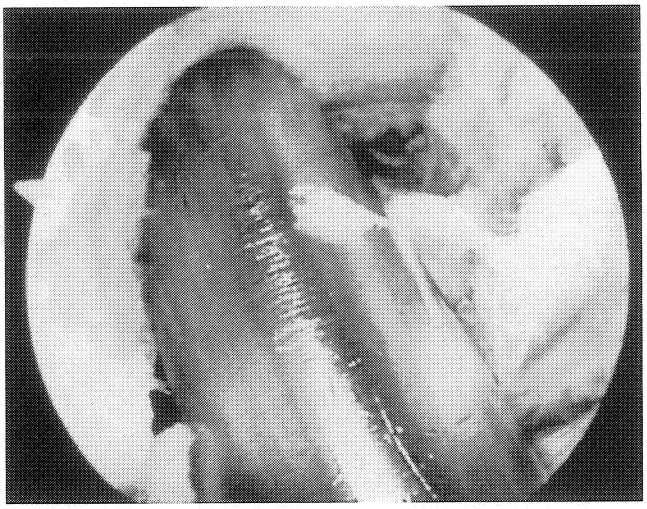

Figure 2
Bone Tunnel Expander (Instrument Makar) within notch to check for impingement, "Impingement test".

the notchplasty, if required. This will ensure that once the graft is inserted and secured, there will be no need for further notchplasty, thereby avoiding iatrogenic graft injury.

Graft Preparation

Fresh frozen bone-patellar tendon-bone allografts that have been harvested under sterile conditions in accordance with the American Association of Tissue Banks (AATB) standards[1] are used. The graft is thawed at the beginning of the case at temperatures below forty degrees to prevent de-naturing the collagen. The graft can be prepared at any time during the case; if operating independently the surgeon may prefer to prepare the graft prior to tourniquet inflation. The central portion of the graft is harvested. Bone plugs are made to fit through the appropriate size drill sleeves. The femoral bone plug is cut to the approximately measured length. A single large retention suture (#5 Ticron) is placed through the tibial bone block (which will be inserted into the femur), and two retention sutures are passed at ten and twenty mm

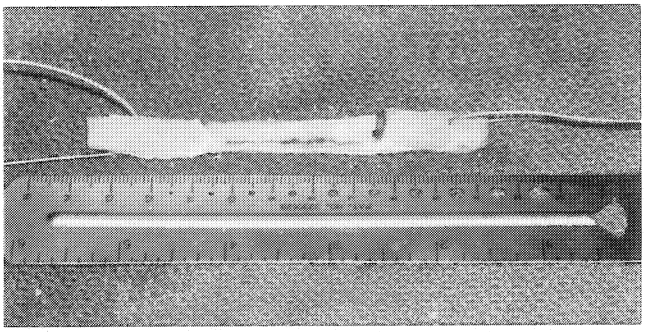

Figure 3
Bone-patellar tendon-bone allograft.

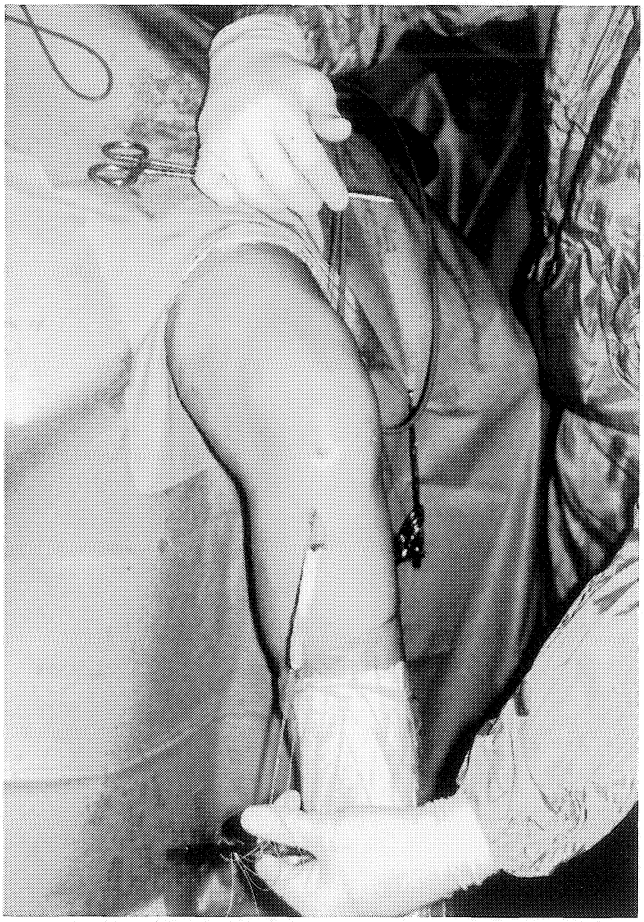

Figure 4
Graft passage technique via endoscopic method.

intervals from the distal end of the patellar bone block at ninety degrees to each other (inserted into the tibia). The tendon-bone interface is demarcated with a sterile marking pen on the cancellous side for accurate assessment of complete graft seating to the level of the bone-tendon tunnel junction (Figure 3). Finally the graft is marked at one cm intervals from the femoral bone plug to aid in isometric positioning after femoral fixation.

Graft Passage and Femoral Fixation

A Beath needle is passed through the tibial tunnel, the femoral tunnel, and out through the skin of the lateral thigh. Failure to keep the knee in the amount of flexion in which the femoral tunnel was created can result in the needle exiting proximal to the leg holder or tourniquet. The graft is attached to the opposite end of the needle and is passed under direct arthroscopic visualization through the tibial tunnel, in the notch, and into the femoral tunnel (Figure 4). A grasper may be used to ensure placement of the ligamentous portion of the femoral bone block posteriorly. Therefore at the time of interference screw placement, abrasion or amputation of the tendonous portion of

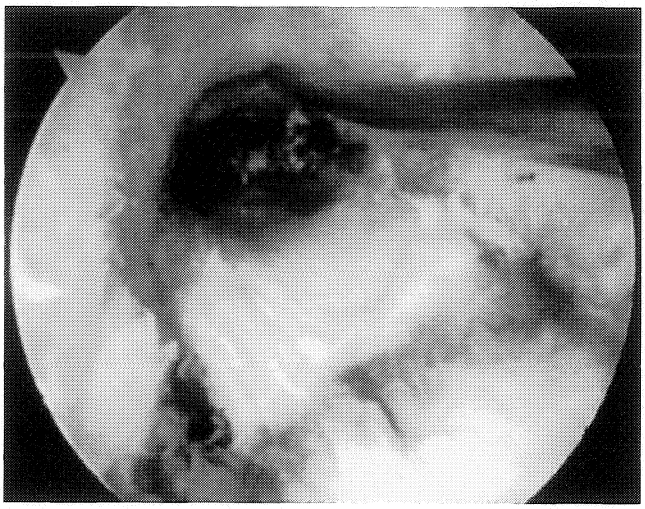

Figure 5
Guide wire placement prior to femoral interference screw fixation.

the graft is less likely to occur. When the bone plug is seated within the tunnel, the graft is palpated to verify that the mark at the tendon-bone junction is at the level of the tunnel outlet. Fixation of the femoral bone plug is achieved with a seven by twenty mm cannulated interference screw. Frequently the medial arthroscopic portal needs to be extended inferiorly to allow for easier passage of the screw. With the knee hyperflexed (120°), a flexible guide pin is inserted through the anteromedial portal and passed anterolateral to the bone plug (Figure 5). The guide wire should be parallel to the bone plug and should not slip through the hole made for the retention suture and get trapped during screw insertion. The screw is then passed over the guide wire and seated. Care is taken to enter the bone-to-bone interval parallel to the bone graft with the tendinous portion safely posterior to the screw. Failure to appropriately hyperflex the knee may result in screw divergence and possible posterior cortical screw breakout. If the screw is inserted too far and leaves one of the suture holes and bone plug exposed, a possible fracture can occur through this screw hole. This can be avoided if the screw is simply approximated to the graft throughout its length. Once secured, the knee should be multiply cycled through a range of motion with tension being placed on the tibial plug sutures to confirm rigid fixation.

Isometry Check

Isometry is checked by applying tension to the tibial bone block retention sutures and observing movement of the graft as the knee is taken through a full range of motion. The markings which were made on the allograft facilitate direct arthroscopic visualization of graft motion relative to the articular surface of the tibial tunnel (Figure 6). We commonly see one to two mm of elongation (tightening) in terminal degrees of extension. This closely

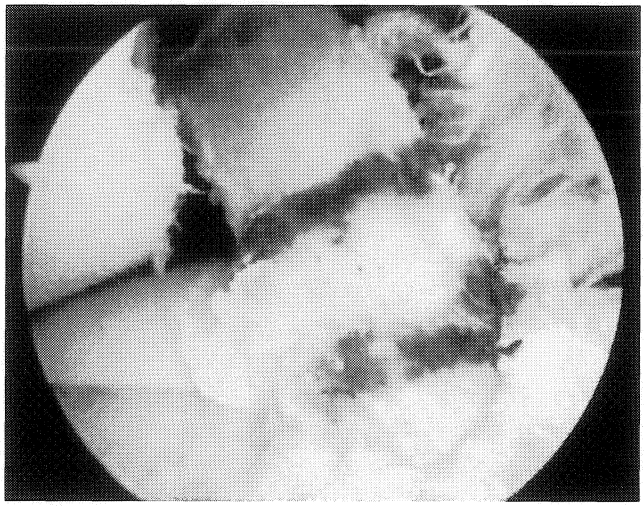

Figure 6
Allograft in place with markings to check "isometric" position prior to tibial fixation.

mimics the normal behavior of the ACL[3]. Fine-tuning of graft isometry can be done by repositioning the orientation of the collagen fibers in the tibial tunnel prior to fixation. However, we do not feel that adjustments in the tibial tunnel should be performed for a malpositioned femoral tunnel.

Tibial Fixation

Tibial plug fixation is performed with the knee in fifteen degrees of flexion while applying graft tension via the retention sutures. Two sutures were previously placed in the tibial bone plug to prevent loss of graft tension if inadvertent suture amputation occurs during interference screw insertion. If the gap between the bone surfaces is less than three mm, a seven mm interference screw is used. If greater than a three mm gap exists, a nine mm screw is inserted. Occasionally, the graft length is excessive and requires the creation of a tibial metaphyseal bone trough with either an osteotome or a gouge, and fixation with staples.

The screw is placed anteromedially on the cortical edge of the tibial bone plug (Figure 7). This is done to promote greater biologic incorporation (increased surface area of bone-to-bone contact) and prevent the screw from diverging with resultant anteriorization of the graft. Care is taken to avoid advancement of the screw tip beyond the bone-tendon junction and possibly abrading the tendon[10]. A final arthroscopic exam is performed as the knee is placed through a full range of motion to check for graft dislodgment and possible impingement. The periosteum over the tibial tunnel is carefully closed for a water-tight seal. The wounds are irrigated and closed in the routine fashion.

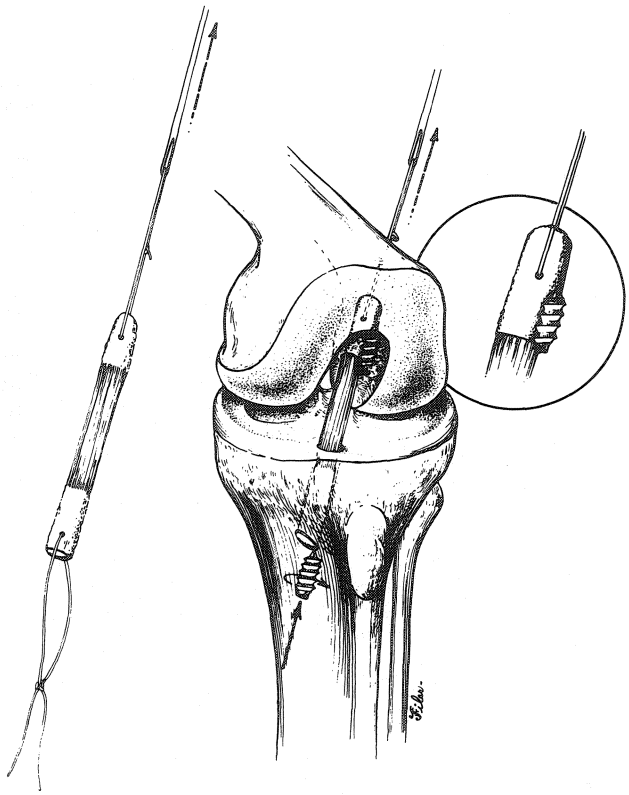

Figure 7
The femoral bone block is fixed with an interference screw. After the graft has been appropriately tensioned, a second interference screw fixes the tibial bone block.

Dressing/Rehabilitation

A sterile dressing is applied as well as a toe-to-groin elastic stocking. A Cryocuff compression sleeve (Aircast, Inc., Summit, New Jersey) is applied over the stocking, and a simple post-op brace locked in full extension is used. An aggressive rehabilitation program is begun immediately which emphasizes control of the inflammation, early restoration of full extension, early range of motion, quadriceps exercises, and restoration of normal gait[2].

DISCUSSION

The advantages of the endoscopic method using fresh frozen allograft tissue are numerous. First, the method greatly minimizes tourniquet, operating, and anesthetic time. Average operative time for an isolated ACL endoscopic reconstruction is approximately sixty minutes. Secondly, this procedure requires a single three cm anterior incision, and no lateral incision. In addition, there appears to be less tissue morbidity, a more cosmetically acceptable scar, less post-operative pain, less effusion, and earlier quadriceps muscle control and strength allowing for accelerated rehabilitation. However, this method is technically

demanding and fraught with potential complications. Attention to detail is critical when performing this procedure. Precise tibial tunnel starting point placement, knowledge of potential pitfalls of endoscopic femoral tunnel drilling, avoidance of graft impingement, and optimization of graft fixation enhance the likelihood of consistently reconstructing the ACL safely and successfully. If one is to consider this technique, he must be prepared to deal with the myriad of pitfalls outlined in this article. The surgeon should critically assess his or her experience and technical skill before attempting this technique.

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ARTHROSCOPIC VERSUS OPEN DEBRIDEMENT OF PENETRATING KNEE JOINT INJURIES

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Richard A. Marder, M.D.

ABSTRACT

Arthroscopic debridement of penetrating knee joint injuries has become a common treatment method. A comparative study was undertaken to compare this method with open joint debridement. Fourteen penetrating knee joint injuries (fourteen patients) were treated by arthroscopic examination and debridement and were compared to sixteen penetrating knee joint injuries (fifteen patients) treated by open debridement. There were no resultant infections or operative complications in either group. Of note, the arthroscopic debridement group had a shorter postoperative hospital stay [mean of 1.6 days compared to a mean of 2.6 days in the open debridement group ($p < 0.02$)], a significant incidence of additional intra-articular injuries detected ($p < 0.01$), less postoperative pain, and a superior cosmetic result. We conclude that arthroscopic debridement of penetrating knee joint injuries is a safe and effective method of treatment, providing additional diagnostic information while minimizing morbidity and reducing hospital stay.

INTRODUCTION

Penetrating knee joint injuries are the most common open joint injuries seen in a busy trauma center today[5]. The joint contamination from these injuries can result in septic arthritis and lead to joint destruction unless adequate and prompt surgical debridement is performed[2]. Additional damage to articular cartilage, menisci, and ligaments may result from direct or indirect trauma at the time of knee joint penetration.

Traditional treatment has consisted of open surgical debridement and primary or delayed closure. With the development of arthroscopic surgery, many surgeons have adapted this method of treatment for selected open joint injuries. A study was undertaken to compare these two methods of treatment.

MATERIALS AND METHODS

During a two year period, thirty consecutive penetrating knee joint injuries in twenty-nine patients were identified and treated. Injuries with associated fractures requiring internal fixation or cast immobilization were not included in this study. The group treated with arthroscopic debridement consisted of seven motor vehicle "dashboard" injuries, five motorcycle/moped/bicycle injuries and two low velocity gun shot wounds. The mean wound size in this group was 3.8 centimeters (cms) (range one to twelve cms) and the mean patient age was 26.6 (range nine to fifty-twelve years). The open debridement group consisted of eight motor vehicle "dashboard" injuries, five motorcycle/moped injuries, one low velocity gunshot wound, one weed-trimmer laceration, and one power-saw laceration. The mean wound size in this group was 4.6 cms (range one to twelve cms) and the mean patient age was 26.9 years (range fifteen to forty-three years).

Diagnosis was based upon examination of the wound, AP, lateral, and notch radiographic views of the knee, and a saline-methylene blue arthrogram performed in the emergency room. The finding of a visible or palpable laceration extending into the joint, air in the joint on radiographs, or fluid extravasation during saline-methylene blue arthrogram was considered as confirmation of knee joint penetration. All patients received irrigation of the soft tissue wound in the emergency room, application of a sterile betadine soaked dressing, intravenous cefazolin, and tetanus prophylaxis. Surgical debridement was subsequently performed on an urgent basis within eight hours, with the treatment method determined by surgeon preference.

Surgical Procedure

Arthroscopic examination and debridement was performed using standard superomedial inflow and anterolateral viewing portals. An additional anteromedial portal was utilized as necessary for probing and debridement. Open debridement was performed by extending the traumatic wound into a limited medial or lateral parapatellar arthrotomy (depending upon location of traumatic wound). Both arthroscopic and open treatment groups underwent examination of knee stability under anesthesia and irrigation of the joint with six liters of sterile saline. In both groups, a 1/4 inch suction drain was inserted into the joint and brought out through a separate stab incision. Traumatic skin wounds were left open. Postoperatively, all patients received intravenous cefazolin every eight hours until the drain was removed at twenty-four to forty-eight hours (dependent upon drain output). Local wound care was

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begun at the time of drain removal and full weight bearing with early active motion was instituted.

RESULTS

A retrospective review of all study patient's medical records was performed. No acute or subacute infection requiring additional treatment occurred in either treatment group and there were no operative complications. At surgical debridement, eleven out of fourteen (79%) arthroscopically debrided knees had additional intra-articular abnormalities detected; whereas in the group treated by open debridement, only two out of sixteen (13%) knees were noted to have additional intra-articular abnormalities (Table 1). This observed difference between groups was statistically significant ($p < 0.01$) by independent t-test.

The mean postoperative inpatient stay for the arthroscopic group was 1.6 days (range one to two days), compared to 2.6 days (range one to four days) for the open group ($p < 0.02$ by independent t-test). At the initial postoperative visit five knees treated by arthroscopic debridement had full range of motion as compared to none in the open debridement group. This difference, however, was not statistically significant.

DISCUSSION

Although penetrating wounds to the knee joint are the most common open joint injury, sparse literature exists concerning management of these injuries. Those reports which are available advocate conflicting treatment methods.

Based upon his World War II experience, Hampton[2] recommended early aggressive surgical treatment of open joint wounds, but stated that simple penetrating wounds with little or no articular damage should be treated nonoperatively. For those which are debrided surgically, he urged thorough immobilization as an essential deterrent to infection.

Patzakis et al,[5] reported a prospective study in which 129 open knee joint injuries were treated with intravenous antibiotics, open arthrotomy, and closed suction irrigation. In this series, a high proportion of the cases resulted from gunshot wounds or co-existed with open fractures extending into the knee joint. With their closed suction irrigation system, a fourteen percent wound contamination rate was noted. The authors concluded that arthrotomy followed by primary closure of the joint without a drain was the treatment of choice.

Gunshot wounds to the knee, although a minority of the patients in our study (ten percent), represent a special case. A bullet which traverses the joint may contaminate the wound directly or from clothing fragments and skin flora. Autosterilization of low velocity bullets has not been shown to occur[8]. Other problems occur when retained intra-articular lead fragments become solublized[3], leading to chronic synovitis, hypertrophic arthritis, or even systemic lead poisoning[6]. Meticulous debridement is therefore mandatory with gunshot wounds to the knee, and may be carried out by either open arthrotomy[1] or arthroscopically[4,7].

SUMMARY AND CONCLUSIONS

Penetrating knee joint injuries require early debridement to prevent the development of joint infection and its sequelae. In our series, surgical debridement (open or arthroscopic) with joint closure over a suction drain in conjunction with twenty-four to forty-eight hours of intravenous antibiotics resulted in no joint infections. With arthroscopic debridement the hospital stay was reduced, a higher incidence of associated intra-articular abnormalities was detected, postoperative pain was reduced, and cosmesis was improved.

Arthroscopic debridement of penetrating joint injuries is therefore a safe and effective treatment method of treatment. It allows for a more complete examination of the knee with the potential to detect and treat associated injuries which may not be evident with open debridement.

Table 1
ADDITIONAL INTRA-ARTICULAR ABNORMALITIES DETECTED

	Arthroscopic	Open
Articular Injury	5	2
Chondromalacia	2	0
Partial Anterior Cruciate Ligament tear	2	0
Meniscal tear	2	0
	11	2

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LIMB SALVAGE TUMOR SURGERY IN CHILDREN

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ABSTRACT

Limb salvage tumor surgery in children is quite popular. Low local recurrence rates and patient survival statistics prove it is a viable option for many patients. We review the major changes in medicine and paramedical fields that have allowed limb salvage to develop. We then discuss patient selection, limb salvage goals, review the surgical staging system, and examine common errors in evaluating a tumor patient. A discussion (with case examples) of various surgical options and a brief look at some current trends is then presented.

INTRODUCTION

We define limb salvage tumor surgery as local resection of a tumor with reconstruction that preserves a functional distal limb. Preservation of satisfactory function necessitates intact neural and vascular structures.

Fifteen years ago amputation was the usual treatment of choice for malignant musculoskeletal tumors in the extremity[21]. In the past ten to fifteen years pioneering development in four separate fields have allowed limb salvage to be a viable option for many of these tumor patients.

Advances in radiological imaging allow accurate margin definition as well as identification of skip and metastatic lesions. This imaging includes the use of computer tomography (CT), magnetic resonance imaging (MRI), and bone scintigraphy (bone scan). With accurate staging and margin definition, much of the guess work of deciding who qualifies for limb salvage is eliminated.

Bioengineering has made great strides in the development of biomaterials as well as computer aided design (CAD) custom prostheses. A custom made prosthesis can now be available at the time of surgery rather than waiting days or weeks post-resection for the implant. Better designs and materials improve the quality of fit and longevity of the implant.

Advances in chemotherapy have contributed greatly to limb salvage. New medications and more effective combinations provide better control of both local and metastatic lesions. A good example is the change in 5-year survival rates for patients with Ewing's sarcoma. The treatment of Ewing's sarcoma has evolved from the traditional chemotherapy and radiation therapy. Complications of radiation include growth arrest, radiation osteitis, pathologic fractures, and post-radiation sarcoma. The favored protocol is now pre-operative chemotherapy (to shrink the tumor mass) followed by surgery for local control and post-surgical chemotherapy[13]. Five year survival has improved from less than 5% to greater than 50% with this treatment regimen[8]. Advantages of preoperative chemotherapy include reduction of edema, destruction of occult microextension and occult metastases, and assessment of drug effectiveness by examining the surgical specimen. In addition, the chemotherapy can be modified if the surgical specimen reveals a poor response.

Finally, improvements in our own field of orthopaedics have contributed to the development of limb salvage. Microvascular surgical techniques and a surgeon's imagination allow free tissue transfers (fibula, iliac crest) as spacers for bone defects and provide soft tissue coverage (lastissimus dorsi) for large soft tissue defects. Bone banks provide allografts for reconstruction. Additionally, materials and techniques for internal fixation and external fixation are allowing greater options in limb salvage. The revolution of bone transport via the Ilizarov and similar techniques is currently being adapted to the tumor patient. The applications of this new technique are not yet fully realized, but they may change our concepts of limb salvage.

Patient Selection

Despite criticism by some regarding local recurrence rate and disfigurement, limb salvage does not necessarily have an adverse effect on survival[1,10,21]. Realizing this, one must select the appropriate patients. A standard approach should be used and the following three questions should be answered: What is the tumor? Where is the tumor? What stage is the tumor? Knowing the type of tumor is the key to understanding its natural history. Aggressive tumors are more likely to invade extracompartmental structures and metastasize. A tumor's location is both a clue to its histology and the possibility of limb salvage. Finally, the staging is critical in deciding on limb salvage. Multiple metastatic lesions constitute a contraindication to limb salvage. With this information the next three questions must be addressed: What margins are needed? What structures must be sacrificed? Can the vital neural and vascular structures be preserved? The surgical

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margin necessary for cure is related to the aggressiveness of the tumor. With the assistance of the radiologic images a surgical map can be drawn with the appropriate surgical margins. If vital structures can be spared a limb salvage procedure becomes an option.

Without a careful, methodical approach to each tumor, judgment errors are likely to occur with suboptimal results. If the patient is a limb salvage candidate, realistic goals must be agreed upon by the physician, patient, and the patient's parents.

Goals

Basic goals of the limb salvage must be established for both the upper and lower extremity (Table 1). For instance, an insensate stiff hand with good sensation does not allow adequate function and therefore undermines the purpose of limb salvage. If a major nerve must be sacrificed, a nerve graft and/or tendon transfers should be included in the pre-operative plan. The lower extremity goals all contribute to an energy efficient gait pattern and must be appreciated. Limb lengths can frequently be addressed with lifts but joint stability may be more difficult to control. For example, quadriceps preservation helps with gait but is not essential for limb viability or walking. With these goals in mind one must also consider the individual patient's needs.

Table 1
Limb Salvage Goals

Upper Extremity
- Preservation of Sensation
- Preservation of Dexterity

Lower Extremity
- Limb Length
- Stability of Major Joints
- Preservation of Active Knee Control

Pediatric patients differ from adults in several ways when considering limb salvage. The growing skeleton and the irresponsible nature of children make limb salvage a challenge. Parents are often available to help with early mobilization, but a durable reconstruction is needed to allow for long term use and recreational abuse. Adults, however, are more concerned with maintaining their independence and preserving their occupational skills. As the child enters the teen years and beyond, cosmesis will play a significant role in selecting the type of surgery.

Staging and Margins

We will briefly review the surgical staging system adopted by the Musculoskeletal Tumor Society in 1980[7,9]. The staging system is based on three separate factors. The histologic grade (G) is first. Low grade tumors are rated G-1 and high grade tumors are G-2. There are charts available which divide most of the musculoskeletal tumors into histologic grades[8,9]. Surgical site (T) is the second factor. T-1 is an intracompartmental lesion and T-2 is an extracompartmental lesion. Compartmental charts are also published and available[8,9]. Metastatic disease (M) is the final factor. M-0 presents no metastasis and M-1 means nodal or distant metastatic disease is present. Combining these three factors gives us the staging system in Table 2.

Table 2
Staging of Musculoskeletal Malignancies

Stage	G	T	M
I - A	Low (1)	Intra (1)	M-0 (None)
I - B	Low (1)	Extra (2)	M-0 (None)
II - A	High (2)	Intra (1)	M-0 (None)
II - B	High (2)	Extra (2)	M-0 (None)
III - A	Low/High (1/2)	Intra (1)	M-1
III - B	Low/High (1/2)	Extra (2)	M-1

When performing a local resection or amputation for an extremity tumor, there are four different types of surgical margins one can achieve[21]. To fully appreciate the significance of these margins one must remember a malignant lesion involves the tumor itself, its surrounding pseudocapsule, and a reactive zone with occult tumor microextensions. An intralesional/intracapsular (biopsy, curettage) resection is through the lesion and leaves macroscopic disease. A marginal resection is through the pseudocapsule/reactive zone and leaves behind occult microextensions and satellite lesions. A wide resection includes a surrounding cuff of normal tissue several centimeters wide. This resection may still miss skip lesions. The fourth type is a radical resection which involves resection of the entire compartment(s) involved with tumor. Most limb salvage surgery involves wide or marginal (around vital structures) resection.

Pitfalls

The patient who presents with an extremity lesion requires a diagnosis and staging prior to definitive treatment. A thorough history and physical exam, good quality imaging studies including angiography when indicated, and a proper biopsy with cultures are required.

The senior author's experience reveals the following common errors in the evaluation of tumor patients: 1) Relying on poor quality imaging studies for diagnosis. This is especially seen in spine tumors where CT, MRI, or tomograms may be required. 2) Failure to recognize when a fracture is pathologic. A detailed history and careful scrutiny of any old radiographs as well as the current films will help prevent this problem. 3) Ignoring the natural history of a lesion. Over treating a benign latent lesion or

under treating a highly malignant lesion may result in an unacceptable outcome. 4) Poor biopsy technique resulting in extensive local contamination. 5) Being unprepared for hemorrhage from a "simple biopsy". Most malignant tumors are vascular and can bleed extensively. 6) Confusing fracture repair, myositis ossificans, and osteogenic sarcoma. A careful history must be taken and the radiologist and pathologist must be involved early. 7) Failure to culture an "obvious tumor" or biopsy a "definite infection". Infection is in the differential for nearly every radiographic lesion so both a biopsy and culture should be done whenever the other is planned.

Surgical Options

If the decision for limb salvage has been made based on the diagnosis, staging, patient's desires and reasonable functional goals, a surgical reconstruction plan must be established. The plan must be flexible to prevent surgical margin compromise in order to make reconstruction easrier. Resection with inadequate margins will increase the local recurrence rate and affect long term survival. There are numerous options for reconstruction (Table 3).

One option is to simply resect all or a portion of the involved bone. Some bones can be sacrificed with minimal to moderate functional loss. Many complications of reconstruction (infection, fractures, non-unions, prosthesis loosening) can be avoided. Examples include resection of the clavicle (Figure 1), the fibula (Figure 2), an internal hemipelvectomy (Figure 3), the Tikhoff-Limberg resection of the shoulder, and removal of the radius or ulna (one bone forearm).

The second option is to fill in the defect with a "spacer". The spacer may be temporary or permanent, and can be metal, plastic, ceramic, or bone. The bony spacer may be autograft, allograft, or autoclaved tumor bone. Finally, the autograft may be vascularized or non-vascularized. This obviously gives a tremendous number of potential reconstruction options and constructs.

The simplest option is to fill the defect with autogenous bone graft (Figure 4). With large intercalary defects this is often not possible. A sliding autograft, allograft, vascularized autograft (Figure 5), or a combination of these may be used. Capanna et al., have recently reported using a vascularized fibula with an allograft in their reconstructions. They feel this provides better size and strength than just a fibula and better healing than with an isolated allograft[3,4].

When resection involves a joint surface the options change. Joint fusion with autograft or allograft will provide a stable limb but some functional compromise. More popular is the use of a prosthesis. There are two basic types. The conventional prosthesis is the typical adult-type joint replacement, frequently custom made to fit the surgical defect (Figure 6). The second is an expanding prosthesis designed to "grow" with the child. Either can be combined with an allograft (Figure 7) to help replace lost bone.

A relatively new area being explored is the use of distraction osteogenesis and bone transport to fill in defects. This potentially eliminates the need for allografts, vascularized autografts, and internal prostheses. However, it is not a panacea, and not without its own set of risks and complications.

A final option for the distal femur, proximal tibia, proximal femur, and iliac tumors[6] is a rotationplasty. Originally used for infected knee joints, this procedure was popularized for use in patients with proximal femoral focal deficiency. It has been adapted to tumor patients with good success (Figure 8). The major drawback is cosmesis. The advantage over an above knee amputation is the retention of a functional knee joint. With a modern prosthesis these patients function very well[14].

Table 3
RECONSTRUCTION OPTIONS

- **I. Resection of Bone**
  - eg: hemipelvis, fibula, clavicle, ulna, radius
- **II. Resection with Spacer**
  - A. Autograft
    1. Massive Corticocancellous Graft
    2. Vascularized Bone Graft
    3. Non-vascularized Bone Graft
    4. Sliding Autograft
    5. Autoclaved Bone
  - B. Allograft
    1. Diaphyseal (intercalary segment)
    2. Hemi-joint
    3. Entire joint
       a. With arthrodesis
       b. Without arthrodesis
  - C. Internal Prosthesis—Conventional or Expanding
    1. Diaphysis
    2. Joint
    3. Entire bone
  - D. Combinations
    1. Vascularized autograft and allograft
    2. Internal prosthesis and allograft
    3. Internal prosthesis and autograft
- **III. Other**
  - A. Van Nes Rotationplasty
  - B. Modified Rotationplasty
  - C. Ilizarov Bone Transport

Recent Ideas and Trends

When the Sixth International Symposium on Limb Salvage was held in Montreal (1991) several trends and themes were highlighted.

The use of allografts is often associated with a high complication rate[2,5,12,16,19,24]. Delayed healing (up to eighteen months with chemotherapy), fracture, and infection

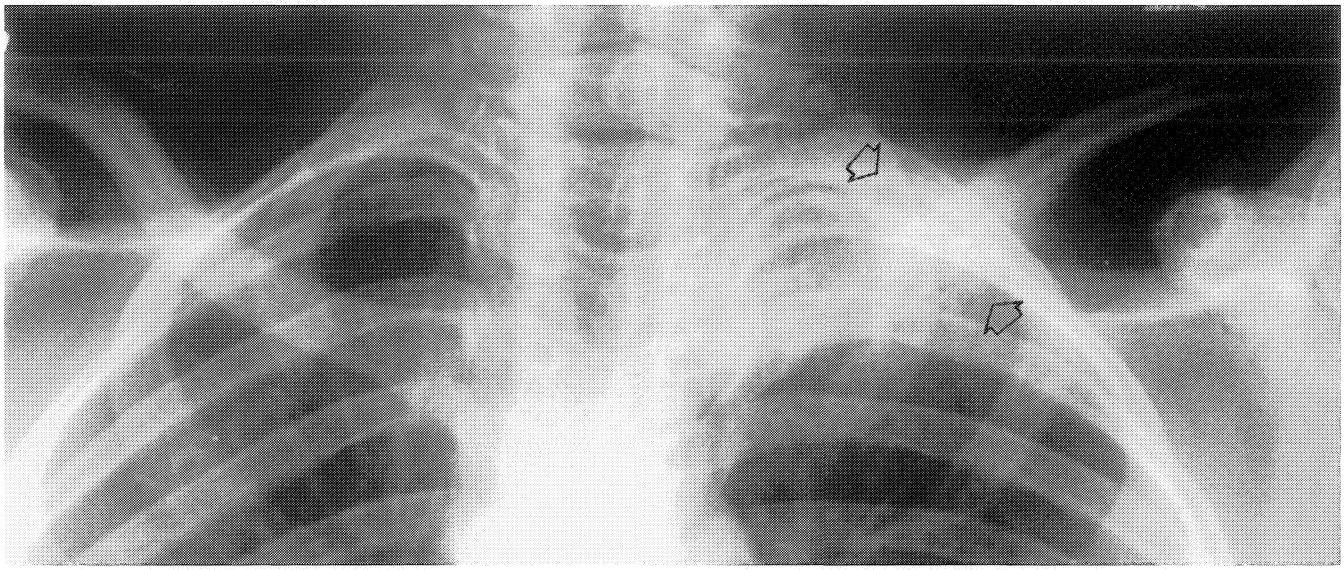

Figure 1A
AP radiograph of a fourteen year old male with a postirradiation osteogenic sarcoma of the left medial clavicle. Radiation therapy had been prescribed for lymphoma at six years of age.

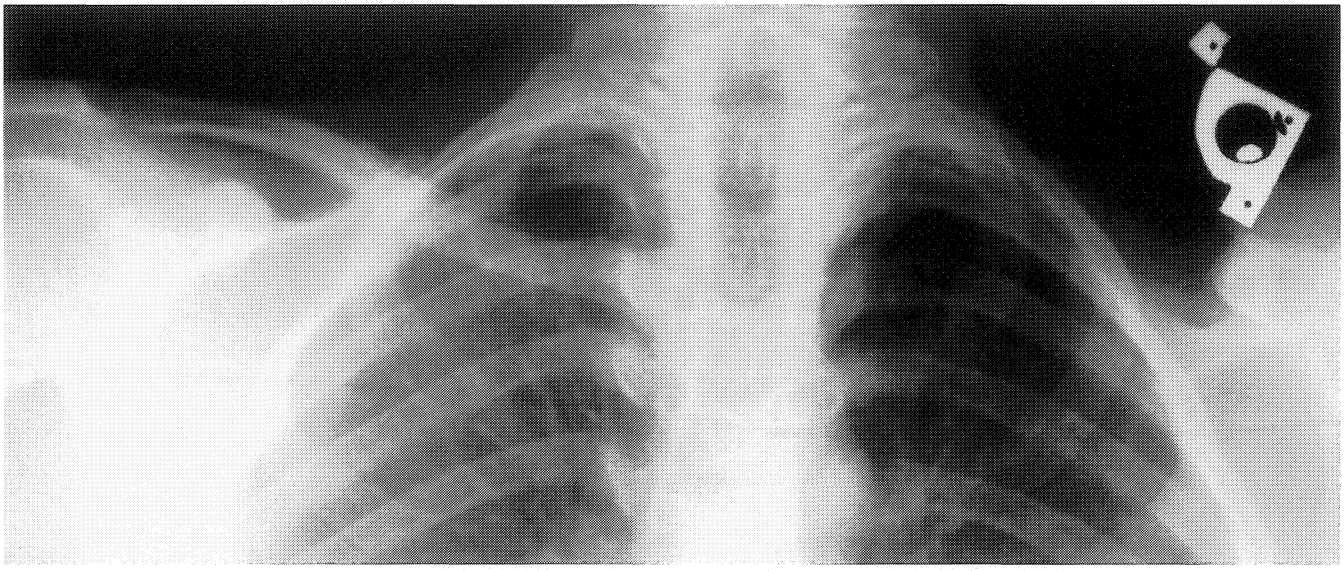

Figure 1B
AP radiograph after resection.

can involve up to 50% of allografts. (Muscular coverage of allografts helps decrease infection rate.) Although complications with allograft use are frequent, proximal femoral replacements combining an allograft with a prosthesis have better strength and stability when compared with a megaprosthesis[15,18,23,25] Loosening is less frequent with proximal versus distal femoral replacements[22].

Allograft arthrodesis appears to be a viable option in both the shoulder and knee. In proximal humerus resections this is a better option than joint reconstruction, especially if the abductor mechanism is sacrificed[17,20]. In knee joint resections allograft arthrodesis may have a better long term outcome than an internal prosthesis with or without allograft[1,2]. When using allograft, it is desirable to cement the allograft to the prosthesis but not the prosthesis to host bone. This will decrease loosening between prosthesis and host secondary to cement fragmentation and particulate debris formation.

The use of a prosthesis in a skeletally immature patient combines the problems of standard prosthesis use (particulate debris and subsequent loosening) with increasing shear forces (secondary to muscle imbalance from surgical

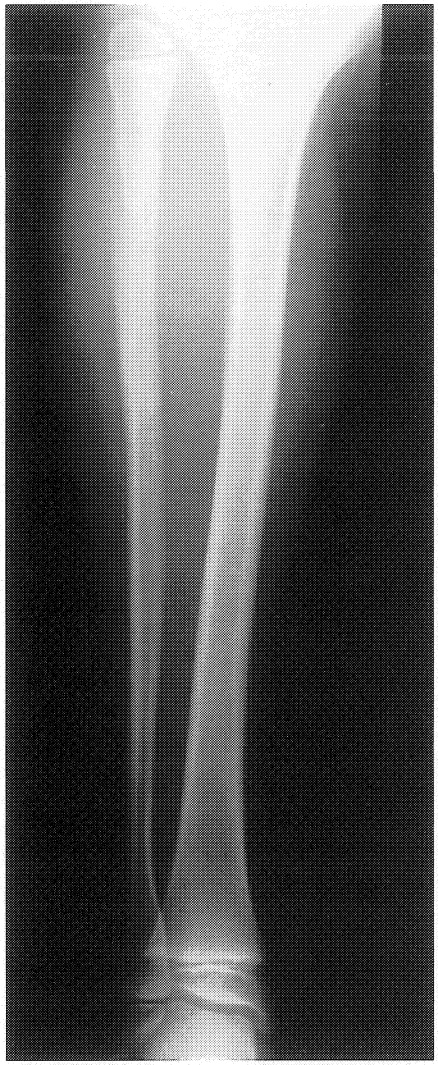

Figure 2A

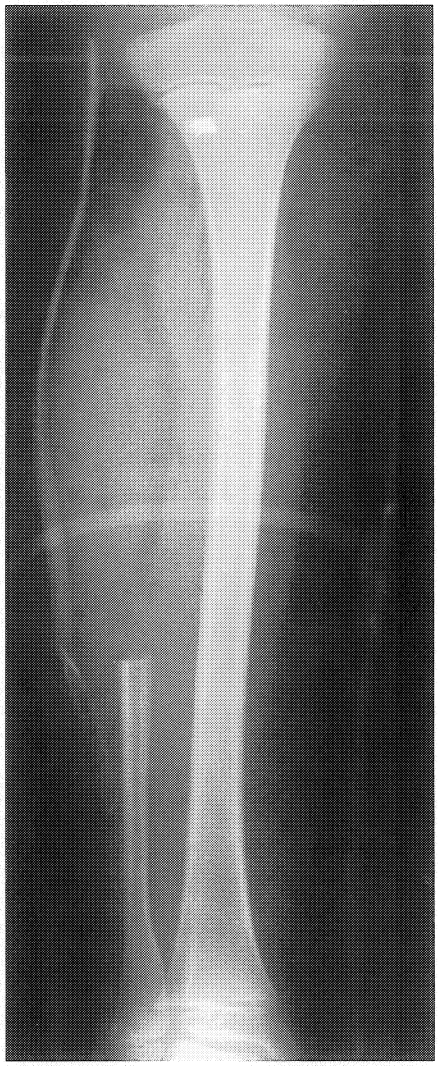

Figure 2B

Figure 2
Fibula resection.

Figure 2A
AP radiograph of the right leg of a nine year old male with Ewing's sarcoma involving the proximal fibula.

Figure 2B
Immediate post-operative AP radiograph showing resection of the affected fibula.

Figure 3 (below)
Hemipelvectomy.

Figure 3A
MRI of the pelvis of a sixteen year old male with Ewing's sarcoma. Note large intrapelvic mass with involvement of the pubic ramus.

Figure 3B
Immediate post-operative AP radiograph showing complete right internal hemipelvectomy.

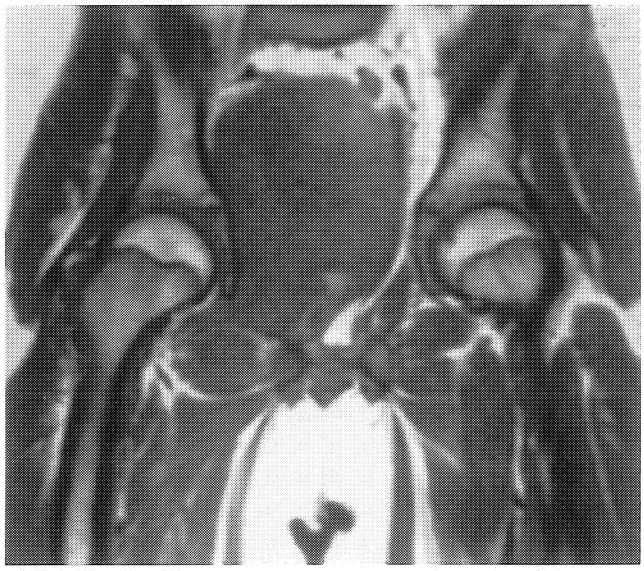

Figure 3A

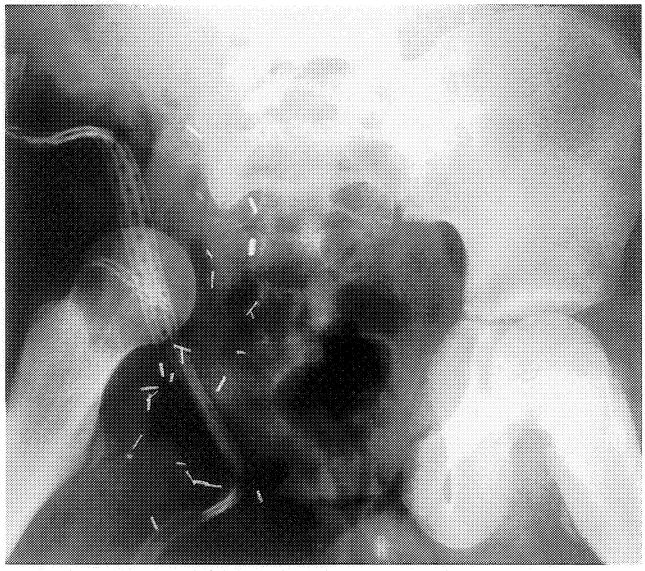

Figure 3B

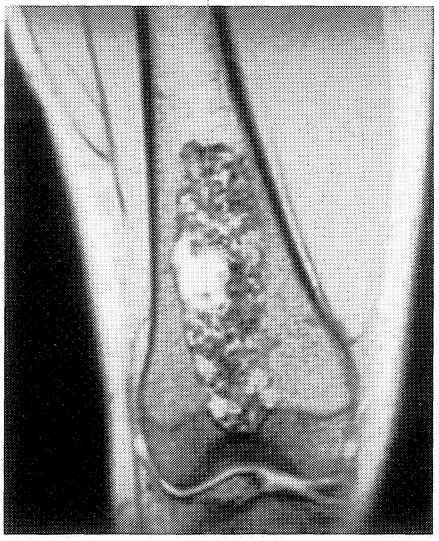

Figure 4A

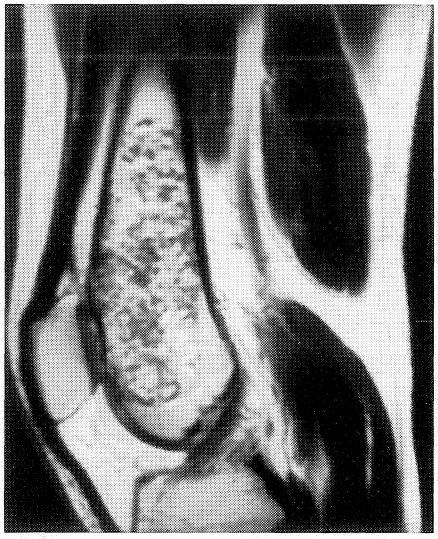

Figure 4B

Figure 4
Corticocancellous Bone Growth.

Figure 4A and B: Coronal and sagittal plane MRI of distal femoral lesion in a seventeen year old female semi-professional dancer. Diagnosis was low grade chondrosarcoma.

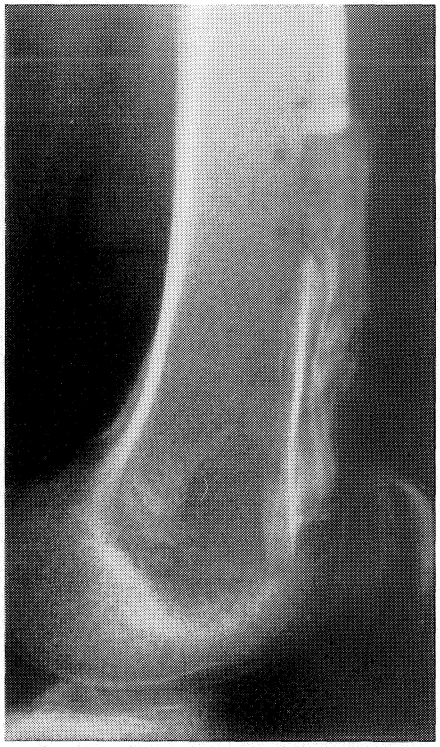

Figure 4C: Lateral radiograph after resection and bone grafting, holes in cortex were used to anchor dexon mesh that secured the graft. Patient had refused prosthesis or fusion as primary procedure.

resection and subsequent gait abnormalities) and the increased growth of the patient. New prosthesis designs, better cement technique, and bony ingrowth prostheses are possible solutions to these problems[11].

CONCLUSION

Limb salvage surgery is an accepted standard of care for many extremity tumors. This change from amputation to reconstruction has resulted in an explosion of new surgical procedures as well as new applications of old methods and materials. We have outlined the decision making process for selecting limb salvage surgery as well as reviewed common pitfalls in tumor patient evaluation. We outlined the common surgical options, presenting examples of some of these. Finally, we briefly looked at some recent trends in limb salvage as reported at The International Symposium on Limb Salvage in Montreal.

ACKNOWLEDGMENT

We wish to thank Dr. Jeffrey Kneisel for contributing case examples presented in Figures 6 and 7. The assistance of Maureen McCloskey in preparing this manuscript is gratefully acknowledged.

Figure 5
Vascularized Fibula.

Figure 5A: Lateral radiograph of a sixteen year old male with a large chondrosarcoma of the proximal femur.

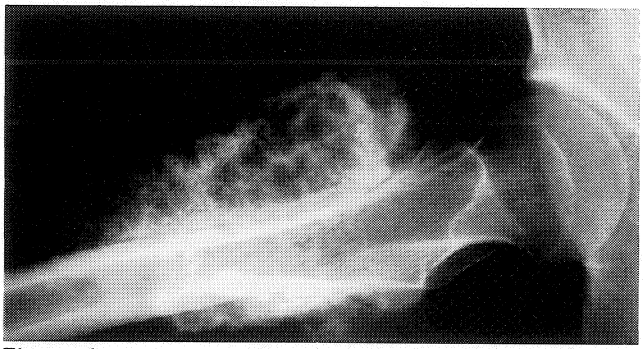

Figure 5A

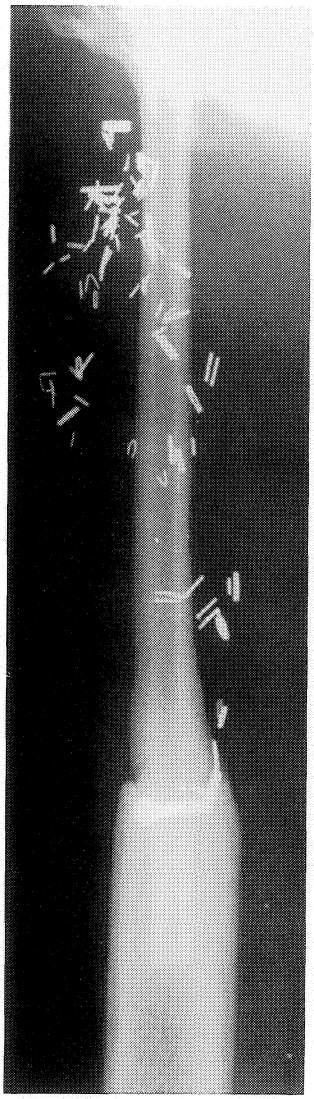

Figure 5B: AP radiograph of a vascularized fibular spacer six months after resection.

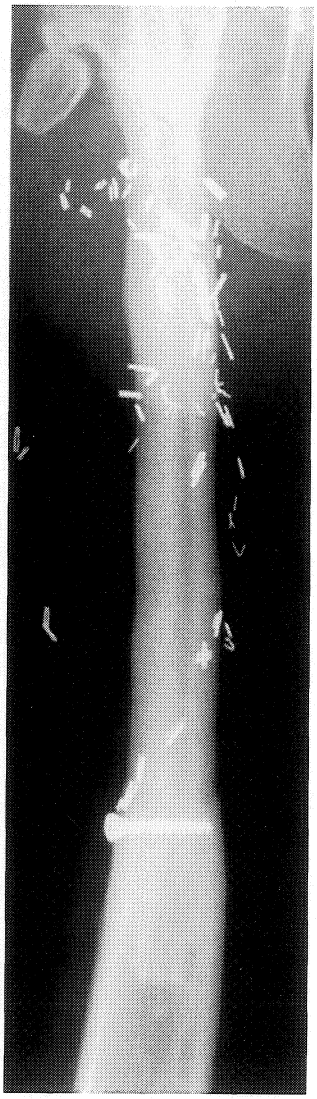

Figure 5C: AP radiograph at seventeen months post-resection and reconstruction. Note hypertrophy of fibula.

Figure 6
Custom Prosthesis.

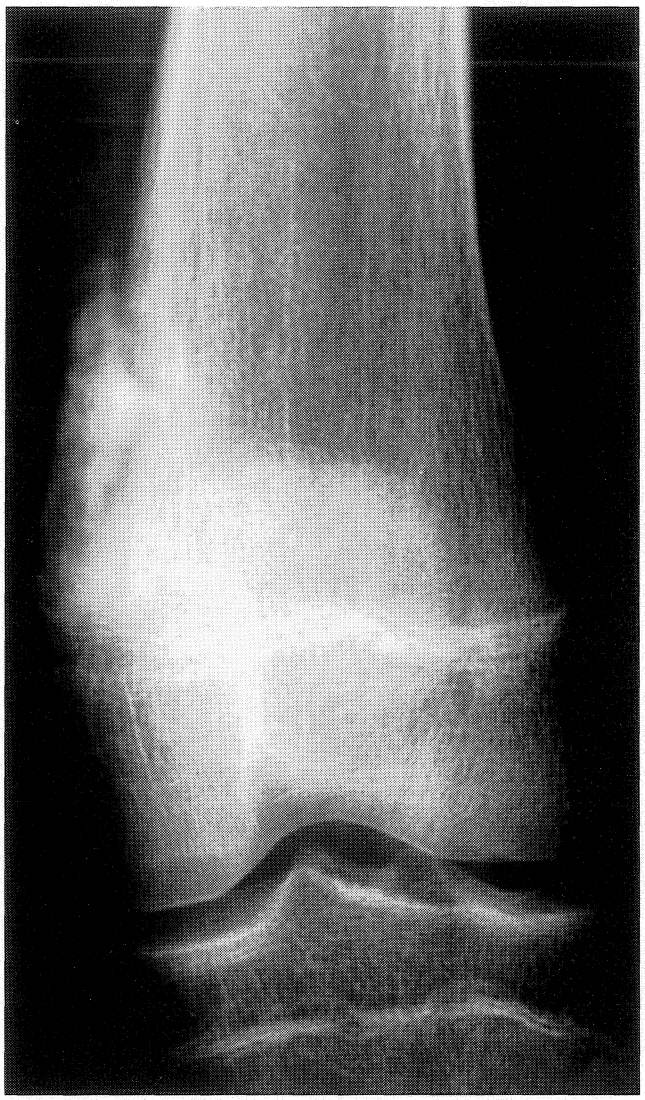

Figure 6A
AP radiograph of a twelve year old female with osteogenic sarcoma of the distal femur.

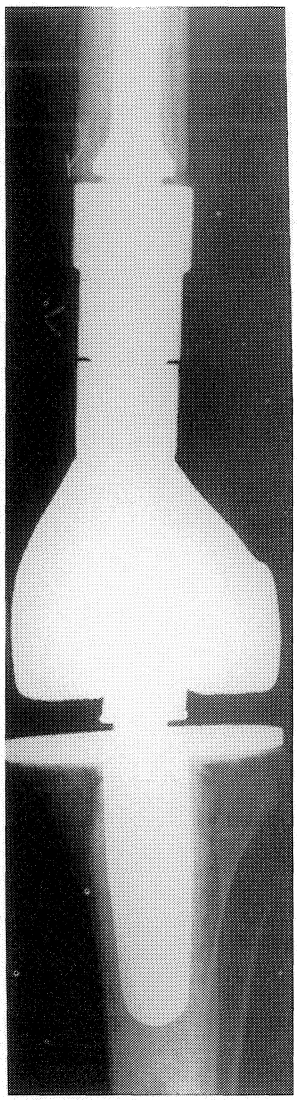

Figure 6B
AP radiograph of the patient after reconstruction with prosthesis.

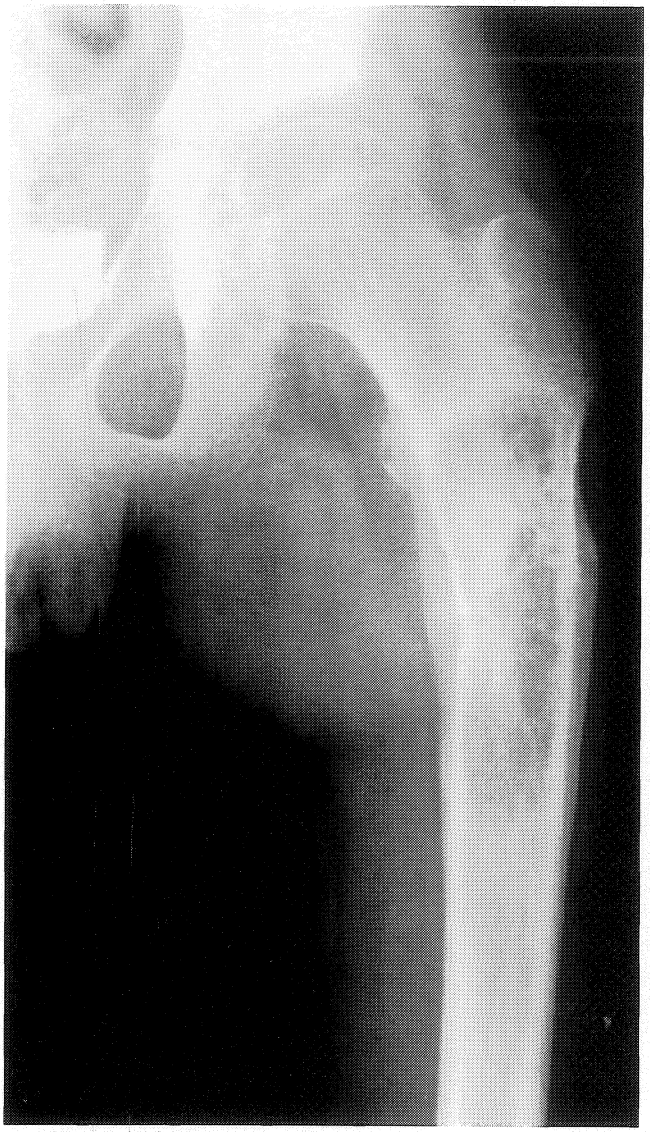

Figure 7A
AP radiograph of a thirteen year old male with Ewing's sarcoma of the proximal femur.

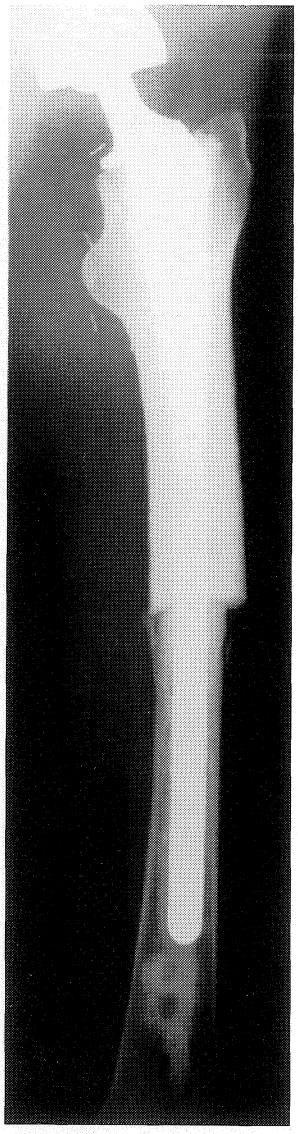

Figure 7B
AP radiograph after reconstruction with a long hip prosthesis and allograft bone.

Figure 7
Prosthesis with Allograft.

Figure 8
Van Nes Rotationoplasty.

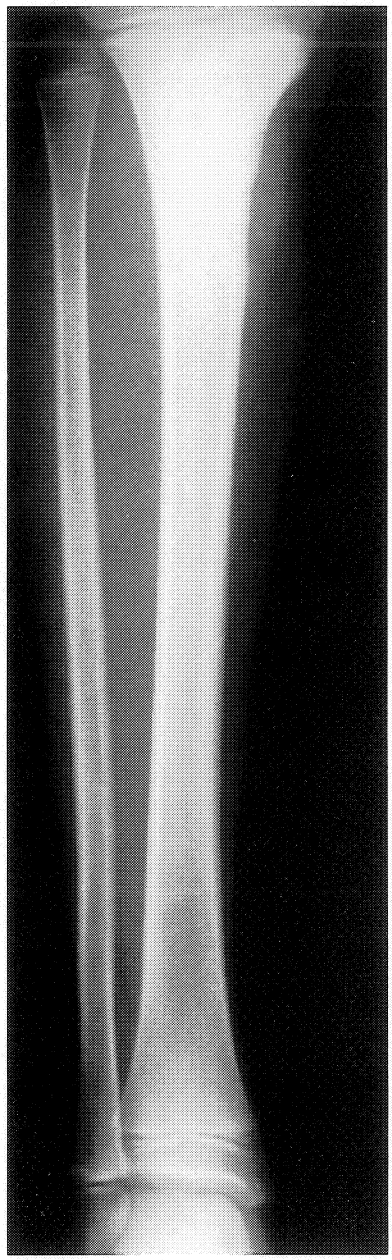

Figure 8A
AP radiograph of a nine year old male with osteogenic sarcoma of the proximal tibia.

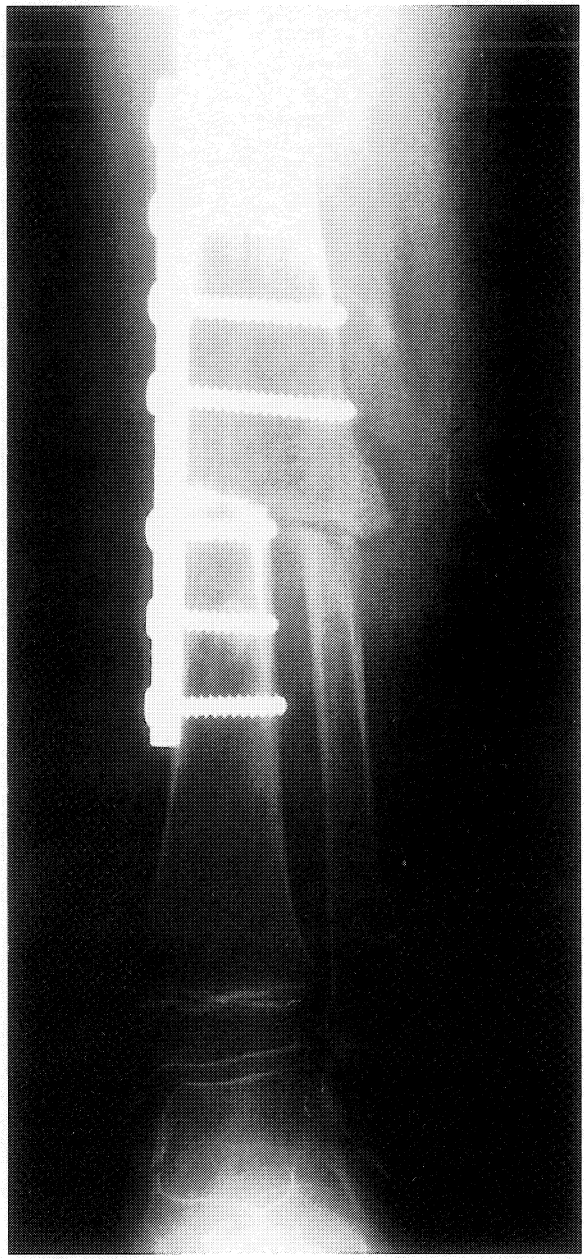

Figure 8B
AP radiograph after resection, 180 degree rotation and fixation of femur to tibia with internal fixation.

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A PRIMER OF OSTEOTOMY OF THE WEIGHT BEARING LONG BONES IN CHILDREN

Frederick R. Dietz, M.D.

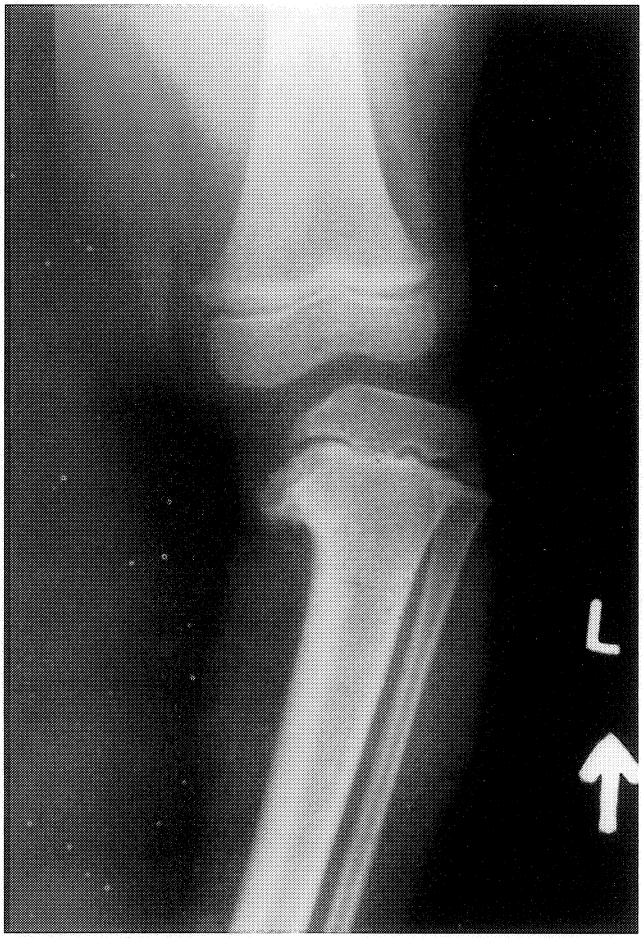

Figure 1 A. AP radiograph of a 3 1/2 year old girl with Stage III Blount's disease.

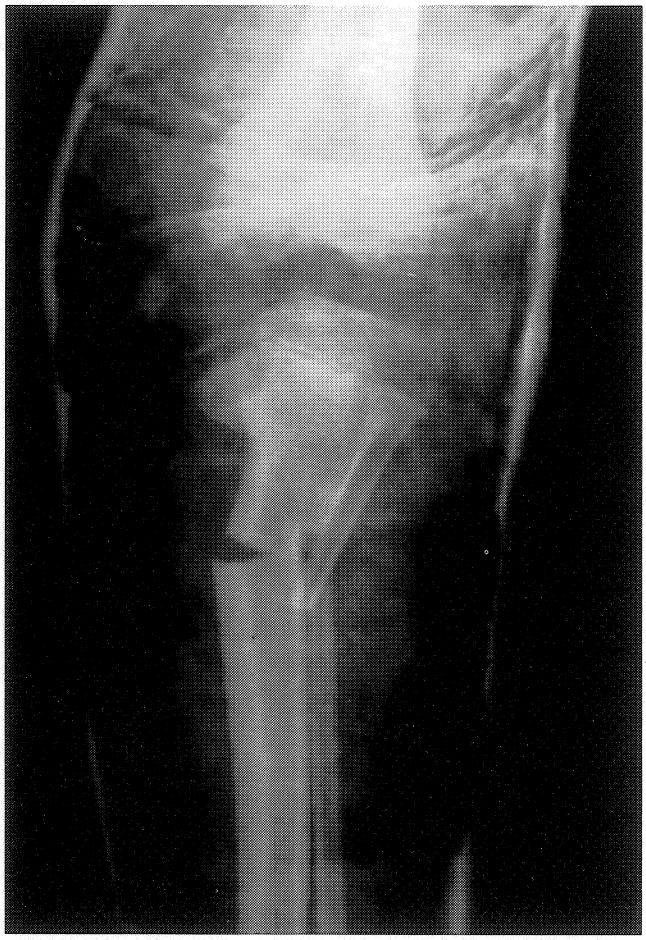

Figure 1 B. AP radiograph immediately after correction using a "spike" osteotomy.

INTRODUCTION

Osteotomy of the femur and/or tibia is a commonly performed operation in children. The causes of a treatable deformity are many, and include skeletal dysplasias, metabolic disorders, trauma, infections, tumors, and idiopathic angular deformities. Deciding whether, when, and how to perform an osteotomy depends on the severity of deformity and the natural history of the underlying cause. In spite of the need for flexibility in treating individual patients, general principles exist to guide appropriate treatment. Equally important is a clear recognition of the areas of decision where empirically derived principles do not exist. The decision to perform an osteotomy and the technique used should be carefully considered, since a high rate of complications occur including recurrence of deformity[3,16,24,30]. This paper will outline factors that should be considered in treating deformities of the weight bearing long bones in children and adolescents. The focus will be on varus and valgus deformities. Flexion, extension, and rotational deformities require somewhat different analyses and will not be specifically addressed in this primer. This is an admittedly idiosyncratic approach and is not meant to be all inclusive. Rather, the hope is to be provocative and to encourage careful consideration of the reasons behind performance of osteotomies in children.

The University of Iowa

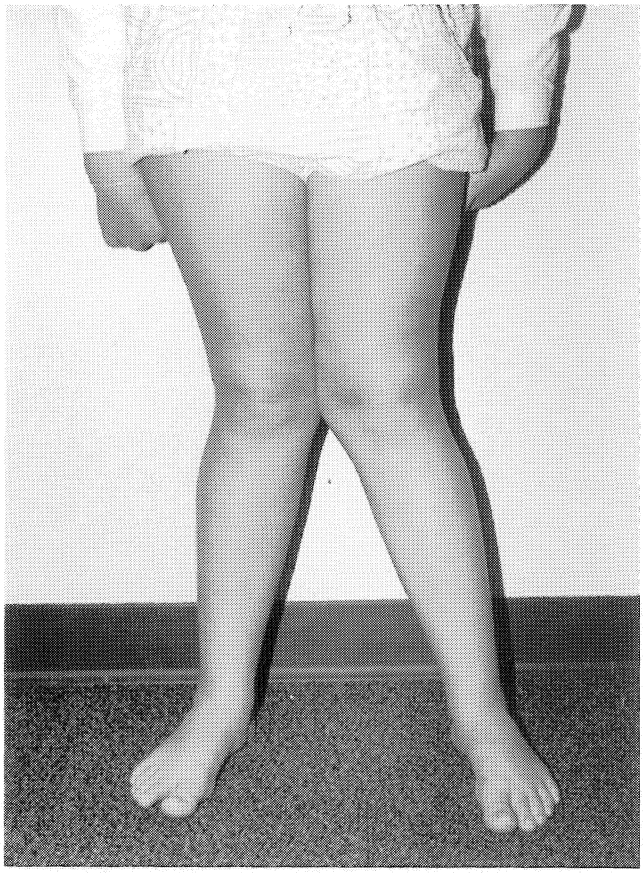

Figure 2 This ten year old girl was found to have mild hypophosphatemtic rickets after presenting for genu valgum. In spite of the severe valgus, the child was an excellent multiple sport competitive athlete. Her only complaint was a medial thigh chafing after prolonged running.

I. WHY PERFORM AN OSTEOTOMY?

Progressive Deformity

Certain conditions have a natural history of increasing deformity for which osteotomy may be curative if performed at an appropriate time. Blount's disease and Congenital Coxa Vara are two such conditions (Figure 1)[5,7,11,12,20,21,29,31]. If untreated they result in progressive, severe deformity with compromise of joint function. This type of progressive disorder constitutes an absolute indication for osteotomy.

Functional Disability

Sufficient deformity that compromises limb function rarely occurs in children (Figure 2). Feet or knees that strike the other limb during walking or running due to rotational or angular malalignment are uncommon. However, parents often attribute age-appropriate clumsinesses in young children to these deformities. Knowledge of normal motor coordination at different ages is necessary to evaluate these problems[23]. The spontaneous resolution

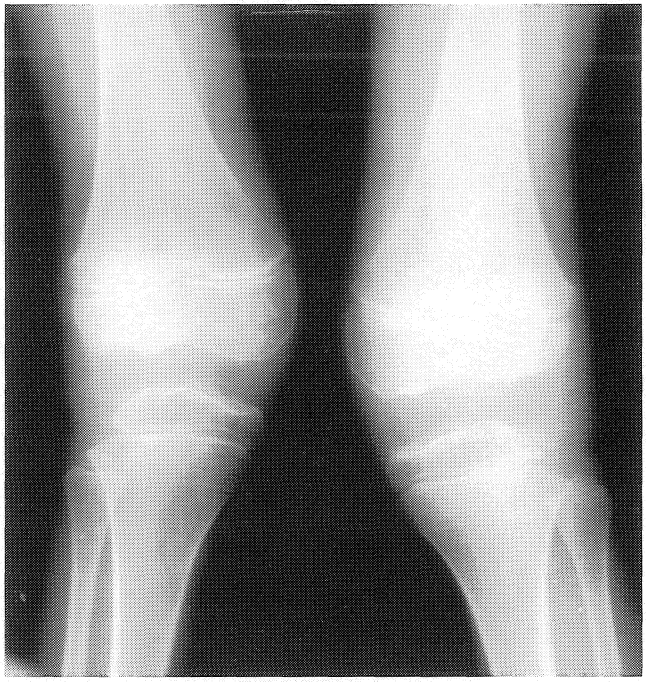

Figure 3A. AP radiograph of the knees of an eight year old boy with Morquio's Syndrome. Note the moderate genu valgum and the beginning epiphyseal fragmentation.

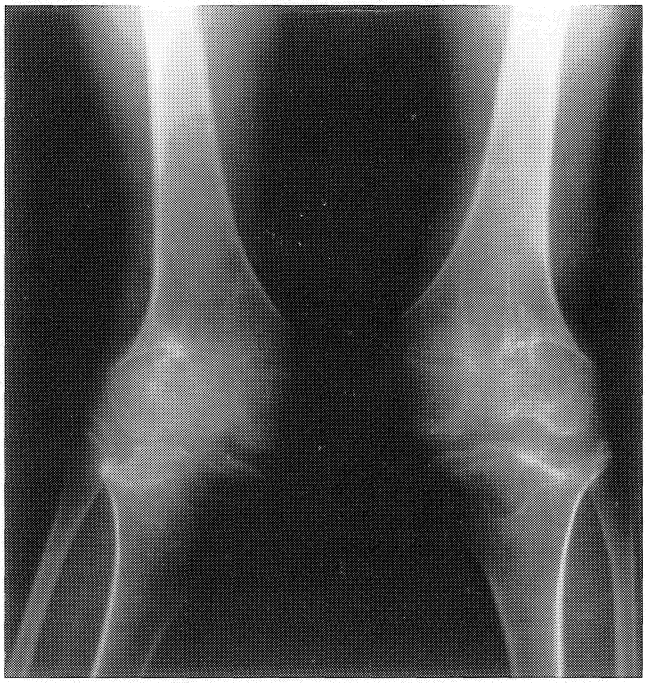

Figure 3B. AP radiograph of the same knees 12 years later. The joints are severely degenerated. Whether osteotomy would slow the degeneration of these joints is not known; but is unlikely.

of physiologic bowlegs and knock knees must always be considered. However, the combination of marked internal rotation of the femur and severe genu valgum may occasionally create a significant functional disability.

Osteotomy may be required after maximum spontaneous correction has occurred.

Joint Dysfunction

Pain, swelling, or giving way of the knee or ankle joints due to angular malalignment are rare complaints in childhood and adolescence. The degeneration of knee or hip articular cartilage in adults rarely occurs in children. Such degeneration is observed in metabolic bone diseases and in skeletal dysplasias (e.g. spondyloepiphyseal dysplasia, Morquio's syndrome) or after articular cartilage trauma (Figure 3)[1,26]. Osteotomy will probably not significantly alter the natural history of these disorders.

Progressive Ligamentous Laxity

Angular malalignment may cause progressive ligamentous laxity. This is primarily a concern about the knee. Large varus or valgus stresses may cause medial or lateral collateral ligament stretching. An extension moment (that produces a "back kneeing" gait) may cause progressive posterior knee joint laxity. Such laxity of ankle ligaments without repetitive injuries is rare.

Progressive laxity is uncommon; unfortunately, the present assessment of ligamentous laxity is a clinical estimate. No data exists which correlates the magnitude of deformity and age of patient with the propensity to develop progressive ligamentous laxity. One would surmise that young children with deformity would have a low tendency to develop laxity since the force applied to ligaments would be small relative to ligament strength. Increasing the size of the child and deformity might be expected to predispose to laxity. However, increased strength of ligaments with age might counteract these forces.

In children whose deformity is likely to recur if osteotomy is done in early childhood, the prevention of progressive laxity as the sole indication for osteotomy should be cautiously considered. Such children (e.g. achondroplasts and pseudoachondroplasts) should be followed yearly to assess ligamentous laxity.

Adaptive Joint Deformity

"Adaptive joint changes" may be defined as alteration in joint morphology or change in the soft tissue constraints to joint motion that result as compensation for a deformity. Adaptive joint changes may lessen the range of joint motion or alter the arc of motion. The best example of this phenomenon is an alteration in the motion of the subtalar joint due to deformity of the limb above. If a deformity causes the ankle joint to be in varus with respect to the weight bearing line, the subtalar joint must lie in valgus so the foot can be placed flat on the ground. Conversely, a valgus deformity may result in some varus positioning of the subtalar joint (Figure 4). If such positions are allowed to persist indefinitely, the soft tissues and/or articular surfaces may remodel so that a fixed valgus or varus deformity of the subtalar joint results. This occurs most commonly when a severe varus deformity of the tibia causes a valgus subtalar compensation. If the valgus attitude of the hind part of the foot becomes fixed, an osteotomy of the tibia to correct the varus results in a severe valgus foot that is unsightly and potentially painful due to an abnormal weight bearing pattern.

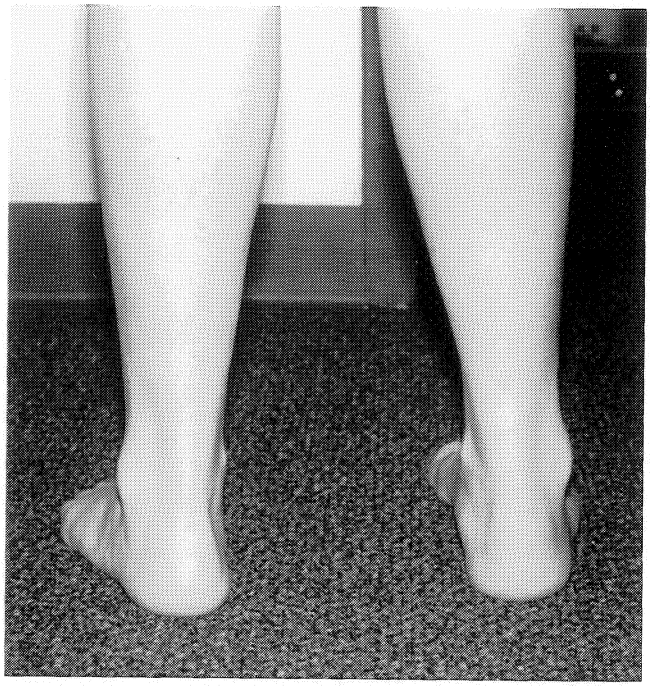

Figure 4 This normal 15 year old girl has moderately severe idiopathic genu valgum. Note the varus position of the heel. The varus of the hind part of the foot might become fixed after a long period of time.

The development of adaptive joint changes occurs over years rather than months. Such development can be assessed by clinical examination. An osteotomy is indicated to prevent the evolution of an adaptive deformity.

In children with ligamentous laxity (especially in skeletal dysplasias), such fixed compensation rarely develops. It is far more likely to occur in normal children after a deformity acquired through growth plate arrest or fracture, or among patients with neurological and neuromuscular disorders.

Osteoarthritis

Long bone malalignment resulting in weight bearing joints that are not perpendicular to the weight bearing force is recommended as an indication for osteotomy[2,9,13,28]. A joint surface that is not perpendicular to the weight bearing force is said to be predisposed to premature osteoarthritis. Two hypothesis are advanced to

support this position. First, angulation is believed to decrease the joint contact area and increase local joint pressure, thereby causing premature cartilage degeneration. Second, joints that are not perpendicular to the weight bearing force of the body result in shear stresses on the articular cartilage which is designed to resist compressive, not shear forces. Cartilage degeneration and premature osteoarthrosis will result if shear forces persist over a long period of time. Although these arguments are intuitive, clinical evidence is inconclusive. The experimental evidence to support this sequence of clinical events is weak as well.

Experimental evidence to support the relationship of joint malalignment and degeneration exists in three areas: whole cadaver bone and joint preparation, isolated articular cartilage preparation, and in vivo animal studies after surgically created angulation. Tarr et al, showed a decrease in ankle joint contact area using pressure-sensitive film in simulated angulation of cadaver tibia-fibula-foot preparations[28]. This effect was only found for distal one-third of the tibia fractures, within the range of angulation explored. Another similar model investigated the effect of simulated tibial angulation on cadaver knee joint contact area and pressure[13]. Little effect on contact area was found, but an increase in contact pressure was found in the compartment toward which the load-axis was shifted. Studies using static cadaver joint preparations have several major potential flaws. They do not account for the biological ability of joints to remodel both the subchondral bone and the articular cartilage. They ignore the possibility that an individual's neuromuscular system may alter load-axes in ways not predictable by such static models. Harrington used a force-platform cinephotographic technique to assess the relationship between angulation, magnitude of load, and the location of the load within the knee in patients with deformity of the knee and normal controls[6]. He concluded that "the assessment of load in the knee joint by static methods is unreliable . . . individuals can modify force transmission by adopting compensatory mechanisms that result in unloading of the knee."

Animal models with long bone deformity have predictably created measurable changes in articular cartilage and/or subchondral bone, but have not created osteoarthritis. Osteotomies of thirty degrees in rabbits and/or dogs have produced cartilage clefting, fibrillation, and chondrocyte cloning without radiographic evidence of joint space narrowing[8,18,33]. Small marginal osteophytes have been an inconsistent finding in these studies. Whether the cartilage lesions seen by these investigators would progress to osteoarthritis is unknown. True osteoarthritis has not been created in animals in the absence of meniscectomy or instability. Simon et. al., investigated the

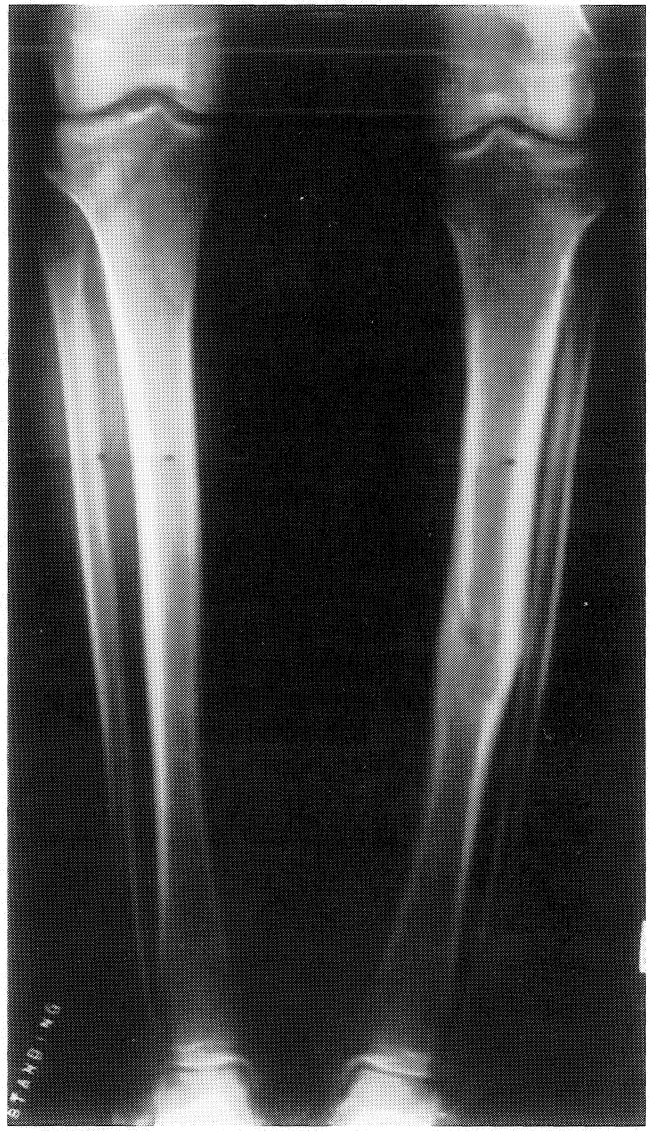

Figure 5 AP radiograph of a 54 year old man who suffered a left tibia fracture at 19 years of age. In spite of nearly 10 degrees of varus compared to the other tibia, he has no pain, functional limitations, or signs of osteoarthritis on radiograph.

effect of cyclic shear on osteochondral plugs obtained from bovine tibial plateaus[22]. The mechanical shear properties (loss and storage moduli) of the articular cartilage decreased 35% in the first 90,000 cycles with very little decrease thereafter, and no change after 108×10^4 cycles. No structural defects in the cartilage could be found by electron microscopy even after the maximum number of cycles. Whether one interprets this as evidence for or against the hypothesis that articular cartilage tolerates shear stresses poorly is a matter of opinion at present. Further work in this area is clearly needed.

Only one clinical study has purported to show a relationship between the development of osteoarthrosis and joint malalignment. Unfortunately, the study's methodol-

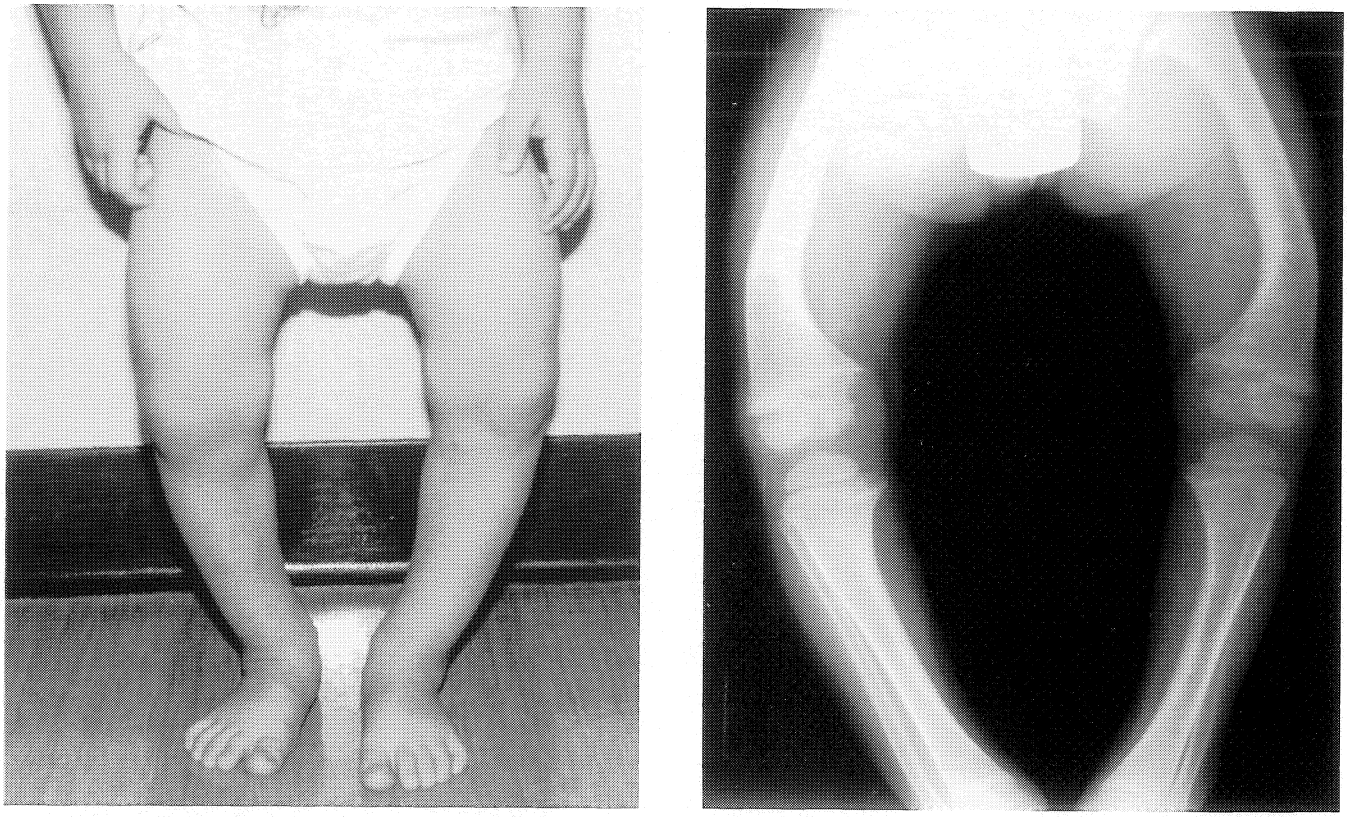

Figure 6A and 6B: This three year old boy with hypophosphatemic rickets has severe bowlegs. The parents strongly desired correction prior to his going to pre-school. We planned distal femoral and proximal tibial osteotomies for correction.

ogy is severely flawed[9]. The authors collected a series of thirteen patients who had both joint malalignment and osteoarthrosis; they assumed a causal relationship, and then described this relationship mathematically. The authors for this paper have taken two conditions whose intersection was found in only a small number of patients that were assembled from throughout the world. Without a population study indicating the incidence of both joint malalignment and osteoarthrosis, this data is meaningless. The association of joint malalignment and osteoarthrosis must be considered a chance occurrence in this small number of patients.

Two long term follow-ups of patients who suffered tibia fractures failed to find any increase in the instance of osteoarthrosis with increasing malalignment[4,10,14]. In one study, patients with a mean tibial angulation of 15 degrees (±15.9 degrees) faired no worse with respect to development of osteoarthrosis than tibia fractures angulated less than 5 degrees at a mean 30 year follow-up (Figure 5). It is possible that a dose/time effect exists and that the more severely angulated of these joints will develop premature osteoarthrosis. A long term follow-up of patients with Blount's disease did demonstrate the development of premature osteoarthrosis[7]. However, the incidence and severity of osteoarthrosis did not correlate with the amount of angulation. Rather, it correlated with the irregularity of the joint surface. There is no evidence in the literature that people with achondroplasia have an increase risk of osteoarthrosis in spite of very common residual malalignment of the weight bearing joints.

Given the immediate and real risks associated with osteotomy, one must carefully consider whether the prevention of osteoarthrosis is an appropriate indication for long bone osteotomy. At present, the amount of angulation compatible with long term good joint function without development of premature osteoarthrosis is unknown.

Studies that fail to show an association of joint malalignment with osteoarthrosis all involve people with normal articular cartilage and normal subchondral and metaphyseal support[4,10,14]. Patients with metabolic bone diseases such as Morquio's syndrome, and skeletal dysplasia such as spondyloepiphyseal dysplasia, do develop premature osteoarthrosis, and joint malalignments are common (Figure 3)[1,26]. Whether restoration of normal alignment in these joints will slow down the development of osteoarthrosis is not known. One can argue that given their predisposition to osteoarthrosis, the most normal alignment should be sought. Alternatively, one could argue that the underlying biochemical abnormalities resulting in pre-

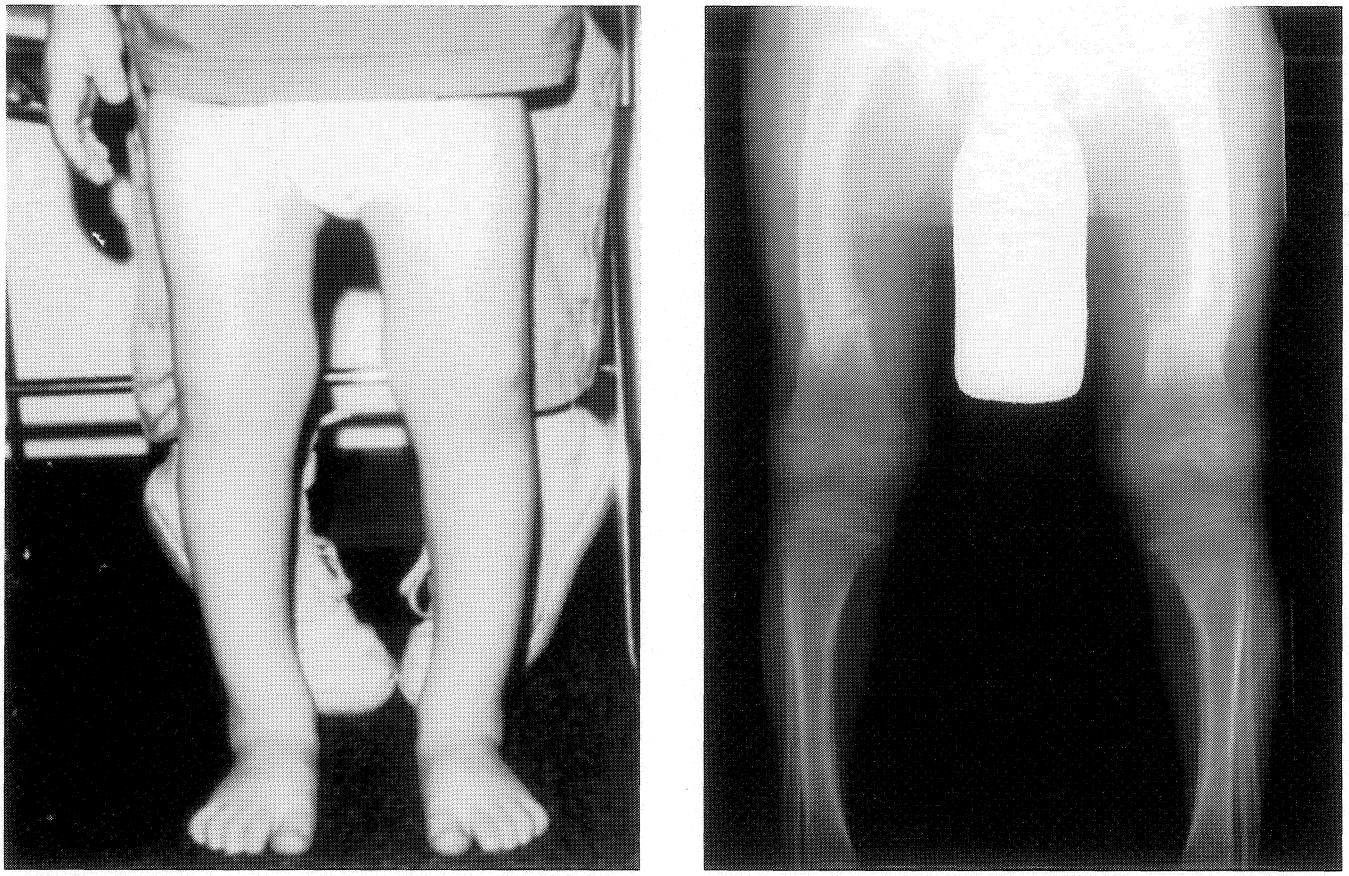

Figure 6C and 6D: Since the worst deformity was in the femur, correction was performed using a spike osteotomy. Since recurrent deformity is common with this form of rickets, tibial correction was deferred.

mature arthrosis are the determining factors and that alterations of alignment will have minimal if any effect on the long term survival of these joints. At present there is no evidence that osteotomy to improve joint alignment will result in avoidance or delay of osteoarthrosis in these patients.

Cosmesis

A major indication for an osteotomy of weight bearing bones is the appearance of the limbs. A severe varus or valgus deformity, especially if asymmetric, may cause significant distress to the affected child and his or her family. Cosmesis is not to be considered lightly and is an important reason for performing osteotomy. Unfortunately, cosmesis remains in the eye of the beholder. A deformity considered acceptable by a particular child and family is often unacceptable to another child and family. These issues must be carefully explored and negotiated with the family (Figure 6). However, it is not the physicians responsibility to perform an osteotomy whenever the alignment is felt to be unsatisfactory by the family. The physician has a knowledge of the potential complications of treatment that may be difficult to communicate to some families in spite of prolonged and intense discussion. A complication from surgery could compromise limb function that would otherwise be normal throughout life.

II. WHEN TO PERFORM AN OSTEOTOMY

Progressive Deformities

Whenever a progressive deformity is identified for which osteotomy is curative, an osteotomy should be performed. When the diagnosis of Blount's disease is made, for example, there is no advantage in delaying the performance of an osteotomy (Figure 1). If a physeal arrest has been identified and bar excision is feasible and appropriate, it is generally prudent to perform the osteotomy to correct malalignment at the time of the bar excision. If the physeal bar resection fails, recurrent deformity may still develop. If the patient is closely monitored, however, completion of the physeal arrest can avoid significant recurrent deformity. Situations occur in young children in which slowly progressive deformities should not be treated for a period of time so that the minimum number of osteotomies are necessary during the growing period. This is especially true of some dwarfing conditions such as pseudoachondroplasia wherein an osteotomy done at a very early age will commonly require repeat osteotomy later in childhood.

Functional Disability

In the rare cases where angular malalignment causes functional disability, it is appropriate to perform the osteotomy if the natural history of the malalignment is not spontaneous correction. For example, severe genu valgum and inward rotation of the femur in a 3 year old might cause a functional problem with running, yet resolve completely with continued growth. After 8 years of age spontaneous correction of angular and rotational malalignment cannot be expected, and osteotomy for correction is warranted in the rare patient with functional disability.

Ligamentous Laxity

Progressive ligamentous laxity due to deformity is uncommon. The difficulty with this indication for surgery lies in quantitating the amount of ligamentous laxity. This is a clinical assessment at present which depends on repeated examinations over a period of time. Instruments that measure ligamentous laxity are generally "sized" for adults and are not useful for quantitating laxity in children. Recording the amount of medial and lateral joint opening with stress is probably the best clinical technique available to assess laxity.

Adaptive Joint Deformity

Adaptive joint deformity is rarely an indication for osteotomy. Since adaptive joint changes develop slowly, preventive osteotomy is unnecessary. The best method for assessing the development of compensatory joint deformity is repeated clinical examination. For example, a single examiner estimating the amount of subtalar motion will be able to pickup a loss of motion on serial examinations before any functional consequences occur.

PREVENTION OF OSTEOARTHROSIS

There are no guidelines at present for the timing of prophylactic osteotomy to avoid premature osteoarthrosis. Less than 20 degrees of deformity at the ankle may be accepted and possibly as much at the knee according to existing clinical data (Figure 7)[4,10,14,17].

Appearance (Cosmesis)

Cosmesis is an acceptable indication for osteotomy when the surgeon and the family agree that the psychosocial costs of the deformity out weigh the risks of surgery. This must be an individualized decision. The risks of osteotomy and the possible need for repeat osteotomy must be fully explained. Many parents will delay osteotomy in a toddler for a deformity that is cosmetically unacceptable in order to decrease the likelihood that repeat osteotomies will be necessary.

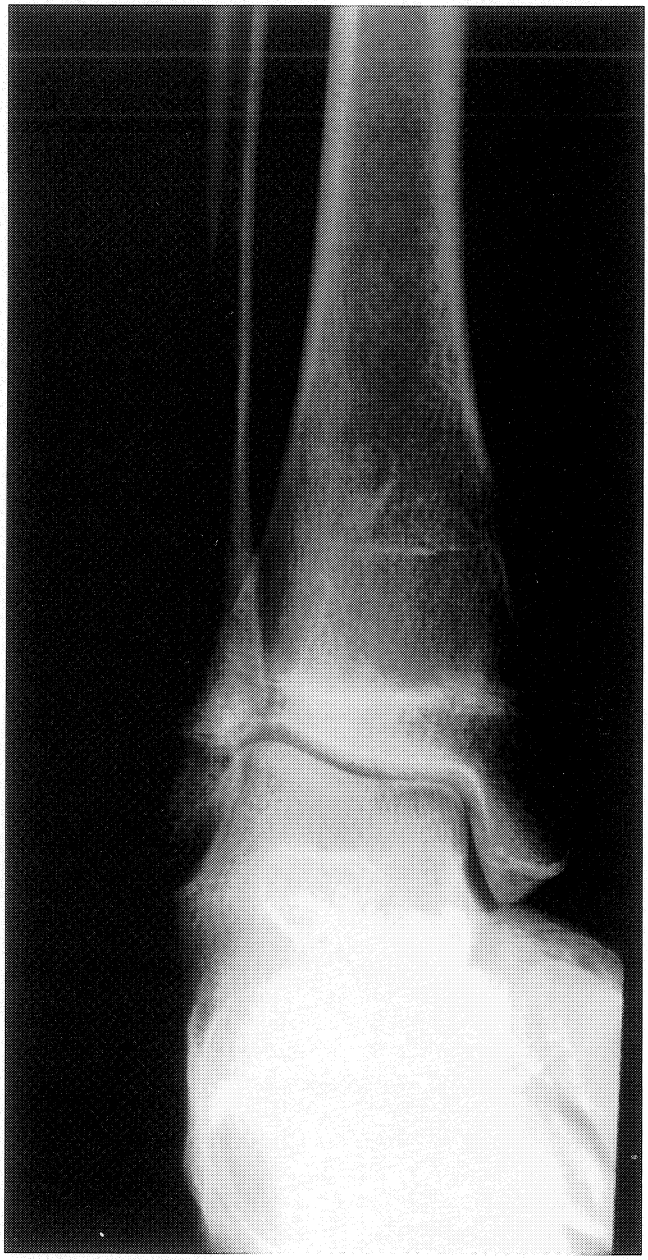

Figure 7 AP radiograph of a 15 year old boy who suffered a distal tibial physeal arrest after an ankle fracture. This resulted in 14 degrees of valgus compared to the other side. He had no pain or laxity, full range of motion, and a nearly normal appearance of the ankle. Given the available information, we did not counsel the family that osteotomy was necessary to avoid premature osteoarthritis.

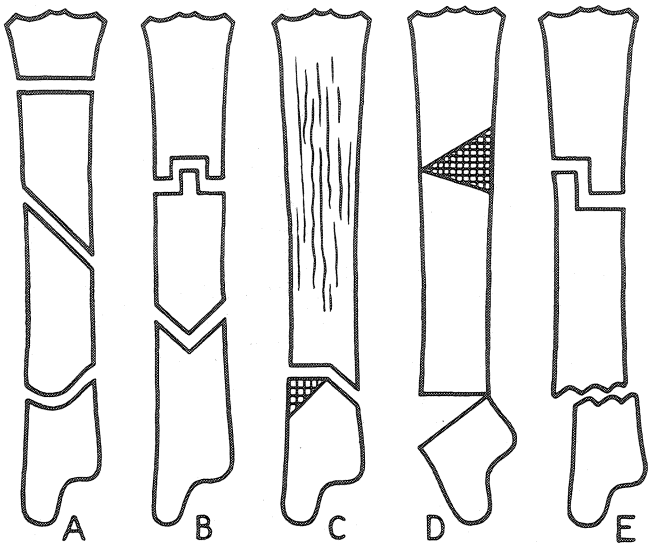

Figure 8 Some of the many osteotomies designed for correction of long bone deformity. (Reproduced with permission from: Milch, H.: Osteotomy of the Long Bones, p. 16, Springfield, Illinois, Charles C. Thomas, 1947.)

III. HOW TO PERFORM AN OSTEOTOMY

A bewildering array of technical options exist when the decision is made to perform an osteotomy (Figure 8). This section will not cover all aspects of osteotomy technique, since specific problems will require specific and sometimes unusual solutions. An attempt will be made to cover general principles important in all osteotomies.

The issues that must be considered include the level and number of osteotomies, the specific technique, the method of fixation, and the potential complications. It is axiomatic that the simplest and safest effective technique is best. Therefore, the specific goals of the osteotomy must be kept clearly in mind when choosing a technique.

It is best to perform an osteotomy at the level of the deformity. In multiplane deformities, a compromise level must be chosen. A common problem occurs when the deformity is at or adjacent to a joint. The osteotomy must then be placed away from the maximum deformity to avoid physeal injury. Another common problem occurs if there is deformity on both sides of a joint or if there is a long curved deformity throughout the bone. Multilevel osteotomies must then be considered.

Two arguments are commonly made to support the use of multilevel osteotomies in spite of an increased incidence of complications and the requirement for more rigid fixation. The first argument is that joint surfaces must be made perpendicular to weight bearing line. The limitations of the evidence supporting this argument have already been discussed. Nonetheless, few surgeons would choose an osteotomy that significantly increased the obliquity of a weight bearing joint for fear of long term joint dysfunction. The second argument for multilevel osteotomies is that the mechanical axis should be restored. Restoration of the mechanical axis in complex deformities, especially those associated with skeletal dysplasias and metabolic bone disease, often requires multilevel and multiplanar osteotomies. No evidence exists that restoration of the mechanical axis improves the functional outcome for the lower extremity except in total knee arthroplasties (Figure 9). It is clear, however, that multilevel osteotomies run a higher risk of limb function compromise through a higher risk of stiffness from scaring, nerve injury, and possibly infection.

The technique of osteotomy should be chosen to accomplish specific goals with the fewest complications. The most common osteotomy techniques at present are opening wedge osteotomies, closing wedge osteotomies, and dome osteotomies.

Closing wedge osteotomies have the advantages of good immediate stability and a diminution of tension in the soft tissues. This is particularly important when soft tissue stretching risks nerve injury, such as when a valgus deformity of the tibia or femur is corrected acutely. The disadvantages of closing wedge osteotomies are that they generally require internal fixation and they shorten the involved bone.

Opening wedge osteotomies have the obvious problems of stretching the soft tissues, delayed healing, collapse of the grafted bone, and the need for prolonged internal or external fixation. These osteotomies should be considered when length is important enough to outweigh these added risks. Soft tissue stretch causing nerve or blood vessel injury and skin compromise must be carefully considered when performing an opening wedge osteotomy.

The Dome osteotomy is probably the most commonly utilized technique for children at present. This osteotomy has fairly good intrinsic stability and does not result in net shortening or lengthening of the bone. Most authors recommend internal fixation (at minimum with pins) until healing has occurred[2,15,19,25].

A popular osteotomy at the University of Iowa is the spike osteotomy (Figure 3B)[3]. This osteotomy has sufficient intrinsic stability that only plaster cast immobilization is required in the majority of cases, and rapid healing can be expected. This osteotomy is the technique of choice for single level uni-planer deformities. In addition to being stable and healing rapidly, this osteotomy does not change the bone length.

A newer technique of osteotomy based on the principles of limb lengthening developed by Illizarov employs a corticotomy with gradual displacement of the bone ends, allowing bone to regenerate as displacement occurs. This technique has the advantage of allowing correction of complex, multiplaner deformities while protecting nerves and blood vessels from injury by the gradual nature of the correction. This technique is particularly valuable when

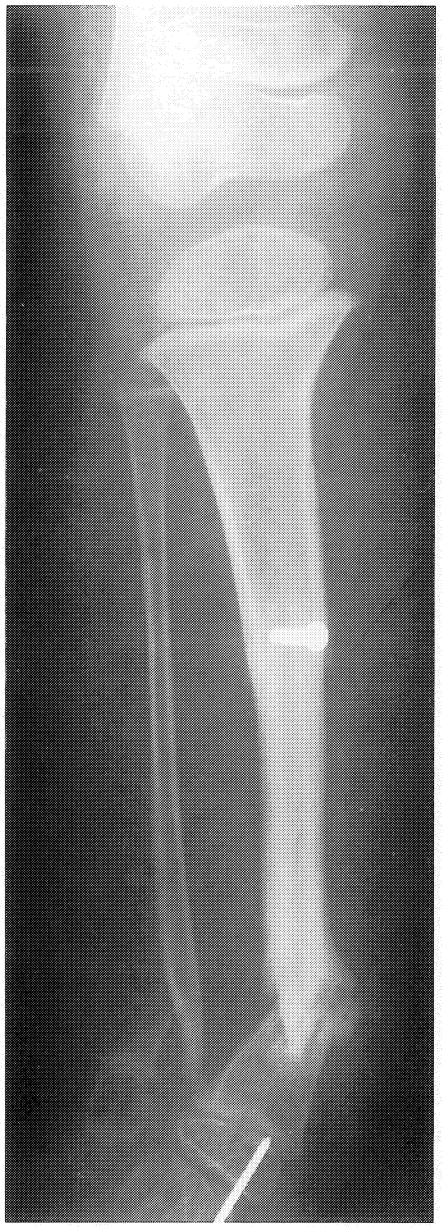

Figure 9a

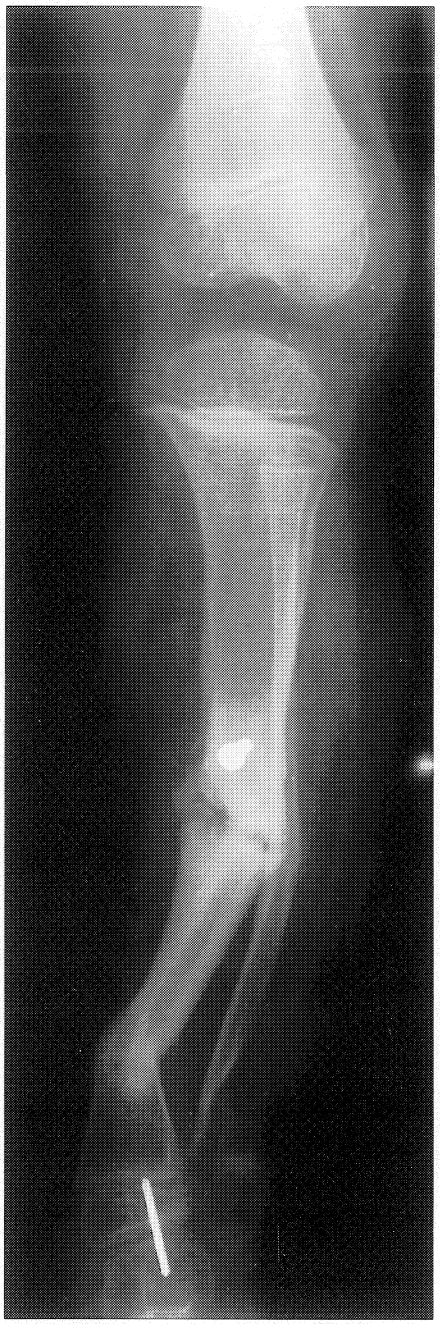

Figure 9b

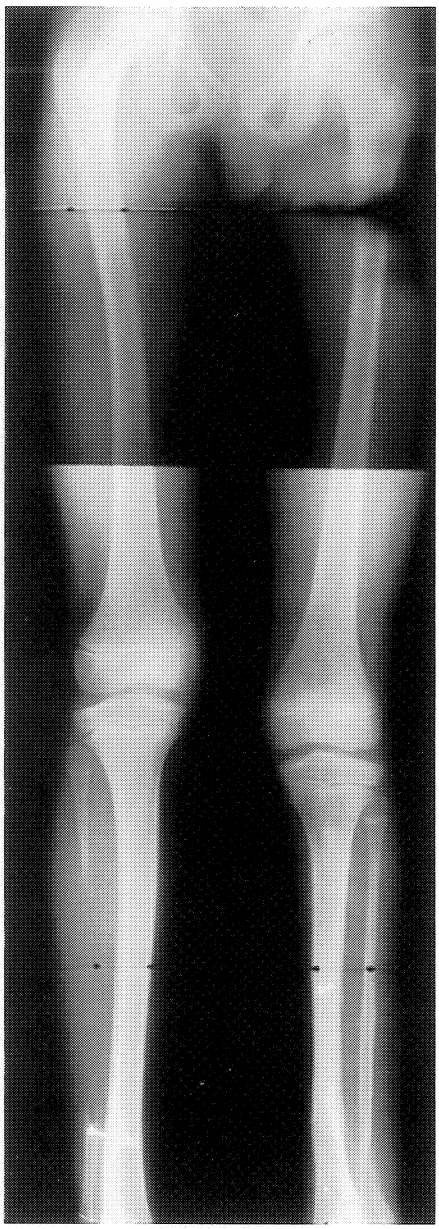

Figure 9c

Figure 9 A. AP radiograph of a 4 year old boy who has obtained union of a distal tibial congenital pseudarthrosis after a vascularized fibula transfer. Severe valgus exists, however, at the site of the pseudarthrosis. B. AP radiograph after a proximal osteotomy was performed to avoid disturbing the prior pseudarthrosis site. This resulted in a zig-zag deformity with offset of the ankle relative to the knee and a worsened deviation from a normal mechanical axis. C. AP radiograph at follow-up. With time and remodeling the overall alignment of the tibia is much improved; including the mechanical axis. Although the mechanical axis is not "perfect", it may be "perfect enough" for good limb function. In selecting osteotomy techniques, one must consider the growth and remodeling potential of children's bones.

limb lengthening must be employed in addition to osteotomy to accomplish the goals of limb reconstruction. Some advocates of this method have found that the gradual correction of deformity in addition to lengthening is unacceptably complicated. These surgeons recommend acute correction of the deformity followed by a gradual distraction for length as the safest and most effective technique. However, in situations such as a severe valgus deformity, gradual correction to avoid peroneal nerve palsy should be considered. One may consider the use of intraoperative nerve conduction velocities to assess stretch of the peroneal nerve during acute correction of severe valgus deformities about the knee.

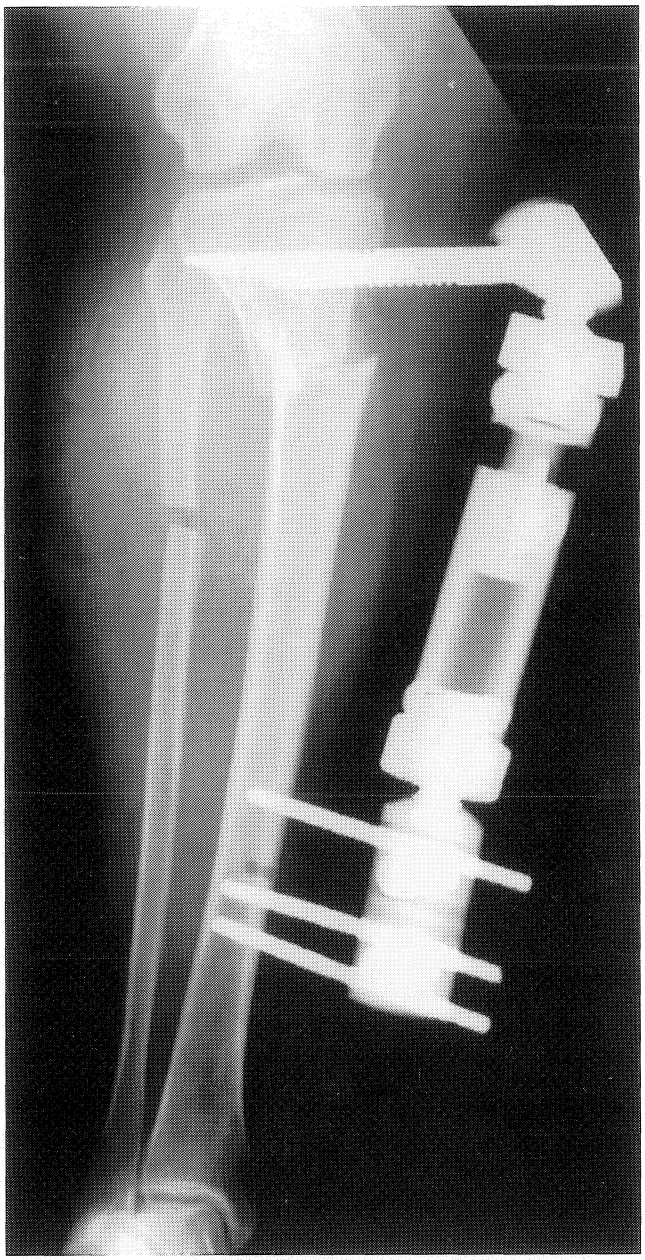

Figure 10 AP radiograph following the use of an external fixator to stabilize a tibial osteotomy in a short, obese female with hypophosphatemic rickets. This allowed adjustment of angulation after the patient was upright, as perfect alignment was difficult to assess in the operating room. Furthermore, her obesity would have made the application of an adequate cast very difficult. The amount of valgus was decreased post-operatively from that shown in the photograph.

The disadvantages of external fixation techniques are the relative technical complexity of their application and the higher incidence of complications. Infected pins, unwanted deformities developing during correction, need for re-osteotomy of premature consolidation, and delayed healing are common reasons for unexpected re-operations. They also have the cosmetic disadvantage of leaving a number of pin holes in the limb and a longer treatment time when compared with other osteotomy techniques. In spite of these complications, some complex deformities are optimally treated by external fixation.

Another use for external fixation is in very heavy children and adolescents in whom cast fixation alone or even cast fixation with pins across the osteotomy may not provide sufficient rigidity to maintain alignment. External fixation can provide stable, relatively rigid fixation in such patients (Figure 10).

Determining the level and amount of osteotomy angle can be difficult. Radiographs that demonstrate the hip, knee, and ankle on the same film are helpful in this regard. In patients with joint laxity, especially those with dwarfing conditions, the apparent deformity while weight bearing may be much greater than the intrinsic bony malalignment due to this laxity. If correction of the full apparent (weight bearing) deformity is undertaken, an opposite deformity may develop with the bony alignment being excessive in the other direction. For example, excessive valgus may be present after correction of a varus deformity. Non-weight bearing full leg radiographs are helpful in these cases (Figure 11). Intraoperative arthrograms to assess the joint "fit" may also help determine the appropriate magnitude of osteotomy. The amount of correction sought also varies with the disease process. For example, in Blount's disease or developmental coxa vara, over-correction of the deformity is often desirable.

SUMMARY

A. Why Perform An Osteotomy In Childhood or Adolescence?

1. Progressive deformity for which osteotomy is curative.
2. Cosmesis.
3. To prevent the development of late osteoarthritis (unproven).
4. Progressive ligamentous laxity (rare)
5. Functionally limiting deformities (rare).
6. Development of adaptive joint deformity (rare).

B. When To Perform An Osteotomy

1. Immediate correction for progressive deformities or functionally limiting deformities that can not be expected to improve by the natural history of the deformity.
2. Immediate correction if the problems listed under "A" are present such as the development of adaptive joint deformity or progressive ligamentous laxity.
3. If no indication for osteotomy exists except cosmesis, consider delaying correction to avoid the need for repeated osteotomies in disorders for which recurrence is common.

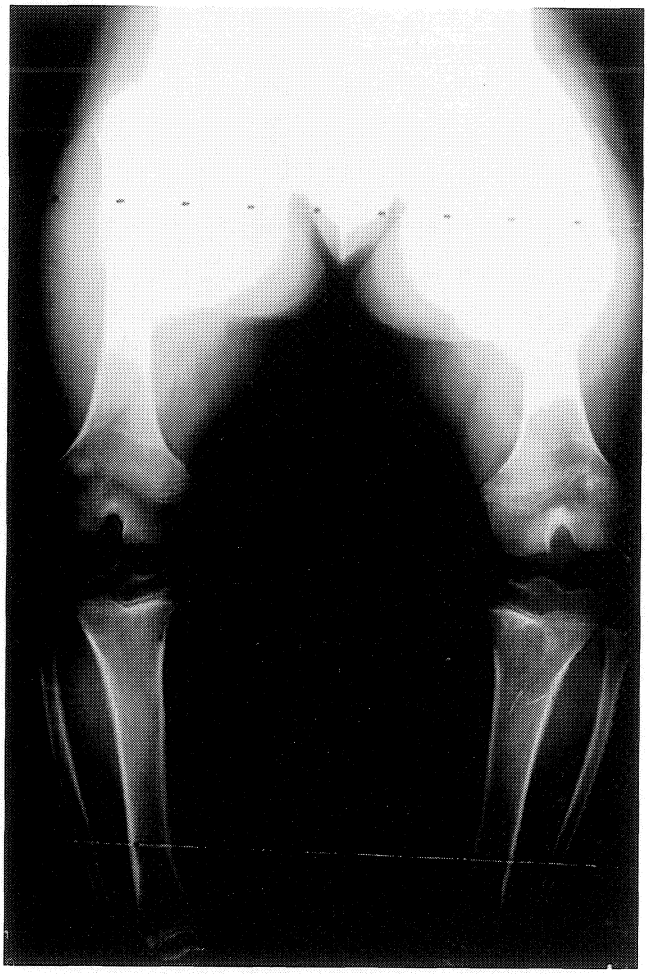

Figure 11A Marked lateral knee joint laxity exaggerates the "intrinsic" bony varus on standing radiographs.

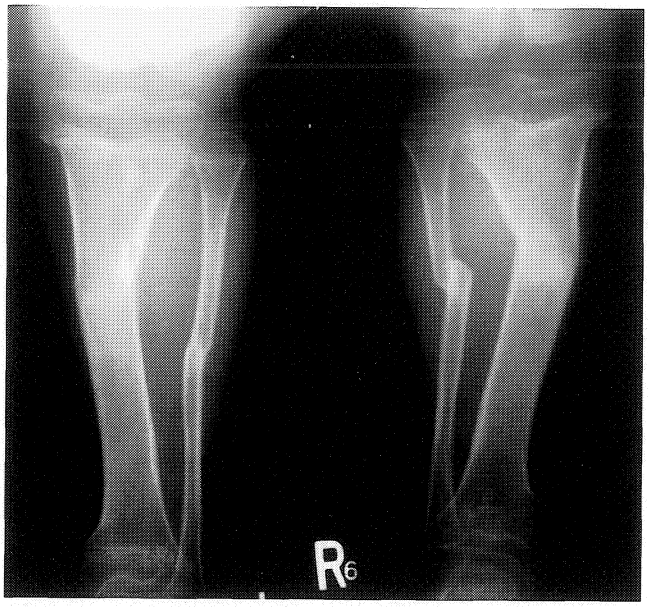

Figure 11B Supine radiographs after corrective osteotomy are shown to compare the joint space with the standing radiographs.

Figure 11 These AP radiographs are of an 11 year old boy with achondroplasia.

C. How To Do The Osteotomies

1. Perform the osteotomy at the level of the deformity when possible.

2. Choose the simplest, safest method that will accomplish specific goals.

3. Complex restoration of perfect mechanical axis and joint alignment may not be necessary. The lesser risks of simpler techniques should be considered.

4. The specific technique chosen will depend on the experience and abilities of the treating physician.

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MECHANORECEPTOR ENDINGS IN HUMAN CERVICAL FACET JOINTS

Robert F. McLain, M.D.*

ABSTRACT

Normal cervical facet capsules, taken from three human subjects, were examined to determine the density and distribution of three types of mechanoreceptive nerve endings. Clearly identifiable mechanoreceptors were found in 80% of the specimens and were categorized according to the classification of Freeman and Wyke. Eleven Type I, twenty Type II, and five Type III receptors were identified, as well as a number of small, unencapsulated nerve endings. Type I receptors were small, globular structures measuring 25-50 microns in diameter. Type II receptors varied in size and contour, but were characterized by their oblong shape and broad, lamellar capsule. Type III receptors were relatively large, oblong structures with a thin, amorphous capsule, within which a reticular mesh-work of fine neurites was embedded. Free (nociceptive) nerve endings were found in sub-synovial, loose areolar, and dense capsular tissues.

The presence of mechanoreceptive and nociceptive nerve endings demonstrates that cervical facet capsules are monitored by the central nervous system, and implies that neural input from the facets is important to the function of the cervical spine. Previous studies have suggested that protective muscular reflexes modulated by these types of mechanoreceptors are important in preventing joint instability and degeneration. The complex neural elements identified in this study may be damaged inadvertently when surgically exposing the cervical spine. Care should be taken, during posterior approaches, to avoid excessive stripping of the facets in portions of the spine not being fused.

INTRODUCTION

Encapsulated mechanoreceptors and free nerve endings have been identified in the periarticular tissues of all major joints of the body, and in every articular tissue except cartilage[11,25,26,31]. These receptors monitor joint excursion and capsular tension, and may initiate protective muscular reflexes that prevent joint degeneration and instability. Recent electrophysiological studies have demonstrated the presence of proprioceptive endings in facet and paraspinous tissues, as well as documenting their response to pressure and facet motion[10,32]. Although mechanoreceptors have been histologically identified in some spinal tissues, receptor type and distribution have not been analyzed.

The purpose of this study was to characterize the types and distributions of mechanoreceptive nerve endings in normal human cervical facet joints.

MATERIAL AND METHODS

Twenty-one cervical facet capsules were taken from three normal human subjects. Thirteen capsular specimens were removed from one donor at necropsy (C1-2 to C7-T1). Four capsules each were removed from two other normal subjects at the time of cervical stabilization for traumatic injuries (C4-5 to C5-6 in one, and C3-4 and C5-6 in the other). Only intact capsules at levels adjacent to injury were included. The cervical joints were examined at each sampled level to rule out pre-existing degenerative changes; all facets were free of joint degeneration based on radiographic and direct visual examination. None of these patients had any systemic disease which might be associated with neuropathy or cervical arthropathy.

Facet capsules were excised en-bloc with a sharp scalpel and forceps; care was taken to avoid crushing or distorting the tissue. The electro-cautery knife was not used. Tissue specimens were frozen in saline prior to processing with the modified gold chloride technique[7]: After thawing, each specimen was placed, whole, in a solution of three parts commercial lemon juice and one part 88% formic acid. The specimen was held in solution for fifteen minutes at room temperature, then removed and dried, and placed in a 1% solution of gold chloride for fifteen minutes in darkness. The gold chloride solution was decanted, and the specimens were dried and placed in a 25% formic acid solution for four to six hours. They were then rinsed twice in ethanol and placed in glycerol over night. After removal from the glycerol the specimens were dehydrated and embedded in paraffin. Twenty-five micron serial sections were cut and mounted on glass slides. After de-paraffinization the specimens were cover-slipped in Permount. No post-staining was necessary. No sampling techniques were applied in this study; because of uncertainty about the expected density of receptors in these tissues, each specimen was entirely sectioned, and every section reviewed. Hence, the likelihood of sampling error in this study was eliminated.

All sections were analyzed under light microscopy using an Olympus BH-2 microscope with a photographic head. No section was excluded because of inadequate staining or artifact. Each section was examined under 140x final

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TABLE 1
Classification of Nerve Endings According to Freeman and Wyke[11]

TYPE	MORPHOLOGY	PARENT NERVES	FUNCTION
I	Globular corpuscles in clusters. Thinly encapsulated	Small myelinated fibers.	Static, dynamic; low threshold, slowly adapting.
II	Conical corpuscles, thickly encapsulated.	Medium myelinated fibers.	Dynamic receptors; low threshold, rapidly adapting.
III	Fusiform corpuscles, thinly encapsulated.	Large myelinated fibers.	Dynamic receptors; high threshold, very slowly adapting.
IV	Free nerve endings.	Very small myelinated. Unmyelinated.	Pain receptors; high threshold, non-adapting.

magnification for the presence of large and small nerves, fine myelinated and unmyelinated axons, free nerve endings, and encapsulated nerve endings. Encapsulated endings were classified according to Freeman and Wyke[11] (Table 1). In order for an identified structure to be confirmed and classified as a mechanoreceptive nerve ending three criteria had to be met: 1) it had to have evidence of encapsulation; 2) it had to have a morphology consistent with previously described receptors; and 3) it had to be identifiable on consecutive sections without evidence of artifacts. In addition, for most confirmed receptors, a parent axon could be identified leading to the structure. The presence of a parent axon allowed the observer to definitively differentiate between nerve endings and artifacts or vessels. Oil-emersion (700x) magnification was used to verify encapsulation and to distinguish between axon filaments, the central axon core, and the darkly stained lumen of small caliber vessels. The presence of free nerve endings was also noted.

RESULTS

Mechanoreceptor endings were found in seventeen out of twenty-one capsule specimens. Six capsules contained only a single receptor, five capsules contained two receptors, four capsules contained three, one capsule contained four, and one contained five receptors which could be classified. When capsules contained more than one receptor, the receptor types were usually different. The majority of endings resembled the Type II mechanoreceptors identified in cat joints[11,25,26]. Fewer Type I receptors were identified, and only a handful of Type III receptors were found in any of the tissues. Parent axons were identified for the majority of these receptors and usually measured three to five micrometers in diameter.

Type I mechanoreceptive endings, which are small globular endings measuring twenty-five to fifty microns in diameter, were found in both loose areolar tissue and dense capsular tissue. They were always solitary receptors. No clusters of Type I nerve endings were found in these specimens (Figure 1).

The Type II mechanoreceptive endings varied considerably in size and shape (Figures 2A and 2B). The largest of the endings measured 500 microns in its greatest length and was characterized by a broad capsule with numerous lamellae. Cross-sections through the capsule of Type II receptors revealed concentric layers of faintly pink material alternating with light grey lamellar lines stained with gold chloride (Figure 3). These receptors were found in dense, fibrous capsular tissue, usually along a thin cleft of fatty tissue through which the parent axon and microvasculature passed.

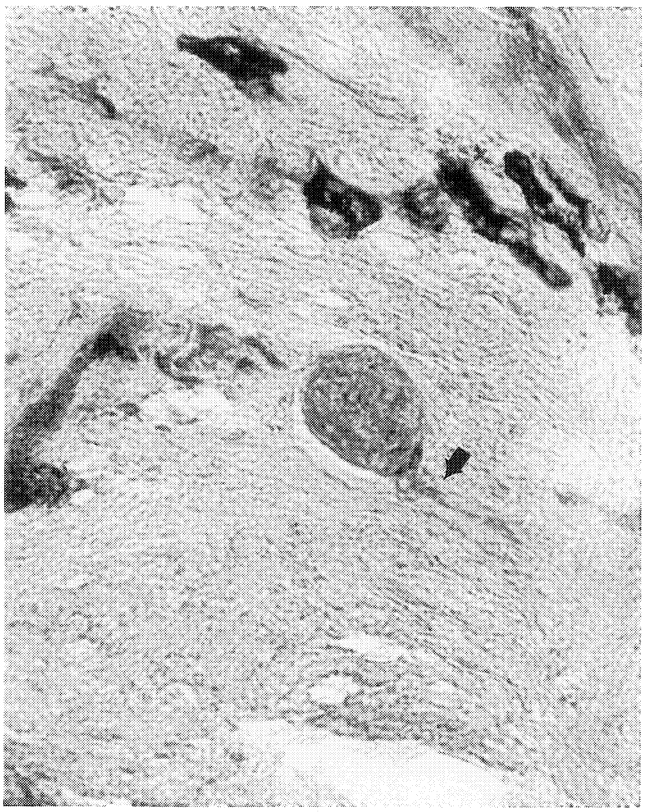

Figure 1
Type I Mechanoreceptor. This small, globular receptor was found in the dense connective tissue of the capsule of a C2-3 facet joint; it measures approximately 35 microns in greatest diameter. Fine filaments within the capsule are just visible at this magnification, and the parent axon is visible (arrow).

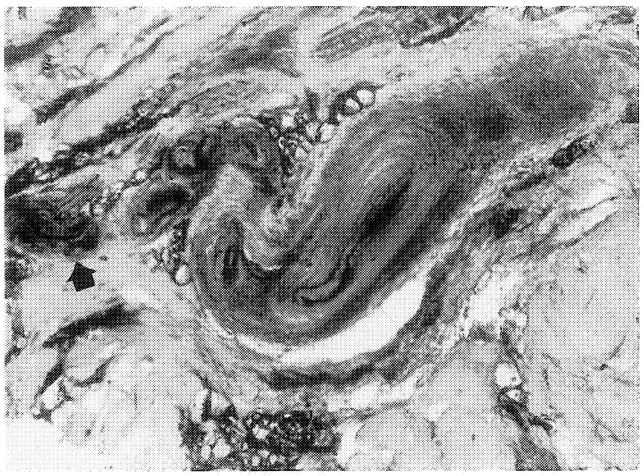

Figure 2

Type II Mechanoreceptors. Type II receptors vary considerably in size and shape and are the most frequent type found in cervical facets. They are characterized by a thick capsule (10-12 lamina thick) and a fusiform or cylindrical shape.

Figure 2A

This large, encapsulated mechanoreceptor (350 microns in length) came from the dense connective tissue of a C1-2 cervical facet capsule. The parent axon and its accompanying capillaries traverse the capsule along a narrow cleft of fat. The parent axon (arrow) enters the receptor at one end.

Figure 2B

A smaller, oblong receptor (250 microns in length) from the C1-2 level, is shown with its parent axon (arrow). The multiple lamellae are typical of Type II capsules.

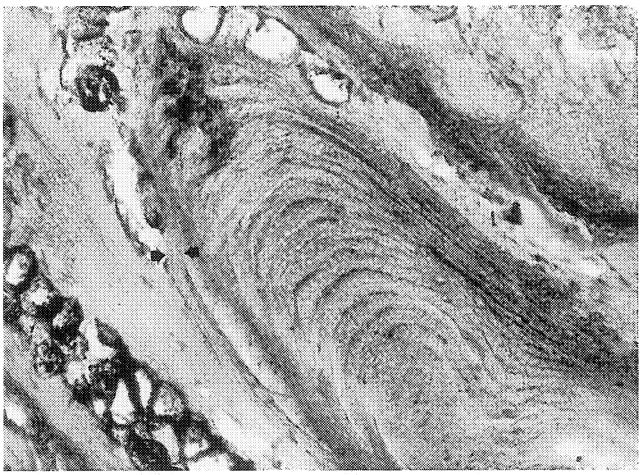

Figure 3

Dense lamellar lines alternate between layers of lighter staining material in this high magnification view of the capsule of a Type II mechanoreceptor. These lamellae are arranged concentrically around the central axon core, producing an onion-skin appearance. An outermost lamina separates the structure from the surrounding connective tissue and a small aggregation of fat.

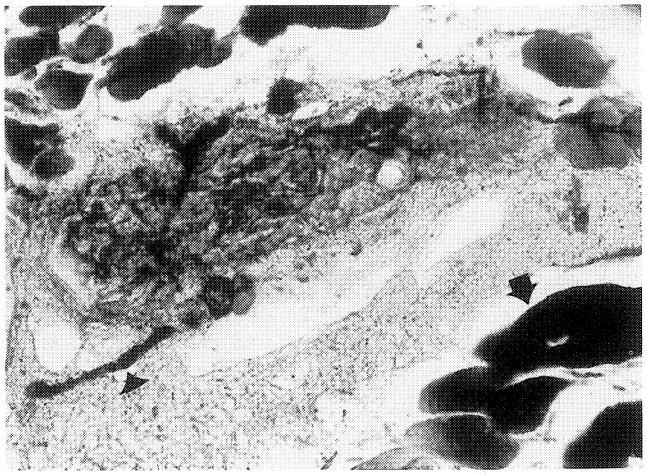

Figure 4

Type III Mechanoreceptor. This complex web of fine neural elements is separated from the surrounding connective tissue by a thin, amorphous capsule, which does not have distinct lamellae like the Type II capsule. This receptor, (350 microns in length), taken from a C3-4 facet joint, is located at the junction of the capsular connective tissue and the attachment of the paraspinous musculature. Skeletal muscle fibers (arrow) are seen insinuating themselves between strands of the fibro-fatty connective tissue. The parent axon is seen at lower left (arrowhead).

Type III mechanoreceptors were found at the junction between the dense fibrous capsule and the loose areolar, sub-synovial tissue. Inspection of Type III receptors at higher magnification revealed a reticular mesh-work of fine neurites contained within an oblong capsule (Figure 4). The receptor capsule was amorphous, lacking lamellar divisions, and extended just beyond the internal neural network.

Fine, unmyelinated nerves consistent with Type IV receptors (less than five microns) were identified in both the dense capsular tissues and in the synovial and areolar tissues; these fine filaments most likely represent nociceptive nerve endings (Figure 5). Such fibers also accompanied many of the vessels within the dense fibrous tissue of the capsule.

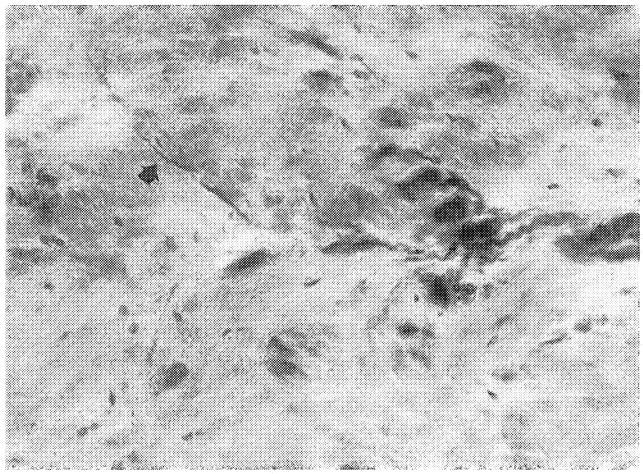

Figure 5
Type IV Mechanoreceptor. This high power photomicrograph demonstrates a free nerve ending (arrow) in the dense connective tissue of a C5-6 facet capsule. Nociceptive free nerve endings are unencapsulated, and found throughout the capsule, synovium, and loose areolar tissue of normal facet joints.

There were no apparent differences in the distribution of receptors between upper cervical versus lower cervical levels. No specific level appeared to be more richly innervated than any others. Likewise, there was no apparent difference from individual to individual with respect to the density of receptors found in their facet capsules.

DISCUSSION

Synovial joints enjoy a dual pattern of innervation: *primary articular nerves* are independent branches from larger peripheral nerves which specifically supply the joint capsule and ligaments; *accessory articular nerves* reach the joint after passing through muscular or cutaneous tissues to which they provide primary innervation[12,31]. These primary and accessory articular nerves terminate in a variety of encapsulated and unencapsulated nerve endings.

Freeman and Wyke described four basic types of afferent nerve endings in articular tissues, and documented the presence of those endings in several different joints[11]. Histological studies have demonstrated these receptors in a variety of tissues, including ligaments[7,13,25], joint capsule[11,17], meniscal tissues[26], and periarticular fat and muscle[8,11]. Types I through III are encapsulated receptors thought to be involved primarily in proprioception. These endings respond more to the extremes rather than the mid-range of joint motion[4,5,16]. It is also thought that these mechanoreceptors may influence the activity of muscles crossing the joint, modulating protective muscular reflexes important to joint function and stability[1,2,3,9,28,30]. Type IV receptors are unencapsulated free nerve endings found in soft tissues throughout the musculo-skeletal system. With recent advances in neurohistochemical techniques, investigators have demonstrated greater numbers of these small diameter nerve fibers in the facet capsule and synovium than previously identified[19,22]. While the Type IV receptors are the only ones known to be exclusively nociceptive, it is thought that some encapsulated mechanoreceptors may function as nociceptors in the face of excessive joint excursion.

Focusing primarily on the knee joint, electrophysiological studies have demonstrated that altered mechanoreception has a direct effect on reflex activity of muscle groups crossing the joint[27,28]. DeAndrade and Kennedy both found that knee joint effusion produces significant reflex inhibition of the quadriceps muscles, probably because of distension of the posterior capsule[6,21]. Using a dog knee model, O'Connor et al. have demonstrated that deafferentation of peripheral joints may accelerate articular degeneration in joints rendered mechanically unstable[27].

Although the innervation of peripheral joints has been studied extensively, the spinal facet joints have only recently begun to attract the attention of investigators. Freeman and Wyke previously documented the presence of receptors in the posterior elements of the spinal column, but did not comment on the density of the receptor population or the distribution of the different receptor types[11,31]. Giles and others demonstrated nociceptive free nerve endings in capsular tissue of human facets, and reported numerous fine nerves and free nerve endings in the facet synovium[14,15,20]. These investigators failed to identify encapsulated nerve endings in the facet capsular tissue, and focused their attention on the lumbar spine. No one has yet addressed the innervation of other segments of the spine. This study found nerve endings in the capsule of normal human cervical facets consistent with those seen in the articular tissues in the cat, dog, and human[11,24,25,29].

Because the authenticity of "putative receptors" reported in some previous studies has been questioned (the structures illustrated in some reports have resembled vessels and fixation artifacts rather than genuine mechanoreceptors)[7,29], endings seen in this study were not confirmed or classified unless specific criteria were met. If the differentiation between a myelinated axon and a vessel was found to be difficult, review of serial sections usually brought the observer to a branch point in the vessel or a lumen which would make identification possible. If the structure was an axon, following it through serial sections frequently brought the observer to a node of Ranvier, which again confirmed the identification.

The small number of mechanoreceptor endings encountered in the facet capsules suggest that individual receptors have a relatively large receptive field. If this is the case, then damage to even a small part of a capsule might

destroy the innervation to that articular structure. Facet denervation could have important implications for long term joint function. If the relationship between joint afferent innervation and protective muscular reflexes pertains in the cervical spine, then it is most important that surgeons protect the cervical facets during operative procedures such as laminectomy or laminoplasty.

Free nerve endings were identified in the loose areolar tissue, dense connective tissue, and synovial lining of the cervical facets. Other authors have identified Substance P immuno-reactivity in encapsulated receptors of the posterior longitudinal ligament and the periosteum of long bones, suggesting that encapsulated mechanoreceptors also play a role in nociception[18,23]. Whether these receptors are active in pain production in the cervical spine at the extremes of joint motion is not known, but it is likely that the facet capsule and its lining play a role in neck pain in otherwise normal patients.

CONCLUSIONS

Small numbers of encapsulated mechanoreceptors are consistently found in normal human cervical facets, indicating that the mechanical state of the capsule (position, tension, pressure etc.) is under the surveillance of the central nervous system. Whether the information collected concerns joint motion during routine daily activities, or relates to potentially damaging stresses at the extremes of motion is unknown. It is likely that different receptors respond to different states of excursion, or tension, and that these receptors play a role in protective muscular reflexes and in pain caused by excessive motion. While muscular reflexes initiated by these receptors may not be important in routine activities, they may be crucial to protecting the injured or unstable joint from early degenerative disease.

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EFFECTS OF VARIATION OF CEMENT THICKNESS ON BONE AND CEMENT STRESS AT THE TIP OF A FEMORAL IMPLANT

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J.H. Keyak, M.D.

ABSTRACT

With the resurgence of the use of bone cement in total hip arthroplasty, a renewed concern in techniques or designs that may reduce cement fixation failure has arisen. Analysis of the stresses at the tip of the prosthesis may suggest strategies to reduce loosening. Using a three-dimensional finite element model this study analyzed stresses in the bone, cement, and prosthesis near the tip of a femoral component as a function of cement thickness. A section of an idealized circular femoral shaft with implant prosthesis and cement was modeled with loading conditions representing the stance phase of gait.

Increasing cement thickness is predicted to significantly reduce stress in the cement mantle of a femoral implant. Tensile stress is reduced by fifty percent while shear stress is reduced at least twelve percent. Peak tensile stresses occur on the medial side at the tip of the prosthesis in a transverse direction, indicating likelihood of failure due to debonding. Local shear stress peaks also occur at the tip.

Shear stresses in the cement mantle are in the same range as the tensile stresses and must be considered when analyzing the possible modes of failure. However, the mode of failure in shear is complex, and shear strength of the stem-cement interface is unknown at present.

INTRODUCTION

Failure of the cement mantle is a major complication in cemented total hip arthroplasty. Bone cement is generally recognized as the mechanical "weak link" in the longevity of the bone-cement-prosthesis construct. The importance of reducing cement stress in prolonging the service life of the cemented arthroplasty is highlighted by the work of Davies and Harris, which shows the fatigue behavior of cement. Previous studies have shown that during single-leg stance the highest cement strains and stresses occur at the tip of the prosthesis[4,10,5,6]. Increasing the thickness of the cement mantle significantly reduces peak cement strains[4]. Also, the cement strain gradient has been mapped for constant thickness[5]. Cement-metal debonding has been analyzed and shown to increase the likelihood of failure[8].

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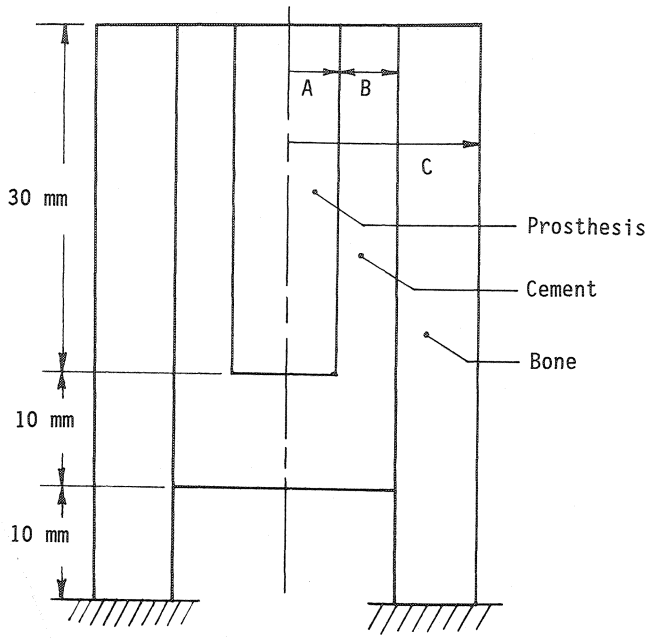
The effects of cemented femoral component arthroplasty on the stress in bone have been studied by finite element analysis[14] and strain gauge studies[11]. These studies indicate that the stress bypasses or is shielded from the proximal femur and is transmitted to the bone in the vicinity of the prosthesis tip. Periosteal cortical hypertrophy has been observed at the tip of the prosthesis in eleven percent of Charnley arthroplasties and is probably a result of this stress bypassing or stress shielding[2].

Increasing the cement thickness seems to be a promising approach to reducing cement stresses and strains, and therefore may decrease the likelihood of failure. It is known that the peak principal strains in the cement near the tip are reduced by increasing the cement thickness. However, the change in the shear and principal stress distributions around the tip with varying cement thickness is unknown.

This study addresses the effects of cement thickness and prosthesis size on cement and bone stress using a three-dimensional finite-element model of an implanted femur near the tip of the prosthesis. Of concern is the optimization of bone and cement stress to improve the longevity of total hip replacement.

MATERIALS AND METHODS

A section of an idealized circular femoral shaft with implanted prosthesis and cement was modeled. Dimensions are shown in Figure 1A, and the model itself is shown in Figure 1B. The section extended from twenty millimeters (mm) distal to the tip of the prosthesis to thirty mm proximal to the tip of the prosthesis for a total length of fifty mm. The outer diameter of the bone was thirty-three mm, and the bone was seven mm thick. The thickness of the cement was varied from two to five mm, and the prosthesis diameter was varied accordingly from nine to fifteen mm. Cement was modeled ten mm below the tip of the prosthesis, and marrow was assumed distal to the cement. The tip of the prosthesis was modeled as a flat end of a cylinder. The distal end of the bone was fixed. The elastic moduli used were 200 GPa for the prosthesis, 15 GPa for the bone, and 2 GPa for the cement, and materials were assumed to be isotropic. The model



	2 mm cement model	5 mm cement model
A	7.5 mm	4.5 mm
B	2.0 mm	5.0 mm
C	16.5 mm	16.5 mm

Figure 1A

Figure 1 Dimensions of the model (A) and a view of the model (B)

consisted of 8,762 nodes and 8,100 8-nodes linear hexahedral elements.

A load corresponding to the stance phase of gait was applied at the superior surface of the section. The distribution of this load was estimated from the results of a previous finite element study[15] of a whole femur with an implant prosthesis. The stress distribution and surface area at a cross-section midway down the femoral shaft of the previous model were used to calculate the compressive and tensile load distribution. In the previous model body weight was assumed to be 1,070 newton's (N), and a compressive load of approximately three times body weight was applied to the femoral head. A tensile load of approximately two times body weight was applied to the greater trochanter. The model was analyzed on a Sun Microsystems SPARC station I computer (Mountain View, CA 94043) using ABAQUS software (Habbitt, Karlsson & Sorenson, Inc., Providence, RI 02906).

RESULTS

Figures 2A and 2B show principal stress distributions along the coronal midplanes for the two mm and five mm cement mantle models. At each node, magnitude and direction of the principal stresses are indicated by vectors. Solid lines indicate compressive stresses while dotted lines indicate tensile stresses. Very high stresses resulting from end effects of the load were found at the proximal

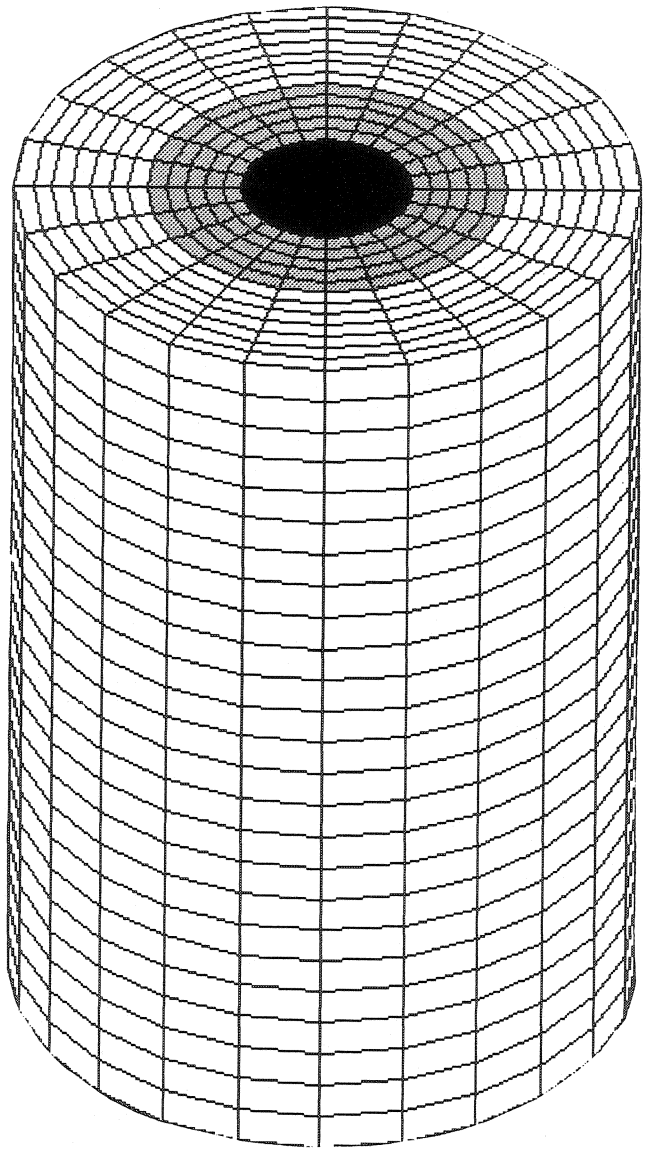


Figure 1B

surface. For this reason six mm of the proximal end is omitted from the figures.

Peak tensile stresses of 7.4 and 3.2 mega pascals (MPa) for the two mm and five mm cement models are found in regions of high transverse tensile stress on the medial side near the prosthesis tip. A peak compressive stress of 6.5 MPa for the two mm cement model is found in a region of high transverse compressive stress on the lateral side near the prosthesis tip. The peak compressive stress in the corresponding region of the five mm cement model is found to be 2.5 MPa, but this stress is only a local peak. The overall peak compressive stress of 6.1 MPa for the five mm cement model is found at the proximal end of the model.

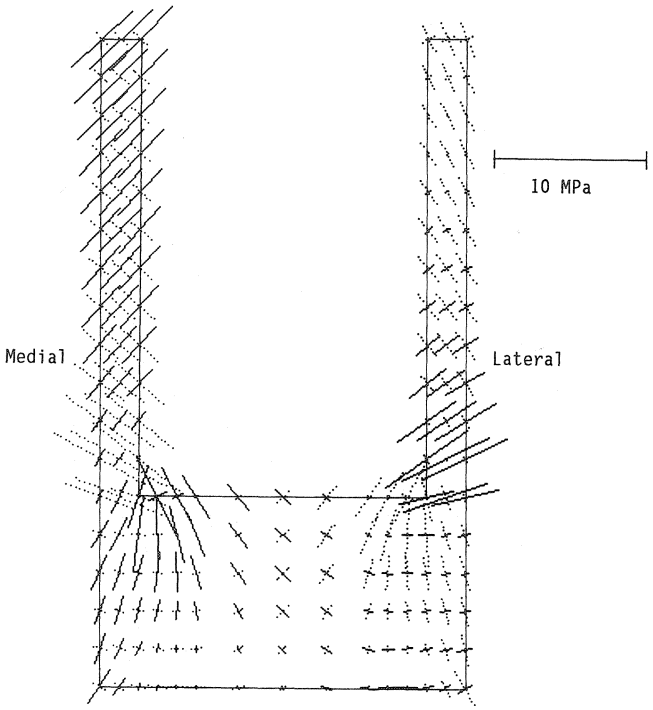


Figure 2A

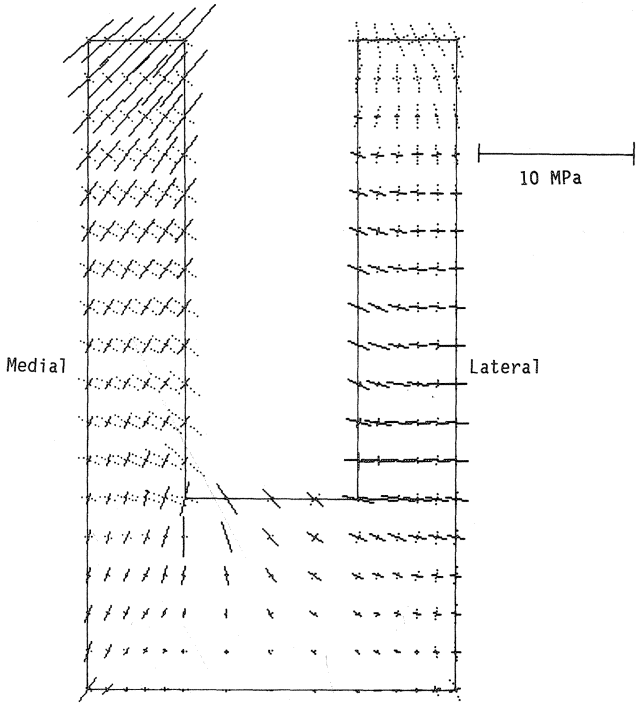


Figure 2B

Figure 2 Principal stress distribution in the coronal plane with a two mm cement mantle (A) and a five mm cement mantle (B).

The stress distributions in the two mm and five mm models are similar, with the stress magnitudes in the five mm model significantly lower. High compressive stresses on the medial side and high tensile stresses on the lateral side are found near the proximal end. These stresses are oriented approximately forty-five degrees from the shaft axis, and decrease distally to a local minimum just proximal to the prosthesis tip. However, just distal to the tip there are regions of high axial compressive stress on the medial side; and in the two mm cement model, high axial tensile stress on the lateral side (a high tensile stress region at the prosthesis tip on the lateral side of the five mm model is not observed). In the two mm model, local peaks in these compressive and tensile stress regions are found to be 4.2 and 6.0 MPa, respectively. In the five mm model, the local peak in the compressive stress region is 2.5 MPa.

In contrast, tensile stresses on the medial side and compressive stresses on the lateral side are low near the proximal end and increase distally, peaking just proximal to the prosthesis tip in regions of high transverse stress. These regions contain the overall peak cement mantle stresses mentioned previously.

Figures 3A and 3B show shear stress distributions along the coronal midplanes for the two mm and five mm models. The vectors are angled for visibility, not to indicate direction. The magnitude of each vector indicates the magnitude of the maximum shear stress. Again, six mm of the proximal end of the model is not shown. The model with two mm cement mantle predicts a peak stress of 4.2 MPa at the tip. In the 5 mm model a peak shear stress of 3.7 MPa occurs at the proximal end, while a local peak shear stress of 2.1 MPa is found at the tip.

DISCUSSION

The region of high axial compressive and tensile stresses found near the tip of the prosthesis on the medial and lateral sides of the cement mantle are in agreement with the strain results of Estok, et. al. The high transverse tensile and compressive stresses found near the tip on the medial and lateral sides of the cement mantle are consistent with the results of Harrigan and Harris. Our results support the finding of Estok, et. al. that a thicker cement mantle significantly reduces cement stresses.

Increasing cement thickness seems an effective way to reduce stress in the bone and cement. Peak tensile stresses and peak shear stresses at the tip are reduced by 50% in the five mm model as compared to the two mm model. It is unclear whether the peak shear stress found at the proximal end in the five mm model is an artifact of the end effects, as the stress does decrease steadily to very low levels in the middle of the shaft. However, the peak shear stress in the 5 mm cement model is still 12% lower than the peak shear stress in the two mm cement model. A larger model must be analyzed to determine the scope of the end effects.

The peak tensile and shear stresses found are all below 8 MPa, significantly lower than reported static values of

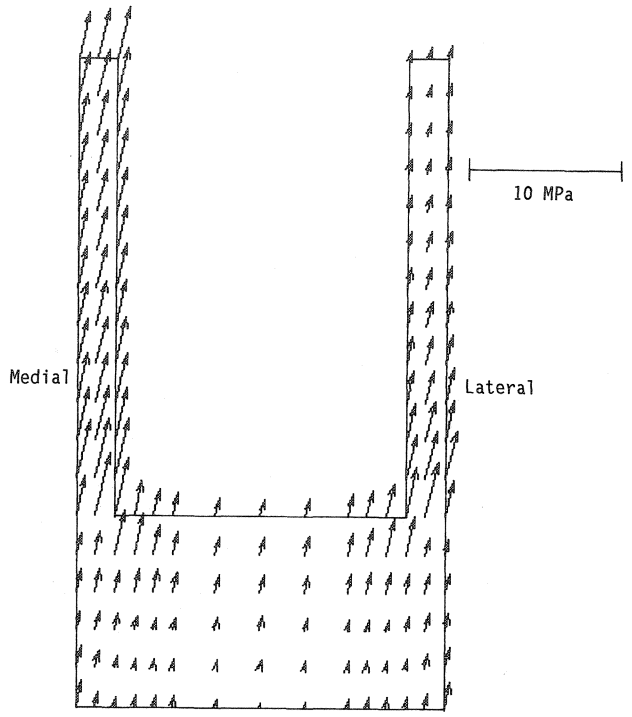


Figure 3A

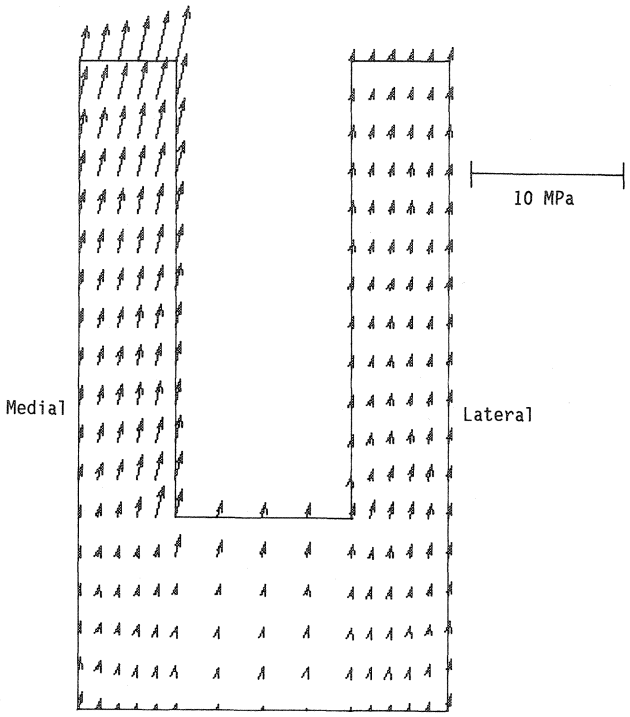


Figure 3B

Figure 3 Shear stress distribution in the coronal plane with a two mm cement mantle (A) and a five mm cement mantle (B).

tensile strength from 21.7 to 48.2 MPa and shear strength from 37 to 41 MPa[13]. However, the peak tensile stress of 3.2 MPa in the five mm cement model is in the range of the tensile fatigue endurance limit of about 4 MPa reported by Davies et al. for uncentrifuged cement. The peak tensile stress of 7.4 MPa in the two mm cement model is well above this value. Our model predicts that centrifuged cement is at risk of early fatigue failure, especially in two mm cement mantle, and uncentrifuged cement would be even more likely to fail in this manner. A higher safety factor for the fatigue is present with a thicker cement mantle and centrifuged cement.

The maximum tensile and shear stresses occur at or very near the stem-cement interface, indicating the likelihood of debonding. Debonding has been proposed as an initiating mechanism in the stem loosening process[9]. Raab et. al. have reported static tensile strength of the dry cement-prosthesis bond of about 7 MPa and a tensile fatigue strength of 2 MPa for cobalt-chromium alloy (assuming ASTM standard F87 surfaces)[7,12]. The maximum tensile stresses of 7.4 and 3.2 MPa for the two mm and five mm cement models support the finding of Harrigan and Harris that debonding at the tip is likely. Since the shear stresses predicted in this study reach levels comparable in magnitude to the normal stresses, shear strength of the prosthesis-cement interface would also be a concern. A clear value for shear strength is not yet given in the literature due to difficulty in interpreting the mixed modes of failure inherent in shear strength testing[7].

It has been shown that a flat prosthesis tip such as that modeled in this study increased stress up to 35% compared to a hemispherical tip[4]. We predict that the high stresses at the edge of the prosthesis tip would be reduced by rounding the tip of the model.

CONCLUSIONS

Increasing cement thickness is predicted to significantly reduce stress in the cement mantle of a femoral implant. Tensile stress is reduced by fifty percent while shear stress is reduced at least twelve percent. Peak tensile stress occurs on the medial side at the tip of the prosthesis, in a transverse direction, indicating likelihood of failure due to debonding. Local shear stress peaks also occur at the tip.

Shear stresses in the cement mantle are in the same range as the tensile stresses and must be considered when analyzing the possible modes of failure. However, the mode of failure in shear is complex and shear strength of the stem-cement interface is unknown at present.

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OSTEONECROSIS OF THE FEMORAL HEAD: CURRENT CONCEPTS AND CONTROVERSIES

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INTRODUCTION

Osteonecrosis (ON) of the hip is a disease in which the living elements of bone in the femoral head die. Although it has been a well-known clinical entity for greater than 100 years, only in the last 20 years has a significant amount of research been devoted to this condition. The interest in ON has produced a recent explosion of literature concerning its pathogenesis, diagnosis, natural history, and treatment. However, this information has done little to clarify current concepts. In fact it has only heightened the controversy due to the variability in approaches to ON and the results of treatment. The purpose of this summary is to highlight past ideas, outline some current concepts, and explore recent controversies regarding osteonecrosis of the femoral head.

HISTORY

A German surgeon named Franz Konig is given credit for the first description of ON of the femoral head in 1888. The German pathologist George Auxhausen described the histology of bone necrosis and was the first to use the term aseptic necrosis to describe an anemic bone infarct. Dallas Phemister, influenced by Auxhausen's work, studied bone necrosis and bone grafting extensively in the 1930's and 1940's. He made tremendous strides in correlating the clinical findings, radiographic changes, and pathology of bone necrosis. Phemister believed in the concept of an ischemic bone infarct, and later reported on the treatment of necrosis by femoral head drilling and tibial bone grafting in 1949. In 1935, Chandler proposed that ON of the femoral head was analogous to a myocardial infarction and termed it "coronary artery disease of the hip"[30]. Based on work from these early pioneers, many investigations have been carried out to determine the pathogenesis, natural history, and treatment of ON of the femoral head. Although our knowledge of etiologic factors, stages, and the natural history in some cases has advanced, little concrete information regarding the actual pathogenesis or best form of treatment has been emerged since these early works.

Etiology and Pathogenesis

Many etiologies and associated factors have been identified in ON of the femoral head. Trauma such as an intracapsular femoral neck fracture or dislocation of the hip can interrupt the blood supply and result in ON. Osteonecrosis is a well-known complication of allograft organ transplantation with steroid administration used for a variety of conditions. Alcohol abuse is one of the most common causes of ON today. Dysbaric phenomena such as Caisson's disease, sickle cell anemia, and Gaucher's disease are well-known, although less common causes of ON of the femoral head. Other possible factors include gout and hyperuricemia, radiation, osteoporosis, hypophosphatemia, hyperparathyroidism, and connective tissue diseases. Often, many factors are present at one time, such as a patient with a connective tissue disease treated with corticosteroids who develops renal failure and undergoes renal transplantation. In addition, up to one-third of all cases may be truly idiopathic, without an identifiable cause, associated factor, or clear pathogenesis.

Much has been written, but little has been learned about the pathogenesis of most cases of ON of the femoral head. It seems plausible that the blood supply would be disrupted in displaced femoral neck fractures and hip dislocations, with subsequent development of ON. However, there are cases of certain complete circulatory disruption which do not lead to ON, and some cases where the blood supply should theoretically remain intact, yet ON develops.

In sickle cell disease and dysbaric phenomena, a thromboembolic mechanism is thought to occur. In sickle disease this would result from sludging of red cells, and in Caisson's disease this represents nitrogen bubbles. In infiltrative disorders such as Gaucher's disease, it is thought that the circulation is encroached with subsequent compromise of nutrition to the osteocytes and marrow.

Intravascular fat has been proposed as a cause of ON in steroid treated patients. This fat has been demonstrated histologically in animal experiments and in humans on steroids. However, no documentation of histologic necrosis has been found despite extensive fat emboli in these studies. Humans treated with steroids have been found to have fat emboli at autopsy, but this has not been correlated with necrosis[10,14]. Spencer, et al. suggested necrosis might occur from a direct cytotoxic affect of steroids on osteocytes or the interference with interosseous microcirculation in the subchondral bone. These conclusions were drawn from abnormalities in the microcirculation found in autopsies of patients on high dose corticosteroids[43,44]. Fat emboli have also been implicated as the cause of alcohol-associated ON.

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Arlot, et al. proposed that osteoporosis or osteomalacia may underlie ON in almost all patients, based on histomorphometry of iliac biopsies from patients with a variety of diagnoses[3]. Andresen and Nielsen believed that an abnormal status of the bone [i.e. osteoporosis] at the time of organ transplantation was important in the subsequent development of ON[2]. Elmstedt proposed that microfractures in osteopenic bone produced vascular changes and eventual ON[12]. Boettcher, et al. presented laboratory data supporting a coagulopathic state as the underlying cause in a series of patients with nontraumatic ON[6]. Saito, et al. supported a theory of single episodes of infarctions from the interruption of a segmental blood supply as the cause of necrosis in cases they called minimal osteonecrosis (MON)[40]. Finally, Ficat and Hungerford believe that intraosseous pressure elevation interferes with blood flow, analogous to a Starling resistor. They liken the femoral head to a compartment and the elevated pressure to a compartmental syndrome of bone[13,20].

It is clear that no theory on pathogenesis is widely accepted, nor can one theory explain all the causes of ON. Any theory must take into account the 80 percent incidence of bilateral disease in patients and the variety of clinical and radiographic presentations. It must explain the multitude of factors associated with the disease process, and the final common pathway of bone necrosis followed by repair and often collapse. Importantly, many surgical procedures have been adopted to address proposed pathogenetic mechanisms, often with little scientific foundation. Theoretically any intervention should address a proven pathogenesis to be successful.

Diagnosis

The diagnosis of ON is made in a patient at high risk who has typical radiographic findings. However, there is no "gold standard" in the diagnosis of early lesions. The history and physical exam are often non-specific, and radiographs are negative in early disease. Magnetic resonance imaging has recently emerged as the most sensitive, specific, and widely used diagnostic tool in these cases. In most reports, MRI can diagnose very early lesions with a greater than 90 percent specificity and sensitivity based on histology or eventual progression[4,5,18,33,34,38,44]. However, one study has not verified this degree of accuracy in early diagnosis, and debate exists regarding the usefulness of MRI in staging lesions, predicting progression, or following response to treatment[9,15,33,52,55]. With further experience, the accuracy should increase and the cost decrease. The added benefits of convenience and lack of ionizing radiation will solidify it as the diagnostic test of choice for suspected early ON.

Previously, plain radiographs, bone scans, CT scans, and tomograms have been used to diagnose osteonecrotic lesions. The accuracy of these tests done alone or in combination has not equaled MRI in comparative studies[33,34]. However, bone scans are useful in early diagnosis. Plain radiographs have a role in diagnosing later stages and in following progression. Other tests may have a role in selective cases and in preoperative planning. The functional exploration of bone (FEB) is a procedure that measures intraosseous pressure, employs venography, and includes a core biopsy (described later). Popularized and described as highly accurate by Ficat, its role in diagnosis has recently been questioned[33].

As part of a prospective protocol using a multimodality approach to the diagnosis in suspicious cases, Stulberg, et al. found MRI better than bone scans, SPECT scans or functional explorations of bone to diagnose asymptomatic lesions[49]. However, bone scanning was the most cost effective method for all cases. It had comparable sensitivity, specificity and predictive value and was recommended as the preferred test in symptomatic cases. The functional exploration of bone had the lowest sensitivity and specificity and was not well-tolerated by patients.

It is generally agreed that early diagnosis is essential in ON. Plain radiographs can be used to follow lesions once they appear radiographically, but this is relatively late in the course. Magnetic resonance imaging has superceded previous tests as the most accurate method of diagnosing lesions early and should be used in all symptomatic patients considered at high risk. Whether or not MRI should be used as a screening tool in all patients considered at high risk is debatable[9].

Classification

Three accepted classifications of ON have been described in the literature. These are shown in table form (see Tables I, II, and III). The Ficat system is the most widely accepted and seems to delineate the usual course of progression[13]. Marcus and Enneking's system does not include preradiographic phases with or without symptoms[28]. The Steinberg classification includes a stage 0 which he describes as a clinically suspicious hip without symptoms, MRI findings or radiographic changes[47].

No study has been done to compare the three staging systems. However, it appears that most patients go through an asymptomatic period followed by a symptomatic and preradiographic period. Therefore, the Ficat classification seems most appropriate.

Natural History

It is generally believed that ON of the femoral head will inexorably progress to collapse and advance to degenerative changes of the hip. This natural history has been demonstrated several times for hips in all radiographic stages[21,25,35,46]. Musso demonstrated that only three of

Table I
FICAT STAGING[28]

Stage	*Clinical*	*Radiographs*	*Bone Scan (MRI*)*
0+	Asymptomatic	Negative	Usually++
I	Pain	Negative	Positive
II	Pain	Mottling cysts	Positive
III	Pain	Collapse	Positive
IV	Pain	Degenerative changes	Positive

+Ficat originally described the four advanced Stages. Hungerford later identified Stage 0.
++Although Ficat believed Core Biopsy was the only method able to diagnose Stage 0, most would agree that the bone scan, and certainly the MRI, would be positive.
*MRI has superceded Bone Scan in the Early Detection of ON.

Table II
STEINBERG STAGING*[47]

Stage	*Criteria*
0	Normal or nondiagnostic radiographs, bone scan, and MRI
I	Normal radiographs, abnormal bone scan, and/or MRI
II	Abnormal radiographs (cystic, sclerotic changes without collapse)
III	Subchondral collapse
IV	Flattening of the femoral head without joint space narrowing or acetabular involvement.
V	Joint narrowing and/or acetabular involvement
VI	Advanced degenerative changes

*Steinberg also advises staging the extent of the lesion by letters A, B. and C depending on size by MRI or x-ray.

Table III
MARCUS STAGING[13]

Stage	*Clinical*	*Radiographs*
I	Asymptomatic	Mottled densities
II	Asymptomatic	Infarcted demarcated by density
III	Pain—mild and intermittent	Crescent sign
IV	Pain with activity	Depression of infarct
V	Pain with activity	Flattening and compression
VI	Pain at rest	Degenerative arthritis

50 hips remained stable at an average follow-up of 16 months[35]. Despite bed rest, crutch walking, non-steroidal anti-inflammatory drugs (NSAID's), and analgesics, 47 out of 50 progressed. Thirty-eight out of 50 either underwent total hip arthroplasty (THA) or had it recommended. Steinberg, et al. presented similar data on 44 out of 48 lesions with radiographic progression[46]. Hungerford and Zizic reported 21 of 22 Ficat Stage II lesions progressed over time with conservative management[21]; all 11 Stage III lesions progressed. Glimcher and Kenzora reported that "following femoral head collapse, conservative measures will not provide symptomatic relief"[16], the majority will require THA. The only study apparently demonstrating resolution of radiographic changes in femoral head osteonecrosis was reported by Andresen and Nielsen. Regression occurred in seven out of 25 lesions, and stabilization in 10 out of 25 femoral heads following renal transplantation. These were all diagnosed in late stages by the radiographic density changes and/or collapse[2].

Osteonecrosis can now be diagnosed earlier with MRI and other studies. It has been assumed that the natural history in these lesions would be as unfavorable as those diagnosed in late radiographic stages. However, the minimal evidence that is available would suggest the contrary. Saito, et al. has described minimal osteonecrosis (MON) of the femoral head as a small and eccentrically localized lesion with clinically benign features[40]. They found MON in 19 hips out of 275 diagnosed with osteonecrosis. Of these 19 cases diagnosed by radiographs, scintigraphy and biopsy, none progressed to collapse or showed any increase in the size of the osteonecrotic lesion over a follow-up period of three to 13 years. Kopecky, et al. reported a prospective study using serial MRI's from the time of renal transplant to 24 months after transplant. Of the 25 hips with apparent ON by MRI, 11 showed regression or disappearance of the lesion without treatment, at an average follow-up of 16 months[23].

Although current recommendations are for early diagnosis and intervention, the actual natural history of early lesions is unknown and may be benign in the majority of cases. Several studies have demonstrated adverse effects of associated factors such as steroids or alcohol abuse[7,27,53]. Most recently, an increased size and location of a lesion in the weight-bearing portion of the femoral head has predicted a poor outcome in both the natural history and in treated cases[47].

TREATMENT

As described earlier, no form of conservative management such as bed rest, protective weight-bearing, analgesics, or NSAID's has proven effective in treating ON of the femoral head[35,46]. Most cases will lead to collapse and advanced degenerative changes; however, the natural history and response to conservative measures of early ON is not known.

The only universally accepted treatment of ON is THA, which is usually reserved for advanced stages. Because the disease most commonly affects young adults, who are

not good candidates for joint arthroplasty, the emphasis has been on procedures to halt progression of early stages. The following is a brief description and reported results of some of these procedures.

Structural Bone Grafting

Phemister first reported the technique of drilling holes and inserting rectangular bone pegs into the femoral head in 1949[37]. He believed that drilling removed dead bone, allowed rapid invasion of a healing response, and prevented fracture and collapse of the head. His anecdotal results on post-traumatic necrosis supported this technique which is still used today in some centers. At the University of Iowa, Bonfiglio and Boettcher modified Phemister's technique and demonstrated an 80 percent satisfactory "healing rate" in approximately 150 patients with traumatic and nontraumatic necrosis of the femoral head[6]. Poor results were attributed to poor technique in half their cases. Later follow-up in 1980 demonstrated continued good results if no or minimal femoral head collapse was present during the index operation[42]. Graft placement into the subchondral cortex was found to be critical to success. Nelson and Clark reviewed the more recent Phemister procedures done at the University of Iowa, with a minimum two year follow-up[36]. Fifty-two percent of patients required THR and between 82 and 95 percent progressed at least one Marcus stage.

At the University of Florida, Marcus, Springfield, and Enneking have supported cortical strut grafting in patients with Stages I and II (Table III)[28,45]. They reported a success rate of up to 90 percent in these early stages. Buckley, et al. recently reported on 20 operatively treated patients with a follow-up of greater than two years, again with a 90 percent success rate[7]. They also attributed failures to improper graft placement, poor surgical indications, continued steroid use, or alcohol abuse.

Core decompression without bone grafting has been the most popular and controversial treatment of ON of the femoral head. Described and popularized by Ficat, the procedure involves removing two separate 6-10 mm core tracks from the femoral head and neck. He believes that the pathogenesis of ON relates to interosseous hypertension, which causes impaired circulation and ischemia, similar to a compression neuropathy or compartment syndrome. As stated before, this is typically done as part of a three part procedure called the functional exploration of bone (FEB): 1) Interosseous pressure measurement (IOP)—a manometer is used to measure the pressure in the intratrochanteric region and femoral head. Resting pressure should be about 20 mm of mercury and greater than 30 mm of mercury is considered abnormal. Five cc's of saline is then injected, and the pressure is measured again. A greater than 10 mm of mercury difference after 5 minutes is considered abnormal. 2) Venography—contrast material is injected into the proximal femur and the venographic appearance assessed. Venostasis and reflux are considered characteristic for ON. 3) Core biopsy—the material removed during coring is assessed histologically for necrosis. Ficat and later Hungerford staged lesions based on results of the FEB, symptoms, and radiographic findings (Table I). They felt that the earliest treatment is best to avoid the complications of advanced disease, and recommend core decompression if the IOP or venography is abnormal[13,20,21]. Ficat has reported 94 percent good or excellent results in Stage I, and 82 percent good or excellent results in Stage II[13].

Ficat's early reports spawned great enthusiasm; however, several surgeons have reported extreme variability of both the success rate and complication rate of this procedure. Hungerford and Zizic reported on both alcoholic patients and patients with lupus treated with core decompression. They strongly supported the use of the FEB as the diagnostic procedure for ON and had results very similar to those of Ficat for halting progression of treated hips. Warner, et al. found that core decompression "prevented" collapse of femoral heads in 15 of 24 patients with Stage I and Stage IIA (which they called sclerotic predominant)[56]. They were unable to prevent progression in lesions beyond Stage II, and they failed to demonstrate a reliable association between elevated IOP, abnormal stress tests, and venography with ON. This and other reports have limited the enthusiasm for using core decompression beyond Stage II. Tooke, et al. reported that core decompression prevented progression of Stage I hips in all of 10 cases, and 15 of 26 Stage II hips with a short term follow-up[53]. They found much worse results if patients continued on steroids.

In the only known study to directly compare nonoperative and operative treatment (core decompression), Stulberg, et al. found a significantly better outcome in Ficat Stage II and III hips that underwent coring than prolonged protective weight-bearing[50]. This study is important for several reasons: 1) Patients were prospectively randomized to either nonoperative or operative treatment. 2) Each stage was randomized separately to allow direct comparison for a given stage. 3) A strict protocol for the operative procedure and method of nonoperative treatment was followed. 4) Results were analyzed with regard to clinical outcome, radiographic progression, and prevention of further procedures. Stage II and III hips that were treated operatively had statistically better results in all three of these categories. The results for Stage I patients were similar, but statistical significance was not achieved. The numbers of Stage 0 and Stage IV patients were not enough to allow statistical validity. Unlike many other reports, no subtrochanteric fractures or other operative complications were found. Although this study provides

the best evidence of efficacy in core decompression, criticisms can be made. The diagnosis of ON was based on a diagnostic protocol set forth by the same authors using a multimodality approach. Although they state the diagnosis of preradiographic stages required two positive tests, the numbers show that the results of these tests (bone scans, MRI, biopsy, etc.) often did not agree. Secondly, radiographic progression occurred in 70 percent of Stage II and all Stage III lesions in the surgically treated group versus 57 percent of Stage II and 30 percent of Stage III lesions in the conservatively treated group. The short minimum follow-up of 18 months (average 27 months) might indicate that the core decompression only temporarily relieves symptoms and delays the ultimate progression of disease. This discrepancy of clinical versus radiographic outcomes has been noted before[13]. Recently, Hungerford reported a 13 year follow-up of patients treated by core decompression with excellent results in the early stages[57]. However, it must be kept in mind that this and most reports of success using core decompression have focused on the earlier stages when the natural history of these lesions is not truly known. In addition, many recent reports have demonstrated poor results and high complication rates using core decompression.

Camp and Colwell showed a 60 percent rate of clinical and radiographic progression of Stage I and II hips followed for an average of 18 months after core decompression[8]. They found the FEB did not add to the accuracy of conventional diagnostic imaging. They also report the highest incidence of subtrochanteric fractures, ten percent. Hopson and Siverhus also found a low success rate in treating early lesions with core decompression[19]. In their series of 21 predominantly steroid-treated patients, all but one had histologic confirmation of necrosis and only 40 percent failed to progress or require a second operation. They had only one peri-operative fracture (4.7 percent). Learmonth, et al. found that clinical or radiographic progression occurred in 34 out of 41 hips in Stage I and Stage II[24]. Finally, Seiler, et al. had similar disappointing results in Stage I and Stage II disease with an average follow-up of only 12 months[41]. They concluded, as had the previous authors, that core decompression had an unexpectedly low success rate for halting progression, and in fact may not improve the natural history of the disease whatsoever. Additionally, these studies reported a significant morbidity, specifically peri-operative fracture through the core track.

ELECTRICAL STIMULATION

Steinberg, et al. has advocated the use of electrical stimulation in combination with core decompression and cancellous bone grafting. In their most recent article, they reported significantly improved results with coring, grafting, and electrical stimulation compared with coring and grafting alone or non-surgical treatment in unmatched, nonoperated controls[46-48]. Although the majority of surgically treated hips had radiographic progression, they did not clinically deteriorate. The follow-up of only 44 months may be too short to judge long-term success. Aaron, et al. reported that electrical stimulation was superior to the natural history or core decompression for both Ficat Stage II and III. Other studies have proposed a beneficial effect of electrical stimulation, but the data is confused by combinations of procedures and a lack of controls. A controlled, longer term follow-up is needed to assess the effectiveness of electrical stimulation in halting progression of ON.

Osteotomy

Osteotomies have many potential advantages in treating ON: 1) They preserve the hip joint by removing the necrotic segment from the weight-bearing forces. 2) They induce hypervascularity and may have a role in relieving interosseous hypertension.

In a retrospective review comparing "joint preservation operations", Saito found poor results with core decompression with or without cancellous bone grafting at average follow-up of four years[39]. Results were somewhat better with osteotomies, but only for those patients with localized lesions.

D'Aubigne' et al. reported using either varus or varus-rotational osteotomies in 56 patients, achieving satisfactory results in 47[11]. These authors recommended an osteotomy for the younger patient with minimal or no collapse and lesions "without marked extension". Maistrelli, et al. reported 106 osteotomies followed for a mean of 8.2 years (81 valgus and 25 varus), achieving a 58 percent good or excellent rating[27]. Better results were found in younger non-alcoholic patients with early necrosis that was limited in size. Sugioka devised and popularized the transtrochanteric rotational osteotomy that rotates the femoral head up to 90° (usually anteriorly)[51]. He found minimal complications and excellent clinical results. Other authors have found the technique very demanding, with high complication rates and only fair results[29,39,54].

Some general comments can be made concerning osteotomies for treating ON. The lesion must be small and rotated out of the weight bearing area. This treatment should be reserved for younger patients; and results have been fair to good with a tendency to deteriorate with time.[17]

Vascularized Bone Grafting

Meyers has reported excellent results in treating patients with Marcus Stage I and Stage II ON using a vascularized quadratus femoris muscle pedicle bone graft from the posterior femur[31,32]. Poor results followed

treated of Stage III or greater ON. Lee and Rehmatullah have reported on ten patients treated with a similar procedure. Seventy percent of patients with Marcus I and II stages had good results[26]. A long postoperative recovery period is required after this extensive procedure. Long-term follow-up will be necessary to determine if a vascularized graft offers an advantage over structural or cancellous grafting, or core decompression alone.

From the previous discussion, it seems clear that no surgical procedure is completely satisfactory in the treatment of ON. Core decompression has become the most popular and controversial procedure, but its current role in early treatment is uncertain due to the disparity of reported results. Structural bone, cancellous bone, and vascularized bone grafting procedures are generally advocated by those who report their use but confirmatory studies are lacking. Osteotomies have limited indications, and at best fair success. Total joint arthroplasty is indicated for advanced symptomatic lesions, but should be avoided as long as possible in young patients.

SUMMARY

Despite many investigations into ON of the femoral head, many issues remain unresolved. The pathogenesis in most cases is only speculative and may involve intravascular factors such as microemboli or extravascular factors such as increased interosseous pressure. MRI has emerged as the diagnostic test of choice for suspected early lesions, and radiographs should be used to diagnose and follow advanced lesions. Bone scanning can be useful for early diagnosis and CT scanning or tomography may help plan surgical procedures. The role of the functional exploration of bone is controversial. The natural history of early lesions is unknown; this makes it difficult to evaluate results of treatment. Radiographic ON will usually progress to collapse and arthrosis if treated nonoperatively. The role of core decompression or other joint preserving operations to prevent collapse is controversial, since the reports of success and complication rates have been extremely variable. These procedures are ineffective if used after radiographic collapse. Total hip replacement is the only satisfactory treatment for advanced symptomatic stages, but is relatively contraindicated in young active patients.

Further research is needed to assess the natural history of early ON and evaluate the role of surgery in preventing progression.

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LATE RUPTURE OF THE POSTERIOR CRUCIATE LIGAMENT AFTER TOTAL KNEE REPLACEMENT

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ABSTRACT

To our knowledge there have been no reports of late rupture of the posterior cruciate ligament (PCL) as a cause of instability in PCL-retaining total knee prostheses. In our experience of 150 total knee replacements using PCL-retaining prosthesis, three cases (2.0%) of late rupture of the posterior cruciate ligament have occurred, each leading to chronic instability, disabling pain, and revision arthroplasty. In each case rupture of the posterior cruciate ligament was confirmed at the time of revision arthroplasty. The use of a more constrained prosthesis led to a successful outcome in each case.

INTRODUCTION

The major goal of total knee arthroplasty (TKA) is to provide a stable, painless knee with a functional range of motion. The reported overall failure rate has ranged from 5% to 13.5% and is secondary to component loosening, infection, or instability. Failure due to instability usually arises early in the post-operative course, and stems from pre-existing instability, soft tissue imbalance, or patellar tracking problems. Much attention has been directed to more common problems such as loosening and infection. A relatively rare cause of instability is late rupture of one or more ligaments of the knee. To our knowledge there have been no reports of instability from rupture of the posterior cruciate ligament (PCL) in PCL-retaining total knee prostheses.

In our experience of 150 total knee replacements using a posterior cruciate retaining prosthesis, three cases (2.0%) of late rupture of the PCL have occurred, each leading to severe instability and revision arthroplasty using a more constrained prosthesis. In each case, rupture of the PCL was confirmed at the time of revision surgery.

CASE REPORTS

Case #1

A seventy-two year old male with psoriatic arthritis had staged, bilateral, cemented total knee replacements with the Whiteside total knee prosthesis (Dow Corning, Arlington TN.). At three years, both knees were stable and painless with a range of motion of 0 to 90 degrees (Figure 1-A).

Four years postoperatively, the patient developed the insidious onset of vague posterior right knee pain. There was no history of trauma or infection of the knee. Physical examination revealed a mild effusion. Posterior drawer and tibial sag signs could be elicited. This posterior subluxation was manually correctable. These findings had not been present on previous examinations. In addition, 25 degrees of valgus laxity with the knee in extension was now present. Radiographic examination did not reveal any evidence of loosening of the components, but the lateral projection demonstrated marked posterior subluxation of the knee (Figure 1-B).

A nine month trial of bracing and quadriceps strengthening failed due to increasing knee pain and instability. The patient eventually underwent a revision arthroplasty with a posterior stabilized prosthesis (Kinemax, Howmedica, Rutherford, N.J.). At surgery the PCL was found to be ruptured in its midportion. Two years after revision, the patient has a stable, painless knee with a range of motion of 0 to 110 degrees (Figure 1-C).

Case #2

A seventy-nine year old Peruvian female with severe tricompartmental osteoarthritis of both knees underwent staged bilateral total knee replacements using the Porous Coated Anatomic (PCA) total knee prosthesis (Howmedica, Rutherford, N. J.).

One year after replacement of the left knee, the patient developed an intermittent discomfort in the popliteal and posterolateral calf regions. The pain was exacerbated by ambulation and extension of the knee, and relieved by rest with the knee in flexion. There was no history of locking, giving way, or instability. Examination revealed no evidence of instability. The range of motion of the knee was 0 to 90 degrees with pain on full extension. Radiographs revealed well-placed components without signs of loosening or malalignment. The posterior knee discomfort continued intermittently for over a year.

Approximately two and one-half years postoperatively, the patient slipped and sustained a mild hyperextension injury of the left knee. She experienced an immediate exacerbation of her knee pain and was unable to flex her

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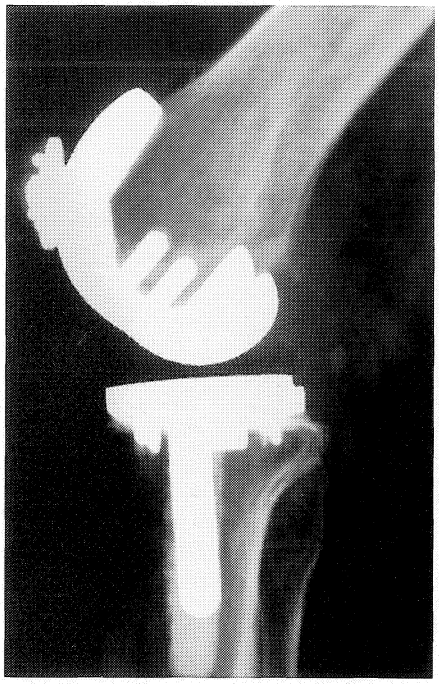

Figure 1-A: Three years postoperatively a lateral radiograph showed no evidence of posterior subluxation. Clinically the patient had a stable, painless knee.

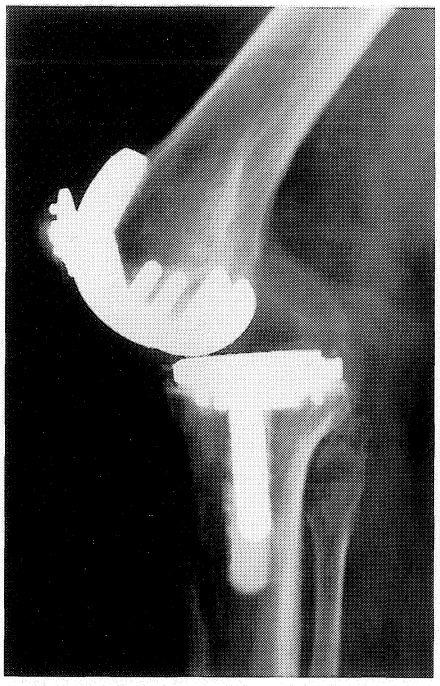

Figure 1-B: At four years he developed pain and instability, and a lateral radiograph revealed significant posterior subluxation of the knee.

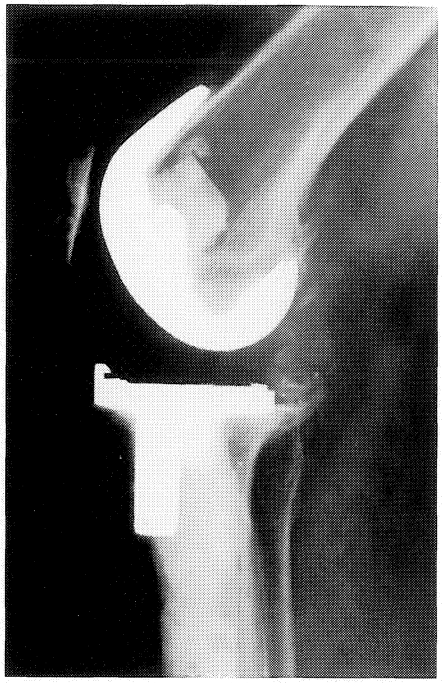

Figure 1-C: Lateral radiograph after revision arthroplasty with a posterior stabilized prosthesis.

knee from a position of full extension. Physical examination revealed a definite posterior sag sign and spontaneous posterior subluxation of the knee with attempted flexion. Radiographs confirmed new posterior subluxation but did not show any sign of component breakage or loosening.

The patient was treated with a cast-brace with motion restricted from 0 to 45 degrees. However, after two months, she developed a spontaneous complete foot drop and subjective sensory loss in the common peroneal nerve distribution. Electromyographic studies confirmed denervation of the tibialis anterior and peroneus longus muscles. Nerve conduction tests showed a partial block of the common peroneal nerve at the knee with evidence of mild axonal degeneration. The cast-brace was discontinued, as it was apparent that continued posterior subluxation was taking place despite the use of a brace.

During revision arthroplasty intraoperative examination confirmed the presence of a complete midsubstance PCL tear with posterior subluxation of the tibia. Although all of the components were well fixed, there was substantial wear of the anterior aspect of the polyethylene tibial insert. The components were replaced with a posterior stabilized prosthesis (Kinemax, Howmedica, Rutherford N.J.). Two years after revision, the patient had a range of motion from 0 to 90 degrees without instability or discomfort. The peroneal nerve palsy completely resolved after several months.

Case #3

A sixty-two year old white male suffered from gouty arthritis and severe degenerative joint disease of both knees. He had undergone a right arthroscopic debridement and chondroplasty five years earlier, and an arthroscopic synovectomy and intravenous antibiotic treatment for a septic knee (Beta haemolytic Streptococcus) three years earlier. The infection resolved, but the degenerative arthritis continued to progress until the pain in his right knee became unbearable and limited his activities of daily living.

After two sterile knee joint aspirations, the patient underwent a cemented right total knee replacement with a PCA prosthesis. Intraoperative cultures and tissue for pathology were taken at the time of arthroplasty. These proved to be negative for infection.

Although the early postoperative course was uneventful, a fascial dehiscence developed and was repaired six weeks postoperatively. Lateral subluxation of the patellofemoral joint was identified and corrected by lateral retinacular release and medial capsular plication four months postoperatively. At surgery, the posterior cruciate ligament was found to be normal. Just prior to the six month postoperative visit, the patient developed painful instability of the knee. Clinical examination disclosed significant posterior drawer and sag signs, but no other instability. Radiographic examination revealed no evidence of component loosening or malposition, however posterior

subluxation of the knee joint was noted. The patient subsequently underwent revision arthroplasty with insertion of a thicker, more constrained polyethylene tibial insert. Inspection of the PCL at surgery revealed a complete midsubtance rupture. Intraoperative cultures were negative at the time of revision. After placement of the new tibial implant, the knee was brought through a range of motion and found to be stable from full extension to a least 90 degrees of flexion. Postoperatively the knee was placed in a bulky dressing and knee immobilizer. Continuous passive motion was started on the fourth postoperative day. However, on the fifth day as he rolled over in bed, the patient experienced an uncomfortable locking of the operated knee. Clinical examination demonstrated that a posterior subluxation had occurred. The subluxation was reduced by gentle longitudinal and anterior traction of the tibia. The subluxation became a recurrent problem over the next several days and the limb was placed in a cylinder cast for six weeks. One year postoperatively, the knee was stable and demonstrated a range of motion of 20 to 80 degrees.

DISCUSSION

Subluxation and dislocation after TKA are relatively rare[1,2,3,5,7,10,12,13]. Bryan and Peterson (1973 and 1979) reported on the postoperative complications of 450 polycentric knee arthroplasties. Instability was second only to infection as a cause of failure in the three year follow-up period. A total of seven knees were unstable (1.6 percent), six of which presented within the first three months after operation. Six anterior subluxations occurred, and at least two required operative revision. Only one posterior subluxation of the knee was observed. The authors felt posterior subluxation resulted because all the ligaments had been cut to gain motion in a locked knee. The knee was eventually fused.

Skolnick et al (1975) reported on 119 Geometric total knee arthroplasties. Dislocation occurred in one case of advanced rheumatoid arthritis with complete absence of the cruciate ligaments. The patient was able to voluntarily displace the tibia posteriorly and medially by contracting the hamstring muscles with the knee partially flexed. However this patient denied instability. Skolnick et al (1976) later reported a dislocation rate of 1.4% after 500 total knee replacements using the polycentric prosthesis. Five of the dislocations were anterior, one was posterior and one was lateral. Three of the seven dislocating knees required more surgery.

Insall and coworkers (1979) identified six subluxations in their review of 461 total condylar knee arthroplasties. All cases were posterior subluxations and occurred during knee flexion. In two knees this represented only a minor inconvenience and both knees had a good rating. However, in four knees, the symptoms resulted in a poor rating; two of these four were eventually revised to a more stabilized prosthesis.

Cameron and Hunter (1982) reviewed the outcome of 700 total knee arthroplasties and found an overall failure rate of 13.4 percent. Late rupture of the medial collateral ligament occurred in seven (1.0 percent), and lateral subluxation of the tibia on the femur occurred in five knees (0.7 percent). Rotational subluxation occurred in three knees (0.4 percent) and was felt to be due to rotational malposition of the tibial component. Instability in the anteroposterior plane was found in two knees (0.3 percent), but no details concerning the specific type of prosthesis, the symptomatology or the etiology were given.

Interestingly, Galinat and coworkers (1988) reported the dislocation of two posterior stabilized total knee arthroplasties. Both patients had valgus deformities which required extensive release of the contracted lateral soft tissues. The dislocations occurred with slight flexion and external rotation. The prosthetic tibial spine had become locked posterior to the femoral cam. Sidney and colleagues (1989) have also reported two cases of posterior dislocation of the constrained Insall-Burstein Total Condylar III Prosthesis.

The three patients reported in our series all had stable knee arthroplasties with a functional range of motion prior to the occurrence of posterior subluxation. Immediately prior to the episodes of subluxation, two of the three patients complained of a vague ache in the popliteal area. Whether this was due to stretching of the PCL or posterior capsule, or early subluxation of the knee is unknown. In the second case, the subluxation probably stretched the peroneal nerve, leading to the neuropraxia. Fortunately, this resolved when mechanical stability of the knee was re-established with a posterior stabilized prosthesis. The third patient had a difficult postoperative course requiring further operative procedures on the knee. The PCL may have been injured during these subsequent procedures.

The etiology of PCL rupture after TKA is unknown. Interestingly, in all three cases the PCL had sustained a midsubstance tear. This may be related to compromised vascularity of the ligament after surgery or to inadvertent injury during the surgical procedure.

The posterior cruciate ligament is larger and roughly twice as strong as the anterior cruciate ligament. It is more vertically oriented and forms the axis around which rotation of the knee occurs. Its most important function occurs in flexion where it provides rotational stability and prevents posterior displacement[6,11]. Noyes et al (1974 and 1976) have reported that the PCL accounts for 89 percent of the resistance to posterior translation of the tibia on the femur. The PCL also acts to check hyperex-

tension only after the anterior cruciate ligament (ACL) has been ruptured or surgically removed. Therefore, in ACL-sacrificing total knee prostheses, the PCL must endure increased stress in flexion as well as in extension. This may lead to degenerative changes and eventual rupture of the ligament.

The vascularity of the PCL has not been studied as extensively as that of the ACL. The blood supply is classically attributed to vessels entering the intercondylar notch near the femoral attachment, and intraarticular capsular vessels near the tibial attachment[11]. There may be a significant contribution from vessels in the soft tissues and synovium which envelop the ligament's intraarticular portion. If the blood supply to the PCL is disrupted during a surgical procedure, rupture of the ligament may result.

Finally, the PCL may be damaged inadvertently by the oscillating saw, osteotomes or other sharp surgical instruments.

These cases illustrate late rupture of the PCL following total knee arthroplasty. In our experience, conservative treatment such as bracing and physical therapy has ended in failure. Surgical revision to a more stabilized prosthesis has been successful.

SUMMARY

To our knowledge there have been no reports of late rupture of the posterior cruciate ligament as a cause of instability in PCL-retaining total knee prostheses. In our experience this has occurred in 2.0% of cases and has led to severe instability. In each case, the PCL had sustained a midsubstance rupture. There was no associated loosening or malalignment of the prostheses. Revision to a more stabilized prosthesis has led to a successful outcome.

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THE ENIGMATIC DIAGNOSIS OF POSTERIOR TIBIALIS TENDON RUPTURE

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Michael E. Pfister, M.D.

ABSTRACT

Posterior tibialis tendon rupture is a diagnosis that is often missed. This is thought to be secondary to nonspecific clinical findings and the lack of any laboratory or radiographic test to reliably confirm the diagnosis. We report sixteen cases of surgically confirmed posterior tibialis tendon rupture. Based on our review of these patients, the diagnosis of posterior tibialis tendon rupture should be strongly suspected in the adult patient presenting with a history of a twisting ankle injury and generalized medial ankle pain and swelling. A flexible, asymmetric pes planus and forefoot pronation deformity with absence of posterior tibialis tendon function on manual testing is seen on examination. This is associated with loss of ipsilateral heel inversion on bilateral heel rise. The patient is usually unable to perform ipsilateral single leg heel rise and has less severe pes planus of the contralateral foot. This study reviews the presentation, pathophysiology, diagnosis, and treatment of posterior tibialis tendon rupture.

INTRODUCTION

Injuries of the foot and ankle are among the most common activity related injuries in every age group. Posterior tibialis tendon rupture is seen almost exclusively in the adult population and can be seen with traumatic ankle injury. It is however, a diagnosis often missed. In 1985, Mann and Thompson reviewed seventeen cases of posterior tibialis tendon rupture in which only thirteen reported a specific antecedent injury to the affected ankle[14]. The average patient age was fifty-seven years. Each had seen at least one previous physician, and only two had been correctly diagnosed. Most sought initial emergency care several weeks after the injury. The average time to presentation and diagnosis after onset of symptoms was 43 months. Furthermore, Woods and Leach (1991) pointed out that "the key" to successful treatment is early diagnosis[23]. When the diagnosis is missed or significantly delayed, a severe pes planus and pronation deformity can often develop, with degenerative change in the subtalar and midtarsal joints.

The difficulty in clinical diagnosis lies in the nonspecific findings in the acute setting. Patients with complete and partial tendon ruptures, as well as patients with stenosing tenosynovitis, have been reported to present similarly[6,9,13,15,21]. Patients with acute rupture have diffuse medial ankle pain that is usually misdiagnosed as an ankle sprain. Radiographic analyses such as magnetic resonance imaging (MRI) are often helpful, but currently not reliably diagnostic[1,4,5]. The picture unfortunately clears only after the resulting deformity progresses. Typically the pain is unresolved and a progressive unilateral pes planus with forefoot pronation deformity occurs[2,4,6,7,9,10,11,13,14,15,21,23]. Later, lateral pain may develop secondary to an impingement deformity[10].

The purpose of this study is to investigate the diagnostic criteria identifying posterior tibialis tendon rupture, review the likely pathophysiology, and to report the findings and surgical treatment in sixteen patients treated by the senior author (R.E.M.)

MATERIALS AND METHODS

Sixteen patients with surgically confirmed absence of posterior tibialis tendon function were treated between 1987 and 1990 in the Foot and Ankle Division of University Hospitals of Cleveland (Table 1). There were five men and eleven women, with an average age of fifty-five years (range: 35-67 years). The right lower extremity was involved in ten, and the left in six patients.

The time from onset of symptoms to initial presentation ranged from immediate to years, with all but four patients being seen within six months of symptoms. However, the time from onset until accurate clinical diagnosis averaged nineteen months (range: 3-50 months). On average, two physicians were consulted before the accurate diagnosis was made. Only in two cases were other physicians not seen previously (cases 1, 5). Most patients had previous radiographic evaluations of the affected ankle and foot. One patient underwent aspiration of the ankle joint and blood tests for infection and arthritis (case 3). Another had computerized tomography (CT) and bone scans (case 6), and another had MRI prior to presentation (case 12). Previous diagnoses were most commonly ligamentous sprains, while others included posterior tibialis tendonitis, gouty arthritis, plantar fasciitis, anterior tibialis tendonitis, degenerative arthritis, rheumatoid arthritis, and pes pla-

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TABLE 1
Preoperative Death

Case	Age,Sex	Associated Problems*	Trauma	Time to Presentation**	No. of Previous Physicians***	Previous Diagnoses	Previous Treatment
1(DB)	60,F	Lumbar Fusion,Cervical Discectomy/Fusion,	Twisted Ankle (Felt Pop)	3 months	0	PT Rupture	Orthotic,NSAID,PT
2(RB)	38,M		Twisted Ankle (volleyball)	18 months	3	Ankle Sprain	Orthotic,NSAID,PT, Air Splint
3(PC)	54,F	OA (L knee)	None	36 months	1	Gout,Plantar Fasciitis	Orthotic,NSAID
4(PC)	64,F		Twisted Ankle (Felt Pop)	17 months	2	Ankle Sprain	Orthotic,NSAID,PT, Air Splint
5(ZAD)	61,F	Hypothyroidism	None	2 months	0	PT Rupture	Orthotic,NSAID,PT
6(BG)	41,F	Idiopathic Scoliosis	Twisted Ankle (aerobics)	10 months	3	Ankle Sprain	Orthotic,NSAID, Air Splint
7(SG)	54,F	Mild Tarsal Tunnel Syndrome,Obesity	Twisted Ankle	30 months	4	Ankle Sprain,Anterior Tibialis Tendinitis,Achilles Tendinitis	Orthotic,NSAID,PT, Steroid Injetion
8(DH)	61,F		None	50 months	2	Pes Planus with Posterior Tibialis Tendinitis,Ankle Sprain	Orthotic, NSAID
9(JM)	60,F	Obesity	None	48 months	2	DJD	Orthotic,NSAID
10(GM)	60,M	Gout	Twisted Ankle	24 months	2	Ankle Sprain	Orthotic, NSAID
11(JN)	52,M		Twisted Ankle	8 months	1	Ankle Sprain	NSAID
12(ER)	53,F		Twisted Ankle (Tennis)	4 months	3	Ankle Sprain,Probable PT Rupture	Orthotic, NSAID
13(ES)	67,F		Twisted Ankle	12 months	2	Ankle Sprain	Orthotic, NSAID,Air Splint
14(DT)	54,F	RA,Obesity, Hypothyroidism	Twisted Ankle	24 months	1	RA	Orthotic,NSAID,Gold
15(DV)	63,M	Psoriatic Arthritis, Asthma,Hypothyroidism	Twisted Ankle	3 months	1	Ankle Sprain,Tendinitis	Orthotic, NSAID,Air Splint,Steroid Inj.
16(WW)	35,M	Obesity	Twisted Ankle (felt pop)	20 months	2	Ankle Sprain,Pes Planus,Posterior Tibialis Tendinitis	Orthotic, NSAID,Air Splint,Steroid Inj.

*Obesity was considered as a weight > 200 lbs.
**Time to presentation is the time in months from onset of symptoms until seen by R.E.M..
***Both case 1 and 5 were initially seen by R.E.M. and both were initially treated conservatively.

nus. The correct initial diagnosis of posterior tibialis tendon rupture by an outside physician was made in only one case (case 12).

Upon presentation to us, eleven patients reported an antecedent twisting injury to the ankle. All but two patients noted diffuse, poorly localized pain in the medial hindfoot. The remaining two patients reported pain laterally. Three noted both medial and lateral ankle and foot pain. Medial swelling about the foot and ankle was reported by fifteen patients. An asymmetric pes planus and forefoot pronation was described in six patients. Ten reported to have noticed a change in gait secondary to pain and deformity.

On physical examination fifteen patients had slight asymmetric forefoot pronation. Fourteen had an asymmetric pes planus in the affected limb. Ten patients had tenderness to palpation over the posterior tibialis tendon behind the medial malleolus. Posterior tibialis tendon function was abnormal in all cases. Fifteen patients could not perform the single leg heel rise[14]. All patients had full mobility across the ankle, subtalar, and midtarsal joints. All patients demonstrated loss of ipsilateral heel inversion on bilateral toe rise.

All patients had AP and lateral roentgenograms taken prior to treatment. Nine had an abnormal sag in the talonavicular joint. One patient had a bony avulsion noted while all other patients had normal studies (Figure 1).

On surgical exploration (Table 2), fourteen patients were found to have complete longitudinal and complex tears of the posterior tibialis tendon. All patients ruptured within six centimeters of the insertion, the majority (ten patients) between two and three centimeters. A large amount of effusion was uniformly noted upon entering the synovial capsule. In each case there was no elasticity to the posterior tibialis muscle, and scar formation around the ruptured tendon was present.

Surgical treatment consisted of tendon transfer utilizing the flexor digitorum longus. In each case the flexor digitorum longus was detached distal to the knot of Henry and the proximal end was re-routed along the previous path of the posterior tibialis tendon. Special care was taken to preserve at least three to four centimeters of the posterior tibialis flexor retinaculum. After passing the flexor digitorum longus under the retinaculum, the tendon was passed inferior to superior through a drill hole in the navicular. It was sutured back onto itself while the foot was in maximum equinus and varus. The distal end of the

flexor digitorum longus was transferred to the flexor hallucis longus. In every case microscopic pathologic examination revealed degenerative fibrotendinous and fibrocartilaginous tissue. Two patients were noted to have chronic proliferative synovitis (case 3, 11).

One patient (case 12) had a nonfunctional tendon with longitudinal tears within the tendon and stenosis at the retinaculum. Degenerative changes in the tendon were noted at the stenotic area. There was good elasticity to the posterior tibialis muscle and the tendon was intact. A mass (2.0 x 1.5 x 1.0 centimeters) was noted at the insertion of the tendon. The mass was excised and the patient underwent tenolysis of the posterior tibialis tendon, with debridement of the interstitial tears. On pathologic review this patient was noted to have had an angioleiomyoma, and chronic proliferative synovitis.

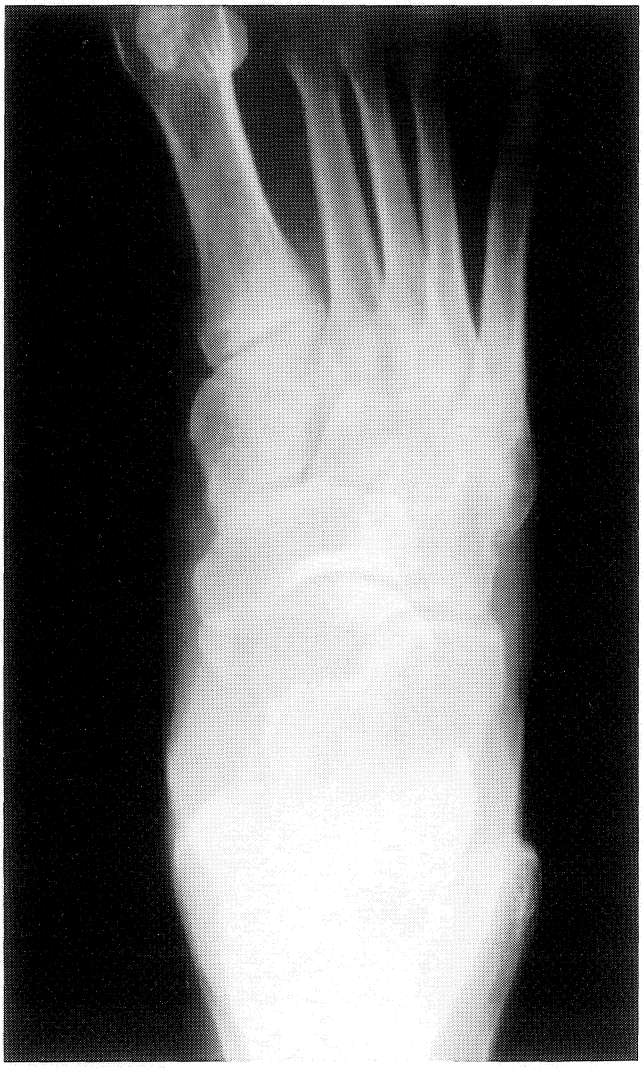

Figure 1
AP radiograph of case 14 showing the avulsion fracture of the navicular. Patient had completely avulsed the insertion of the posterior tibialis tendon.

The remaining patient with a navicular avulsion fracture (case 14) was found to have a scarred and immobile posterior tibialis tendon with an osseous fragment distally. The muscle was noted to have good elasticity. The navicular fragment was excised and the posterior tibialis tendon was reattached in the method described earlier. Pathologic review noted degenerative osteoarthropathy of the navicular fragment, along with degenerative fibrocartilage and fibrotendinous tissue.

Postoperatively the patients were immobilized in a short leg cast in maximum equinus and varus. At four weeks the patients were placed in a short leg walking cast in neutral ankle alignment and allowed to weight bear as tolerated. At ten weeks the cast was removed and the patients were advanced to an orthotic insert with physical therapy for range of motion and strengthening exercises.

RESULTS

The duration of follow-up averaged forty months (range: 24 to 67 months). Subjective satisfaction was evaluated with regard to pain, swelling, deformity, and gait. Tendon strength was determined by inversion power on manual testing and the ability to perform ipsilateral single leg heel rise.

Two patients were considered failures. Both had relief of preoperative medial ankle pain, but continued to have weak tendon function. One patient (case 1) began to develop increasing deformity seven months postoperatively. At one year follow-up, the patient had developed lateral ankle pain. With combined progressive deformity and gait disturbance, triple arthrodesis was performed fourteen months postoperatively. A second patient (case 7) developed lateral ankle pain four months postoperatively. This pain continued despite conservative efforts including physical therapy. Pain began to affect her gait. At one year, degenerative changes were noted at the subtalar and midtarsal joints on radiographic evaluation. Triple arthrodesis was performed fourteen months postoperatively. In both cases the tendon transfer was surgically intact at the time of arthrodesis. At follow-up both patients were pain free and ambulating independently with solid fusions.

Relief of preoperative medial ankle and foot pain was marked in the remaining fourteen patients. Each patient reported a great improvement in gait and a return to functional activity. Many returned to recreational endeavors such as golf, swimming, cross-country skiing, and bicycling. One patient (case 12) noted a return to unlimited activity, including tennis, and another (case 11) returned to running two miles every other day. All but one patient (case 5) noted marked relief of swelling.

Subjective improvement of the pes planus deformity was noted in all but one case (case 8). At follow-up each patient maintained a slightly improved but static deformity.

TABLE 2
Surgical Findings and Treatment

Case	Posterior Tibialis Muscle Elasticity	Surgical Findings	Rupture location	Initial Surgical Treatment
1(DB)	None	Complete Rupture, Accesory Navicular	6 cm Proximal to Insertion	Removal of Accesory Navicular, Tendon Transfer
2(RB)	None	Complex Tear with Adhesions	3 cm Proximal to Insertion	Tendon Transfer
3(PC)	None	Complex Tear with Adhesions, Chronic Proliferative Synovitis	1 cm Proximal to Insertion	Tendon Transfer
4(PC)	None	Complex Tear with Adhesions	2 cm Proximal to Insertion	Tendon Transfer
5(ZAD)	None	Longitudinal Tear	3 cm Proximal to Insertion	Tendon Transfer
6(BG)	None	Longitudinal Tears	3cm Proximal to Insertion	Tendon Transfer
7(SG)	None	Longitudinal Tear	3 cmProximal to Insertion	Tendon Transfer
8(DH)	None	Longitudinal Tear with Adhesions	3 cmProximal to Insertion	Tendon Transfer
9(JM)	None	Longitudinal Tears with Adhesions	2 cm Proximal to Insertion	Tendon Transfer
10(GM)	None	Longitudinal Tear with Adhesions	3 cm Proximal to insertion	Tendon Transfer
11(JN)	None	Complete Rupture with Adhesions, Chronic Proliferative Synovitis	4 cm Proximal to Insertion	Tendon Transfer
12(ER)	Good Muscle	Intact Tendon with Interstitial Tears and Stenosis at Retinaculum, Angioleiomyoma at Insertion,Chronic Proliferative Synovitis	InterstitialTears Posterior to Malleolus	Removal of Tumor, Synovectomy and Removal of Adhesions
13(ES)	None	Complete Rupture with Adhesions	3 cm Proximal to Insertion	Tendon Transfer
14(DT)	Good Muscle	Navicular Avulsion with Adhesions	Avulsion at Insertion	Removal of Navicular Fragment, Tendon Advancement
15(DV)	None	Complete Rupture	1 cm Proximal to Insertion	Tendon Transfer
16(WW)	None	Complete Rupture with Adhesions	2 cm Proximal to Insertion	Tendon Transfer

All but two patients (case 9, 12) continued to use a soft total contact orthotic with medial arch support for extended weight bearing activities. None of the fourteen patients were noted to have ankle swelling or tenderness on examination. The tendon transfer was noted to be fully functional on manual testing in every case. In addition, each one of these patients was noted to have full subtalar, midtarsal, and ankle mobility.

DISCUSSION

Rupture of the posterior tibialis tendon is often misdiagnosed[10,14]. This may be the result of the physician's failure to consider the diagnosis when evaluating an ankle complaint. The misdiagnosis rate is also secondary to the nonspecific clinical presentation, especially in the acute setting.

The initial history often includes previous eversion injury to the ankle and generalized medial ankle pain with swelling. A flexible asymmetric pes planus and forefoot pronation deformity is seen. Absent tendon function and an inability to perform a symmetric single heel rise are usually seen on examination. A patient that presents with these findings who has been unsuccessfully treated for other ankle or foot problems should be strongly suspected of having a posterior tibialis tendon rupture.

Chronic tenosynovitis has been reported in the pathophysiology of posterior tibialis tendon rupture[7,8,11,13,21,22]. It has been postulated that tenosynovitis either contributes to the tendon pathology, or results from it secondary to deformation of normal anatomy[11,13]. Surgically confirmed stenosing tenosynovitis has been reported to present in a similar fashion to confirmed tendon rupture. This similarity is likely due to the nonfunctional posterior tibialis tendon which is the common result in either case. One distinguishing factor in patients with a diagnosis of tenosynovitis is the marked tenderness of the posterior tibialis tendon[3,7,11,13,21]. Nine of our patients noted some tenderness on palpation of the tendon preoperatively, three of these were diagnosed with chronic proliferative synovitis (cases 3,11,12). In the cases of acute rupture, the synovitis was most likely a consequence of the injury.

Posterior tibialis tendon rupture is more often reported as a result of chronic overuse in the adult population[6,7,9,10,11,13,14,15,23]. Collagen maturation[18], decreases in water content, and an increase in elastin coarseness will decrease tendon compliance. Loss of strength, a decrease in lower limb flexibility, and reduced impact absorption capability have been speculated to account for the higher frequency of foot and ankle injuries in older individuals[17]. However, in the physically active adult, age-related biochemical and biomechanical changes

in connective tissue will reverse with a continuous training program. The collagen maturation process is eliminated by the high rate of collagen turnover associated with continued physical activity[18].

In active individuals chronic overuse is not likely to be an etiologic factor for tendon pathology. In this and previous reports, antecedent trauma is seen in patients with confirmed posterior tibialis tendon rupture[4,6,9,10,11,12,13,14,15,19]. One review of injuries in the active adult population noted no difference in frequency of tendon pathology among the two age groups studied with mean ages of 30.4 and 56.9 years[17].

Diffuse medial ankle pain and swelling were the two most common symptomatic complaints in our review. Although the pain in most reports is described as diffuse, medial ankle swelling was a consistent finding in other studies. Fifteen of our patients were noted to have asymmetric forefoot pronation, fourteen of these also had an asymmetric pes planus deformity. This deformity is easy to recognize, but requires careful examination of the patient's gait and stance (Figure 2). Under these circumstances a close examination of the tendon is warranted. Manual testing will likely reveal a nonfunctional tendon; this finding has been reported to be diagnostic of rupture in 90% of cases[2]. Every patient in our review had similar absence of posterior tibialis tendon function (Figure 3). Other signs of tendon rupture include an inability to perform the single heel rise and loss of normal heel inversion[2,6,10,14,23]. As expected with a dysfunctional posterior tibialis tendon, there is an inability to stabilize the transverse tarsal joints and subsequent decreased transmission of plantarflexion force through the forefoot[20]. All but one patient was noted to fail at this test in our study (Figure 4).

Lateral ankle impingement and pain should imply severe progression of valgus deformity[10]. In our review, five patients presented with lateral ankle pain that improved after treatment. After reconstruction two other patients developed progression of their deformity. Subsequent triple arthrodesis became necessary to relieve the pain.

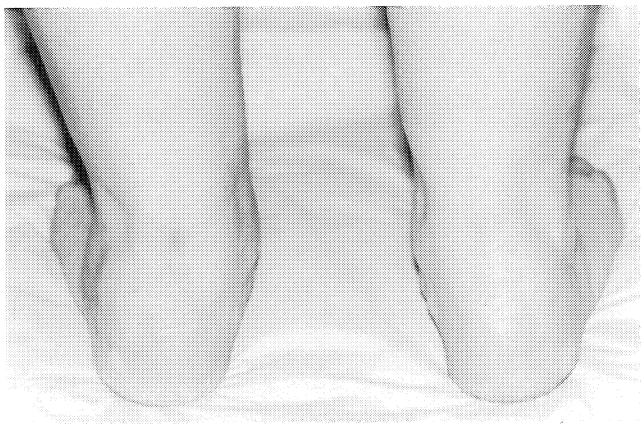

Figure 2
Patient with asymmetric pes planus and posterior tibialis tendon rupture on the right side.

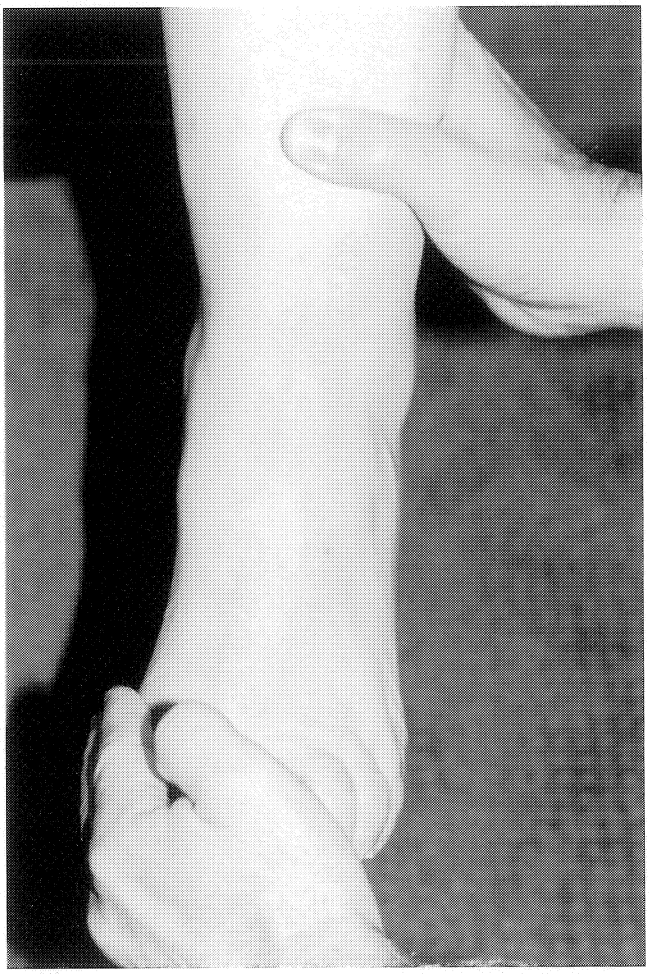

Figure 3
Manual testing of the posterior tibialis tendon function. With the patient sitting and knee flexed, the ankle is placed in equinus and the foot in inversion. The examiner then attempts to evert the foot.

Anteroposterior and lateral standing radiographs are obtained to evaluate the severity of the deformity and identify gross bony pathology[2,6,10,14]. In our study the standing radiographs were of little diagnostic value. Talonavicular sag was seen in only three cases. Presence of a perinavicular fracture should alert the clinicians for presence of posterior tibialis tendon avulsion. Magnetic resonance imaging has been reported in the recent literature as helpful in both tenosynovitis and rupture[1,2,4,5]. Newer techniques such as thin slice acquisition, improved resolution with surface coils, and modified coronal, oblique T2-weighted views increase diagnostic accuracy[5]. Ultrasound imaging is a promising diagnostic adjuvant as well[4]. Downey et al noted the usefulness of both static and dynamic ultrasound imaging[4]. Tenographic studies have not been very useful, and are of academic interest only[2,4].

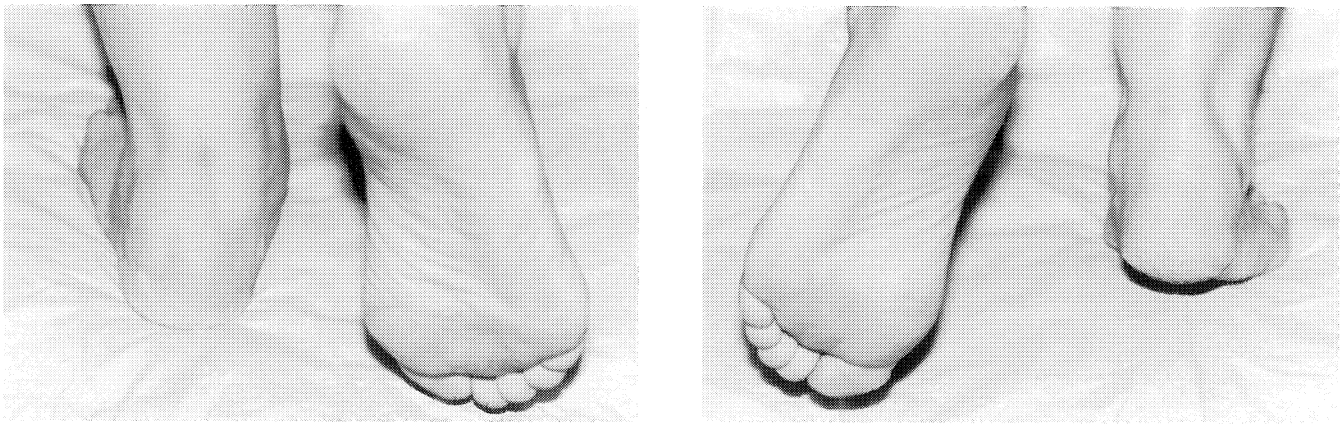

Figure 4
Single leg heel rise test
A. Patient unable to raise up the left heel due to ruptured posterior tibialis tendon.
B. Patient able to raise up the right heel with intact tendon function.

Use of tendon transfer has dominated the literature as the preferred surgical treatment of posterior tibialis tendon rupture[2,4,7,9,10,14,15,24]. The choice of tendon to transfer has varied between the flexor hallucis longus and the flexor digitorum longus tendons. The potential moment of the flexor hallucis longus approaches that of the posterior tabialis, unlike the flexor digitorum longus which has approximately one third of the potential moment of the posterior tibialis[20]. However, most believe that successful reconstruction must balance the effect of the peroneus brevis. The flexor digitorum longus does this nicely, with a similar moment[14,20]. Opting for neutralization of the deforming imbalance across the foot and maintenance of the stabilizing effect of the flexor hallucis longus, the flexor digitorum longus was utilized for tendon transfer in our study. Excellent results were seen postoperatively. No restrictions were placed on the patient's level of activity after rehabilitation with physical therapy; however, use of an orthotic with medial arch support was recommended for extended weight bearing activities.

CONCLUSIONS

Some physicians may feel that posterior tibialis tendon rupture is a rare condition, one that they have never seen. Our review would suggest, as Jack Hughston would say, "they may not have seen it . . . . . but it probably has seen them!"

Posterior tibialis tendon rupture is strongly suspected in patients presenting with a complex of findings. These include:

1. Eversion ankle injury.
2. Generalized medial ankle pain and/or swelling.
3. Flexible, asymmetric pes planus and forefoot pronation.
4. Nonfunctional posterior tibialis tendon on manual testing.

The first three findings are nondiagnostic; however, when seen together they are helpful in identifying patients with posterior tibialis tendon pathology. Manual testing of the posterior tibialis tendon should be performed in the presence of these findings to confirm the tendon's functional status. Other associated findings include gait disturbance secondary to deformity, tenderness along the posterior tibialis tendon, and a talonavicular sag on lateral standing radiograph.

A trial of conservative therapy with orthotics, nonsteroidal antiinflammatory drugs, and physical therapy may be instituted in patients suspected of posterior tibialis tendon rupture. However, close follow-up is important and the patient should be explored surgically if progression of symptoms or deformity occurs. In neglected cases of posterior tibialis tendon rupture, a severe pes planus and pronation deformity is often seen. This may result in degenerative changes of the subtalar and midtarsal joints. This progression will not respond to soft tissue procedures, and fusion is often necessary[10,14].

Flexor digitorum longus tendon transfer is suggested with absent posterior tibialis muscle elasticity. In most cases tendon transfer will result in relief of pain and return of function. The resultant deformity is rarely corrected to normal, and although improved, a static deformity generally remains[6,14].

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FRACTURE OF THE HOOK OF THE HAMATE: RADIOGRAPHIC VISUALIZATION

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Jeffrey F. Lakin, M.D.

ABSTRACT

The diagnosis of fracture of the hook of the hamate is rarely made at the time of the initial injury. Routine roentgenograms of the hand in the standard three positions do not visualize this structure. The carpal tunnel view (CTV) with hyperextension of the wrist may be too painful to position. A computerized tomography of the wrist in the transverse or axial plane will clearly and painlessly identify the fracture. Placing both hands and wrists in the praying position gives excellent comparison and documents any developmental bone abnormality. Scintigraphy, when positive, must be followed by tomography or a carpal tunnel view.

INTRODUCTION

Fracture of the hook of the hamate is infrequently diagnosed when pain in and about the wrist and hand is the presenting complaint. As fractures of the hamate constitute only two percent of all carpal fractures[6], it is often not considered in a differential diagnosis. Most frequently the patient does not recall a specific incident of trauma such as a fall or crushing blow to the hand. A high percentage will present with complaints of chronic pain or weakness in grip. Unfortunately the hamulus is not well visualized on standard roentgenograms such as the posteroanterior, lateral, and oblique views of the hand and wrist. In the initial examination, the pain of trauma often limits forced hyperextension of the wrist. This paper reviews techniques of visualization which should prove helpful in diagnosing hook of the hamate fractures.

MATERIAL AND METHODS

The authors have reviewed 180 cases reported in the literature that identify a fracture of the hamulus. Table 1 shows what radiographic studies were used to identify fractures of the hook of the hamate in acute cases when the initial standard three radiographic views were negative. All positive bone scans thereafter had either a CTV or tomogram for positive identification. We have added to that the experience of twenty-five cases treated by our Hand Service. We were interested in radiographic visualization of the fracture should one be suspected. Of the 180 cases reported, only thirty-five were identified and treated acutely. In our own twenty-five cases, eight were diagnosed acutely and two were treated here initially by the authors.

DISCUSSION

It is common practice to obtain a standard set of radiographs to evaluate the hand after direct trauma or continued pain. The positions are posteroanterior, lateral and forty-five degrees pronated in relation to the tube-cassette axis. Experts agree that these views fail to outline the hook of the hamate. Norman et al felt that such a fracture may be seen on the PA view when outlining the "eye" or base of the hamulus[15]. This sign has not been helpful to us. Other roentgenographic views are more definitive.

A useful view of the osseous outlines of the carpal canal and especially the hook of the hamate and the pisiform, is the CTV. This is now a standard position accepted by radiologic technicians. The view was initially suggested by Hart and Gaynor in 1941[8] and later amplified by Wilson in 1954[21]. As anomalies of the carpal bones may occasionally appear, a standard review of these will be found in other sources[3,16]. The wrist and forearm are placed in a pronated position on the cassette. The fingers and hand are hyperextended and held by the other hand. The tube is then directed at the wrist with an angle that will outline the carpal canal (Figure 1). This standard view is presented in radiology texts and in the publications of Murray[14], Parker[17], and Stark[19]. Another method of obtaining this view is to have the patient stand and place the pronated hand on the cassette. The wrist is then extended and the tube angulated from behind the patient (Figure 2).

In 1934, Milch[12] described the difficulties in obtaining a good projection of the hamulus by x-ray. He noted that several oblique positions were necessary. Several authors have advised that the best visualization of the hook of the hamate is the projection taken with the hand and wrist in forty-five degrees of supination[1,10]. This is a useful position and is helped if the hand is held in slight radial deviation.

For many years advances in radiographic techniques have been developed to focus on one level of anatomy[5]. In 1935 Grossman developed tomography by moving the tube and plate to blur unwanted structures and therefore

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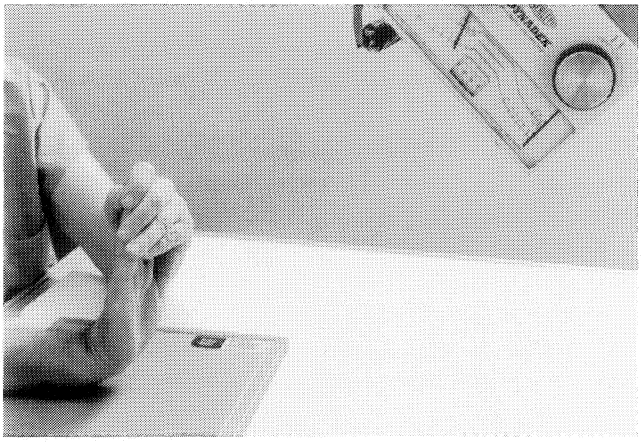


Figure 1
The most common position for visualization of the carpal canal is the pronated forearm with fingers and hand held in hyperextension on the cassette. The tube can then be angled to outline the carpal canal.

identify a selected level[7]. Tomography is of great value in detecting injuries to the hand. Planar and trispiral modalities are equally effective[11,20]. Eaton and Morris specifically pointed out the use of tomography in a fracture of the hamulus[4]. Computed tomography (CT) was first introduced in 1972 by Hounsfield[9] to give a two-dimensional view of a cross sectional level of anatomy. It was developed from the 1917 work of the mathematician Radon[18], and extended by the work of Cormack in 1964. The image can be obtained without pain in an acute fracture of the hamulus, and is most accurate in visualizing old suspected fractures.

The development of nuclear medicine in radiology is a fascinating story that began with the work of Rutherford in 1919[5]. With the introduction of radionucleides that have a short half-life, no beta emission, and a gamma emission of 140 KeV, a safe method of identifying abnormal activity in the skeleton has evolved. Belsole in 1981[2] pointed out the value of technetium-99m administration in the diagnosis of a fractured hamulus of a golfer. It is, however, necessary to obtain a CTV or tomogram to properly identify the exact level of fracture. Murakami[13] found scintigraphy helpful in identifying the presence of a fracture of the hamulus in five of his nineteen cases.

From the literature and our own experience, we realize that a fracture of the hook of the hamate is rare and the diagnosis is often difficult to make. With no fracture seen on the standard three x-ray views, two alternatives are offered in two cases that we have treated primarily:

1) A twenty-nine year old orthopaedic resident fell on his outstretched hand while playing roller hockey. Pain and swelling were present on the ulnar side of the wrist and palm. He was able to hyperextend his wrist and a clear CTV was obtained to show the fracture of the hamulus (Figure 3).

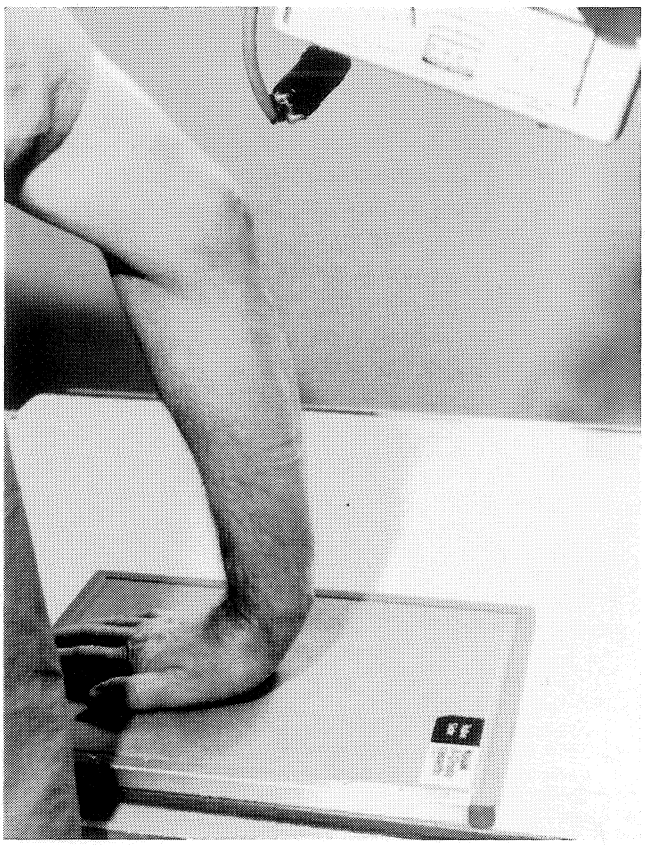


Figure 2
The standing position is favored by some technicians as the hand does not shift or waiver as it might when held by the patient.

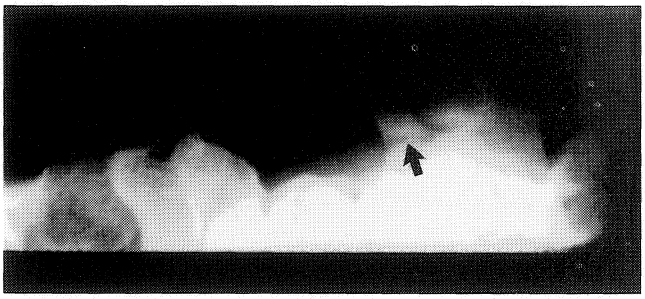
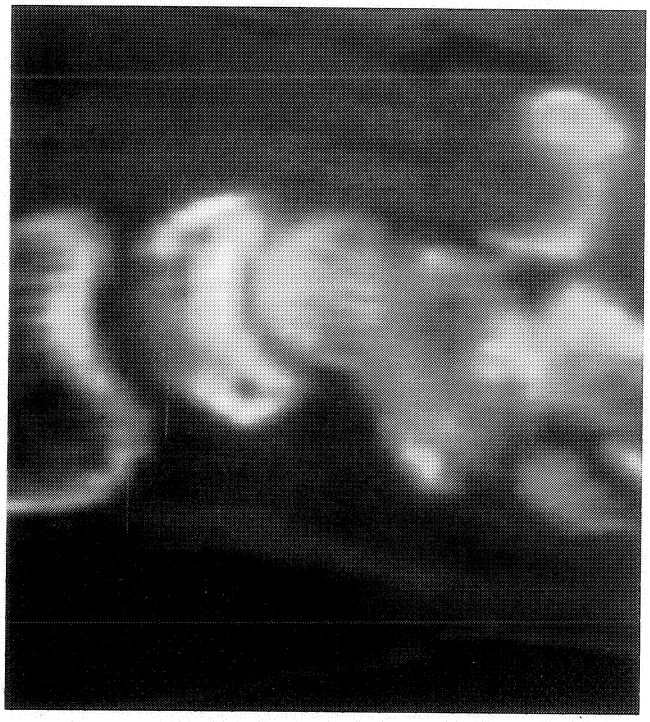


Figure 3
The patient was able to extend his wrist in spite of pain to obtain a CTV showing the fracture of the hamulus (arrow).

2) A sixty year old housewife fell in the street on her outstretched hand. The ulnar side of the palm was swollen and painful. A CTV was difficult to obtain secondary to discomfort and a CT scan showed the fracture nicely (Figure 4).

Traditionally the diagnosis of fracture of the hamulus has not been made acutely but after a long period of chronic pain. A CTV is considered a very specific method of plain radiography, but fracture of the hamulus can be missed on this view. In seven of twenty-three cases of patients with



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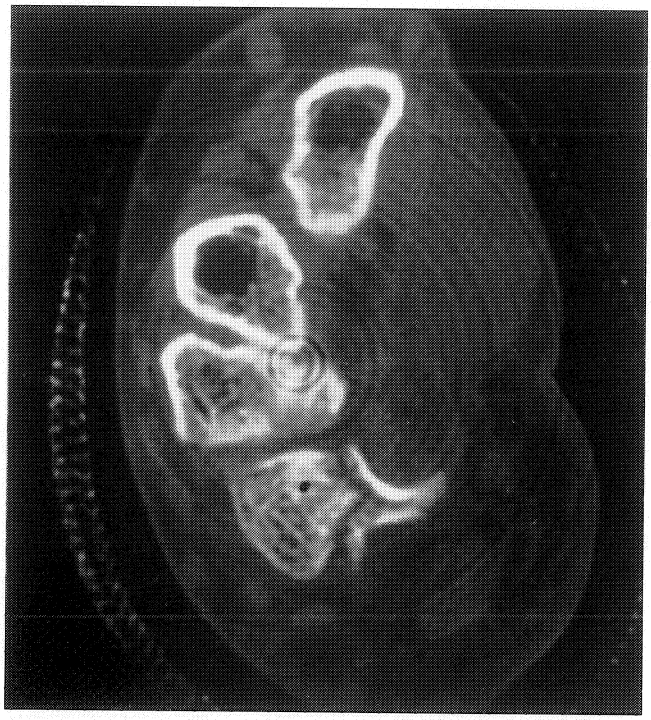

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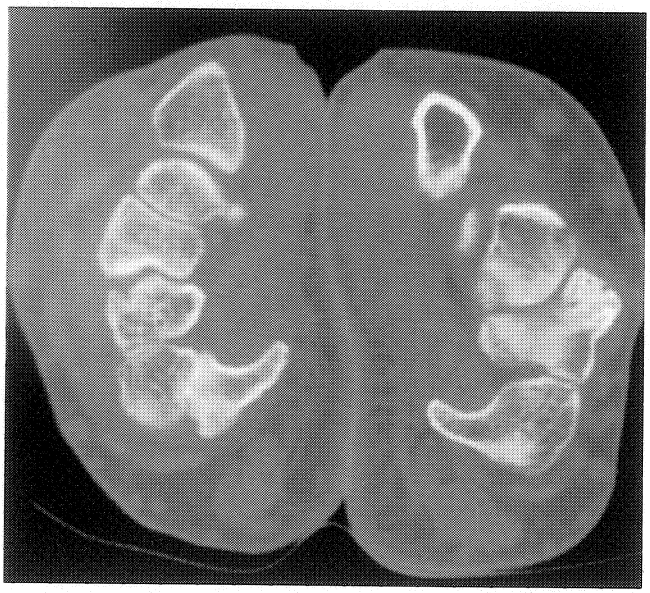

C

Figure 4

Due to pain in the hand and wrist this patient could not extend her wrist. A CT scan was painless and identified the fracture of the hamulus on lateral or sagittal view (a) and transverse or axial view (b). The authors prefer the axial view with both hands in the "praying" position. It allows the best comparison for healing as shown in (c) at 3 months. It is also useful when anomalies of the carpal bones are suspected.

persistent pain in the wrist treated by us, the CTV had to be repeated to bring out the fracture.

We have presented this paper to identify techniques of radiography that make this elusive diagnosis more easily and painlessly in the acute setting, as well as in patients with chronic pain in the ulnar wrist and palm.

X-ray Positions of the Hand & Wrist in Acute Cases (literature)

Author	Year	Cases	Carpal tunnel view	45° supinated oblique	Tomogram	Computed Tomography	Bone Scan
Milch	1934	1		1			
Torisu	1972	1		1			
Nisenfield	1974	1	1				
Stark	1977	1	1				
Roach	1978	1		1			
Egawa	1983	6	6				
Foucher	1985	1	1				
Norman	1985	2			2		
Parker	1986	3	1	1			1
Bishop	1988	2			2		
Stark	1989	10	8	1		1	
Murakami	1991	3					3
Whalen	1992	6			6		

Table 1

This table identifies the type of radiograph used by each author for his initial identification when a fracture of the hamulus was suspected. All patients had a negative set of standard views. When scintigraphy was used, either tomograms or a CTV followed to clearly identify the lesion.

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MANAGEMENT OF MAJOR ARTERIAL INJURIES OF THE LIMBS IN 166 CASES

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ABSTRACT

The charts of 166 patients with major arterial injuries (175 arterial repairs) inflicted during periods of peace and war time were reviewed. One hundred and sixty-seven (95%) of the arteries repaired remained patent. Nine patients had limbs which developed ischemic contracture. Nine patients required amputations. Follow-up data was obtained from seventy-five patients with an average follow-up of five years and one month (six months to thirty years). Early diagnosis, prompt treatment including fasciotomies when indicated, complete debridement, appropriate coverage, meticulous surgical technique, and simultaneous treatment of concomitant injuries are all crucial factors in successful limb salvage.

Major arterial injuries of the limb occur frequently in our patient population, especially in time of war. Successful treatment of major arterial injuries may be life-saving, as well as allowing limb salvage and restoration of limb function. The purpose of this study is to review our experience with repair of major arterial injuries.

CLINICAL DATA

We reviewed the charts of 166 patients with major arterial injuries from 1959 through May 1991 who were treated at this Institute. Major arterial injuries are defined in this study as injuries of peripheral arteries above the brachial artery in the upper extremity or above the popliteal artery in the lower extremity. Other major injuries include simultaneous injury to the radial and ulnar arteries or anterior and posterior tibial arteries.

There were 144 male and twenty-two female patients. The ages ranged from seven to seventy-one years. There were 121 patients (73%) within the age range of 20-45 years. The causes of arterial injury are listed in Table I.

Table I
Causes of Major Arterial Injury

Causes	No. of Cases
Laceration	62
Bullet	26
Vehicle	22
Machinery	19
Crush	19
Others	18
Total	166

Anatomic distribution is noted in Table II.

Table II
Anatomic Distribution of Arterial Injuries

Artery	*No.*	*%*
Subclavian	8	5
Axillary	9	5
Brachial	59	35
Radial & Ulnar	11	7
Brachial, Radial & Ulnar	1	1
External iliac	9	5
Femoral	31	19
Bilateral femoral	1	1
Popliteal	37	22
Total	166	100

Types of arterial injury are noted in Table III.

Table III
Types of Arterial Injuries

Types	*No.*	*(%)*
Transection	112	62
Partial	26	15
Contusion with subsequent thrombosis	15	8
Spasm	7	4
Compression from fracture	3	2
False aneurysm	13	7
Arteriovenous fistula	4	2
Total	180	100

Fourteen of the patients presented with late arterial injury, defined as untreated or treated inadequately for four weeks after trauma. Associated injuries were found in 135 patients (see Table IV).

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Table IV
Incidence of Associated Injuries Among 135 Acute Arterial Injuries

	No.	%
Shock	48	36
Fracture	56	42
Dislocation	6	4
Vein injuries	53	39
Severe muscle injuries	23	17
Nerve injuries	96	71

In addition, there were two patients with hemopneumothorax and one with abdominal visceral injuries.

Methods of Management

The methods for end-to-end anastomosis, autovein graft, and hydrostatic dilation are illustrated in Figures 1, 2, and 5A. Methods of management are listed in Table V. Five ulnar and radial arteries were ligated.

Table V
Methods of Management

Method	Treated No.	Patent No.	(%)
End-to-end anastomosis	69	65	(94)
Autovein graft	85	82	(96)
Bypass graft	1	1	
Vein patch	1	1	
Prosthetic graft	1	0	
Lateral suture	8	8	
Intraluminal hydrostatic dilation	7	7	
Decompression by reducing fracture	3	3	
Total	175	167	

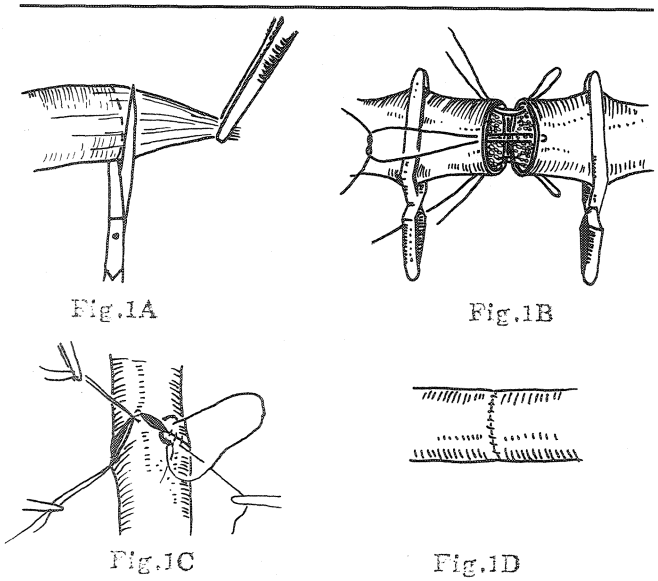

Figure 1
Illustrations for the procedure of end-to-end anastomosis. Adventitia excised (A). Three-point mattress fixation suture (B). Simple running suture (C). Finished (D).

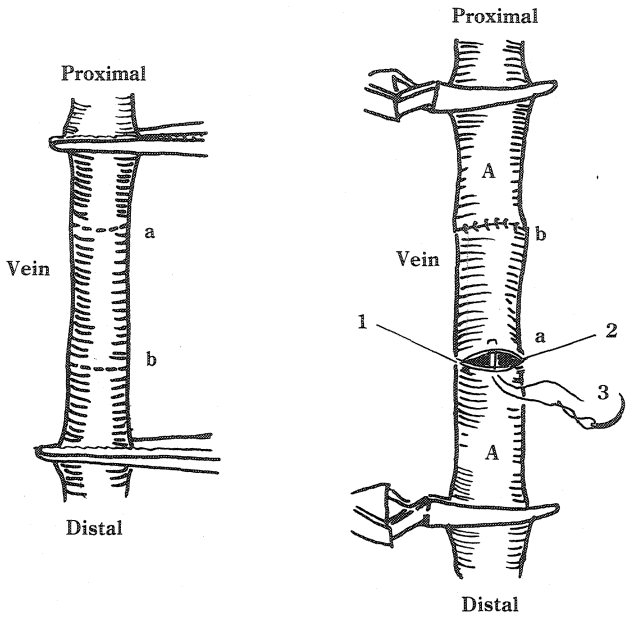

Figure 2.
Illustrations for the procedure of vein graft. Vein segment excised (A) reversed and inserted into artery (B).

RESULTS

Early Results

1. Patency: One hundred and sixty-seven (95%) of 175 arteries treated remained clinically patent.

2. Incidence of Ischemic Contracture: Nine patients (5%) suffered ischemic limb contracture. In this group, eight popliteal arteries and one femoral artery had been initially injured.

3. Amputation: Nine patients (5%) required an amputation. Common causes were delay in diagnosis and management, massive soft tissue destruction, lack of coverage by healthy muscle, and renal failure after revascularization.

4. Mortality: No deaths occurred in this series.

Long-term Results

One hundred and fifty-seven patients had limb survival. One hundred and fifty had follow-up of greater than six months. Data was obtained by chart review or follow-up examination for seventy-five patients, with an average follow-up of five years and one month (range six months to thirty years).

1. Blood supply and function of the limbs: Of the seventy-five patients with longer follow-up, seventy-three had excellent circulation in the affected limb, while two had mild ischemic pain. Sixty-four patients (85%) had satisfactory function in the affected limb. Eight patients required orthopaedic procedures for lower limbs with ischemic contracture. Nine patients had limbs with partial nerve deficit.

2. Patency Documented by Doppler-Ultrasonic Imaging Studies: Eighteen patients with 18 grafts were examined by doppler-ultrasonic imaging. Sixteen grafts remained patent. Two patients with occluded grafts had overall excellent circulation and satisfactory function. In one of these patients an arteriogram demonstrated rich collateral circulation.

Nine of these cases are examined in greater detail in Figures 3 through 11.

DISCUSSION

Prompt and accurate diagnosis is essential for treatment of major arterial injuries. Clinical characteristics such as pulsatile massive hemorrhage or hematoma, swelling, lack of warmth, loss of capillary refill, and absent or diminished distal arterial pulses help to diagnose major arterial injuries. Doppler-ultrasonic imaging provides an accessory method for diagnosis and location of lesions. Surgical exploration should be performed as soon as major arterial injuries are suspected.

Priority must be given to management of life-threatening injuries. Pressure dressings should be applied to wounds in the field as early as possible. Primary debridement and ligation of injured vessels are often helpful methods of initial stabilization prior to transfer to a regional center for definitive care.

Time is vitally important in the management of arterial injuries. Wolma et. al. found that delay in management of more than six hours was associated with an amputation rate of 50%, whereas intervals of less than six hours between injury and definitive treatment led to an amputation rate of only 7% (1). The relationship between time interval and result in 135 acute arterial injuries from this series is presented in Table VI.

We advocate prompt and early surgical treatment, preferably within twelve hours of injury, but in our experience the time factor is not absolute. Nine cases in this series were successfully treated after a delay of more than twenty-four hours (Figure 3).

Table VI
Relationship of Interval between Injury and Operation to Result in 135 Acute Arterial Injuries

Time (hr)	No. Cases	Ischemic Contracture No. (%)	Amputation No. (%)
<12	89	1 (1)	1 (1)
>12	46	8 (17)	8 (17)
Total	135	9 (7)	9 (7)

The damaged vessels were repaired after prompt and thorough debridement. The ideal method of repair varies depending on the extent of injury. Adequate soft tissue coverage is important, and we prefer healthy muscle when available. End-to-end anastomosis and vein grafting were the most common methods of treatment in our series. The method of three-point mattress fixation and simple running suture was designed by the authors. This method is convenient, allows easy visualization of the luminal surface, and allows the surgeon to avoid constriction at the site of anastomosis.

Autogenous vein graft is the material of choice for reconstruction of peripheral arteries[2,3,4]. Eighty-five (49%) of 175 damaged major arteries in our series were repaired with autovein grafts (Figures 4 through 10). Early clinical patency rate was 97%. Long-term follow-up results for autovein grafts showed a patency rate of 89%, without any pseudoaneurysms.

Simultaneous treatment of associated injuries to veins, nerves, and fractures is essential to successful arterial repair. One patient had a popliteal artery transection and a comminuted femur fracture from a collapsed wall. The popliteal artery was repaired with autovein graft and the femur fracture managed by sliding traction. At follow-up the patient had excellent circulation and function in his involved limb (Figure 6).

The literature reports a high amputation rate with popliteal arterial injury[5]. There were 37 patients (22%) with popliteal artery injury in our series, five (13%) of which required an amputation. Inadequate treatment of popliteal arterial injuries among our patients often resulted in ischemic pain and intermittent claudication (Figure 10).

Injury to the subclavian artery often causes exsanguinating hemorrhage and hypovolemic shock. Prompt exploration and control of massive bleeding are crucial for survival in this situation[6,7]. The location of the subclavian artery makes surgical exposure difficult. One of our patients had a subclavian artery injury and a large associated hematoma. Exposure was easily obtained through a median sternotomy incision and a dilated autogenous saphenous vein was used to repair the artery successfully (Figure 8).

Fasciotomy can be an important adjunctive measure for relief of intracompartmental pressure and salvage of an ischemic extremity. The eighteen patients in our series who were treated with fasciotomy had good limb function at follow-up (Figure 7).

Surgical intervention is indicated for late arterial injury associated with severe ischemia. Collateral circulation to the limb should be preserved carefully when an operation for late arterial injury is performed. There are two options for revascularization. The first is to use autogenous vein graft, most commonly the saphenous vein. The second is to perform a bypass, thus avoiding the scarred area

Figure 3
(Case 1) Thirty-three year old male with popliteal artery transection. Operation was performed forty-eight hours after injury. Follow-up was twenty-six years, with satisfactory function.

Figure 3A Twenty-six years postoperative.

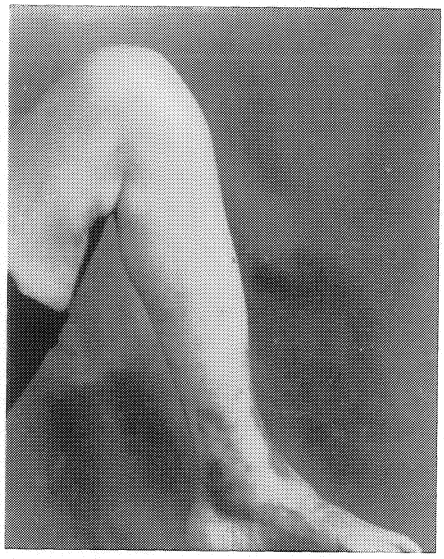

Figure 3B Normal flexion of knee joint.

Figure 4 (below)
(Case 2) A twenty-four year old male suffered injuries to the left brachial artery and median and ulnar nerves. Autovein graft was used to repair a six centimeter defect in the artery.

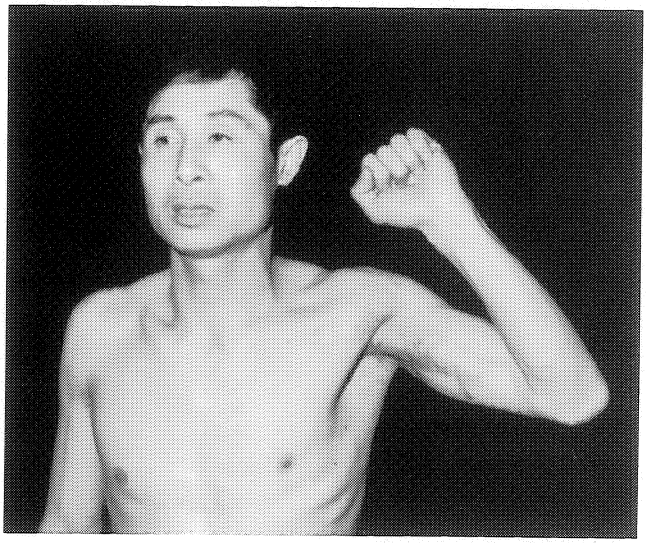

Figure 4A At thirteen year follow-up the patient had excellent function.

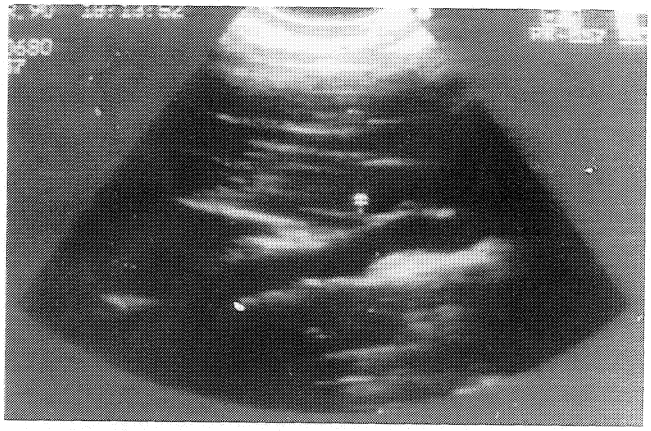

Figure 4B Ultrasonic imaging shows patent graft.

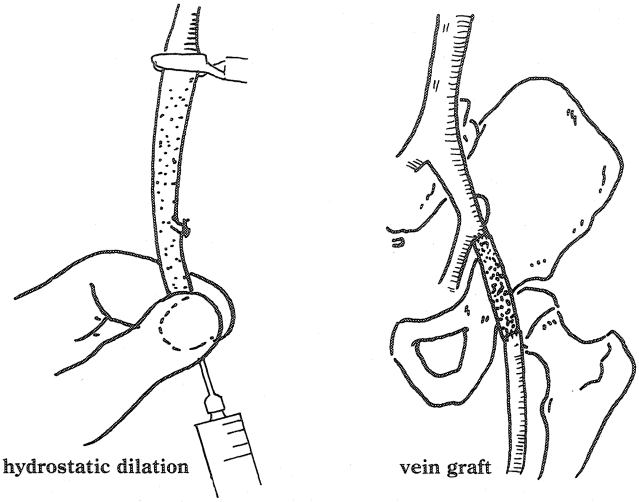

Figure 5A Illustration for hydrostatic dilation of vein graft.

Figure 5
(Case 3) A twelve year old boy suffered an injury to his left external iliac artery. Autovein graft (4.5 cm) was used in the repair after hydrostatic dilation. He was followed for twelve years with excellent function.

(Figure 10). In our review, one patient had a femoral artery repaired, but developed ischemic pain in his leg. The involved artery was documented to be occluded on arteriogram. He underwent a revision of the repair with autovein graft. Eleven years after the second operation his limb was functioning well and the graft was patent (Figure 9).

In the management of psuedoaneurysm and arteriovenous fistula, excision of the lesion and restoration of vascular continuity should be performed early (Figure 11). It is harmful to wait for collateral circulation to appear. In this series, seventeen patients were treated early with excellent results.

CONCLUSION

Injuries to major arteries generally carry with them a high amputation and mortality rate if not properly treated. The results of treatment in our series were satisfactory for early and long-term follow-up. Early diagnosis, prompt treatment including fasciotomies when indicated, complete debridement, appropriate coverage, meticulous surgical technique, and simultaneous treatment of concomitant injuries are all crucial factors in successful limb salvage. Late arterial injury associated with a painful ischemic limb is an indication for surgical intervention.

ACKNOWLEDGMENT

The authors would express the thanks to Dr. Wang Zhen for his drawing of the ischemic diagrams.

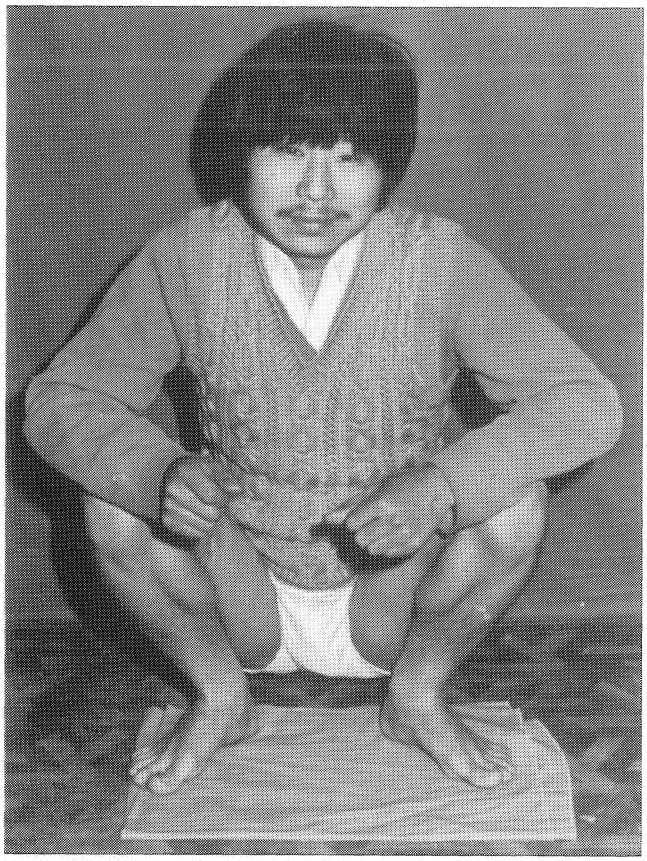

Figure 5B Twelve years postoperative.

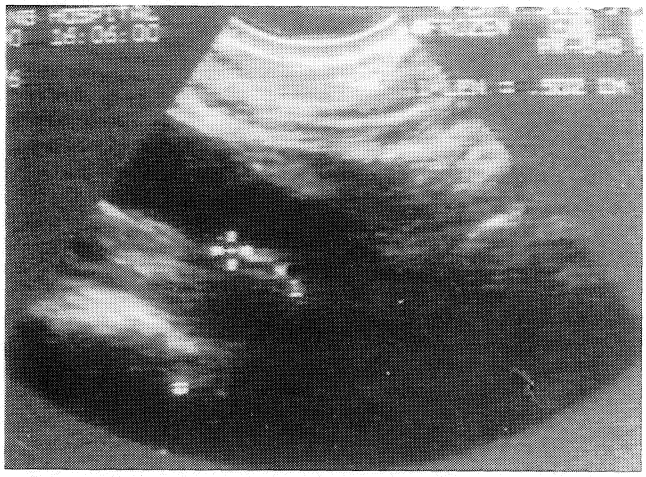

Figure 5C Ultrasonic imaging shows patent graft.

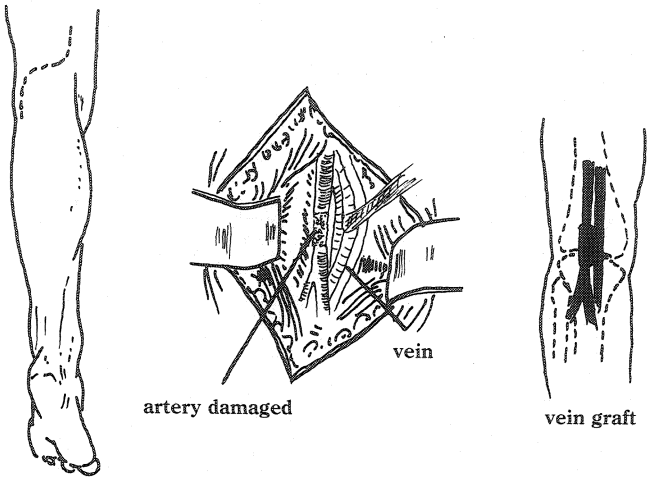

Figure 6A Illustration for injury and repair with vein graft.

Figure 6B At twelve years postoperatively, the patient had excellent function in the affected limb.

Figure 6
(Case 4) A twenty-nine year old male suffered a popliteal artery transection, with associated femur fracture. Autovein graft (7 cm) was used and the femur fracture was managed by sliding traction.

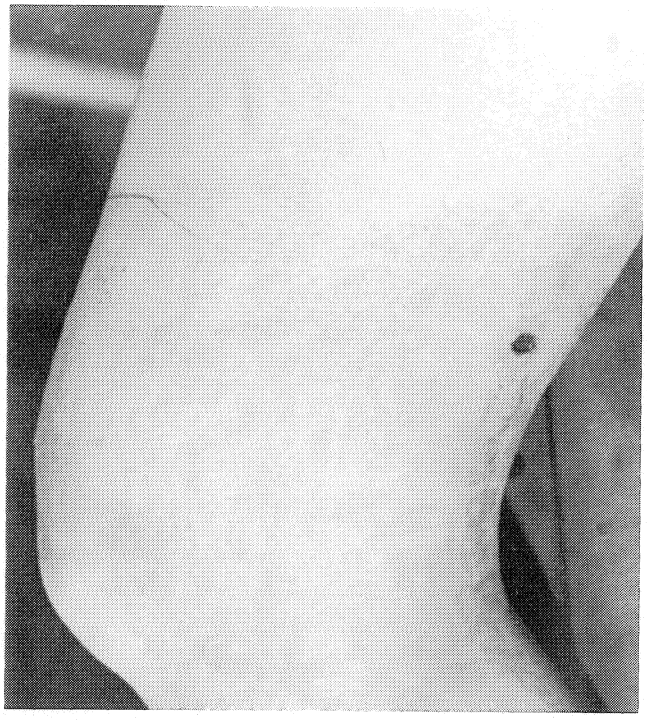

Figure 7A, 7B Injury caused by bullet wound.

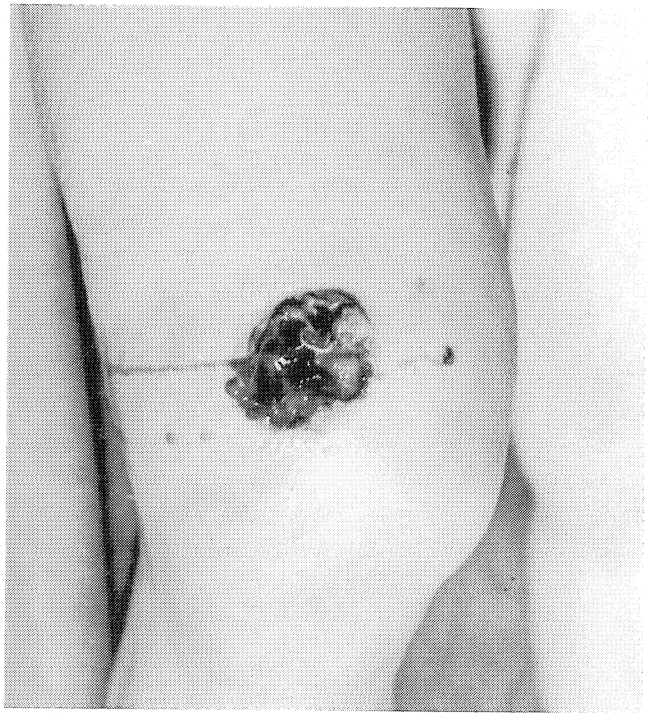

Figure 7B

Figure 7
(Case 5) This patient is a twenty-one year old male with a popliteal artery injury associated with a communited femur fracture secondary to a bullet wound. The involved artery was repaired with a seven centimeter autovein graft.

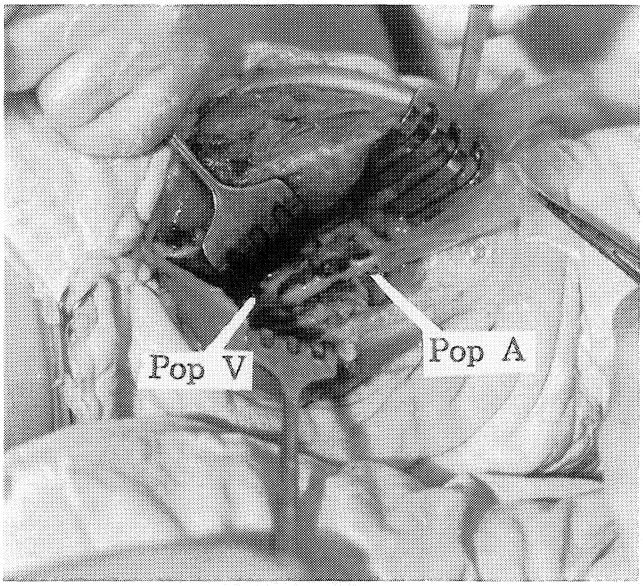

Figure 7C Operative appearance.

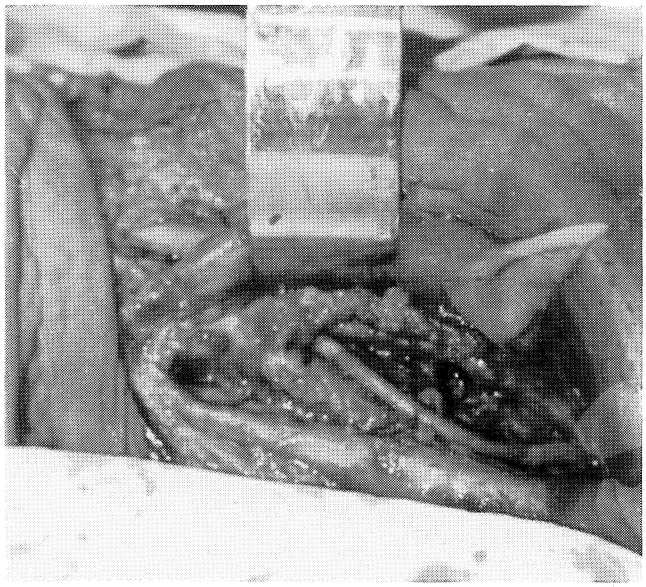

Figure 7D Vein graft transplanted.

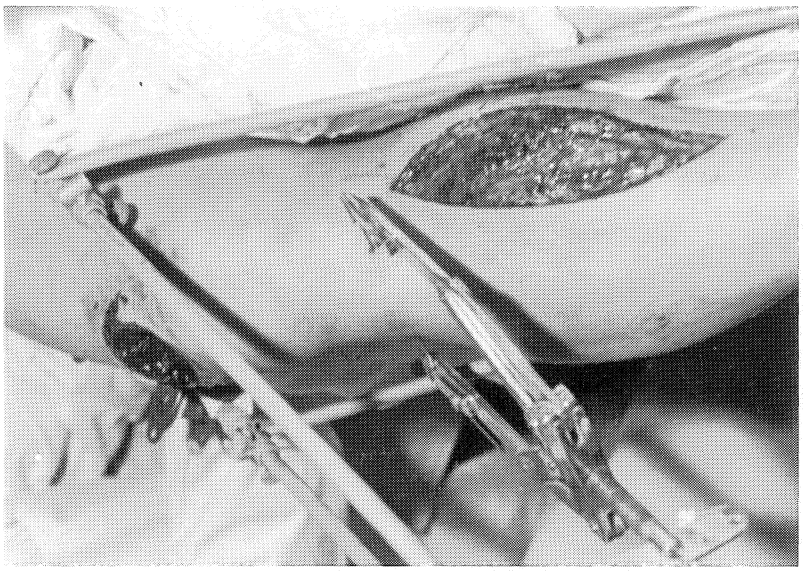

Figure 7E Fasciotomy and continuous traction.

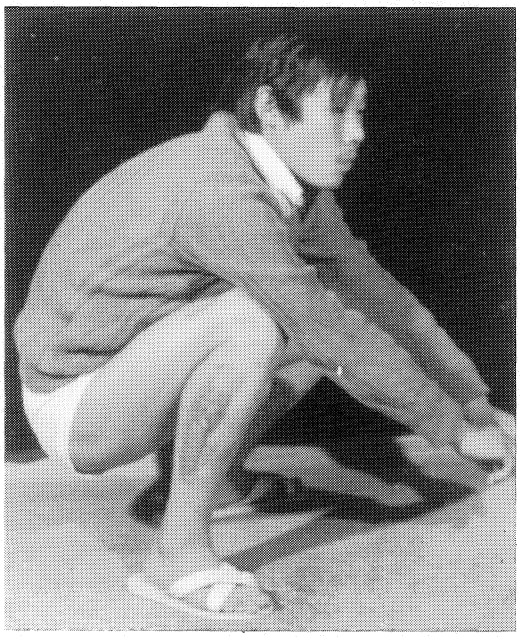

Figure 7F Two years postoperative. Patient had excellent function.

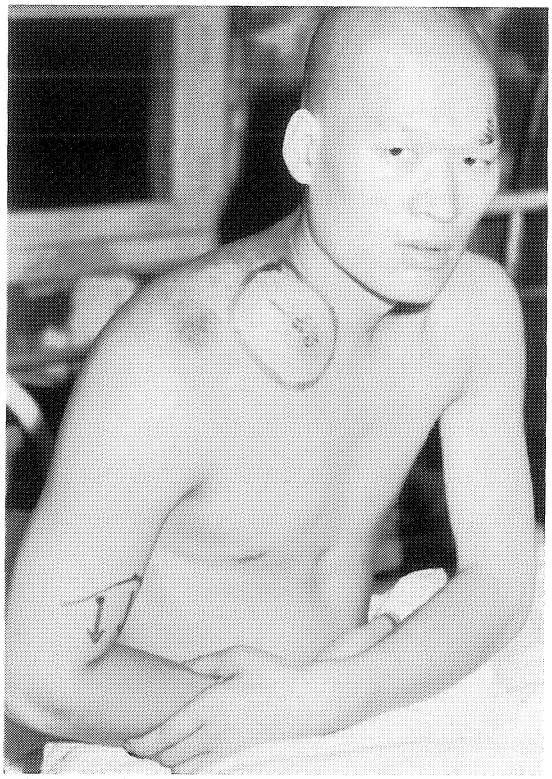

Figure 8A Preoperative appearance seven days after original injury.

Figure 8

(Case 6) This twenty-five year old male had injured his subclavian artery. Exposure of the right subclavian artery was obtained through a median sternotomy incision. Dilated autovein graft (Diameter seven mm, length five cm) was used to repair the artery (Diameter eight mm).

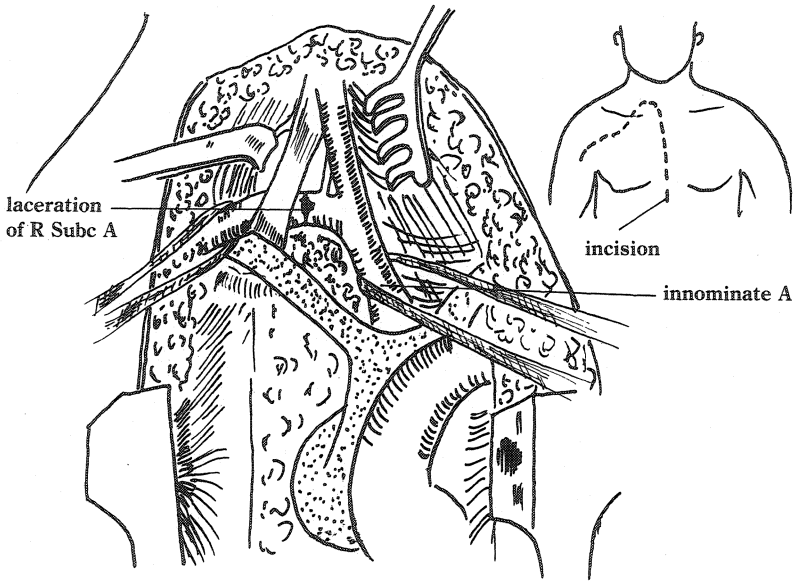

Figure 8B Illustrations for incision and exposure.

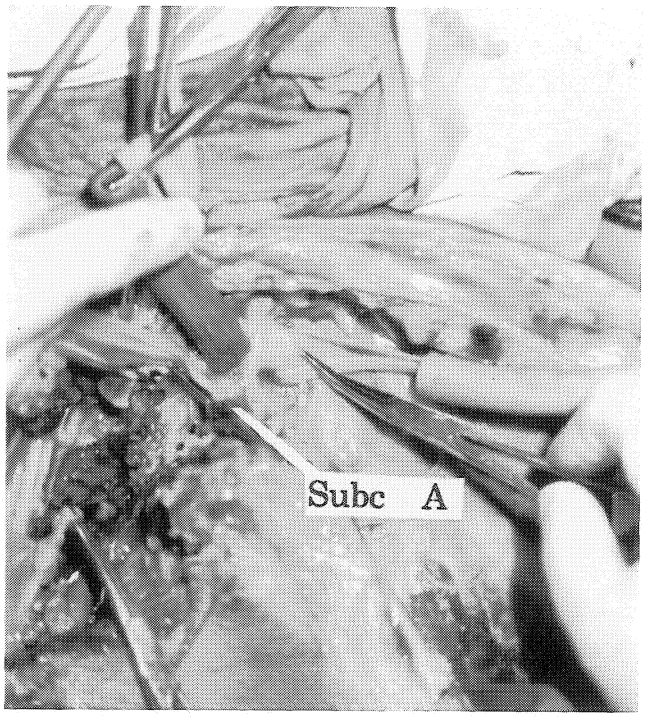

Figure 8C Operative appearance. A large hematoma had formed.

Figure 8D Vein graft transplanted.

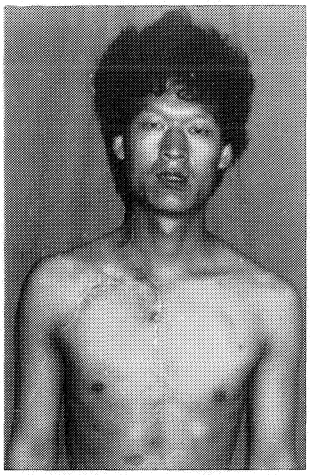

Figure 8E Nine months postoperative.

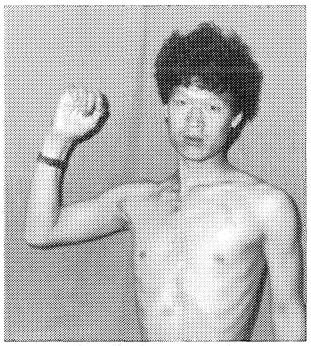

Figure 8F Nine months after surgery he continued to regain function.

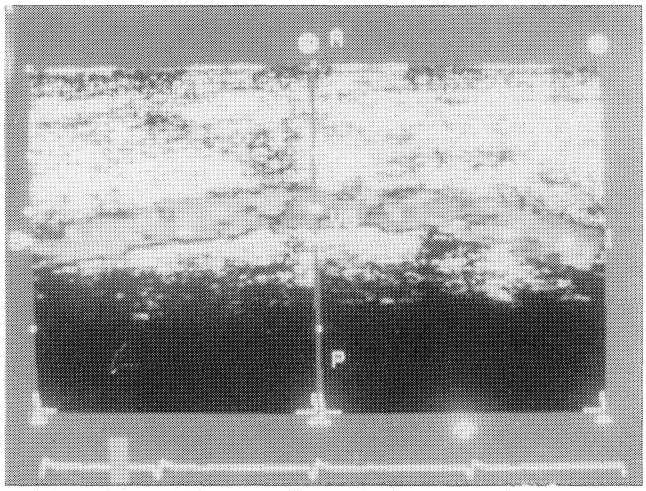

Figure 8G Ultrasonic imaging shows patent graft.

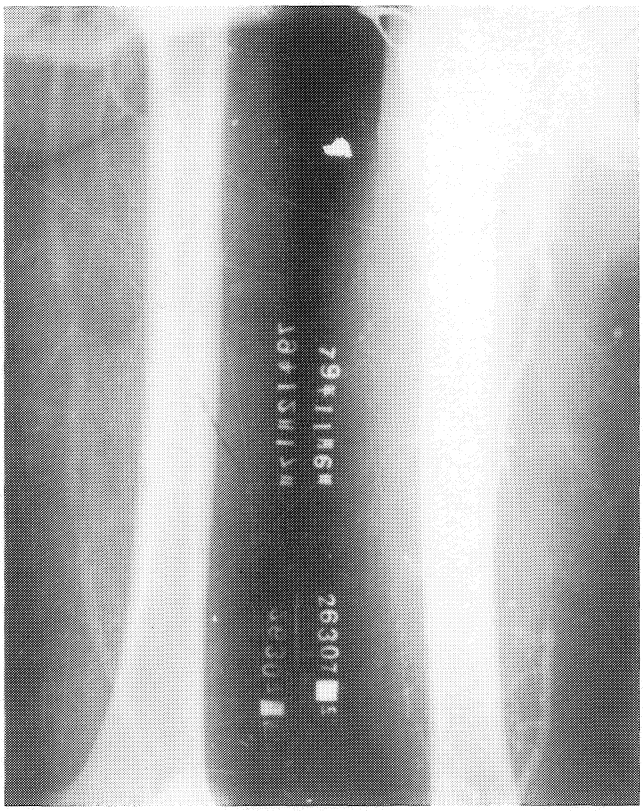

Figure 9A Preoperative (right) and postoperative (left) arteriograms.

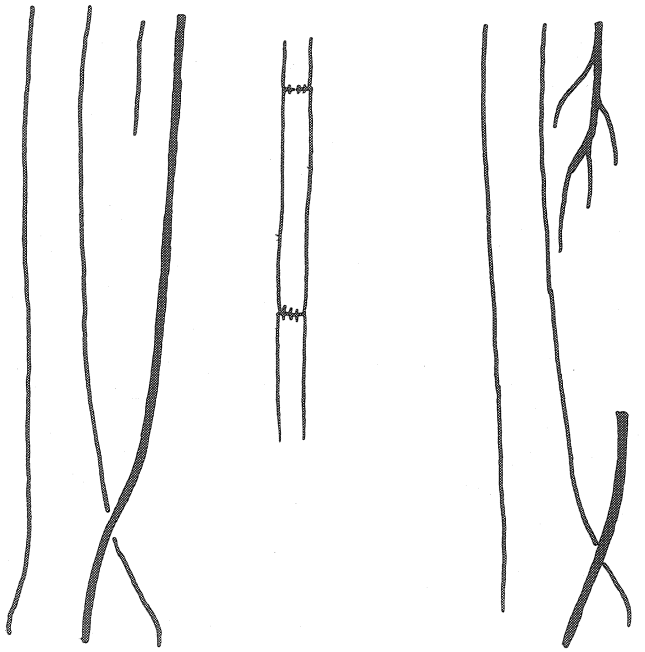

Figure 9B Illustrations for preoperative (right) and postoperative (left) arteriograms.

Figure 9

(Case 7) A twenty-three year old male had ischemic pain after repair of his femoral artery. A second operation was performed with use of autovein graft (two centimeters) At eleven years follow-up he showed excellent function and a patent graft.

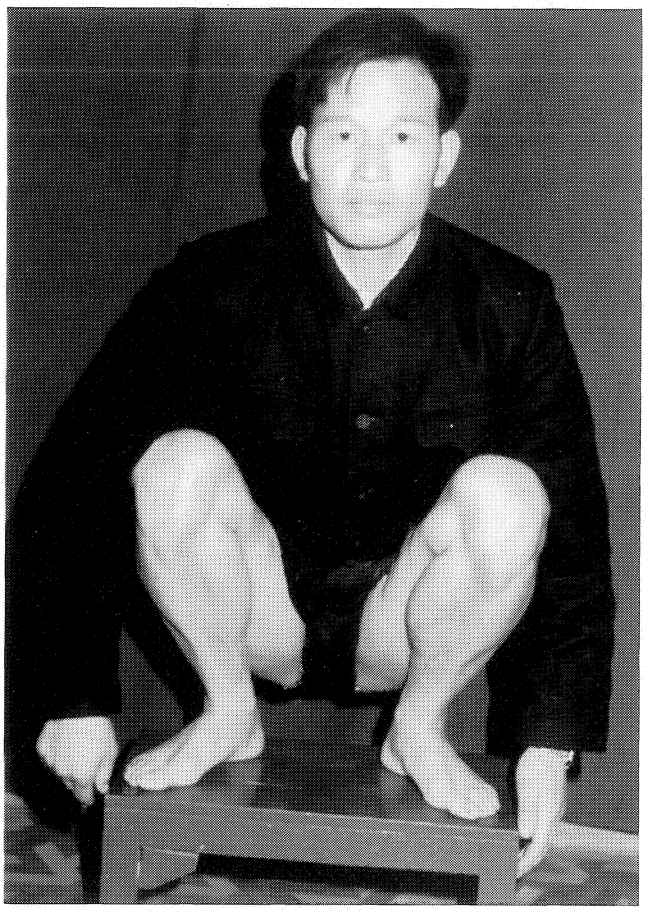

Figure 9C Eleven years postoperative.

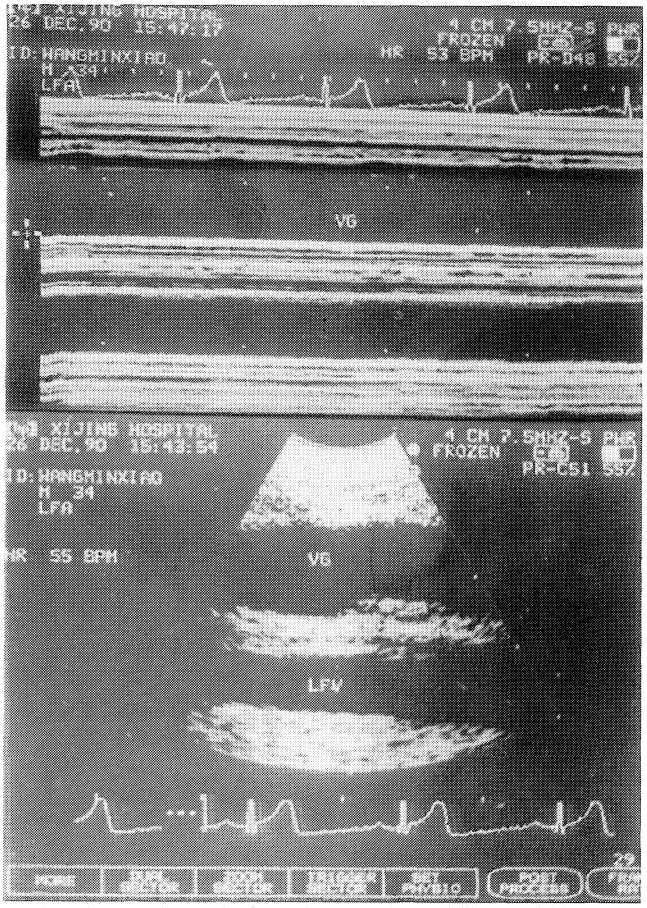

Figure 9D Doppler-ultrasonic imaging shows patent graft.

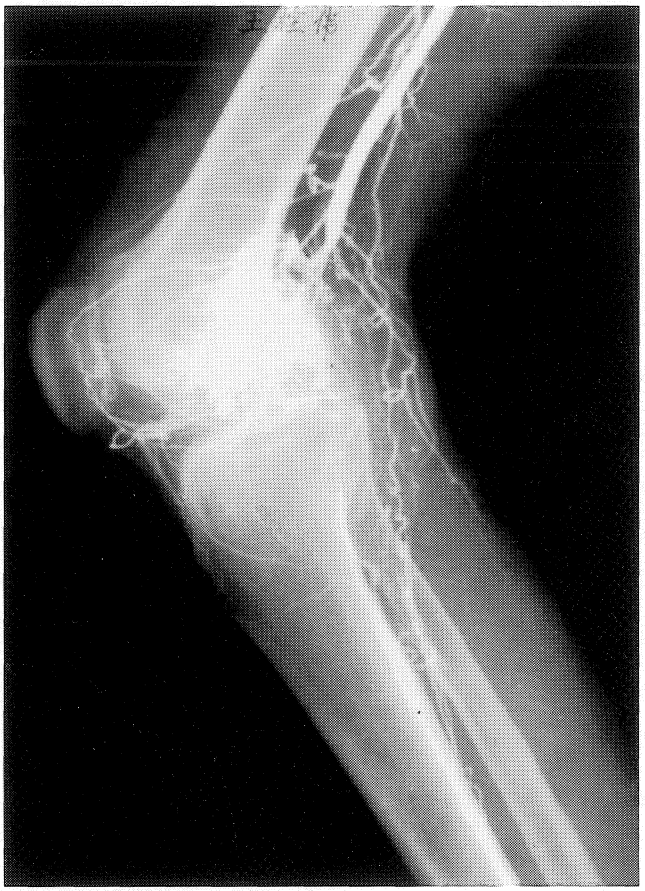

Figure 10A Preoperative arteriogram.

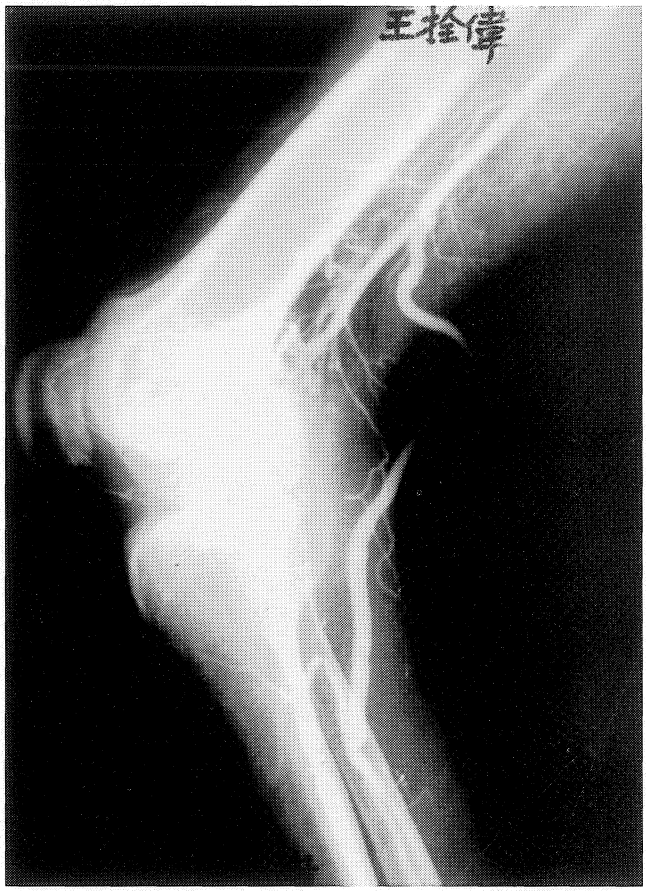

Figure 10B Postoperative arteriogram.

Figure 10
(Case 8) A thirty-two year old male had ischemic pain and contracture following repair of a popliteal artery injury. Reoperation was performed with use of autovein graft bypass (two centimeters). At 9 months follow-up he has satisfactory circulation.

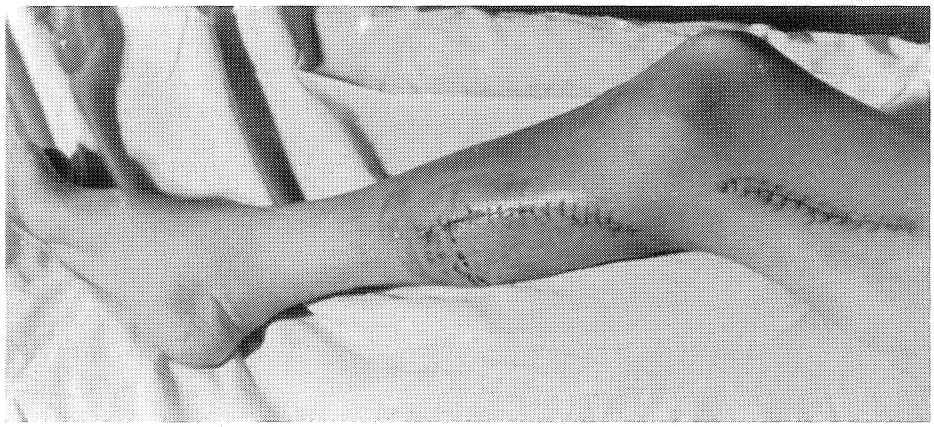

Figure 10C Postoperative appearance and incision

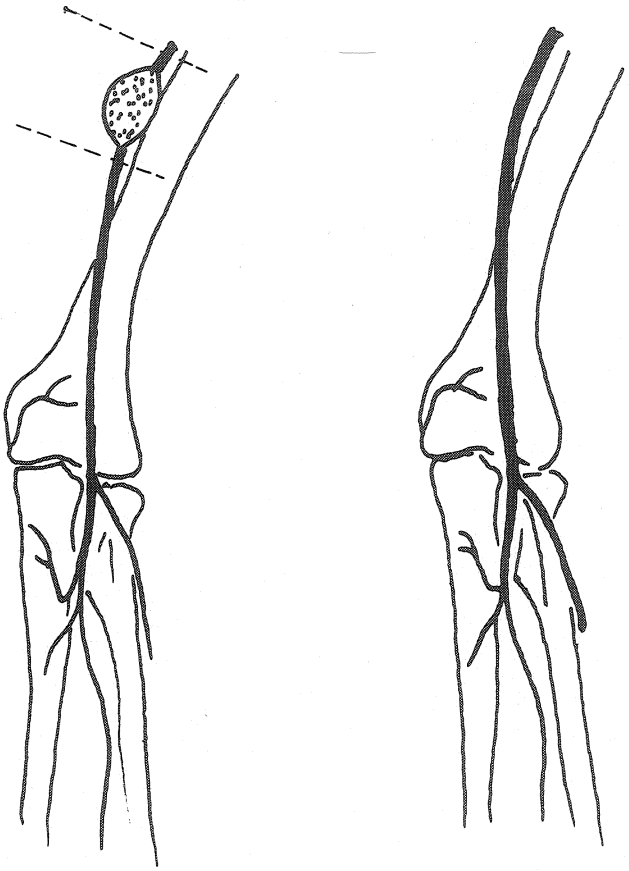

Figure 11A, 11B Illustrations for aneurysm excision and anastomosis.

Figure 11C 26 years postoperative.

Figure 11
(Case 9) This thirty year old male had pseudoaneurysm formation at the anastomosis site of a brachial artery repair. Excision of the aneurysm and reanastomosis was performed. The patient had excellent function at twenty-six years follow-up.

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TREATMENT OF DIFFICULT FRACTURES AND NONUNIONS OF THE HUMERUS AND ELBOW WITH A MODIFIED KUNTSCHNER NAIL

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Byron P. York, Jr., M.D.**

ABSTRACT

A series of fourteen difficult fractures and nonunions of the humerus and elbow have been treated over a period of twenty-eight years with a modified Kuntscher Nail. A total of twenty-six operative attempts had been previously made in this group of fourteen patients. One patient had eight failed surgeries prior to treatment. Slots were placed along the spine of the nail for transfixion with screws. In two instances additional modification of the Kuntscher nail was made by attaching a plate to the end of the nail for fixation to the ulna after retrograde insertion into the humerus. One such device was used to fuse the elbow. The other was used to stabilize a low nonunion in which the elbow was already fused.

Union was obtained in nine cases with failure in five. Four of the failures united with one additional surgery. The one failed case had a surgical neck fracture which was eventually treated with a Neer prosthesis. The method described may not be superior to other methods; however, it can be successful in obtaining union in difficult elbow and humerus fractures or nonunions resulting from multiple failed prior procedures.

INTRODUCTION

Treatment of humeral nonunions utilizing intramedullary rods without fixation to bone has shown less success than plate fixation[1,2,3,4,5,6,9,10,11,13]. There are few references in the literature describing the use of transfixed intramedullary rods in the treatment of fractures and nonunions of the humerus[7,12,14]. Results have generally been good.

This series of cases is being reported because the authors have found this method to be successful in the treatment of complex fractures and persistent nonunions of the humerus. It has been most helpful in nonunions with many previously failed procedures. In the last twenty-eight years, fourteen complicated nonunions and fractures of the humerus and elbow have been treated with a slotted Kuntscher nail transfixed with screws above and below the fracture site. The construct is coupled with autogenous bone grafting. The first time this technique was utilized was in 1962 by Dr. I.S. McReynolds. The patient had eight prior operative attempts to obtain union following a humerus fracture. Using this method he obtained union on the first attempt.

MATERIALS AND METHODS

Rod Construct

In all cases a Kuntscher nail was modified by grinding slots at intervals across the spine of the rod (Figure 1). In two cases a plate was fixed to the distal rod for fixation at the ulna during elbow fusion (Figure 2). In the early cases the rods were custom made (Howmedica, 359 Veterans Boulevard, Rutherford, New Jersey 07070) with slots placed where needed. Later, a standardized nine or ten millimeter (mm) Kuntscher rod was furnished by the manufacturer with the slots placed one centimeter apart.

Operative Technique

After a standard surgical approach the nail was inserted through the greater tuberosity in nine cases. Retrograde insertion was performed in five cases. When the elbow was beyond salvage the nail was inserted retrograde through the olecranon. In two of these cases an additional plate was attached to the ulna from the rod at the angle of the fusion desired (Figure 2). In one case the rod was inserted through the nonunion site.

Minimal periosteal stripping is necessary with this technique; only the fracture or nonunion site is exposed for bone grafting. This is in contrast to the extensive exposure required when using a plate for fixation. Reaming was necessary in one patient when the shaft diameter was too narrow. Reaming was chosen in another patient who had persistent nonunion after rodding. Removal of the smaller rod, reaming and placement of a larger device eventually led to union. In the other cases, a nine or ten mm Kuntscher nail easily fit inside the medullary canal without reaming. Therefore the rods were inserted unreamed in hopes of preserving endosteal blood supply.

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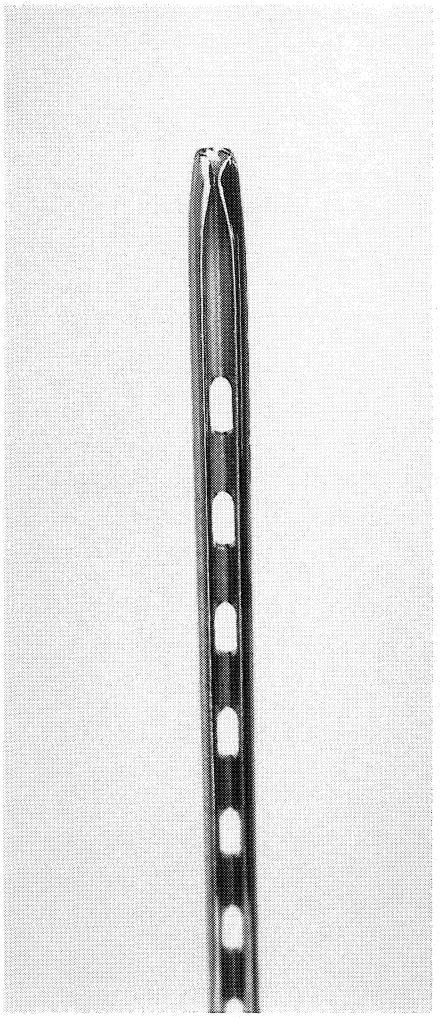

Figure 1
Standard Kuntscher rod with one centimeter slots ground at one centimeter intervals across the spine of the rod.

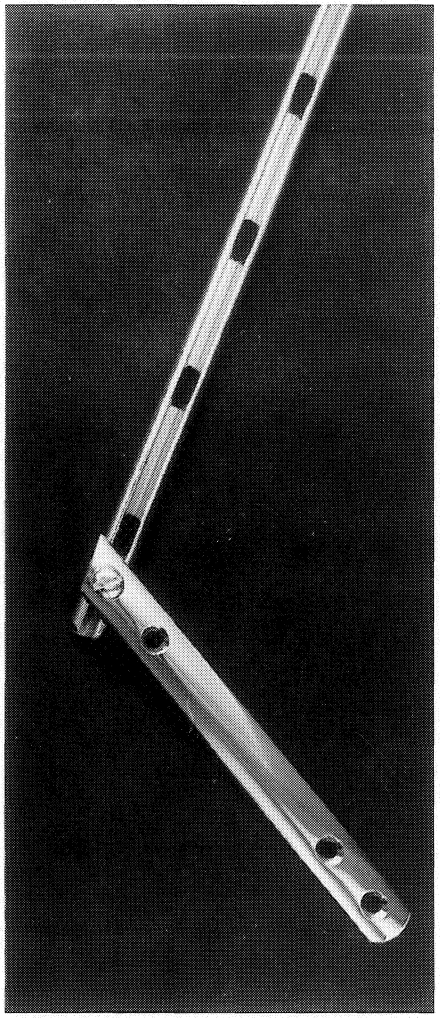

Figure 2
Modified Kuntscher rod with attached plate for elbow fusions. The plate is attached to the ulna after retrograde insertion.

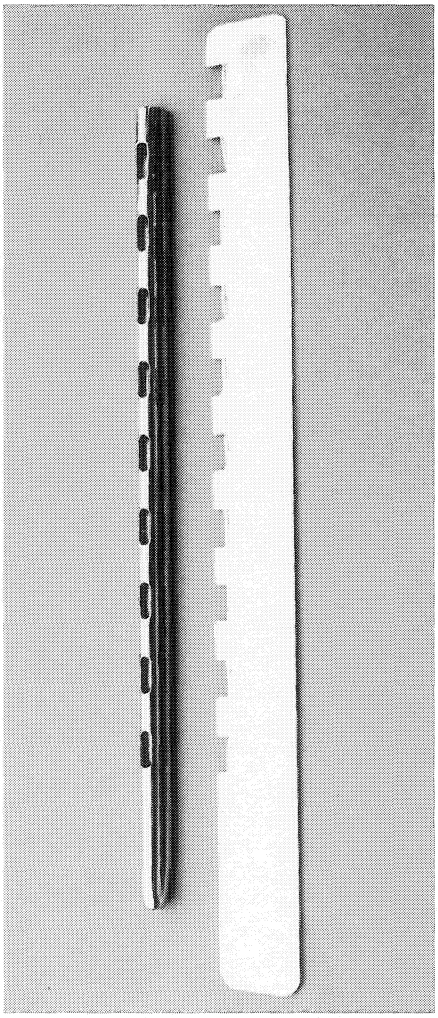

Figure 3
A custom made rod with homemade metal template used to assist interlocking screw placement.

Autogenous bone grafting was used in twelve of fourteen cases. The authors believe that this is necessary in the treatment of these difficult fractures and nonunions.

Location of the slots for fixation of the rod with interlocking screws is accomplished in various ways. In general, drilling through the slots is not difficult. Slots are easier to find than holes due to their increased size. Because the slots are made through the relatively thin metal of the Kuntscher nail, considerable leeway is available in passing the screws through the slots. The drill holes can be angled through the slots vertically or horizontally. Often the slots are visualized at the nonunion site, making it easy to drill through the near cortex. For slots at a distance from the nonunion site a template can be used (Figure 3). Because we had two cases where the slot was missed using the template alone, we recommend routine use of image intensification.

This method prevents rotation, distraction and angulation of the fragments, yet it is not a completely rigid system.

Patient Selection

For complete listing of patients included see Table 1. The following cases are included in greater detail.

Case 1

The patient was a twenty year old male who sustained a fracture of the distal humerus in an oilfield accident. Prior to treatment with a modified Kuntscher nail he had previously failed eight surgeries (See Figure 4a). These included two platings, three bone grafts, three pinnings with rush rods or steinmann pins and two iatrogenic lacerations and repairs of the radial nerve. Four years after injury, intramedullary rodding with interlocking

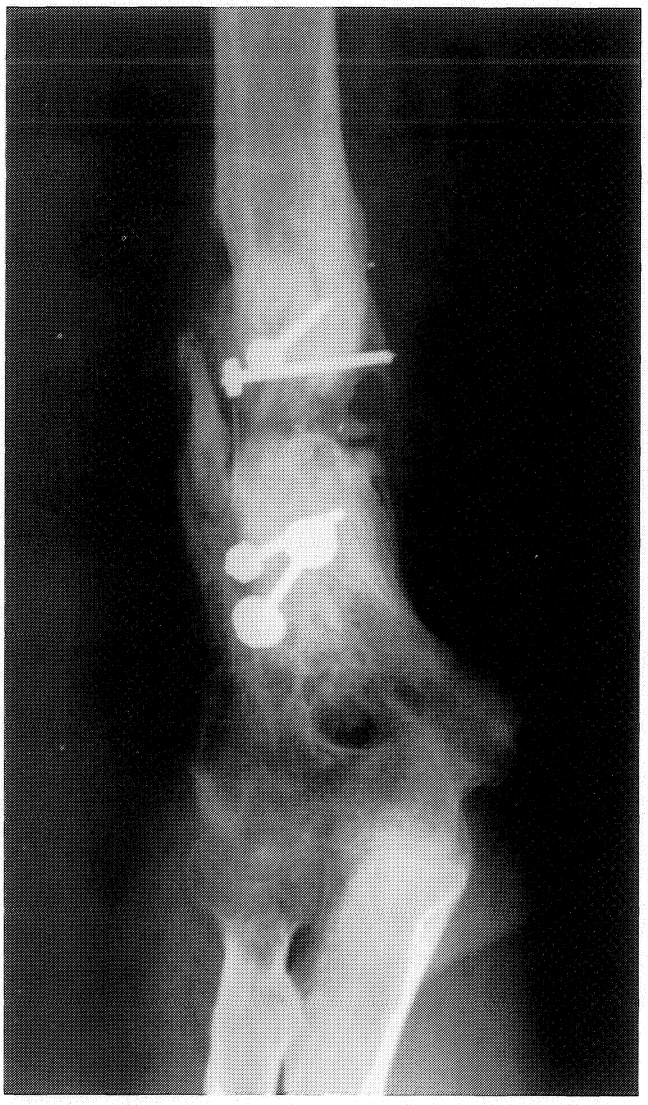

Figure 4a
AP radiograph of the distal humerus prior to treatment with a modified Kuntscher nail.

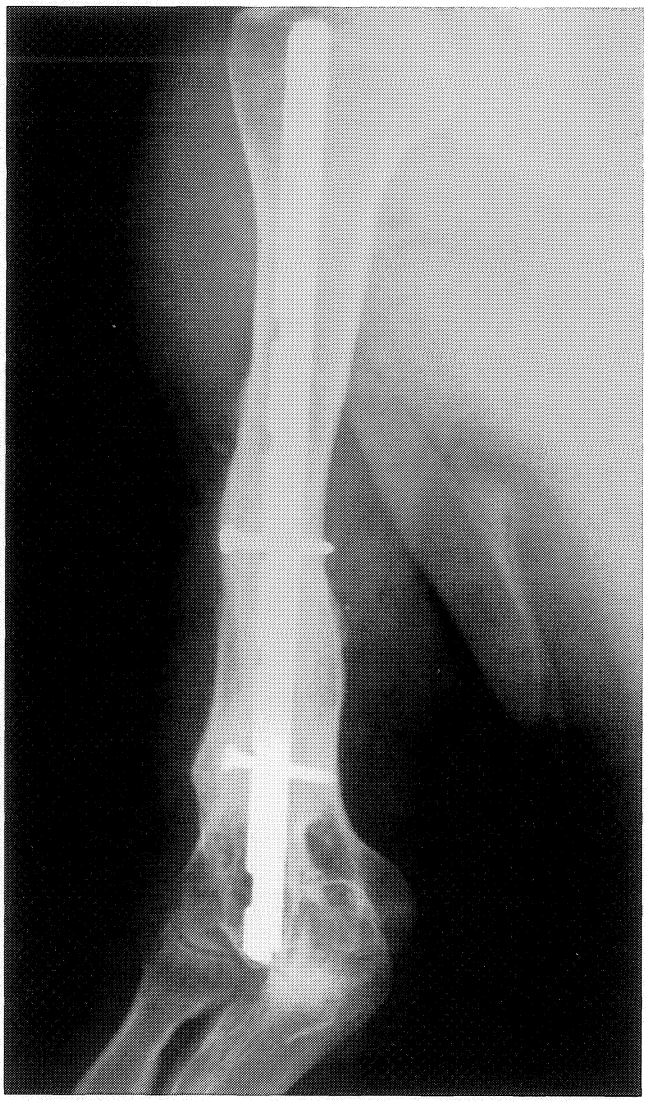

Figure 4b
AP of the humerus after treatment with modified Kuntscher nail.

screws and bone grafting was performed. He subsequently went on to union within seven months and following appropriate tendon transfers he was able to return to work as an oil driller (See Figure 4b).

Case 2

A sixty year old male automobile dealer suffered an open fracture of the distal humerus and ipsilateral proximal ulna in an automobile accident. Prior to definitive treatment he had four operations including one debridement, two pinnings, and transposition of the ulnar nerve (Figure 5a). Eleven months later a retrograde elbow fusion was performed using a plate attached to the intramedullary rod at 130 degrees. Autogenous bone graft was placed in the nonunion site as well as the elbow joint. Elbow arthrodesis was included in the procedure since the joint already displayed fibro-osseous ankylosis.

The patient developed motion at the fracture site and at reoperation the single proximal interlocking screw was noted to be loose. The screw was replaced and the nonunion site was again bone grafted. Four months later he achieved radiographic union (Figure 5b).

Case 6

A fifty-three year old woman suffered a closed humeral shaft fracture that was initially treated with compression plating and bone grafting. She progressed to radiographic union but suffered a refracture and plate failure after a fall. She developed a nonunion after plate removal (Figure 6a). Antegrade intramedullary rodding with interlocking screws and bone grafting was performed. The patient was

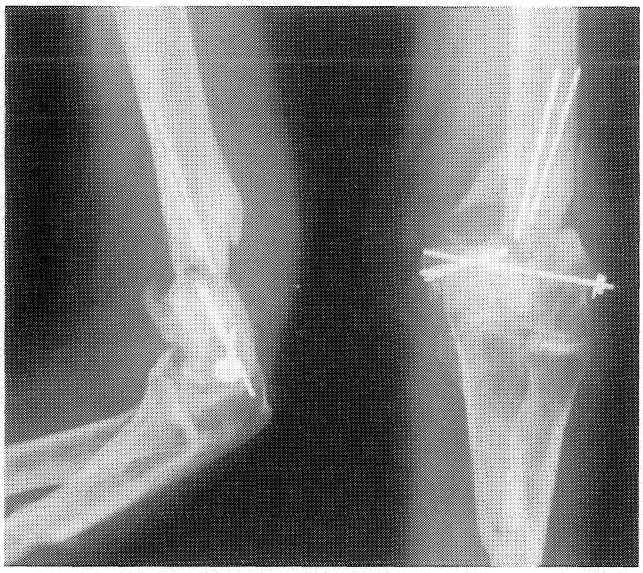

Figure 5a
AP and lateral radiographs of the distal humerus prior to elbow fusion with a modified Kuntscher nail.

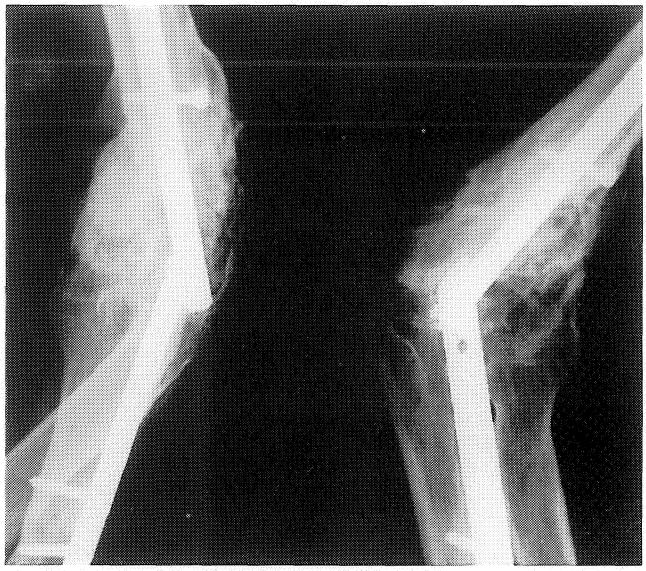

Figure 5b
Two oblique radiographs of the elbow demonstrating solid fusion and elbow arthrodesis.

Figure 6a
AP radiograph of the humeral shaft with nonunion.

Figure 6b
AP radiograph six months after rod exchange and bone grafting.

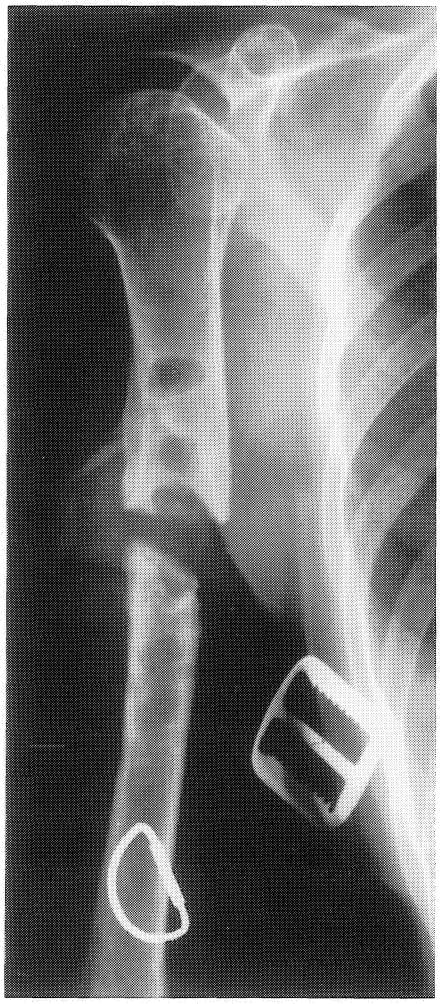

Figure 6a

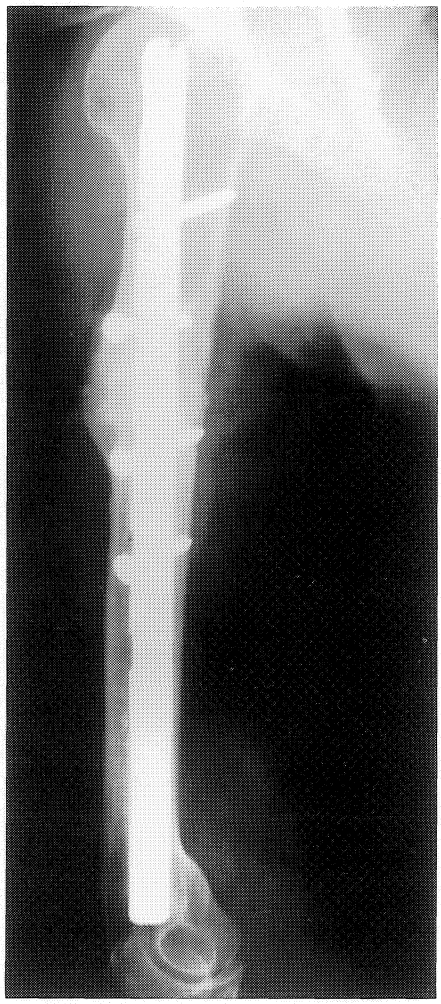

Figure 6b

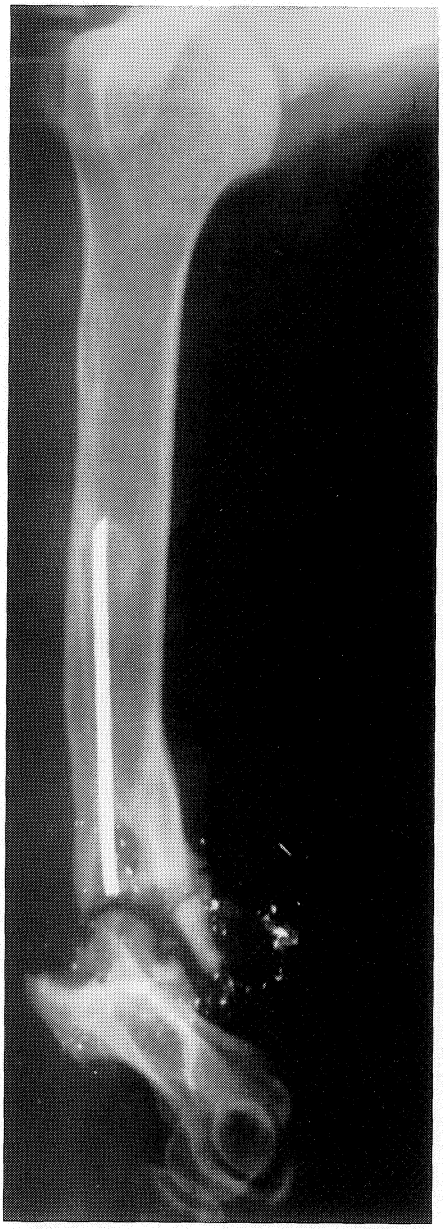

Figure 7a

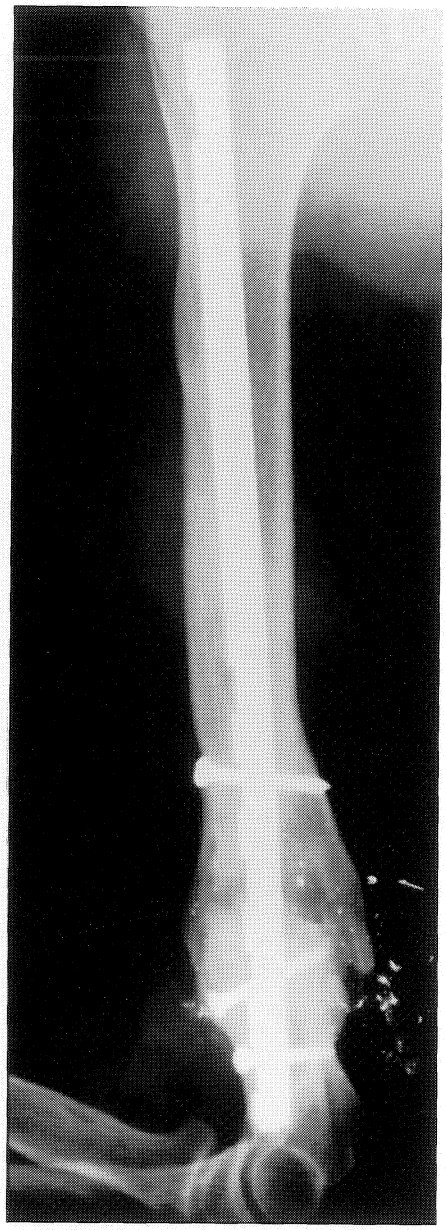

Figure 7b

Figure 7a
Lateral radiograph of distal humerus revealed persistent nonunion two years after initial injury.

Figure 7b
Lateral radiographs nine months after rodding and bone grafting.

lost to follow-up for nine months. When she returned radiographs demonstrated a persistent nonunion. She subsequently underwent rod removal, reaming, placement of a larger rod, and repeat bone grafting. At six months she developed radiographic union (Figure 6b).

Case 8

A forty-one year old male was shot in the right arm with a .44 caliber pistol during a robbery. He developed nonunion after three operations including rush rodding, hardware removal with bone grafting, and delayed bone grafting (Figure 7a). Two years after his initial injury an antegrade nailing with a modified Kuntscher nail, interlocking, and bone grafting was performed. Nine months after surgery he developed radiographic union (Figure 7b).

Case 9

A twenty-eight year old man suffered a severe gunshot wound to the elbow from a 30-06 hunting rifle. He had complete arterial laceration that required repair. The elbow was beyond salvage and the patient was indicated for arthrodesis.. A retrograde rodding of the humerus through the olecranon using a modified Kuntscher nail was perfromed (Figure 8a). We were concerned with the modest distal fixation and therefore a large iliac crest corticocancellous graft was used to supplement the back of the arthrodesis. Union occurred at three months (Figure 8b) and the patient has returned to work as a grocery checker, lab technician and house painter.

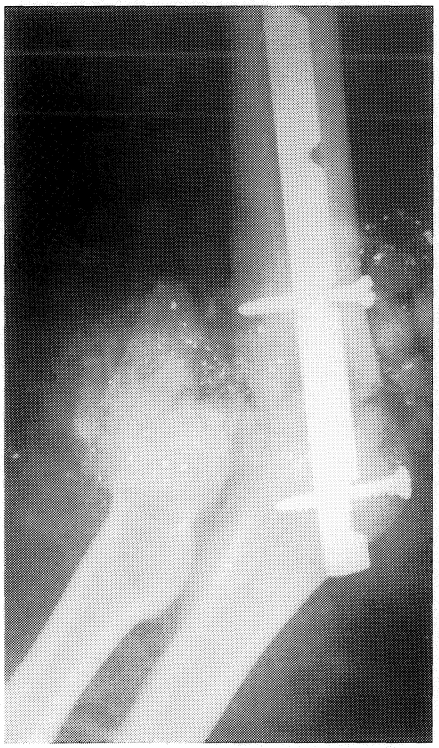

Figure 8a
AP radiograph of the elbow after initial fixation with a modified Kuntscher nail. Note relatively tenuous fixation distally.

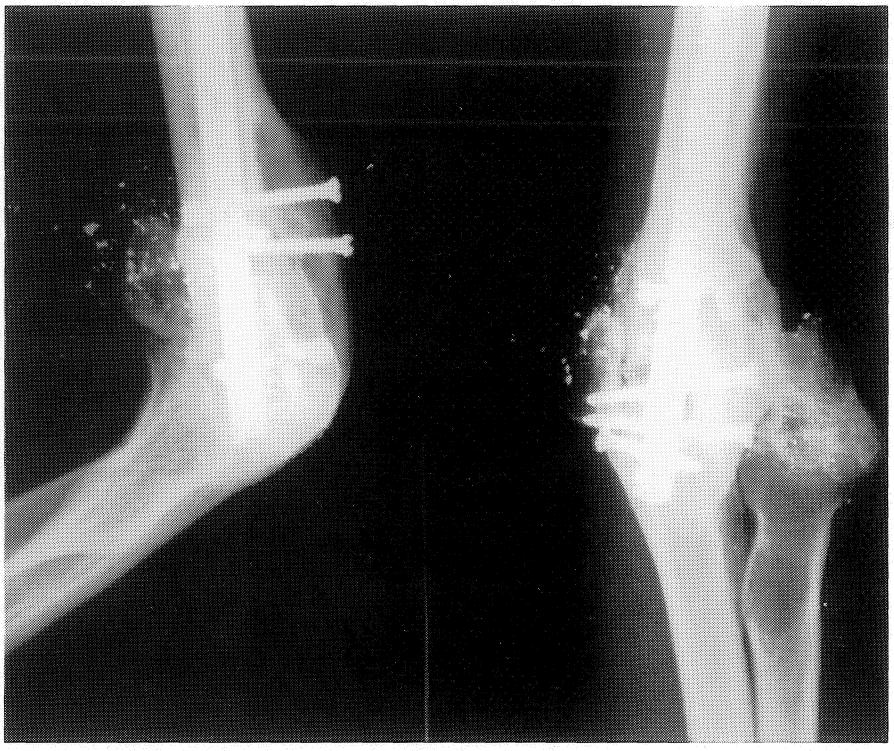

Figure 8b
AP and lateral radiographs three months after rodding with subsequent corticocancellous bone grafting and screw fixation. Note solid union.

RESULTS

The fourteen cases treated since 1962 are summarized in Table 1. Fracture union was accomplished after the first attempt utilizing the technique described in nine cases. Four cases solidly healed after one additional procedure. In one case union was never achieved and a proximal humerus hemi-arthroplasty was eventually required.

Complications

We had no infections, permanent nerve injuries or significant rotatory or angular deformities using this method. Several patients had shortening but this was not a significant problem for them. We had no breakage of rods or screws. The complicated nonunions reported here resulted from inadequate stabilization. In our five failures, adequate stabilization was lost and had to be reestablished before union occurred. Adequate fixation could not be established in one case with proximal nonunion.

One rod was removed because it projected above the greater tuberosity and caused irritation. Since then, all rods have been placed level with the greater tuberosity, or slightly below it. All patients who had insertion of the rod through the greater tuberosity regained normal shoulder motion.

DISCUSSION

There were twenty-six previous failures of operative treatment in this series. One case had eight previously failed surgeries. Union was obtained in nine cases with failure in five. Four of the failures united with additional surgery. The one failed case was surgical neck fracture of the humerus, which was eventually treated with a Neer prosthesis.

The authors believe that these results are better than they appear. The four cases which failed, but united after one additional surgery were due to preventable incidents, or to patient noncompliance. In patient No. 2, in which a plate was attached to the distal end of the rod with a screw, the screw became loose and this allowed motion between the plate and the rod. This problem was eliminated in a later case by making the plate an integral part of the rod. In patient No. 9, the lower screw was placed in the top of the slot and this allowed gravity to pull the distal bone fragment distally. This resulted in distraction at the fracture site as well as allowing motion. This was also recognized early, and success was achieved with a secondary operation. The screws should be placed in the proximal end of the slot in the proximal fragment, and in the distal end of the slot in the distal fragment. This will prevent any migration of the screws in the slots. Patient noncompliance was a factor in patient No. 6. The patient

Table 1
Summary of Cases

Case	*Age/Sex*	*Injury*	*Number of Previous Surgeries Performed*	*Procedure Performed*	*Subsequent Surgeries*	*Time to Union*	*Other Complications*
1	20/M	Distal Humerus Fracture Non-union	8	Rodding and Bone Grafting	Tendon Transfers	7 months	— —
2	60/M	Distal Humerus Fracture Non-union	4	Elbow Fusion	Screw Replacement and Bone Grafting	14 months	— —
3	63/F	Monteggia Fracture Non-union	4	Elbow Fusion	Ulnar Nerve Transposition	3 months	— —
4	48/M	Midshaft Humerus Fracture Non-union	1	Rodding and Bone Grafting	— — — — —	8 months	— —
5	41/M	Distal Shaft Humerus Fracture Non-union	1	Rodding and Bone Grafting	Rod Removal	6 months	Impingement of Proximal Rod
6	53/F	Upper Shaft Humerus Fracture Non-union	2	Rodding and Bone Grafting	Rod Exchange Bone Grafting	6 months	— —
7	35/M	Transverse Shaft Fracture	0	Rodding and Bone Grafting	Wound Exploration	6 months	— —
8	41/M	Distal Shaft Gunshot-Fracture Non-union	3	Rodding and Bone Grafting	Screw Removal	9 months	— —
9	28/M	Shattered Elbow Rifle Injury	0	Elbow fusion	Repeat Bone Graft With Screws	3 months	— —
10	59/F	Midshaft Humerus Fracture Non-union	1	Rodding and Bone Grafting	— — — — —	4 months	Transient Radial Nerve Palsy
11	66/F	Humerus Shaft Fracture Non-union	0	Rodding and Bone Grafting	— — — — —	4 months	— —
12	63/F	Humerus Shaft Fracture Non-union	2	Rodding and Bone Grafting	— — — — —	6 months	— —
13	62/M	Communited Humerus Fracture Gunshot	0	Rodding and Bone Grafting	Repeat Bone Graft	10 months	— —
14	77/M	Surgical Neck Fracture Non-union	0	Rodding and Bone grafting x 3	Attempted ORIF Eventual Hemi-arthroplasty	— — —	— —

was treated with a transfixed intramedullary rod and was lost to follow-up for nine months before returning with a nonunion. Union was achieved when the smaller rod was removed, the humerus reamed, and a larger nail placed after bone grafting. Patient No. 13 had a failure of the original treatment with a transfixed intramedullary rod. He healed after a secondary bone grafting, but no cause was found for the original failure.

The only ultimate failure in the series, patient No. 14, was likely due to the very proximal location of the fracture.

In evaluating this method, the severity and chronicity of these difficult cases must be considered. In any case with multiple previous failures, the treatment method is put to a severe test. The authors feel that obtaining union in 13 out of 14 very difficult cases is encouraging, despite the fact that four cases required secondary surgery. Although more sophisticated internal fixation devices exist, the authors will continue to use this method for selected difficult fractures and nonunions of the humerus.

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TOMÂS TORQUEMADA AND SOME ORTHOPAEDIC CPT CODING PROBLEMS

(God is a comedian, playing to an audience who is afraid to laugh!) Voltaire

William O'Connor, M.D.*
Richard I. Jacobs, M.D.

Many of you will recall reading, in the National Geographic, of the recent unearthing of the correspondence between the Vatican and Galileo at the time of their contremps in the 15th century. By a strange coincidence, we have come to possession of similar manuscripts of correspondence of Tomâs Torquemada with the Vatican in the era of Christopher Columbus. We have the correspondence of Torquemada to the Vatican, but only one of the replies from Innocent VIII. History does repeat itself, and the following letters may be of great value to any involved in Quality Assurance programs. The translation is free, not literal.

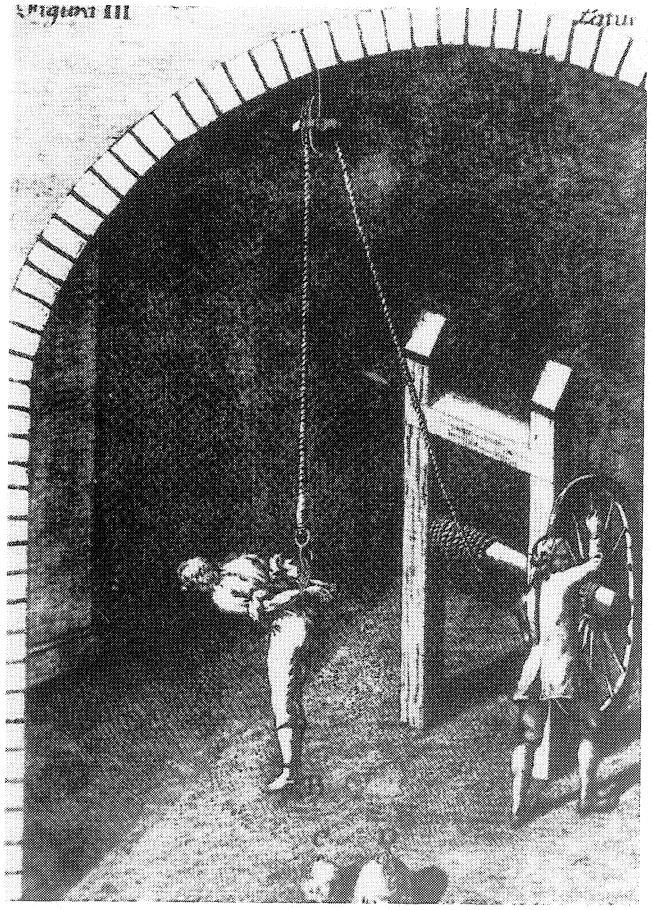

Figure 1
El Pendulo (Strappado), page 95; *Inquisition.*

July 2, 1489

Pope Innocent VIII
The Holy See
Rome
Holiness;

I have received your recent correspondence concerning funding and the necessity for documentation of my activities in the Spanish Inquisition. First, a brief outline of the history of our organization as a prelude to further discussion.

As you will recall Lucius III issued the Papal Decretal "Ad Abolendum" in 1184 A.D., which founded the Inquisition. This came to its first great fruition in the time of Gregory IX in 1231 when the Papal Inquisition was actually founded. This first furnished the boon of the death penalty for heretics in Germany and France.

In the time of Sixtus IV, at the request of our holy Catholic Kings, the Spanish Inquisition (1 November 1478) was established[3]. Sad to say, some excesses were committed in the name of God. Pursuant to this, I was named the Grand Inquisitor of the Holy Office of the Spanish Inquisition Against Heretical Depravity in 1483 A.D.. My mandate was nothing less than establishing the propriety, the "norms" of the institution[5,6]!

I now understand that because of various papal demands and budgetary stringency, you require annotation or documentation of our activities in order that we may obtain funding. In subsequent letters I will inform you of our progress and try to conform to your newly suggested coding system, the "CPT" (Catholic Procedural Terminology).

In his name, Tomâs.

Pope Innocent VIII
The Holy See
Rome
Holiness;

I wish to introduce a most excellent technique to discourage apostasy. We term it "El Pendulo" (Strappado);[2,4] wherein the heretics hands are tied behind

*Albany Medical College

him and thence to a rope suspended from a pulley in the ceiling (Figure 1). When the rope is tightened and the patient suspended from it, work is done upon the shoulders that is literally indescribable. I would like to suggest that for funding, this procedure be coded 23020, "Capsular Contracture Release". We can be quite sure that the subjects will not only praise God, but have excellent mobility of both shoulders. I hope that this meets with your approval.

Sincerely Tomâs

Pope Innocent VII
The Holy See
Rome
Holiness;

A sure remedy for those guilty of simony, we term it quemadero (the roasting kiln)[4]. With this device one can be sure that souls are speeded straight to heaven, purged and cleansed of original sin, free of intent of simony (Figure 2). We would like to code this as 16000, "initial treatment, first degree burn". This is a start. The technology of "roasting to a turn" yet eludes us, but I can promise, it is coming! This supersedes previous technology. As you will recall, earlier this year I burned the mathematician Valmes at the stake. He claimed to have solved the quartic equation. I informed him that it was the will of God that such a solution be inaccessible to human understanding!

The lesson will not be lost on others!

Another excellent device is the "Spanish Spider". A sharp traction device, it can be used to grasp various soft parts of the anatomy to apply vigorous traction[4]. These can be used cold or first brought to a white heat. It has elicited redemption in the majority of cases. We would like to code this under 20650, application of traction device (Figure 3).

A profoundly effective passive device is called a "Heretic's Fork"[4]. This device abuts upon the sternum, with a sharp supportive device lined directly beneath the mandible. It offers the benefit of rigid anterior support to the cervical spine during periods of questioning. Prolonged survival can assure quotidian use. We would like to code this as 22845, "anterior spinal instrumentation" (Figure 4). More will follow with the evolution of time.

Yours in work. Sincerely, Tomâs Torquemada.

Figure 2
The Spanish Quemadero or slow roasting of heretics in brick kilns, page 80; *Inquisition.*

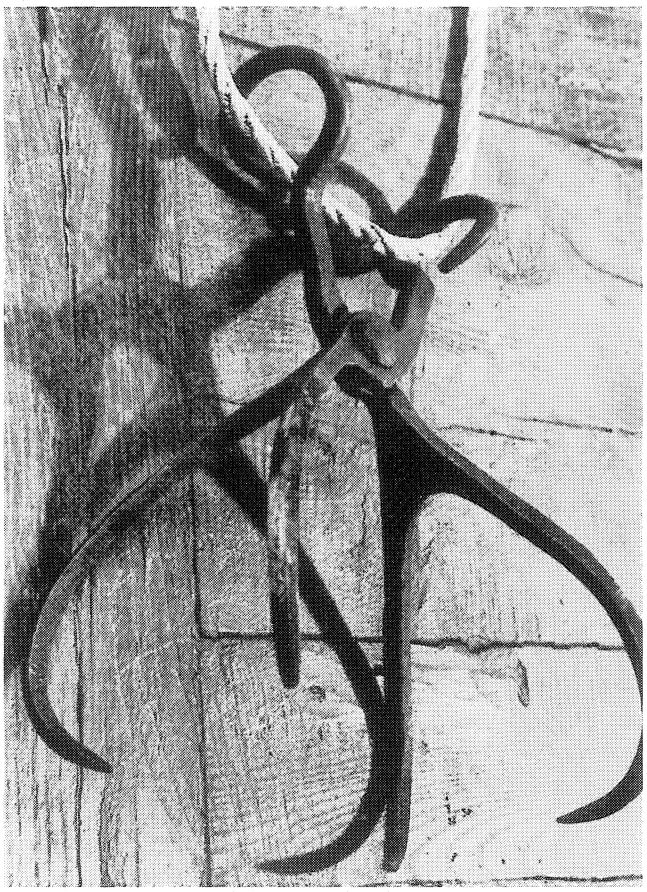

Figure 3
Spanish Spiders—Used both cold and red hot to lift victims, page 59; *Inquisition.*

Figure 4
The Heretic's Fork, page 79; *Inquisition.*

Pope Innocent VIII
The Holy See
Rome
Holiness;

Things are going quite well here. We have enlarged our armamentarium of devices for correction of the soul. A new, excellent device is called "The Skull Splitter"[4] (Figure 5). This device encircles the patient's skull with a ring of iron, with in-pointing spikes. The headband can gradually be tightened, each time to the benefit of the deviant soul. After each consecutive tightening, vigorous motion is imparted to the skull through the offices of the attached handle. The end effect, should the sinner fail to repent, is the everlasting stigma of complete loss of skull cap as the cranial vault is sundered. We would like to code this as "application of halo device" 20661.

An alternative device is termed "The Rack"[4]. Longitudinal distraction is afforded to the axial corpus as well as the four members. A simple ratchet and pawl work to sublime purpose (Figure 6). The gratitude of the subjects, having completed their treatment, is immeasurable! We would like to code this as 97122, "Traction, manual".

Sincerely, Tomâs.

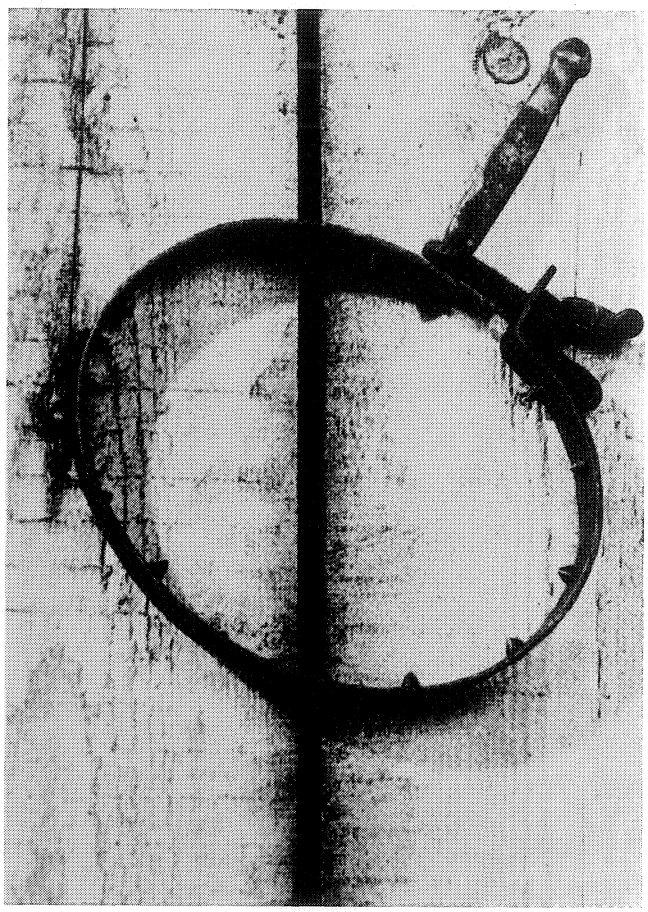

Figure 5
Skull Splitters, page 65; *Inquisition.*

And then came a final simple letter.

1984

Tomâs Torquemada
The Grant Inquisitor
Granada, Spain
Tomâs—

In the name of God, stop!

Your friend,

Innocent

And thus, the correspondence ended.

We feel sure that the proceeding documentation will help afford some direction to those involved in our present drive toward rationalization of medicine and documentation with Quality Assurance. Don't be frustrated with "QA"; although it is now at an inchoate stage, we now see what can develop with persistence and determination!

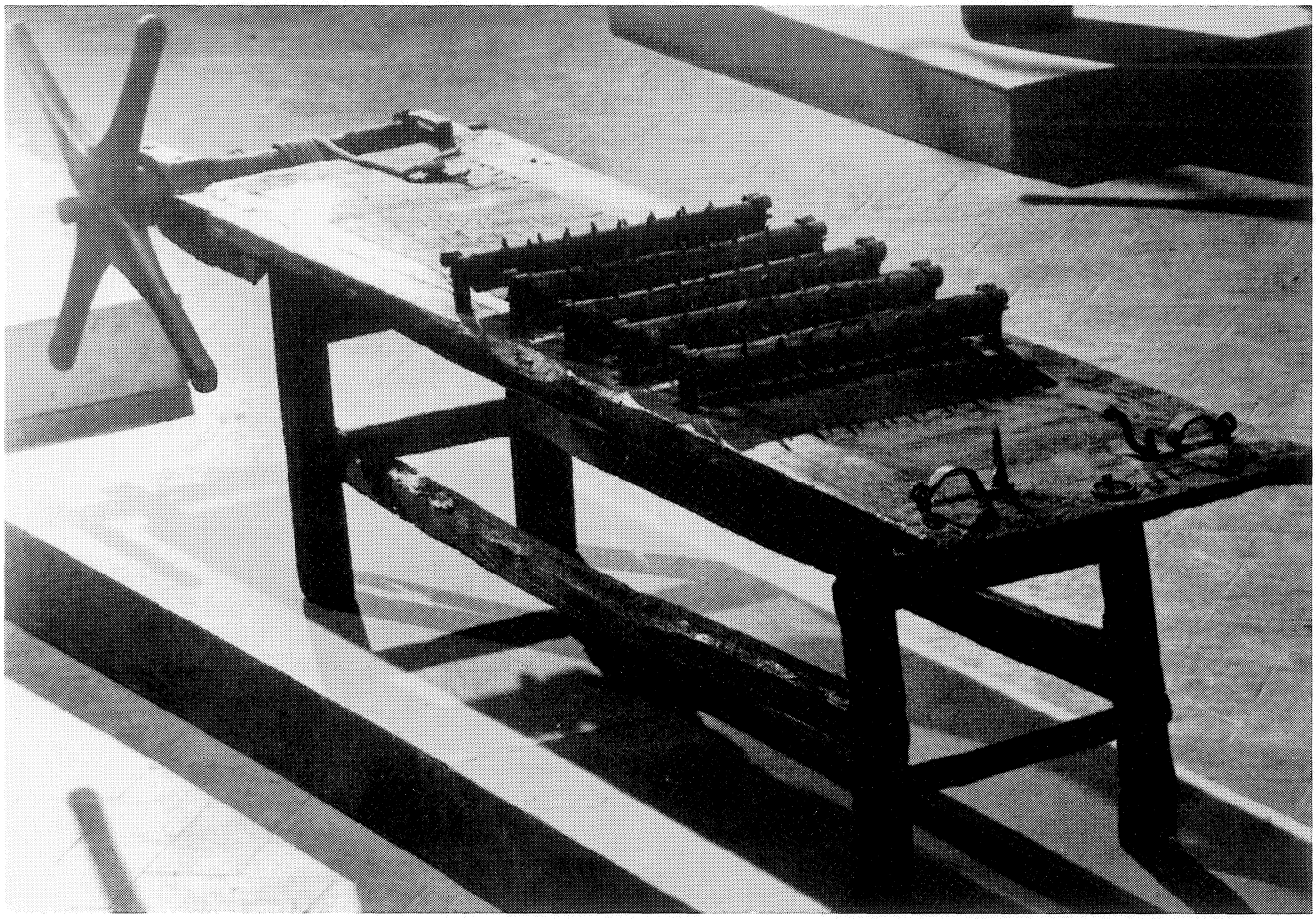

Figure 6

The Rack, page 91; *Inquisition.*

POSTSCRIPT

Tomâs Torquemada died in 1498, having secured his place in history. To this day, one who is considered guilty of excesses is sometimes termed a "terrible Torquemada". (This document is respectfully dedicated to all the courageous men and women serving as chairman of resurgent quality assurance committees throughout our great country.)

We would like to gratefully acknowledge Mr. Robert Held, Certaldo, Italy, for permission to use these illustrations. He is internationally known for his efforts to abolish torture and capital punishment.

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ULNAR SUBLUXATION OF THE EXTENSOR DIGITORUM COMMUNIS TENDON: A CASE REPORT AND REVIEW OF THE LITERATURE

Robert J. Andruss, M.D.*
James H. Herndon, M.D.

INTRODUCTION

Ulnar subluxation of the common extensor tendon at the metacarpophalangeal (MCP) joint of the hand is a relatively uncommon problem in the non-rheumatoid patient. Four etiologies have been described in the literature: post-traumatic, spontaneous or degenerative, congenital, and focal epileptic dislocations[19,23]. The disorder most commonly involves an oblique or longitudinal tear of the radial sagittal fibers of the long finger[1,7,14,32]. Reported treatment options include splinting,[1,2,4,26] realignment and direct repair,[13,14] and various forms of tendon reconstruction[2-4,7,8,10,19,20,32].

We report a case of a chronic post-traumatic ulnar subluxation of the extensor tendon to the long finger, its surgical treatment, and a review of the literature surrounding this disorder.

CASE REPORT

A thirty-two year-old right hand dominant male sustained an injury to his right long finger when a truck spring fell onto his hand. He had immediate pain and swelling about the dorsal aspect of the third MCP joint. His pain persisted and became disabling. Eventually, a "tendon repair" procedure was performed at another institution. Postoperatively, he was immobilized in a short arm cast for four weeks. Despite treatment, he had no relief of his symptoms and continued to complain of pain and "locking" of the MCP joint of his long finger. He denied any symptoms prior to the above mentioned injury.

On examination, a healed 1.5 centimeter tranverse scar was noted over the MCP joint of the long finger of his right hand associated with mild soft tissue swelling. There was no joint effusion or sign of infection. He had full active and passive ranges of motion of the MCP joint. There was, however, a visible and palpable "snapping" of the extensor tendon as the MCP joint was extended. The tendon was visibly subluxated into the ulnar gutter between the metacarpal heads of the long and ring fingers (Fig. 1). There was no pain or tenderness on the palmar side, and no triggering of the flexor mechanism. Motor and sensory examinations were within normal limits.

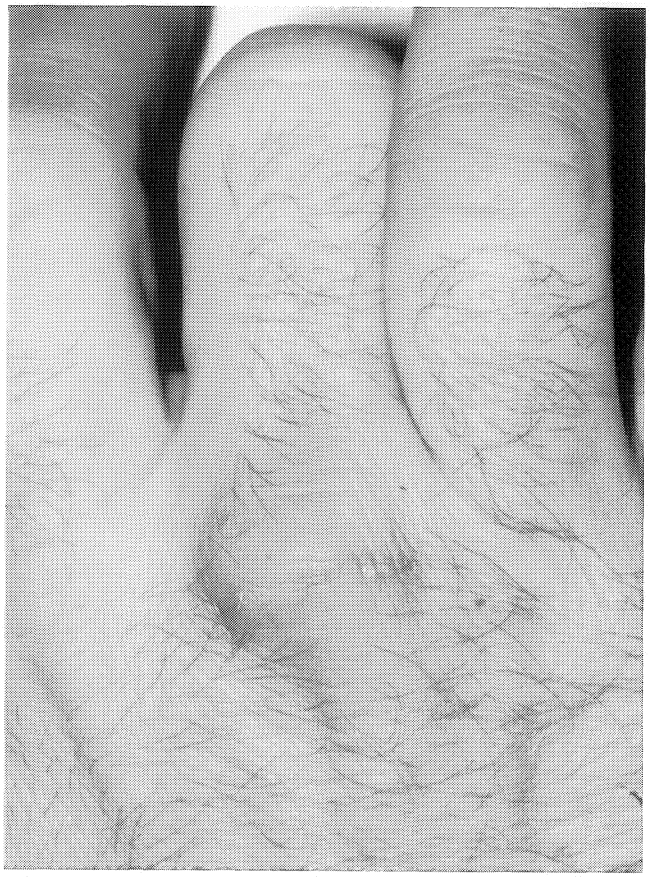

Figure 1
Preoperative photograph of the dorsal aspect of the long finger MCP joint of the right hand. Notice the periarticular swelling and ulnar displacement of the extensor tendon.

Surgical exploration revealed a markedly attenuated and scarred radial sagittal band with an ulnarly dislocated extensor tendon (Fig. 2). The ulnar sagittal fibers needed to be released before the tendon could be reduced. The sagittal fibers of the radial side were trapped in scar and also required release. An attempt was made to use a small junctura tendon between the extensor tendons of the long and ring fingers to correct the ulnar subluxation of the involved tendon. However, this caused the tendon to subluxate into the radial gutter with flexion of the MCP joint. Therefore, the junctura tendon was repaired to its origin on the ring finger. A proximally based slip from the radial side of the central tendon of the long finger extensor was created to act as a "check rein" (Fig. 3). This was dissected free, passed down beneath the radial lateral

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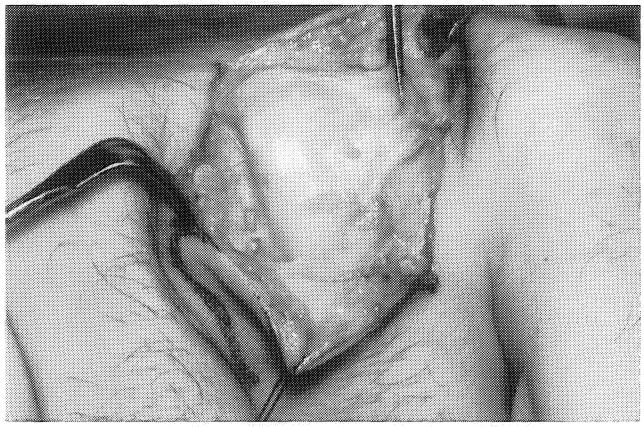

Figure 2
Intraoperative photograph of the flexed long finger MCP joint illustrating the ulnarly subluxated extensor tendon with the attenuated radial sagittal fibers. [Fingers down, index to the right.]

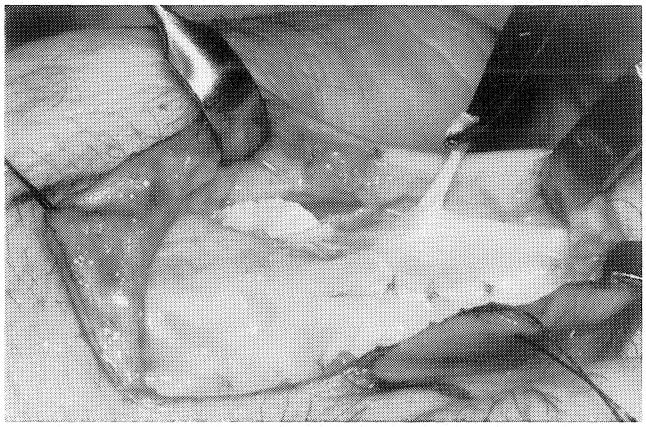

Figure 4
Intraoperative photograph demonstrating the slip of extensor tendon passed beneath the radial lateral band.

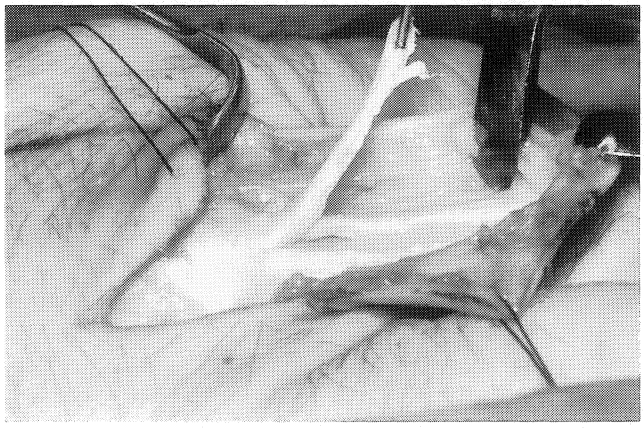

Figure 3
Intraoperative photograph demonstrating the proximally based, radial slip of the central tendon to be utilized as a "check rein" to prevent recurrent ulnar subluxation of the extensor tendon.

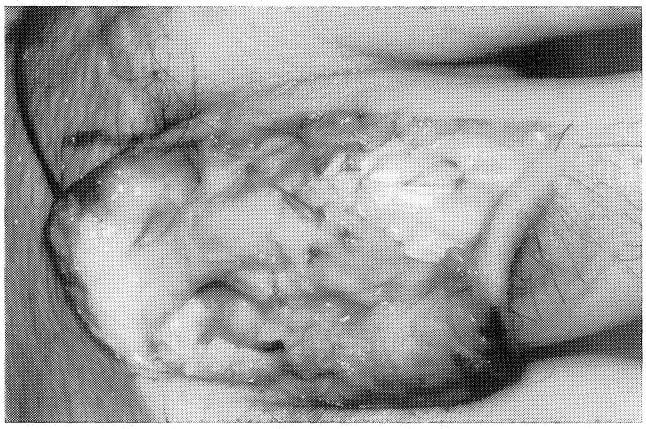

Figure 5
Intraoperative photograph showing completed reconstruction.

band (Fig. 4) and sutured back upon itself to prevent ulnar subluxation of the extensor tendon. The sagittal band of the radial side was imbricated with a "pants-over-vest" technique. The ulnar sagittal fibers were repaired to a flap created from the dorsal capsule (Fig. 5). The long extensor tendon was then stable in flexion and extension over the center of the metacarpal head.

Postoperatively, the patient was maintained in a volar splint with the MCP joints slightly flexed for four weeks. Gentle active range of motion exercises were then initiated. At twelve months follow-up, the extensor tendon to the long finger remained centered over the MCP joint (Fig. 6a and 6b). At last follow-up, he had an active range of motion of five to ninety-five degrees of MCP flexion and was pain free.

DISCUSSION

The first report of this condition was published by Legouest[16] in 1868. Sir James Paget[24] and Marsh[17] each followed with a small series in 1875 and 1896 respectively. Further reports and discussion of the pathology and treatment have followed.

Patients most often present with complaints of pain and/or swelling at the MCP joint or spontaneous dislocation of the extensor tendon. These symptoms may be associated with complaints of "catching" or "locking" and the inability to fully extend the digit. As noted above, four etiologies of extensor tendon subluxation have been reported:

(1) Traumatic dislocation is usually described as being caused by a direct injury to the dorsal aspect of the incompletely flexed finger. The digit is driven into further flexion and ulnar deviation against a strongly contracting

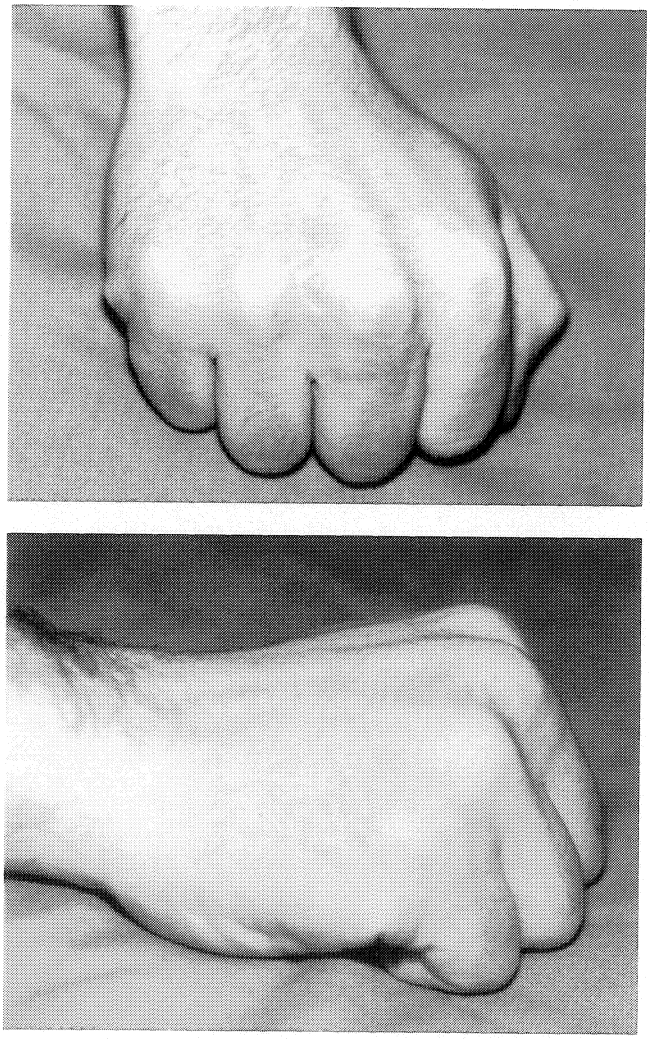

Figure 6a and 6b
One year follow-up photographs showing extensor tendon to the long finger of the left hand centrally located over the MCP joint.

extensor muscle[3,32], this creates a longitudinal or oblique rent in the radial sagittal fibers[1,7,14,32].

(2) Spontaneous or degenerative dislocations are likely due to underlying laxity of the joint capsules[8,11,12] or atrophic changes in the sagittal fibers[8,12]. Other factors include variation in the anatomic configuration of the extensor mechanism, especially as it pertains to the long finger extensor tendon passing over the MCP joint[13,14,32].

(3) Congenital extensor tendon subluxation is thought to be secondary to congenitally weak periarticular structures[19,32].

(4) Chronic, persistent myoclonic jerks in a patient with focal myoclonic epilepsy has also been reported to cause extensor tendon dislocation[23].

Combining the forty-seven patients compiled by Araki, et al,[1] with other reported cases in the literature, we were able to identify eighty-one patients with ninety-four dislocated extensor tendons. All but four were ulnar dislocations, and 73% involved the long finger. Fifty-three patients suffered traumatic injuries, twenty-four had spontaneous dislocations, with congenital and epileptic dislocations being diagnosed in the remaining four patients.

Smith demonstrated that the principal function of the sagittal band was to extend the proximal phalanx[29]. The sagittal bands and extensor hood also act to stabilize the extensor tendon in the midline over the dorsal aspect of the MCP joint[29]. Zancolli analyzed the dynamic forces contributing to this central stabilization[33]. He found the tendon to be stabilized throughout flexion and extension by the radial fibers of the hood and the sagittal bands. The radial interosseous muscles were also found to contribute through their pull on the extensor hood[33]. Tubiana and Valentin observed the sagittal bands arising from the transverse metacarpal ligament and passing dorsally to and over the extensor tendon[31]. Ishizuki showed that the sagittal band could be further disected into superficial and deep layers which encompass the tendon[13]. In this series it was noted that those patients with spontaneous dislocations invariably disrupted only the thin, superficial layer of the sagittal band just radial to the extensor tendon. Traumatic dislocations on the other hand, ruptured through both the superficial and deep layers. This usually occurred at a distance of several millimeters from the radial border of the tendon[13]. Cross-sectional anatomic studies have demonstrated that the tendon of the long finger is more rounded, sits higher on the transverse fibers, and has a relatively looser fibrous attachment when compared to the more oval tendons of the index, ring, and small finger[14,32]. Additionally, a more distal incorporation of the long finger extensor tendon into the extensor hood was noted[14]. This is felt to explain the relatively increased tendency of the long extensor tendon to sublux[14,32].

In the normal situation, the force tending to displace the extensor tendon in an ulnar direction is greatest in full extension, decreases as flexion proceeds from zero to sixty degrees, and then increases again from sixty to ninety degrees of flexion. Once ulnar subluxation has begun, the force required to prevent further displacement increases significantly[14]. Koniuch et. al. demonstrated in fresh cadaveric specimens that two thirds of the radial sagittal fibers needed to be cut before the extensor tendon would dislocate. Even then dislocation would occur only if the MCP joint was flexed ninety degrees with a minimum of sixty degrees of palmar wrist flexion[15]. Morohashi, et al, stated that for a complete dislocation of the extensor tendon to occur, the intertendinous fascia must be completely torn with a partial tear of the sagittal fibers[21]. Radial dislocation could not be created even by complete transection of the ulnar sagittal fibers with the joints of the wrist or fingers in any position[15]. This was felt to be

secondary to the tethering effect of the juncturae tendons and the normal slight ulnar deviation at the MCP joint[15]. Araki was able to demonstrate radial subluxation of the long extensor tendon in a fresh cadaveric hand by transection of the intertendinous fascia and the ulnar sagittal band, followed by flexion of the long finger MCP joint while applying resistance to all the extensor tendons[1]. The authors concluded that the unbalanced pull of the radial intertendinous fascia was able to dislocate the tendon into the radial gutter between the index and long finger metacarpal heads[1].

The majority of reported cases have undergone surgical treatment of this lesion either primarily or following failure of immobilization. However, there are reports of successful treatment of dislocated extensor tendons via splinting alone, especially in the acute setting[1,2,4,5,18,26,28]. Bunnell recommended nonoperative initial management with prolonged splinting to allow injured structures to heal[2]. In a recent paper, Ritts, et al, reported good results in two patients treated with extension splinting of the MCP joint initiated within ten days of injury[26]. Araki's review entailed a total of thirteen patients treated nonoperatively with splint immobilization[1]. Four of his own patients did well when treatment was initiated within ten days of injury. However, he also reported four treatment failures in this group. Of these, three were not immobilized until at least three months after injury. Four patients who were simply observed all had poor results. Carroll, et al, also presented seven patients who were treated with six weeks of splint immobilization[4]. Four did well, regaining full range of motion without recurrence of subluxation, while three patients subsequently required operative intervention. The length of time from injury to initiation of treatment in this series was not mentioned.

Successful surgical treatment has been reported and advocated by multiple authors with several surgical procedures being described. Haberern[10] in 1902, and later Razemon[25] and Straus[30] had success with reattaching the extensor tendon to the radial aspect of the hood via flap construction with the ulnar side sutured over the top of the tendon. Bunnell in 1948 simply translated the tendon radially on the aponeurosis without the ulnar flap[2]. Simple tendon realignment and repair of the sagittal fiber defect has been advocated as "both simple and effective" by Kettelkamp et al,[5] and is recommended by Ishizuki[13] for acute traumatic and spontaneous cases. Kettelkamp reported full, painless range of motion without recurrence of subluxation in five patients with up to five year follow-up[14]. Ishizuki performed direct repair of thirteen patients with acute traumatic or spontaneous ulnar dislocations of the long extensor tendon[13]. These authors reported good results and no recurrences with a minimum follow-up of one year. Wheeldon used a "conveniently situated" juncture tendon which he detached from the ring finger extensor tendon, crossed over the extensor tendon of the long finger from the ulnar side, and anchored into the radial side of the sagittal fiber defect to augment its repair[32]. He reported no recurrences at one year. Fitzgerald[9] and Culter[6] each reported using a strip of fascia while Bunnell[3] used a piece of palmaris longus tendon as a check rein to prevent recurrent subluxation. Elson used a distally based slip of the extensor tendon itself which he looped laterally and sutured into the deep palmar tranverse ligament in one patient with good results. A similar operation was described by Michon and Vichard, whereby the distally based extensor tendon slip was anchored to the radial side of the capsule[20]. McCoy and Winsky, in two patients, utilized a proximally based tendon slip from the radial side of the tendon which they passed deep to the lumbrical tendon and sutured back upon itself[19]. They also reported no recurrences at fifteen and twenty months follow-up. Carroll et al used a distally based ulnar portion of the extensor tendon which was passed deep to the tendon and sutured down around the radial collateral ligament[4]. Their patients all had previously failed conservative management with splinting of the MCP joint for six weeks. Satisfactory results without recurrences were observed in these three patients with five tendon dislocations.

In the case presented, the patient had already failed a direct repair of the sagittal fibers and was left with a chronically dislocating extensor tendon. As noted above, he had a scarred, attenuated radial sagittal band which was insufficient to hold the extensor tendon over the MCP joint. We thus initially attempted to correct this problem by suturing a junctura tendon from between the long and ring fingers to the extensor tendon of the index finger. However, this actually caused the extensor to dislocate into the radial gutter. The more involved reconstruction detailed above was carried out with satisfactory results.

Postoperatively, most authors recommend protective immobilization of the MCP joints for a period of four to six weeks, followed by a range-of-motion program. Recently, Koniuch has advocated the use of dynamic splinting in the immediate post-operative period[15].

SUMMARY

Ulnar subluxation of the extensor digitorum communis tendon at the MCP joint occurs infrequently in the non-rheumatoid patient and is secondary to one of four reported etiologies: traumatic, spontaneous, congenital, or epileptic. If symptomatic, patients may present with pain, swelling, a sensation of the tendon "snapping", "catching", "locking", or the inability to fully extend the MCP joint. Conservative and operative interventions have been recommended as treatment options. In the acute traumatic dislocation (less than ten days post injury), satisfactory results may be obtained with simple splinting with the

MCP joint in extension. Patients who have failed conservative management or have a more chronic or degenerative dislocation may require surgical correction. The successful surgical repair must meet two requirements: (1) the tendon must be accurately aligned over the MCP joint to diminish the forces causing the dislocation to occur, and (2) the repair must be able to withstand the ulnar forces incurred during flexion of the joint[14]. Realignment of the extensor tendon and direct repair of the radial sagittal band may be sufficient in acute traumatic, congenital, or spontaneous cases if the tissue is sufficient. In chronic dislocations or in cases with atrophic or degenerative tissue, reconstruction with augmentation of the radial restraints to the extensor hood is advised.

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BONE METASTASIS FROM RETINOBLASTOMA

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William Kapp, M.D.

INTRODUCTION

Retinoblastoma is a rare intraocular tumor of children which can occur spontaneously or be inherited as an autosomal dominant trait[5,12,19]. Advances in surgical and radiotherapeutic techniques have led to survival rates exceeding ninety percent[4,13]. Late extraocular metastases are rare; however, survivors are at a substantially increased risk for the development of independent second tumors with osteosarcoma being the most common[1-3,6,10,14,16]. We report a case of late metastatic retinoblastoma with osseous involvement. In addition, we review the genetic association of retinoblastoma and the development of second primary malignancies.

CASE REPORT

The patient is a thirteen year old black female with a history of unilateral retinoblastoma treated with enucleation at twelve months of age. Three years prior to presentation she had sustained a twisting injury to the right ankle and developed intermittent pain and swelling which became persistent over the last year. During the last six months she also experienced ipsilateral knee pain without swelling and contralateral wrist pain. She experienced a five kilogram weight loss with a normal appetite over the last six months. Her past medical history is significant for sickle cell trait and positive reactivity to PPD testing. Family history was negative for retinoblastoma, bone and neural tumors.

Ophthalmologic examination showed no evidence of recurrent retinoblastoma. An orthopaedic examination demonstrated a tender left wrist and right knee with mild warmth but no effusion. The right ankle was diffusely erythematous, warm, swollen, and tender to palpation. Diffuse mild atrophy was present in the right lower extremity. Neurovascular examination of the extremities was normal.

Initial radiographs of the right ankle demonstrated both sclerotic and radiolucent areas in the right distal tibial metaphysis and epiphysis with posterior bone destruction and a large soft tissue mass (see Figures 1A, 1B). Right knee, left wrist, chest radiographs, and computer tomography of the chest were normal. Laboratory tests revealed a mild microcytic anemia (hemoglobin 10.4), sickle cell trait, and an erythrocyte sedimentation rate of 40 mm/hour. Three phase bone scintigraphy showed increased tracer activity within the right ankle, right knee, and left wrist. Magnetic resonance imaging revealed areas of expansile osseous destruction including the epiphysis and metaphysis of the right distal tibia and femur (see Figure 2). Involvement was also seen in the left distal radial metaphysis with epiphyseal sparing.

Incisional biopsy of the distal tibial epiphysis and metaphysis demonstrated a highly cellular neoplasm infiltrating trabecular bone (see Figure 3). The tumor consisted of a sea of small blue cells with moderate pleomorphism, low mitotic index, and several atypical mitotic forms such as ring mitoses. The hyperchromatic nuclei exhibited minimal chromatin clumping and the eosinophilic cytoplasm was scant. There was no architectural substructure to the tumor such as rosetting. Immunohistochemical staining for neuron specific enolase was strongly positive. Stains for keratin, desmin, actin, leukocyte common antigen, and chromogranin were negative. Electron microscopy showed the presence of intercellular junctions and scattered neurosecretory granules. Very few differentiated features or organelles were noted. These findings were suggestive of a primitive neuroectodermal tumor such as retinoblastoma. In consideration of the patient's previous history of retinoblastoma and the multifocal nature of the lesions, the process was felt to be most consistent with metastatic retinoblastoma.

The patient is currently undergoing a combined radiation and chemotherapeutic regimen.

DISCUSSION

Retinoblastoma, an intraocular tumor of children, accounts for one to two percent of pediatric neoplasms with an incidence of approximately one in 20,000 live births. Both hereditary and sporadic forms have been recognized with ages of onset of ten and eighteen months, respectively[4,5]. Bilateral tumors occur in approximately forty percent of cases and twenty to twenty-five percent of these present with a positive family history. Bilateral disease is usually considered hereditary in nature, whereas unilateral retinoblastoma more often occurs sporadically[9,11-13].

Early detection and treatment of retinoblastoma is critical. When confined to the ocular region, the tumor is

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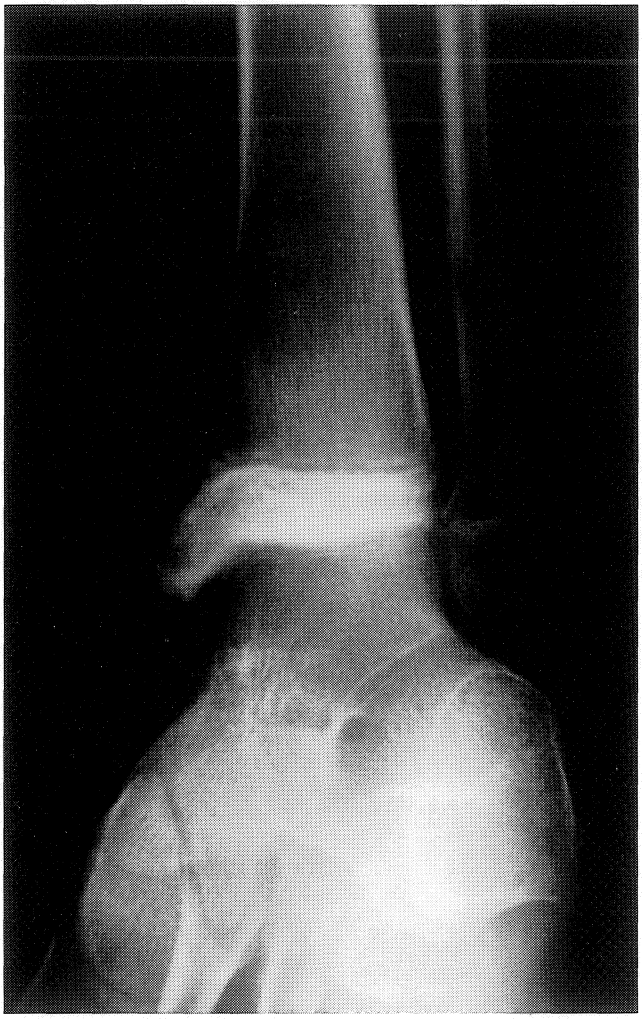

Figure 1A

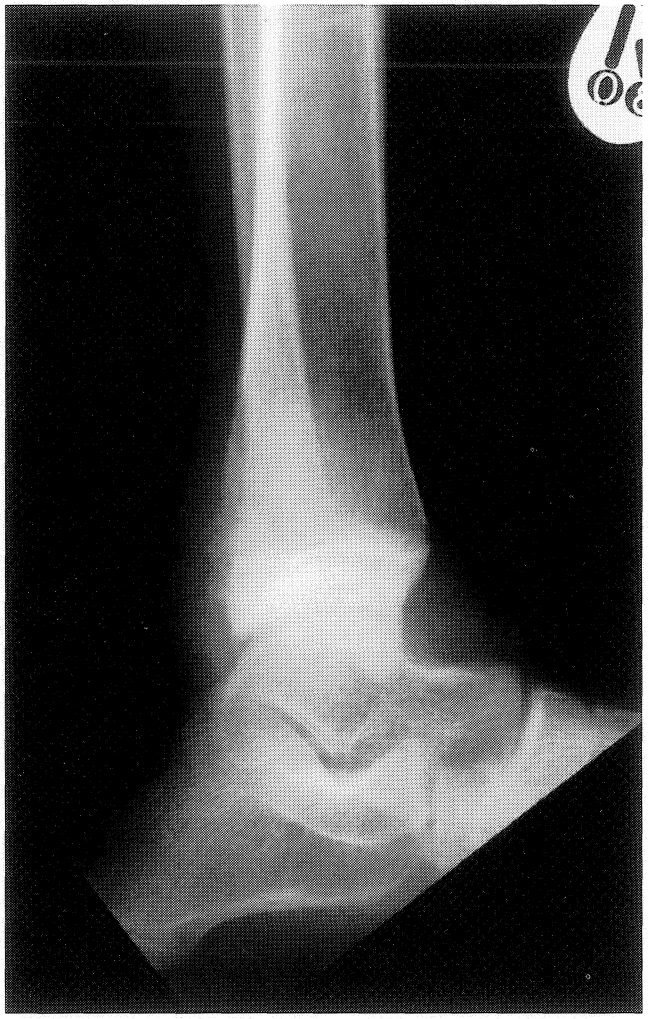

Figure 1B

AP and lateral radiographs of the right ankle exhibiting radiolucent and sclerotic changes of the tibial epiphysis and metaphysis. Posterior cortical destruction and an associated soft tissue mass with fine spicules of calcification are also noted.

readily curable and survivorship approaches ninety-five percent. Once extraocular metastases occur, the prognosis worsens and few survive metastatic disease[4,11,13]. Dissemination occurs most commonly by direct extension along the optic nerve and less commonly via lymphatic or hematogenous spread[7]. Mortality can be divided into two groups: early within two years of diagnosis with death secondary to metastatic extraocular disease, and late death secondary to metastasis of second primary neoplasms at sites within or outside a radiation field.

Segregation analysis of pedigrees in both familial and sporadic retinoblastoma has suggested that a germ cell mutation is the primary event when the disease is bilateral. This mutation is expressed in the heterozygous state resulting in an autosomal dominant inheritance pattern and an expected manifestation nearing 100%[12,19]. However, this is not the case in all families, indicating incomplete or variable penetrance of the retinoblastoma gene. The penetrance of the gene in the hereditary form of retinoblastoma varies between eighty-five and ninety-five percent[4,5,8,12]. In unilateral disease, the genetic influences are not as clearly understood; however, statistics indicate a relationship between the two forms of the disease. It is estimated that ten to fifteen percent of patients with unilateral disease actually have the hereditary form and represent the variable expressitivity of the mutant gene[9,19]. Chromosomal studies of retinoblastoma patients and their tumors have localized the retinoblastoma tumor locus to the long arm of human chromosome thirteen (q14 band)[8,12,19].

The successful introduction of radiotherapy, light coagulation, and cryotherapy in the 1930's resulted in populations of long term survivors in which the biological effects of genetically associated tumors can be observed[4,12]. Following radiotherapy, retinoblastoma patients were noted to be at risk for the development of secondary

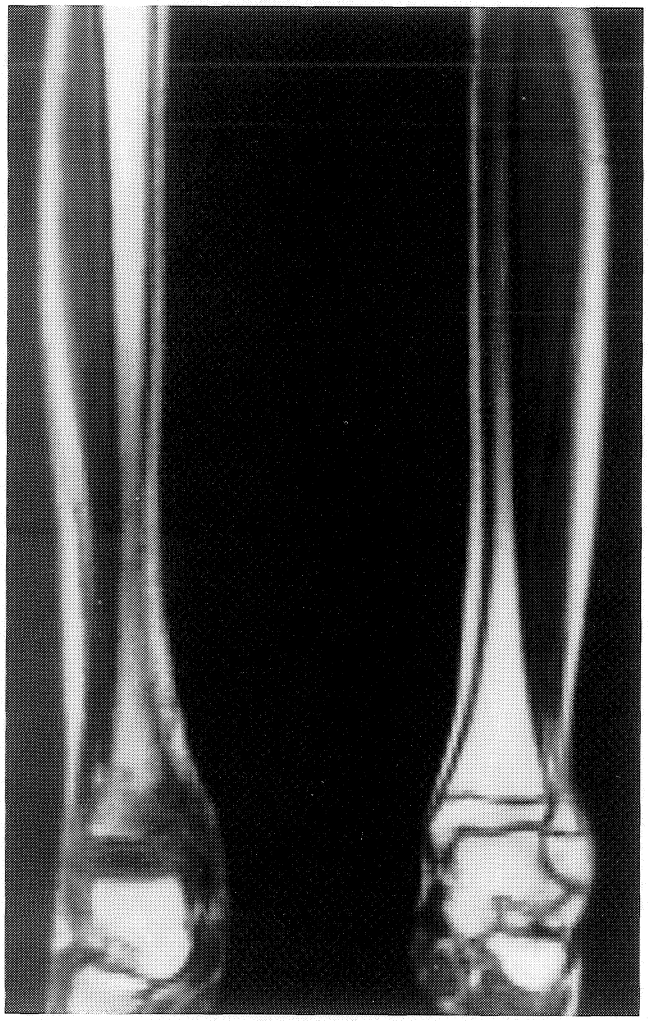

Figure 2
Coronal T1-weighted MRI scan of the legs showing destructive changes involving the right tibial epiphysis and medial tibial metaphysis.

tumors within the radiation fields[15,17]. As the population of long term survivors increased, it was recognized that survivors of genetic retinoblastomas maintain an increased incidence of secondary malignancies, osteogenic sarcoma being the most common[1-3,6,8-10,14,16]. Reports of the actuarial incidence of secondary tumors varies, and is believed to be related to the difference in therapeutic regimens utilized in the initial treatment of retinoblastoma[1,6]. Most recently, Smith et al. (1989) found a six percent incidence of secondary neoplasms at ten years, nineteen percent at twenty years and thirty-eight percent at thirty years. The latent period from treatment of retinoblastoma to the diagnosis of malignancy ranged from 5.2 to 36.2 years with the mean of sixteen years. Reports indicate that fifty to seventy-five percent of the secondary tumors occur within the radiation field, whereas the remainder are distant from the initial treatment site[3,10,18]. These secondary malignancies include, in addition to osteogenic sarcoma, soft tissue sarcoma, squamous epithelioma, Wilms' tumor, thyroid adenocarcinoma, malignant lymphoma, and Ewing's sarcoma[1,11-13]. A recent review of genetic retinoblastoma survivors indicated a 400-fold increase in the expected number of osteosarcomas when compared to the the general population[10].

The patient in the current report presented with a history of unilateral retinoblastoma treated solely with enucleation. She had no family history of retinoblastoma, indicating that her disease was most likely sporadic or spontaneous in nature. Enucleation as treatment for unilateral retinoblastoma confined to the ocular cavity is appropriate and has resulted, with careful follow-up, in long term survivors of the disease. The development of metastatic disease later than three years following early enucleation is extremely rare, and to date there have been no reports of metastatic retinoblastoma presenting twelve years following initial diagnosis[1,4,14]. The present case

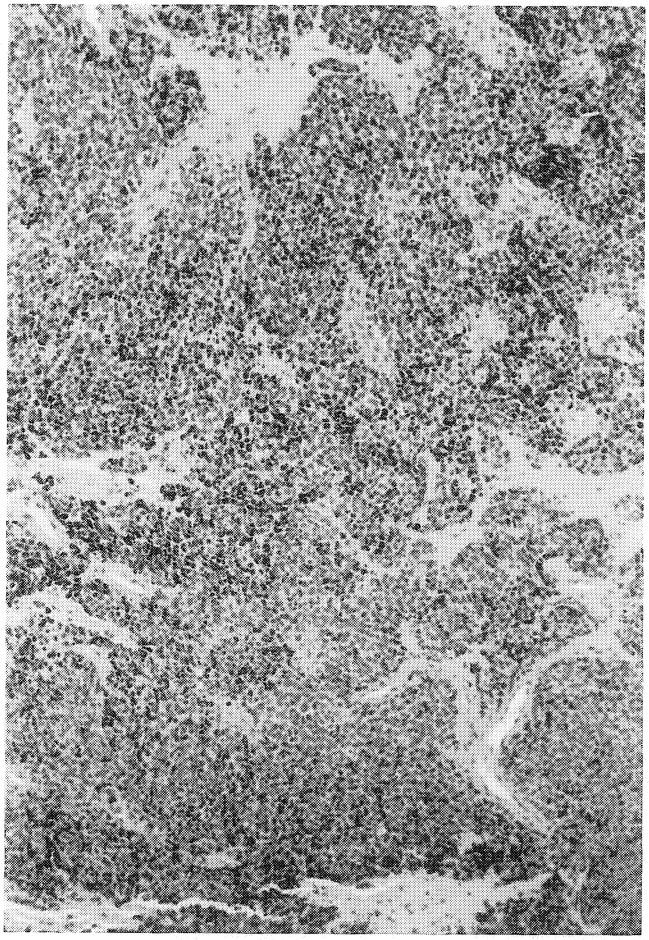

Figure 3
Histological section of the right tibial epiphysis showing numerous small cells, scant cytoplasm, moderate pleomorphism, and low mitotic index (hematoxylin and eosin, original magnification 10X).

illustrates the unpredictable nature of this rare tumor. Despite excellent long-term survivorship, late metastases can occur. Additionally, the ability of the retinoblastoma gene to predispose individuals to second primary malignancy necessitates continued long term follow-up.

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SIMULTANEOUS IPSILATERAL FRACTURE OF THE SUPRACONDYLAR HUMERUS AND FOREARM IN A CHILD: A CASE REPORT

J.E. Mumford, M.D.*

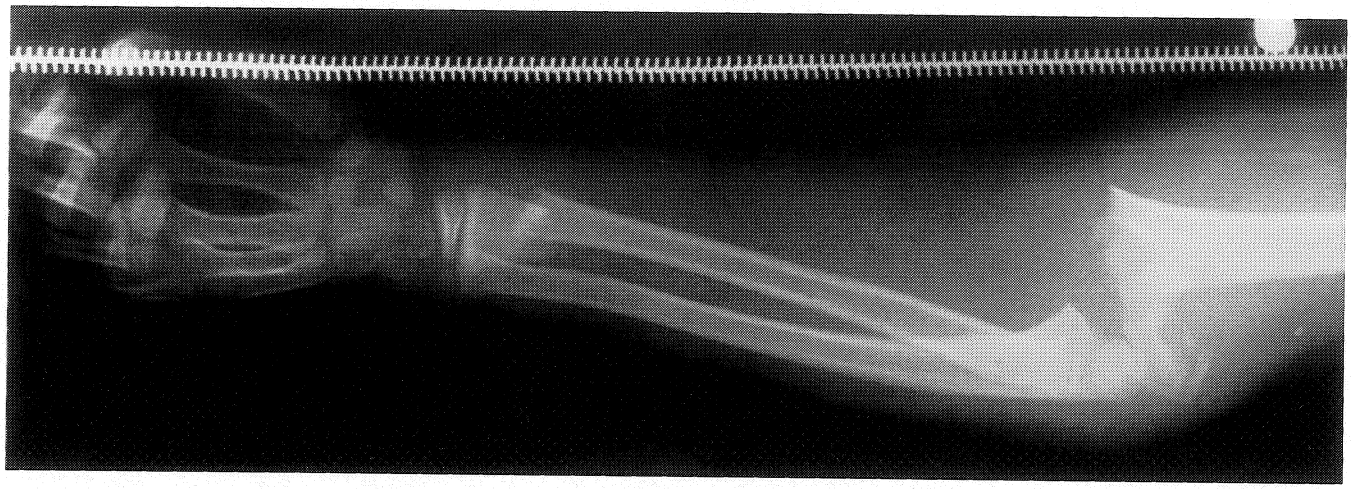

Figure 1
Anterior-posterior radiograph of upper extremity. Supracondylar humerus fracture with Type III displacement and ipsilateral distal third both bone forearm fracture with volar and ulnar angulation.

In the overall pattern of pediatric extremity fractures, the relatively high incidence of supracondylar humerus fractures is second only to fractures of the forearm[7]. Conversely, the simultaneous occurrence of these two injuries in the same extremity is considered an unusual event[6,7]. This report concerns the case of a child who sustained a simultaneous ipsilateral fracture of the supracondylar humerus and forearm, hereafter referred to as the "combined injury"[5].

CASE REPORT

A healthy six-year-old right-handed girl fell off school playground equipment and landed on an outstretched left upper extremity. She had pain and obvious deformity which prompted temporary splinting in an air cast and rapid transport to the nearest emergency facility. Upon arrival, inspection of the extremity revealed two points of angulation about the elbow, giving it an "S" shaped configuration, as well as volar and ulnar angulation of the distal third of the forearm. The digits exhibited brisk capillary refill and radial pulse was weakly palpable. The patient could fire wrist flexors and extensors as well as hand intrinsic and extrinsic motor groups. Sensation was intact to pin prick and light touch and two point discrimination was less than five millimeters. The skin was intact with no dimpling noted above the antecubital fossa. Radiographs confirmed a completely displaced (Type III)[2] supracondylar humerus fracture with a displaced fracture of the distal third of both forearm bones (Figure 1). Reduction of the humerus fracture by closed manipulative methods was carried out first. Crossed K wires, inserted percutaneously through the epicondyles, were used to secure the distal fragment. The forearm fracture was then reduced with gentle longitudinal traction. Counter traction, applied by an assistant through the proximal forearm, was used to avoid redisplacement of the fixed supracondylar humerus fracture. After reduction, a K wire was placed distal to proximal to prevent redisplacement. A well padded posterior plaster splint was applied with the elbow flexed to ninety degrees and forearm in neutral rotation (Figure 2). The patient was admitted overnight for monitoring of neurocirculatory status which remained intact throughout treatment.

Ten days after injury, the posterior splint was removed and a circumferential long arm cast was applied. The cast and pins were removed at six weeks after injury and the patient was protected an additional three weeks in a bivalved long arm cast which could be removed for active range of motion exercises. The most recent radiographs and clinical photos, nine weeks following injury, are depicted in Figures 3 and 4.

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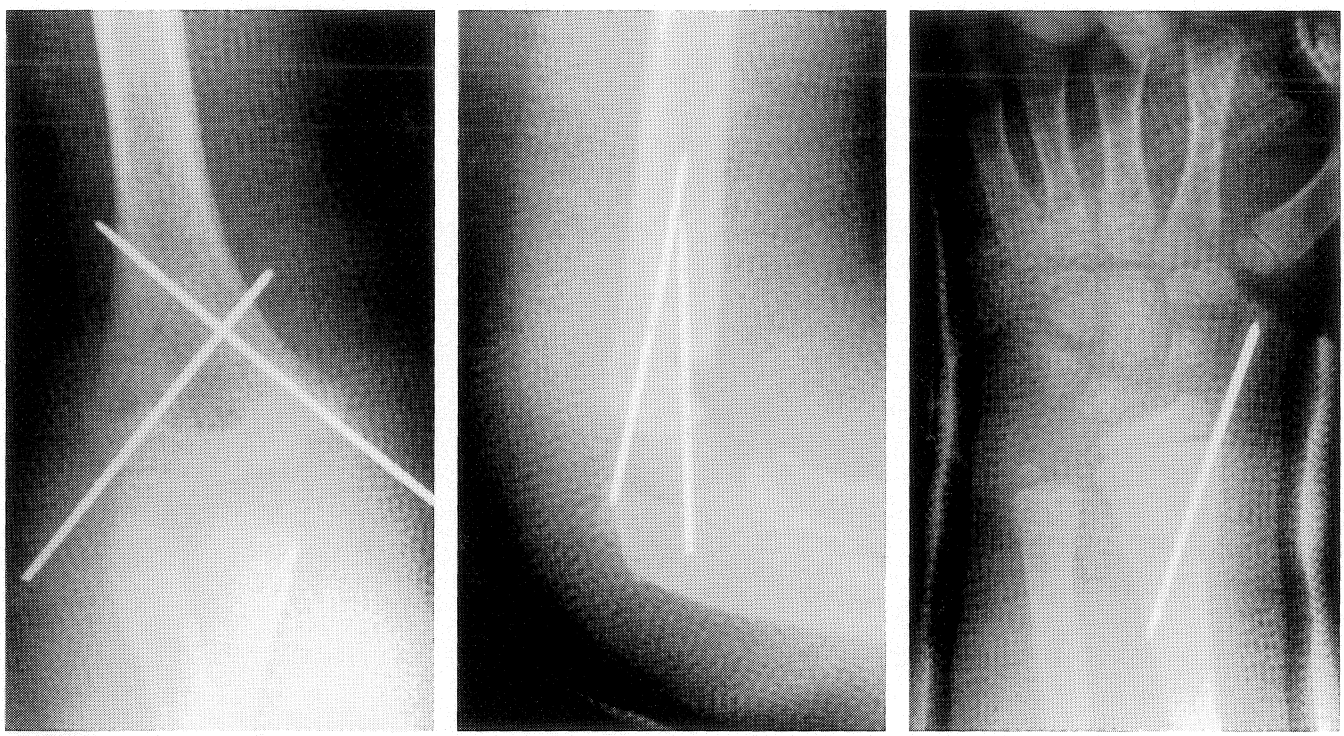

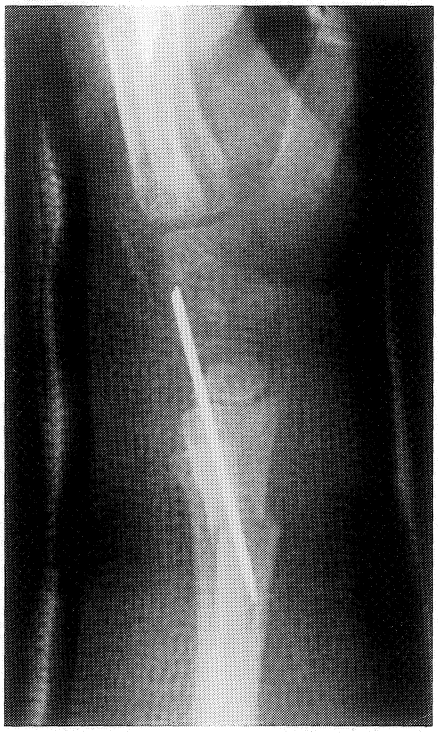

Figure 2
Postoperative radiographs in a splint.

DISCUSSION

While many large series of pediatric supracondylar humerus fractures make no mention of the combined injury, several reports have noted simultaneous ipsilateral forearm fractures accompanying five to fourteen percent of the cases[1,3,4,5,6]. In general, the supracondylar component shows complete (Type III) displacement and the forearm fracture is generally seen in the distal one third. The mechanism of injury is speculative and felt to occur after a fall of considerable magnitude on an outstretched hand with supination of the forearm, hyperextension at the wrist and elbow, and adduction of the elbow. The impact is shared unequally at the wrist and the elbow[5].

Treatment options used in managing the combined injury are varied; most series report results based on a combination of several techniques used by a variety of surgeons. Closed reduction and casting, Dunlap's skin traction, olecranon pin traction, and closed reduction with percutaneous pinning of one or both fractures have all been used successfully[4,5,6].

Though the segmental nature of the injury may cause significant neurovascular compromise at either or both fracture sites, the overall incidence of complications is the same as seen with isolated fractures of similar magnitude. One notable exception is a higher incidence of cubitus varus in patient's with a combined injury[5]. A higher malunion rate may be related to internal rotation of the distal fragment when a pronation force is applied during reduction of a distal forearm fracture[5]. This complication can be minimized by reducing the forearm first and stabilizing the injury with plaster or pins before addressing the humerus. Another option, recommended here and by others,[6,7] includes adequate reduction and stabilization of the supracondylar humerus fracture first with medial and lateral percutaneous pins, followed by manipulation of the

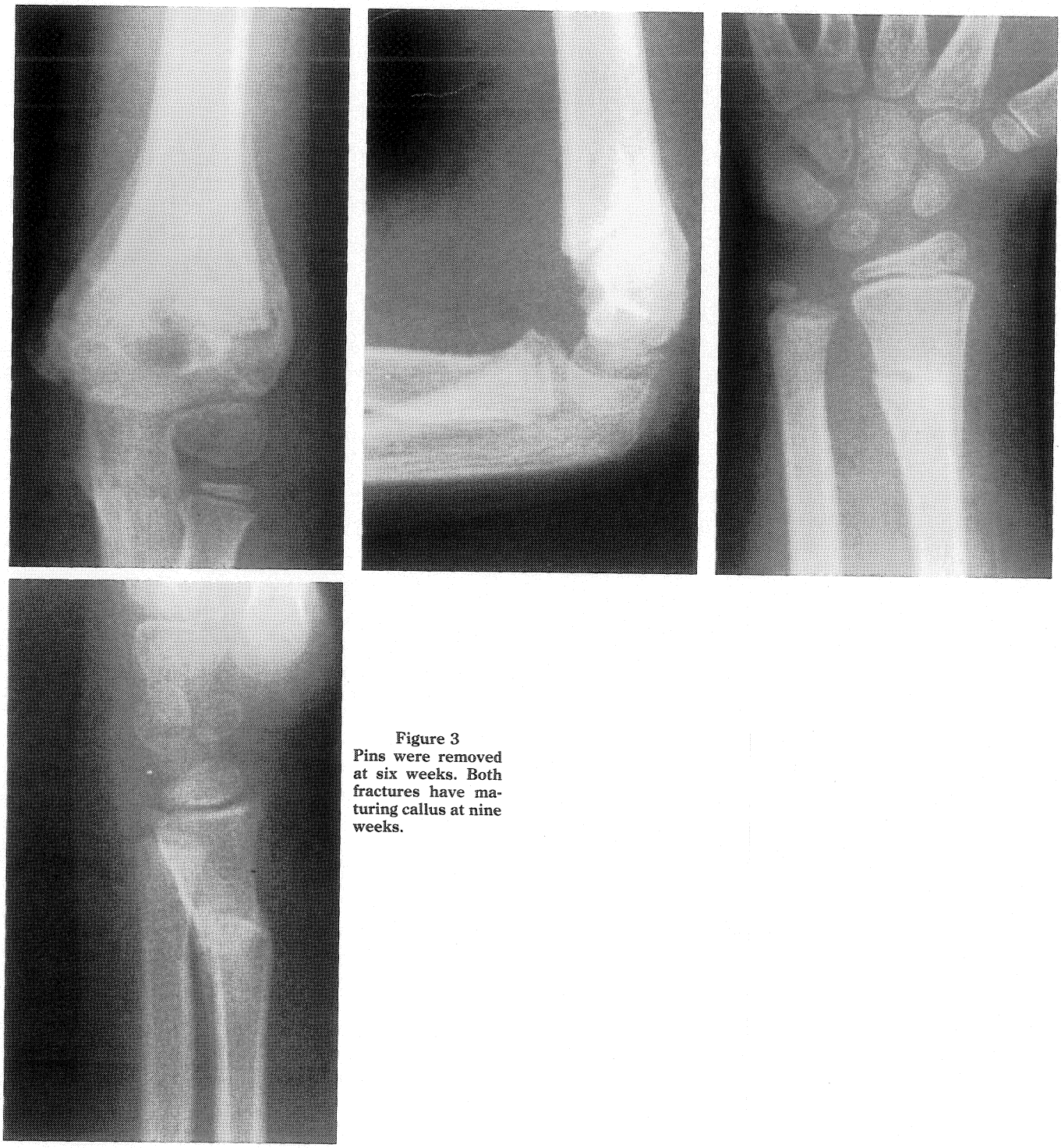

Figure 3
Pins were removed at six weeks. Both fractures have maturing callus at nine weeks.

forearm. When reducing the forearm, care should be taken to have an assistant apply counter-traction to the proximal forearm to protect the humeral fixation. As long as there is adequate reduction of the forearm, a single pin across the radius fracture provides satisfactory fixation.

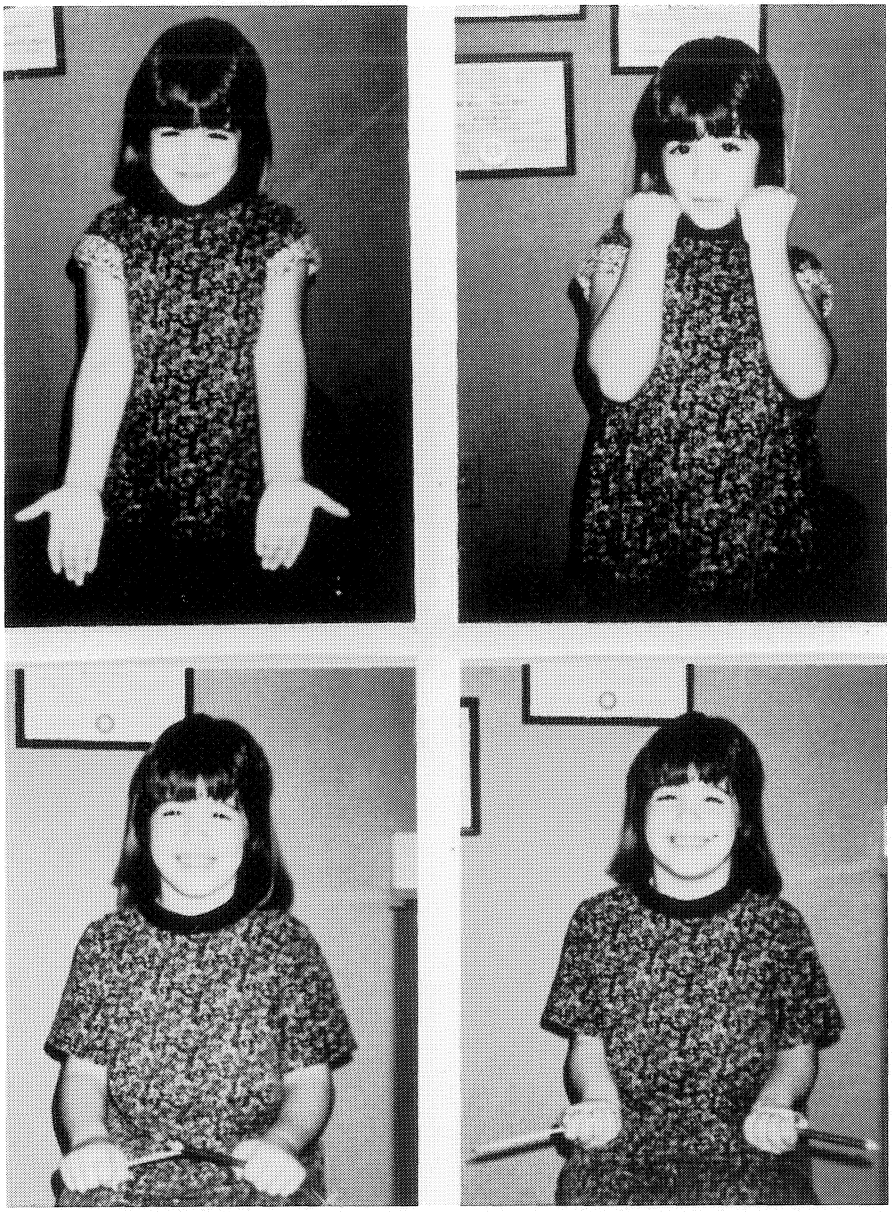

Figure 4
Clinical photos three weeks following initiation of protected motion exercises (nine weeks after injury). Patient enjoys full forearm pronation and supination, elbow extension to 0° (compared to 5° of recurvatum on the right), elbow flexion to 150°, and a symmetric carrying angle.

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ANTERIOR ELBOW DISLOCATION ASSOCIATED WITH OLECRANON FRACTURES – REVIEW OF THE LITERATURE AND CASE REPORT

Ricky D. Wilkerson, D.O.

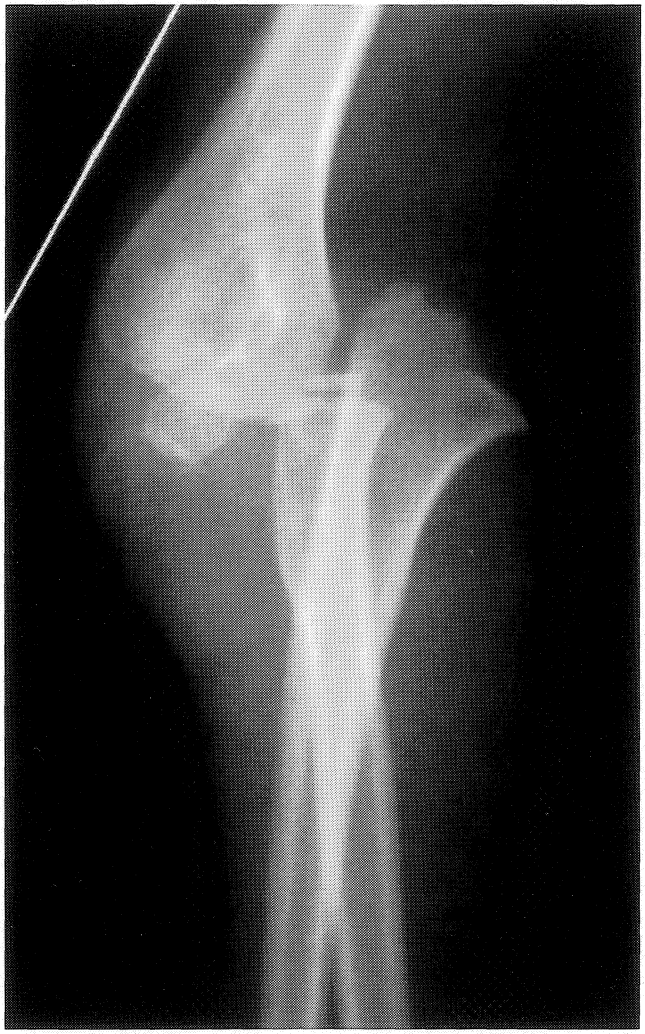

Fig. 1A Oblique radiograph of the right arm following injury.

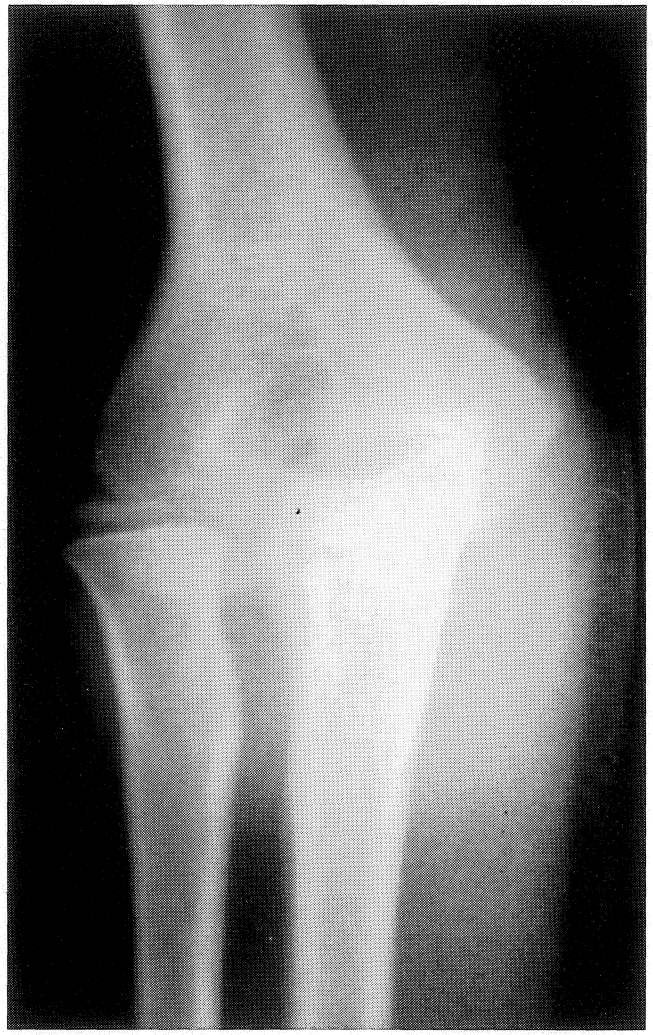

Fig. 1B Anterior-posterior radiograph of the right arm following injury.

Elbow dislocation, simple or complicated by an associated fracture, is not an unusual injury. Anterior elbow dislocation, however, has been described infrequently[1]. The original description was published by Evers in 1785[2] and translated and published by Cohn in 1922[3]. Before 1850, the combination of olecranon fracture and anterior dislocation was never reported. In fact, the possibility of an elbow dislocation anteriorly was apparently not recognized in a thorough publication by Sir Astley Cooper on the topic of dislocations in 1832[4].

Wheeler in 1967 reported on a series of thirty-seven fracture dislocations of the elbow[5]. In five of these patients the dislocation was associated with an olecranon fracture. In 1977, Neviaser reviewed a series of 115 elbow fractures in two separate New Orleans medical centers over a ten year period. Three were anterior dislocations, and only two of the these elbow dislocations were associated with olecranon fractures[6]. Anterior elbow dislocation associated with an olecranon fracture is therefore infrequently seen by most orthopaedists.

The mechanism of injury for this complex fracture-dislocation is one of hyperextension. The olecranon is levered within the olecranon fossa and fractured; continuation of this force displaces the more distal aspect of the

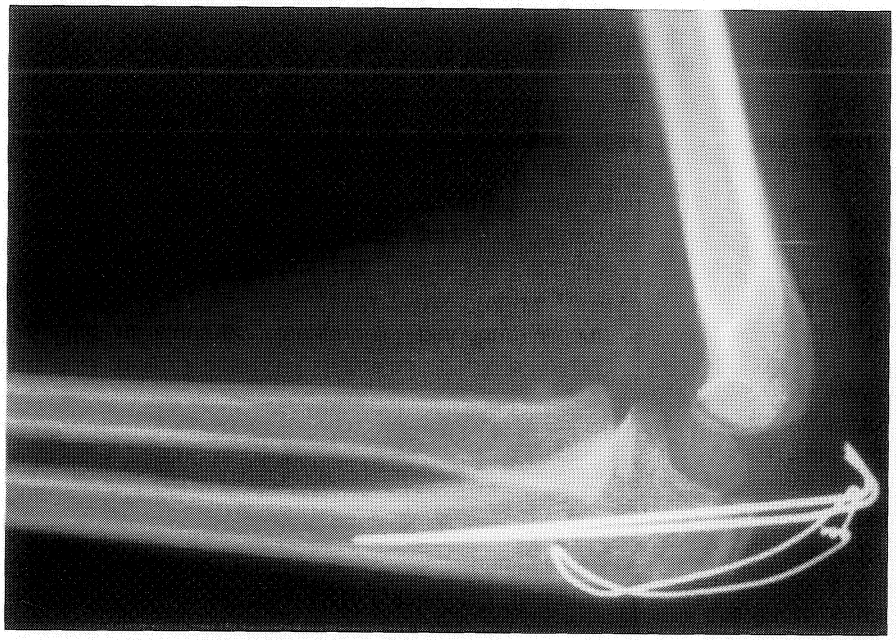

Fig. 2A Lateral radiograph after tenson-band wiring.

ulna anteriorly, leaving the olecranon fragment with the triceps mechanism posteriorly.

We report a case of anterior elbow dislocation with associated olecranon fracture. The anteriorly displaced ulna was button-holed through the anterior capsule of the elbow, thus preventing a closed reduction.

CASE REPORT

A seven year old boy was playing on a swing set when he fell from the swing at the peak of the arc and landed on his outstretched hand. He had immediate pain and obvious deformity. He was taken to the local physician who splinted the elbow and referred him for definitive care.

On evaluation in the emergency room the child was found to be in significant pain with an obvious deformity of the elbow and significant swelling. The skin and neurovascular status were intact. Radiographs revealed a displaced olecranon fracture with anterior dislocation of the forearm on the humerus. (Figures 1A and 1B).

The patient was taken to the operating room where several attempts at closed reduction under general anesthesia were unsuccessful. Open reduction was then performed.

A longitudinal posterior incision was made, and the triceps mechanism with the olecranon fragment was retracted proximally. This allowed excellent exposure of the elbow joint. The distal portion of the ulna had button-holed through the anterior aspect of the capsule of the elbow joint, thus locking the forearm in a displaced anterior position. A Freer elevator was then utilized to lever the capsule over the end of the distal ulna to allow reduction.

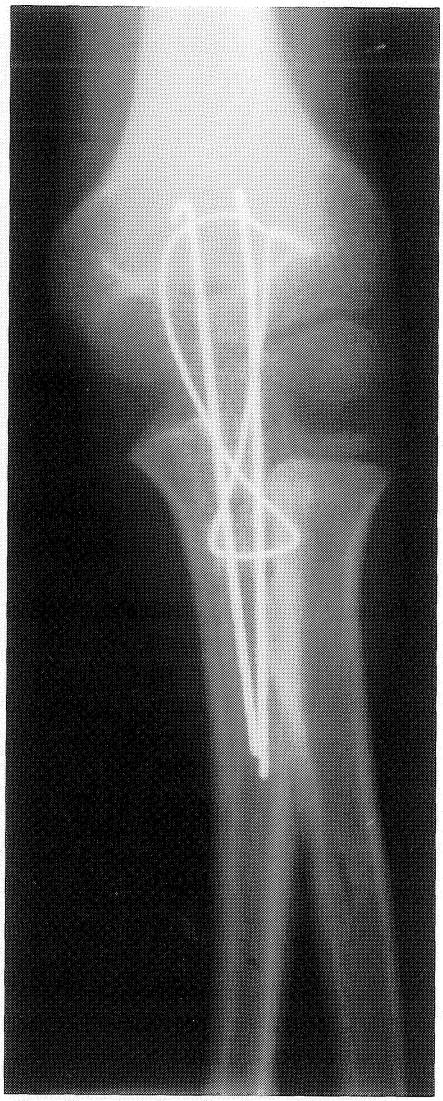

Fig. 2B Anterior-posterior radiograph after tenson-band wiring.

Freeing the ulna allowed anatomic reduction of the ulnar-trochlear and radial-capitellar articulations. The olecranon fracture was fixed with a tension band wire technique. Prior to closure the elbow had full, stable range of motion. Radiographs confirmed the reduction and fixation. (Figures 2A and 2B)

The patient began active range of motion on postoperative day three with progression as tolerated. After twelve weeks the tension band wire was removed in hopes of preventing growth arrest of the olecranon apophysis. The patient's range of motion is presently ten to 120 degrees with good stability.

DISCUSSION

This case illustrates the most commonly recognized and described mechanism of injury for anterior dislocation of the elbow, that of hyperextension. The olecranon process is fractured as the end of the ulna is levered into the olecranon fossa, leading to anterior dislocation of the more distal fragment. Review of the literature dealing with this injury has revealed no previous case of an irreducible anterior dislocation due to button-holing of the ulna through the anterior capsule. Mercer Rang has recommended the use of tension band wire technique for this fracture, a treatment which we found effective[7]. We removed fixation in the in hopes of preventing growth arrest of the olecranon with subsequent "capture" of the trochlea within the articular surface of the ulna. Wilkins, however, has stated that growth arrest of the physis of the olecranon has never been reported, so this concern may be ill-founded[8].

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INTRAARTICULAR DISLOCATION OF THE PATELLA

Kary R. Schulte, M.D.*
David S. Tearse, M.D.

Intraarticular dislocation of the patella is a rare traumatic condition. In a review of the literature, there have been a total of twenty-seven cases of intraarticular dislocation of the patella recorded[1-11]. In light of the striking radiographic appearance and initial presentation as a locked knee, we report the case of an additional patient and review the literature.

CASE REPORT

W.L. is a thirty-five year old male who injured his left knee on November 29, 1992. The patient was injured while urinating beside his car on a public highway. He slipped on a patch of ice, fell down the roadside embankment, and struck the superior pole of his patella on the side of a parked highway maintenance vehicle. He had immediate onset of pain, and his knee was locked in a position of 90 degrees of flexion. In the emergency room, any attempts to extend or further flex the knee were strongly resisted secondary to severe pain. AP and lateral radiographs of the left knee revealed an intraarticular dislocation of the patella. The patella was rotated on its horizontal axis and lodged between the femoral and tibial articular surfaces in the intercondylar notch of the knee. (Fig. 1) There was moderate knee swelling and the overlying skin was intact.

The patient was taken to the operating room and after induction of general anesthesia the knee was easily fully flexed and extended passively. However, attempts at manipulating the patella into its normal position were unsuccessful. An open reduction was performed through an anterior mid-line longitudinal incision. Inspection revealed the quadriceps tendon to have been torn from the superior pole of the patella; the patellar tendon as well as

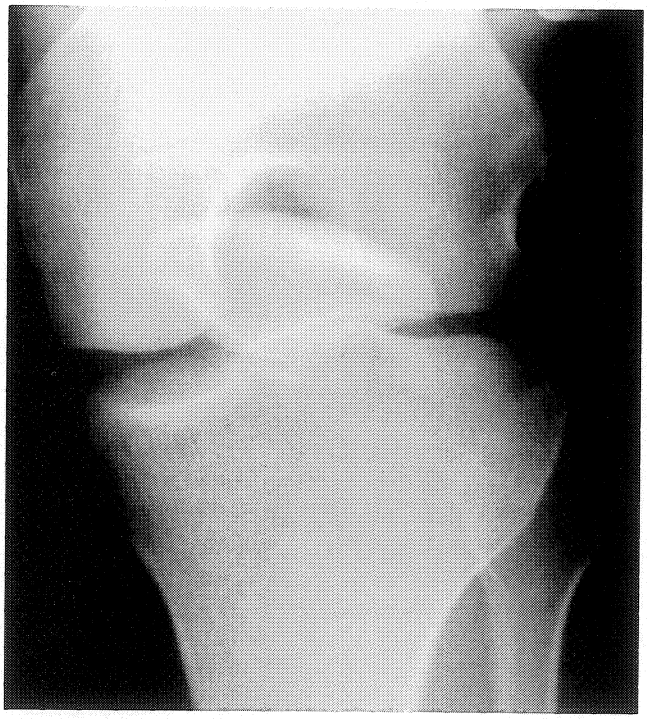

Figure 1A

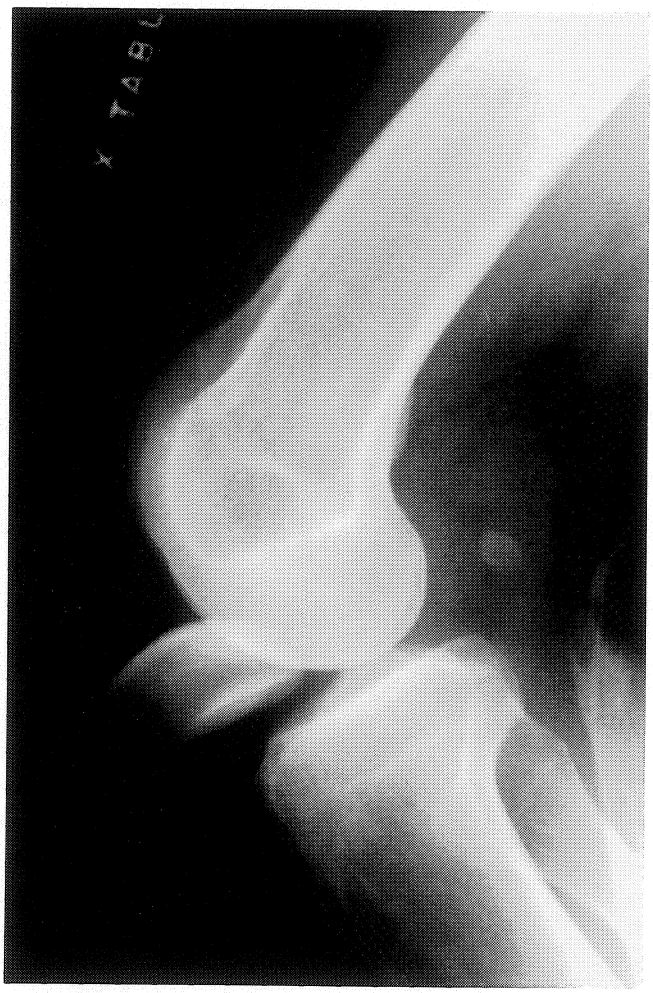

Figure 1B

Figure 1 AP (A) and lateral (B) radiographs of the left knee upon presentation.

*University of Iowa, Department of Orthopaedic Surgery, Iowa City, Iowa 52242

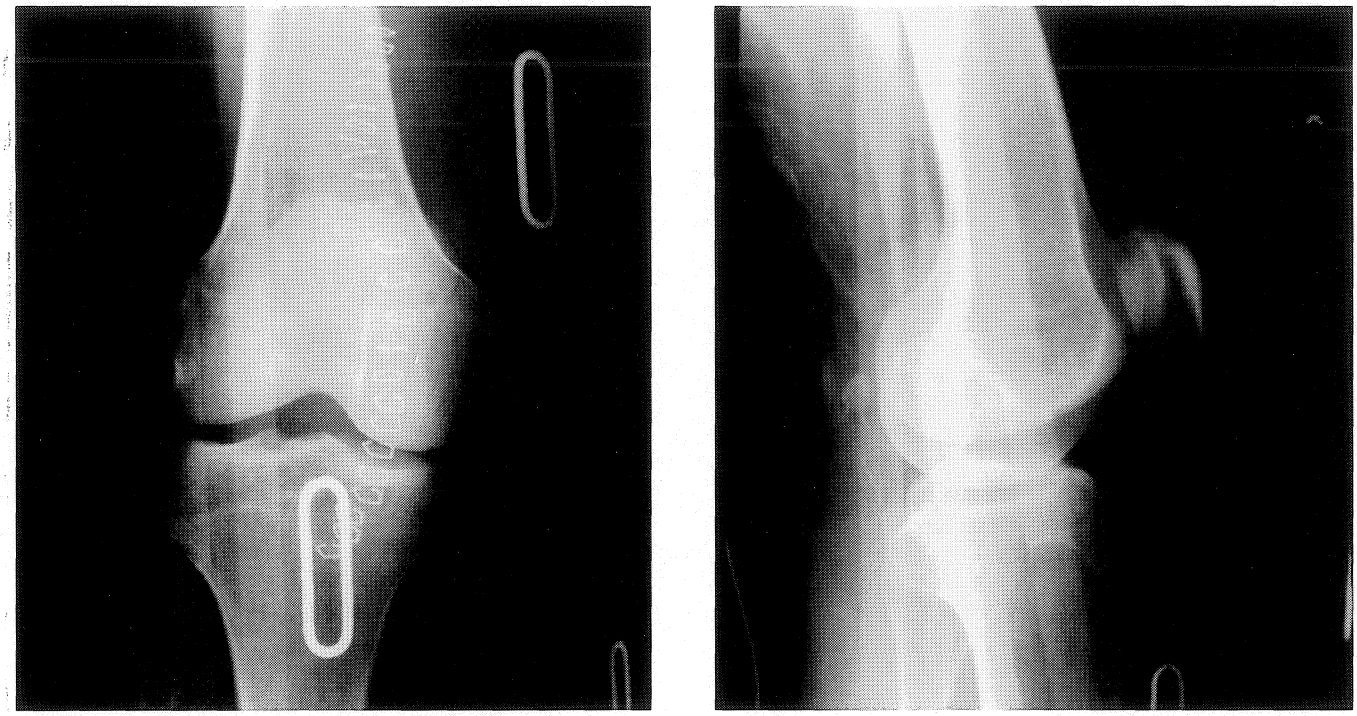

Figure 2 AP (A) and lateral (B) radiographs of the left knee postoperatively.

the medial and lateral retinaculae were intact. Attempts to manipulate the patella into its normal anatomic position through the disrupted quadriceps mechanism were unsuccessful. A release of the lateral retinaculum was performed and the patella was reduced to its anatomic position (Fig. 2). The quadriceps tendon was repaired to the patella using modified Bunnell stitches passed through the tendon and into drill holes made in the patella. Passive range of motion from zero to 125 degrees confirmed an excellent repair. The incision was closed in a routine manner and the left leg was placed in a knee immobilizer postoperatively. The repair has remained intact in early follow-up.

DISCUSSION

Intraarticular dislocation of the patella is a rare injury. Including the present case, a total of twenty-eight such injuries are reported in the literature. In this case a rupture of the quadriceps mechanism allowed rotation of the patella on its horizontal axis with subsequent intraarticular displacement. Although the majority of cases have included rupture of the quadriceps mechanism, several authors have reported cases with an intact quadriceps mechanism. In those patients, most of whom were twenty years old or younger, the superior pole of the patella was stripped from the extensor apparatus[2,6,7,8,9,10]. Surgery was required for reduction in all but four cases. Descriptions of successful closed treatment make it evident that a tremendous amount of force is required to obtain reduction. In light of the significant force needed and the probable damage to the extensor mechanism of the knee, open reduction and repair of the extensor mechanism is recommended.

The age range of patients reported in the literature is seven to sixty-four years old. In all but two cases, the superior pole of the patella was detached from the extensor mechanism and the patella rotated about a horizontal axis to a position between the femur and tibia. In two cases, the articular surface faced superiorly[9,10]. In the majority of the cases, the injury was caused by a direct blow to the superior pole of the patella.

Although this is a very rare injury, intraarticular dislocation of the patella should be included in the differential diagnosis for a locked knee. The radiographic findings are striking and diagnostic. In agreement with most previous reports of this injury, we believe open reduction and repair of the quadriceps mechanism is necessary to restore the normal anatomic position and function to the patella.

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