Temple University Journal of Orthopaedic Surgery & Sports Medicine

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A John Lachman Society Publication

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Letter from the President of Temple University

Temple's founder, Russell H. Conwell, once said, "Many of us spend our lives searching for success when it is usually so close that we can reach out and touch it." As we move forward this academic year, the search for success points to Temple. New faces, innovative capital projects, a talented and diverse student body, and our commitment to academic excellence are winning positive recognition for the University on all levels. So much is happening right here in our backyard. I take pleasure in providing you with a summary of some of our recent accomplishments, with particular regard to the Schools of Medicine and Health Professions.

This fall, we welcomed 4,000 freshmen and 2,650 transfer students to begin their education here at Temple. Our vibrant campus community thrives with about 10,000 students living on and around the Main Campus. Once again, we had an influx of the area's best and brightest students with an average high-school GPA of 3.26 and SAT score about 60 points above the national average. I am proud to report that the 2006 edition of *The Best 361 Colleges,* the annual guide from the *Princeton Review,* ranked Temple No. 2 on the list of most diverse student populations.

Governor Edward G. Rendell and other dignitaries joined us on November 1st of last year for the ceremonial ground breaking of the largest construction project in Temple University history — a 480,000-square-foot, $150 million, new facility for the School of Medicine. Slated to open in 2009, the new building will become the primary teaching and research site for the school, securing its position as a premier urban academic medical center. The Temple University Office of Technology Transfer has been making remarkable contributions in fields as biomedicine, engineering, and technology. For example, the distribution of a new drug to help leukemia patients is currently in development. Other projects include treatment for restless leg syndrome, the reduction of crude oil thickness, X-rays that can detect the transmission of dangerous materials, among many other findings that impact the world outside of our doors. Independence Blue Cross (IBC) has pledged $3.5 million to support the new building and heart research at Temple University's School of Medicine. The IBC Cardiovascular Research Center will occupy the top floor in the new $150 million state-of-the-art medical teaching and research facility. The College of Health Professions now ranks number eight in funding from the National Institutes of Health. Dave Devereaux, SBM '86 recently committed $1.5 million to establish an endowed chair in the Nursing Department in the College of Health Professions. He chose that particular college to benefit from his gift because he spent the majority of his career in the healthcare field. He chose the Nursing Department in honor of his mother, who just celebrated 50 years in nursing.

I would like to congratulate the John Lachman Society, the Department of Orthopaedic Surgery, and Joe Thoder, MD, Chairman for the publication of this journal which highlights the academic and research activities of those components of the University that relate to orthopaedic surgery and sports medicine.

Ann Weaver Hart

Letter from the Chairman

The Department of Orthopaedic Surgery and Sports Medicine is proud to present Volume II of our *Journal.* The Department of Orthopaedic Surgery and Sports Medicine continues to grow in its role as an integral part of Temple University School of Medicine and Temple University Health System. Our mission is to deliver the highest level of healthcare in the city of Philadelphia and the Delaware Valley. From primary care and outreach programs, to the local population of North Philadelphia, to regional tertiary care, Temple continues to be a leader. We continue to have the full support of the Temple University Health System and the School of Medicine in pursuing new programs. This year, with the cooperation of both the School of Medicine and the Health System, we have initiated a program with the Philadelphia Archdiocese to coordinate orthopaedic services to their high schools, including Certified Athletic Trainers for their athletic programs. Through this outreach program, Temple Sports Medicine is able to serve the community with screening programs, general musculoskeletal wellness education, and acute, timely care for the injured athlete.

On the educational side of North Broad Street, Temple University School of Medicine has broken ground for the new medical school. This new facility will not only provide Temple medical students with state of the art educational tools, but will also provide new laboratory and research space for the entire Health Science campus. This new facility, along with continued collaborative projects with the Department of Anatomy and Cell Biology, will further enhance our research opportunities.

The Department of Orthopaedics Surgery and Sports Medicine here at Temple University continues to take great pride in its tradition of excellence in clinical service, education and research. I would like to acknowledge the Temple/Shriners Orthopaedic Alumni Association and The John Lachman Research Foundation for their continued support of resident education. Through their support this year, residents have had the opportunity to present their research projects at the Pennsylvania Orthopaedic Society, the Eastern Orthopaedic Society, Arthroscopy Association of North America, and the American Academy of Orthopaedic Surgeons annual meetings.

Volume II of the *Journal* is a tangible representation of our continued efforts to maintain the high standard of excellence that has been set here at Temple. It would not have been possible without the efforts of several key members of the Department. In particular, I would like to recognize Drs. Kristofer Matullo and Simon Chao for their tireless efforts to be sure manuscripts were compiled and deadlines were met and Drs. Joseph Torg and Saqib Rehman for their oversight of the project. We are pleased to present volume II of our *Journal.* Once again, I am confident that you will find the contents educational, informative and reflective of our mission to our academic and clinical communities.

Joseph Thoder, MD

Letter from the Editors

We are proud to present the second edition of the *Temple University Journal of Orthopaedic Surgery and Sports Medicine.* This journal was inspired by our chairman, Dr. Joseph J. Thoder, and it strives to uphold the "Tradition of Excellence" begun by Drs. John Royal Moore, John Lachman, and Michael Clancy. The goal for our index edition was to construct a high quality journal that would encompass a broad spectrum of musculoskeletal pathology, engineering, and basic science. We feel that we have succeeded in this endeavor. The content of our journal was representative of the spirit, insight, and diligence of our department, with contributions from the Department of Anatomy and Cell Biology, and the Department of Kinesiology of Temple University. The feedback from our index edition was overwhelmingly positive and we have added a few changes to this edition to present more of our work. We are proud to feature more extended abstracts this year as well as standard abstracts whose manuscripts are currently under review for publication in other peer-reviewed journals. Along with these scientific endeavors, we continue to offer insight into our world by providing a snapshot of the lives of our residents, alumni, and staff.

A work of this size is again not possible without the help of many individuals. We would like to thank Synthes for their continued support of our journal and educational advancement, our faculty for their guidance and support, the editorial board for their insight, and the authors for allowing us to share their work. We would also like to recognize many other companies whose donations to the journal allow us to distribute our work to orthopedic colleagues around the country. Two people deserve particular attention: first, our Chairman, Dr. Joseph Thoder, whose initiative has propelled this project forward and kept the spirit of success alive, and second, Dr. Joseph Torg, whose personal commitment and direction led to the journal's continued excellence. We are pleased that the Journal has grown in both quality and breadth this year, and hope it continues to do so in future years to come.

In closing, we are proud to present the second edition of the *Temple University Journal of Orthopaedic Surgery and Sports Medicine.* Thank you for allowing us to share some of our academic endeavors with you.

Sincerely,

Kristofer S. Matullo, MD

Simon Chao, MD

LATCH IN HIS TEACHING MODE: Charlie Parsons' excellent drawing depicting "Latch in his teaching mode" has been adopted as the Society's logo. We are happy to inform that those who have accepted membership in the Society with a monetary commitment have received prints of Charlie's drawing autographed by Latch and suitable for framing.

Message from the John Lachman Society

The John Lachman Society was founded in 2004 to honor Dr. Lachman and propagate his principles of integrity, teaching, and excellent patient care. The Society will also provide discretionary funds for the Chairman to promote and support the academic mission of the Department including student and resident research. The mechanism to accomplish these goals will be through the Society's support of the John Lachman Orthopedic Research Fund (JLORF), incorporated in Pennsylvania as a non-profit corporation. The Internal Revenue Service has determined that The John Lachman Orthopedic Research Fund is exempt from federal income tax under 501 (C) (3) of the Internal Revenue Code and that contributions to the fund are tax deductible.

The mission of The John Lachman Society is twofold: 1) to promote the Lachman principles of integrity, resident training, and quality patient care by various proactive means and programs; and 2) to provide discretionary funds for the chairman to foster and support both the academic and research mission of the Department. Of the total contributions received in any calendar year, 75% will be invested in an endowment to be determined by Finance Committee with the approval of the Board of Trustees and 25%, plus interest on the endowment, will be used to support the aforementioned mission. Proposals for appropriation of funds may be initiated by Executive Committee, working committees of the Society, or the Chairman and will require the approval of both the Chairman and 51% of the Board of Trustees or two-thirds of the Board of Trustees.

Membership in The John Lachman Society will include the following groups and initiation contribution levels:

1) Physician group — $5,000.00 over five years;
2) Scientists and allied health professionals — $1,250.00 over five years;
3) Friends of John Lachman — $5,000.00 over five years.

Once an individual has met his or her initiation contribution, he or she will be a member in perpetuity. It is anticipated that contributions to the fund will also be forthcoming from satisfied patients and members of industry. Checks should be made payable to The John Lachman Orthopedic Research Fund and forwarded to P.O. Box 7283, St. Davids, PA 19087.

Those interested in membership in The John Lachman Society should contact the Chairman of the Membership Committee, Philip Alburger, MD, c/o The John Lachman Society, P.O. Box 7283, St. Davids, PA 19087.

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Gerald Williams, MD
Steven Wolf, MD
John Wolf, MD
Thomas Yucha, MD

RESIDENT RESEARCH SUPPORT: The following resident research projects have been or are currently being supported by The John Lachman Orthopedic Research Fund:

1) Anbari, A: "The Relationship Between Posterior Shoulder Capsular Tightness and SLAP Lesions".
2) Hsu, V.: "Elastic Instability, Columnar Buckling, and OrthopedicInjury".
3) Yucha, D.T., Junkin, D.M., Ilyas, A., D'Addesi, L.L., Purchase, R.J.: "Evaluation of the Relationship of the Dorsal Sensory Branch of the Ulnar Nerve to the 6U and 6R Arthroscopic Portals-An Anatomic Study".
4) Junkin, D.M.: "The Arthroscopic Anatomy and the Closure of the Rotator Interval".

5) Junkin, D.M., D'Adessi, L.L.: "Distal Radio-ulnar Joint Subluxation Resulting from Proximal Migration of the Radius — Defining the Pathologic Lesion and Treatment".
6) Purchase, R.J., Hsu, V.: "12–15 Year Follow-up of High Density Polyethylene Prosthetic Anterior Cruciate Ligament Reconstruction."
7) Reish, M.: "Intermediate Term Results of Arthroscopic Cuff Repair: Correlation of Outcome and Degree of Humeral Head Coverage".
8) Reish, M.: "Outcome of Arthroscopic Repair of Massive Rotator Cuff Tears".
9) Hsu, V.: "Functional Outcomes Following Radial Head Replacement".
10) Ilyas, A.M.: "Intramedullary Fixation of Distal Radius Fracture: A New Technique for an Old Problem".
11) Matullo, K.S., Sewards, J.M.: " Proximal Carpectomy: A Novel Surgical Technique".
12) Chao, S., Yucha, D., Thomas, S.: "The Effects of Scapular Fatigue on Shoulder Motion".
13) Chao, S., Thomas, S., Yucha, D: "The 'Bear Hug' Test in Detecting Upper Subscapularis Insufficiency — An EMG Study".
14) Matullo, K., Codjoe, P.: "Refurbished Drill Bits: Effectiveness in the Operating Room".
15) Matullo, K., Ilyas, A., Thoder, T.: "First Carpometacarpal Arthroplasty: A Review of Treatment Options and Introduction of a Limited Incision Technique".
16) Matullo, K., Samdani, A., Betz, R.: "Low Back Pain and Unrecognized Cobb Syndrome in a Child Resulting in Paraplegia".
17) Matullo, K., Sewards, M., Coll, A., Thoder, J.: "Proximal Row Carpectomy: Clinical Evaluation of a Novel Surgical Technique".

WEBPAGE: The John Lachman Society web page is a reality and can be entered at www.johnlachmansociety.org.

JOHN LACHMAN LECTURE: The inaugural John Lachman Lecture was held in conjunction with the Pennsylvania Orthopaedic Society annual meeting on Friday November 11, 2004 at the Bellevue Hotel in Philadelphia. David Apple, MD, medical director of the Shepherd Spine Center in Atlanta, Georgia presented "Practical Ethics in Orthopaedic Practice". Dr. Apple is a member of the Academy's Committee on Ethics and has published on the subject matter.

John Bergfeld, MD, Medical Director of the Cleveland Clinic Sports Medicine Program presented the second annual John Lachman Lecture entitled "Should the Team Doctor Pay to Play?" at the annual meeting of the Pennsylvania Orthopaedic Society in Pittsburgh on November 18, 2005. The lecture format was pro-con and John was suitably provoked by Joe Torg. We believe that this is a timely topic with profound ethical implications. The Pennsylvania Orthopaedic Society has made this lectureship a permanent part of their annual meeting itinerary.

The Third Annual John Lachman Lecture was presented by Michael A. Smerconish, well known radio talk show host, Philadelphia Inquirer columnist, and author of "Flying Blind" and "Muzzled." In addition, he is a frequent guest on several of the nationally televised news commentary shows. In view of the fact that most orthopaedic surgeons are primarily pre-occupied with such matters as the anterior cruciate ligament, total joint arthroplasty, and tort reform, it seemed appropriate to indulge in matters of public policy and in the arena, Smerconish excels. The title of his talk was "Fifteen Points of Current Interest." The venue for the talk was the Friday luncheon at the fall meeting of the Pennsylvania Orthopaedic Society, before a capacity audience in the ballroom at the top of the Bellevue. Smercornish opinioned on fifteen topics: Smerconish's talk was well delivered, well received, and reflected most favorably on the John Lachman Society!

RESIDENTS DISSERTATIONS: The second annual John Lachman Society Research Day was held on April 8, 2006. Eight resident research papers were presented (see attached). Guest judges included Fayaz Safadi, PhD, Scott Kozin, MD, and Roy Reinus, MD. Dave Yucha and Simon Chao shared first prize award of $500. Kris Matullo and Matt Reish were awarded the second best paper for their subscapular nerve block technique study and Matt Reish received honorable mention for his report on Intermediate term results for arthroscopic rotator cuff repairs. Dave and Simon also placed first in the Philadelphia Orthopaedic Society for Sports Medicine (POSSM) residents' research program.

PRESENTATIONS:

1) **Suprascapular Nerve Block Technique: An Anatomic Study**
   Matullo K, Reish M, Kelly J
2) **13–15 Year Results of a High Density Polyethylene Prosthetic Anterior Cruciate Ligament Reconstruction**
   Purchase R, Mason R, Hsu V, Torg J
3) **Intramedullary Fixation for Distal Radius Fractures: A New Technique for an Old Problem**
   Ilyas A, Coll A, Thoder J
4) **Intermediate Term Results of Arthroscopic Rotator Cuff Repairs: Correlation of Outcome and Degree of Humeral Head Coverage**
   Reish M, Richmond J, Kelly J
5) **Performance Analysis and Morphology of Unused versus Reproduced Drill Bits**
   Rehman S, Darvish K, Shafieian M, Matullo K, DeLong W
6) **An Electromyographic Assessment of the "Bear Hug": A New Technique for Evaluation of the Subscapularis**
   Chao S, Thomas S, Yucha D, Kelly J
7) **Functional Outcomes Following Radial Head Arthroplasty**
   Hsu V, Thoder J
8) **Intramedullary Nailing of the Fractures and Malunions of the Distal Radius**
   Reish M, Thoder J

TEMPLE UNIVERSITY JOURNAL OF ORTHOPAEDIC SURGERY AND SPORTS MEDICINE: A major accomplishment of the society was sponsorship of the first annual Temple University Journal of Orthopaedic Surgery and Sports Medicine. Thirty-five hundred copies of the journal were distributed to members of The John Lachman Society, Temple University medical faculty and key University Administrators, members of the Pennsylvania Orthopaedic Society, Temple University School of Medicine alumni who trained elsewhere in orthopaedic surgery, Chairman and Directors of orthopaedic programs with residency training programs, selected members of the general orthopaedic community, selected members of the National Athletic Trainers Association, and selected referring physicians. The Journal was well received by and we believe clearly established the creditability of our academic program.

RESIDENTS LIBRARY SUPPORT: In keeping with the request of the Director of the residency program, The John Lachman Orthopaedic Research Fund is committed to a $2,500 a year expenditure for texts and other educational materials.

SYNTHES AWARD: Synthes has awarded The John Lachman Orthopedic Research Fund $20,000 to support the research and academic activities of the Department of Orthopaedic Surgery.

FINANCIAL SUMMARY: Since its inception in 2005, the JLS has exceeded expectations in generating contributions to the JLORF sufficient to cover the Fund's operating expenses and more than double its endowment, now over $150,000. Outstanding pledges in excess of an additional $200,000 bring the endowment to thirty-five percent of our goal of $1,000,000, which, conservatively invested, should yield enough money to carry our current level of annual expenses in perpetuity. This provides resources to the Department of Orthopedic Surgery at Temple University School of Medicine for research funding, resident education-related travel expenses, and publication of this Journal for the foreseeable future.

Joe Torg, MD
Secretary

John Chaney in charge

Dedication

John Chaney: Coach, Mentor, Educator, Social Activist, and Athlete

John Chaney was all of these. A gifted basketball player at Ben Franklin High School in Philadelphia, he was named the best player in the city in 1951.

Despite his success and obvious talent, his opportunities were limited because of race and the times. With the strong encouragement of a caring high school coach, he was afforded one opportunity to attend Bethune-Cookman College in Florida.

Taking advantage of the one road given, he built on that to be a collegiate All-American and an NAIA Tournament MVP. Following college, he played one year with the Harlem Globetrotters. He then became a player-coach in the Eastern Professional league where he was a seven time All Star and twice MVP of the league. His playing career ended when he was severely injured in an auto accident while traveling between games.

John returned to Philadelphia to teach and coach, starting at Simon Gratz High School in 1966. Success at Gratz became an opportunity to become the coach at Cheyny State in 1972. While there, he won a Division II national championship and also earned a teacher of the year award; he acknowledged this award made him realize "that education can save your life." Appreciating the unique combination of intelligence, foresight, and leadership qualities in the man, Dr. Peter Liacouras, the Temple University President persuaded John to come coach the Temple basketball team in 1982. The chronicled success of his tenure at Temple thereafter has assured that the name and face of John Chaney will be forever united with Temple University.

Honed by his own experiences, John has become the champion of persons from compromised background and limited opportunities. All he asks is that the individual is given a chance, not a free ride, not perpetual mom's apple pie, but an open door to those who are willing to try. Trying in Chaney philosophy is working early and working hard — a similar dictum for an orthopedic practice, certainly one for an orthopedic resident. However, the logic behind his early practices was meant to afford structure enabling his student athletes to best fulfill his demands as well as their academic responsibilities. John's famous mantra, "winning is an attitude" was meant primarily to reflect winning at life, not that he was unaware that winning on the court afforded even greater opportunities. With this in mind, his main objective for his student athletes was to become productive, integral members of society.

If you live what you believe, you can teach it. John's beliefs, and his passion for them, are in his face and in all his words. Caring and kindness, honesty and fairness, persistence and focus on a goal are the core of his existence. His life is a testimony of his adherence to these principles.

In our country's Romanesque personality, success in athletics affords the greatest and most immediate visibility, as John Chaney's success has afforded our University. Temple University may be ever proud that Coach has projected a perfect reflection of founder Russell Conwell's, original objectives, to allow dreams of opportunity to become acres of diamonds. John is that diamond, and we are all proud to be able to claim him as one of us.

Ray A. Moyer, MD

Commentary

An Editorial on Gunshot Violence

This summer, I witnessed (again) a young man's needless departure from this life. Another victim of the senseless gunshot violence which has besieged our city. As an Orthopedic surgeon at Temple University 'on call', I ambled into the emergency room before I left for home that evening, to be sure that no orthopedic care would be needed for the "trauma category one" I heard announced throughout the hospital. When I entered the 'trauma bay' of our emergency department, I became mesmerized by the competent trauma surgeons trying heroically, albeit unsuccessfully, to revive an unresponsive young man, fatally wounded in the chest from a presumed close range, large caliber missile. In the cacophony of a life and death rescue attempt, I couldn't help but overhear a nurse exclaim 'there is another gunshot wound to the abdomen on the way'. The poor lifeless body I beheld was essentially 'dead on arrival'. I was overwhelmed by the childlike countenance of this poor victim who was reportedly 21 years of age, but, in my estimation, appeared still an adolescent.

I remember my 21st year with the fondest of memories — family, friends, romance, sports, college and the prospects of going to medical school. I grieve this young man's truncated existence — the loss of yet another precious life, a life which will never experience the full joys of early manhood, of vocational calling, of marriage and parenthood — all the things I revere about my Blessed life.

With every gunshot related death I read about or discover on TV, there always seems to be a continual lament: 'this violence and senseless killing must stop'. With the recent and honorable death of officer Gary Skerski (mismatched against a criminal carrying a 'sawed-off shotgun'), and the commemoration of officer Daniel Faulkner's slaying, the public outcry against gun violence seems to have reached its Zenith.

Alas . . . nothing has changed.

One blatant truth remains — there are too many guns. A wounded culture simply does not need more weapons to "act out" its' conflicts. Until this truth is embraced and conquered, the carnage will continue.

The state of Pennsylvania eased restrictions on gun permits in 1985. Since then, the number of citizens authorized to carry a handgun rose from 700 to 32,000. Guns are simply too accessible and are the 'default' option for conflict resolution. Our beloved city saw 380 homicides in 2005, the deadliest year since 1997. 208 deaths were over 'disputes' and drug related killings accounted for only 13 percent. This year, we are on track to surpass this figure easily. Our country experiences 40,000 to 50,000 firearms related deaths each year — a figure that equals the number of Americans lost in Vietnam. The estimated cost to society, including loss of productivity, pain and suffering and reduced quality of life has been estimated at 63.4 billion dollars per year.

Contrast these figures to countries with strict handgun prohibitions, where the numbers of gunshot related deaths are but a 'handful'.

It is time we embrace the obvious. Unless we make it more difficult (if not impossible) to carry a concealable firearm, the loss of precious life will inexorably continue. Guns will remain the option of choice for many when anger arises. Our beloved Police force (whom I have the profound privilege of caring for the last sixteen years) risk their lives everyday for society, and will continue be outmanned by their foes. One more senseless killing is one too many.

Let's get back to the fundamentals: life is more important than distorted 'second amendment rights' or special interest groups. Society's cultural ills and the degradation of mores will not be cured overnight; in the meantime, more guns lead to more killings.

Excuse me for now; I must rest and prepare for the next 'call'. I pray my spirit can withstand what befalls my eyes my next sojourn to the ER.

John D. Kelly, IV, MD

Clinical Research

Biceps to Triceps Transfer for Elbow Extension in Persons with Tetraplegia

LL D'Addesi,[1] SH Kozin,[2,1] MJ Mulcahey[3]

[1]*Department of Orthopaedic Surgery, Temple University,*
[2]*Department of Orthopaedic Surgery,* [3]*Rehabilitation Services and Clinical Research, Shriners Hospitals for Children, Philadelphia, PA*

Abstract

Purpose: People with C5-C6 level tetraplegia lack triceps function which leads to significant disability. Regaining elbow extension allows those with tetraplegia to be independent, performing activities such as pressure relieving maneuvers, propelling a wheelchair, performing independent transfers, eating, self-grooming, and self-catheterizing. Although a posterior deltoid to triceps transfer has been the standard transfer to reconstruct elbow extension, the senior author has shown excellent results in a previous study using the biceps to triceps transfer. A retrospective study was done to assess functional outcome of biceps to triceps transfers as a follow-up to this previous study.

Methods: Biceps to triceps transfers were done in 42 arms of 27 patients. Inclusion criteria consisted of: less than antigravity triceps muscle strength, active brachialis and supinator muscle, and less than a 20 degree flexion contracture at the elbow. Outcome measures were elbow extension strength using manual muscle testing (MMT), and performance and satisfaction with self-selected goals.

Results: No patient expressed subjective complaints of decreased elbow flexion or forearm supination strength. Stringent MMT for elbow extension revealed an average muscle strength of 3.1 (range; 0 to 4). Performance and satisfaction were greatly improved in pushing a door open, manipulating elevator buttons, adjusting blankets while in bed, using a computer, styling hair, brushing teeth, feeding self, playing games, and reaching up towards a high shelf. After tendon transfers, the total mean score increased from 3.3 to 5.7 and from 2.6 to 5.5 for performance and satisfaction, respectively. Complications were few and related to post-operative infection, olecranon bursitis, compartment syndrome, and attenuation of the transfer during rehabilitation.

Conclusion: Biceps to triceps transfers in tetraplegic patients has been found to be a useful transfer to reconstruct elbow extension. Our patients were extremely satisfied and complications were few. We recommend its use as the preferred technique to reconstruct elbow extension in persons with tetraplegia.

Introduction

Over 100,000 Americans live with tetraplegia.[1] Tetraplegia can be highly disabling, requiring a great deal of assistance. Spinal cord injuries resulting in tetraplegia commonly occur at the C5-C6 level. This level usually leaves the patient with a lack of elbow extension as well as wrist and hand function, and the inability to perform simple, essential tasks, such as pressure relief maneuvers, feeding, self-catheterization, and perineal care. Those who are incapacitated by tetraplegia wish to live independently and increase their mobility.[2] Patients with tetrapelgia have regarded surgical reconstruction of the upper extremity as highly important, and have appreciated elbow extension more than any other restored function.[3–8] Although some independence can be gained by restoration of hand function, these improvements are impaired by the need to rely on gravity for elbow extension.[9] Voluntary control of triceps is a significant determinant in the ability to perform self care tasks.[10] The benefits of retaining elbow extension are to increase strength and control of motion, perform overhead activities, push up to a sitting position, perform pressure relief maneuvers, propel a wheelchair, transfer independently, feed oneself, self-catheterization, and drive[4, 11–18] (Figure 1).

Two options for reconstructing elbow extension include the posterior deltoid to triceps transfer, and biceps to triceps transfer.[19] In the past, the senior author (SHK) has performed mainly deltoid to triceps transfer, and limited the biceps to triceps transfer to patients with elbow flexion and forearm supination contractures and elbow flexion spasticity, or in cases of failed deltoid transfers. Anecdotal evidence from these procedures has supported varied results with deltoid to triceps transfers and more consistent results after biceps to triceps transfer. Recently, a prospective, randomized study was performed to compare the posterior deltoid to triceps and biceps to triceps transfers. The biceps to triceps transfer more reliably produced antigravity elbow extension strength, while both groups had an equal loss of elbow flexion torque.[20]

The senior author (SHK) is now exclusively using the biceps to triceps transfer for the reconstruction of elbow extension. The purpose of this paper is to assess the results of biceps to triceps transfers with regards to the ability to achieve antigravity strength and patient satisfaction.

Figure 1A. Sixteen-year-old male status post bilateral biceps to triceps transfer with full extension against gravity.

Figure 1B. A 19-year-old female status post biceps to triceps transfer with improved ability to reach shelves.

Material and Method

A retrospective review was completed of all biceps to triceps transfers performed over the last six years at the Shriners Hospital for Children, Philadelphia, PA. Biceps to triceps transfers were done in 42 arms of 27 patients. Eight of the patients were female and 19 were male. Fifteen patients had bilateral biceps to triceps procedures (11 were done simultaneously). All patients had mid to upper level cervical spinal cord injury resulting in tetraplegia, and specifically loss of elbow extension. The average age at the time of surgery was 16 years.

Active brachialis and supinator muscles are prerequisites to biceps transfer to maintain elbow flexion and forearm supination.[16, 18, 21] The evaluation of their integrity requires a careful physical examination of elbow flexion and forearm supination strength. The brachialis and supinator muscles can be palpated independent of the biceps muscle. Effortless forearm supination without resistance induces supinator function that can be palpated along the proximal radius. Similarly, powerless elbow flexion incites palpable brachialis contraction along the anterior humerus. Equivocal cases require additional evaluation to ensure adequate supinator and brachialis muscle activity. We prefer injection of the biceps muscle with a local anesthetic to induce temporary paralysis and allow an independent assessment of brachialis and supinator function. A supple elbow with near complete range of motion is also required. All patients with an elbow contracture underwent therapy to resolve the contracture. Surgery was delayed until the contracture was less than 20 degrees. Numerous modalities were used to correct the contracture, with serial casting being the most efficacious.

The surgical procedure and postoperative rehabilitation protocol has been described previously.[20, 22] A three-centimeter anterior transverse incision across the antecubital fossa is performed. A second seven-centimeter longitudinal incision is made along the medial intermuscular septum (Figure 2). The biceps tendon is routed along the medial side of the arm superficial to the nonfunctional ulnar nerve (Figure 2). A third seven-centimeter posterior incision is made over the distal third of the triceps and curved around the olecranon. The triceps is sharply split over the tip of the olecranon and a self-retraining retractor placed between the incised tendon (Figure 3). The biceps tendon is subsequently passed obliquely through the medial portion of the triceps tendon using a tendon braider. The end of the tendon is then placed into a unicortical hole made within the olecranon, large enough to accept the tendon. Sutures within the tendon are passed through two small drill holes through the opposite posterior cortex. The sutures are used to pull the tendon into the unicortical hole (Figure 3). The elbow is placed into extension as the tendon is advanced into the unicortical tunnel and the sutures are tied over the bone. This maneuver automatically sets the tension within the tendon transfer. Additional sutures are added between the biceps and triceps tendon.

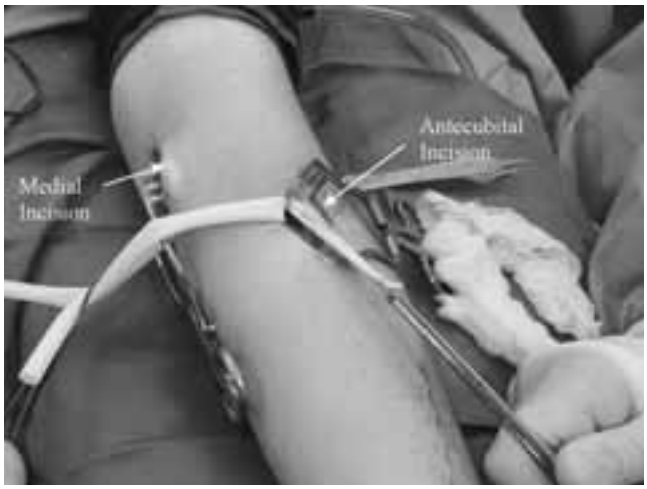

Figure 2A. Antecubital incision. The biceps tendon has been released from the radial tuberosity via the antecubital incision. A medial skin incision has been made on the medial aspect of the arm. The biceps tendon will be tunneled subcutaneously from the antecubital fossa through the medial skin incision.

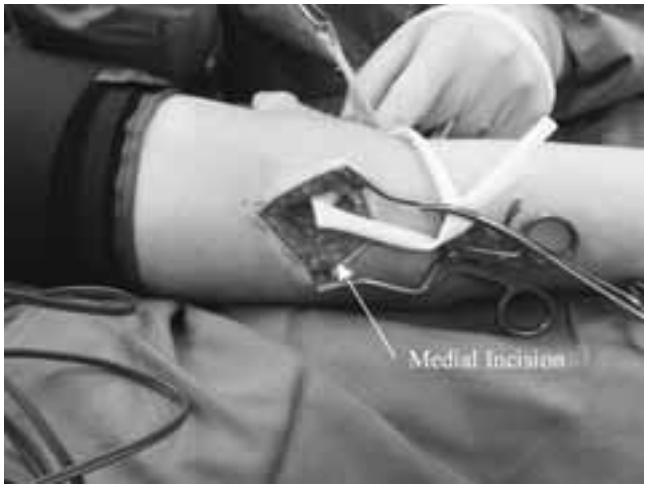

Figure 2B. Medial skin incision. The biceps tendon, as shown here, is being presented through the anterior antecubital incision. The tendon will be retrieved through the medial incision, and then passed to the posterior incision subcutaneously to be attached to the triceps.

After surgery the elbow is casted in full extension for four weeks. An elbow extension splint in full extension is then fabricated for nighttime use. A dial-hinge brace (e.g., Bledsoe brace Systems, Prairie, Texas) is fitted for daytime use and acts as a flexion block at 15 degrees. The brace is adjusted each week to allow an additional 15 degrees of flexion. The brace is not advanced if an extension lag develops. Tendon transfer firing is started in an antigravity plane. The medially routed biceps can be palpated along the medial humerus during active elbow extension. Verbal prompting of active elbow flexion and active forearm supination facilitate motor learning. Additional modalities, such as biofeedback, are used in patients that have difficulty with firing. Functional activities of daily living are incorporated into the therapy as elbow

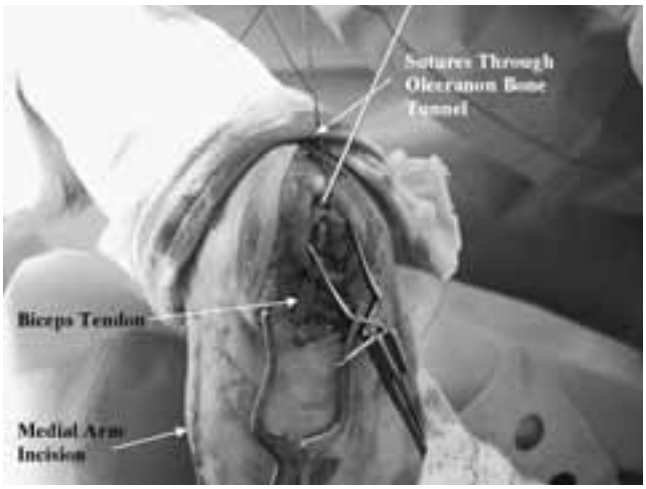

Figure 3. Posterior skin incision. The biceps tendon is tunneled subcutaneously from the medial skin incision to the posterior elbow incision to be woven through the triceps tendon. The tendon is then passed into the olecranon using the suture passer.

flexion increases each week. A dial-hinge brace is continued until 90 degrees of elbow flexion is achieved without an extension lag. A nighttime extension splint is maintained until 12 weeks after surgery. Strengthening is started three months after surgery.

Other procedures that were performed simultaneously in appropriate patients included brachioradialis (BR) to flexor pollicus longus (FPL), BR to extensor carpi radialis brevis (ECRB), split FPL transfer, thumb metacarpophalangeal (MCP) joint fusion, humeral osteotomy, radius and ulna osteotomy, extensor carpi ulnaris (ECU) to ECRB, ECRB to flexor digitorum profundus (FDP), extensor carpi radialis longus (ECRL) to FDP, intrinsic releases, first dorsal interossei to thumb adductors, and implantation of functional electrical stimulation electrodes.

Our outcome measures were elbow extension strength and performance and satisfaction with self-selected goals. Manual muscle testing was performed by the main author (S.H.K.) as well as certified occupational therapists. Subjects were tested for isotonic elbow extension and elbow flexion strength before and after surgery. Manual muscle testing (MMT) was performed according to accepted standards. The ability of a subject to impart resistance against gravity or with gravity minimized was assessed through the available arc of passive movement. Unwanted substitution patterns, such as shoulder external rotation with attempted elbow extension, were prevented. In patients that demonstrated full active movement with gravity minimized, and incomplete motion against gravity, a MMT grade of 3-/5 was assigned. When the patient was able to move through the entire arc of available motion against gravity, then resistance was imparted against gravity and the muscle strength re-graded.

A grade 3 implies full motion against gravity with the inability to extend the elbow against resistance. A grade 4

indicates the ability of the patient to exert moderate resistance throughout the available arc of passive movement. This strict grading criteria eliminates confusion regarding the results of MMT and prohibits a patient from being scored a grade 4 strength unless they have achieved full available motion against gravity (grade 3 strength). Therefore, many patients with a 3- MMT had some capacity to apply resistance within some points of their available motion.

Results

Manual Muscle Testing (MMT)

All patients regained full elbow flexion and forearm supination against gravity and ability to impart resistance to manual muscle testing (grade 4 or 5). No patient expressed subjective complaints of decreased elbow flexion or forearm supination strength. Stringent MMT for elbow extension revealed an average muscle strength of 3.1 (range; 0 to 4) (Figure 5). Thirty two arms (76%) were able to extend completely against gravity (MMT 3 or greater). Ten arms (24%) were unable to extend completely against gravity (MMT < 3) of which 5 arms were directly related to complications listed below. Most patients with less than grade 3 strength were able to impart resistance through some arc against gravity.

Performance and Satisfaction with Self-Selected Goals

Among 9 subjects, 75 goals for surgery were established. Following the biceps to triceps transfer, improvement in performance and satisfaction was identified in 78% and 89% of the subjects, respectively. Performance and satisfaction were greatly improved (as defined by improvement of at least four points) in pushing a door open, manipulating elevator buttons, adjusting blankets while in bed, using a computer, styling hair, brushing teeth, feeding self, playing games, and reaching up towards a high shelf. Although improvement was seen in 2 subjects in lower body dressing, a decline in performance was seen in 3 subjects. Wheelchair transfers also showed a mixed result with 2 subjects citing improvement, 3 a decline in performance, and 2 no change in performance or satisfaction. After tendon transfers, the total mean score increased from 3.3 to 5.7 and from 2.6 to 5.5 for performance and satisfaction, respectively. This demonstrates a positive change in client-perceived performance of and satisfaction with self-identified goals after tendon transfers.

Complications

Complications were related to the surgery or the rehabilitation process. One patient developed a post-operative infection that required irrigation, debridement, and intravenous antibiotics. His follow-up MMT was 3-/5. One patient developed an unrecognized compartment syndrome that resulted in denervation of his transfer and a grade 0/5 manual muscle score. A relatively frequent problem after surgery was olecranon bursitis from the suture used to secure the tendon within the olecranon, as persons with tetraplegia tend to lean on their elbows. This complication was later minimized by placement of the two small posterior cortex holes off to the side of the olecranon to avoid suture knot placement directly beneath the skin. Four patients attenuated their transfer during rehabilitation. One patient was diagnosed immediately after the event by the development of an extension lag during weight lifting against advice. Clinical evaluation did not infer complete disruption of the transfer and the rehabilitation process was slowed. His final MMT score was 3-/5 on the attenuated side compared to 4-/5 on the contralateral side. The other patients retrospectively reported the occurrence of an extension lag when queried about their asymmetric results. Two patients have undergone revision and reattachment of the biceps to the olecranon.

Discussion

Patients with mid-level cervical SCI, such as the most common C5-C6 level, are left with good shoulder function, elbow flexion (biceps brachii and brachialis muscles), brachioradialis muscle, possibly weak or no wrist extension (extensor carpi radialis longus and brevis muscles), and possibly an intact supinator muscle. Elbow extension as well as extrinsic and intrinsic hand function is lacking. In 1975, Moberg[11] renewed interest in the treatment of mid-level cervical SCI by describing a new philosophy in reconstructing upper extremity function that included elbow extension reconstruction. Moberg recognized the benefit of restoring elbow extension and pioneered efforts in the tetraplegic population. The technique utilized the posterior part of the deltoid transferred to the aponeurosis of the triceps using toe extensors as a free graft. The biceps brachii to triceps transfer is an alternative transfer to reconstruct elbow extension. However, the posterior deltoid to triceps transfer has been the most common reconstruction for elbow extension.[19, 20]

Each procedure has specific indications, with inherent advantages and disadvantages to each. Posterior deltoid to triceps transfers are indicated in patients that have a posterior deltoid muscle strength grade 4 or above, lack an active supinator muscle, have an elbow flexion contracture less than 15 degrees, and have full range of motion of the shoulder.[4, 12, 23] The biceps to triceps transfer is indicated in patients that have posterior deltoid muscle strength grade less than 4, an active supinator and brachialis muscle, an elbow flexion contraction of greater than 20 or 30 degrees often combined with a supination deformity, biceps spacticity, paralysis of the costal head of the pectoralis major, strong elbow flexion, and in cases where a deltoid to triceps transfer has failed.[5, 14, 16, 18–21, 24]

Advantages of using the posterior deltoid consist of easy activation as an elbow extensor and no risk of functional loss in case of failure, while the disadvantages include the need for tendon grafting, the potential for elongation of the graft at two repair sites, insufficient strength, attenuation over time, and long immobilization time and course of rehabilita-

tion.[4, 8, 11, 19, 25–27] The biceps to triceps transfer requires only one tendon junction (since a tendon graft is not part of the procedure) with less attenuation and lack of elbow extension over time, allows patients with elbow flexion contractures to be surgical candidates, requires less operative time than deltoid to triceps transfer, is technically easier to perform with less risk of damage to the motor nerve, and allows earlier post-operative mobilization; there is a risk, however, of losing elbow flexion strength, and re-education for elbow extension may be more difficult post-operatively.[16, 19–20]

Moberg[11] described the posterior deltoid to triceps transfer using toe extensors as the free graft to attach the deltoid to the aponeurosis of the triceps. The elbow was immobilized in 10 degrees of flexion for 6 weeks. Flexion was then advanced up to 10 degrees per week. Many reports subsequent to Moberg's[11] initial description have focused on fine tuning graft options, surgical technique, and post-operative rehabilitation to improve reliability of the posterior deltoid to triceps transfer.[3, 8–9, 13, 26, 28–30] Attempted earlier mobilization protocols have led to poor results with this transfer.[4, 12, 31]

The first report of the biceps to triceps transfer in tetraplegics was given by Dr. Leo Mayer, as published by Friedenberg.[32] Dr. Leo Mayer performed bilateral biceps to triceps transfer in a tetraplegic patient with a fracture-dislocation of C3-C4 using the lateral route. Flexion contracture of 50 degrees was present bilaterally, which made ambulation with crutches impossible. Post-operative elbow extension was 150 degrees. Functionality improved except the patient was unable to ambulate with crutches because of marked weakness of the trunk and other upper extremity muscles. Friedenberg, using Dr. Leo Mayer's approach, performed bilateral biceps to triceps transfers in a patient with poliomyelitis who had upper and lower extremity involvement, stating that the biceps to triceps transfer was a stronger transfer than the other described transfers at the time. The biceps to triceps transfer allows the patient to lock the elbows in extension in order to transfer to a wheelchair or to ambulate with crutches.

There have been contradictory statements made about each transfer. Zancolli[12] preferred the biceps to triceps transfer over the posterior deltoid in patients with an intact supinator muscle because of its simplicity. Zancolli also stated that reeducation of elbow extension was very simple, and elbow flexion is not significantly weakened. However, at the Second International Conference on Surgical Rehabilitation of the Upper Limb in Tetraplegia in 1986, Zancolli reported that elbow flexion power is reduced by 24% after biceps to triceps transfer.[5] Lamb and Chan[31] found the one case in his series where the biceps to triceps was performed resulted in failure. Ejeskar[33] stated that the biceps to triceps transfer is the procedure of choice because it only involves one tendon junction. Moberg[14] stated the biceps to triceps to be less reliable.

It has been difficult to evaluate the results of posterior deltoid to biceps transfer studies since the outcome measures have varied. Manual muscle testing, functional testing, or patient satisfaction questionnaires have been inconsistently used, although elbow extension torques equal to or greater than antigravity strength with few complications have been reported (Table 1).

The technique for transferring the biceps to the triceps has changed from routing the biceps tendon laterally,[32] where the radial nerve may be compressed, to routing the biceps medially.[16, 20] The radial nerve may be providing some sensory innervation to the hand, whereas the ulnar nerve on the medial aspect of the arm is usually nonfunctional in tetraplegics. Revol et al.[21] performed 13 biceps to triceps transfers using the medial route in patients with a paralyzed upper part of the pectoralis major muscle and strong elbow flexion. The average extension torque was 3.7 Nm, mean range of motion of 6–137 degrees, and a 47% reduction in elbow flexion power. No patient complained of this loss of power.

Comparative reports between posterior deltoid to triceps transfer and biceps to triceps transfer have been discordant. Ejeskar[33] compared the biceps to triceps transfer with two different grafting techniques using the posterior deltoid to triceps transfer and found better results with the biceps to triceps. Hentz et al.[34] did not find the biceps to triceps transfer as strong as that anticipated from the deltoid to triceps. Kozin and Schloth[35] salvaged failed bilateral posterior deltoid to triceps transfer with biceps to triceps transfers with success. We reported a prospective, randomized study using 16 arms split into a posterior deltoid (8 arms) and biceps (8 arms) group.[20] Seven of 8 arms in the biceps group at 24-month follow-up were found to have antigravity strength, whereas only 1 of 8 arms in the deltoid group produced antigravity strength. Three months following surgery, the biceps group showed a 52% loss of elbow flexion torque and the deltoid group showed a 51% loss of elbow flexion torque. By 24 months following surgery both groups improved but still showed loss of 47% (biceps) and 32% (deltoid). However, no subject appreciated any functional consequences. This study changed our treatment recommendation for elbow extension. Subsequently, only the biceps to triceps transfer was recommended for restoration of elbow extension.

The strength results of deltoid to triceps transfer in our prospective study was unparalleled in other studies (Table 1). We believe this is due to a more stringent protocol for manual strength testing. Our stringent methodology during manual muscle testing lends confidence to the accuracy and repeatability of our strength results.

Our results of biceps to triceps transfer in our cohort have shown to reliably produce elbow extension strength of grade 3 or better in 76% of patients. Even with the positive reports of the deltoid transfer, there are clear advantages of the biceps transfer with regards to surgical technique, outcome, and rehabilitation. The procedure is straightforward and easily reproducible without requiring a graft. The post-operative restrictions are less demanding. There is no difficulty in re-educating the biceps as an extensor. The rehabili-

Table 1. Results of Deltoid to Triceps Transfers

Author	Graft Type	Number of Transfers	Outcome Measure	Mean Outcome Score (range)	Mean Active Extension Deficit (deg)	Condition
Bryan 1977	Toe extensors	14	Powderboard	10.2 kg		Outcome measures not universally measured in all patients
DeBenedetti 1979	Toe extensors	14	MMT, extension power	3.6 (2.5–4.5), 4.6 lbs (1–9)		
Lamb et al. 1983	Toe extensors	16	MMT	4.3 (3–5)	15	Eight elbows had grade 5 power after the transfer
Raczka et al. 1984	Toe extensors	19	MMT	2.9 (0–4)		Three failures due to attenuation of graft, infection, and worsening neurologic status, recommended 15 weeks of immobilization
Freehafer et al. 1984	Tibialis anterior	15	ADLs	Successful		No quantitative data given, patients able to transfer from bed to chair
Lacey et al. 1986	Tibialis anterior	16	MMT, extention torque, ROM	3.4 (2–4), 36.4 kg-cm (21–58), 16–144 deg		Only one transfer had strength less than 3
Ejeskar 1988	23 toe extensor, 7 reflexed triceps tendon	30	ADLs	Improved	some > 60 deg	Toe extensor group: 10 arms > 60 deg extension lag; reflexed triceps group: 6 arms > 60 deg lag
Freehafer 1991	Tibialis anterior or fascia lata	38	ADLs	Improved		No quantitative data given; 2 failures, one from weakness of transfer, one that "did not work"
Mohammed et al. 1992	Tibialis anterior	34	MMT	3.0 (0-4)		Only 24 of 34 arms tested; one arm had strength of 0 from ruptured anastamosis
Paul et al. 1994	Tibialis anterior	9	MMT, extension torque	3.4 (2-4), 10.5 Nm (1.4-27.1)	8.3	2 arms had grade 2 strength; ROM was full in 4 arms and extension lag of 10–20 deg in 5 arms
Dunkerley et al. 2000		10	MMT	1.9 (1–3)		This study compared functional measures between a surgical and nonsurgical group and found no difference
Lieber et al. 2003	Tibialis anterior	40	Extension moment	5.89 Nm		No difference in measurements from post-op and 1 year

The outcome measures vary between studies making comparison difficult. Abbreviations: MMT = manual muscle testing, ADLs = activities of daily living, ROM = range of motion, deg = degree, FIM = functional independence measure.

tation period is shorter than is required for posterior deltoid to triceps transfer, there is significantly less risk of elongation of the transfer. We believe that because of the aforementioned advantages, the biceps to triceps transfer is a better choice for reconstructing elbow extension, and propose expanding its indications as the primary transfer when the brachialis and supinator are active.

Conclusion

We retrospectively reviewed the results of the biceps to triceps transfer performed in a cohort of patients with tetraplegia from a midcervical SCI. Our results show that the biceps to triceps transfer can reliably produce antigravity strength. Because of the advantages of this procedure over the deltoid to triceps transfer, we recommend its use as the preferred technique to reconstruct elbow extension in persons with tetraplegia.

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Clinical Research

A Retrospective Study of the Effect of BMI on Knee Osteotomies Performed for Osteoarthritis

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Abstract

The literature concerning knee osteotomy procedures is unclear regarding the effect of body mass index (BMI) on surgery outcomes. A retrospective chart review was performed to gain insight into this issue. Knee osteotomy procedures for osteoarthritis performed on 47 patients by the Department of Orthopaedic Surgery at Temple University Hospital from November 1999 through December 2005 were analyzed with regard to surgical complications and re-interventions. For surgical candidates with a BMI over 43, the incidence of complications increased significantly (p=.0172). An ANOVA test revealed that mean BMI was significantly higher (p=.033) among those with re-interventions. These results suggest the possibility of a clinical correlation between high BMI and poor outcomes.

Introduction

Osteotomy of the knee, first described in 1958, has been employed for patients with unicompartmental arthritis who are younger than 60 years old and intend to retain a degree of physical activity. This is an alternative to total knee arthroplasty, which may result in activity restrictions. However, the efficacy of knee osteotomies in overweight patients has also become an issue of concern.

The literature is unclear regarding the role of BMI in the outcome of knee osteotomy. Giagoundis et al.[1] found that osteotomized knees of patients above the normal BMI experienced a reduced pain-free postoperative course when compared to patients with normal BMI. Marti et al.[2] remarked on obesity's contribution to the failure of the planned angular correction in high tibial osteotomies. Matthews et al.[3] found that obesity "severely decreases the duration of function of the osteotomy treated knee." Spahn et al.[4] reported a significant difference in post-surgical KOOS scores between obese patients and non-obese patients, with the obese population having an inferior outcome (p<.05). Similarly, Coventry,[5] Pfahler et al.,[6] and Brueckmann et al.[7] all observed a negative impact of obesity on surgical success.

However, this result is not unanimous. Bauer et al.[8] could not find any significant correlation between obesity and osteotomy failure, while Naudie et al.[9] hold that increased BMI is actually a better predictor of successful outcome over the long term compared to normal BMI. In this study, a Kaplan-Meier survivorship curve was employed to show that over 20 years, time to failure was less for patients with a BMI below 25. This is in accordance with Jakob and Murphy[10] who reported that heavier patients are better suited for high tibial lateral closing wedge osteotomies.

Our goal was to examine this variation in results. Differing definitions and modes of analyzing failure contribute to the contradictory results. Therefore, we constructed a more complete definition of failure using elements from previous studies. The goal of this study was to investigate the potential relationship between BMI with the rate of complications and surgical re-intervention after high tibial osteotomy procedures.

Materials and Methods

For the purpose of our study, 29 patients who underwent high tibial osteotomies for medial compartment osteoarthritis, 13 patients who underwent tibial tubercle osteotomies (Macquet's and Fulkerson's) for anterior compartment osteoarthritis, and 5 patients who underwent femoral osteotomies for lateral compartment osteoarthritis were selected based on the availability of their operative reports, clinic notes, and inpatient charts. As no clinic notes or operative reports could be retrieved prior to 1999, the study was limited. The operative reports and inpatient charts of those patients who had their procedures after December 2004 were retrieved from the Alpha Systems computer program. All other operative reports and inpatient charts were obtained from the archives of Temple University Hospital department of Medical Records. Clinic note transcriptions were retrieved from the Cbayscribe dictation service at www.cbayscribe.com from the years 2001 to March 2004, and from September 2004 through the end of 2005. The clinic note transcripts between March and September of 2004 were retrieved from a departmental electronic file.

Age, gender, height, weight, time since surgery, osteotomy type, presurgical angle of genu alignment, post surgical angle of genu alignment, complications, and surgical re-interventions were recorded. Angles of alignment were measured via calibration of knee x-rays off of the IDXRAD

computer system. Pre-surgical angles of alignment were measured from the last available knee x-rays before osteotomy, while post-surgical angles were measured from the latest available film of the osteotomized knee. All varus angles were multiplied by negative one during analysis to distinguish between varus and valgus alignments. Complications were classified as loosening/destruction of inserted screws, destruction of inserted plates, osteotomy collapse, wound infection, deep vein thrombosis, pulmonary embolism, non-union/delayed union, peroneal nerve palsy, tibial plateau fracture, and death. Surgical re-intervention included re-osteotomy, TKA, removal of hardware, and bone grafting.

Data were analyzed using Data Description Inc.'s Data Desk statistical package. Using BMI as a discrete variable, we performed a Fisher Exact T-test comparing complication frequencies at multiple BMI cut-off points. An ANOVA test was used to test if patients with complications had a significantly higher mean BMI. Similarly, the Fisher Exact test was used to assess whether there was a specific BMI above or below which the rate of surgical re-intervention was significantly higher. An ANOVA was performed to test whether the patients who underwent surgical re-interventions had significantly higher BMIs. Chi-Square and ANOVA tests were used for subgroup analysis among the three osteotomy types. The effect of confounding variables such as pre-surgical genu alignment angle and post-surgical genu alignment angle were analyzed by generalized linear models. One patient had to be removed from the analysis after recognizing that he did not meet the criteria for patient inclusion (did not have the osteotomy for osteoarthritis).

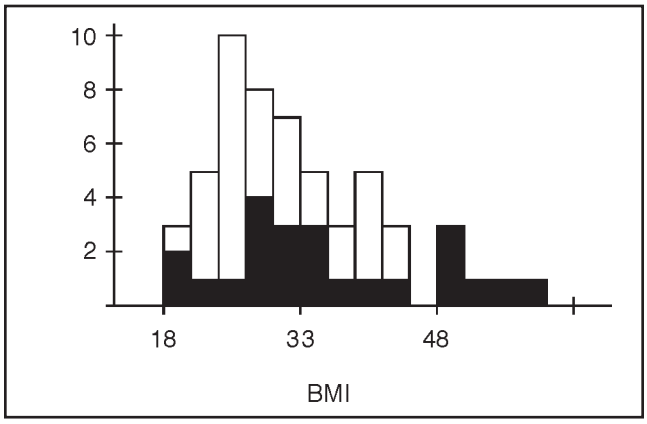
Results

A significantly higher frequency of complications was found at a BMI of 43 and above (p=.0172) [Graph 1, Graph 2]. This was not the case for BMI greater than or equal to 29 (p=.3730) or 40 (p=.4796). The ANOVA test comparing BMI values to existence of complication revealed that the patients with complications did not have a significantly higher mean BMI (p=.0805). Unfortunately we were unable to look at correlations with any specific complication because the sample size was too small and precluded this type of statistical analysis (there were only five patients with BMI of 43+).

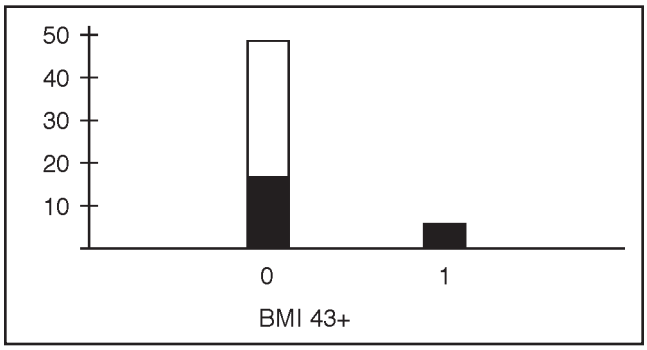
There was no discrete BMI value identified above or below which there was a significant rate for increased surgical re-intervention. BMI values of 43+, 29+, 27+ were statistically evaluated. However, interestingly, ANOVA showed that the group of patients that had surgical re-interventions had a significantly higher mean BMI (39.9) than the group of patients that had no further surgery (31.5, p=.0326) [Graph 3].

When looking at each type of osteotomy by itself, we found that 81.8% of the complications occurred in patients who underwent high tibial osteotomies, 18.2% occurred in patients who underwent tibial tubercle procedures, and 0% occurred in the femoral osteotomy group. Taking into account relative proportions of people in each osteotomy group, 62.1% of high tibial osteotomy patients in the study suffered complications, 30.8% of the tibial tubercle patients had complications, and 0% of femoral osteotomy patients had complications. This latter result proved to be significant (p=.0146).

Graph 1. Histogram of BMI: Dark Portions Represent Those Patients with Complications

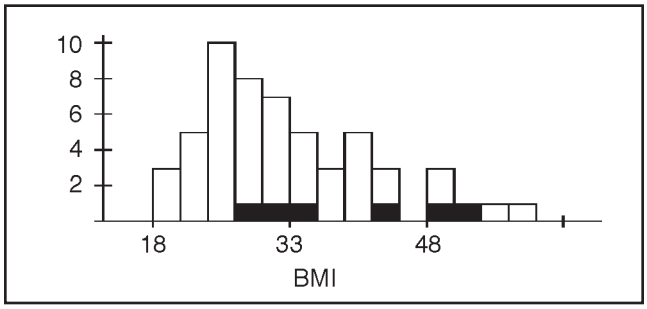


Graph 2. BMI as a Discrete Variable vs. Complications



(0=below BMI 43, 1=BMI 43+, Dark=complications)

Graph 3. Histogram of BMI: Dark Portions Represent Those Patients with Surgical Reinterventions



The ANOVA shows significance with a mean BMI of those who did not have further surgery at 31.5 and those that did at 39.9 (p=.033). The best split value we found was at BMI 29+, but this split was still not perfect (p=.067) at our sample size. However, no one with a BMI of 26 or less had any further surgeries.

The mean BMI of patients with complications in the high tibial osteotomy group was 34.65 versus a mean BMI of 31.76 for those without complications in this group (p=.44). In the tibial tubercle osteotomy group, those with complications had a mean BMI of 36.67, and those without had a mean BMI of 29.92 (p=.18). None of the patients in the femoral osteotomy group had a complication (mean BMI for group=28.59).

Possible confounders that were analyzed in this study included time since surgery, and pre and post surgical angles of genu alignment. Time since surgery was not found to have any significance in predicting the occurrence of complications (p=.3384).

Pre- and post-surgical angle of alignment showed no significant correlation (p=.3126) with complication occurrence, as most patients were brought to an alignment of between four and seven degrees valgus.

Discussion

The findings in this study support the hypothesis that BMI has an effect on surgical outcome. With regard to complications, we were able to find a discrete value of BMI (43) above which there was a significant increase in complication rate (p=.0172). With regard to surgical re-intervention, patients who underwent re-intervention were found to have a significantly higher mean BMI (p=.033). When looking at this data alone, it seems reasonable to suggest that patients with a BMI of above 43 should be excluded as potential candidates for knee osteotomy procedures. Similarly, they should warn their patients that increased BMI is associated with increased risk of surgical re-intervention (8% more likely). When combining these caveats with the significance we report for a greater occurrence of complications in patients undergoing high tibial osteotomies as compared to the other two surgical groups (p=.0146), we can modify our assertion to say that patients with BMI over 43 should not be considered for high tibial osteotomies.

The data become less clear when we see that ANOVA does not support the claim that patients with complications tend to have a higher BMI (p=.0805) and that a Fisher Exact test does not support the claim that patients above a certain BMI tend to have a greater need for surgical revision (p=.067 at BMI cut point of 29). Despite the lack of statistical significance in these two tests, clinical significance is possible when they are viewed in concert with the former two tests that did yield statistical significance (p=.0172 and p=.033). By simply looking at the data in these two failed tests, it is apparent that an association exists between BMI and complications and surgical revision. Small population size in this study, confounded by systematic limitations, played a role in preventing statistical significance from being achieved in these two results.

Conclusion

This study was able to show a significant correlation between BMI and both complications and surgical re-intervention. For complications, we were able to find a cut-off BMI of 43, above which surgery should not be pursued, while we were not able to find such a cut point for re-intervention. The inability to prove significance for all of our tests of association with BMI and complications, and BMI and re-intervention forces us to conclude that uncertainty in this area still exists. Certainly the study should be revisited with a larger sample size to determine definitively, the exact nature of these relationships.

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Clinical Research

Fourteen Year Prospective Results of a High Density Polyethylene Prosthetic Anterior Cruciate Ligament Reconstruction

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Abstract

Our experience with an ultra-high molecular weight polyethylene (UHMWPE) braided graft indicates that this prosthetic ACL is not associated with the complications of other prosthetic ligaments and has equivalent results compared to an autograft. In a prospective cohort study, nine patients underwent ACL reconstruction with the UHMWPE prosthetic graft, and seven patients received an autograft. Each patient completed a questionnaire regarding their symptoms and a Tegner and Lysholm Score. Objective results included a physical examination and KT1000 measurement. Noninferiority statistical methods were utilized to determine a clinically significant difference between the groups. Six individuals from the prosthetic group and six of the controls were followed for an average of 14.1 years. In the prosthetic group, one graft failed at 9.5 years after the reconstruction. There were no sterile effusions. There were no graft failures or sterile effusions in the controls. Tegner scores were equivalent (control = 5.5, prosthetic = 5.0) as were the Lysholm scores (control = 82, prosthetic = 80.5). KT1000 testing for the prosthetic group (mean side-to-side difference in maximal displacement = 1.75mm) was not inferior to the control group (2.0mm). In this prospective study with 14 years of follow-up, the subjective and objective results of a prosthetic ACL compared favorably to an autograft, suggesting a reconsideration of the use of prosthetic grafts as an alternative to ACL reconstruction with autograft.

Introduction

It is estimated that 80,000 people injure their anterior cruciate ligament (ACL) each year in the United States, and approximately 50,000 undergo reconstructive surgery.[4,5] The main problems with the use of autografts are donor site morbidity and availability. Cost, potential for disease transmission, and shortages of supply deter the use of allograft tissue.[2] We believe a prosthetic ligament would avoid these problems as well as allow for early, aggressive physical therapy and return to activity.[8] Past experience with the gore-tex graft was unsuccessful due to unacceptable rates of both sterile effusion and early failure, and its use was discontinued.[8]

The senior author (JST) has noted good, long-term functional results in a group of patients reconstructed with a braided ultra-high molecular weight polyethylene (UHMWPE) graft (Fig. 1) at 5–7 years of follow-up (unreported data). These patients were a part of a prospective multicenter study of the UHMWPE graft. It was discontinued prematurely in light of the poor results of the gore-tex ligament. Recent technological advances include the implantation of a material similar to the UHMWPE braid for knee ligament reconstruction and spine dynamic stabilization procedures. These applications have had clinical success. This prospective study deals with the question of whether the UHMWPE braided prosthetic ACL could provide a durable repair, free of the complications typical of other prosthetic ligaments, and provide equivalent results compared to an autograft.

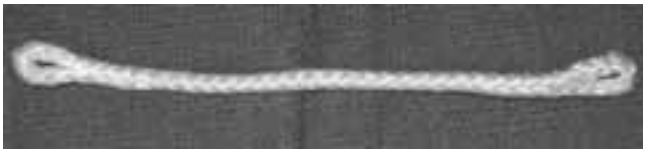

Figure 1. The ultra-high molecular weight polyethylene (UHMWPE) braided graft manufactured by Smith & Nephew Richards, Inc. (Memphis, TN)

Materials and Methods

The prosthetic ligament device that was used consists of braided ultrahigh molecular weight polyethylene (Richards Prosthetic Anterior Cruciate Ligament, Smith & Nephew, Memphis, TN). The braid consists of more than 4,000 individual fibers each with a tensile strength of 435,000 psi. The ultimate tensile strength is 9,000 N and stiffness is 240 N/mm. The device is 4.6 mm in diameter and comes in varying lengths to accommodate various sized knees. The graft is looped at each end to secure bone fixation with a screw and washer.

This study was originally designed to be a randomized prospective study. However, it was terminated early due to concerns regarding the safety of the current allografts. This report represents the long term comparative results of the prosthetic and control group. The experimental group consisted of 9 patients with ACL insufficiency that underwent ACL reconstruction with the Richards Prosthetic Anterior Cruciate Ligament between May 1991 and February 1993. All ACL-injured patients that presented to the senior author were considered candidates for the prosthetic ligament except patients with an isolated ACL rupture. The mean age of the prosthetic group was 27 years old. Seven of the nine had a prior procedure, and the average time from injury to reconstruction was 61.4 months. At the time of reconstruction six of the nine had evidence of degenerative changes.

The control group consisted of 7 individuals with ACL insufficiency who received an ACL reconstruction with an autograft. The control group was formed by including the patient who most closely followed a prosthetic reconstruction that was willing to participate. These included 5 bone-patellar-bone grafts and 2 hamstring grafts. The mean age of the control group was 20 years old. Only 1 had a prior surgical procedure, and the average time from injury to reconstruction was 20.7 months. One patient had evidence of early degenerative changes at the time of reconstruction.

The prosthetic ACL reconstruction was performed using a trans-tibial tunnel and the "over the top" technique on the femoral side (Fig. 2a, 2b). All others from the multi-center study used a trans-tibial and trans-femoral tunnel technique. The tibial tunnel was placed over the posterolateral footprint of the ACL insertion on the tibia to ensure that the graft would not impinge on the notch or the lateral femoral condyle. A notchplasty of the lateral femoral condyle was performed to prevent graft impingement. The graft was routed "over the top" of the lateral femoral condyle utilizing an incision over the distal lateral thigh. The graft was fixed first on the femoral side with a bicortical screw and washer. The tibial side was tensioned and fixed with a bicortical screw with the knee flexed in 30 degrees of flexion.

Postoperatively, all patients underwent an aggressive rehabilitation protocol which included early range of motion, strengthening, and progressive return to general athletic activity. Sport-specific activity was the last phase of the protocol in those patients returning to high level sports activity.

This analysis will report the results of two evaluations. The earlier follow-up data of 5–7 years represents previously unpublished results of the experimental group. Extensive pre-operative and post-operative subjective and objective evaluations were performed on each patient. The subjective evaluations included visual analog scales of symptoms (pain, swelling, locking, catching, and giving way) and function. The subjective functional questions related to problems with stair ascent/descent and instability during multiple activities, e.g. walking level and uneven ground, changing directions, twisting or pivoting, squatting, jumping, running, and carrying objects. Tegner activity scales and Lysholm knee scores were also collected.[6, 15] Objective evaluations included KT-1000 arthrometer testing, Cybex testing, single leg hop test, and X-ray evaluation. Physical examinations consisted of the Lachman test, the anterior drawer test, the pivot shift test, varus and valgus stress testing, and assessments of effusion, tenderness, and range of motion. Data were analyzed using paired sample t-test (2 tailed) and repeated measures analysis of variance (ANOVA). Pre-operative values were compared to the 5–7 year follow-up data for significant differences.

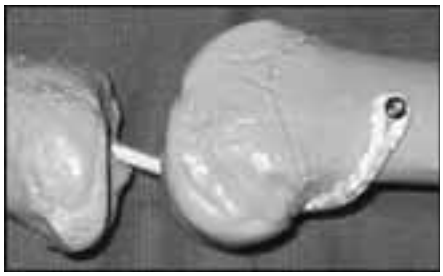

Figure 2a. A lateral view of the prosthetic ligament in the "over the top" position.

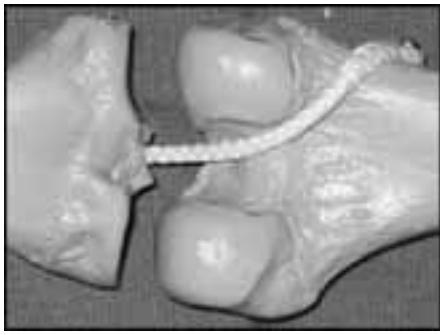

Figure 2b. A posterior view of the prosthetic ligament in the "over the top" position.

The most recent follow-up data of 14.1 years compares the experimental group to the control group in a noninferiority statistical evaluation. A noninferiority trial is indicated when it would be unethical or impractical to include a placebo group, such as leaving a young, active adult with an unreconstructed ACL when it is otherwise indicated. The objective is to show that the experimental treatment is not inferior to the standard treatment. Instead of utilizing a null hypothesis, noninferiority trials attempt to disprove an alternative hypothesis which states that there is a difference between the two cohorts.[10, 13] The value generated by noninferiority statistical methods is a power value. This power value is a measure of the ability to detect a clinically relevant difference between an experimental and a control group via the recorded parameter.

In order to calculate this power value, the investigator must establish a value for a clinically relevant difference between the cohorts that is reasonable to detect. There are two acceptable methods to determine the clinically relevant

difference. One is to rely on a foundation of clinical experience to set reasonable values. The other is to utilize the medical literature to facilitate the determination of this value. The clinically relevant difference used in this study was generated after reviewing several prospective randomized trials comparing non-operative versus operative treatment of ACL injuries.[1, 3, 7]

Attempts were made to contact all patients. Each patient was asked specifically about swelling, instability, and subsequent surgery. Each patient was asked to complete two validated outcome measures, the Tegner score and Lysholm score. A physical examination was performed with the main objective parameter measured being maximal anterior displacement of the reconstructed leg versus the uninjured leg, measured with a KT1000 arthrometer.

Results

At the earlier follow-up, 8 of the 9 patients were available for evaluation at a range of 63–84 months. Seven of the patients were able to return for examination, one was available for a phone interview, and one patient was lost to follow-up 18 months after surgery.

The results of the visual analog scale revealed significant improvement in all symptoms assessed at 5–7 years as compared to pre-operatively ($p<0.05$). The functional scores likewise showed significant improvement in all categories ($p<0.05$). The mean follow-up Tegner score was 6.3. This was significantly improved from the average pre-operative Tegner score of 4.2 ($p<0.05$). The mean pre-operative Lysholm score was 73. This increased to an average post-operative Lysholm score of 93. This was a significant difference ($p<0.01$).

On KT-1000 testing the mean pre-operative side-to-side difference in maximal manual displacement was 6.3mm. This value was significantly decreased to 2.5mm at final follow-up in this report of 5–7 year results ($p<0.01$). A positive Lachman exam was elicited on each patient pre-operatively (avg = +2.2). This dropped to an average Lachman test of +0.7 at the 5–7 year follow-up. This was significantly different ($p<0.01$). The mean anterior drawer test was 1.1 pre-operatively which improved to 0.6 at 5–7 years. A significant difference in the pivot shift was also found with a pre-operative average of 1.9 versus a 5–7 year average of 0.6 ($p<0.05$). The Cybex results on the reconstructed knees revealed a decline in quadriceps and hamstring strength at 5–7 years compared to the pre-operative values. Interestingly, the unaffected limbs showed a similar decline. In fact, Cybex testing of peak hamstring torque revealed no significant difference between limbs ($p>0.5$), and there was only a 7% difference in quadriceps peak torque. Single leg hop tests revealed significant improvement in the affected limbs when compared to pre-operative values ($p<0.05$) (Fig. 3).

For the most recent evaluation, attempts were made to contact each patient from the control and the prosthetic group. Six patients from the prosthetic ligament group provided Tegner and Lysholm scores, including the patient with a documented graft failure. Four were available for a physical examination. Six patients from the experimental group provided Tegner and Lysholm scores while five were available for a physical examination. The average follow-up was 14.1 years.

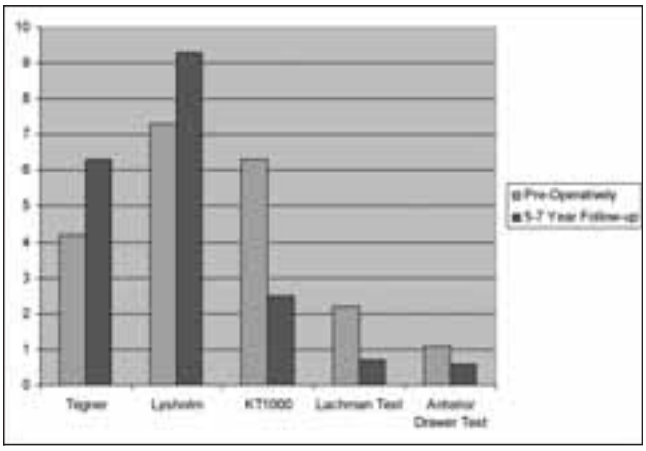

Figure 3. Pre-operative compared to 5–7 year follow-up data.

There was three second-look arthroscopies in the prosthetic group. Two of the second-looks, 18 months and 5.5 years after the reconstruction, documented an intact ligament. The only graft failure occurred 9.5 years after the reconstruction and was documented by a diagnostic arthroscopy. In the control group, there were no second-look arthroscopies. While there were no documented symptomatic failures in the control group, one patient had a non-functioning ACL as manifested by a side-to-side difference in maximal displacement greater than 3mm (4mm) on KT-1000 testing.

The Tegner and Lysholm scores between the two groups were similar (Figure 4). The Lysholm score for the prosthetic group was 80.5 while it was 82 in the control group. The Tegner score for the prosthetic group was 5.0 and 5.5 for the controls. While the $Power_{Lysholm}$ was 23% to detect the minimal clinically relevant difference of 6, the $Power_{Tegner}$ was 88% to detect the minimal clinically relevant difference of 3.

	Tegner	Lysholm	KT1000 (mm)
Prosthetic	5.0 +/– 1.79	80.5 +/– 14.72	1.75 +/– 0.5
Control	5.5 +/– 1.64	82 +/- 8.69	2.0 +/– 1.2
Power	88%	23%	95%

Figure 4. 14 year results of the prosthetic group and control group.

Similarly, the KT1000 data between the groups were nearly equivalent (Fig. 4). The mean side-to-side difference in maximal displacement was 1.75 mm in the prosthetic group and 2.0 mm in the control group. The $Power_{KT1000}$ was 95% to detect the minimal clinically relevant difference of 3 mm.

Discussion

It appears that a UHMWPE prosthetic ligament for ACL reconstruction has several advantages. Not only would a prosthetic option avoid the disadvantages typical of autografts and allografts, it has a unique advantage. A synthetic graft does not require revascularization and allows early, aggressive rehabilitation with more rapid return to pre-injury level of function. All patients in the prosthetic cohort return to their desired level of activity within 3–6 months (Fig. 5) while none of the controls returned to their desired level of activity prior to 9 months.

Figure 5. A 16-year-old skier from the prosthetic group who was allowed to resume full activities, including skiing, 12 weeks from his prosthetic ACL reconstruction.

Two recent clinical applications of a material similar to the UHMWPE, the DYNESYS system (Zimmer, Warsaw, IN) and the Ligament Advancement Reinforcement System (LARS) artificial ligament (Canada), are enjoying clinical success. The DYNESYS system utilizes a polyethylene terephthalate cord to help dynamically stabilize unstable spine segments. It has been used extensively in Europe and recently in the US with no reported implant failures secondary to the polyethylene cord.[11, 12, 14] The LARS ligament, also a terephthalic polyethylene cord, is a prosthetic ACL currently being implanted in Canada. A report of two-year follow-up shows excellent stability with no effusions.[9] Data from patients who had a reconstruction with the LARS ligament suggest that an interval from injury to return to play of 6 weeks to 3 months is possible.

When the prosthetic group was evaluated at 5–7 years post reconstruction, pre-operative and post-operative data for the visual analog scale, Tegner scores, Lysholm scores, and KT1000 data revealed significant improvements in all parameters.

At the most recent follow-up of 14.1 years, there is considerable power to detect a clinically relevant difference in regards to both Tegner scores and KT-1000 data while the power to detect a clinically relevant difference with the Lysholm score was not as great. The literature supported clinically relevant differences of the Tegner score of 3 and KT-1000 of 3mm but only of 6 in regards to the Lysholm score. When the scale of the Tegner score (10) and the Lysholm score (100) is considered, it becomes apparent that a change of 3 in the Tegner score represents a 30% alteration whereas a change of 6 in the Lysholm score represents a change of only 6%. While the Lysholm score was evaluated in this report because it allowed comparison to prior data, it is not as sensitive of a marker of clinical improvement after an ACL reconstruction as the Tegner score.

In this study comparing a prosthetic ligament to an autograft, there were no occurrences of early prosthesis failure. The one failure occurred 9.5 years after the reconstruction. There were no sterile pyarthroses. The subjective scores and the objective results between the groups are equivalent.

Based on this result, we reject the alternative hypothesis which states that there is a difference between the prosthetic ligament group and the autograft group. Therefore, it is our belief that the clinical results, specifically patient-rated satisfaction and functional results as well as a long-term objective measure of stability, were similar between this prosthetic ligament and an autograft. This data justifies reconsideration of a UHMWPE prosthetic graft utilizing the over-the-top technique as an ACL reconstructive option.

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Clinical Research

Combined Gunshot Femur Fractures with Associated Vascular Injury: A Retrospective Analysis

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Abstract

Gunshot femoral fractures with vascular injury are limb-threatening injuries which require rapid and coordinated interdisciplinary care in order to achieve acceptable outcomes. Most controversy regarding the management of these injuries is focused on the sequence of surgical intervention with regards to orthopaedic and vascular surgery. We performed a retrospective study of twenty four patients treated at an urban level 1 trauma center over a ten-year period with low velocity gunshot wounds resulting in femur fractures and major vascular injury. Data was stratified according to sequence of surgical intervention. The mean age was 31.3 years. Mean time to revascularization was highest in patient undergoing definitive orthopaedic fixation first (660 minutes) and lowest in patient undergoing shunting first (210 minutes). Most complications were in patients undergoing vascular repair first, including two disrupted repairs requiring immediate revision after subsequent orthopaedic fixation. Other complications included compartment syndrome and one amputation. Surgical sequence did not affect the incidence of limb loss.

Introduction

Femoral fractures with combined vascular injury from low energy gunshot wounds are uncommon injury patterns seldom seen in most trauma centers. When encountered, however, these patients require rapid, coordinated interdisciplinary care in order to achieve acceptable outcomes. Low velocity gunshot wound (GSW) femoral shaft fractures are successfully treated with intramedullary nailing without formal debridement of the wound or fracture site.[17, 18] However, vascular injuries require emergent revascularization and therefore take precedence over definitive fracture stabilization.[2, 4, 5, 7–10, 14, 17] This creates a shift in priorities with multiple methods available to achieve rapid revascularization with skeletal stability. Management of these injuries is critical in both civilian and military gunshot injuries. The current controversy centers on the proper sequence of surgical intervention with regards to orthopaedic and vascular stabilization.

The three commonly described approaches to the management of these injuries include 1) Definitive vascular repair followed by definitive orthopaedic fixation; 2) Vascular shunting followed by definitive orthopaedic fixation followed by definitive vascular repair; and 3) Orthopaedic fixation followed by vascular repair. Temporary orthopaedic stabilization in the form of an external fixator followed by definitive vascular repair followed by delayed definitive orthopaedic fixation is also a described treatment option.

While several studies have investigated either combined femur fractures with vascular injury or long bone fractures and vascular injuries from gunshot wounds, few have focused on only femur fractures with vascular injuries from gunshot wounds.[2, 4, 5, 7–10, 12, 17, 18] The purpose of this study was to retrospectively investigate the experience at one urban level one trauma center with these injuries and to examine the implication of surgical sequence with regards to short-term complications and ischemia time.

Materials and Methods

The medical records of all patients admitted to Temple University Hospital, a level one trauma center in Philadelphia, PA, from January 1996 to December 2005 were searched for the following combination of injuries: gunshot wound, vascular injury, femur fracture. IRB approval was obtained for this study. Patients with gunshot wounds and vascular injury in a limb other than the limb with the femur fracture were excluded. High energy gunshot injuries such as those by shotgun were excluded. All femoral fractures, including peritrochanteric and supracondylar femur fractures were included if accompanied by vascular injury. Major venous injuries were also included if accompanied by ipsilateral femur fractures. All patients were civilians.

Data was recorded directly from chart review to a database with ID numbers assigned to each patient. Patient age, sex, and mechanism of injury were recorded. The type of fracture (eg. femoral shaft, supracondylar, etc) as well as the type of vascular injury (eg. superficial femoral artery transection, superficial femoral vein transection, etc) was noted. The method used to diagnose the vascular injury was noted in all cases. Multiple time points were measured: 1) time from injury until arrival in the emergency department; 2) time from injury until diagnosis of vascular injury; 3) time from injury until re-vascularization completed; 4) time from injury until fracture stabilization; 5) total surgical time; and

6) length of stay. The incidence of fasciotomies, whether prophylactic or for compartment syndrome, were noted. The surgical sequence of events with regards to orthopaedic and vascular intervention was recorded. Finally, complications such as compartment syndrome, amputation, vascular repair disruption, and death were recorded.

Results

Thirty one patients with femoral fractures, gunshot wounds, and vascular injury were identified by the search of the medical records. Five patients were excluded due to femoral fractures in an opposite limb from the vascular injury. These were patients with multiple gunshot injuries resulting in femur fractures on one limb and vascular injuries without fractures in the other limb. One patient had a shotgun injury and was therefore excluded. One patient with multiple low velocity gunshot wounds to bilateral lower extremities was excluded due to intraoperative death. This patient presented without a palpable blood pressure and had injuries to the left common femoral artery and bilateral common femoral veins. The patient expired in the operating room before revascularization was completed and fracture stabilization could be performed. This left a total of twenty four patients for further consideration.

The mean age was 31.3 years (median 29, range 17–71). All injuries were characterized as low-velocity gunshot wounds. The anatomic location of the femur fractures included femoral shaft (12 cases), the peritrochanteric region (5 cases), and the supracondylar region (7 cases). Vascular injury location included the superficial femoral artery (6 cases), superficial femoral vein (3 cases), superficial femoral artery and vein (3 cases), popliteal artery (4 cases), popliteal artery and vein (5 cases), profunda femoris artery (3 cases), external iliac vein (1 case).

Arteriograms were used pre-operatively for diagnosis in three cases, whereas arteriogram was performed intraoperatively in six cases. The diagnosis was made in most cases by the clinical findings of pulselessness and negative arterial dopplers, pulsatile bleeding, or massive hematoma in patients with penetrating wounds in proximity to major extremity vessels.

Because of the perceived importance of surgical sequence, we stratified our data according to the surgical sequence of events in a similar fashion to that of McHenry, et al.[9] The three main groups encompass the three main modes of treatment commonly described: orthopaedic fixation followed by vascular repair/reconstruction (Group 1), vascular repair/ reconstruction followed by orthopaedic fixation (Group 2), and vascular shunting followed by orthopaedic fixation followed by vascular repair/ reconstruction (Group 3). We also decided to include patients who did not fall into any of these categories (Group 4). These mainly included patients with major venous injuries or arterial injuries treated by interventional radiology (eg. profunda femoris branches).

Time to revascularization, time until fracture stabilization, and total surgical time for all four groups are outlined in Table 1.

Table 1. Time for Revascularization, Fracture Stabilization, and Total Surgical Time for All Patients

Group	Mean Time Until Revascularization (Median)	Mean Time Until Fracture Stabilization (Median)	Mean Total Surgical Time (Median)
Group 1 (n=3)	550 (660)	390 (405)	380 (360)
Group 2 (n=12)	315 (285)	1826 (545)	468 (390)
Group 3 (n=4)	233 (210)	435 (375)	810 (720)
Group 4 (n=5)	217 (210)	620 (500)	475 (480)

Group 1: Orthopaedic followed by vascular
Group 2: Vascular followed by orthopaedic
Group 3: Shunting followed by orthopaedic followed by vascular
Group 4: Other

Eleven of the 24 patients underwent prophylactic fasciotomies. Complications included two revised vascular repairs, five compartment syndromes, and one below knee amputation. One patient also developed acute renal failure and one patient developed a superficial wound infection. The complications are outlined in Table 2.

Table 2. Complications for All Groups

Group 1 (n=3)	None
Group 2 (n=12)	2 leg compartment syndromes, 1 thigh compartment syndrome, 2 revision vascular repair, 2 foot drop, 1 fasciotomy wound infection
Group 3 (n=4)	1 compartment syndrome, 1 acute renal failure
Group 4 (n=5)	Peripheral neuropathy, 1 compartment syndrome, 1 below knee amputation

Though there were several complications as listed in Table 2, the rate of limb salvage was excellent regardless of the sequence of surgical intervention. The lack of complications in Group 1 likely is due to the small number of patients. The long time to revascularization in this group (Table 1) is due to late diagnosis and presentation. One patient in this group had a late diagnosis of a vascular injury, which turned out to be a pseudoaneurysm treated with vascular reconstruction. Another patient had a delayed presentation after being transferred from another institution. Nevertheless, this patient was treated with orthopaedic fixation first.

Complications were numerous in Group 2, in which vascular repair was performed first, followed by orthopaedic fixation. Though eleven of the twenty-four patients in this study did have prophylactic fasciotomies, three patients in this group who did not have prophylactic fasciotomies developed compartment syndrome and subsequently had fasciotomies. Perhaps more notable is the incidence of two patients in which the vascular reconstruction had to be revised after orthopaedic fixation. At the time of writing this manuscript, we have treated another patient who would have been placed

in Group 2 who also had a revision of a vascular reconstruction after orthopaedic fixation. Though this danger has been recognized as a theoretical risk of manipulating and pulling an unstable limb out to length which has a fresh vascular repair or reconstruction, other studies have not reported this complication.[9, 10]

One patient in Group 3 had only one kidney, and subsequently developed acute renal failure which resolved. One patient in Group 4 had severe neurovascular injuries from the gunshot wound (popliteal, tibial, peroneal arteries and veins, tibial nerve) and underwent IM nailing of the femur and a below knee amputation due to the extent of injuries.

Discussion

This study represents the largest group of low velocity gunshot femur fractures with vascular injury reported in the English literature to our knowledge. It is generally agreed upon that revascularization of the ischemic limb is of paramount importance. However, some believe that this should be performed in a stabilized limb. The current controversies regarding this injury, therefore, center on the proper surgical sequence of events.

Surgical sequence of intervention has long been a controversy in the management of these injuries.[1, 2, 4–18] There have been advocates of vascular intervention first as well as fracture fixation first. In a recent study, McHenry retrospectively reported on 27 patients with both lower and upper extremity gunshot injuries with fracture and vascular injury over a 10 year period.[9] They stratified patients into three groups that were identical to our Groups 1–3. There were no cases of vascular repair disruption after fracture fixation in 22 cases. Four of five patients who underwent orthopaedic fixation before revascularization required fasciotomies while only 8 of 22 patients who were revascularized first required fasciotomies. They therefore concluded that revascularization should be done before orthopaedic fixation. It should be pointed out again, however, that this study included only 16 lower extremity injuries and the anatomic locations were not mentioned. The shortening and subsequent traction required to reduce a femoral fracture can be considerably more than that for a humerus or tibia fracture.

Many other studies have supported the claim that orthopaedic fixation should be done after vascular repair. Menzoian retrospectively reported in 1982 on 56 patients with vascular injuries below the inguinal ligament, some without fractures.[10] 15 of these patients had fractures (4 of the femur) and the vascular injuries included all mechanisms. They advocated generous use of external fixation and performed vascular repair both before and after orthopaedic fixation with presumably similar results. However, they clearly advocated the revascularization of patients with severely ischemic limbs prior to orthopaedic fixation. Ashworth retrospectively reported in 1988 on 25 patients with vascular injuries of the lower extremity, 12 of which had fractures with only 2 of these being femoral fractures.[2] Two fractures required external fixation before revascularization due to gross instability. Otherwise, vascular repair was done first followed by orthopaedic fixation. A 96% limb salvage rate was reported with 84% of patients ambulating independently. Their conclusion was that vascular repair should be done before orthopaedic stabilization, even though they acknowledged that in cases of gross displacement or instability that external fixation may need to be done first. Wiss also reported on five patients with 5 patients with gunshot femur fractures and vascular injury treated with IM nailing after vascular reconstruction.[18] All the fractures united without any vascular insufficiency.

In contrast, Starr examined 26 patients with femur fractures and vascular injuries. 13 patients had gunshot injuries while the rest were from blunt trauma.[17] External fixation was used in six patients who generally had high MESS and ISS scores and could not undergo an IM nailing procedure. 10 patients had IM nailing done before vascular repair while 9 patients had IM nailing done after vascular repair. They noted equivalent limb salvage rates whether the fixation was performed before or after vascular repair. In a related study, Nowotarski reported on 54 patients with femur fractures treated with temporary external fixation, 8 of whom had vascular injury.[12] These eight patients underwent rapid unilateral external fixation to bring the fracture out to length prior to definitive revascularization. Patients were brought back to the OR at approximately seven days for conversion to an intramedullary nail with good clinical results.

There were no incidences of limb loss in our study regardless of the sequence of surgical intervention. Nevertheless, the 23% incidence of compartment syndrome in patients who did not undergo prophylactic fasciotomy indicates the likely need for more prophylactic fasciotomies in these patients. Also, two patients in this study and the more recent patient mentioned earlier had vascular reconstructions revised after orthopaedic fixation due to disruption attributed to manipulation and lengthening. Though there were no obvious immediate sequelae from either of these complications, these are only a few cases and the long term consequences are unknown. In one case, the vascular surgeon was not made aware of the fracture until the vascular reconstruction was completed. This was a highly comminuted femoral shaft fracture with shortening. This was treated with rapid external fixation which caused the interposition graft to fail, which required revision. A comminuted and shortened femoral fracture was present in the second and third cases. Closed reduction and intramedullary nailing was performed in both cases which similarly resulted in graft failure requiring revision. None of the cases had catastrophic disruption with pulsatile bleeding. But in light of the recognition of disrupted vascular repairs reported in Group 2, we have now shifted towards more use of the technique of initial rapid external fixation followed by vascular repair/reconstruction whenever possible. Recognition of the disrupted repair is important to diagnose while the patient is still on the operating room table. External fixators can be safely converted to IM nails in the face of a vascular repair if done at approxi-

mately 7–10 days. This has been shown in studies by both Iannacone and Nowotarski.[8, 17]

Arterial shunting as a temporary measure is somewhat controversial but was successful in this series as well as in other studies.[9, 13] Certainly, the group in this study with the shortest revascularization times was Group 3, in which arterial shunting was performed. Venous shunting can also be performed, if possible.[16] However, shunting does lead to increased operative times as shown in Table 1.

Fasciotomies were performed frequently in our series, yet there were still five cases of compartment syndrome, including both the leg and the thigh. Retrospectively, it seems as if we should have performed more fasciotomies. However, it should be recognized that one wound infection did occur post fasciotomy. Furthermore, more long term data is required to determine the morbidity of fasciotomies in this patient population.

Mention should be made of the diagnosis of arterial injury in this study. All three cases that had pre-operative arteriogram were found to have profunda femoris arterial injuries that were treated with interventional radiologic hemostatic methods. In six cases, arteriography was done intraoperatively. It is our opinion that patients with so-called "hard signs" of vascular injuries such as loss of pulse, pulsatile bleeding, or expanding hematoma in cases of penetrating injuries with large vessel proximity should go to the operating room immediately. In proximity cases with abnormal examination but without hard signs, a trip to the angiography suite may be appropriate. 21 of 24 cases in this study went directly from the emergency room to the operating room.

The few numbers of patients and retrospective nature of this and other studies on this topic make treatment recommendations difficult. In order to improve the statistical significance of the data, we are currently performing a meta-analysis of the literature to help pool the data on this uncommon injury pattern. Furthermore, we are developing a prospective study in order to more accurately gather data and be able to examine sequelae with longer followup. Issues such as fracture healing, vascular claudication, and late ischemia can be examined in comparison to a control group.

Conclusions

Gunshot femur fractures with vascular injuries are limb-threatening injuries that can be treated appropriately with interdisciplinary intervention between trauma surgeons, orthopaedic surgeons, and vascular surgeons. The rate of limb loss does not appear to be affected by the sequence of surgical intervention. However, revascularization of a grossly unstable limb does pose a real risk of vascular repair disruption after subsequent fracture fixation. This surprisingly has not been previously reported. The reason that the issue of surgical sequence has been controversial is partly due to the fact that significant differences have not been noted in outcomes in this or other studies. Furthermore, this is a rare condition and these studies have been retrospective. They do not have statistical power and clinical significance is questionable. We advocate an individualized approach to each case with ischemia time kept in mind and a goal of revascularization by six hours post injury. Fasciotomies should be performed, particularly in cases of prolonged ischemia. In unstable femur fractures that are diagnosed and brought to the operating room early, rapid external fixation followed by definitive repair is likely the best option. However, in cases of severe ischemia or delayed diagnosis, definitive repair or shunting followed by fracture fixation is probably a better sequence to pursue. Therefore, the answer is not orthopedic first or vascular first, but rapid revascularization preferably in a stable limb.

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Clinical Research

Intra-Articular Civilian Gunshot Wounds to the Knee: Initial Management and Early Treatment

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Abstract

Introduction: No standardization of care for intra-articular gunshot injuries has been established. Many advocate surgical irrigation and debridement. Prophylactic antibiotics are recommended due to the lack of autosterilization of a discharged bullet. The purpose of this retrospective study was to review trends in treatment of knee intra-articular gunshot injuries at Temple University Hospital and the incidence of acute joint sepsis.

Methods: Using a database of all patients treated by the Orthopaedic Department, 22 patients that had sustained an intra-articular injury of the knee were identified, 18 with an isolated injury. Of the 18 identified, 14 (77%) were treated with surgical irrigation and debridement with or without internal fixation of adjacent fractures. All patients were treated with an intravenous antibiotic. No cases of joint sepsis were recorded after an average of 16.6 days of follow-up after discharge from the hospital.

Conclusion: We recommend the use of a first-generation cephalosporin antibiotic for the treatment of an intra-articular knee gunshot injury. Surgical irrigation and debridement with or without fracture stabilization can be safely performed within 48 hours after injury. Administration of antibiotics immediately upon arrival to the emergency department may be more important for the prevention of joint sepsis than emergent surgical management.

Introduction

According to the U.S. Department of Justice in the National Crime Victimization Survey (NCVS) in 2005, 477,040 victims of violent crimes involved an offender with a firearm compromising 9% of the 4.7 million violent crimes committed.[1] Federal Bureau of Investigation estimates for 2004 report that approximately 66% of the 16,137 murders during that year were committed with firearms.[1] Victims of gun violence are on the rise nationally resulting in more emergency room visits annually secondary to gunshot wound injuries. Physicians are faced with treating such wounds. There continues to be a debate regarding the proper treatment modalities of gunshot wounds to the extremities.

Many of the treatment modalities for intra-articular gunshot wounds have been gained solely by clinical and anecdotal experience. A standardization of treatment is lacking in the literature. A debate continues regarding the need for antibiotic prophylaxis and the duration of such treatment if deemed necessary. Early theories that the thermocapacity of the missile at the time of firing rendering the bullet tract sterile,[8, 9] has been disputed by studies by Thoresby and Darlow et al.[10] which documented the contamination of wounds by the vacuum effect of low-velocity missiles. Their work was later validated in an animal model study by Tian et al.[12] in 1988 with positive bacteriologic cultures obtained immediately after injury and a peak in cultured counts 12 to 24 hours after injury. In 1978 Wolf et al.[11] concluded that bullets should not be ruled out as possible sources of infection. Bullets contaminated with *Staph. aureus* prior to discharge were recovered from a sterile sand target proved culture positive of the original contaminate.

Despite the scientific evidence for the potential for joint contamination by a traversing low-velocity bullet no consensus is available in the current literature of appropriate antibiotic prophylaxis. The variability of the injury sustained to the knee joint compounds the question of appropriate antibiotic therapy. In attempts to simplify the treatment Ganocy et al.[6] in 1998 devised a classification system for intra-articular low-velocity gunshot injuries, which accounted for the location of the bullet, contamination of the wound and associated fractures. These 3 combined parameters, graded on a level of increasing severity 1 to 3, could then be used to guide treatment. Despite demonstrating the need for antibiotic prophylaxis, no consensus of the appropriate antibiotic agent was determined.

Numerous reports advocate the necessity of immediate joint irrigation and debridement, as well as removal of any retained bullet fragments, loose pieces of bone, and foreign material carried intra-articularly by the "vacuum effect" that follows low velocity missile penetration. Complications of retained bullet fragments include mechanical destruction of the articular cartilage, leading to noninflammatory arthritic change, and obstruction of normal joint motion.[20, 21] Furthermore, these fragments can become a nidus for infection and eventually leading to septic arthritis. Contamination can result not only in septic arthritis, but chondrolysis, periarticular fibrosis, joint destruction and lead toxicity.[14, 19, 20] Lead intoxication can occur within days to several years after a gunshot wound, with peak blood lead levels occurring at 4 to

6 months according to an animal model.[15, 17,19] Retained bullet fragments within a joint may dissolve within the synovial fluid and deposit lead within the subsynovial tissues. This can lead to arthritic changes, even in the absence of fragment-induced mechanical wear, and the systemic effects of plumbism.[18, 19]

The purpose of the study was to review current trends in early treatment of intra-articular gunshot injuries to the knee at Temple University Hospital and the effectiveness of treatment in preventing septic joint arthritis.

Material and Methods

Approval by the Institutional Review Board was obtained to conduct a retrospective review of the treatments provided for intra-articular gunshot injuries to the knee. A prospective patient database search was conducted to identify all patients that had sustained a gunshot wound from April 2004 to September 2006. Two hundred sixty-three patients that had sustained a gunshot injury were identified using "gsw" and "gunshot" as search criteria. This was then narrowed down by the addition of the term "knee" to the search, resulting in the identification of 26 patients. Those with documented involvement of the knee joint on the orthopaedic assessment (initial consult or history and physical) were identified. Inclusion criteria included either radiographs demonstrating intra-articular bullet fragments or either a positive saline load test through the entry or exit wound.

Results

Twenty-two patients were identified with mean age of 31.14 years (20 to 44 years). Eighteen (82%) had sustained an isolated gunshot injury to a knee, 2 had associated bowel injuries, 1 with an associated scrotal injury and 1 patient had sustained multiple bilateral lower extremity gunshot wounds necessitating bilateral leg fasciotomies 3 days later (Table 1).

Table 1. Additional Injuries

Tibial Plateau Fracture	4
Distal Femoral Fracture	5
Femoral Shaft Fracture	1
Fibular Head Fracture	1
Patellar Fracture	2
Osteochondral Fracture	1
Abdominal Wounds	2
Urologic Injuries	2
Compartment Syndrome	1

All patients received appropriate tetanus prophylaxis and antibiotic prophylaxis upon evaluation in the emergency department, an average of 19.8 minutes after being triaged. A first-generation cephalosporin antibiotic was administered to all but two patients who received clindamycin secondary to a stated penicillin allergy (Table 2). The two patients with abdominal wounds were also administered a second-generation cephalosporin. Eighteen patients with an isolated knee involvement were continued on intravenous antibiotic treatment with either a first-generation cephalosporin or clindamycin for an average of 71.3 hours during the hospital stay. Fourteen of 18 (77%) patients underwent surgical debridement with 5 patients simultaneously having open reduction internal fixation of adjacent fractures or soft tissue repair (Table 3). These patients were taken to the operating room an average of 21 hours 43 minutes after arriving in the emergency department (range 2 hours 12 minutes to 79 hours 54 minutes). The remaining 4 patients had no radiographic evidence of retained bullet fragments and were treated with local wound care.

Table 2

Time of Initial Abx After Arrival to ER (minutes)	Antibiotic Prophylaxis	Timing of Operative Treatment (hr:min)
19.8 minutes	Ancef 1gram*	21 hours 43 minutes

*2 patients received Clindamycin 600 milligrams because of allergy

Table 3. Surgical Treatments

Arthroscopic Debridement & ORIF of Distal Femur and Proximal Tibial
Arthroscopic Debridement
Open Debridement, Bullet Removal & Femoral Retrograde IM rod
Arthroscopic Debridement & ORIF of Distal Femur
Arthroscopic Debridement & Delayed ORIF Tibial Plateau
Arthroscopic Debridement
Open Debridement, Bullet Removal & ORIF of Distal Femur
Arthroscopic Debridement
Open Debridement
Arthroscopic Debridement & Bullet Removal
Open Debridement & Bullet Removal
Open Debridement, Bullet Removal & ORIF of Tibial Plateau
Open Debridement & Bullet Removal
Arthroscopic Debridement & ORIF of Osteochondral Fracture
Open Debridement
Open Debridement & Partial Patellectomy

After discharge all patients treated surgically were placed on an oral antibiotic (1st generation cephalosporin versus clindamycin) for 5 to 7 days per the decision of the treating surgeon. Of the 4 patients treated non-operatively one (25%) was continued on an oral first-generation cephalosporin for one week.

Review of the medical records revealed initial patient follow-up after discharge was a mean of 16.6 days (range 10 to 24). There were no recorded signs and symptoms consistent with infection. All patients denied the presence of fevers. Subjective pain evaluation was variable and not accurately recorded.

There were no reported cases of subsequent septic arthritis in the 22 patients treated in this series. Also currently there is no reported hardware infections in the 5 patients that underwent internal fixation of adjacent periarticular fractures (Table 3).

Discussion

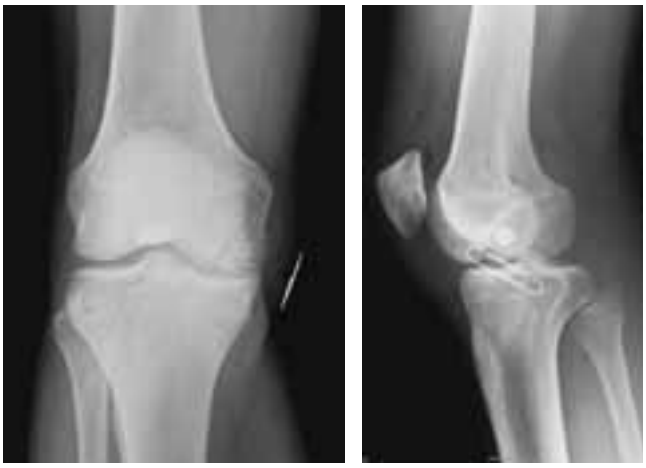
With the increasing rate of gun violence, gunshot injuries to the musculoskeletal system are becoming an increasing concern to the treating physician. A standardized treatment for intra-articular low-velocity missile injuries is lacking. Simpson et al. in a review of the literature concluded that intra-articular injuries require a minimum of 24 to 48 hours of an intravenous first-generation cephalosporin.[13]

There is no literature or protocol regarding the surgical treatment. There are only recommendations by many authors to irrigate and debride the joint with specific attention to removing all retained bullet fragments in the joint cavity. Timing of operative treatment still remains unclear. Many consider missile injuries to joints a surgical emergency while others believe that when no gross contamination of the wounds is present, the surgical irrigation and debridement can be addressed in a non-emergent manner.

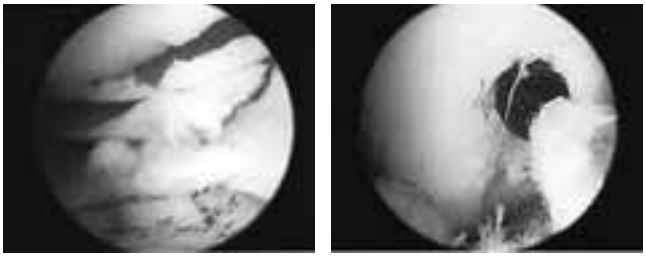
In a retrospective study by Tornetta et al., arthroscopic evaluation of the knee following a gunshot injury was effective in identifying additional intra-articular soft-tissue injuries (chondral and meniscal), and debris (bone, bullet fragments, skin, and clothing) not evident on radiographs. In addition, he was able to conclude arthroscopy aided in the removal of bullet fragments and surgical irrigation and debridement.[4] No discussion of timing to surgical intervention was included. Cho et al. in case report treated an intra-articular gunshot injury of the knee with arthroscopy including tractoscopy of the bullet tract.[3] By including tractoscopy, they concluded that a thorough debridement can be carried out ensuring removal of all particulate debris not only within the joint but retained within the bullet tract.

Our experience with low-velocity gunshot injuries to the knee demonstrates that emergent irrigation and debridement of the joint may not reduce the risk of infection. Those treated surgically after 6 hours from the time of injury and those treated non-operatively had no incidence of septic arthritis or wound infection. Antibiotic prophylaxis administered within 6 hours of injury may be an important factor to reduce joint sepsis with low-velocity gunshot injuries to the knee. A larger series may confirm such theory.

The results of this study are however limited by the inherent weaknesses of a retrospective, case series study design. Specifically, the results are subject to distortion by the interplay of confounding factors and bias, due to the absence of control groups and randomization. The presence or absence of joint sepsis in the subjects was based on the subjective self report of symptoms without the use of a validated survey instrument. It also relied on subjective physical exam data recorded by multiple clinicians without the use of a validated or standardized physical exam methodology. Objective lab data was not used as diagnostic criteria for the determination of joint sepsis.



A 37-year-old male with a bullet within the joint. The patient underwent an arthroscopic debridement, bullet extraction, internal fixation of an osteochondral fracture of the medial femoral condyle and medial meniscus repair.



Arthroscopic views demonstrating the osteochondral fracture and medial meniscus tear. The second image shows stabilization of the osteochondral fracture with bioabsorbable pins.

The small number of study subjects and the poor post-treatment follow-up were other significant factors that introduce bias into the study. Of the 22 subjects, 12 did not attend their initial follow-up appointment and 2 more subjects were lost to follow-up after this initial appointment. This high number of subjects lost to follow-up may have significantly affected the study's internal validity because this group's decision not to continue medical care is unknown.

Future directions include identifying more cases and expanding the study cohort; adding validated instruments, like the Oxford Knee Score and Tegner Activity Level Scale, in order to evaluate the post-treatment recovery; comparing various antibiotic protocols with and without irrigation, surgical debridement, and arthroscopic knee examination in order to help further elucidate an evidence-based treatment protocol that is most effective for the management of intra-articular knee gunshot injuries.

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Clinical Research

Comparison of Preliminary External Fixation Versus Ulnar Osteotomy with External Fixation for the Treatment of Radial Dysplasia Using the Resolved (3-D) Total Angle of Deformity

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Introduction

Radial dysplasia is a deformity of the upper extremity characterized by hypoplasia or aplasia of the radius.[1–5] Neither the assessment nor the treatment of radial dysplasia has been well established.

Radiographic measurements of radial dysplasia have included the wrist angle (WA), ulnar bow (UB) and total angle (TA), which is the sum of the wrist angle and ulnar bow. All of these angles have been defined in only one plane and do not take into account the 3 dimensional nature of the deformity.[12–23]

Treatment of radial dysplasia historically has been associated with a high complication and recurrence rate. New techniques including the use of external fixation have been employed in an attempt to re-establishing the length and alignment of radial dysplasia.

The purpose of this study is to: 1) define the 3 dimensional deformity in radial dysplasia by measurement of the resolved total angle (RTA) of deformity; and 2) use this measurement to compare preliminary external fixation and centralization to ulnar osteotomy with external fixation for the treatment of radial dysplasia.

Methods and Materials

This is a retrospective review of patients with radial dysplasia. IRB approval was obtained for this study.

Defining the 3-D Deformity

To assess the 3-D deformity in radial dysplasia, the radiographs of 15 patients (23 limbs) with radial dysplasia were measured. 11 patients had undergone previous centralization, with recurrence. The group was composed of 8 females and 7 males with a mean age of 9.6 years.

The 3-D deformity was measured by the resolved total angle of deformity (RTA). This angle was determined by first measuring the total angle of deformity on orthogonal (anteroposterior and lateral) radiographs, then resolving a maximal total angle of deformity by using a nomogram. The resolved angles defined the magnitude and the plane of the maximum deformity of the radial dysplasia for each limb.

Specifically, the measurements were made measuring the angles between three lines drawn on the anterior-posterior (AP) and lateral radiographs (Fig. 1). The first line was made parallel to proximal ulna. It was determined by a point at the center of the ulna at the level of the olecranon fossa and another point 1 cm distal to the coronoid fossa. The second line was drawn parallel to the distal ulna. This parallel line was determined by two points at the center of the ulna, 1 cm and 2 cm respectively, proximal to the distal physis. The angulation between these two lines was the ulnar bow. A third line was drawn through the third ray in a parallel plane. The angle between the line through the third ray and the distal ulna was the wrist angle. The total angulation of the deformity was the sum of the wrist angle and the ulnar bow. Identical measurements were made on the AP and lateral radiographs. An average angle of deformity was determined for the ulnar bow, wrist angle, and total angle for the measurements in each plane of the twenty-three samples. Lastly, the average angular deformities of ulnar bow and the wrist angle in the two planes were resolved using a nomogram as described by Bar and Breitfuss.[24]

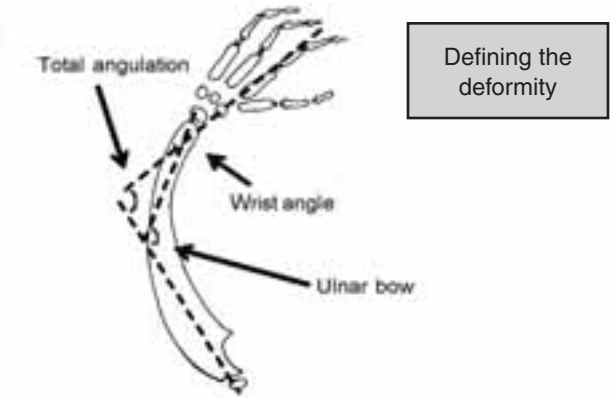

Figure 1. Demonstrates the angles measured on the AP and Lateral x-rays for each patient (lateral radiograph depicted)

We showed that the total resolved angle of deformity (TRA) best describes the maximum clinical deformity and therefore we chose to utilize this measurement to evaluate the results of our surgical treatment.

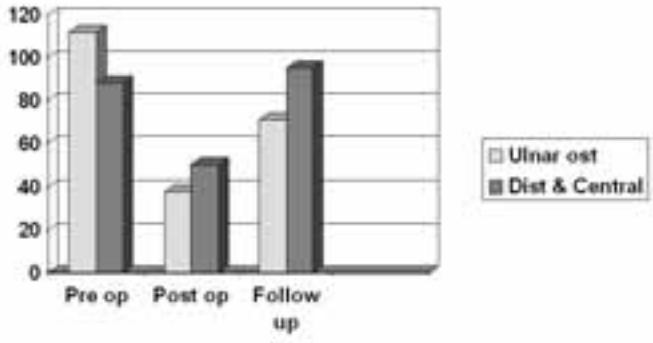

Figure 2. Total Resolved Angle for Ulnar Osteotomy vs. Distraction & Centralization

Comparison of Preliminary External Fixation Versus Ulnar Osteotomy with External Fixation for the Treatment of Radial Dysplasia

Eleven patients (16 limbs) underwent surgical treatment for the correction of their deformity. Six patients (8 limbs) underwent correction of their angular deformity with preliminary external fixator distraction followed by centralization. There were 3 females and 3 males, and 4 had undergone previous centralization.

Five patients (6 limbs) underwent ulnar osteotomy with external fixation. There were 2 females and 3 males, and 4 had undergone previous centralizations. For all patients, we made preoperative and postoperative measurements of the wrist angle, ulnar bow, and total angle in two planes. These angles were used to determine the total resolved angle and plane of maximum deformity for each sample preoperatively, postoperatively and at follow up. Then, all of the values in each treatment group were used to calculate a mean.

Results

Defining the 3-D Deformity

The magnitude of the deformity is shown in Table 1. Using a nomogram for two-plane deformity with the sagittal angle on the x-axis and the coronal angle on the y-axis, the resolved deformity for wrist angle was 55°, ulnar bow was 31°, and the total angulation was 85° in a plane 51° from the AP plane.

Table 1. Average Resolved Three-Dimensional Deformity for All Subjects

	Wrist	Ulna	Total
Resolved angle	55°	31°	85°

*51° from AP plane

Evaluation of Correction (Centralization Versus Osteotomy)

The mean age for the group that underwent distraction and centralization was 8.8 years, with a mean follow up of 40 months (range 26–59). Initial total resolved angle (TRA) was 112° for this group, which improved to 38°, with a worsening over time to 71°. There was a decrease in the total resolved angles of 41° at follow up, 67% of which was correction of the wrist angle.

The mean age for the group that underwent ulnar osteotomy with overcorrection of the deformity through the ulna, was 10.1 years and the mean follow up was 43 months (range 23–59 months). Initial total resolved angle (TRA) was 88° for this group, which improved to 50°, with a worsening over time to 95°. There was therefore an *increase* in the total resolved angle of 7° at follow up, with an increase in both the wrist angle of 15°, and the ulnar angle of 5°.

The total angle, which is the summation of the wrist and ulnar bows, is less than the sum of each angle because the direction of the angulation (wrist versus ulnar bow) was opposite; this reflects the intentional over correction that was incorporated into the ulnar osteotomy, in an effort to produce overall correction.

The ulna was lengthened a mean of 38 mm for those undergoing ulnar osteotomy. There was little change in the plane of deformity with either procedure. Table 2 illustrates the average of the resolved angles preoperatively and postoperatively.

Table 2. Average Pre- and Post-op Resolved Angles Comparing Distraction and Centralization to Ulnar Osteotomies

	Distraction and Centralization		Ulnar Osteotomy	
Resolved angle	**Pre op**	**Post op**	**Pre op**	**Post op**
Ulnar bow	42°	33°	32°	37°
Wrist angle	76°	50°	65°	80°
Total angle	112°*	71°*	88°†	95°†

Note that the total angles does not equal the sum of the ulnar and wrist angles due to compensating deformities (ie the wrist and the ulnar bows may be in opposite directions).

*$p < 0.05$ †NS

Discussion

Radial longitudinal deficiency is a developmental process that involves the preaxial structures of the upper extremity. The areas that have gained the most focus are the structures of the forearm, wrist, and hand. Bayne and Klug classified the morphologic appearance by the radiographic appearance of the radius.[6] Their classification of the radius was two-dimensional and consisted of 4 types: short distally, hypoplastic, partially absent, and completely absent. James modified the classification to include the clinical and radiographic appearance of thumb hypoplasia and carpal anomalies.[7] However, neither system integrated a quantitative value to describe the three-dimensional deformity of this disorder.

In radial longitudinal deficiency, the surgical objective is correction of the radial deviation of the wrist, balance of the

wrist on the forearm, growth of the forearm, and maintenance of wrist and finger motion.[22] However, this has proved difficult in light of the static multiple skeletal abnormalities and dynamic biomechanical forces.[2–5] The initial treatment of centralization damaged the distal ulnar epiphysis leading to increased shortening.[2, 8] Since then, surgical refinements have included resection of the carpi, use of tendon transfers, radialization, microsurgical transfers and bone distraction techniques.[3, 4, 9–11]

During this surgical evolution, studies have shown a significant improvement in the post-operative angulation.[3, 8, 9, 11, 23] However, the most common problem following centralization is recurrence.[3, 9, 11, 22, 23] Some have found this complication less significant than others.[3, 9, 11] Risk factors for recurrence have included young age at initial surgery, preoperative degree of angulation, intraoperative angular correction, and radially increased forearm-wrist angle postoperatively.[22, 23] However, comparison of the different studies to is difficult because of the variety of surgical techniques and the variations in measurements.

In this study, there were two objectives. The first was to evaluate preoperative x-rays, use the angles on orthogonal views to define the deformity in a three-dimensional manner, and determine the angles with the most deformity. From these measurements, we found the primary deformity was wrist angulation (55°), with a resolved angle that was 65% of the total resolved angle (85°). In addition, the plane of maximum deformity was approximately midway between flexion and radial deviation demonstrating a three-dimensional skeletal morphologic problem in radial dysplasia much larger than described from either of the orthogonal x-rays.

The second objective in our study was to evaluate the treatment results. In the separate treatment groups, total correction was greater for those who underwent preliminary external fixator distraction and centralization (total mean decrease of 41°) than ulnar osteotomy treatment group (total mean increase of 7°). This indicates in our study after longer term follow up, only preliminary distraction and centralization will result in a sustained correction. This differs from our early post operative results (mean of 17 months after surgery) in which the overall correction (both groups) was 48°, with a total correction of 79° for the distraction and centralization, and 38° for the ulnar osteotomy and gradual (over) correction and lengthening. This supports the finding that early good results tend to deteriorate with longer term follow up.[3, 9, 11, 22, 23] Most of the deformity tends to recur in the wrist. Surprisingly the ulnar deformity was measured to be greater in the ulnar osteotomy group post operatively. When we reviewed the films this appeared to be true for two reasons: 1) some intentional over correction was incorporated into the osteotomy to compensate for the wrist deviation. This clinically rarely was successful because the wrist would only deform more with time, negating any correction with the ulna. Additionally in one case, there appeared to be late ulnar deformity, this was not in the area of the previous osteotomy, but appeared to be plastic deformation at the distal aspect of the ulna.

This difference in the total resolved preoperative and postoperative angles for distraction and centralization are similar to those previously published despite the different methods of quantitative assessment of the results. In reviewing the literature for the various treatments of radial dysplasia, there have been a limited number of studies that have quantitatively analyzed the results of the angular correction.[3, 9, 11, 12, 21–23] Lamb used centralization and a change in radial deviation of 78° to 22°.[3] However, he does not describe how he measured the angle. Manske et al. measured hand-forearm angle from the AP x-ray after centralization and gained an average correction of 31°.[11] Another study by Geck et al. found a correction of the hand-forearm angle of 50° and a correction of the ulnar bow of 18°.[23] Most recently, Kawabata et al. used the Ilizarov method after centralization to correct residual deformity.[21] They measured the sum of ulnar bowing and radial deviation of the hand at the wrist with a measured change of 39°. Lastly, Vilkki used distraction and microvascular epiphysis transfer for radial clubhand and gained an average of 20° for the hand-forearm angle and 10° for the ulnar bow.[12]

From this study, we feel that the treatment of recurrent radial dysplasia should consist of a careful quantitative radiographic and a qualitative clinical examination of the upper extremity deformity. The radiographic exam should include AP and lateral x-rays of the extremity, which are evaluated for the wrist angle, ulnar bow, and total angulation. The angles are resolved to determine the plane of the deformity and each component's angular contribution to the total deformity. These quantitative measurements determine whether the deformity is primarily from the wrist or from the ulna and will be combined with the physical exam to direct a treatment plan. During the clinical exam, the entire extremity should be evaluated for deformity with special attention to the elbow, the length of the ulna, the ulnar deformity, and the stability of the wrist. If the elbow is limited to less than 90° of flexion, then the deformity should not be lengthened or corrected. Next, the wrist stability is assessed to aid in the direction of the treatment. If the wrist is stable, with little deformity, then the patient may be lengthened through an ulnar corticotomy, but little long term angular correction can be expected. Angular correction should be performed through the wrist with preliminary distraction followed by centralization (or even radialization), especially if there is wrist instability. Ulnar lengthening or angular correction of the ulna (if significant) should be performed at a later date and great care must be taken to insure that the wrist deformity does not worsen during lengthening.

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Clinical Research

Lateral Joint Findings on MR Imaging of the Acutely Injured ACL Deficient Knee

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Abstract

Purpose: Anterior cruciate ligament injuries are often associated with both meniscal injuries and bone bruises. The purpose of this study was to examine the ability of lateral compartment findings on MRI to predict lateral meniscal tears in the acutely injured knee.

Methods: A retrospective review of ACL injuries with or without meniscal tears was performed. Patients were included in the study if they had no other ligamentous injuries and had MR scans within two weeks of the injury. Thirty-seven patients were identified, their MRI studies were reviewed and evaluated for the size of bone bruise and for the grade of lateral meniscal signal change. Point biserial correlation was performed to determine if the size of the bone bruise was correlated positively or negatively with the presence of lateral meniscus tears. Also calculated was the sensitivity of MR imaging in diagnosing lateral meniscus tears in the acutely injured knee.

Results: There was neither a positive nor negative correlation between bone bruise size and lateral meniscal injury. The sensitivity of the MRI in evaluating the lateral meniscus in acutely injured knees was lower than previous studies have reported. We found a sensitivity of only 19% for grade 3 signal changes.

Conclusion: While a bone bruise may be the result of an anterior translation/rotation mechanism and may indicate a region of articular cartilage injury, its size has no relationship to the presence or absence of a lateral meniscal tear.

Introduction

More than 95,000 anterior cruciate ligament (ACL) injuries occur each year in the United States.[1] MR imaging of these knees allows the physician to confirm the diagnosis of an ACL deficient knee and evaluate the patient for associated meniscal tears. Detection of associated injuries is important for the surgeon to determine indications for surgery, perform pre-operatively planning, and predict the length and type of post-operative rehabilitation program.

The incidence of meniscal injury associated with acute ACL injuries has been reported to be between 45–77%, with lateral tears being more common.[2–5] The ability of MRI to detect isolated meniscal injuries is generally excellent, with sensitivities of 97% for the medial meniscus and 94% for the lateral meniscus.[6] However, while most studies report on isolated meniscal injuries, none that were reviewed reported on the ability to correctly identify a torn meniscus within two weeks of injury. De Semet et al. studied MRI sensitivity for detecting meniscal injuries with concurrent ACL injury and found disappointing results. The sensitivity for detection of meniscal tears in the presence of ACL injury decreased to 88% for the medial side and 69% for the lateral side.[6]

Another frequent MRI finding in the ACL injured knee is the presence of a bone bruise. These often occult subchondral injuries have a reported association in 48–96% of ACL tears.[2, 7, 8] Most commonly they occur on the lateral tibial plateau and lateral femoral condyle.[9] Although their significance is not fully understood, they are thought to represent trauma to the joint surfaces and are associated with deterioration of articular cartilage.[9, 10]

It has been one of the senior authors' experience (RAM) that there are often large tears of the lateral meniscus associated with ACL injuries despite the absence of obvious meniscal injury demonstrated on MRI. Since a chief function of the meniscus is to protect the articular surface, we sought to determine if there was a relationship between the size of the lateral bone bruise and the occurrence of a lateral meniscal injury. Furthermore, we calculated the MRI sensitivity in detecting lateral meniscus lesions in knees with acute ACL disruption.

Methods

A retrospective review of patients who had sustained ACL injuries was performed over a one-year period. The patients who were successfully contacted on the phone were asked to bring their MRI studies for evaluation. Charts and MRI studies were reviewed. Patients with acute, isolated ACL ruptures with no history of a previous knee injury, and MRI studies that were performed within two weeks of the injury were included. Exclusion criteria included chronic ACL injury,

previous knee injury, delayed MRI, and multi-ligamentous injuries.

The MRI studies were evaluated for meniscal injury and size of bone bruise by a single sports medicine surgeon (RAM). The meniscal signal changes were graded as 0, 1, 2, and 3. Grade 0 was assigned if there was no signal change (normal meniscus signal). Grade 1 was assigned if the meniscus was contused with an amorphous interstitial signal. Grade 2 was assigned if the signal change was linear but did not connect to the surface. Grade 3 was assigned if the signal change was linear and connected to the surface of the meniscus. These results were then compared to the operative findings to determine MRI sensitivity.

To calculate the size of the bone bruise, easily identifiable and reproducible landmarks were chosen to define an area around the lateral tibial plateau and the lateral femoral condyle. On the saggital views, the most proximal portion of the posterior projection of the condyle to the articular surface was used for the femoral side, and the tibial tubercle to the articular surface was used on the tibial side. On the coronal views, the epiphyseal scar to the articular surface was used for the femoral side, and the metaphyseal-diaphyseal junction to the articular surface was used on the tibial side. The size of the bone bruise was calculated as a percent of the total size of the landmarks defined above; thereby avoiding the need to account for magnification errors. This calculation was performed for both the saggital and coronal cuts and then multiplied to calculate the relative volume of the joint that was affected by the bone bruise. For example, if the bone bruise occupied ½ of the defined femoral region on the saggital view and ½ of the defined femoral region on the coronal view, there was ½ x ½, or ¼ (25%) volume involvement of the femoral condyle.

An intra-observer reliability test was also performed by having the same surgeon re-evaluate the MRI studies for both bone bruise size and meniscal tear grade.

Results

A total of 37 patients met the inclusion criteria. Twenty-eight (77%) had a detectable bone bruise on MRI. The average bruise size calculated for those with a lateral meniscal tear was 17%, and the average size for those patients without a lateral meniscal tear was 13%. Point biserial correlation was performed to determine if a relationship existed between bone bruise size and the presence of lateral meniscal tear. The correlation value r, calculated for the tibia was 0.03 and that for the femur was 0.2, indicating that there was neither a positive nor negative correlation between the two. In other words, the size of the bone bruise had no relationship to the presence of a lateral meniscal injury.

Review of the arthroscopy reports reveals 16 (43%) lateral tears, 7 (19%) combined medial and lateral tears, 6 (16%) medial tears, and 8 (22%) patients with no meniscal tears. With respect to the sensitivity of MRI in detecting lateral meniscal tears, a sensitivity of 61% was obtained when using signal grades one through three. However, this sensitivity was reduced to 19% when using scans demonstrating the more clinically relevant grade three signal.

The intra-observer reliability was 94% for categorizing meniscal signals, and 93% for calculating bone bruise areas.

Discussion

Bone bruises and lateral meniscal tears are common findings on MRI in patients with ACL injuries. Bone bruises have a reported 48–96% association with acute ACL injury.[2, 7, 8] They frequently affect the posterolateral plateau and central third of the lateral femoral condyle as a result of anterior translation and rotation of the tibia on the femur.[9] While the exact natural history of these bruises is not known, some studies have shown gross and histological changes that occur in the region of the bruise, which persist even into long term follow-up.[9, 10] However, their incidence on MRI has been reported to decreases with time.[2]

Since one of the functions of the meniscus is to protect and cushion the articular surface, we wished to determine if a larger bone bruise could predict concomitant lateral meniscal lesions. We sought to answer the following question: Would the presence of a large bone bruise indicate failure of the meniscus leading to increased forces on the articular surface and therefore a larger subchondral zone of injury or would a small bone bruise indicate that the meniscus absorbed a significant force, protecting the joint surface, but sustaining a tear in the process? Also, is the size of the bone bruise merely a manifestation of the degree of energy of the injury, with higher energy injuries expected to demonstrate more frequent lateral meniscal lesions? Previous investigators have found no relationship between location of the bruise and location of meniscal tear.[2, 11] However, we are unaware of any study that attempted to quantify the size of the bruise and relate it to the presence of a meniscal injury. Our results show that there does not seem to be a relationship between the size of the bone bruise and the presence of a lateral meniscal tear.

A review of the literature on MRI sensitivities in detecting meniscal injuries revealed several studies on isolated tears,[10, 12–14] and only one study that looked at the sensitivity of detecting a meniscal tear in knees with an ACL injury. In the latter study, the sensitivity of MR scanning was significantly decreased for lateral tears, going from 94% for ACL competent knees to 69% for knees with ACL deficiency.[6] Our study had a sensitivity of 61% for all three grades of meniscal tears and 19% for grade three tears alone. One possible reason the MRI scans had lower sensitivity may be due to the fact that only studies performed within 2 weeks of the acute injury were included. The existing literature is not clear on the relationship between the timing of the MRI study and its sensitivity. The studies that do make mention of

time after injury consider scans taken within 6 weeks of injury as acute scans. In our study, we have found that during the initial two-week acute period, the contusion of the meniscus may make detection of a grade 3 signal changes difficult.

Due to strict inclusion criteria, this study represents a small sample size. No attempts were made to characterize the meniscal injury; rather, we compared meniscal signal grading to surgical findings.

Conclusion

This study has demonstrated that while a bone bruise may be the result of an anterior translation/rotation and may indicate a region of articular cartilage injury, its size has no relationship to the presence or absence of a lateral meniscal tear. Furthermore, the ability of MRI scans to detect grade 3 signal changes in lateral menisci in the first two weeks after an acute injury is less than has been reported in the literature.

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Clinical Research

A Survey Assessing Orthopedic Patients' Utilization of Chiropractors

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Abstract

This study reports the findings of a survey that assesses orthopedic patients' prior use of chiropractic services. A total of 637 patients completed the survey, with forty-seven (7.4%) reporting having visited a chiropractor for their presenting symptoms. The chiropractic patients consisted of 27 males (57.4%) and 20 females (42.6%). They had made an average of 10.9 visits to their chiropractors and reported an average 3.3 on a scale of 5 measuring their satisfaction with the chiropractic treatment. Of the 47 patients, 29 patients (61.7%) sought chiropractic help for back and neck disorders for which chiropractors are traditionally trained. Fourteen of these patients (29.7%) were treated for other symptoms involving the upper and/or lower extremities, which chiropractors are not trained to treat. Eighteen patients (38.3%) were evaluated and treated by chiropractors for non-spinal symptoms, i.e., upper and lower extremity problems and other non-orthopedic complaints, such as allergies and health maintenance issues.

Introduction

The growing emphases on market-driven health care and consumer choice have expanded the role of complementary and alternative treatment in the health-care system. The past decade has seen passage of legislation expanding the scope of alternative treatment. However, the extent of the consumer's utilization and the appropriateness of the services these therapists provide have not been fully studied. Chiropractic is the largest alternative health profession in the United States with well over 45,000 chiropractors practicing today.[1] Chiropractors are licensed throughout the United States and subsidized by governmental and private health insurance agencies.[1] However, the discipline of chiropractic has often been labeled as unscientific: due, in part, to the fact that the majority of chiropractors have supplemented their spinal manipulations with physical and pharmacological therapies, and other unstudied treatments.[2] Moreover, some chiropractors have expanded the areas they treat beyond manipulating the lower back to include the neck, head, upper and lower extremities claiming that virtually any illness is caused directly, or indirectly, by derangement in homeostasis that results from vertebral malalignment, also called "subluxation".[1] Some have promoted manipulations for infectious diseases, and claim their efficacy in preventing such diseases such as cancer. They often encourage patients to maintain a regimen of regular adjustments to maintain optimal health.

This study reports the findings of a survey employed to assess the extent and appropriateness of orthopedic patients' use of chiropractic services for orthopedic symptoms prior to presenting as patients in our orthopaedic department.

Materials and Methods

All new patients presenting to the outpatient orthopedic facility at our institution for a 12 week period were asked to fill out an extensive survey. A total of 637 patients agreed to participate in the study. The survey contained the purpose of the study, which was to assess the extent of our orthopedic patients' utilization of alternative therapies. Patients were asked to report if they had received treatment by chiropractors for their current symptoms prior to presenting to our department. They were also asked to enter their age, gender, educational level, type of health insurance, and the anatomic location of their symptoms. Moreover, the patients were asked to report the number of visits they made to their chiropractors, and to rate their quality of treatment on the following scale: 1=poor, 2=below average, 3=average, 4=above average, and 5=excellent. Two pilot studies were conducted to refine the survey's questions.

All completed survey results were entered into Microsoft Excel 2000 for Windows (Microsoft Corp., Seattle, WA). Patients were divided into four groups with respect to their ages. The first group consisted of all the patients under 18 years old. The second group contained the patients with ages between 18 and 29. The third group contained the patients with ages between 30 and 49 and the last group consisted of the patients ages 50 years and older.

Under the education category, patients were divided into three groups. The first group consisted of all patients under 18 who are still in school. The second group consisted of all

adult patients who achieved a maximum of a high school degree. The third group contained the patients who had a college or graduate degree.

As for health insurance, the data were divided into 6 groups, which included Blue Cross/Blue Shield, HMOs, commercial insurance companies, Medicare, medical assistance agencies, and uninsured.

For anatomical locations of symptoms, the patients selected from the following areas: shoulder, arm/elbow, wrist/hand, neck, back, pelvis/hip, leg, knee, ankle/foot, other. After the surveys were entered, the anatomical locations were grouped into upper extremity (shoulder, arm/elbow, wrist/hand), lower extremity (pelvis/hip, leg, knee, ankle/foot), and spine (neck and back). The data were analyzed in Excel using the t-test, and chi-square functions.

Results

A total of 637 patients completed the survey. The sample surveyed consisted of 354 males (55.6%) and 278 females (43.6%). The average age of the patients was 36.8 years (range 1–87). There were 127 patients (19.9%) under the age of 18 years. 103 patients (16.2%) were in the age range of 18–29 years, 255 patients (40.0%) were between 30 and 49 years old, and 150 patients (23.6%) were over the age of 50.

With respect to their educational level, 127 patients (19.9%) were under 18 years old and were therefore still in school. A total of 267 patients (41.9%) obtained a maximum of a high school degree. 153 patients (24.0%) achieved a college or higher degree of education.

Under health insurance, there were a total of 109 Blue Cross/Blue Shield patients (17.1%). 265 patients (41.6%) had HMO insurance, 46 patients (7.2%) had commercial insurance plans, 32 patients (5.0%) had medicare, 26 patients (4.1%) had medical assistance plans, and 18 patients (2.8%) were uninsured.

Forty-seven patients (7.4%) of those surveyed reported visiting a chiropractor for their symptoms before presenting to our department. These patients consisted of 27 males (57.4%) and 20 females (42.6%). They had an average age of 39.5 years (range 8–76). There were 5 patients (10.6%) under the age of 18 years, 7 patients (14.9%) between 18 and 29 years old, 23 patients (48.9%) between 30 and 49 years old, and 12 patients (25.5%) ages 50 and older.

With respect to education, 5 of the 47 patients (10.6%) who visited chiropractors were under the age of 18 and were still in school, 20 patients (42.6%) were adults who completed a maximum of high school, and 19 patients (40.4%) finished college or beyond. As for health insurance, 10 patients (21.3%) had Blue Cross/Blue Shield, 17 patients (36.2%) had HMOs, 9 patients (19.1%) had commercial insurance plans, 1 patient (2.1%) was on medicare, 2 patients (4.3%) were on medical assistance, and 1 patient (2.1%) was uninsured (Table 1).

Table 1. Summary of Survey Results

	General Population	Chiropractic Patients	
Total Patients	637	47 (7.4%)	
Males	354 (55.6%)	27 (57.4%)	
Females	278 (43.6%)	20 (42.6%)	
Average Age	36.8 (range 1–87)	39.5 (range 8–76)	
Age Groups			
Under 18 y.o.	127 (19.9%)	5 (10.6%)	
18-29 y.o.	103 (16.2%)	7 (14.9%)	
30-49 y.o.	255 (40.0%)	23 (48.9%)	
Over 50 y.o.	150 (23.6%)	12 (25.5%)	
Educational Level			
Under 18 y.o. (in school)	127 (19.9%)	5 (10.6%)	
High School	267 (41.9%)	20 (42.6%)	
College and above	153 (24.0%)	19 (40.4%)	(p=0.02)
Health Insurance			
Blue Cross/Blue Shield	109 (17.1%)	10 (21.3%)	
HMO	265 (41.6%)	17 (36.2%)	
Commercial	46 (7.2%)	9 (19.1%)	(p=0.002)
Medicare	32 (5.0%)	1 (2.1%)	
Medical assistance	26 (4.1%)	2 (4.3%)	
Uninsured	18 (2.8%)	1 (2.1%)	

The patients who visited chiropractors made an average of 10.9 visits before they presented as patients to our institution. Their satisfaction with the treatment they received was 3.3 on average, on a scale of 1–5.

Of the 47 patients who reported visiting chiropractors, 29 patients (61.7%) had symptoms that included the spine (back and neck). Two of those patients (4.3%) had a combination of upper extremity, lower extremity and spine problems. Four patients (8.5%) had both upper extremity and spine problems. Eight patients (17.0%) had both lower extremity and spine problems. Only 15 patients (31.9%) had only spinal complaints. The above 29 patients who had a valid spinal complaint made an average of 13.2 visits to their chiropractors and scored 3.4 on the 1–5 satisfaction scale.

On the other hand, there were 18 patients (38.3%) whose symptoms did not include the spine. Of those patients, two patients (4.3%) had a combination of upper and lower extremity complaints. Eight patients (17.0%) had only upper extremity complaints, 7 patients (14.9%) had only lower extremity complaints. One patient (2.1%) reported he visited his chiropractor for "maintenance and allergy relief." This group of patients made an average of 7.9 visits to their chiropractors and scored 3.1 on the 1–5 satisfaction scale. There were no statistically significant differences with respect to either the number of visits or patient satisfaction between the patients who had spinal complaints and those who did not.

Utilizing the chi-square test, the only statistically significant findings found compared to what is expected of the general population were in the following two groups. First, a higher ratio of patients (19.6%) with commercial insurance visited chiropractors than expected in the general population (p=0.002). Similarly, a higher ratio of patients (12.3%) who

have a college or higher degree visited chiropractors than expected in the general population (p=0.02). No other statistically significant deviations from the general population were found in any of the other groups based on gender, age, health insurance or education.

Discussion

Use of alternative therapies has been the focus of numerous studies. A study conducted in 1994 estimated that 10% of the U.S. population visited a professional in one of the following fields: chiropractic, relaxation techniques, massage therapy or acupuncture.[3] In our study, we have estimated that about 7.4% of our orthopedic patients visit chiropractors before they present as new patients to our office. Often, our patients report being asked by chiropractors to get numerous x-rays and other radiographic studies, frequently before every office visit. A number of patients report having been unsuccessfully treated by chiropractors for visceral or non-spinal related musculoskeletal symptoms.

Chiropractors provide most of the manipulative therapy used on patients with low back pain in the United States.[4] In their treatment of low back pain, chiropractors often employ static and motion palpation of the spine, along with some orthopedic, neurologic, and radiologic examinations.[5] Benefit is usually documented within seven visits, but 39% of patients require maintenance treatment. Spinal manipulation is not always a benign procedure. A survey of 177 neurologists reported 55 strokes, 16 myelopathies, and 30 radiculopathies following cervical manipulations by chiropractors. Most of the patients continued to have persistent neurologic deficits 3 months after the manipulation, and about 50% had severe deficits.[6] Another study reported a case of an infant with congenital torticollis who sustained quadriplegia after chiropractic manipulation.[7]

Sometimes chiropractors' limited medical knowledge may lead to serious delays in diagnosis and treatment. Turow described a number of occasions in which patient treatment was delayed because chiropractors continued to insist on the effectiveness of their spinal manipulations. In one case, the patient had low back pain, which was treated by a chiropractor for two months as spinal malalignment and hip asymmetry, and was later diagnosed by the patient's primary care physician as metastatic choriocarcinoma.[8] Another case of metastatic osteosarcoma was treated incorrectly by a chiropractor for 6 weeks before the patient was referred to see a physician.[8]

A large number of chiropractors utilize radiography for complaints which generally do not require radiographic diagnosis. A recent survey conducted in Toronto concluded that 63% of chiropractors use radiography on patients with uncomplicated low back pain lasting less than one week. Their reasons for using radiography have no support in the current literature.[9]

Despite occasional serious side effects and delays in diagnosis, spinal manipulation of low back pain is generally effective and accepted modalities by some medical practitioners especially in patients with acute, uncomplicated low back pain.[4] A recent study reported the practices of physicians in their referral to chiropractors. A total of 820 clinicians who had previously reported interest in alternative therapies were sent a survey to study their referral patterns for chiropractic care. One quarter of respondents reported never referring their patients to chiropractors because they believed physical therapy to be more effective. While unsure of the benefits their patients could get from chiropractic manipulation, half of the respondents do occasionally refer patients to chiropractors for low back pain.[10]

Although chiropractic studies have documented some potential benefit in treating acute low back pain,[4] there has not been a study in the literature that has proven chiropractic's role in upper or lower extremity symptom relief, or with other symptoms such as allergies or headaches. A recent study on episodic tension headaches found no significant difference in symptom relief between patients receiving manipulation and those in the control group.[11] There is a consensus that while chiropractic may be acceptable for low back pain and possibly neck pain, it is likely inappropriate for any other indication.[8] Unfortunately, some chiropractors continue to treat patients for otitis media, asthma, gastrointestinal disorders, enuresis, attention deficit, allergies, and some even go as far as offering health maintenance and regular checkups and preventive manipulations.[8]

Although studies have reported a general decline in the proportion of chiropractors who see patients with problems other than low back pain,[12] we have found this trend not to apply to patients with orthopedic complaints. In this study, 38.3% of patients who sought chiropractic help had inappropriate indications and symptoms for doing so, and as many as 29.7% of patients had additional symptoms of the upper and/or lower extremities for which chiropractors are neither trained, qualified, nor licensed to treat. A study by Coulter et al. reported that about 70% of patients who seek chiropractic help reported their condition to be related to the back or neck.[13] However, 13% were treated for other upper and lower extremity symptoms, and 6% were treated for non-musculoskeletal problems such as allergies, intestinal parasites, etc. The patients' satisfaction with their chiropractors in this study was 87.4 out of 100. Patients presenting with non-musculoskeletal were interestingly more satisfied with chiropractic help with a score of 90.3 out of 100.[13]

Several reasons can be proposed as to why patients seek chiropractic help. The most common[14] cited reasons are word of mouth by friends and relatives, and fear of drug side effects. Some patients prefer to have a more natural drug-free approach to treating an illness, and often appreciate the more personalized attention they get from a chiropractor. Another "psychological" theory argues that chiropractic care is appealing to the "neurotics," the "worried-well," or patients with depression who cannot find a medical doctor to validate their illness.[13] When patients' attitude about chiropractors is

studied, patients who have visited chiropractors report being more impressed and satisfied with the personal attention they receive rather than the actual treatment itself.[15] Studies have suggested that patients' education correlates positively with the increased choice of alternative medicine.[8, 13] Our study agrees with this conclusion. We have found that a larger proportion of patients with higher education seek the help of chiropractors. Another positive correlation was found in patients with commercial health insurance plans. This may be due to the fewer restrictions these commercial plans place on their members with respect to seeking alternative therapies.

It is interesting to note that our patients who sought chiropractic help scored their satisfaction at an average of 3.3 on a scale, between average and above-average, with no significant differences between the patients who had low back or neck symptoms and those who had other non-spinal symptoms. We believe this is mostly due to the attentiveness and/or placebo effect of the chiropractic treatment. In many illnesses, especially in chronic pain, placebos are often effective. The time chiropractors spend with patients and the "hands-on" approach they offer, further enhance the therapeutic relationship and potentiate the placebo effect. Thus, the psychological reaction to spinal manipulation and the patient's confidence in the chiropractor may therefore play a significant role in temporarily relieving symptoms.

This study has some limitations. The survey was given only to new orthopaedic patients. Moreover, the study was conducted in a limited geographic area with referrals from Pennsylvania, New Jersey and Delaware. Therefore, one cannot expand the conclusions made in this study to the population at large. Since this study was conducted in an orthopaedic office setting, our patient sample and their answers may be biased.

Conclusion

It is generally accepted in the medical community that chiropractic treatment for acute low back pain and possibly neck pain could be an acceptable first step. However, chiropractic treatment of other medical problems has no scientific basis. There has not been a single double-blind placebo-controlled study that shows the benefit of using spinal manipulations to heal the body of other medical problems.[16]

In this study, while only 7.4% of our patients had sought chiropractic services before presenting to our office, 38.3% of those did so for inappropriate indications, and 29.7% had additional complaints for which chiropractors are not trained to treat. While the placebo effect of chiropractic treatment may yield above average satisfaction, there are several disadvantages to chiropractic manipulation. First, patients may be placed at high risk for exposure to injury. Some chiropractors have been alleged to give uneducated general medical and drug advice. Also, the impression some chiropractors give regarding their ability to treat non-musculoskeletal illnesses and to provide preventive maintenance through spinal manipulations can lead to a delay in the patient receiving appropriate medical treatment. Chiropractors, as we have observed in our study, are treating symptoms which are not related to low back pain. This can lead to inefficient health care and sizable expenditures on treatments with no scientific basis.

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Clinical Research

Evaluation of HMO Radiographs of Orthopedic Patients

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Abstract

Radiographs provided by HMO radiology centers were compared to radiographs obtained in an in-office academic radiology department at a university hospital.

Twenty-three HMO patient cases and twenty-four non-HMO patient cases were randomly selected. Four reviewers were asked to assess the radiographic studies using four criteria: Appropriateness and Quality on a 10-point scale, and Insufficiency and Excessiveness on an absolute value scale. **Quality** was evaluated based on the brightness and contrast of the x-rays. **Insufficiency** was graded based on the number of additional views needed to make a definitive diagnosis. **Excessiveness** was graded based on the number of unnecessary views. **Appropriateness** was scored based on the observers' overall satisfaction with the x-rays provided.

In terms of actual x-ray count, the academic radiology department produced an average of 2.75 x-rays per patient, and the HMO centers produced an average of 4.48 x-rays,a difference of 1.73 x-rays ($p<0.001$). On the **Appropriateness** scale, the academic department scored 6.93/10 and the HMO centers scored 5.35/10 with a difference of 1.58 ($p<0.001$). The academic department did not differ significantly from the HMO centers with respect to **Quality,** with both groups scoring approximately 6.40/10, ($P=0.96$). **Insufficiency** with both groups being insufficient by approximately 0.40 x-rays per patient. However, while the academic radiology department produced an average of only 0.38 excessive x-rays per patient, the HMO centers produced 1.61 excessive x-rays, which amounted to a difference of 1.23 excessive x-rays per patient ($p<0.001$).

Introduction

Participation in health maintenance organizations (HMOs) has increased rapidly in the past decade, with well over 50 million people currently enrolled.[1] As a cost containing measure, HMOs provide their members with medical care by selective contracting, whereby certain medical and diagnostic centers are capitated to provide services for members at a discounted rate.[2] However, instead of health care providers competing for patients by the quality of care they provide, selective contracting has introduced the concept of price competition and, perhaps, has subjugated quality of care to achieve greater cost savings.

This selective contracting has naturally triggered a number of studies analyzing the quality of care that HMO patients receive in capitated health care facilities. We became interested in this issue from an orthopedic perspective, since no study to date has researched the quality and cost effectiveness of capitated HMO radiology centers with respect to orthopedic studies.

We compared the performance of capitated HMO radiology centers to that of an "in-office" orthopedic radiology facility in an academic institution. We sought to address the question of how capitated HMO radiology centers compare to academic institutions with respect to 1) appropriateness, 2) quality, 3) completeness, and 4) redundancy of radiographs.

Materials and Methods

Random patient cases were pulled from one outpatient academic orthopedic facility. Cases were randomly selected from charts by a non-blinded observer. Patients were divided into two categories: HMO and non-HMO patients. All HMO patients' radiographs were taken at the local capitated HMO radiology facilities. All non-HMO patients' radiographs were taken in an office academic radiology department. HMO patients were excluded from the study if their radiographs were taken at the above academic facility or a non-HMO affiliated radiology center. Non-HMO patients were excluded from the study if their radiographs were taken at an outside facility.

Forty-seven patient cases were selected, consisting of twenty-three HMO patients and twenty-four non-HMO patients (Table 1). Both groups of patients were similar in age and anatomical distribution of complaints (Table 2). Each patient's chart was read and summarized by a non-blinded observer into a presenting chief complaint and brief history in order to convey the nature of disease to the blinded reviewers.

Table 1. List of Cases, Anatomic Location of Complaint, Age, Gender and Type of Patient

1	Knee	28	M	HMO Patient
2	Hip	55	F	Non-HMO Patient
3	Knee	45	F	Non-HMO Patient
4	Shoulder	60	M	Non-HMO Patient
5	Knee	35	M	HMO Patient
6	Hip	24	M	Non-HMO Patient
7	Knee	19	M	HMO Patient
8	Knee	45	F	HMO Patient
9	Knee	66	M	HMO Patient
10	Foot	48	M	Non-HMO Patient
11	Knee	44	M	Non-HMO Patient
12	Shoulder	27	M	HMO Patient
13	Knee	51	M	Non-HMO Patient
14	Ankle	13	F	Non-HMO Patient
15	Knee	40	F	HMO Patient
16	Leg	13	F	Non-HMO Patient
17	Ankle	18	F	HMO Patient
18	Shoulder/Ankle	38	M	Non-HMO Patient
19	Knee	18	M	Non-HMO Patient
20	Low Back	15	M	Non-HMO Patient
21	Hand	42	M	HMO Patient
22	Hand	69	M	Non-HMO Patient
23	Ankle	40	M	HMO Patient
24	Elbow	49	M	HMO Patient
25	Shoulder	16	M	Non-HMO Patient
26	Upper Back/Neck	35	M	Non-HMO Patient
27	Knee	15	F	HMO Patient
28	Knee	34	M	Non-HMO Patient
29	Shoulder	36	F	HMO Patient
30	Ankle	26	M	HMO Patient
31	Fingers	15	F	Non-HMO Patient
32	Ankle	41	F	HMO Patient
33	Low Back	16	M	HMO Patient
34	Ankle	15	F	Non-HMO Patient
35	Hand	54	M	Non-HMO Patient
36	Forearm	40	F	Non-HMO Patient
37	Low Back/Knee	47	F	HMO Patient
38	Foot	35	M	HMO Patient
39	Hand	56	F	HMO Patient
40	Fingers	54	F	Non-HMO Patient
41	Shoulder	57	F	HMO Patient
42	Knee	47	F	Non-HMO Patient
43	Hip	11	M	HMO Patient
44	Knee	19	M	Non-HMO Patient
45	Wrist	14	F	HMO Patient
46	Elbow	29	M	HMO Patient
47	Shoulder	18	M	Non-HMO Patient

Table 2. Summary of Cases

	HMO	Non-HMO
Patients	23	24
Average Age	34	35
Males	13	15
Females	10	9
Upper Extremity	8	8
Lower Extremity	13	13
Spine	1	2
Miscellaneous	1	1

In order to conceal the identity of the patients and the radiology centers where the radiographs were taken and to minimize observer bias, all x-rays were digitally scanned using a Hewlett Packard 4C scanner equipped with a transparency adapter (manufacturer: Hewlett Packard, Inc. Singapore). Image scans were saved on an Apple Power Macintosh computer (manufacturer: Apple Computer, Inc. USA). Before scanning the x-rays, sample x-rays were scanned at a number of different settings (brightness and contrast) and the most optimal setting was used to scan all the x-rays in this study. This allowed us to optimally reproduce and standardize the images. Adobe Photoshop (manufacturer: Adobe Systems, Inc. USA), an image editing software program, was utilized to mask the x-rays' nameplates and location insignia, and to crop the images down to a standard size. This was particularly important because the HMO centers and the academic radiology department use different film sizes.

The scanned x-rays were imported into a multimedia authoring software, Macromedia Director (manufacturer: Macromedia, Inc. USA), and the presenting chief complaints and histories were entered into the program as well. Three orthopedic and one musculoskeletal radiology faculty members participated in the study as blinded observers. For every case, the automated software program, without any participation from the non-blinded observer, presented the observer with the patient's history and pertinent physical exam findings along with the accompanying radiographs. The observer was able to look at all the x-rays in thumbnail (reduced size) format or enlarge every x-ray to full screen size.

The observers were asked to assess the radiographic studies using four criteria: Appropriateness, Quality, Insufficiency, and Excessiveness. **Quality** was assessed by the degree of brightness and contrast of the x-rays and the visibility of the pertinent structures. **Insufficiency** was scored based on the number of additional views the investigator needed to make reasonable radiographic assessment. **Excessiveness** was graded based on the number of unnecessary or superfluous views in relation to the presenting symptoms. **Appropriateness** was treated as a global measure, which encompassed the above three measures in addition to evaluating whether the standard views were correctly obtained, and described the blinded observers' overall satisfaction with the radiographs provided. Both Appropriateness and Quality were graded on a 10 point scale, while Insufficiency, and Excessiveness were graded on an absolute value scale, indicating the number of lacking or excessive views respectively.

HMO and non-HMO patient data were analyzed and compared using Microsoft Excel's paired t-test (Microsoft Corp., Seattle, WA). In order to assess intra-observer reproducibility, the four observers were asked to repeat their assessment of 12 cases four weeks after they first evaluated them. Moreover, observers' assessments were compared to the most senior observer's (R.A.M.) to calculate inter-observer variability. Kappa values were calculated both for intra- and inter-observer consistency.

Results

X-ray Count: The academic radiology center produced an average of 2.75 x-rays per patient, while HMO radiology centers produced an average of 4.48 x-rays. This amounted

to a significant difference of 1.73 x-rays per patient ($p<0.001$).

Appropriateness: The academic radiology center scored 6.93/10 ± 1.25, while the HMO radiology centers scored 5.35/10 ± 1.29. This amounted to a statistically significant difference of 1.58 ($p<0.001$). The intra-observer kappa values were 0.8–0.9, and the inter-observer kappa values were 0.5–0.7.

Quality: There was no statistically significant difference between the HMO radiology centers and the academic radiology center. HMO radiology centers scored 6.39/10 ± 1.26, while the academic center scored 6.41/10 ± 0.93 ($p=0.96$).

Insufficiency: There was no statistically significant difference between the two groups, since HMO radiology centers produced 0.39 ± 0.55 insufficient views, while the academic center produced 0.40 ± 0.55 insufficient views ($p=0.98$).

Interestingly, there was a significant difference between the two groups with respect to **Excessiveness.** HMO radiology centers produced 1.61 ± 1.10 excessive views, while the academic center produced 0.38 ± 0.57 excessive views. This amounted to a statistically significant difference of 1.23 excessive x-ray produced by the HMO centers per patient ($p<0.001$) (Table 3). The intra-observer kappa values were 0.8–0.9, and the inter-observer kappa values were 0.9–1.0. This reflects a high degree of reproducibility and reliability within the group of observers.

Discussion

HMO patients typically need to be examined by a "gatekeeper" primary care physician before they are referred to a specialist. For a patient with orthopedic complaints, the primary care physician may refer the patients to an orthopedist directly, in which case the orthopedist may request specific radiographs, which can only be done at an outside capitated facility. Alternatively, the primary care physician may refer the patient to a capitated radiology center to obtain radiographs first, and then refer the patient to an orthopedist. In both scenarios, the treating orthopedist has diminished control on the radiographic determination.

While requiring HMO patients to use one of the approved HMO radiology centers may be intended to be cost effective without compromising quality, our study demonstrated that this practice actually produced the opposite effect. We found that HMO-produced radiographs were less likely to be appropriate for the clinical presentation, based on the global measure of Appropriateness. This finding is illustrated by a commonly encountered set of patients in our practice, namely, patients with knee complaints who present with AP, lateral and oblique radiographs already taken. The oblique films may not be indicated in every knee complaint and are therefore considered excessive in some cases. It was, therefore, not surprising for us to find out that HMO radiology centers produce x-rays which are about 15.8% less appropriate and more excessive by 1.23 x-rays per patient than those produced in an academic department with the direct input of the orthopedic surgeon. This can partially be explained by the fact that in HMO radiology centers, a pre-determined standard examination is usually performed, as opposed to specified views requested by the orthopedist during the same patient visit. This same standard examination, we believe, is performed whether the radiographs are ordered by the primary care physician or by the orthopedist directly. We did not directly compare HMO radiographs ordered by the primary care physician vs. those ordered by the orthopedist. This distinction may have helped clarify how closely HMO radiology centers follow the radiographic orders they receive from the different referring physicians.

Our study suggests that overall, regardless of the referring physician an excessive number of radiographs are produced by the HMO radiology centers. Moreover, HMO radiographs frequently include a necessary view that is technically suboptimal. The practical result of this is that the patient needs to have the suboptimal views redone, resulting in a higher overall cost, as well as inconvenience to the patient. This is especially true when the patient is required by the HMO to return to the often remotely located capitated center for those additional views.

HMOs and their practice of selective contracting have resulted in a number of research studies evaluating HMO practices in several medical specialties. A study on coronary artery bypass graft surgeries suggested that HMOs do not channel their patients to high-quality, low-mortality hospitals.[2] Instead, it concluded that HMOs in the state of Florida were primarily concerned with reducing cost. The study further stated that HMOs did not conduct formal studies on the quality of care their providers produced, and often used subjective measures such as the reputation of the provider.

A survey conducted by Schmittdiel et al. compared the satisfaction of HMO patients assigned to a primary care physician to patients who were allowed to choose their own physician. It found that patients who chose their own personal physician were 16–20% more likely to rate their satisfaction as "excellent" or "very good" than those patients who had no choice in their physician selection.[3] Moreover, when com-

Table 3. Summary of Results

	Appropriateness	Quality	Insufficiency	Excessiveness	X-ray Count
Non-HMO Cases	6.93 ± 1.25	6.41 ± 0.93	0.4 ± 0.55	0.38 ± 0.57	2.75 ± 1.26
HMO Cases	5.35 ± 1.29	6.39 ± 1.26	0.39 ± 0.55	1.61 ± 1.10	4.48 ± 1.83
p value	< 0.001	0.96	0.98	< 0.001	< 0.001
Difference = (Non-HMO–HMO)	1.58	0.02	0.01	–1.23	–1.73

paring Medicare HMO enrollees to fee-for-service patients, Tudor et al. found that while HMO patients were more likely to be satisfied with the costs of their care, non-HMO enrollees were more likely to indicate better satisfaction with their quality of treatment.[4]

The above and other studies[5–7] have indicated that HMO cost cutting measures may be truly compromising quality and effectiveness of care and may prove ultimately to be more "costly" in the final analysis.

The performance of HMOs has been and will continue to be the subject of intense academic interest and debate. We demonstrated in this investigation that HMO-produced radiographs for orthopedic complaints have lower overall appropriateness as well as higher cost, compared to those produced in orthopedic offices under the supervision of orthopedists. Capitated x-ray facilities, thus, do not demonstrate overall effectiveness in generating cost-efficient and appropriate radiographs. Furthermore, patients may be subject to excessive radiation exposure in the capitated system.

Conclusion

With the rapidly growing market of health maintenance organizations and increased emphasis on cost cutting, there has been an ongoing public interest in monitoring and improving the quality of care. Numerous studies in different medical fields have raised questions about the quality of care delivered by HMOs.

To our knowledge, our study is the first to examine the specific issue of radiographs that HMO patients with orthopedic complaints receive compared to non-HMO patients. Our study demonstrates that local capitated HMO radiology centers often produce both inappropriate and excessive radiographs for common orthopedic afflictions, consequently compromising both the efficiency such plans purportedly uphold. Capitated arrangements further subject patients to needless radiation exposure and inconvenience.

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Clinical Research

Outcome of Arthroscopic Repair of Massive Rotator Cuff Tears

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Abstract

Purpose: To study the results of arthroscopic repair of massive full-thickness rotator cuff tears.

Methods: A retrospective review was performed of patients who underwent arthroscopic repair of massive full-thickness rotator cuff defects. Massive tears were defined with greater than or equal to 5 cm retraction. Patients were evaluated using the UCLA and Simple Shoulder Test questionnaires. The data collected were analyzed to determine the outcome in patients with an average follow up of 3 years.

Results: A total of 74 arthroscopic rotator cuff repairs were performed between March 2000 and September 2003. Of the seventeen patients who had tears defined as massive fifteen were available for evaluation. Preoperatively, the mean UCLA and SST scores were 12.7 and 4.0, respectively. Postoperatively, the mean UCLA and SST scores were 32.6 and 10.7, respectively. Fourteen of the patients reported a UCLA score of at least 30. One patient reported a score of 23. All 15 patients reported feeling "satisfied and better".

Conclusion: In this series, all of the patients reported satisfaction with the procedure. According to the UCLA score, 8 patients had good results and 6 patients had excellent results. Patients with a massive full-thickness defect of the rotator cuff tendon may be effectively treated with an arthroscopic repair with goon and excellent clinical results at an average of three years follow up.

Introduction

Arthroscopic rotator cuff repair is gaining popularity with the advent of new fixation devices, techniques and suture configurations. Arthroscopic repair provides the benefit of same day surgery, decreased postoperative pain, sparing of deltoid morbidity and quicker recovery. Massive rotator cuff tears are problematic since retracted tears are difficult to visualize with open techniques. Arthroscopic treatment of massive rotator cuff tears affords not only enhanced visualization, but also superb cuff mobilization.

Credit for describing the first rotator cuff tear has been given to J. G. Smith in 1834. In the early 1900s Codman and McLaughlin described their experience and observations of the rotator cuff and its pathology and treatment. Neer and Rockwood have contributed greatly to the management of shoulder disorders and have formed the foundation that current treatment modalities have built upon. Since the early 1990s the treatment of rotator cuff tears has progressed from open to open arthroscopic combined to arthroscopic mini open repairs to all arthroscopic. It is the goal of this paper to evaluate arthroscopic repair of massive rotator cuff tears and compare clinical outcomes with previously reported methods.

Methods and Materials

Demographics

Between March, 2000 and September, 2003, 74 arthroscopic rotator cuff repairs were performed by the senior author (JDK IV). A retrospective review of the patients' charts, operative reports, arthroscopic pictures, and implant records were collected and placed in a database.

Massive rotator cuff tears were defined as tears demonstrating retraction equal to or greater than 5 cm. Sixteen patients that met the inclusion criteria and underwent arthroscopic repair. Fourteen of the 16 patients were available for follow up.

Indications for Procedure

Patients were surgical candidates after failing conservative measures. These included activity modifications, physical therapy, NSAIDS, and corticosteroid injections. Indications for surgical intervention were unrelenting pain, weakness, and interference with activities of daily living.

Operative Technique

Patients were placed in a lateral decubitus position using a beanbag. The operative extremity was suspended using 5–10 lbs of weight with the shoulder in 45 degrees of abduction and a slightly forward flexed position. All patients had general anesthesia and a supplemental intrascalene or suprascapular nerve block. The glenohumeral joint was first injected with approximately 50cc of saline. A standard posterior portal was used to perform a diagnostic arthroscopy and the shoulder was systematically evaluated for any pathology. Rotator cuff tears were identified and intraarticular pathology (ie. labral tears) were addressed using a straight anterior portal. If appropriate the undersurface of the tear was debrided with a shaver. Attention was then turned to the

subacromial space where the cuff tear was more fully evaluated. When indicated, a standard subacromial decompression was performed using an arthroscopic Mumford procedure.

Arthroscopic repair was then carried out with limited bursectomy to allow visualization. Arthroscopic releases were performed including coracohumeral ligament and capsular release as necessary for mobilization. Tear configuration was determined and suture arrangement and type planned.

Margin convergence sutures were placed to reduce the edges of the tear without creating a dogear. Braided, nonabsorbable suture of #2 Ethibond or Fiberwire was used for the repair. The defect was closed in this manner until the insertion site for the cuff was approached. At this point, suture anchors were placed on the decorticated cuff insertion site to allow for as near an anatomic repair to the tuberosity as possible.

The quality of tissue and performance of complete vs near complete vs incomplete coverage of the humeral head was noted. The shoulder was drained and the portals closed with prolene suture.

Post-operative Rehabilitation

Patients were placed in an abduction shoulder immobilizer in the operating room which was left in place for 6 weeks. Elbow, wrist, and hand motion were encouraged and begun immediately. Passive external rotation was begun at one week. From two to six weeks postoperatively, patients were instructed to participate in passive external rotation in slight abduction exercises only. At six weeks, formal physical therapy was begun focusing on motion, with strengthening beginning at approximately 10 to 12 weeks.

Patient Evaluation

Evaluation included preoperative and postoperative UCLA Shoulder Form (Table 1) and Simple Shoulder Test Questionnaire (Table 2) as well as examination of both shoulders assessing for pathology, motion and strength. Of note, preoperative assessments that were not available were repeated in a retrospective fashion. Follow up ranged from 13 to 68 months with a mean of 3 years. Office evaluation included presence of impingement sign, ROM including forward flexion, internal rotation, and external rotation in both adduction and abduction with the scapula stabilized. Strength testing of the supraspinatus and infraspinatus were evaluated and a Whipple test was performed. Followup examinations were performed by the senior author (JDK IV) .

Results

Surgical demographics are presented in Table 3. Preoperative and postoperative evaluation was conducted using UCLA and Simple Shoulder Test forms. In addition, a physical examination in the office was performed on patients to evaluate motion, strength, and the presence or absence of impingement.

Table 1. UCLA Shoulder Assessment Score

Pain
1__Present all of the time and unbearable; strong medication frequently
2__Present all of the time but bearable: strong medication occasionally
4__None or little at rest, present during light activities; salicylates frequently
6__Present during heavy or particular activities only; salicylates occasionally
8__Occasional and slight
10__None

Function
1__Unable to use limb
2__Only light activities possible
4__Able to do light housework or most activities of daily living
6__Most housework, shopping, and driving possible; able to do hair and dress and undress, including fastening brassiere
8__Slight restrictions only; able to work above shoulder level
10__Normal activities

Active Forward Flexion
0__Less than 30°
1__30° to 45°
2__40° to 90°
3__90° to 120°
4__120° to 150°
5__150° or more

Strength of Forward Flexion
5__Grade 5 (normal)
4__Grade 4 (good)
3__Grade 3 (fair)
2__Grade 2 (poor)
1__Grade 1 (muscle contraction)
0__Grade 0 (nothing)

Satisfaction of the patient (post-op only)
0__Not Satisfied and Worse
5__Satisfied and Better

Excellent 34–35 Good 29–33 Poor <29

Table 2. Simple Shoulder Test (SST)

Circle YES or NO to whether you can or think you can do the following:

1. Is your shoulder comfortable with your arm at rest by your side?	YES	NO
2. Does your shoulder allow you to sleep comfortably?	YES	NO
3. Can you reach the small of your back to tuck in your shirt with your hand?	YES	NO
4. Can you place your hand behind your head with the elbow straight out to the side?	YES	NO
5. Can you place a coin on a shelf at the level of your shoulder without bending your elbow?	YES	NO
6. Can you lift one pound (a full pint container) to the level of your shoulder without bending your elbow?	YES	NO
7. Can you lift eight pounds (a full gallon container) to the level of your shoulder without bending your elbow?	YES	NO
8. Can you carry twenty pounds at your side with the affected extremity?	YES	NO
9. Do you think you can toss a softball underhand ten yards with the affected extremity?	YES	NO
10. Do you think you can toss a softball overhand twenty yards with the affected extremity?	YES	NO
11. Can you wash the back of your opposite shoulder with the affected extremity?	YES	NO
12. Would your shoulder allow you to work fulltime at your regular job?	YES	NO

Table 3

	L or R	Size of Tear (cm)	Tear Configuration	Fixation Type	Tissue Quality	Head Coverage
1	R	5	Reverse L	2 anchors, 4 margin convergence	Poor	Near-complete
2	L	5	Reverse L	T anchor, 4 sutures	Average	Near-complete
3	R	5	Reverse L	2 anchors, 3 sutures	Poor	Near-complete
4	R	5	Reverse L	2 anchors, 4 margin convergence	Average	Near-complete
5	L	>5	Reverse L	4 sutures, T anchor	Average	Near-complete
6	L	5	Reverse L	3 margin sutures, 1 anchor	Poor	Near-complete
7	L	5	Reverse L	2 anchors, 3 margin convergence	Good	Near-complete
8	L	>5	Reverse L	1 margin convergence, 4 sutures, 1 anchor	Poor	Complete
9	R	5	L	1 anchor, 4 margin convergence	Poor	Near-complete
10	L	5	Reverse L	4 margin convergence, 1 anchor	Poor	Near complete
11	R	>5	Reverse L	5 margin convergence, 1 anchor	Average	Near-complete
12	R	5	Retracted longitudinal	4 sutures, 1 tack	Poor	Incomplete
13	L	>5	Retracted to glenoid	4 sutures, 1 anchor	Poor	Incomplete
14	L	5	Crescent	1 anchor, 4 margin convergence	Average	Near-complete
15	L	>5	Reverse L	4 margin convergence, no anchors	Average	Complete

UCLA scores improved from an average of 13.2 to 32.6 (Table 4). Eighty-six per cent of patients had occasional to no pain and 71% of patients had no pain postoperatively. Eighty-six per cent of patients had slight restrictions to no restrictions of activities and 71% had no restrictions with regard to function. Thirteen of 14 patients had greater than 150 degrees of active forward flexion while the remaining patient was limited to 120–149 degrees. Thirteen of 14 patients had at least 4 out of 5 forward flexion strength with the remaining patient achieving 3 of 5 for motor strength. Thirteen of the 14 patients had a UCLA score that classified as a good or excellent result. The remaining patient had a score of 23. All 14 patients reported feeling "satisfied and better". The only patient to have a setback with respect to the UCLA evaluation was a patient who went from 8 to 6 in function.

Table 4

Patient #	Preop UCLA	Postop UCLA	Preop SST	Postop SST
1	10	33	9	8.5
2	9	35	0	11
3	15	33	4	12
4	2	33	0	12
5	24	30	6.5	9
6	6	30	1	11
7	14	35	4	12
8	3	23	0	6.5
9	21	32	4.5	10.5
10	5	32	0	11.5
11	12	33	3	9
12	26	35	11	12
13	17	35	11	12
14	9	35	3	12
15	17	35	2.5	12

The Simple Shoulder Test scores improved from an average of 4.2 to 10.7. The most common tasks that patients were unable to perform were lifting eight pounds to the level of the shoulder without bending the elbow and tossing a softball over-hand 20 yards with the affected extremity. Postoperatively, only 50% of patients were able to hold eight pounds at the level of the shoulder. Seventy-three per cent of patients were able to toss a softball overhead 20 yards. All patients had an improvement in SST scores except patient #1 who went from 9 to 8.5 overall. This patient lost the ability to carry 20 lbs at the side from preop to postop. Patient #5 recorded losing the ability to place the hand behind the head with the elbow out at the side.

Discussion

This study addresses massive rotator cuff tears, size 5cm or greater, that have been repaired arthroscopically. Techniques of arthroscopic releases, decompression and suture placement have improved allowing for the fixation of larger rotator cuff tears

Favorable results for these massive tears can be attributed to several reasons. First, evaluation of pathology and tear configuration is greatly enhanced with arthroscopic visualization. The ability to evaluate the rotator cuff from both the glenohumeral joint as well as the subacromial space allows superior understanding of the tear configuration. A great deal of advancement in classifying tear configuration has afforded us the ability to better repair tears anatomically. The limbs of the tear can be assessed for mobility and directly visualized.

Secondly, the ability to perform arthroscopic mobilization and arthroscopic releases is crucial to obtaining proper repair and restoring the rotator cuff force couple. Mobilization of the cuff is simpler through the arthroscope and we found a standard coracohumeral release to be easier. Although there were no formal interval slides performed, these have been

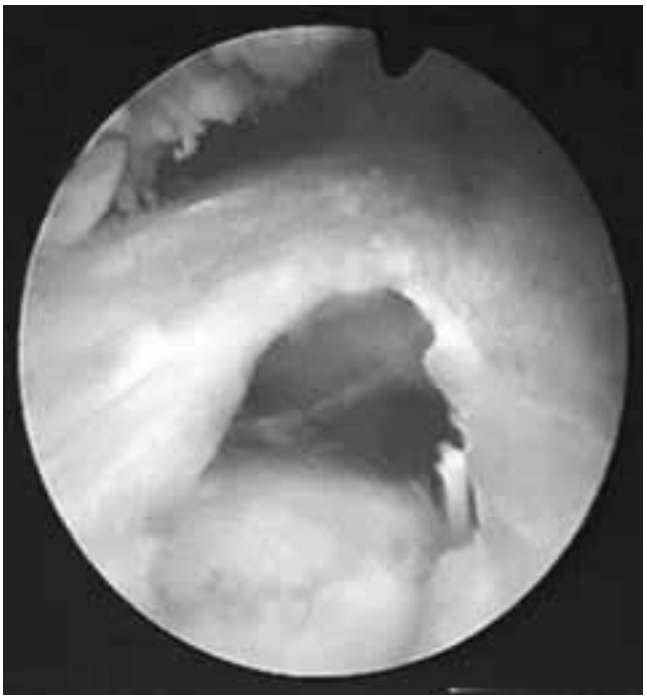

Figure 1. Rotator Cuff Tear

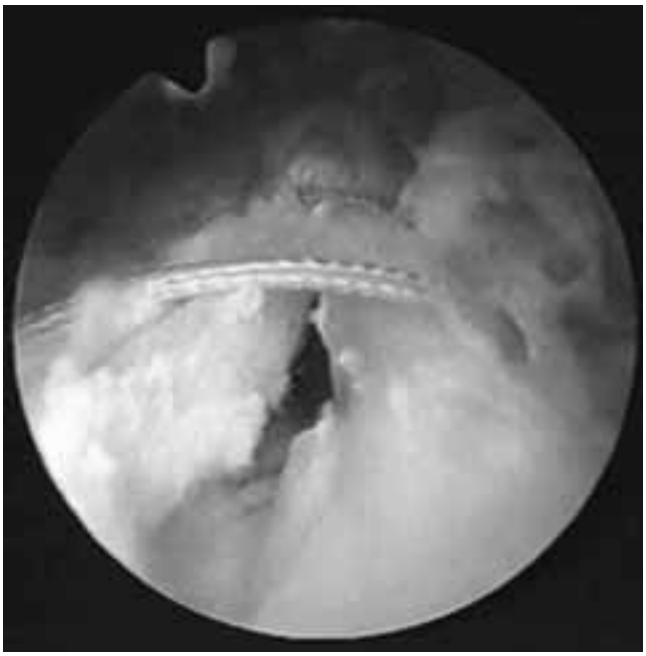

Figure 2. Margin Convergence

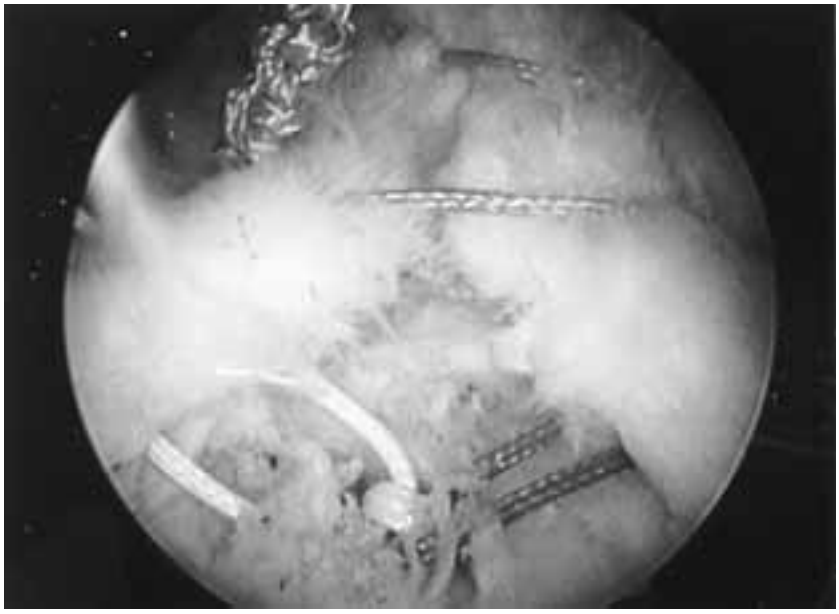

Figure 3. Margin Convergence with Double Limbed Suture Anchor

described in the literature and shown to be technically feasible and beneficial in massive, retracted cuff tears.

The advancement of suture technique and an understanding of proper suture configuration is critical to the repair. Restoring the force couple of the rotator cuff is possible using the technique of margin convergence. This technique allows the shoulder to function closer to the normal physiologic range even if the rotator cuff does not completely cover the humeral head.

Finally, the all arthroscopic technique for repair results in a less traumatic impact on the deltoid and other soft tissues and allows for a quicker recovery and postoperative rehabilitation course. Risk of deltoid injury, detachment and dysfunction are avoided. With less immobilization necessary maintaining and regaining motion and strength can be achieved more readily.

Conclusions

1. Arthroscopic repair of massive rotator cuff tears is feasible.
2. Visualization of the tear and understanding of the tear configuration is enhanced with arthroscopic techniques.
3. Mobilization of the cuff and releases are adequately performed arthroscopically and are facilitated with the arthroscope.
4. Suture techniques, specifically margin convergence to restore the force couple of the rotator cuff while avoiding the "dogear" has allowed for a more anatomic repair and better functional outcome.
5. Massive cuff repairs performed arthroscopically can achieve good and excellent results even in the face of poor tissue quality and only partial humeral head coverage.

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Basic Science Research

Molecular Cross-Talk Between Transforming Growth Factor-Beta 1 (TGF-β1) and Connective Tissue Growth Factor (CTGF/CCN2) Controls Extracellular Matrix Synthesis in Osteoblasts

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Abstract

Introduction: Connective tissue growth factor (CTGF/CCN2) is a cysteine-rich, extracellular matrix protein that acts as an anabolic growth factor to regulate osteoblast differentiation and function. CTGF acts as an effector of transforming growth factor β1 (TGF-β1) in specific cell types. In this study, we examined the role of CTGF as a mediator of TGF-β1 induced extracellular matrix (ECM) production in primary osteoblasts and characterized the CTGF cis-regulatory promoter motifs required for induction by TGF-β1 signaling.

Results: Using primary cultures, we demonstrated that TGF-β1 is a potent inducer of CTGF expression in osteoblasts, and that this induction occurred at all stages of osteoblast differentiation from the proliferative through mineralization stages. TGF-β1 treatment of osteoblasts increased the expression and synthesis of the ECM components, type I collagen and fibronectin. When CTGF specific siRNA was used to prevent TGF-β1 induction of CTGF expression, it also inhibited collagen and fibronectin production, thereby demonstrating the requirement of CTGF for their up-regulation. To investigate the involvement of the TGF-β1 response element (TRE) and the SMAD binding element (SBE) in CTGF induction, we cloned the rat CTGF proximal promoter containing the TRE and SBE motifs into a pGL3-Luciferase reporter construct. Using promoter deletion and site-directed mutagenesis approaches, we demonstrated the requirement of both the TRE and SBE for CTGF induction by TGF-β1 in osteoblasts.

Conclusion: Collectively, these data demonstrate for the first time that CTGF is an essential downstream mediator for TGF-β1 induced ECM production in osteoblasts, and that CTGF induction by TGF-β1 requires both the TRE and SBE.

Introduction

The development (modeling) and maintenance (remodeling) of bone is a complex process that involves the coordinated actions of different cell types and is regulated by a multitude of systemic and locally-produced growth factors. Osteoblasts, the bone forming cells, produce bone through their gradual differentiation and biosynthetic activity that is characterized by a temporal pattern of expression of cell growth genes, osteoblast phenotype-related genes and transcriptional factors.[43] Early stages of osteoblast differentiation are characterized by proliferation of osteoprogenitor cells that gradually differentiate to mature, biosynthetic osteoblasts that produce the components of the bone extracellular matrix (ECM), including type I collagen and fibronectin.[43] Subsequently, in the later stages of osteoblast differentiation this ECM becomes is mineralized to form bone.

One secreted growth factor that is known to modulate the proliferation and differentiation of osteoprogenitor cells and control the synthetic functions of osteoblasts is transforming growth factor-β1 (TGF-β1).[4] TGF-β1 promotes recruitment of osteoblast precursors to sites of bone formation,[39] acts as a regulator of osteoblast replication[6] and stimulates the expression of numerous bone ECM proteins such as type I collagen and fibronectin.[5, 8, 23, 49] However, the molecular mechanism(s) by which TGF-β1 functions in osteoblasts are not fully understood.

Another secreted growth factor with more recently described effects on osteoblast differentiation and function is connective tissue growth factor (CTGF). CTGF is a cysteine-rich, matricellular protein that belongs to the CCN (CTGF, Cyr61, nov, WISP1, WISP2 and WISP3) protein family.[34] CTGF has been implicated as a key regulatory factor in complex biological and pathological processes including wound healing, angiogenesis,[1, 32] fibrotic disorders,[11, 12, 25, 31, 37] carcinogenesis,[18, 30] endochondral ossification[27, 46] and bone fracture repair.[35, 41] Depending on the cell

type, CTGF has been shown to promote cell growth, migration, adhesion, survival, differentiation and the biosynthesis of ECM proteins.[7, 17, 26, 29, 34]

Our laboratory recently demonstrated that CTGF is produced and secreted by osteoblasts where it acts in an autocrine fashion as an anabolic growth factor to regulate osteoblast differentiation and function.[41, 50] Using recombinant CTGF, we demonstrated that CTGF acts to stimulate new bone formation when injected into the marrow cavity in vivo, and has the ability to promote proliferation, matrix formation (nodule formation) and mineralization in differentiating primary osteoblast cultures.[41] These results have been confirmed by similar findings using osteoblast cell lines, where CTGF was shown to promote proliferation, collagen synthesis, alkaline phosphatase (ALPase) activity and mineralization.[36] Collectively, these studies suggest that CTGF plays distinct roles during both early and late events of osteoblast differentiation.

Studies in other connective tissue cell types have shown that TGF-β1 is a potent inducer of CTGF expression,[2] and this induction has largely been attributed to the presence of two cis-regulatory elements, the TGF-β1 response element (TRE) and the Smad binding element (SBE) found in the CTGF proximal promoter.[22, 24] Studies have demonstrated divergent roles for each of these elements in either basal or TGF-β1 induced regulation of CTGF. For example, in scleroderma fibroblasts and chondrocytic cell lines (HCS-2/8) only the TRE is required for TGF-β mediated CTGF induction.[14, 24] However, in normal skin fibroblasts and mesangial cells, the TRE only plays a role in basal CTGF expression and the SBE mediates CTGF induction by TGF-β1.[24] From these studies it has become clear that the mechanism for induction of CTGF expression by TGF-β1 differs depending on the cell type being investigated.

A number of studies have shown that CTGF acts as a downstream mediator of some of the effects of TGF-β1 cellular functions including, cell proliferation, migration, adhesion and matrix production.[3, 13, 20, 21, 42, 44, 45, 47, 48, 51] More recently, a study using CTGF -/- mice demonstrated the requirement of CTGF for TGF-β1 induced responses in embryonic fibroblasts including induction of subsets of genes associated with matrix production such as type I collagen.[42] Although studies have demonstrated the requirement of CTGF expression for TGF-β1 induced responses in specific cells, it is important to note that this TGF-β1/CTGF interaction is highly dependent on cell type. Since TGF-β1 and CTGF have similar effects on osteoblast differentiation by modulating their proliferation and inducing ECM synthesis, the potential interaction between these two factors in osteoblasts merits investigation. Therefore, this study utilized a well-established primary osteoblast culture system to: 1) examine the induction of CTGF expression by TGF-β1 at different stages of differentiation, 2) determine whether CTGF is a downstream mediator of TGF-β1 effects on synthesis of extracellular matrix proteins, and 3) characterize the contributions of the TRE and SBE to CTGF induction by TGF-β1.

Materials and Methods

Animals. Primary osteoblasts were derived from bone (calvaria) of Norway-Hooded rats that are maintained at Temple University School of Medicine (Philadelphia, PA). All animals were handled according to the principles in the NIH Guide for the Care and Use of Laboratory Animals (U.S. Department of Health and Human Services, Publ. No. 86-23, 1985) and guidelines established by the IACUC of Temple University.

Primary Cell Culture. Primary osteoblast cultures were obtained using neonatal rats as previously described.[50] Briefly, parietal calvaria from neonatal rat pups were isolated and subjected to sequential digestions of 5, 15, 15, 25 and 25 min at 37°C in a shaking water-bath with 2% collagenase-P (Roche)/0.25% trypsin (Mediatec). Cells from the first step of digestion were discarded and those released from the 2nd–5th digestions were plated in 100mm dishes (Falcon) at 5×10^5 cells/plate in Earle's Minimal Essential Medium (EMEM; Mediatec) supplemented with 10% fetal calf serum (FCS; Mediatec). The cells were incubated at 37°C with 5% CO_2 with change of media every three days until they reached 80% confluency. Cells were maintained in this state or frozen in EMEM with 10% FCS and 10% DMSO for subsequent utilization. To initiate osteoblast differentiation, the plating medium was replaced with EMEM containing 10% FBS and 50μg/ml ascorbic acid (Sigma) on the 3rd day of culture. Then on the 7th day and every 3 days thereafter the medium was replaced with EMEM containing 10% FCS, 50μg/ml ascorbic acid and 10mM β-glycerophosphate (Sigma).

TGF-β1 Stimulation. Recombinant TGF-β1 (Calbiochem) was diluted to a concentration of 1μg/ml in sterile 4mM HCL containing 0.1% BSA and stored at –20°C. Osteoblasts were serum deprived for 24hrs prior to treatment. Cells were treated with a standard concentration of 5ng/ml of TGF-β1 for a 24hr period unless otherwise stated.

RNA Isolation and Northern Blotting. Total cellular RNA was isolated from cell cultures using Trizol reagent (Invitrogen) according to the manufacturer's directions. For Northern blot analysis, 10μg of RNA was separated on an agarose gel and transferred to a Hybond (Amersham, Biosciences) membrane in the presence of 10x SSC. DNA probes used for hybridization were obtained from the following sequences: rat CTGF,[50] 18S (kindly provided by C. Demers, University of Montreal, Canada); rat type I collagen and rat fibronectin were amplified from a rat cDNA pool. All DNA probes were labeled with ^{32}P-dCTP using Rediprime II Random Prime Labeling System (Amersham Biosciences) according to the manufacturers instructions. Membranes were hybridized at 65°C in Church buffer consisting of 1% BSA, 1mM EDTA, 0.5M NaHPO4 (pH 7.2) and 7% SDS.

After hybridization, the blots were washed twice in 0.1% SDS/2X SSC and twice in 0.1% SDS/1XSSC at room temperature and exposed to Kodak film at –80°C. Changes in mRNA levels were determined following normalization of each sample to the 18S RNA signal in each lane. All data are expressed as a percent of the control for each experiment.

Protein Isolation and Western Blotting. Western blot analysis was performed as previously described.[41] Briefly, cell monolayers were homogenized for 24 hrs at 4°C in protein extraction buffer (RIPA buffer) consisting of 50mM Tris-HCL (pH 7.5), 135 mM NaCl, 1% Triton X-100, 0.1% sodium deoxycholate, 2mM EDTA, 50mM NaF, 2mM sodium orthovanadate, 10μg/ml aprotinin, 10μg/ml leupeptin and 1mM PMSF. Cell lysates were clarified using centrifugation. Total protein concentration from the resulting supernatant was measured using the BCA Protein Assay Reagent Kit (Pierce) according to the manufacturer's instructions. Samples were subjected to sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) on a 7.5% acrylimide gel and transferred to PVDF filters by electroblotting. Blots were incubated with either anti-CTGF (Sigma), anti-fibronectin (Cell Signaling) or anti-actin (Sigma) primary antibody, and then with HRP-conjugated secondary antibody (Sigma). Antigens were detected using the Pierce chemiluminescent substrate system.

Osteoblast Collagen Synthesis. Collagen synthesis was measured by the incorporation of [3H]-proline into collagen as previously described.[52] Primary rat osteoblasts were pulsed with 1 μCi/ml L-[2,3,4,5-^{3}H]-proline (Amersham) and 50μg/ml ascorbic acid in the last 24hrs of the culture. Collagen from the cell layer was solublized using 0.5 N NaOH and proteins were precipitated with 20% trichloroacetic acid (TCA) by centrifugation at 3000g. Samples were split into duplicates, neutralized with HCL and one sample from each pair was treated with collagenase (600U/ml). Following a 2hr digestion at 37°C, acid-insoluble material was precipitated with 10% TCA/0.5% tannic acid. Samples were counted for β-emission. Counts were subtracted from duplicate samples without collagenase and normalized to cell number.

Osteoblast Transfection with CTGF siRNA. Subconfluent primary osteoblast cultures were transfected with siRNA 48hrs prior to treatment with TGF-β1. Transfections were carried out using Lipofectamine 2000 (Invitrogen) and Opti-MEM (Invitrogen) according to the manufacturer's protocol. An siRNA SMARTpool consisting of four target-specific 19-nucleotide siRNA duplexes was manufactured from the open reading frame of rat CTGF (accession number in GenBank: NM_022266) by Dharmacon Research (Lafayette, CO). Control siRNA was siCONTROL non-targeting siRNA #1 (D-001210-0105) (Dharmacon). siGLO RISC-Free siRNA (D-001600-01-05) (Dharmacon) was used to assess transfection efficiency.

Luciferase Reporter Constructs. CTGF promoter deletion constructs: pGL3-T129, pGL3-S164 and pGL3-314 were obtained as a generous gift from Dr. Tom Owen, Pfizer Global Research. pGL3-W787 was constructed from a fragment of the CTGF proximal promoter containing nucleotides –787 to +1 from rat CTGF cloned into the Sma I-Bgl II cloning site of the pGL3-Basic luciferase reporter vector (Promega). This construct was used as a template for PCR amplification to make the mutation constructs. Multiple mutation constructs were created containing a substitution of the SBE (ΔSBE), the TRE (ΔTRE) or both the SBE and TRE (ΔSBE/ΔTRE) with a BamHI restriction site. Figure 7A indicates positions of base substitutions in the SBE and TRE consensus sequences used to create a BamHI restriction site in place of either the SBE, TRE or both. Substitutions were confirmed by BamHI restriction digestion and sequencing. CTGF promoter mutation constructs were constructed using the Quick-Change site-directed mutagenesis kit (Stratagene) using the following oligonucleotides:

ΔSBE: 5'-GGACTGGGGAACTGTGAGGTGTAAGGAGGCCTAGGTTTTCGACCG TGTGAAATCGACC-3', 5'-GGTCGATTTCACACGGTCGAAAACCTAGGCCTCCTTTCACCTCACAGTTCCCCAGTCC-3';

ΔTRE: 5'-CAGACGGAGGAATGTGGAGGATCAGGGGTCAGGATCAATCC-3' 5'-GGATTGATCCTGACCCCTGGATCCTC CACATTCCTCCGTCTG-3';

ΔSBE/ΔTRE: 5'-ATCCGGAGGAATGTGGAGGATCC AGGGGTCAGGATCAATCC-3', 5'-GGATTGATCCTGACCCCTGGATCCTCCACA TTCCTCCGGAT-3'.

pGL3-SV40 and pRL-Renilla luciferase vectors were obtained from Promega.

Luciferase Assays. Luciferase activity was determined using the Dual-Glo Luciferase Assay system (Promega) according to the manufacturer's instructions. Primary osteoblasts were plated in a 96 well microplate ($2.4x10^4$ cells/well) and incubated in EMEM supplemented with 10% FCS for 24hrs. At this point cultures were transfected with 0.4μg of an indicated luciferase reporter vector and co-transfected with 0.2μg of a Renilla luciferase expression vector (internal control). The cells were serum starved for 24hrs and then treated as indicated. To determine the luciferase activity, cells and buffers were equilibrated to RT for 1hr and subsequently lysed in a volume of Dual-Glo luciferase reagent equal to that of the culture media. After a 10 minute incubation, the firefly luminescence was then measured using a Wallac 1420 fluorometer. Next, the firefly signal was quenched using an equal volume of Dual-Glo, Stop and Glo reagent. After a 10 minute incubation the Renilla luminescence was measured. Relative luciferase activity was expressed as a ratio of firefly/renilla luminescence values. All samples were normalized to a blank reaction.

Data Analysis. For all quantitative data, analysis of variance (ANOVA) was employed to evaluate the effect of one variable on two or more independent groups. In the event of a significant group effect, individual pairs of means were compared using the Bonferroni post-hoc test. Data are calculated as mean + SEM, and in some cases, converted to percent of control. A p value <0.05 was used to determine whether differences were statistically significant.

Results

TGF-β1 up-regulation of CTGF expression in primary osteoblast cultures (Figure 1). In the first series of experiments we assessed the ability of TGF-β1 to up-regulate CTGF expression in osteoblasts during three stages of differentiation; proliferation, matrix production and maturation, and mineralization. Preliminary experiments revealed that treatment of osteoblasts with TGF-β1 at a dose of 5ng/ml for a period of 24 hours induced a maximal increase in steady-state CTGF transcript and protein levels compared to mock-treated (diluent only), control cultures. Therefore, these conditions were used in all subsequent experiments unless otherwise stated. Northern blot analysis showed that TGF-β1 stimulation up-regulated CTGF mRNA expression to a similar extent during the proliferative (~day 7), matrix production (~day 14) and mineralization (~day 21) stages as compared to mock-treated controls (Fig. 1A). Western blot analysis confirmed that CTGF protein levels were also increased following TGF-β1 treatment at all stages of osteoblast differentiation (Fig. 1B).

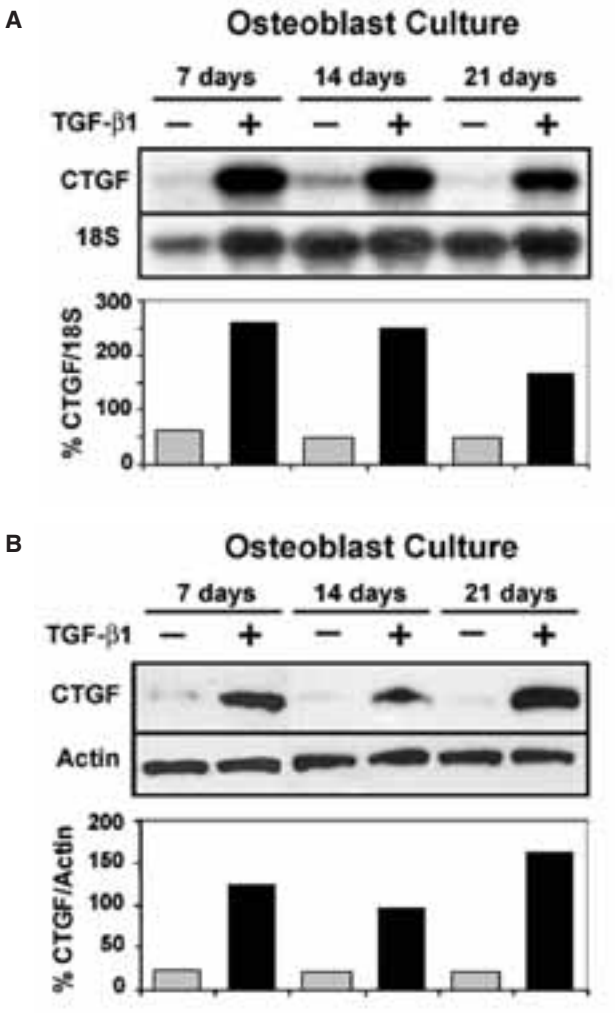

Figure 1. TGF-β1 induction of CTGF expression at different stages of osteoblast differentiation. Primary osteoblasts cultured under differentiating conditions were serum deprived (24hrs) and treated with TGF-β1 (5ng/ml) or diluent only (mock treated control) for an additional 24hrs prior to termination at day 7, 14 and 21. CTGF expression was assessed by Northern **(A)** and Western blot analysis **(B).** Bar graphs represent data from pictured blots. Experiment was repeated three times with similar results.

CTGF siRNA inhibits induction of CTGF expression by TGF-β1 (Figure 2). In order to study if CTGF functions downstream of TGF-β1 in osteoblasts, we used a CTGF siRNA gene knockdown strategy to inhibit TGF-β1 induced CTGF expression. Using fluorescently labeled siRNA duplexes, we were able to achieve a transfection efficiency ranging from 80-85% in primary osteoblast cultures (Fig. 2A). Cell viability was not affected by any of the transfection reagents or the CTGF siRNA as determined by trypan blue exclusion counting and MTT assay (data not shown).

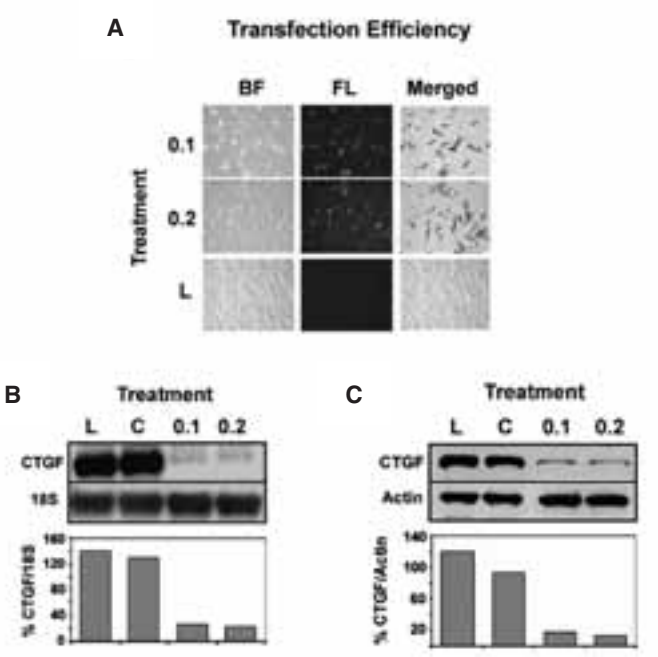

Figure 2. CTGF siRNA inhibits CTGF induction by TGF-β1. **(A)** Osteoblast cultures were transfected with 0.1μM or 0.2μM of fluorescently labeled siRNA duplex or lipofectamine (L) only. After 24hrs, cells were visualized using bright field (BF) and fluorescent (FL) microscopy; digital images from identical fields were merged. (B) Primary osteoblast cultures were transfected with lipofectamine only (L), 0.2μM non-targeting siRNA (C), 0.1μM or 0.2μM CTGF siRNA. Twenty four hours later, the cultures were serum deprived for 24hrs then treated with TGF-β1 (5ng/ml) for an additional 24hrs prior to termination. CTGF expression was assessed by Northern **(B)** and Western blot analysis **(C).** Bar graphs represent data from pictured blots. Experiment was repeated three times with similar results.

We first performed experiments to determine the time frame for maximal inhibition of CTGF expression in cells transfected with CTGF siRNA. The results showed that CTGF expression was maximally inhibited in osteoblasts at 48hrs post-transfection (data not shown). Following these preliminary experiments, cultures were transfected with 0.1μM or 0.2 μM CTGF siRNA, 0.2 μM non-silencing

siRNA, or Lipofectamine only. Following transfection, the cultures were serum-deprived for 24 hours prior to treatment with TGF-β1 for an additional 24 hours. The cultures were terminated and CTGF mRNA and protein levels were assessed by Northern and Western blot analysis, respectively. TGF-β1 stimulation of CTGF mRNA levels was dramatically reduced by 80–85% in the CTGF siRNA transfected cultures compared to the non-silencing (C) and lipofectamine (L) controls (Fig. 2B). Western blot analysis demonstrated that CTGF siRNA blocked the up-regulation of CTGF protein levels compared to the non-silencing (C) and lipofectamine (L) controls (Fig. 2C). The inhibition of CTGF mRNA and protein levels in TGF-β1-treated cultures transfected with CTGF siRNA resulted in levels that were similar to those found in cultures that were not treated with TGF-β1 (compare with Figure 1). To confirm the specificity of our CTGF siRNA, we analyzed its effects on expression of Cyr61, a CCN family member with the closest structural and functional homology to CTGF. CTGF siRNA (0.1μM) did not affect (down-regulate) the expression of Cyr61 even though this same dose effectively inhibited TGF-β1 induced CTGF (data not shown). These data demonstrate that the CTGF siRNA is specific for CTGF expression.

CTGF is a downstream mediator of TGF-β1 induced ECM synthesis in osteoblasts (Figures 3 and 4). Using the siRNA approach to knockdown CTGF, we next evaluated the ability of CTGF to modulate TGF-β1 induced collagen and fibronectin expression in our primary osteoblast culture system. Osteoblasts treated with TGF-β1 demonstrated a dose-dependent increase in type I collagen mRNA reaching maximal levels at doses of 2.5 ng/ml and above compared to mock-treated controls (Fig. 3A). Using a 3H-proline incorporation assay, we measured collagen protein production in TGF-β1 treated cells. Collagen production was also significantly increased at all doses of TGF-β1 following a pattern that was similar to that observed for mRNA levels (Fig. 3B).

To determine if CTGF served as a downstream mediator of TGF-β1 induced collagen synthesis, primary osteoblast cultures were transfected with CTGF siRNA prior to treatment with TGF-β1 to block up-regulation of CTGF expression. Type I collagen mRNA levels and collagen protein production were then assessed in these CTGF silenced cultures compared to control cultures. Collagen transcript levels were dramatically reduced in osteoblasts transfected with CTGF siRNA compared to Lipofectamine only (L) and non-silencing (C) transfection controls (Fig. 3C). Silencing of CTGF expression also reduced collagen production by ~70% as compared to Lipofectamine only (L) and non-targeting siRNA (C) transfection controls (Fig. 3D). The transfection

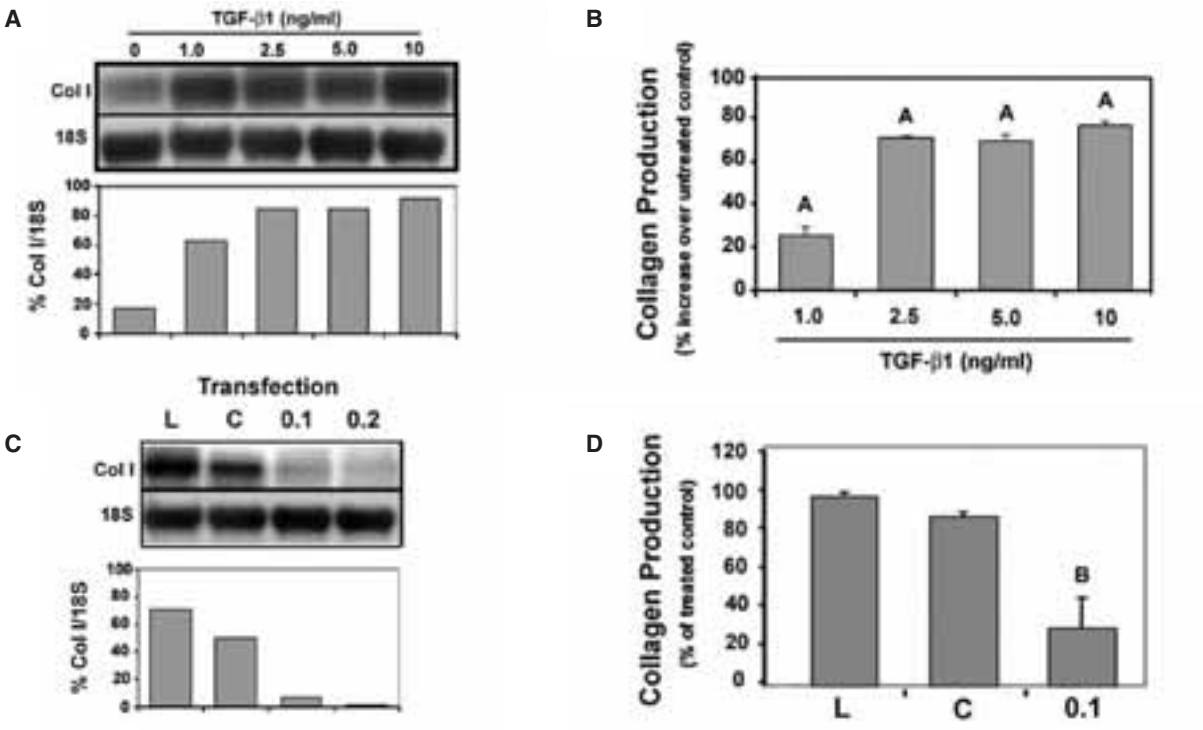

Figure 3. CTGF is a downstream mediator of TGF-β1 induced collagen production. (A–B) Primary osteoblast cultures were serum deprived (24hrs) and treated with increasing doses of TGF-β1 for 24hrs prior to termination on day 5 of culture. **(A)** Collagen mRNA expression was determined by Northern blot analysis. **(B)** Collagen production was measured using L-[2,3,4,5-3H]-proline incorporation β-emission counting. Data are expressed as a percent increase over the untreated (diluent only) control. **(C–D)** Primary osteoblast cultures were transfected with lipofectamine only (L), 0.2μM non-silencing siRNA (C), 0.1μM or 0.2μM CTGF siRNA. Twenty four hours later, the cultures were serum deprived for 24hrs then treated with TGF-β1 (5ng/ml). Collagen mRNA expression was determined by Northern blot analysis **(C)** and collagen production was measured using L-[2,3,4,5-^{3}H]-proline incorporation β-emission counting **(D).** Data are expressed as percent of TGF-β1 treated, untransfected control. (*A* = $p<0.05$ compared with untreated control; *B* = $p<0.05$ compared with non-silencing siRNA control.)

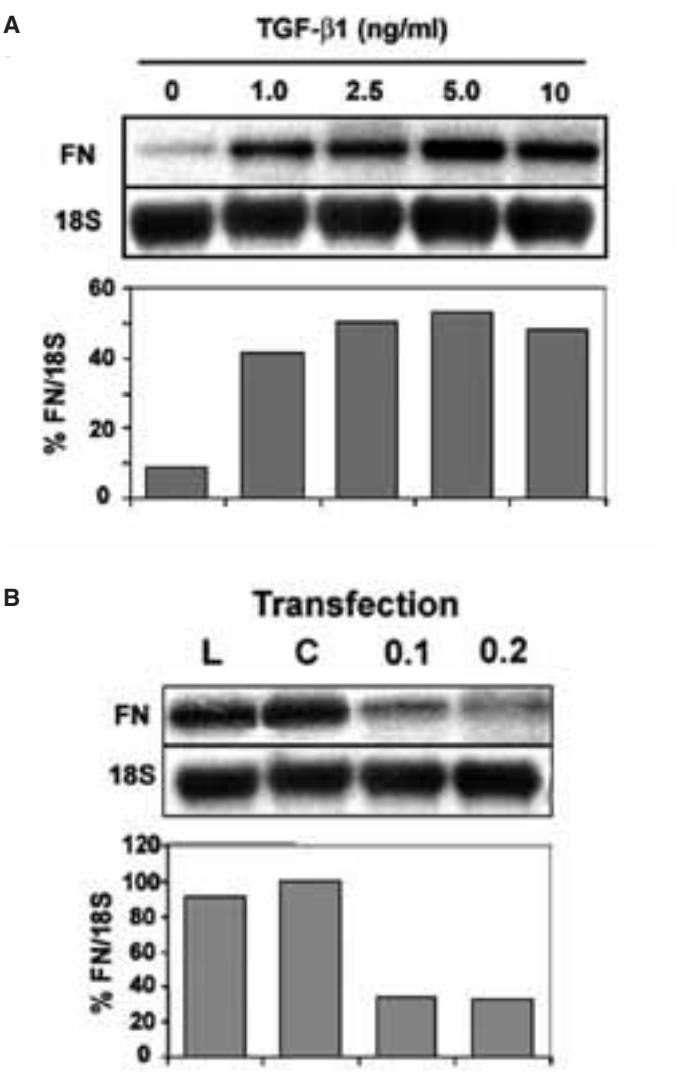

Figure 4. CTGF is downstream of TGF-β1 induction of fibronectin expression. (A) Primary osteoblast cultures were terminated at day 5 after serum deprivation for 24hrs and treatment with increasing doses of TGF-β1 for an additional 24hrs. Fibronectin expression was determined by Northern blot analysis. **(B)** Primary osteoblast cultures were transfected with lipofectamine only (L), 0.2μM non-silencing siRNA (C), 0.1μM or 0.2μM CTGF siRNA. Twenty four hours later, the cultures were serum deprived (24hrs) then treated with TGF-β1 (5ng/ml) for 24hrs. Fibronectin mRNA expression was determined by Northern blot analysis. Bar graphs represent data from pictured blots. Experiment was repeated three times with similar results.

controls were not significantly different from TGF-β1 treated, untransfected controls (data not shown).

Fibronectin transcripts also increased after treatment with TGF-β1 (Fig. 4A). This increase in fibronectin mRNA levels was similar for each of the doses of TGF-β1 used. Transfection with either 0.1μM or 0.2μM CTGF siRNA caused a reduction of approximately 70% in fibronectin transcript levels compared to Lipofecamine only (L) and non-silencing siRNA (C) transfection controls (Fig. 4B). Collectively, the results of these experiments clearly demonstrate that the induction of type I collagen and fibronectin transcripts as well as collagen synthesis by TGF-β1 in primary osteoblast cultures are CTGF dependent events.

The CTGF promoter is TGF-β1 inducible (Figure 5). Several previous studies have identified putative and functional regulatory motifs in the CTGF proximal promoter that

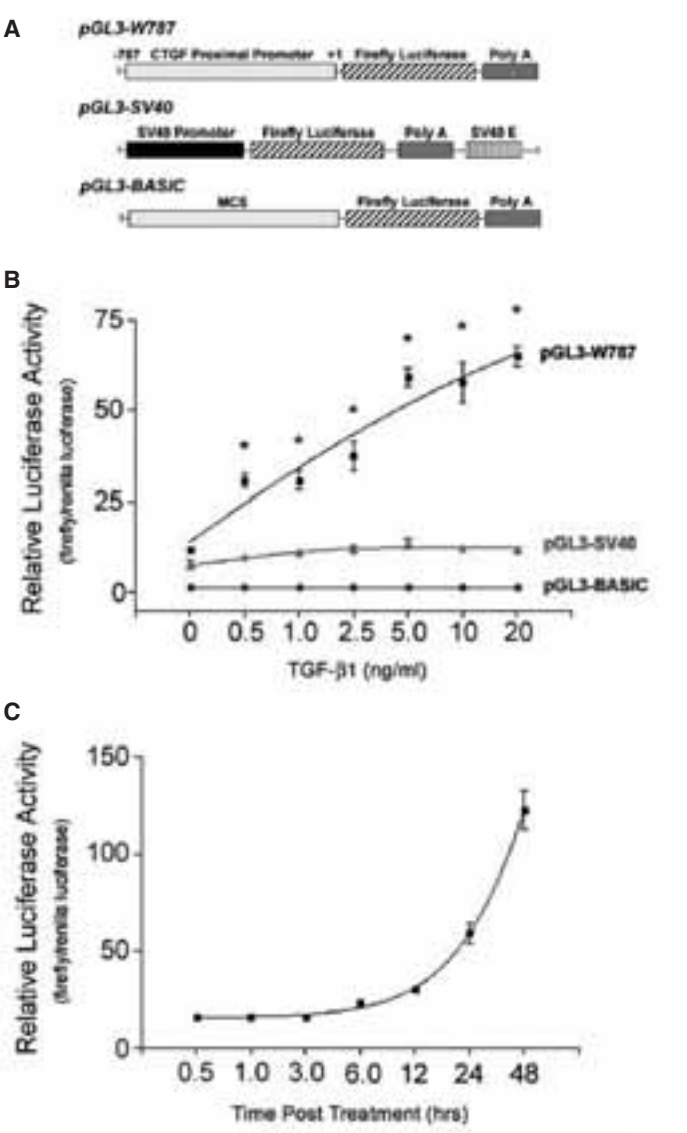

Figure 5. Effect of TGF-β1 on CTGF promoter activity. (A) Structures of the promoter constructs: pGL3-W787, pGL3-SV40 and pGL3-Basic. **(B)** Primary osteoblasts were plated in 96 well microplates and transfected with 0.4 μg of pGL3-W787 (CTGF proximal promoter +1/–787), pGL3-SV40 or pGL3-Basic vector and co-transfected with 0.2μg of a Renilla luciferase expression vector (internal control). The cells were serum starved for 24hrs and then treated with indicated doses of TGF-β1 for 24hrs. Data shown as mean +/– SEM (n=5). **(C)** Primary osteoblasts were plated in 96 well microplates and co-transfected with 0.4 μg pGL3-W787 and 0.2 ug Renilla luciferase expression vector. Cells were serum starved and treated with TGF-β1 (5ng/ml) for indicated times. Data shown as mean +/– SEM (n=5). Relative luciferase activity was expressed as a ratio of firefly/renilla luminescence values. A statistically significant increase in luciferase activity was observed for the pGL3-W787 construct at each dose of TGF-β1 tested compared to pGL3-SV40 or pGL3-Basic (*p<0.001).

are required to confer TGF-β1 responsiveness.[22, 24] The purpose of this experiment was to determine if TGF-β1 treatment induced elevated CTGF promoter activity in osteoblasts. We cloned the entire CTGF proximal promoter spanning nucleotides +1 to –787 into a pGL3-Luciferase reporter vector (W787) and used it to assay for TGF-β1 responsiveness (luciferase activity) in our primary osteoblast culture system (Figure 5A). In these experiments a pGL3-SV40 vector containing the SV40 promoter and the promoter-less pGL3-Basic vector were utilized as negative controls. The pGL3-W787, pGL3-SV40 and pGL3-Basic vectors were transfected into parallel cultures of primary rat osteoblasts. The cultures were serum starved for 24hrs and then treated with increasing doses of TGF-β1 for 24hrs. Luciferase assays revealed that TGF-β1 stimulated the activity of the CTGF promoter in a dose-dependent manner, with a maximal response occurring at doses of 5ng/ml and greater which represented a ~6.5-fold and 30-fold increase over the pGL3-SV40 and pGL3-Basic vectors, respectively (Figure 5B).

We next examined the temporal pattern of CTGF promoter inducibility in TGF-β treated primary osteoblast cultures. There was a time dependent elevation of luciferase activity following TGF-β1 exposure with a maximal response occurring at 48hrs post-treatment (Figure 5C). Since the 24hr time point represented the approximate mid-log phase of the response curve, we used a 24 hour treatment period for all subsequent luciferase experiments.

The SBE and TRE are required for TGF-β1 induction of CTGF (Figures 6 and 7). Previously CTGF induction has largely been attributed to the presence of two proximal promoter elements, the TGF-β1 response element (TRE) and the Smad binding element (SBE).[24] The relative contributions of these two elements in CTGF induction by TGF-β1 vary in different cell types. In these experiments, we evaluated whether the TRE or the SBE were required to confer TGF-β1 responsiveness to the CTGF promoter in primary osteoblasts. To delineate which motif is responsible for induction by TGF-β1, we constructed deletion mutants of the CTGF promoter containing only the first 129 nucleotides (T129), the TRE (S164) or both the SBE and TRE (P314) (Figure 6A–B). Parallel osteoblast cultures were plated and transfected with either pGL3-Basic (negative control), T129, S164, P314 or W787 (positive control). The cells were serum starved for 24hrs and then treated with TGF-β1 (5ng/ml) or mock treated (TGF-β1 diluent) for 24hrs. In all deletion constructs a significant decrease in the basal level of expression was detected compared to the W787 construct (Figure 6C). With respect to TGF-β1 inducibility of the CTGF promoter, inclusion of the TRE alone was not enough to confer TGF-β1 responsiveness. While the P314 construct, which contained both the SBE and TRE, was 3-fold more responsive to TGF-β1 treatment compared to the other deletion constructs, this response was significantly lower (70% decrease) from that of the full length proximal promoter construct (W787).

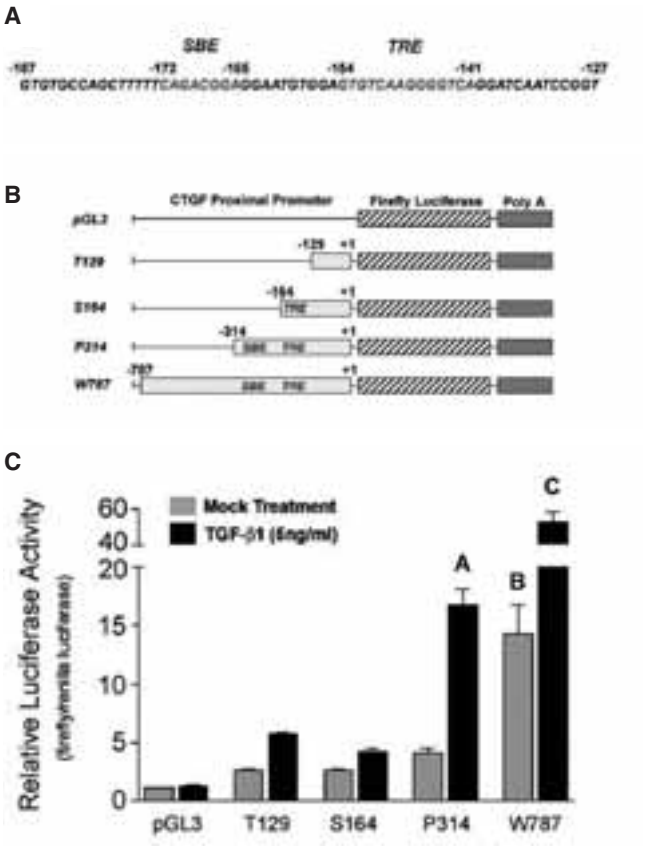

Figure 6. Deletion analysis of CTGF promoter luciferase constructs. (A) Position of the TRE and SBE (shown in red) relative to each other in the CTGF promoter. **(B)** Deletion constructs used to test involvement of the TRE or SBE in CTGF promoter activity by TGF-β1 treatment in osteoblasts. Consensus sequences for the TGF-β1 response element (TRE) and the Smad binding element (SBE) are indicated. **(C)** Primary osteoblasts were plated in 96 well microplates and transfected with either 0.4 μg of pGL3-Basic, T129, S164, P314 or W787 and co-transfected with 0.2μg of a Renilla luciferase expression vector (internal control). The cells were serum starved for 24hrs and then treated with TGF-β1 (5ng/ml) or mock treated (TGF-β1 diluent) for an additional 24hrs. Luciferase activity was then assessed. Data shown as mean + SEM (n=8). Relative luciferase activity was expressed as a ratio of firefly/renilla luminescence values. (*A* = p<0.001 compared to TGF-β1 treated pGL3, T129, S164; *B* = p<0.001 compared to mock treated pGL3, T129, S164, P314; *C* = p<0.001 compared to TGF-β1 treated P314).

These results suggest that: 1) other elements upstream of the SBE are required for TGF-β1 responsiveness, and/or 2) due to the relative proximity of the SBE and TRE, the deletion strategy itself may sterically inactivate these sites. In order to address these possibilities, a mutagenesis approach was utilized that is summarized in Figure 7. Our approach involved mutating the SBE and/or TRE to BamHI sites as previously described 28. Multiple mutation constructs were created containing a mutation of the SBE (ΔSBE), the TRE (ΔTRE) or both the SBE and TRE (ΔSBE/ΔTRE) (Figure 7A–B). Mutations were confirmed by BamHI restriction digestion and sequencing. Interestingly, the ΔSBE, ΔTRE and the

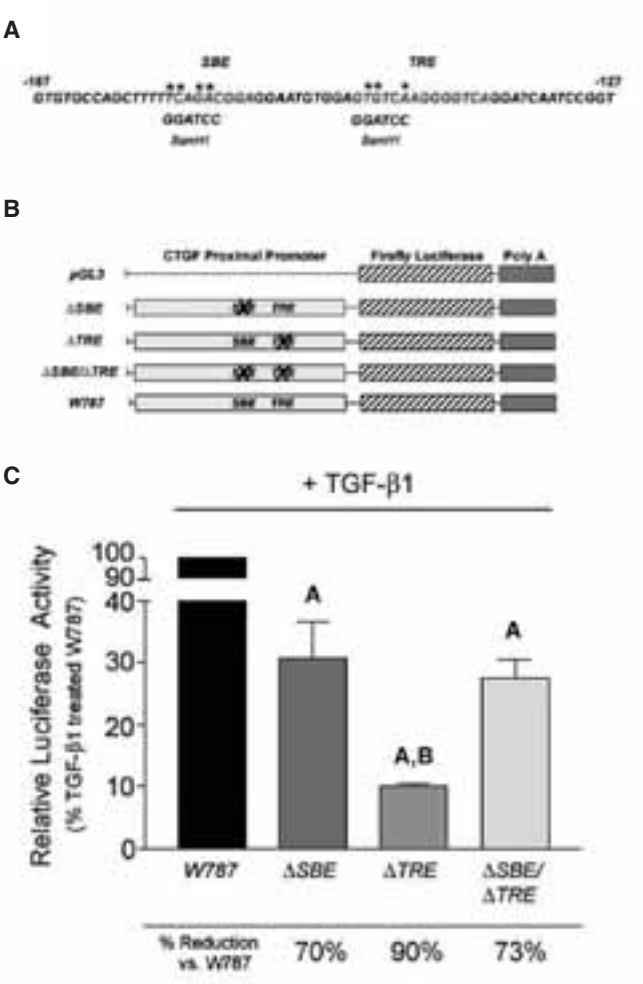

Figure 7. The SBE and TRE are required to confer TGF-β1 responsiveness to the CTGF promoter in osteoblasts. (A) Asterix indicates base substitutions in the SBE and TRE consensus sequences used to create a BamHI restriction site in place of either the SBE, TRE or both. Mutations were confirmed with BamHI restriction digestion and sequencing. **(B)** Multiple mutation constructs were created containing a substitution of the SBE (ΔSBE), the TRE (ΔTRE) or both the SBE and TRE (ΔSBE/ΔTRE). pGL3 (negative control) and W787 (positive control) were used as controls for luciferase activity. **(C)** Primary osteoblasts were plated in 96 well microplates and transfected with either 0.4 μg of ΔSBE, ΔTRE, ΔSBE/ΔTRE, pGL3 or W787 and co-transfected with 0.2μg of a Renilla luciferase expression vector (internal control). The cells were serum starved for 24hrs and then treated with TGF-β1 (5ng/ml) for 24hrs. Luciferase activity was then assessed. Relative luciferase activity was expressed as a % of W787 (+ SEM, n=8). (*A* = p<0.001 compared to W787; *B* = p<0.01 compared to ΔSBE).

ΔSBE/ΔTRE all exhibited a marked decrease in TGF-β1 inducibility by 70%, 90% and 73%, respectively when compared to the full length proximal promoter construct (W787) (Figure 7C). In all mutation constructs a significant decrease was also seen in basal CTGF expression (90%) compared to the W787 promoter construct (positive control) (data not shown). Thus, the presence of both the SBE and TRE are required for basal and TGF-β1 induced CTGF expression in osteoblasts.

Discussion

The recent identification of CTGF as an essential downstream effector of TGF-β1 in certain biological and pathological processes[20, 33, 48, 51] has sparked interest in this novel mechanism of action. Previous studies have demonstrated that both TGF-β1 and CTGF are growth factors affecting osteoblast proliferation/differentiation and function.[19, 41] Specifically, TGF-β1 has bifunctional effects on osteoblast proliferation and induces the synthesis of extracellular matrix components such as type I collagen and fibronectin.[6, 10] CTGF has been shown to promote proliferation and extracellular matrix formation in osteoblastic cells.[36, 41] In this study, we were interested in; 1) characterizing the regulation of CTGF mRNA and protein expression by TGF-β1 in primary osteoblast cultures, 2) examining whether CTGF acts as a downstream effector of TGF-β1 induced extracellular matrix (ECM) production, and 3) determining the promoter motifs responsible for TGF-β1 induction of CTGF expression.

Northern and Western blot analysis revealed that TGF-β1 is a potent inducer of CTGF expression in osteoblasts. The ability of TGF-β1 to induce CTGF expression in osteoblasts is consistent with previous reports that have established TGF-β1 as a potent inducer of CTGF expression in other cell types.[2] A recent study using murine osteoblasts demonstrated TGF-β1 induction of CTGF mRNA expression in primary cultures,[38] though the time course of induction was significantly shorter than the time course demonstrated in this study. The three stages of differentiation in primary osteoblast cultures have been well-characterized and include an initial stage of cell proliferation until the cells reach confluency (~day 7), followed by a phase of matrix production and maturation (~day 7–14), and ending with a stage of mineralization in which minerals accrue in the matrix (~day 14–21). The ability of TGF-β1 to induce CTGF expression was not affected by the stage of osteoblast differentiation and occurred at a similar magnitude during each of these stages.

A major function of TGF-β1 during osteoblast differentiation is to stimulate production of the extracellular matrix components that compose osteoid.[9, 49] In this study, treatment of primary osteoblasts with TGF-β1 significantly enhanced the expression of type I collagen and fibronectin compared to control (non TGF-β1 treated) cultures. This induction of extracellular matrix proteins was effectively inhibited in cultures in which the up-regulation of CTGF expression was blocked using CTGF siRNA. This is the first study demonstrating CTGF as a requisite downstream mediator of TGF-β1 induced ECM protein synthesis in osteoblasts, and these results are consistent with previous reports in other cell systems. In-vitro studies in normal rat kidney fibroblasts demonstrated that TGF-β induced collagen synthesis can be inhibited with anti-sense CTGF or anti-CTGF antibody.[13] Similarly, in rat renal fibroblasts, CTGF anti-sense blocked TGF-β induced fibronectin expression and collagen produc-

tion,[51] and treatment of rat mesangial cells with neutralizing antibodies to CTGF inhibited TGF-β induction of fibronectin synthesis.[3] Although our results clearly demonstrate that CTGF plays an important role as a downstream effecter of TGF-β1 induced ECM synthesis, we cannot rule out the possibility that other CTGF independent mechanisms may also contribute to TGF-β1 induced ECM synthesis.

The mechanism by which TGF-β1 induces CTGF expression in osteoblasts is unknown. A more detailed study of TGF-β1 regulation of CTGF has been performed in other cell types and found that CTGF induction has largely been attributed to the presence of two proximal promoter elements, the TGF-β1 response element (TRE) and the Smad binding element (SBE).[22, 24] To characterize the cis-regulatory motifs involved in CTGF induction by TGF-β1, we used a combination of promoter deletion and site-directed mutant constructs, demonstrating the requirement of both the TRE and SBE for basal and TGF-β1 induced CTGF expression in osteoblasts. This is the first study in which both elements together have been shown to be necessary for induction of CTGF expression by TGF-β1. Previous studies in other cell types have demonstrated divergent roles for each of these elements in either basal or TGF-β1 induced regulation of CTGF.[22, 24]

Despite the unique cooperation of both elements in CTGF induction in osteoblasts it is not uncommon for multiple transcriptional elements to be involved in the context of Smad mediated signaling involving the SBE. Smads themselves have an intrinsically weak DNA binding affinity and are reliant on formation of large nucleoprotein complexes with transcription factors that bind to one or more transcriptional DNA sequences (cognate motifs) adjacent to the SBE to achieve target gene activation.[15] For example, multiple transcriptional motifs have been shown to be required for Smad3-mediated TGF-β1 induced transcription of p15.[16, 40] While the SBE in the p15 promoter has low affinity for Smad3 by itself, juxtaposition of the SBE to the Sp1 motif results in an affinity exceeding that of the Sp1 site or the SBE site alone, demonstrating the requirement of both the SBE and Sp1 to achieve high affinity binding and subsequent p15 expression.[16, 40] Based on these results we hypothesize that in osteoblasts the TRE acts as a cognate motif along with the SBE to achieve CTGF expression. Additional studies are underway to confirm this interaction and elucidate the signaling proteins that bind the TRE and SBE to regulate CTGF induction by TGF-β1 in primary osteoblasts.

In conclusion, our study demonstrates that CTGF is induced by TGF-β1 during all stages of osteoblast differentiation, and that CTGF acts as a downstream mediator of TGF-β1 induced ECM synthesis. Further, we show the requirement of both the TRE and SBE for both basal and TGF-β1 induced CTGF expression in osteoblasts. Additional studies are required to further elucidate the signaling pathways that regulate TGF-β1 induction of CTGF expression, and to understand the similarities and differences in mechanisms of action of these two growth factors as they relate to osteoblast differentiation and function. These studies will not only enhance our understanding of the molecular mechanisms of TGF-β1 and CTGF in osteoblasts, but may also help to identify novel therapeutic targets to control early stages of osteoblast differentiation during bone formation. The eventual clinical application of these studies is to develop new therapeutic approaches that enhance bone formation for the management of patients where this effect would have a favorable clinical outcome, such as osteoporosis, fracture repair and periodontal disease.

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Basic Science Research

Effects of Demineralized Bone Matrix on Human Marrow Stromal Cell Migration and Differentiation: A Comparative Study

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Abstract

Demineralized bone matrix (DBM) has been utilized as an autogenous bone graft substitute in the treatment of fracture nonunions as well as osseous defects. Though numerous DBM products are commercially available, a reproducible *in vitro* test to compare efficacy of products is necessary, though not yet described. We investigated the chemotactic and osteogenic properties of several commercially-available DBM's in an effort to establish such a model. A comparative migration assay was performed using different commercially available DBMs as chemotactic agents (DBX mix, DBX putty, Optium putty, Total Bone Matrix bar, Accell Connexus ISOTIS putty, Accell DBM100 putty, Accell Connexus GEN.SCI putty and AlloMatrix putty). Our results demonstrate that Accell DBM100 putty, Total Bone Matrix bar and Optium putty can stimulate *in-vitro* migration of human mesenchymal stem cells (hMSCs). This suggests that Accell DBM100 putty, Total Bone Matrix bar, and Optium putty might play a role in the chemotactic recruitment of undifferentiated osteogenic cells during bone remodeling and healing. For differentiation, hMSCs treated with AlloMatrix Putty showed more alkaline phosphatase staining in comparison with all the DBMs and controls. This suggests that AlloMatrix Putty stimulates osteoblast differentiation and also acts as an osteogenic agent during bone remodeling and healing.

Introduction

Demineralized bone matrix (DBM) products have been extensively used in recent years as alternatives for bone autografts in the healing of osseous defects and fracture nonunions. Marshall Urist's preliminary studies on the uses of DBM's are the basis of the current clinical use of this form of bone graft in situations in which autogenous grafting is less desirable.[33, 34] Despite the increasing popularity of DBM's, there continues to be confusion regarding the clinical efficacy of demineralized bone matrix for treatment of nonunions and bone defects. Currently, most of the major orthopaedic device and tissue bank companies promote each of their own DBM products to orthopaedic surgeons. It is difficult to compare clinical efficacy between DBM products between companies as well as between specimen lots of each product itself. Efficacy of DBM products is determined by intra-abdominal pouch placement and measurement of subsequent bone formation in the athymic animal model.[11] While this provides a measurable comparison between products, it is time consuming and requires animal experimentation to perform. An *in vitro* test which compares the efficacy between DBM products would be a better way to allow orthopaedic surgeons to select the most effective products from others. The aim of the present study was to develop such an *in vitro* test for this purpose and to subsequently compare several commercially-available DBM preparations.

Materials and Methods

Demineralized Bone Matrix. Each respective company donated DBM products for use in this study. These included DBX mix and DBX putty (Synthes, Paoli, PA), Optium putty (LifeNet, Virginia Beach, VA), Accell Total Bone Matrix bar, Accell Connexus ISOTIS putty, Accell DBM100 putty, and Accell Connexus GEN.SCI putty (ISOTIS OrthoBiologics Inc, Irvine, CA) and AlloMatrix putty (Wright Medical Technology Inc, Arlington, TN). 0.400g of each DBM was homogenized in a Tekmar homogenizer (Tekmar Company, Cincinnati, OH) and diluted in 10 ml of 1X PBS (Cellgro, Herndon, VA).

Cell Culture. hMSCs were cultured in a proliferation media consisting of minimal essential medium alpha (α-MEM) (Cellgro, Herndon, VA) + 20% fetal bovine serum (FBS) + 1% 100 U/ml penicillin/100 μg/ml streptomycin (Invitrogen, Carlsbad, CA) a medium that is capable of maintaining the cell's phenotypes. Cells were harvested at 60%–70% confluence using 0.25% Trypsin EDTA 1X (Cellgro, Herndon, VA) for 5 min at 37°C, and were then either re-plated. Cultures were incubated in a humidified incubator at 37°C and 5% CO_2. Only passages 5–10 were utilized for these studies.

Migration Assay. hMSCs were cultured until 70% confluence in α-MEM + 20% FBS + 1% penicillin/streptomycin, and an overnight serum starvation was performed. Different media were prepared: α-MEM + 0.5% BSA + 1% P/S (Control), or α-MEM + 0.5% BSA + 1% P/S + 150µl of demineralized bone matrix (experimental groups). Twenty-four well transwell culture plates with 8.0µm Pore Polycarbonate Membrane (Corning Incorporated, Corning, NY) were used for the migration assay. Six hundred µl of each different media was added to the lower well of the transwell plate and 100µl of cells (2×10^5 cells) suspended in α-MEM + 0.5% BSA + 1% P/S media was added to the inside of each insert. Cells were then incubated overnight in a humidified incubator at 37°C and 5% CO_2. Media of the lower well and insert was aspirated and rinsed twice with 1X PBS. Six hundred µl of 4% Paraformaldehyde was added to the lower well for 10 minutes at room temperature. Using cotton-tipped swabs, non-migratory cells were removed from the interior of the inserts. Inserts were transferred to a clean well containing 200µl of methylene blue in borate buffer (pH 8.5) for 30 min at room temperature. Inserts were washed several times in a beaker containing ddH_2O and allowed to air dry. Photographs of migratory cells were taken using an inverted phase contrast microscope (Nikon E300).

Methylene Blue Dye Extraction. Transwell inserts were transferred to an empty well and dye extraction was performed using 100% ethanol: 0.1% HCL (1:1v/v) 200 µl/well. This led to protonation of the acidic groups and liberation of methylene blue into the solution. Each plate was then incubated for 20 minutes on an orbital shaker and 100µl was transferred from each sample to a 96-well plate (BD, Franklin Lakes, NJ) and absorbance was measured at 560nm using a micro-plate reader.

Alkaline Phosphatase Staining. hMSCs were cultured in a osteogenic media consisting of minimal essential medium alpha (α-MEM) + 20% fetal bovine serum (FBS) + 1% 100 U/ml penicillin/100 µg/ml streptomycin. On day 3 of culture, the media was replaced with α-MEM + 20% FBS + 1% 100 U/ml penicillin/100 µg/ml streptomycin + 25µg/ml ascorbic acid + 150µl of demineralized bone matrix. On day 7 of culture, the media was replaced with α-MEM + 20% FBS + 1% 100 U/ml penicillin/100 µg/ml streptomycin + 50µg/ml ascorbic acid and 50mM β-glycerophosphate + 150µl of demineralized bone matrix. Cultures were terminated at day 14 and stained using an alkaline phosphatases staining kit (Sigma-Aldrich, St. Louis, MO). Treatments with the demineralized bone matrix were performed only at days 3 and 7 of culture.

Statistical Analysis. Quantitative results were expressed as the mean + standard error of the mean. Statistical evaluation was performed using a one-way ANOVA and Tukey test for individual comparisons; $p < 0.05$ was considered statistically significant.

Results

Migration Assay. Chemotactic cell migration assays of hMSCs demonstrated significant migration of cells with Accell DBM100 putty, Total Bone Matrix bar, and Optium putty (Fig. 1A). Chemotactic response to Accell DBM100 putty was significantly greater than that of both Total Bone Matrix bar and Optium putty, while Total Bone Matrix Bar showed significantly increased chemotactic migration in comparison with Optium putty. 0.5% BSA was used as a negative control for cell chemotaxis. DBX mix, DBX putty, Accell Connexus ISOTIS putty, Accell Connexus GEN.SCI putty and AlloMatrix putty did not demonstrate any significant migration when they were tested. Methylene blue dye extraction was performed and absorbance was measured at 560nm using a micro-plate reader (Fig. 1B). Thus, only three of the eight DBMs stimulated chemotaxis in hMSCs.

Osteoblast Differentiation. hMSCs were cultured in an osteogenic media and on day 3 and 7 the cultured cells were treated with demineralized bone matrix. Cultures were terminated at day 14 during the phase of matrix production and maturation (including nodule formation) (day 7–14). Alkaline phosphatase staining was performed as a marker for osteoblast differentiation. Cells treated with AlloMatrix putty had an increased expression of alkaline phosphatase if compared with control and the other DBMs. Cells treated with Optium putty and DBX mix demonstrated decreased expression of alkaline phosphatase when compared with control and AlloMatrix putty. Cells treated with DBX putty, Total Bone Matrix bar, Accell Connexus ISOTIS putty, Accell DBM100 putty and Accell Connexus GEN.SCI putty did not demonstrate alkaline phosphatase expression. Only cells treated with AlloMatrix putty exhibited an osteoinductive effect in hMSCs when differentiated into osteoblasts *in vitro.*

Discussion

Demineralized bone matrix (DBM) is being increasingly used for various musculoskeletal reconstructive procedures.[9, 10, 28, 32, 38] Most of the major orthopaedic device manufacturers now market their own brand of DBM for clinical use. Marketing strategies often include claims that these products enhance mesenchymal cell attachment, proliferation, and differentiation, resulting in biomaterials that are capable of regenerating bone in fractures, nonunions, cases of bone loss, and fusion procedures. The advantage over iliac crest bone graft is the avoidance of morbidity from the harvest technique.[4] With the increasing use of DBM's, it becomes critical that surgeons be able to distinguish which products will perform better than others.

Recent studies have shown that DBM contains different morphogens, cytokines, and mitogens that play a role in skeletogenesis and angiogenesis. However, the exact role of DBMs in bone remodeling and bone healing still needs to be

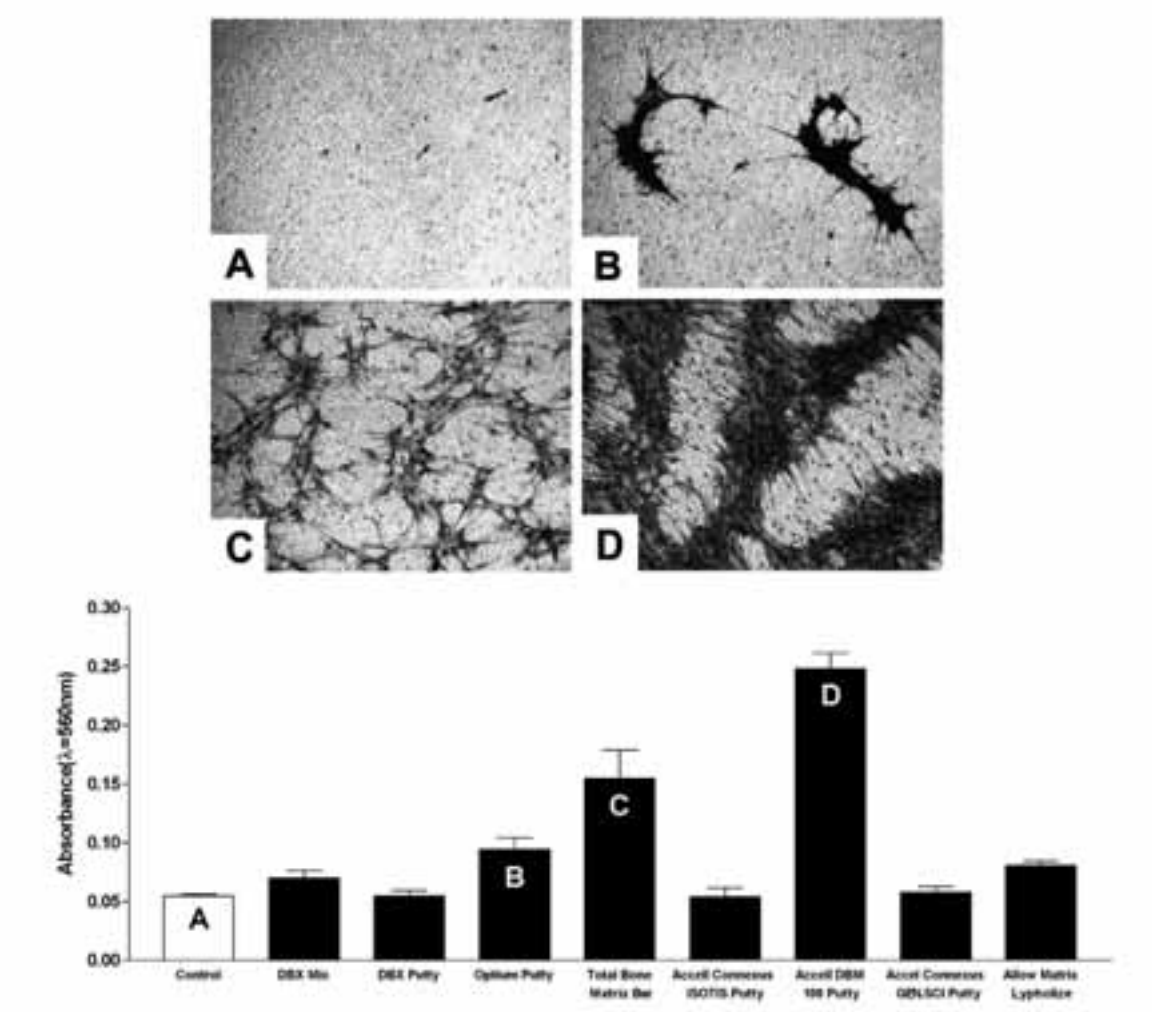

Figure 1. Chemotaxis of hMSC cells exposed to commercial available DBMs. Cell migration was assayed after 12 h. Pictures of migratory cells were taken under bright field microscope. Negative control (A), Optium putty (B), Total Bone Matrix bar (C), Accell DBM100 putty (D). Transwell inserts were transferred to an empty well, dye extraction was performed and absorbance was measured at 560nm using a microplate reader. Each condition was set up in triplicate or quadruplicate per experiment. The data bars are the mean + SEM of three separate experiments.

defined. Migration of osteogenic cells is an important physiological event both during bone healing and bone remodeling.

Fracture healing involves a complex cascade of cellular events; these events include immediate injury response, intramembranous ossification, chondrogenesis, and endochondral ossification resulting in the formation of a fracture callus.[31] This process can be influenced by metabolic alterations as well as poor blood supply and aging, which may lead to the lack of inductive callus and cause delayed union or nonunion.[5, 13]

There are several mechanisms by which DBM can be used to assist in bone regeneration. DBM can be used as a scaffold that is subsequently going to be replaced by new bone.[19, 29, 30] Some studies have demonstrated that the implantation of demineralized bone matrix into subcutaneous or muscular sites induce the proliferation of mesenchymal stem cells, which differentiate to cartilage cells and subsequently undergo endochondral ossification.[12, 14, 27, 33, 34] Different studies have suggested that DBM acts as a stimulant of chondrogenesis or osteogenesis of marrow stromal cells (MSCs), thereby promoting and accelerating the healing of critical size bone defects.[8, 23, 31] Previous *in-vivo* studies have shown that the cellular changes in response to demineralized bone implants include chemotaxis and attachment of the undifferentiated osteogenic cells to the matrix, proliferation and differentiation into chondrocytes, cartilage mineralization, vascularization, osteogenesis and bone marrow formation.[15, 20, 21, 39]

Undifferentiated osteogenic cells particularly depend and function *in vivo* via specific interactions with their substrate that are mediated by their extracellular matrix. The extracellular matrix modulates the nature of cell attachment and the resulting cell shape, cell proliferation and differentiation.[4] The marrow stromal population *in vitro* contains a diversity

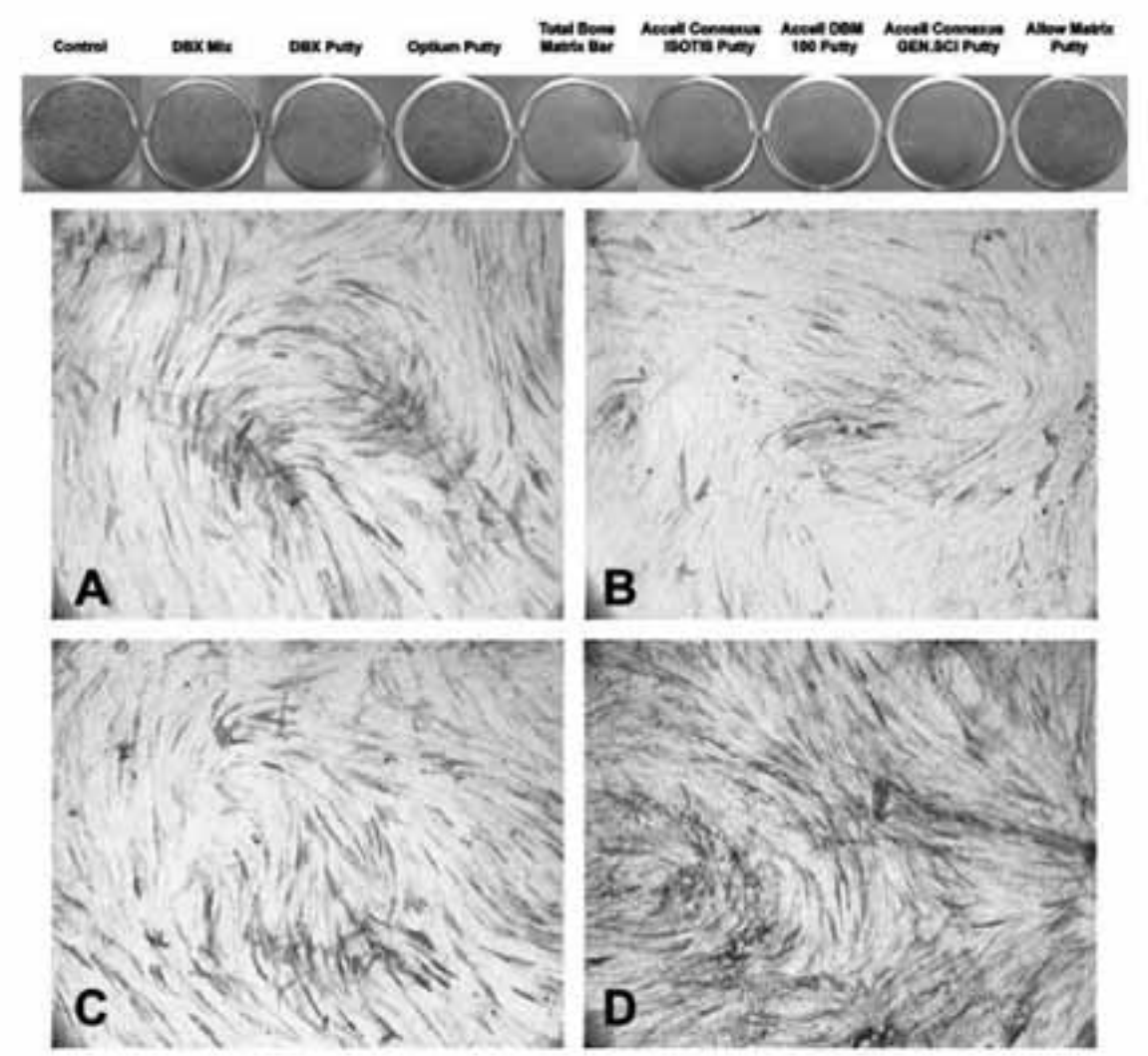

Figure 2. Osteoblast differentiation of hMSC cells treated with commercial available DBMs. hMSCs were cultured in a osteogenic media and on day 3 and 7 of culture cells were treated with demineralized bone matrix. Alkaline phosphatase staining was performed at 14 days. Photographs were taken under bright field microscope, Positive control (A), DBX mix (B), Optium putty bar (C), Allo Matrix putty (D). Each condition was set up in triplicate or quadruplicate per experiment.

of cell types including osteoblastic cells, fibroblastic cells, adipocytes and monocytes/macrophages.[1] hMSCs have the potential to differentiate to lineages of mesenchymal tissues, including bone, cartilage, fat, tendon, and muscle.[23] Migration of osteogenic cells is an important physiological event both during bone healing and bone remodeling. Materials which can enhance this migration are known as osteoinductive.

The need for quantification of osteoinductivity of DBM products has been recognized by both orthopaedic surgeons and the industry itself.[36] Efforts to quantify the BMP content in DBM products have been reported by several authors recently.[17, 25, 26, 37] Since performance of DBM is typically measured by bone formation in the athymic animal model, Honsawek and Zhang et al. both described an ELISA *in vitro* assay and correlated this with the animal model of bone formation.[11, 37] They found that protein measurements of BMP-4 levels in DBM correlated with improved bone formation in an *in vivo* model in matched human DBM donor paired specimens. However, it can be argued that this *in vitro* model does not measure the osteoinductive properties of the DBM products. The chemotactic (osteoinductive) properties of commercially-available DBMs have not been reported to our knowledge. In the present study, we investigated the osteoinductive properties of eight different commercially-available DBMs in two different *in vitro* experiments: human mesenchymal stem cell (hMSCs) differentiation to osteoblasts lineage, and chemotactic migration of hMSCs.

We were able to demonstrate that these DBMs have different osteoinductive effects on hMSCs. Of the eight DBM products tested, only Accell DBM100 putty, Total Bone Matrix bar and Optium putty had a positive effect. The chemotactic response to Accell DBM100 putty was significantly greater than that of Total Bone Matrix bar or Optium putty. Therefore only three of the eight DBMs tested were demon-

strably osteoinductive, and five DBMs were not osteoinductive in our testing.

In the second part of this study, we used hMSCs to assess whether commercial DBMs stimulate osteoblast differentiation *in vitro* by measuring alkaline phosphatase levels. In previous studies, Kasten et al. were able to use hMSCs to compare three resorbable biomaterials for cell penetration into the matrix, cell proliferation, and osteogenic differentiation.[15] Our results demonstrate that cells treated with osteogenic media differentiated into osteoblasts (control), however only AlloMatrix putty seem to have an osteoinductive effect on hMSCs. The effects of DBM proteins depend on the differentiation status of the cells. Woo et al. showed that in undifferentiated hMSCs they increased cell proliferation without affecting cell differentiation whereas in more mature osteoblastic cells, DBM proteins positively increased differentiation.[35] These findings suggest that Optium putty and DBX mix have a moderate osteoinductive effect on undifferentiated hMSCs, but possibly a better effect on more differentiated cells. However DBX putty, Total Bone Matrix bar, Accell Connexus ISOTIS putty, Accell DBM100 putty and Accell Connexus GEN.SCI putty demonstrated no osteoinductive effect on undifferentiated hMSCs in our study. In fact, they may have a negative effect thereby inhibiting the differentiation of hMSCs to osteoblasts. Optium putty had a moderate effect in both migration and differentiation in undifferentiated hMSCs (Table 1). However, AlloMatrix putty is the only DBM that showed a strong osteoinductive effect on undifferentiated hMSCs, suggesting that AlloMatrix putty could be used in conjunction with bone marrow cells to accelerate bone regeneration. It should be noted that correlation of alkaline phosphatase activity in vitro with *in vivo* osteoinductivity has not been clearly established.[16]

Table 1

Treatment	Migration	Differentiation
Control	−	++
DBX mix	−	+
DBX putty	−	−
Optium putty	+	+
Total Bone Matrix bar	++	−
Accell Connexus ISOTIS putty	−	−
Accell DBM100 putty	+++	−
Accell Connexus GEN.SCI putty	−	−
Allow Matrix putty	−	+++

The clinical efficacy of DBMs has not been proven in any large prospective studies. Nevertheless, clinicians continue to use these products with increasing frequency due to the hope that they are as effective as autogenous iliac crest bone graft in treating bone defects and fracture nonunions. However, safety is an issue with DBM products as shown in a recent study demonstrating the nephrotoxicity of the glycerol component of these products.[3] In fact, the FDA has treated these products as bone graft and not drugs or devices. Therefore, no proof of efficacy has been required by the FDA until only recently. As of 2006, the FDA now requires that these products be shown to heal a drill hole in small animal models to prove their efficacy. Whether this method of determining efficacy is sufficient to translate to clinical efficacy in humans is certainly debatable. In our effort to develop an *in vitro* assay to compare the efficacy of several different DBM products, we have shown that several of these do not attract mesenchymal stem cells (ie. not osteoinductive) and in fact are anti-osteoinductive in a dose-dependent fashion. Though this was somewhat surprising, other studies also demonstrated inconsistent performance of DBM products. Peterson et al. compared Grafton Putty, DBX Putty, and AlloMatrix were compared in a spinal fusion model in the athymic rat.[22] Interestingly, the AlloMatrix group demonstrated no evidence of spinal fusion whatsoever. Grafton Putty treatment led to the best spinal fusion as determined by manual testing. However, the control used was a decorticated animal with no application of any graft. As pointed out by Einhorn, an inactivated DBM product would have been a better control since the fusion may have been assisted by the DBM acting as a scaffold (osteoconductive) rather than its osteoinductive properties.[6]

One of the shortcomings of our study was that the experimental findings *in vitro* were not correlated with *in vivo* findings in an animal model and most importantly, clinical findings in human patients. Large prospective clinical studies are still needed to validate the use of these products in human patients. Though similar studies have been done for products such as BMP-2, they are lacking for DBM products.

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Basic Science Research

An Electromyographic Assessment of the "Bear-Hug" — A New Exam for the Evaluation of the Subscapularis Muscle

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Abstract

Rotator cuff pathology is commonly seen in clinical practice. Bennett et al. (2001) demonstrated a 27% incidence of subscapularis involvement in routine shoulder arthroscopy.[1] Subscapularis tears typically originate at the upper margin of the tendon. The Lift-Off and Belly-Press tests are well established maneuvers for evaluation of the subscapularis, however these tests may not be optimal for assessing upper margin subscapularis tears. We sought to compare these traditional tests to the Bear-Hug by assessing electromyographic (EMG) activity of the internal rotators; including the two portions of the subscapularis. Twenty-one healthy male volunteers (age = 22.95 ± 3.71, mass = 73.48 ± 6.93, height = 68.76 ± 2.26) with no previous history of shoulder surgery or injury participated in the study. Fine wire and surface EMG of the glenohumeral joint internal rotators (i.e., upper and lower subscapularis, pectoralis major, and latissimus dorsi) were recorded performing five subscapularis tests (Bear-Hug at 0°, 45°, 90° of shoulder flexion; Lift-Off; Belly-Press). Peak EMG values were normalized to the maximum voluntary isometric contraction (MVIC) for each muscle. A 4 (muscle) x 5 (test) ANOVA with repeated measures on test were used to analyze peak EMG (% MVIC). Follow-up univariate ANOVA and Tukey post hoc analyses were performed for each of the five tests. The Bear-Hug performed at 45° shoulder flexion revealed significantly greater peak EMG in the upper (107.64% ± 63.52) and lower (85.75% ± 64.69) subscapularis compared to the pectoralis major (41.43% ± 25.42) and latissimus dorsi (20.32% ± 15.70; $p < 0.05$). The Bear-Hug performed at 90° shoulder flexion showed significantly greater peak EMG in the lower subscapularis (166.0% ± 132.71) compared to the upper subscapularis (97.23% ± 70.78), pectoralis major (50.63% ± 29.60) and latissimus dorsi (17.56% ± 13.64; $p < 0.05$). The Belly-Press test revealed significantly greater peak EMG in the upper (77.88% ± 53.233) and lower (71.82% ± 46.49) subscapularis compared to pectoralis major (18.49% ± 14.85) and latissimus dorsi (34.85% ± 27.73; $p < 0.05$).

The Bear-Hug examination performed at 45° of shoulder flexion and the Belly-Press test may be valuable diagnostic tools in the clinical evaluation of the upper subscapularis muscle. Also, the Bear-Hug at 90° of shoulder flexion may be a valuable diagnostic tool in assessing the lower subscapularis.

Introduction

Few clinical tests are currently used to evaluate the integrity of the musculotendinous unit of the subscapularis muscle. Gerber et al. described the Lift-Off test and Belly-Press test as reliable maneuvers to detect subscapularis injury.[2–3] The Lift-Off test is performed by placing the dorsum of the patient's hand on their back at the lower lumbar level, and inability of the patient to actively lift the involved hand off the back constitutes a positive test. The Belly-Press test requires the patient to place the palm of the affected extremity on the abdomen, just below the xiphoid process. The patient is asked to press upon the abdomen while maintaining the elbow in the coronal plane. Subscapularis weakness is indicated by any compensatory wrist flexion or inability to maintain elbow position in the coronal plane.

An electromyographic study reported by Tokish et al. revealed that the Belly-Press test selectively activated the upper subscapularis muscle significantly more than the Lift-Off test and the Lift-Off test activated the lower subscapularis muscle more than the Belly-Press test.[4] However despite the current evidence validating these clinical tests, they may be of limited value in patients who cannot bring the affected arm into the starting position required to perform each test due to restricted range of motion and/or pain.

Burkhart et al. recently described the "Bear-Hug test" in an attempt to detect relatively small tears of the upper subscapularis.[5] The test is performed with the palm of the affected arm held on the opposite shoulder while the elbow is held in a position of maximal anterior translation. The examiner applies an external rotation force perpendicular to the forearm at the wrist of the affected arm, as the subject attempts to hold the starting position through resisted internal rotation (Figures 1–3). A positive test results when the patient dem-

Figure 1. Bear-Hug at 0 degrees of shoulder flexion

Figure 2. Bear-Hug at 45 degrees of shoulder flexion

Figure 3. Bear-Hug at 90 degrees of shoulder flexion

onstrates difficulty in holding the hand on the shoulder with the applied external rotation force of the examiner or weakness when compared to the contralateral side. According to the senior authors of that study, this test has anecdotally been more accurate in diagnosing upper subscapularis tears upon arthroscopic evaluation than the other two standard tests. The authors proposed that as in the Belly-Press test, recruitment of the upper subscapularis fibers is increased with the elbow anterior to the midline in the sagittal plane.

The purpose of our study was to use electromyography to evaluate different variations of the Bear-Hug test compared to the Belly-Press and Lift-Off test, and to verify existing clinical data to see which exam is more for useful for the evaluation of the upper and lower subscapularis muscles.

Methods and Materials

Twenty-one healthy male volunteers (age = 22.95 ± 3.71, mass = 73.48 ± 6.93, height = 68.76 ± 2.26) with no previous history of shoulder surgery or injury participated in the study. Informed consent was obtained by all subjects, and research was conducted in accordance with our institution's Internal Review Board.

In all patients, a complete physical examination of both shoulders was performed, with specific attention to the integrity of the rotator cuff and any restriction of active and passive range of motion.

For each subject, fine wire and surface EMG activity of the glenohumeral joint internal rotators (i.e., upper and lower subscapularis, pectoralis major, and latissimus dorsi) were recorded while performing five tests to evaluate the subscapularis muscle (Bear-Hug 0°, 45°, 90°; Lift-Off; Belly-Press). Pre-gelled silver-silver/chloride bipolar surface electrodes (Noraxon USA, Inc, Scottsdale, AZ) were used to measure the muscle activity of the pectoralis major and latissimus dorsi. Surface electrode placement was made in line with the direction of the muscle fibers with a center-to–center interelectrode distance of approximately 25 mm, as described by Basmajian and Deluca.[6] Indwelling electrodes for the upper and lower subscapularis muscles were placed within the muscle substance as described by Kadaba et al.[7] Standard anatomic references for the placement of surface and indwelling electrodes have been previously described in other studies. Accurate placement of electrodes was confirmed by manual muscle testing of each individual muscle.

Peak EMG values were first normalized to the maximum voluntary isometric contraction (MVIC) for each muscle. The standard MVIC procedures and protocols have been previously reported by Decker et al.[8] and Hintermeister et al.[9]

EMG data was collected using a Noraxon Telemyo 8-channel, 12 bit-analog FM-FM electromyography transmitter (Noraxon USA, Inc., Scottsdale, AZ). Data was transferred to a receiver where raw EMG data was sampled, then transferred to a PC for analysis and integration using Myoresearch electromyography software (Noraxon, USA, Inc.)

The Lift-Off and Belly-Press tests were performed as previously described. Variations of the Bear-Hug test were performed with flexion of the shoulder at 0, 45, and 90 degrees in the sagittal plane with the palm of the tested arm on the contralateral shoulder.

Maximal EMG reference values were calculated for each muscle by using the average of four peak EMG signals and represented 100% MVC. Average EMG amplitudes were calculated during the middle 50% of each trial for all clinical tests and expressed as percentage of MVIC.

Group means and standard deviations were calculated from the three trials of EMG data (% MVIC). A 4 x 5 (clinical test by muscle) repeated-measures analysis of variance was used to determine muscle activation differences (% MVIC) within and between tests. Post hoc analyses were scrutinized with the univariate analysis of variance and Tukey post hoc method for each of the five clinical tests.

Results

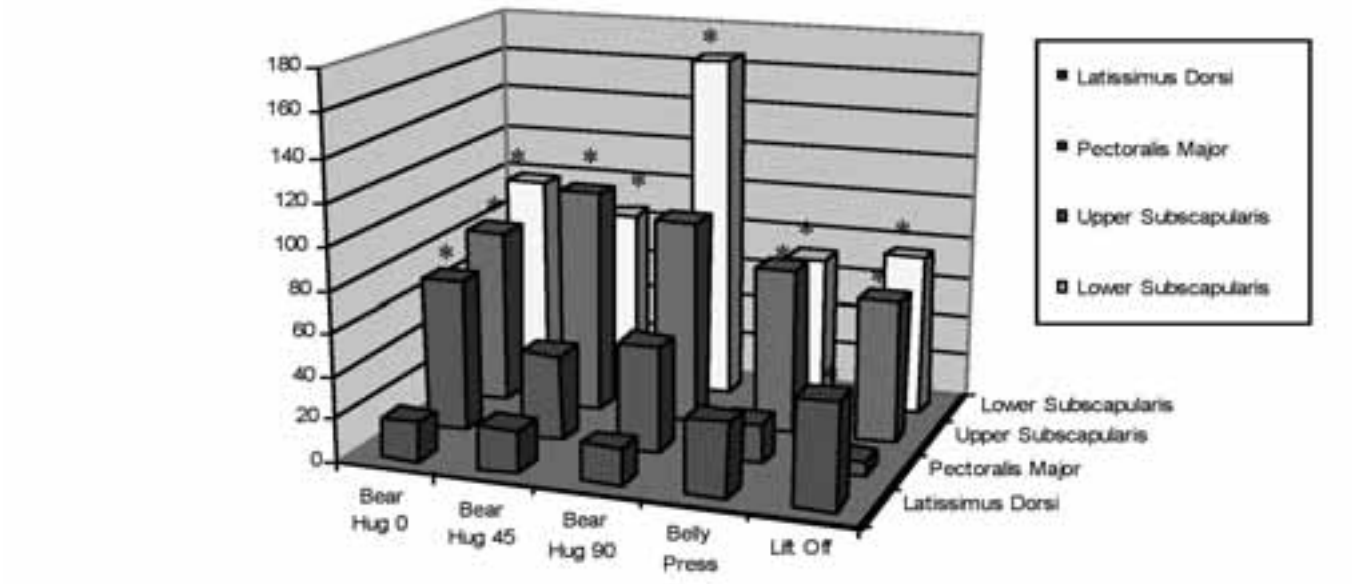
The results of integrated EMG data of active internal rotation are shown in Tables 1 and 2. The Bear-Hug performed at 45° shoulder flexion revealed significantly greater peak EMG in the upper (107.64% ± 63.52) and lower (85.75% ± 64.69) subscapularis compared to pectoralis major (41.43% ± 25.42) and latissimus dorsi (20.32% ± 15.70; $p < 0.05$). The Bear-Hug performed at 90° shoulder flexion showed significantly greater peak EMG in the lower subscapularis (166.0% ± 132.71) compared to upper subscapularis (97.23% ± 70.78), pectoralis major (50.63% ± 29.60) and latissimus dorsi (17.56% ± 13.64; $p < 0.05$). The Belly-Press test revealed significantly greater peak EMG in the upper (77.88% ± 53.23) and lower (71.82% ± 46.49) subscapularis compared to pectoralis major (18.49% ± 14.85) and latissimus dorsi (34.85% ± 27.73; $p < 0.05$).

Discussion

As the largest muscle of the rotator cuff and the main internal rotator of the humerus, the function and importance of the subscapularis is well understood. However, rotator cuff tears involving the subscapularis are less common and difficult to diagnose. Several studies have recorded the incidence of subscapularis tears. Smith et al. was likely the first to document the occurrence of subscapularis tears in a post mortem cases study of seven rotator cuff tears which showed that all tears involved the subscapularis.[10, 11] Frankel et al. found that subscapularis tears occurred in approximately 8% of patients with rotator cuff tears.[12] Depalma et al.[13] reported on the incidence of subscapularis tears in anterior shoulder dislocations and found that among 18 patients with fresh dislocations, 3 patients had subscapularis tendon avulsions, and in 12 patients, the subscapularis was stretched over the humeral head. Depalma also showed a 20% incidence of subscapularis tears in cadaveric dissections.[13] In arthroscopic studies, Bennett and Barth et al., in independent studies, reported a prevalence of subscapularis tears to be 27% and 29%, respectively.[1, 5] Various other studies have illustrated associated pathology with these tears including biceps tendon disorders, superior glenoid labrum tears, and fluid within the subscapularis recess or subcoracoid bursa.

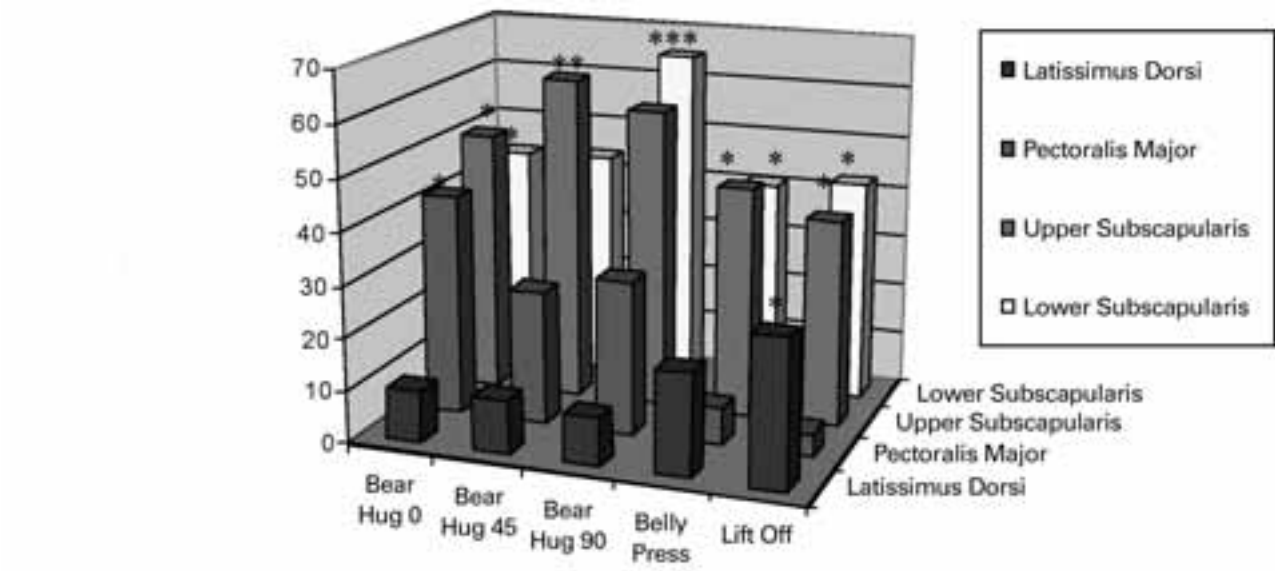
Technically we believe that variations of the Bear-Hug are easier to perform than the Belly-Press and the Lift-Off tests. Having the patient place the palm of the affected extremity on the contralateral shoulder, and asking them to hold this position while the examiner applies an external rotation

Table 1. Peak Average EMG Amplitudes (as % of MVIC)



*Indicates statistical significance

Table 2. Average EMG Amplitudes (as % of MVIC)



*Statistical significance
**Statistical significance b/w upper subscap and lats
***Statistical significance b/w lower subscap and pec major and lat dorsi

force perpendicular to the forearm at the wrist provides a simple means to examine the subscapularis complex. It requires the least amount of active participation from the patient. The Belly-Press requires the patient to actively internally rotate the shoulder while maintaining the wrist in a neutral position. The Lift-Off may be difficult to perform secondary to additional pathology about the shoulder exacerbated by extremes of abduction and internal rotation.

Our work showed that when performing the Bear-Hug at 0 degrees of shoulder flexion, the latissimus dorsi was significantly less active than the other 3 muscles tested. The test did not single out the upper and lower portions of the subscapularis from the pectoralis major. We feel that this test at 0 degrees is not significant for specifically testing the upper or lower subscapularis muscles, nor did it show much differentiation between the subscapularis complex and the pectoralis major. However, the Bear-Hug at 45 degrees of shoulder flexion showed significantly greater activity in the upper and lower subscapularis than the pectoralis major and latissimus. Although there was no statistically difference between the upper and lower subscapularis, the upper subscapularis demonstrated 20% more muscle activity than the lower subscapularis. Currently we are not aware if this is clinically significant.

Tokish demonstrated that the Lift-Off had greater lower subscapularis activity, and we have discovered that the Bear-Hug performed at 90 degrees also shows significantly greater muscle activity of the lower subscapularis.[4] We therefore feel that it is a reliable test for lower subscapularis integrity. Barth et al. performed their Bear-Hug at 90 of shoulder flexion, and found that the test was the most sensitive test in their series for detecting subtle injuries to the subscapularis complex. They, however, concluded that the examination at 90 degrees of shoulder flexion better detected lesions of the upper subscapularis.[5] Our data showed greater activity in the lower subscapularis than the upper subscapularis at 90 degrees of shoulder flexion. This examination at 90 degrees may therefore evaluate a region of the extraarticular portion of the subscapularis, thus a region of the complex not readily examined at arthroscopy. Extensive involvement of the lower subscapularis in an injury to the subscapularis complex may therefore require an open rather than an arthroscopic surgical repair.

Clinically, we believe that this test may be easier to perform for the patient than the Lift-Off and therefore may be better able to evaluate pathology involving the subscapularis. Difficulty in performing the Lift-Off test may be related to other factors such as adhesive capsulitis, subacromial impingement, or glenohumeral arthritis. These conditions would not likely be symptomatic during the Bear-Hug maneuver.

The **Bear-Hug** at 45 degrees of shoulder flexion demonstrated that the upper and lower subscapularis complex exhibits significantly greater activity than the latissimus dorsi and the pectoralis major. This test best isolated the subscapularis as a unit. We did note, however, 20% greater activity in the upper compared to the lower subscapularis in the raw data, no statistical significance could be demonstrated. The ability to design a test to evaluate the upper subscapularis complex may be of some clinical benefit. DiGiovine et al. showed that in the late cocking/early acceleration phases of the pitching cycle, the upper subscapularis

complex exhibits greater activity than the lower subscapularis.[14] It is during these two phases of throwing that a number of injuries occur to both the shoulder and elbow (SLAP lesions, medial collateral ligament/valgus instability injuries to the elbow). Early detection via physicial examination and subsequent rehabilition/repair of the lesions may prevent further injury to the extremity, as well as facilitating faster return to competition.

The **Belly-Press** test showed upper and lower subscapularis activity significantly greater than pectoralis and latissimus dorsi. The upper subscapularis showed 8% greater activity than the lower; however this data was not statistically significant. Our results differ somewhat from the data obtained by Tokish.[4] He found in his study that upper subscapularis showed 32% greater activity with the Belly-Press as compared to the lower subscapularis. This may be due to the high variability involved with fine wire EMG.

The **Lift-Off** test demonstrated significantly less activity in the pectoralis major than all other muscles, however showed no difference among the upper and lower subscapularis, and the latissimus dorsi. As previously stated, we feel that not only does the Lift-Off not isolate the subscapularis complex, but it is a maneuver that may be difficult to perform in the presence of additional shoulder pathology.

Conclusions

A few different clinical tests have been described in the literature to evaluate the function and integrity of the subscapularis muscle. This report is an electromyographic study of the Bear-Hug, a new clinical diagnostic test for detecting subscapularis tears. We have also compared this new test to the Lift-Off and Belly-Press tests for the evaluation of the upper and lower subscapularis.

We conclude that the Bear-Hug examination performed at 45° of shoulder flexion and the Belly-Press test may be valuable diagnostic tools in the clinical evaluation of the upper subscapularis muscle. Also, the Bear-Hug at 90° of shoulder flexion may be a valuable diagnostic tool in assessing the lower subscapularis.

The Bear-Hug is an efficient and new clinical exam to assess the integrity of the subscapularis. Further investigations will be needed to assess its sensitivity and specificity in the diagnosis of upper and/or lower subscapularis tears. Correlation is also needed to compare the clinical findings to imaging studies and those seen at arthroscopic and open surgery.

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Basic Science Research

Dose-Response Relationship Between Reach Repetition and Indicators of Inflammation and Movement Dysfunction in a Rat Model of Work-Related Musculoskeletal Disorder

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Abstract

We have developed an *in vivo* rat model of work related musculoskeletal disorders of the upper extremity in order to determine the extent to which repetitive tasks cause motor decrements and inflammation in musculoskeletal tissues and systemically. This study compares the effects of high and low reach rates on serum and tissue inflammatory responses and on reach performance. Forty-seven rats reached repetitively for 2 hours/day, 3 days/week for 3–8 weeks at a high or low rate. Forelimb musculotendinous tissues were examined for macrophage infiltration, and proteins indicative of inflammation and injury (COX2 and hsp 72). Reach rate and abnormal movement patterns were recorded. Serum was assayed for IL-1 alpha and IL-1 beta. Tissue inflammation was evident by week 3 in the high repetition rats, and peaked in week 6. High repetition animals also experienced a 2-fold decline in reach rate, and strong emergence of a raking movement. Serum IL-1 alpha, but not IL-1 beta, increased in the high repetition group, but decreased in the low repetition group. This model provides evidence that both local and systemic inflammation and motor decrements increase with high repetition, responses that are attenuated at the lower rate. These findings support the use of risk reduction in WMSD prevention.

Introduction

Work-related musculoskeletal disorders (WMSDs) accounted for 30% of all injuries and illnesses with days away from work reported by US private industry in the year 2005.[1] Epidemiologic and field studies suggest that there is a relationship between the onset and severity of these disorders and the performance of highly repetitive and/or forceful tasks.[2–4] A clear relationship between the amount of exposure and the pathophysiological findings is still under investigation.[5, 6]

Repeated damage to musculoskeletal and neural tissues leads to macrophage infiltration.[7–11] Structural damage to most tissues results in an infiltration of phagocytic cells such as macrophages. Macrophages respond to tissue damage by upregulating production of cytokines. Cytokines released during the acute inflammatory phase, such as IL-1 alpha and IL-1 beta, mediate the proliferation and further infiltration of macrophages, which leads to inflammation, enhancement of nociception, and induction of pain-related proteins as well as direct tissue damage from phagocytosis.[12, 13] IL-1 also enhances the expression of cyclooxygenase-2 (COX2), an enzyme with an important role in the synthesis of prostaglandin E2.[14] Additionally, injuries such as ischemic damage and inflammation lead to increased cellular expression of the inducible members of a family of "heat shock" proteins, also known as chaperonins and stress proteins.[15]

Animal models of WMSDs are an ideal means to study the link between exposure to physical risk factors and resulting physiological and behavioral responses. We have developed such a model in the albino rat and have shown that performance of a highly repetitive reaching and grasping task leads to both localized and systemic inflammation and behavioral degradation.[5, 7–10] The purpose of this study was to compare the effects of high and low repetition exposures over an 8-week period on serum levels of proinflammatory cytokines interleukin 1 alpha (IL-1 alpha) and interleukin 1 beta (IL-1 beta) and on reach performance in a rat model of a repetitive reaching and grasping task with negligible force.

Materials and Methods

Animals. Fifty-seven adult Sprague-Dawley rats (age 12–14 weeks at onset of experiment; Ace) were used. Forty-seven rats were trained to perform a repetitive forelimb reaching and grasping task with negligible force for up to 8 weeks. Ten rats served as age-matched controls and did not participate in the performance of the task regimen. The experimental animals were food restricted so that they maintained ± 5% of full body weight as defined by weights of age-matched controls. Experiments were approved by the Temple University IACUC in compliance with NIH guidelines for the humane care and use of laboratory animals.

Immunochemical Analysis. Groups of 3–5 animals were euthanized at 3, 6 and 8 weeks of task performance using

Nembutol (120 mg/kg body weight) and perfused intracardially with 4% paraformaldehyde in phosphate buffer. Forelimb flexor muscles and tendons were collected "en bloc" and frozen sectioned. Tissue sections were treated with 3% H_2O_2 in methanol for 30 min, 1% Pepsin in 0.01N HCL for 20 min, and then blocked with 4% goat serum for 30 min. Tissues were incubated with monoclonal antibodies against ED1 (Chemicon; diluted 1:250 in PBS), cyclooxygenase 2 (COX 2; Cayman; diluted 1:2000 in PBS) and heat shock protein 72 (hsp72; Sigma; diluted 1:1000 in PBS) overnight at 40°C, then washed in PBS. Sections were incubated with horseradish peroxidase-conjugated secondary antibodies (Jackson Immuno; diluted 1:100 in PBS) for 2 hours. Bound antibody was detected using standard diaminobenzidene detection (Sigma).

For quantitative analysis, sections were analyzed as a thresholded cell count per mm^2 using the Bioquant image analysis system. Three adjacent fields were measured per tissue. For further explanation, see Barbe et al., 2003.[8] Findings were analyzed by one-way ANOVA across weeks for the high and low repetition groups. A p value of ≤0.05 was considered significant. Post hoc analyses were carried out using the Bonferroni method for multiple comparisons. Group means + SEM were calculated and graphed.

Behavioral Task. The rats were placed in operant test chambers for rodents (Med. Associates, VT). At one end was a portal fitted with a 1.5 cm wide and 2.5 cm long tube that sloped downward 10° with respect to the chamber floor and was located at the animal's shoulder height. Food pellets (45 mg, Biosource) were dispensed (Pellet dispenser, Med. Associates) every 15 seconds (high repetition group, HR, n=39) or every 30 seconds (low repetition group, LR, n=8) during the reach sessions. An auditory indicator (Stimulus clicker, Med. Associates) provided a cue that a pellet had been dispensed, thereby instructing the animal to attempt a reach. Animals performed the task for 2 hours/day, 3 days/week for 3–8 weeks. An observer using a hand-operated counter and reach distance criteria logged the number of reaches. Gross movement patterns were examined for deviations from normal movements comprising reaching in rats.[16] Reach rate was defined as the average number of reaches performed per minute and was analyzed by repeated measures analysis of variance (ANOVA) across weeks of task performance for the high and low repetition groups. From a previous study (Barr et al., 2000), two distinct alternative reach movement patterns were defined as scooping in which the semi-open forepaw is placed over the food pellet and the pellet is dragged along the bottom or side surface of the tube and "scooped" into the mouth. Raking is an inefficient extreme of scooping in which repeated unsuccessful attempts to contact the food pellet with the semi-open forepaw result in repeated back and forth movements that resemble a raking motion. These behaviors were noted as present (>1/minute) or absent (<1/minute) at the end of each task week. They are expressed as the percentage of animals in which the behaviors were present.

Protein Isolation and Cytokine Analyses. Serum IL-1 alpha and IL-1 beta levels were examined in rats that had performed the task for 0, 6 and 8 weeks (n=3–8/group). Blood samples were collected from the heart, centrifuged, serum aspirated and total protein determined using BCA-200 protein assay kits (Pierce). Fifty µl aliquots were utilized for measuring IL-1 alpha and IL-1 beta using enzyme-linked immmunosorbant assay (ELISA) kits (Biosource International, Camarillo, California) according to the manufacturer's protocol. ELISA data were normalized to mg protein. Each sample was run in triplicate and data are presented as mean pg/µg total protein + 1 SD. Serum levels of IL-1 alpha and IL-1 beta protein were analyzed by one-way ANOVA across weeks for the high and low repetition groups. A p value of ≤0.05 was considered significant. Post hoc analyses were carried out using the Bonferroni method for multiple comparisons.

Results

Macrophage Infiltration. There were increased numbers of ED1 immunoreactive (IR) macrophages in distal regions of the forelimb flexor tendons and their surrounding loose connective tissues of high repetition (HR) rats in weeks 3, 6 and 8 (Figure 1). The numbers of ED1-IR macrophages peaked at week 6 of performance of the HR task. Increased numbers of ED1-IR macrophages were also found within flexor muscles and in the radiocarpal ligaments (data not shown). No significant increases in ED1-IR macrophages were seen in the low repetition (LR) rat tissues (Fig. 1).

COX2 and hsp72 Expression. At 3 weeks in the HR rats, cyclooxygenase-2 (COX2) immunoreactive (IR) cells were present in the connective tissues surrounding muscles and tendons of the distal forelimb (data not shown). By 6 weeks, COX2-IR cells were present in distal forelimb muscles, tendons and radiocarpal ligaments. Several COX2-IR cells were double-labeled for ED1 indicated that activated tissue macrophages are producing the COX2 protein. COX2-IR cells increased above control levels in the untrained distal forelimbs of these same HR rats, suggestive of a systemic inflammatory response. There were no COX2-IR cells in the musculotendinous tissues of the control animals.

Heat-shock protein-72 (hsp72) IR cells increased substantially over control levels by 3 weeks in lumbrical myofibers collected from HR rats (data not shown). By 4 weeks of the HR task, hsp72-IR cells dramatically increased in tendons and flexor muscle bellies of the distal forelimb. There were no hsp72-IR cells in the tissues of control animals.

Motor Performance Changes. The mean reach rate for the HR group in week 1 (baseline) was 8.27 reaches/minute ± 0.66 SEM. There was a significant decrease in reach rate at the end of week 5 to 6.82 reaches per minute ± 0.66 SEM (p=0.0028, n=31) and week 6 to 6.12 reaches per minute ± 0.51 SEM (p=0.0070, n=26). This decrease in reach rate

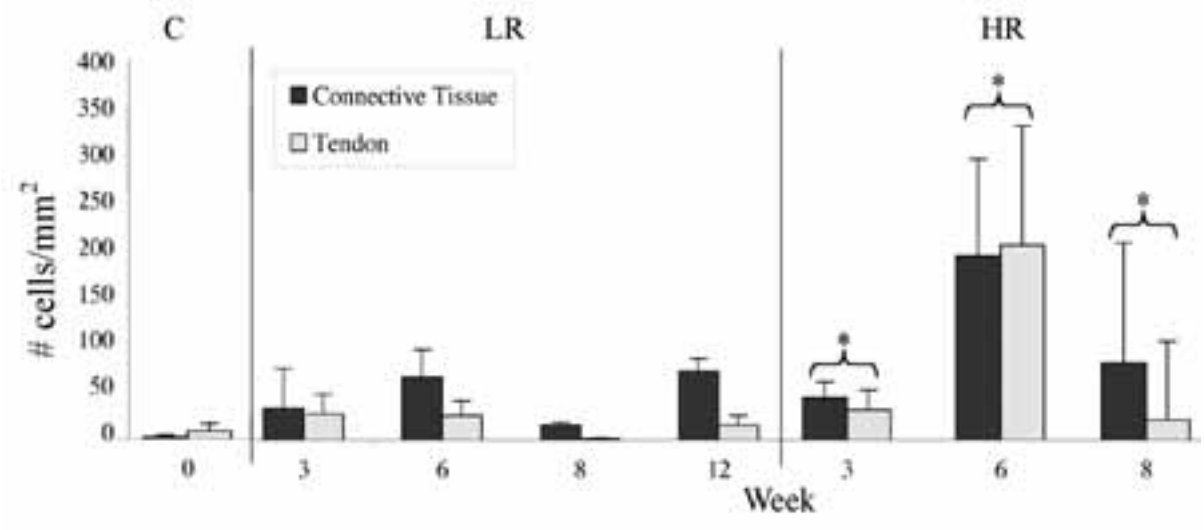

Figure 1. Number of activated macrophages in flexor forelimb tendons and in surrounding loose areolar and synovial connective tissues in rats that performed either the low repetition (LR) or the high repetition (HR) tasks. *p=0.01 compared to control rats (C).

continued through week 7 (5.96 reaches per minute ± 0.51 SEM, not significant) and was followed by an increase toward baseline to 6.90 reaches per minute ± 1.18 SEM in week 8 (not significant) (Barbe et al., 2003; Clark et al., 2003). For the LR group, there was no significant difference in reach rate across weeks (p=0.140) and the mean reach rate was 3.01 reaches per minute ± 1.03 SEM.

In the HR group, scooping emerged first as an alternative reaching pattern, peaked in 47% of animals by week five, and decreased to 26% of animals by week 8 (Figure 2). Raking continued to increase beyond 5 weeks and was present in 100% of HR group animals in weeks 7 and 8. In the LR group, the emergence of scooping also preceded that of raking (Figure 3). However, both alternative movement patterns were present in fewer animals in this group. Scooping peaked in 20% of animals by week 3; raking peaked in only 60% of animals in weeks 7 and 8.

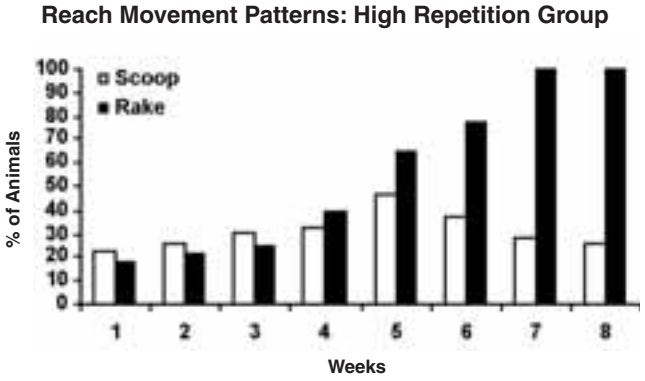

Figure 2. Alternative reach movement patterns in high repetition group (n=39, week 1; n=19, week 8). Data are expressed as the proportion of animals observed to perform the behaviors at a rate > once per minute. Note that 100% of all animals engage in raking by week 7.

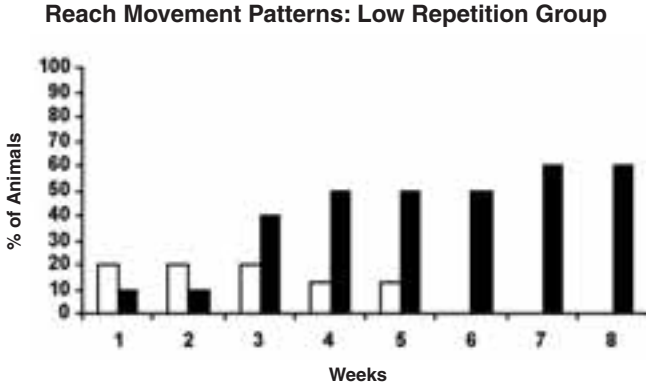

Figure 3. Alternative reach movement patterns in low repetition group (n=8, week 1; n=5, week 8). Data are expressed as the proportion of animals observed to perform the behaviors at a rate > once per minute. Note that 60% of all animals engage in raking by week 7.

Serum Cytokine Increases. In the HR group, serum levels of IL-1 alpha increased significantly by 27% above control levels in week 8 (Figure 4). The serum levels of IL-1 beta, however, were not significantly different over weeks of task performance (p=0.3987) (Figure 5). There was a trend toward decreasing levels of IL-1 beta in week 8 by 6% as compared to either week 0 or 6.

In the LR group, serum levels of IL-1 alpha did not change significantly over weeks of task performance (p=0.5103) (Figure 4). In contrast to the HR group, there was a trend toward a decrease in IL-1 alpha by 34% in week 8 as compared to controls. There was no significant change in serum levels of IL-1 beta over time in the LR group as compared to the control animals (p=0.3028) (Figure 5).

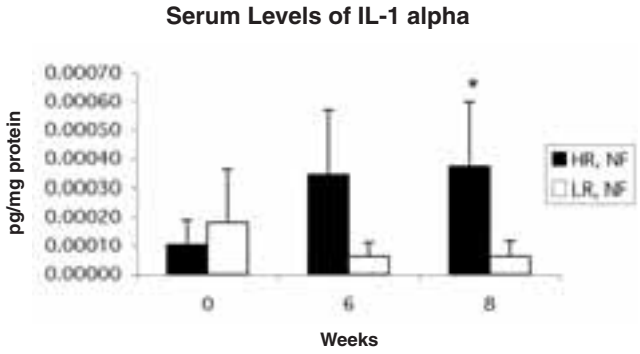

Figure 4. Serum levels of IL-1 alpha expressed as pg IL-1 alpha/µg total protein. HR, NF = high repetition group; LR, NF = low repetition group. IL-1 alpha is significantly greater at 8 weeks (p=0.0188) in the high repetition group as compared to controls (0 weeks) *p=0.01.

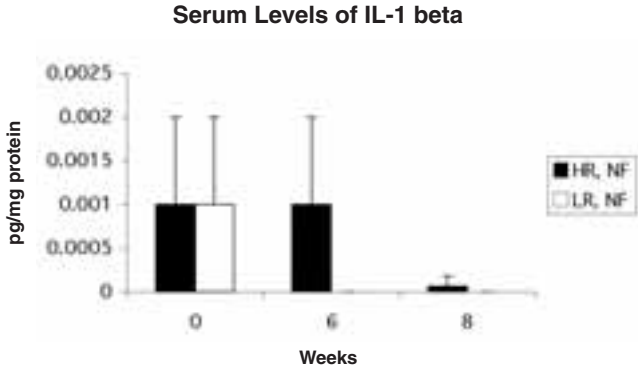

Figure 5. Serum levels of IL-1 beta expressed as pg IL-1beta/µg total protein. HR, NF = high repetition group; LR, NF = low repetition group. Note the decrease in IL-1 beta levels in the high repetition group in week 8 as compared to controls (p=0.3028). There were no measurable levels of IL-1 beta in 6 and 8 week in the low repetition group.

Discussion

Our results show a dose-response relationship between reach rate and both behavioral and physiological responses to a repetitive reaching and grasping task in rats. Even in a relatively low repetition reaching task with negligible force and a reach cycle time of 15 to 20 seconds, behavioral changes suggest physical discomfort that coincides with physiological evidence of diffuse and systemic inflammation. Increasing reach cycle time approximately twofold, to 7 to 10 seconds, further increased the magnitude of the behavioral response. The proportion of animals observed to scoop in week 5 was 2.35 times greater and the proportion observed to rake in week 7 was 1.67 times greater in the high repetition group as compared to the low repetition group. Although it is tempting to conclude from these results that the behavioral response to the performance of a repetitive task may linearly increase with increased repetition rate, the physiological response observed in these experiments suggest a more complex dose-response relationship.

Macrophages play an important role in modulating the inflammatory response. They secrete pro-inflammatory cytokines, e.g. IL-1 alpha when activated.[7, 8] IL-1 alpha induces further production and secretion of itself as well as production of other cytokines by macrophages. In this study, ED1-IR macrophages increased dramatically from 3 to 6 weeks of high repetition task performance in musculotendinous and ligamentous tissues of the trained and untrained palm and distal forelimb, and remained elevated in week 8 compared to control levels. This continued elevation of activated macrophages may have contributed to the significant increase of IL-1 alpha in serum in week 8 in the high repetition rats.

COX2 expression is highly induced by pro-inflammatory cytokines.[18] COX2 expression was detectable in endothelial cells, macrophages, and connective tissue cells after 3 weeks of task performance. This primary inflammatory mediator converts arachidonic acid from damaged cellular membranes into vasoactive prostaglandins, with free radicals as by-products.[14] These products of COX2 have been implicated in progressive vascular damage and cytotoxicity. The prolonged expression of COX2 seen in this injury model may worsen tissue pathology. Expression of hsp72 is a fundamental feature of cellular response to injury.[15] The increase of hsp72-IR cells in musculotendinous tissues of the HR rats indicate that these tissues were injured by the high repetition task regimen.

As has been previously shown, the primary tissue response to exposures to repeated, posturally constrained reaching movements in this rat model is inflammation resulting in reduction in physical performance.[5, 7–11] This inflammatory response, which includes an increase in IL-1 alpha in the high repetition group, occurs both locally in the tissues directly involved in task performance as well as at more distant tissue sites and in serum.[8] In this study, the serum levels of IL-1 beta decrease with task performance in both the high and the low repetition groups. IL-1 alpha increases in the high repetition group, but decreases in the low repetition group. These discrepancies are probably due to different mechanisms of the overall inflammatory response elicited by tissue injury. This response includes the production of both pro- and anti-inflammatory proteins. The IL-1 cytokines are pro-inflammatory proteins and are synthesized and secreted by osteoblasts, fibroblasts, myoblasts, synoviocytes, infiltrating monocytes and macrophages in response to mechanical tissue injury.[8, 17, 19] The macrophages also produce pro-inflammatory cytokines through autocrine mechanisms. Inflammatory mediators such as the cytokines also induce the production of anti-inflammatory cytokines as part of homeostatic repair and regeneration elicited after injury. Upregulation of these anti-inflammatory cytokines usually stimulates a down regulation of the pro-inflammatory cytokines.[20, 21] We know from our histological examination of tissues from these animals that several anti-inflammatory cytokines have been upregulated with task performance, at least in neural tissues.[17] We hypothesize that the net cytokine pro-

duction in the low repetition group allows for the maintenance of homeostasis through the resolution of an acute inflammatory response. The level of repeated incidents of mechanical injury to the tissues in the high repetition group, on the other hand, leads to a net production of IL-1 alpha, which is increasing in the blood serum and is indicative of a chronic and systemic inflammatory phase in which some pro-inflammatory cytokines remain elevated as task performance continues. Furthermore, animals in the high repetition group are not able to maintain a consistent reaching pace over time.

Our findings indicate that performance of highly repetitive tasks is associated with both local inflammation in musculotendinous tissues and systemic inflammatory responses that may result in decrements in task pace and movement efficiency. At higher repetition rates, a chronic inflammatory response may develop. These findings support a preventive approach to WMSDs in which exposure to risk factors, such as repetitiveness, are reduced through job redesign. They also suggest a role for the management of systemic inflammation in affected workers.

Acknowledgements

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Basic Science Research

Suprascapular Nerve Block Technique: An Anatomic Study

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Abstract

This study describes a technique to block the suprascapular nerve utilizing readily identifiable and reproducible anatomic landmarks. Using eighteen embalmed cadaveric shoulders, dissection allowed identification of the acromioclavicular (ACJ) joint, the superomedial angle of the scapula (SMA), and their bisection point. An eighteen gauge spinal needle was introduced into these three landmarks along the scapular plane. Blunt dissection was carried out to the level of the suprascapular notch and needle placement within the notch was confirmed. Total distance between ACJ and SMA, ACJ and the suprascapular notch needle (NCH), and between NCH and SMA were recorded. The average distance between the SMA and ACJ was 80.9 mm, between ACJ and NCH was 39.5, and NCH and SMA was 41.4. The average ratio of ACJ to NCH to the total distance was 0.49. The placement of the needle along the plane of the scapula was an average of 1.9 mm posterior to the line connecting ACJ and SMA. The needle was located within the notch 94% of the time. With the combined rise in popularity and the increasing indications for arthroscopic and reconstructive shoulder surgery, one can easily and reproducibly block the suprascapular nerve within its notch utilizing this method.

Introduction

With a rise in arthroscopic shoulder surgery and an increase in the number of outpatient procedures performed at surgical centers, a technique that provides an easily reproducible way of providing pre-operative, intra-operative, and/or post-operative pain relief could allow an increase in patient satisfaction and a decrease in both time to discharge and the number of patients requiring surgery in an inpatient setting. The suprascapular nerve is a branch off of the superior trunk of the brachial plexus and provides motor innervation to the supraspinatous and infraspinatus muscle, as well as sensation to the posterior shoulder capsule, acromioclavicular joint, subacromial bursa, and coracoclavicular ligament.[1] A suprascapular nerve block is an effective technique that allows for adequate pain relief for both arthroscopic and nonarthroscopic shoulder procedures.[1, 3, 4, 7]

Other nonoperative indications for the utilization of a suprascapular nerve block have included relief of pain and discomfort from advanced osteoarthritis of the shoulder joint,[6] an adjunct when combined with therapy for treatment of rotator cuff tears,[5] or analgesia allowing increased performance in home exercise programs for patients with frozen shoulder.[2]

The current method of blockade of the suprascapular nerve utilizes either a nerve stimulator or a field block where the needle is inserted 1 cm medial to the junction of the clavicle and scapular spine, directed toward the coracoid, until bone is reached, then walked anteriorly until no osseous resistance is felt.[1] This paper describes a novel method of suprascapular nerve blockade where the needle is introduced directly into the suprascapular notch, prior to the divergence of sensory branches, utilizing directly palpable osseous structures and an easy method of determining the site of needle introduction. No nerve stimulation conformation is necessary and a field blockade is still produced with confidence that the location of the anesthetic is within the suprascapular notch.

Methods and Materials

Eighteen nondissected, embalmed cadaveric shoulders of varying soft tissue coverage and body habitus were selected for study. The study group consisted of 10 male and 8 female cadavers, divided into 17 right shoulders and 1 left shoulder. The acromioclavicular joint (ACJ) and the superomedial angle of the scapula (SMA), Figure 1, were palpated through the overlying skin and the center position of these landmarks was determined. An 18 gauge spinal needle was inserted perpendicular to the scapular plane to mark the location of these landmarks. Visually, the distance between these two landmarks was bisected, and a third spinal needle was introduced at this bisection point, directed slightly posterior until the scapula was palpated. The needle was then gently walked in an anterior direction, perpendicular to the plane of the scapula until no bony resistance was felt, the needle was then withdrawn by approximately 1 mm and left in place. This third spinal needle (NCH) was our intended injection site for the nerve block.

Utilizing a caliper and ruler, the distance between the ACJ and SMA were measured and recorded. The distance between

Figure 1. Location of needle placement. ACJ = acromioclavicular joint, SMA = superomedial angle of the scapula.

the ACJ and NCH as well as the distance between NCH and SMA were also determined. A line connecting the ACJ and the SMA needle was then drawn and the location of the NCH needle off of the line in an anteroposterior direction was recorded with the posterior direction recorded as a positive value. Dissection was then performed to verify the location of the spinal needles, repeat the above mentioned measurements, and record whether the NCH needle was truly located within the suprascapular notch. Each measurement (ACJ to NCH, NCH to SMA, total distance, and anteroposterior location) both prior to and after dissection was determined three times using a caliper by two authors (KSM and MWR). An average value was then recorded for each measurement.

Results

The average distance between the SMA of the scapula and the ACJ measured 80.9 mm (range 74 mm to 100 mm), with the average distance from the ACJ to the NCH and the NCH to the SMA as 39.5 mm (range 26 mm to 55 mm) and 41.4 mm (range 34 mm to 54 mm) respectively. The ratio from the ACJ to NCH distance divided by the total distance from ACJ to SMA was 0.49 (range 0.33 to 0.55). This measurement indicated that the location of the notch needle was approximately 49% of the way between the ACJ and the SMA.

The anteroposterior location of the NCH needle off of a line drawn in the plane of the scapula from the ACJ needle to the SMA needle was 1.9 mm in the posterior direction (range 6 mm posterior to 8 mm anterior). The NCH needle was located within the suprascapular notch in 17 out of 18 shoulders, for a 94.4% success rate. The results indicate that if a needle is introduced at approximately half of the distance between the SMA and the ACJ, approximately 2 mm behind a straight line connecting these two points, the needle with be within the suprascapular notch 94% of the time. All data is summarized in Table 1.

Discussion

The use of a suprascapular nerve block has already proven its effectiveness as a provider of pain relief in both surgical and non surgical settings. However, a more anatomic method of nerve blockade would allow confident usage of this technique without a need for nerve stimulation to confirm placement. The orthopaedic surgeon can perform this technique, either within the operating room or the office, with confidence that it will effectively block the suprascapular nerve.

The method described utilizes two bony landmarks that reside on the same osseous structure that rotate with the scapula regardless of the position of the upper extremity. Therefore, the positioning of the limb within space will not change the distance between these two structures or the location of the suprascapular notch. Also, the superomedial angle and the acromioclavicular joint are readily palpable, subcutaneous structures which are easily felt on patients with even the most challenging of body habitus. By placing the thumb on one structure and the middle finger of the same hand on the other, the physician can easily visually bisect these two structures and introduce a spinal needle slightly posterior to a line connecting these two fingers.

The relative dangers of this technique are the introduction of the needle within the anterior structures of the thorax (subclavian artery, subclavian vein, and lung fields); however, by inserting the needle slightly posterior to the bisection point, and then walking the needle in an anterior direction until no resistance is felt, we feel that the technique is safe. First, if the starting point is too posterior, the only structure that will be violated is the supraspinatous muscle. Second, the depth of the spinal needle to the bony architecture can be noted, in this manner when the needle is slowly walked anteriorly, when bony resistance is lost at the needle tip, the physician will know how deeply to introduce the needle. With these points in mind, the likely event of damaging the anterior structures is reduced.

A field block of the nerve is still performed, introducing a large volume of local anesthetic to the area of the notch, (we typically use between 10-20 cc) so if the needle is one of the few not located directly within the notch, it is in close proximity and allows the volume of anesthetic to affect the nerve. Aspiration prior to injection of the anesthetic is recom-

Table 1. Cadaver Data

Total Distance (mm)	ACJ to NCH (mm)	NCH to SMA (mm)	Ratio ACJ to NCH/ Total Distance	Anteroposterior Needle Location (mm)	Needle Located Within Notch
75	41	34	0.547	4	Yes
88	43	45	0.489	2	Yes
75	36	39	0.480	2	Yes
75	38	37	0.507	0	Yes
78	26	52	0.333	5	No
74	35	39	0.473	2	Yes
74	34	40	0.459	3	Yes
75	36	39	0.480	–1	Yes
74	38	36	0.514	6	Yes
79	37	42	0.468	0	Yes
100	46	54	0.460	0	Yes
83	41	42	0.494	6	Yes
80	38	42	0.475	5	Yes
100	55	45	0.550	–2	Yes
89	47	42	0.528	4	Yes
76	38	38	0.500	–8	Yes
76	39	37	0.513	3	Yes
85	43	42	0.506	3	Yes
Average 80.9	Average 39.5	Average 41.4	Average 0.49	Average 1.9 Posterior direction is (+) Anterior direction is (–)	Average 17/18 within notch 0.94

mended as the suprascapular artery courses superior to the transverse scapular ligament.

Suprascapular nerve block allows immediate post-operative neurologic evaluation of the musculocutaneous, axillary and lateral antebrachial cutaneous nerves, structures that are at potential risk for iatrogenic injury given their proximity to the surgical site. An interscalene block causes paresthesias and paralysis to these nerves. Depending on the anesthetic used, a reliable examination may not be possible for hours post procedure.

The utilization of embalmed cadavers for this study should not negatively affect the data, given that the structures are osseous in nature and their location is not altered by cadaveric preservation or time to dissection. The width of the anatomic landmarks utilized within this study may vary the anteroposterior location of the bisection point (i.e. if the physician utilizes the mid portion compared to the anterior or posterior portion of the acromioclavicular joint), however, if the needle is introduced slightly posterior to the bisection point and slowly walked anteriorly, the notch should still be located, as demonstrated in our study with a 94% confidence.

We conclude that two easily palpable anatomic landmarks, the superomedial angle of the scapula and the acromioclavicular joint, can be readily palpated and bisected with a spinal needle to provide a safe, confident, and reproducible method for blockade of the suprascapular nerve in either a surgical or office setting.

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Case Report

Resorption of Iliac Crest Allograft and Failure of Fusion Mass Formation in Posterior C1-C2 Cervical Spine Fusion Using Recombinant Human Bone Morphogenic Protein 2

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Introduction

Bone morphogenetic proteins (BMPs) have been shown to increase fusion rates in animal models and in humans. Recombinant human BMP-2 (rh-BMP2), in particular, has been widely studied and has been approved by the Food and Drug Administration for use in single level anterior lumbar interbody fusions.[4, 6, 8–9, 13] Vertebral body bone resorption has been described after transforaminal lumbar interbody fusion supplemented with rhBMP-2. This paradoxical response is thought to be due to dose and environmental dependent effects on the bone formation-bone remodeling continuum.[15]

Off-label use of rhBMP-2 in the cervical spine has been associated with a high rate of soft tissue swelling and dyphagia in anterior cervical fusions.[2, 16, 19] To our knowledge, however, graft resorption with the use of rhBMP-2 has not been reported in the cervical spine. Here, we report a case study of posterior cervical spine fusion with tricortical allograft, local autograft, and rhBMP-2 (Infuse, Medtronic Sofamor Danek, Minneapolis, MN), which subsequently developed graft resorption.

Case Report

A 46-year-old male presented two weeks after a fall, with a chief complaint of suboccipital pain. The past medical history was significant for 25 pack years of cigarette smoking, and a shot gun injury to the head with retained shotgun pellets.

On physical examination, he had diffuse tenderness in the cervical spine and neck pain. Motor and sensory examination was intact. No upper motor neuron signs were present. Deep tendon reflexes were intact. Plain radiographic and CT assessment revealed a minimally displaced type II odontoid fracture (Figure 1). CT angiogram demonstrated no injuries to the vertebral arteries.

The patient underwent a posterior C1-C2 fusion with sublaminar wires, allograft, local autograft, and rhBMP-2 delivered in a collagen soaked sponge (Infuse, Medtronic Sofamor Danek, Minneapolis, MN) (Figure 2). The fusion bed was prepared in standard fashion, with exposure of 5mm of the ring of C1 on either side of the midline, and exposure of the posterior elements of C2, taking care not to disrupt the C2-C3 facet capsules or the posterior cervical attachments at C2. A small laminotomy was performed at C2-C3 and double strand titanium cables (Songer cables, Medtronic Neurological, Minneapolis, MN) were passed antegrade under C2 and C1. The cables were divided and one taken to either side of the midline. The graft bed was lightly decorticated with a high-speed burr and tricortical allograft with local autograft was wrapped in a collagen sponge soaked with rhBMP-2. The constructs were placed in the prepared bed and the cables tightened using the tensioner and crimped following radiographic confirmation of cable position. The dose of BMP used was 2.0 mg/cc, for a total dose of 40 mg, 20 mg per side. No drains were used and the wound was closed in the usual manner.

Figure 1. AP and lateral radiographs as well as sagittal CT scan showing minimally displaced Type II odontoid fracture.

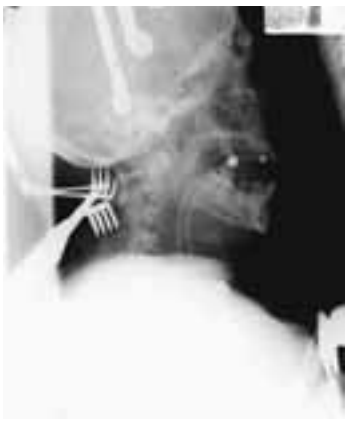

Figure 2. Intraoperative lateral radiograph with sublaminar wires and bone graft in place.

Postoperatively, a halo was placed for eight weeks, followed by a Miami J collar for an additional four weeks. Standard postoperative analgesia was used with Patient Controlled Analgesia (PCA) for 48 hours followed by transition to oral agents. The patient was discharged to home on postoperative day three.

The patient was followed in a standard manner. At no time postoperatively were oral steroids or non-steroidal anti-inflammatory agents used. Unfortunately, the patient continued to smoke 1–2 packs of cigarettes per day.

Over the ensuing months, the fusion mass, including the graft, subsequently resorbed (Figure 3). Fortunately, the fracture went on to uneventful, bony union.

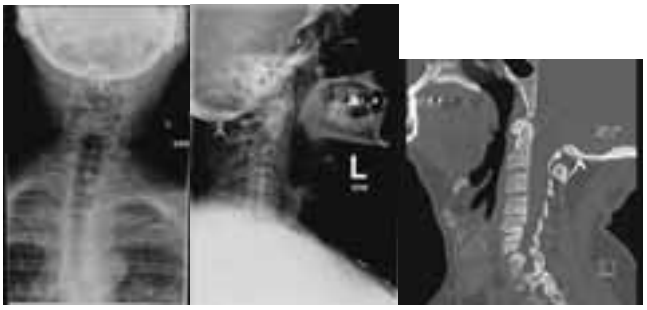

Figure 3. Postoperative AP and lateral radiographs as well as coronal and sagittal CT scans showing resorption of bone graft and stable union of odontoid fracture.

Discussion

The benefits of rhBMP-2 in animal models and in humans undergoing lumbar spinal fusion surgery have been well documented.[1, 3–7, 10–14, 19] Rh-BMP-2 is approved by the Food and Drug Administration (FDA) for clinical use only in anterior lumbar spine fusions, specifically in combination with a threaded titanium cage and collagen sponge carrier. Similar benefits have been suggested in posterolateral fusion trials, specifically using rhBMP-7.[14] It has, however, been observed in nonhuman primates that posterolateral lumbar fusions supplemented with rhBMP-2 using a collagen sponge carrier was not effective in achieving bony fusion.[12] It has been hypothesized that the compression of the sponge by the surrounding musculature found in the posterior spine inhibited bone formation, either directly or by local dilution.[1, 7, 18] In a primate model, Boden et al. demonstrated that rhBMP-2 delivered with a compression resistant carrier composed of biphasic tricalcium phosphate was associated with solid bony fusion.[6] Additionally, Dimar et al. reported an 88% fusion rate using a biphasic calcium phosphate compression resistant matrix with rh BMP-2 for posterolateral fusion, compared to a 73% fusion rate in the autograft group. The dose of BMP used in this study was higher than that in the commercially available mixture (2.0 mg/cc, for a total dose of 40 mg, 20 mg per side, versus 1.5 mg/cc, total dose of 12 mg, 6 mg per side).[10]

With regard to the cervical spine, Shields et al. recently reported an increased risk of significant hematomas, a higher rate of postoperative respiratory and swallowing difficulty, and extensive soft tissue edema with the use of rhBMP-2.[19] This was not noted by Baskin at al, in a similar study.[2] This complication may be the result of a difference in the local concentration of rhBMP-2 used. Shields et al. used a concentration of 2.1 mg/level of rhBMP-2 in comparison to the 0.6 mg/level used by Baskin et al. The local concentration of rhBMP-2 in the anterior cervical spine seems to play a critical role in the development of postoperative morbidity and is likely dose dependent.[2, 12, 17, 19–20] The precise etiology of this is unknown. It is our opinion that, in addition to the dose factors noted above, that the initial inflammatory response seen with the resorptive phase of BMP may be concentrated in the relatively closed compartment of the prevertebral space. This local mechanical effect combined with the exuberant vascularity of this area may result in the observed swelling.

To our knowledge, there are no randomized, prospective studies evaluating the use of rhBMP-2 in posterior cervical spine fusion surgery. The present report suggests that, like posterolateral fusions in the lumbar spine, there may be a compressive effect of the surrounding musculature that inhibits bone formation and precludes the use of a carrier composed of only collagen. In this instance as well, the collagen sponge may have been compressed against the relatively more rigid allograft. This mechanical effect, combined with the exuberant blood supply of the upper cervical spine may have led to a relative dilution of the BMP. With a relatively lower concentration, it would appear that the synthesis-remodeling balance might have been disrupted as well, resulting in resorption.

The resorption of the tricortical iliac crest allograft observed in this case may also be due to the inflammatory reaction initiated by the rhBMP-2. Vertebral body osteolysis has been observed in adjacent levels after anterior lumbar interbody fusions. One theory of the pathogenesis of this osteolysis involves the upregulation of local inflammatory mediators. When this occurs in combination with limitations of a collagen-only matrix unable to contain rhBMP-2, a local environment hostile to osteogenesis may result. Due to dilution, the remaining concentration of rhBMP may simply be insufficient to overcome the cellular environment of inflammation. In the absence of the secondary osteogenic phase, resorption is the net result.

Conclusion

We report a case of posterior C1-C2 fusion with sublaminar cables, allograft, local autograft and rhBMP-2, delivered by a collagen sponge. While the C2 fracture healed, the graft was totally resorbed. Possible explanations for this include local blood supply, compression of the collagen sponge and insufficient dosage of BMP, leading to an imbalance in remodeling osteogenesis, and subsequent resorption. Additionally, the patient continued to smoke postoperatively. A multiplicity of factors may have contributed to fusion failure. While all the possible mechanisms noted above are dis-

cussed in the literature, the contribution of each theory *per se* remains, in this case, speculative, and awaits further investigation.

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Case Report

A Femoral Head Fracture with Posterior Hip Dislocation While Playing Basketball in a 31-Year-Old Male: A Case Report

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Introduction

Sports-related injuries are a common presentation to both orthopaedic clinics and emergency departments. In a recent survey of sports- and recreation-related injuries in 1997–1999, Americans suffered an average annual estimate of 7.0 million sports-related injuries.[4] Basketball was the most frequently mentioned activity of those injuries, accounting for 14.4% of the total. Most sports-related injuries occurred to the extremities, with the lower leg and ankle most often affected in the lower extremity, and the hands, wrists or fingers commonly injured in the upper extremity. The most frequent diagnosis was sprains and strains accounting for 31.5%, followed by fractures accounting for 22% of the diagnoses. The major mechanisms of injury were struck by/against, fall, and overexertion/strenuous movement.[4]

Nearly 70% of all dislocations of the hip are associated with motor vehicle accidents.[14] Approximately 80% of all hip dislocations and 90% for sports-related hip dislocations are posterior. A significant force of more than 400 N (90 lb) is necessary to disrupt the joint to cause the femoral head to distract from the acetabulum.[14] This event occurs when a when the hip is flexed, adducted and internally rotated and the knee strikes the dashboard. A similar mechanism is seen in contact sports (football, rugby) when a running player is tackled and falls onto a flexed knee. Conversely, anterior dislocations occur with a flexed, abducted and externally rotated hip. Such injuries are more common in sports involving jumping and landing as in gymnastics or basketball.

This paper presents an uncommon sports-related injury: a femoral head fracture with posterior hip dislocation in a 31-year-old male basketball player who sustained this injury after landing on his right leg following a jump shot.

Case Report

A 31-year-old male presented to the emergency department complaining of severe right groin pain and a tingling sensation in his right foot after landing on his right leg after a basketball jump shot. Physical examination revealed severe pain with hip motion in all planes, and mild internal rotation of the entire lower extremity without noticeable shortening. Ankle dorsiflexion and extension of the hallux were weak, and sensation was intact in all foot dermatomes. Radiographs of the right hip demonstrated a posterior hip dislocation with fracture of the femoral head (Fig. 1A–C).

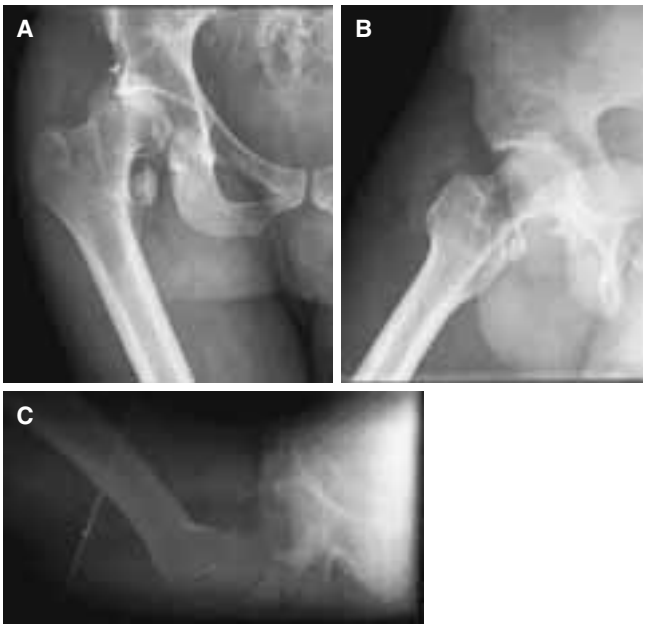

Figure 1. Emergency Room X-Rays showing the posterior dislocation of the hip with a femoral head fracture. Also note an avulsion fracture of the lesser trochanter.

Closed reduction under conscious sedation in the emergency department was unsuccessful. An emergent CT (Fig. 2A–E), performed prior to taking the patient to the operating room, showed the distal fragment to be perched on the posterior wall of the acetabulum without an acetabular fracture. The smaller proximal fragment was contained with acetabulum. The femoral head fracture line was in the sagittal plane involving approximately 20 to 25% of the weight-bearing surface of the femoral head.

The patient was taken to the operating room where closed reduction under general anesthesia was again unsuccessful. A posterior (Southern) approach to the hip was performed. Once reduced, the hip was re-dislocated posteriorly. The ligamentum teres was transected in order to reduce the proximal fragment to the femoral head. The fracture was stabi-

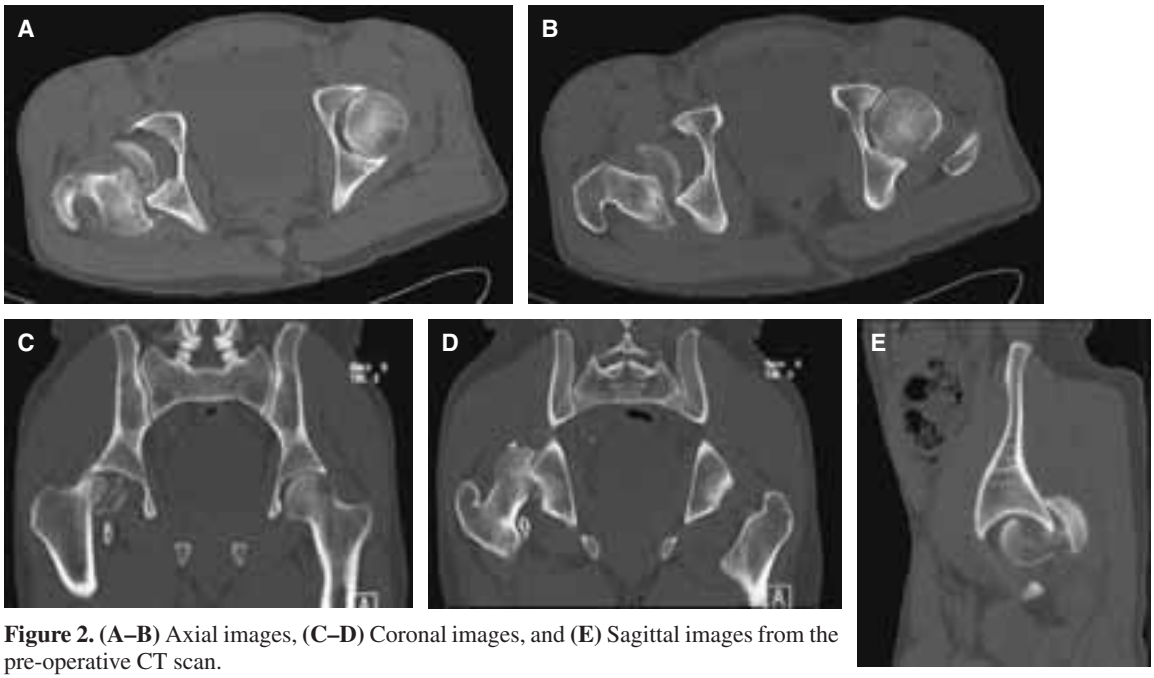

Figure 2. (A–B) Axial images, **(C–D)** Coronal images, and **(E)** Sagittal images from the pre-operative CT scan.

lized with headless compression screws (Zimmer, Warsaw, IN). The hip was then reduced and a capsular repair was performed (Figure 3A–C).

Post-operatively the patient was instructed to maintain hip dislocation precautions and to be nonweight-bearing on the right lower extremity. Physical examination revealed weakness of right ankle dorsiflexion and extension of the right hallux. The patient was fitted for a multi-podus boot to prevent Achilles contracture.

Figure 3. Post-operative X-Rays and axial CT images.

Three weeks after his injury, while bending over to tie his shoes, the patient described feeling a "pop" in his right hip and the immediate onset of severe pain. X-ray examination revealed a posterior hip dislocation with loss of fracture reduction (Fig. 4). Closed reduction in the emergency department with conscious sedation was successful. A post-reduction CT scan revealed a concentric reduction of the joint and fracture (Fig. 5A–B). Physical examination remained unchanged from the immediate post-operative period. The patient was fitted for a hip abduction brace and instructed to continue his nonweight-bearing status.

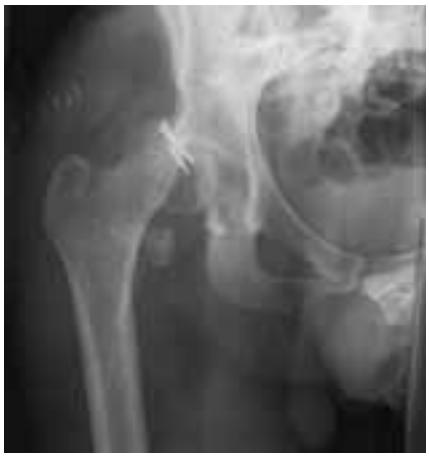

Figure 4. Patient represented 3 weeks after open reduction, internal fixation with a posterior dislocation and loss of fracture reduction.

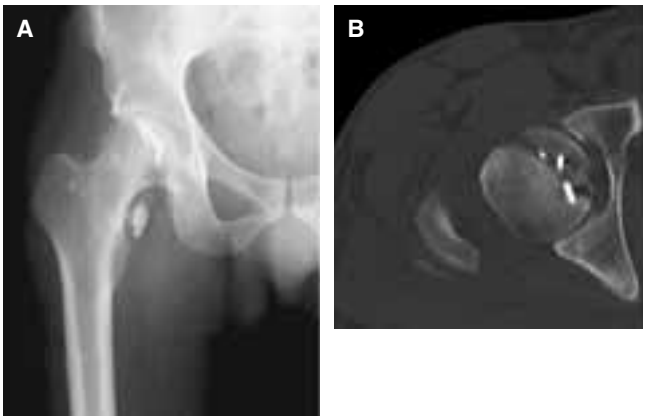

Figure 5. After closed reduction in the emergency department with conscious sedation. AP X-Ray and axial CT images showing adequate reduction.

Three months after injury, the patient was pain-free though he continued to have weakness of ankle dorsiflexion and EHL extension. Radiographs revealed fracture healing without evidence of osteonecrosis (Fig. 6A–B). The weight-bearing status was advanced to toe-touch, which the patient tolerated well with minimal discomfort. During the patient's one year follow-up evaluation, he reported an increase in pain both at rest and with ambulation. Radiographs showed evidence of fracture nonunion with joint space narrowing and sclerosis of the femoral head consistent with Ficat Stage IV osteonecrosis (Fig. 7).

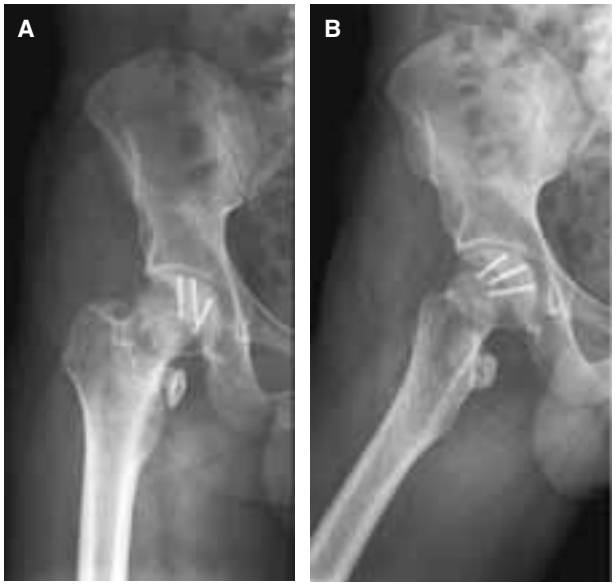

Figure 6. AP and lateral radiographs at 8 month follow-up demonstrating maintenance of reduction, fracture healing and no evidence of osteonecrosis of the femoral head.

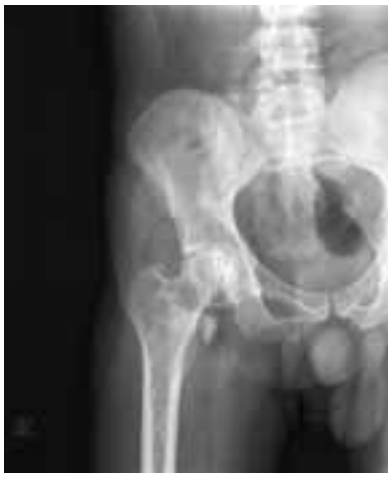

Figure 7. 1 year follow-up, increasing pain and radiographic evidence of osteonecrosis of the femoral head.

Fourteen months after the initial injury, a ceramic on ceramic total hip arthroplasty (Wright Medical Technology, Inc, Arlington, TN) was performed (Fig. 8). Intra-operative findings confirmed an ununited femoral head fracture with osteonecrosis. The patient did well with his arthroplasty for the first eight weeks and began ambulating post-operative day one without difficulty and was able to go home without needing therapy. However, further complicating his course, he fell onto his operative side causing a stable, nondisplaced periprosthetic fracture which was reduced and fixed using an AO plate with screws and cerclage cables (Synthes, Paoli, PA). The three-month evaluation after his last surgery reports the patient his ambulating without aids and is pain-free (Fig. 9).

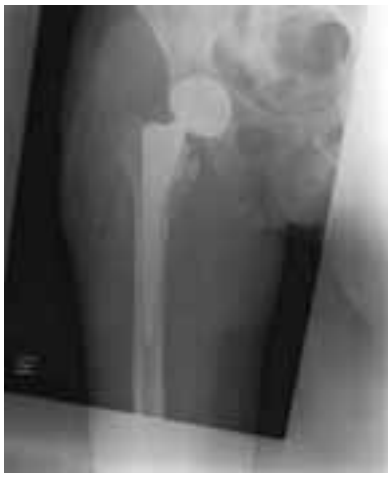

Figure 8. After ceramic on ceramic Total hip arthroplasty.

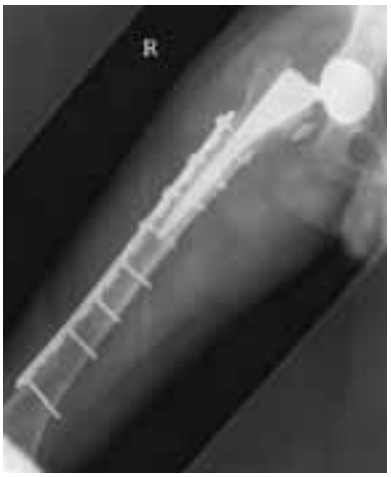

Figure 9. After open reduction internal fixation of late periprosthetic fracture.

Discussion

Traumatic dislocation/fracture-dislocation of the hip is an orthopaedic emergency with considerable morbidity and mortality. Unfortunately, the incidence of this injury is on the rise due to the increasing frequency of its most common cause — the high-speed motor vehicle accident. Hip dislocations, especially the posterior type, occur most often in motor vehicle accidents due to the high-energy impact of the flexed knee on the dashboard while the hip is flexed and adducted. This injury is less common in sports injuries, which are typically low energy in nature, but has been reported in basketball, rugby, football, gymnastics, skiing, biking, and jogging.[3] To our knowledge, a posterior hip dislocation with femoral head fracture has never been reported in the English literature in association with basketball. However, there has been one report of a posterior hip dislocation during basketball in the literature. Tennent et al. described a 22-year-old club basketball player who slipped after a jump shot resulting in a posterior hip dislocation. The injured hip was closed reduced within 2 hours after evaluation by an orthopaedic surgeon. The posterior hip dislocation caused a palsy of the deep peroneal branch of the sciatic nerve demonstrated by a foot drop. Unfortunately, the foot drop persisted following the reduction and at the 6 month follow-up.[12]

In 1970, Lamke studied traumatic hip dislocations and reported a 5.5% incidence due to sports-related injuries.[7] In a more recent review, Chudik et al. reported 2–5% of hip dislocations resulting from athletics. In sports-related hip dislocations, the flexed, adducted hip is at risk, with mechanisms including a forward fall on the leg with flexed hip and a hit from behind while the injured is on all fours.[3] In all studies reviewed, the posterior traumatic hip dislocation was the most common type, and men were more often affected than women.

There are several classification systems to describe both anterior and posterior hip dislocations and hip dislocations with associated femoral head fractures. The most commonly used classification system for hip dislocations was described by Steward and Milford after a study they conducted on fracture-dislocations of the hip published in 1954.[11] Their classification system divides anterior and posterior dislocations into four grades. Grade I includes a simple dislocation without fracture or with a chip from the acetabulum. Grade II includes a dislocation with one or more large fragments, but with sufficient socket remaining. Grade III is an explosive or blast fracture with disintegration of the rim of the acetabulum. Finally, Grade IV is a dislocation with a fracture of the neck or head of the femur.[9]

Within the literature, there is controversy over the classification system of posterior hip dislocations with associated femoral head fractures. Two different classification systems are well described in the literature: that described by Brumback and that described by Pipkin. In a study of the functional outcome of patients with femoral head fractures associated with hip dislocations by Stannard et al., it was reported that the Brumback classification system offered greater differentiation of the fracture types in contrast to the Pipkin classification system.[10] However, in another study by Marchetti et al., it was reported that the Pipkin classification system was a more accurate predictor of outcome compared to the Brumback classification system.[8] In that study, patients with Pipkin types 1 or 2 had statistically significant better outcomes when compared with those sustaining Pipkin types 3 or 4. This case used the Pipkin classification to describe the injury, which is as follows. Type I is a posterior dislocation with femoral head fracture caudad to the fovea capitas femoris. Type II is a posterior dislocation with femoral head fracture cephalad. Type III is a type I or II with associated fracture of the femoral neck while Type IV is a type I or II with associated fracture of the acetabular rim.[9] The patient in our case report sustained a Pipkin type II hip fracture-dislocation.

Although all studies reviewed agree that early diagnosis and prompt reduction are keys to good long-term outcomes, a definitive treatment strategy has yet to be determined for posterior hip dislocations. The treatment options can be broadly classified into two categories, closed reduction and open reduction. Bauer et al. wrote that the decision of which approach to utilize depends on several factors, including the type of injury, the patient's age, the patient's preexisting health, and the concomitant injuries sustained.[1] Epstein performed a long term study of outcomes for posterior fracture-dislocations of the hip and reported satisfactory results in 12% treated with closed reduction, 63% treated by closed reduction followed by open reduction, and 63% treated by primary open reduction.[6] However, in a study performed by Sahin, 74% of those patients treated with closed reduction had very good or good-medium results compared to only 66.6% of those treated with ORIF.[9] Regardless of the method

used, all studies reported better long term outcomes with prompt, stable, and accurate reductions by any method.

The choice of a posterior surgical approach was at the discretion of the operating surgeon is this case to achieve open reduction of the dislocation and then fixation of the femoral head fracture. Controversy regarding surgical approach is evident in the literature as the risk of osteonecrosis and further damage to the blood supply of the femoral head is of great concern. Many argue against a posterior approach due to the theorized risk of further damage to the femoral head blood supply. McKee et al. in 1998 concluded after a retrospective study of irreducible posterior hip fracture-dislocations, that "it is generally difficult to reduce the hip from an isolated anterior approach."[15] After reduction through a posterior approach, the femoral head fracture can be fixed with the aid of internal rotation of the hip or, if necessary, through a separate anterior approach.[15] There is no mention of integrity of ligamentum teres in the McKee series or whether the ligamentum should be preserved or may be transected to aid in reduction. The intact ligamentum teres may provide the only remaining blood supply to the femoral head. Anatomic studies of the arterial supply to the femoral head by Wertheimer et al. concluded ligamental arteries are always present, but in approximately two-thirds of the specimens studied the arteries were of such small caliber (<200 micrometers) that it only supplies a small area of the fovea.[17]

Epstein, after a retrospective study of irreducible posterior hip dislocations with associated femoral head fracture concluded that an anterior approach is contraindicated as this may disrupt any remaining blood supply to the femoral head.[16] In their series 5 of 6 patients who were treated with an anterior approach resulted in osteonecrosis of the femoral head. With an anterior iliofemoral approach, the ascending branch of the lateral circumflex artery must be ligated and the capsule must also be incised to expose the femoral head, thus jeopardizing the blood supply.[16]

The outcome of this case was an ununited fracture most likely due to osteonecrosis of the medial femoral head fragment. It can be presumed that the transection of the ligamentum teres, done in order to achieve fracture fixation in this case, may have damaged the remaining blood supply to the medial fragment leading to the nonunion and collapse. This case may represent a contraindication against fixation via a posterior surgical exposure. A separate second anterior incision for fracture fixation may have resulted in a better outcome as the ligamentum teres could have been preserved. A second posterior dislocation 3 weeks after the initial injury and surgery indicates the instability of the hip and potentially further disrupted any remaining vascularity to the femoral head. A poor prognosis is associated with these severe injuries to the hip joint.

There are multiple long term complications that may occur after sustaining a traumatic hip dislocation. Osteonecrosis of the femoral head, as previously mentioned, is the most common. Posttraumatic arthritis of the hip joint, and neurologic injury have also been reported. In his follow-up, Sahin reported 16.1% of his patients developing late post-traumatic osteoarthritis of the hip and 9.6% developing osteonecrosis of the femoral head.[9] In this study, there was also a notable increase in incidence of these complications by delayed reduction, regardless of treatment type. This conclusion is confirmed in a different study in which 17.6% of the patients who experienced osteonecrosis of the femoral head underwent reduction in under 12 hours compared to 56.9% in those undergoing reduction after 12 hours.[2] Similarly, the percentage of patients with post-traumatic arthritis of the hip was reduced with anatomic reduction and removal of all bony fragments from the joint.[6]

This patient in our case had a sciatic nerve palsy, a common complication in these injuries, though in this case ultimately resolved. In a literature review performed by Cornwall et al., it was noted that the incidence of neurologic injury is roughly 10% in adults and 5% in children.[5] Cornwall also noted that at least 60–70% of patients sustaining neurologic injury as a result of traumatic hip dislocations regain nerve function regardless of treatment type. However, early rehabilitation prevents adverse outcomes as a result of the neurologic injury, such as equinus foot deformity.

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Review

First Carpometacarpal Arthroplasty: A Review of Treatment Options and Introduction of a Limited Incision Technique

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Abstract

Arthritis of the first carpometacarpal (CMC) joint of the hand is a common and often debilitating disease. Diagnosis can be readily made with history, physical exam, and radiographic evaluation. Patients with advanced disease who have failed conservative treatment modalities have multiple surgical options including ligament reconstruction, resection arthroplasty, silicone implantation, tendon interposition, or total joint arthroplasty. A limited incision tendon interposition and ligament reconstruction arthroplasty has been developed that involves a smaller dorsal incision with a novel approach to the trapeziometacarpal joint. This technique offers comparable results with less operative and post-operative complications given the more direct and minimally invasive approach necessary to reach the joint space.

Introduction

The carpometacarpal (CMC) joint of the first ray is a diarthrodial saddle joint comprised of two major articulations, the trapeziometacarpal articulation to the thumb and the scaphotrapezial articulation; as well as two lesser articulations, the trapeziotrapezoid and the trapeziometacarpal to the index finger, determined by the axis of thumb compression and function.[1] Given the myriad of activities that an average human undertakes, the CMC joint can be stressed repeatedly in its three main planes of movement: abduction-adduction, flexion-extension, and opposition. An individual with laxity of the ligamentous stabilizers of the CMC joint, namely the anterior oblique (volar beak) ligament and the dorsoradial ligament, can develop synovitis and wear of the cartilage of the CMC joint, with eventual degenerative changes including joint space narrowing, osteophyte formation, ligament attenuation, and eventual dorsal radial subluxation of the joint.[1, 2] During active pinch and release, dorsal enlargement of the hand is indicative of a translation of the metacarpal on the trapezium. If the volar beak ligament was eliminated, the dorsal translation and articular contacts were similar to the values obtained on a patient with known osteoarthritis of the carpometacarpal joint.[3] With continued use of the hand, the patient can then develop metacarpophalangeal hyperextension, which further increases pain while decreasing pinch strength and range of motion.

Diagnosis

The typical presentation is a female between 50–70 years of age, complaining of an insidious radial thumb pain that originally worsened with use, has decreased her ability to perform activities of daily living, decreased strength, and decreased dexterity. Activities requiring opposition, such as writing, opening jars, or carrying heavy objects between the thumb and fingers, are instigators of pain. Common complaints include an increasing weakness of pinch, progressing to stiffness and cramping with increasing stage. As time progress, the pain may exist at rest. A more atypical presentation can occur in patients in their third decade. Typical complaints include pain in the thenar eminence or cramping in the first web space resulting from joint laxity and synovitis of the trapeziometacarpal joint. This pain may radiate up the radial aspect of the forearm with activities such as writing. Regardless of the patient's age, the pain is often mitigated by rest and analgesics.

Physical examination often reveals a dorsoradial prominence of the thumb metacarpal base secondary to subluxation due to ligamentous laxity and the pull of the adductor pollicus longus muscle. There is tenderness to palpation at the trapeziometacarpal joint, as well as the scaphotrapezial joint in advanced stages of disease. Crepitus may often be felt and the "grind test" of the trapeziometacarpal joint will often produce pain. One can check for translation and ligamentous laxity of the joint with stabilization of the metacarpophalangeal and the interphalangeal joints. Pain with distraction of the trapeziometacarpal joint can help differentiate synovitis.

Radiographs of the CMC joint in the standard AP, lateral, and oblique view can help confirm suspicion of the diagnosis. A stress view of the joint, obtained by a 30 degree PA view centered on the thumbs while the patient presses the thumb tips together, can help assess the degree of joint space loss and subluxation of the trapeziometacarpal joint.[1, 4] One must be careful to make a diagnosis, not only radiographic evidence, but also by patient complaints and physical exami-

nation. Approximately 25% of women and 8% of men will develop radiographic evidence of degeneration of the CMC joint, yet most remain asymptomatic, and when questioned, only 28% of women with carpometacarpal and 55% with carpometacarpal and scaphotrapezial arthritis will admit to pain.[5]

The differential diagnosis of thumb CMC arthritis is broad in spectrum. Arthritic diseases, such as rheumatoid arthritis, rheumatoid synovitis, tenosynovitis, osteoarthritis, and septic arthritis; autoimmune diseases, such as systemic lupus erythematosus, psoriatic arthritis, Reiter syndrome, or scleroderma; inflammatory arthridities, such as gout and pseudogout; as well as gamekeeper's thumb, carpal tunnel syndrome, de Quervain disease, and trigger thumb must all be remembered during the evaluation process.[16]

Classification

There are two major classification systems for thumb CMC arthritis. The first system was developed by Burton, and utilizes clinical signs, patient symptoms and radiographs (Table 1). Stage I involves early degeneration of the trapeziometacarpal joint characterized by patient reported pain, a positive grind test, and ligamentous laxity, demonstrated by subluxation in a dorsoradial direction. Stage II demonstrates increased instability, chronic subluxation, and degenerative changes as evidenced by radiographs. Stage III is a further progression of the degeneration to involve the scaphotrapezial joint, with stage IV as either stage II or III with metacarpophalangeal joint changes.[6, 7] This classification depends on subjective symptoms and objective signs rather than pure radiographic changes, and makes large transitions from stage to stage in terms of degenerative changes on radiographs without subclassifying finer differences.

Table 1. Burton Classification System for Trapeziometacarpal Arthritis[6, 7]

Staging	Characteristics
Stage I	• Pain • Positive Grind Test • Ligamentous Laxity • Dorsoradial Subluxation of the trapeziometacarpal joint
Stage II	• Instability • Chronic Subluxation • Radiographic Degenerative Changes
Stage III	• Involvement of the scaphotrapezial joint or less commonly the trapeziotrapezoid or trapeziometacarpal joint to the index finger
Stage IV	• Stage II or III with degenerative changes at the metacarpophalangeal joint

The Eaton classification (Table 2) relies on radiographic changes only, removing the patient's subjective complaints and objective findings from the staging protocol. This is the staging system that, in our experience, is most useful clinically, and therefore will be the system utilized in the remainder of this paper. Stage I demonstrates a normal to slightly widened trapeziometacarpal joint secondary to ligamentous laxity or effusion, normal articular contours, and up to one third subluxation. Stage II is progression to involve a narrowing of the trapeziometacarpal joint and osteophytes or loose bodies less than 2 mm in diameter. Stage III is marked by a further decrease in the trapeziometacarpal joint space, subchondral cysts, sclerosis, osteophytes or loose bodies measuring 2 mm or more in diameter, and subluxation measuring greater than one third of the joint space. Once the degeneration involves the scaphotrapezial joint, stage IV is declared.[1, 6–8]

Table 2. Eaton Classification System for Trapeziometacarpal Arthritis[1, 6–8]

Staging	Radiographic Characteristics
Stage I	• Normal or slightly widened trapeziometacarpal joint • Normal articular contours • Trapeziometacarpal subluxation (if present up to one third of the articular surface)
Stage II	• Decreased trapeziometacarpal joint space • Trapeziometacarpal subluxation (if present up to one third of the articular surface) • Osteophytes or loose bodies less than 2 mm in diameter
Stage III	• Further decrease in trapeziometacarpal joint space • Subchondral cysts or sclerosis • Osteophytes or loose bodies 2 mm or more in diameter • Trapeziometacarpal joint subluxation of one third or more of the articular surface
Stage IV	• Involvement of the scaphotrapezial joint or less commonly the trapeziotrapezoid or trapeziometacarpal joint to the index finger

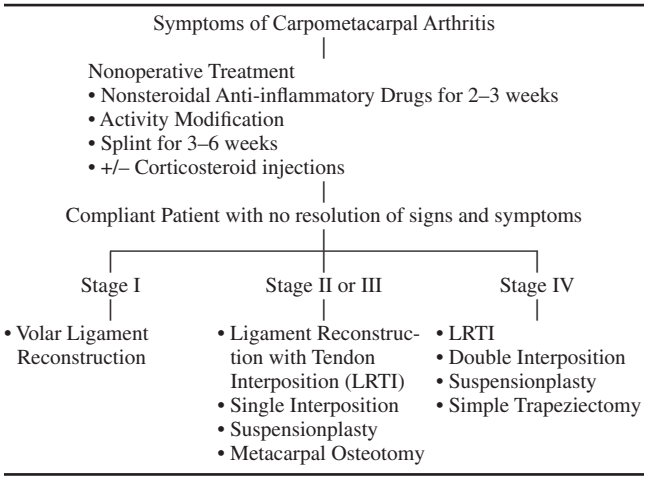
Non-Surgical Treatment Options

All patients, regardless of their initial staging, require a trial of conservative therapy prior to any operative considerations. This management consists of activity modification, joint protection, thenar muscular strengthening, NSAID use, and splinting with a long opponens, or thumb spica, for three to four weeks. If the patient must continue work, a short thumb-stabilizing splint can be utilized during work hours and switched for the thumb spica during off hours. If symptoms improve, the patient can be gradually weaned from the splint. If initial conservative management fails, physical therapy and corticosteroid injections into the CMC joint may be utilized for pain relief. Corticosteroid injections should be used with caution in patients with early stage disease given their potential to accelerate arthritic degeneration. Once conservative treatment has failed in a compliant patient, surgical intervention is the next step (Figure 1).

Surgical Options

Indications for surgical intervention in first CMC arthritis are similar to the indications for arthroplasty of most any joint: persistent pain, decreased function, instability, and failure of conservative management. Since modern surgical intervention for first CMC arthritis approximately five

Figure 1. Flow Diagram for Treatment of Carpometacarpal Arthritis[1]



decades ago there have been multiple techniques introduced. Surgical options vary with the stage and nature of the disease. In early stages, trapeziometacarpal ligament reconstruction[9, 10] has been shown to provide good symptomatic relief while also preventing further degenerative changes secondary to ligamentous laxity. For severe or late stage disease, some have advocated arthrodesis of the first CMC joint assuming that there is good mobility of the joints above and below it. In the light of the success of the various arthroplasty techniques, arthrodesis has fallen out of favor and is generally reserved for post-traumatic or rheumatoid arthritis limited only to the first CMC joint of the hand.

Arthroplasty techniques have ranged from simple partial or complete trapeziectomy to various implant and ligament interposition and reconstructions. These techniques have been generally indicated for Stage II or greater disease having failed conservative management.

Excisional arthroplasty involves the removal of the trapezium, and a substitution of the flexor carpi radialis (FCR) tendon into the empty space to help decrease the loss of joint space resulting from the surgically induced bone loss. The incision is made from the dorsal base of the thumb to the scaphoid tuberosity on the volar aspect of the wrist and continued proximally along the FCR tendon. The superficial radial nerve branches are protected, the volar capsule around the trapeziometacarpal joint is incised, and the abductor pollicis longus (APL) and extensor pollicis brevis (EBP) tendons are retracted dorsally after release of the first dorsal compartment. The radial artery is identified and protected at this step. Following this exposure, the trapezium is excised from dorsal to volar. Attention is then turned to the FCR tendon, with a 5 cm strip incised, being careful to preserve its distal attachment. The tendon is rolled into a ball and inserted into the space from the excised trapezium.[2] Results indicated an average relief of pain for six years, with a reduction of pinch strength and joint height of 30% and 50% respectively. This technique was later modified to excision of only one half of the trapezium to try to maintain joint height.[11, 12] While this technique removes the trapezium (entirely or partially) in an attempt to reduce the pain associated with the disease, complications include a reduction of pinch strength as well as a loss of joint height with full excision and a technique that does not correct pantrapezial involvement with partial excision.

Tendon interposition arthroplasty with ligament reconstruction was described by Burton and Pellegrini utilizing an incision extending from the base of the thumb to the interval between the APL and EPB. The trapezium is excised completely in stage IV disease or limited to the distal portion in stage II or III. The incision is then extended over the thumb metacarpal to expose the base and proximal shaft, which is debrided of all diseased articular cartilage. A small drill hole is created in the radial base of the thumb shaft and a segment of the distal portion of the FCR tendon is passed through. The tendon segment is approximately 10–12 cm in length and split distally until its insertion site. The tendon is tensioned with the thumb in traction and abduction, and then secured to the lateral periostium and itself. The remainder of the tendon is inserted into the defect from the excised trapezium and secured to itself and the deep palmar capsule. The incision is closed in layers, and the thumb is immobilized for four weeks, followed by four weeks of protected range of motion.[2, 13] Short and long term follow-up demonstrates favorable results with regards to pain, function, and satisfaction. There is an increase in grip and pinch strength from baseline, an increase in the ease of performing activities of daily living, and a subjective decrease in pain with a majority reporting no pain, or pain with exertional activities only.[13–15]

Double arthroplasty is an extension of the previous LRTI, and involves resurfacing of both the trapeziometacarpal and the scaphotrapezial joint. The thumb is approached from a volar curvilinear Wagner incision extending from midmetacarpal to the proximal pole of the scaphoid. The thenar muscles are elevated from their origins to gain exposure to the trapeziometacarpal and scaphotrapezial joint, the first metacarpal base, and the FCR tendon. After inspection of the scaphotrapezial joint through a radial arthrotomy, a long strip of FCR is obtained with its distal one third left attached to its insertion. The proximal two thirds is detached and set aside. The distal one third of the FCR is passed through the first metacarpal shaft to perform the trapeziometacarpal joint interposition as above. The detached tendon is folded to create a double layer and inserted into the space created by the distally resected portion of the trapezium in the trapeziometacarpal joint. The remainder of the tendon is layered and added to the scaphotrapezial joint for resurfacing. If additional tendon is necessary, the flexor carpi ulnaris (FCU) is utilized. The results demonstrated a stable joint with near normal range of motion (90%), an increase of grip strength of 30%, and a slight increase of pinch strength of 12%. Patients graded their results as excellent or good in 96% of the procedures.[16] Livesey reported similar results with resto-

ration of a more dynamic hand, increased strength, and better preservation of joint space as compared to reconstruction with a single palmaris longus tendon alone.[17]

Mennon introduced an arthroscopic interpositional procedure in 1996 in which the patient's thumb is hung from finger traps in slight pronation and flexion with five pounds of traction.[18] Two portals are made along the trapeziometacarpal joint line, one dorsal and one volar to the APL, separated by one centimeter. The base of the metacarpal, trapezium, volar ligament, and the dorsal ligament are examined, and following inspection, the articular surface of the trapezium is debrided using a round burr until cancellous subchondral bone is visualized. FCR, palmaris longus tendon, fascia lata allograft, or a Gortex patch can be used as interpositional material, the capsule is closed and the patient is placed in a thumb spica splint. Results from the procedure revealed no loss in range of motion, an average increase in pinch strength in 20 of 31 patients, no loss of pre-operative range of motion, and subjective reports of no pain or pain with exertional activities in 88% of patients.[18] Benefits of this technique involve a smaller incision, less extensive dissection, ability to examine the articular cartilage under surgical magnification; however, at this time, there is no reported technique to allow for ligament reconstruction arthroscopically.

Silicone implant arthroplasty showed initial promising results in terms of improved function and decreased subjective pain. The technique was described by Swanson[2, 6, 19] as involving a straight incision parallel to the EPL over the trapezium, with identification and protection of the superficial radial nerve branches. The capsule over the trapeziometacarpal joint is opened with careful attention not to damage the radial artery. The trapezium is removed and the base of the first metacarpal is squared. A hole is made in the intramedullary canal to receive the prosthesis that is secured with a tight capsular closure. Original results from the procedure demonstrated an increase in grip and pinch strength, range of motion, and decrease in subjective pain.[19]

Although initial results were excellent, long-term results yielded high incidences of subluxation, cold creep, silicone wear, synovitis and bony erosion. Creighton reported on 151 implants in 1991 and found a 56% incidence of scaphoid cysts as well as 74% metacarpal lucencies or cysts with evaluation suggesting silicone synovitis; however, the results did not correlate with clinical signs as 84% of patients reported satisfaction with the surgery.[20] Other long term studies demonstrate similar results with complications including prosthetic wear, radiographic evidence of loosening, and even prosthetic fracture.[21, 22]

Currently, the idea of total joint arthroplasty is in the new and experimental stages. In the past, there have been implants, such as the Ledoux, Niebauer, and de la Caffiniere, which were tried and have major limitations. These included silicone synovitis, high rates of loosening, and subluxation requiring revision with other materials.[23–27] Hemiarthroplasty with the Swanson titanium implant, and total arthroplasty with the Braun titanium and polyethylene prosthesis are still being investigated with reasonable results to date.[28, 29]

Limited Incision Technique

Our proposed technique utilizes a more limited incision, with a unique approach to the trapezium, which not only allows easier identification and excision, but also protects the radial artery and the superficial branches of the radial sensory nerve.

The hand is prepped and draped in the usual sterile fashion, utilizing a tourniquet preset to 250 mmHg and an extremity drape. A 2 cm V-incision, apex dorsal, is marked over the first CMC joint. 1% Marcaine is injected, the hand is exanguinated, the tourniquet is inflated, and the incision is created (Figure 2) with a 15 blade through the skin to the underlying fascia. Dissecting scissors are used to expose the APL and EPB tendons (Figure 3), the APL is retracted volarly while the EPB is retracted dorsally. The tendons are held in this position with a self-retaining retractor. This position also moves and protects the lateral branch of the superficial radial nerve volarly as well as the radial artery and dorsal branches of the superficial radial nerve dorsally, allowing full access to the joint without fear of injury to neurovascular structures.

Figure 2. Apex dorsal incision over first dorsal compartment.

Figure 3. Split the APL and EPB tendons.

The CMC capsule is capsulotomized with a Beaver #64 blade and dissected off volarly and dorsally for later repair. The full extent of the trapezium is identified (Figure 4), and morsilized with a small osteotome and mallet (Figure 5). Care is taken no to extend too ulnarly and damage the FCR tendon. The trapezium is then completely resected with a ronjeur and confirmed with palpation. The FCR tendon should now be visible at the base of the surgical field (Figure 6).

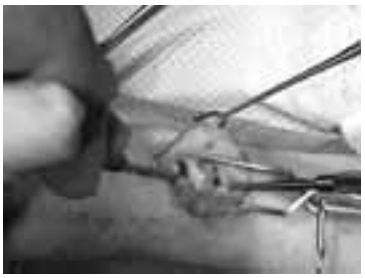


Figure 4. Expose the trapezium.

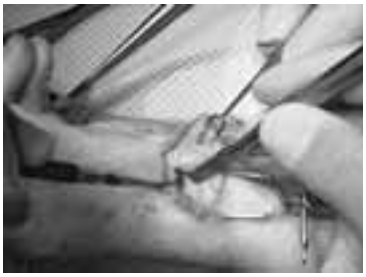


Figure 5. Morsilize and remove trapezium.

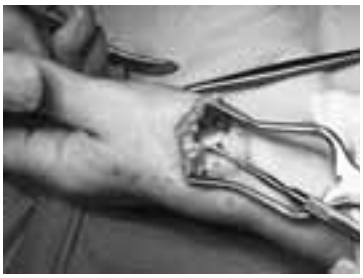


Figure 6. The FCR tendon.

Attention is then turned to preparing the first metacarpal for reception of the FCR tendon by drilling two tunnels, one through the metacarpal shaft, and the second through the dorsal aspect of the base (perpendicular to the nail bed) with a 3mm drill bit. The two tunnels should create a 90 degree angle, and they are cleared of remaining debris with a small courette with care to avoid destruction of the bone bridge just created (Figures 7–9).

The FCR tendon is now ready for harvest. The distal aspect of the tendon sheath is opened utilizing dissecting scissors and a tendon passer is inserted (Figure 10) and passed proximally. The volar aspect of the forearm is incised (approximately 1 cm) over the tip of the tendon passer and dissection is carried out to identify the FCR tendon sheath and tendon. The FCR proximal to the tendon passer is secured completely with a hemostat to prevent splitting of the tendon proximally. The ulnar half of the FCR tendon just distal to the hemostat, at the end of the tendon passer is incised and split distally. This ulnar half is inserted into the tendon passer and pulled distally with slow, constant pressure until it is split completely to its insertion at the base of the second metacarpal.

Figure 7. Maintain the thumb planar to your OR table.

Figure 8. Create a canal down the metacarpal shaft.

Figure 9. Your second hole should be perpendicular to the thumb.

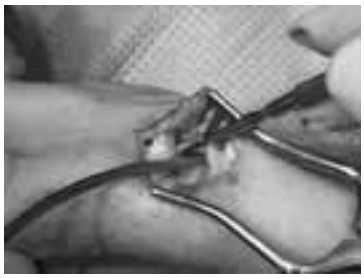


Figure 10. Retrieve the FCR tendon and complete the standard volar beak ligament reconstruction.

The free FCR tendon is now passed through the shaft and out the dorsal aspect of the first metacarpal utilizing the previously drilled passage. The tendon is tensioned, allowing the metacarpal to assume an abducted and slightly flexed position and secured in place with 3-0 Ethibond suture. The remainder of the tendon is wrapped around the intact FCR tendon and secured again with 3-0 Ethibond suture, creating a type of "anchovy." The site is irrigated, the capsule and subcutaneous tissue is closed with 2-0 Vicryl suture, and the skin is approximated with 5-0 Nylon suture. A thumb spica splint is applied and worn for two weeks.

Conclusion

Numerous techniques have been described in the literature to correct the common problem of basal joint arthritis. Multiple options can therefore be used depending on the surgeon's education, comfort level, and experience. Our repair incorporates the fundamentals of previously proven techniques utilizing reconstruction of the volar "beak" ligament and tendon interposition of the remaining FCR tendon to create an "anchovy" to help limit joint space collapse. Our approach, however, gives direct access to the trapezium, and effectively separates the overlying EPB and APL tendons to protect them during the procedure. This separation also manages to protect and remove the radial artery and superficial radial nerve branches from the operative field, allowing the surgeon to work unhindered and without fear of damage to these structures, once protected.

While this technique is utilized in our institute for management of all operative cases of carpometacarpal arthritis, it is not the only repair that is effective. This approach has yielded results that are comparable in short term follow-up (five years) in terms of function and subjective pain relief, as compared to the more traditional approaches and studies (study in progress). It is a tool for a general orthopaedic surgeon, as well as a hand specialist, to place in his toolbox to provide options for management of a fairly common arthritic complaint.

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Technique

Intramedullary Nailing After a Radial Shortening Osteotomy for the Treatment of Kienböck's Disease

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Introduction

Surgical options for Stage II and IIIa Kienböck's disease has been limited to revascularization of the lunate, proximal row carpectomy and radial shortening in order to preserve wrist motion. Revascularization has been met with mixed results and proximal row carpectomy results in decreased strength. In patients with a negative ulnar variance a radial shortening osteotomy has been supported by many. Koh et al.,[2] in a 10 year follow-up review, concluded radial osteotomy is a reasonable treatment option with clinical improvement in 96% of patients. Severe osteoarthritic changes and proximal migration of the capitate were avoided with their course of treatment.

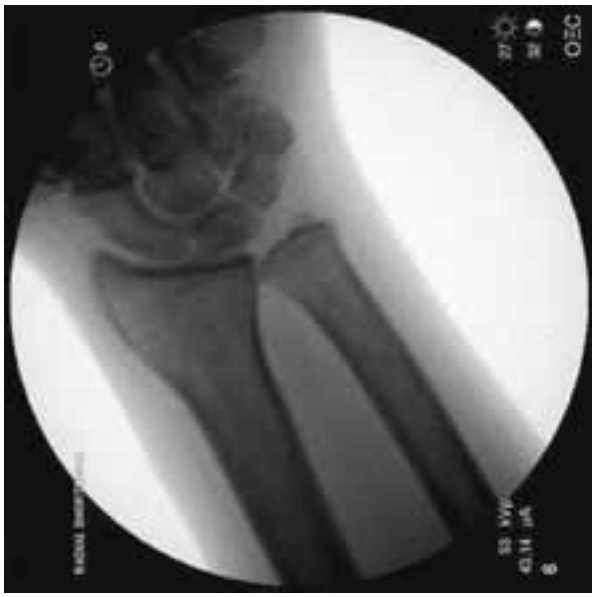

Figure 1. PA view demonstrating the negative ulnar variance.

A radiovolar approach for a radial recession osteotomy was described by Edward Almquist, MD through the flexor carpi radialis tendon sheath and elevating the pronator quadratus subperiosteally off the volar surface of the distal radius.[3] Almquist and Burns recommend a diaphyseal osteotomy to allow enough length of the distal fragment for fixation.[3] A 6-hole compression plate is positioned and the radius is marked for resection and rotational alignment. The distal-most screw is inserted and the markings are re-confirmed. The plate and screw are removed for the segmental resection to be performed with a sagittal saw. The plate is then reapplied and distal-most screw reinserted. The proximal screws are placed in a compression fashion.[1] With this protocol Almquist and Burns reported a satisfactory result in 11 of 12 patients at a 10 year follow-up with functional improvement including increased grip strength and wrist range of motion.[1]

Preliminary reports of intramedullary fixation of distal radius fractures with the Micronail (Wright Medical, Memphis TN) are promising, providing a stable construct and earlier initiation of post-operative rehabilitation. It has been this early success with the micronail for fracture fixation and malunion repair[4] by the surgeon in this series (JJ Thoder) that has led to its use for radial shortening osteotomy in the treatment of Stage II and IIIa Kienböck's disease.

Procedure

A longitudinal incision is carried out over the dorsal aspect of the radius from the level of Lister's tubercle distally for approximately 4 cm (Figure 2). It is carried down through skin and subcutaneous tissues. The interval between the first and third compartments is identified proximally at the level of the intersection, and then the second and third compartments distally to the level of the retinaculum. The retinaculum is not violated.

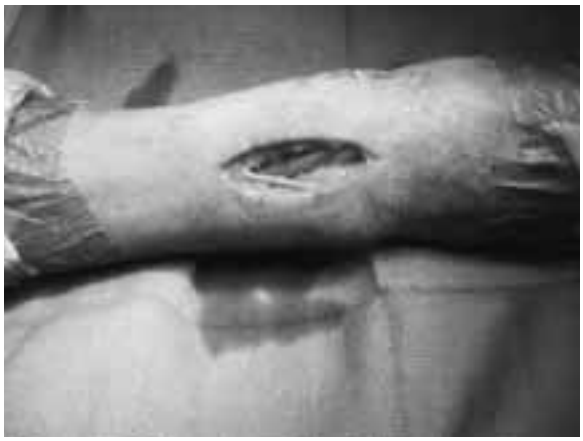

Figure 2. Clinical photos of dorsal incisions.

The dorsal aspect of the radius is identified and reverse retractors are placed on either side of it and with aid of mini C-arm the osteotomy site is selected at the metaphyseal-diaphyseal juncture. An oscillating saw is used to remove an appropriately sized wafer of bone from the radius, being sure to be perpendicular to both long axis of the radius and transverse axis of the radius in both cuts. The "lifesaver"-type portion of bone is removed by releasing it from its soft tissue attachments and radius is shortened by direct contact (Figure 3). The position of the radius is secured with percutaneous 0.062" k-wire on the dorsal ulnar corner of the radius through the 4th–5th dorsal compartment interval, across the osteotomy site to engage the volar cortex (Figure 4). Anterior-posterior and lateral radiographs are takes to assure that there is adequate shortening with good bony contact.

Attention is then directed toward the interval between the first and second dorsal compartment, where a separate 3–4 cm incision is made over the radial styloid and carried down through skin and subcutaneous tissue (Figure 5). The dorsal sensory branch of the radial nerve is identified and retracted out of harms way within the dorsal flap. The flap/bare area between the first and second compartments is identified at the level of the radial styloid and a guide pin is inserted with x-ray, confirming that the guide pin is in the midline of the shaft of the radius on the lateral projection (Figure 6). The reamer is used to enlarge the hole as well as a rongeur. Then, in a serial fashion, a trial size of rasps for the intramedullary nail of the radius are inserted (Figure 7). With a well fitting rasp inserted, anterior-posterior and lateral radiographs show this is an appropriate canal size and the holes are in a position that is safe in terms of remoteness from the osteotomy site itself, and the position of the osteotomy can be maintained (Figure 8).

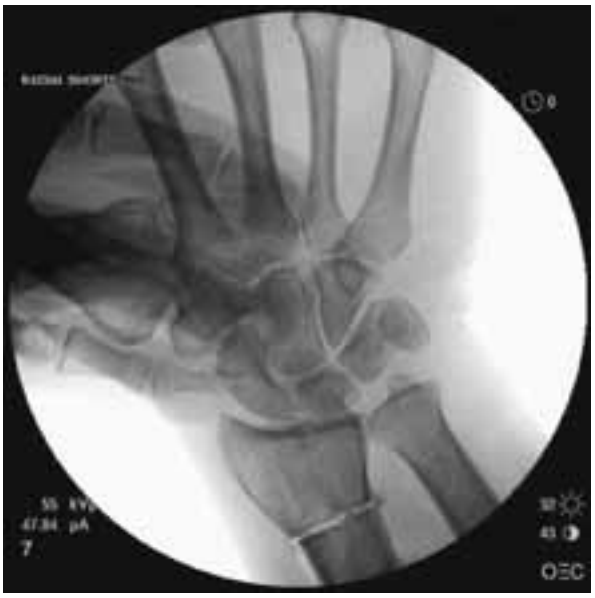

Figure 3. PA after the completed osteotomy.

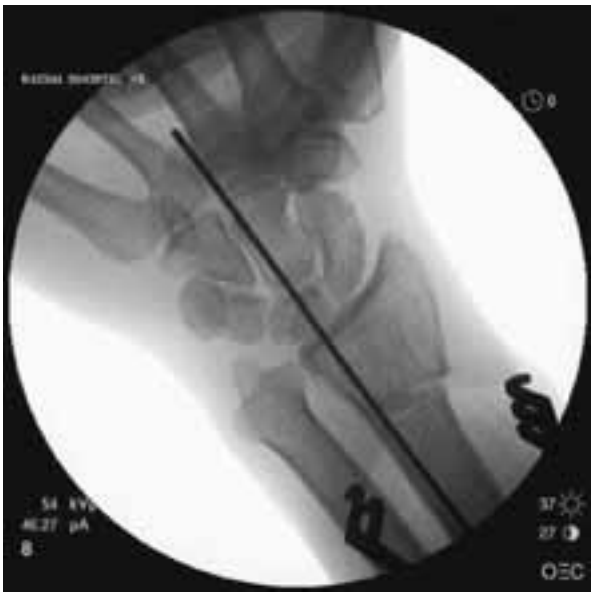

Figure 4A. PA view with ulnar-sided dorsally placed pin.

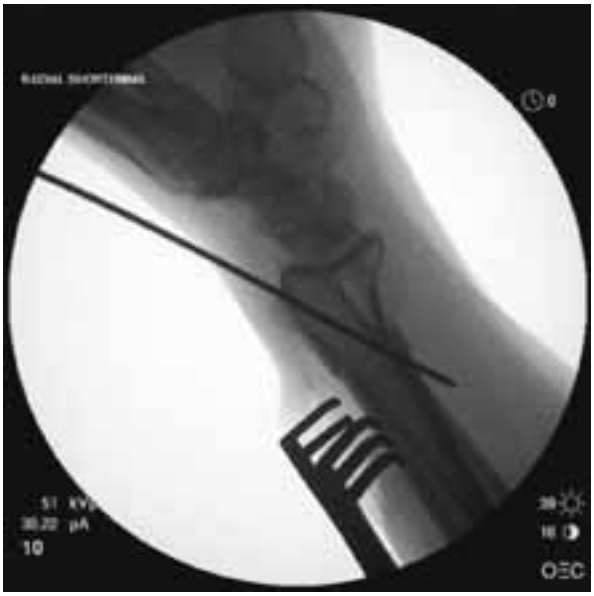

Figure 4B. Provisional fixation with dorsal pin.

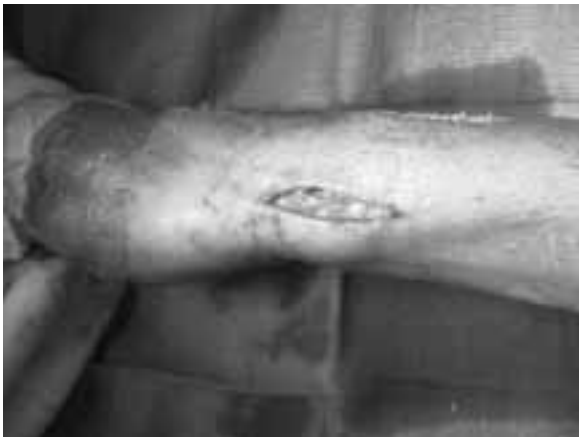

Figure 5. Clinical photo of radial styloid incision.

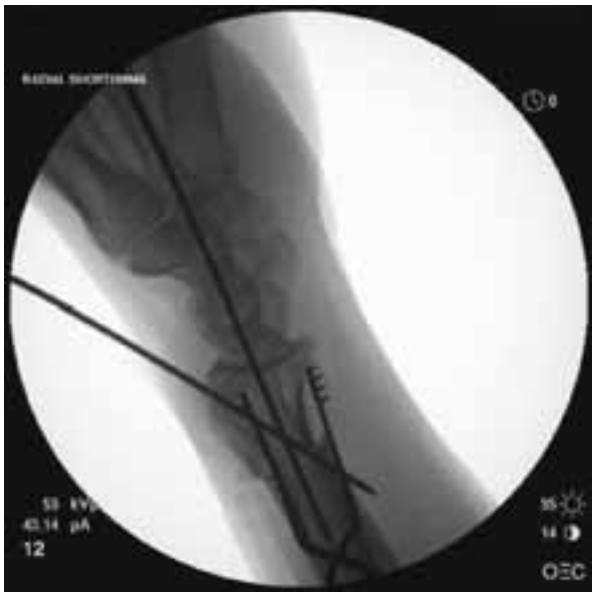

Figure 6A. After placement of the radial styloid pin.

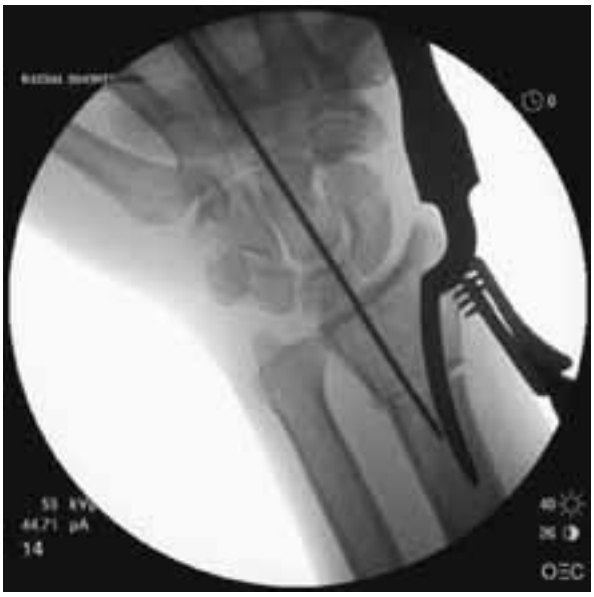

Figure 7. Placement of first rasp.

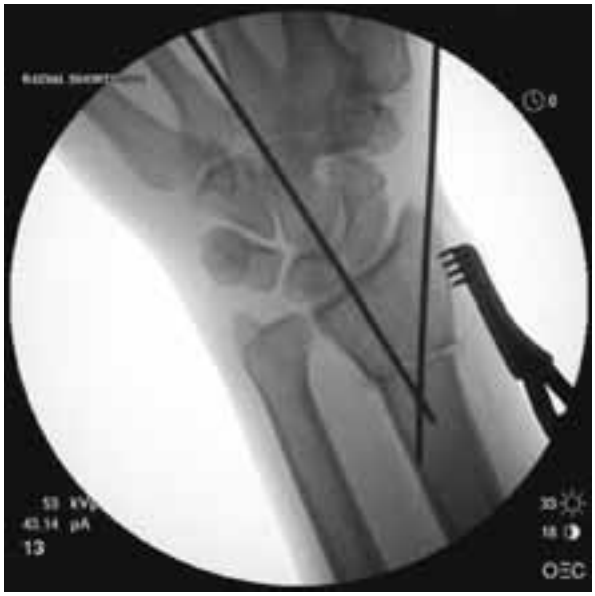

Figure 6B

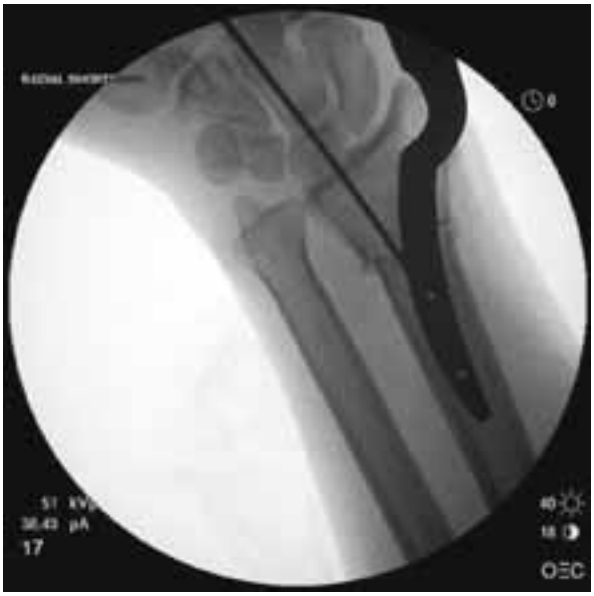

Figure 8. Final rasp with appropriate canal fit.

An appropriate sized Micronail is then assembled on the introducer and inserted. It is locked distally, radiographs are again checked to be sure that the osteotomy site has not shifted (Figure 9). It is compressed and the proximal locks are inserted. Anterior-posterior and lateral radiographs with the introducing device removed are taken again to be sure that the osteotomy site is secured, screws are of appropriate length and that there is no penetration of the distal radial ulnar joint (Figure 10). The wrist is checked to confirm that full flexion, extension, and full pronation and supination are maintained. Under fluoroscopy the wrist is passively flexed and extended to be sure that there is no motion at the osteotomy site. The dorsal cortex of the osteotomy site will be congruent.

Post-operatively the patient is maintained in a volar short arm splint for two weeks and then initiation of wrist motion after two weeks. The patient is instructed to maintain a non-weightbearing status for 6 to 8 weeks.

Discussion

Intramedullary fixation with the Micronail for displaced extra-articular and simple displaced fractures of the distal radius has been established as an effective treatment modality providing a rigid construct. The Micronail's use has been demonstrated in the correction of malunited fractures at our institution.[4] The rigid construct provided by the Micronail enables early rehabilitation and the hypothesized benefit of a

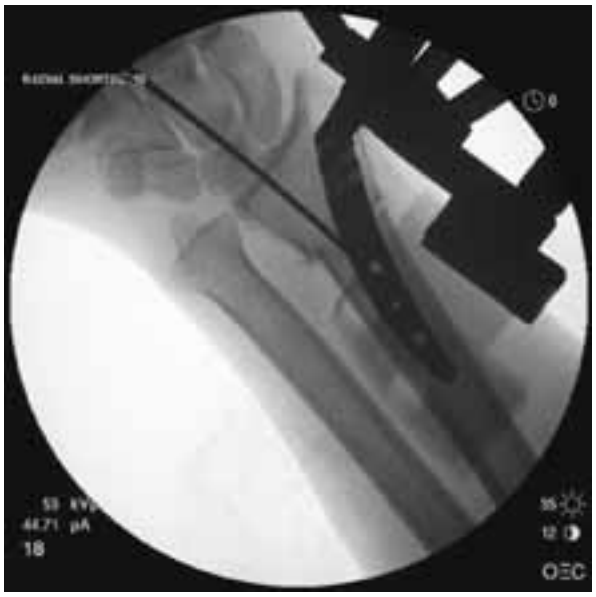

Figure 9. PA view after placement of the nail with insertion device.

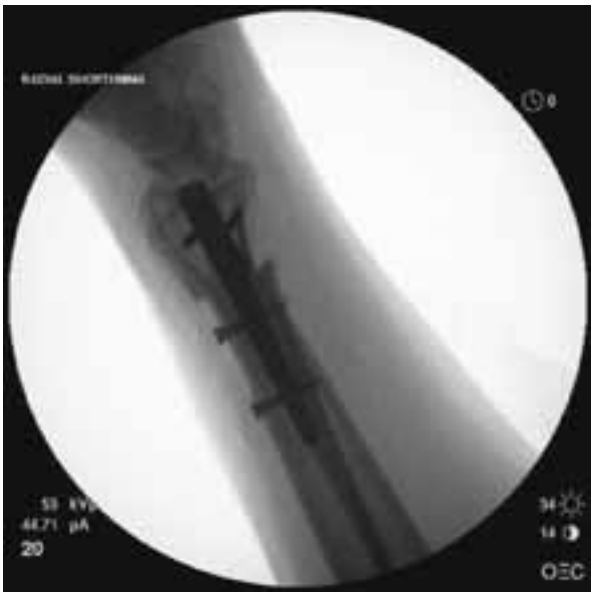

Figure 10B

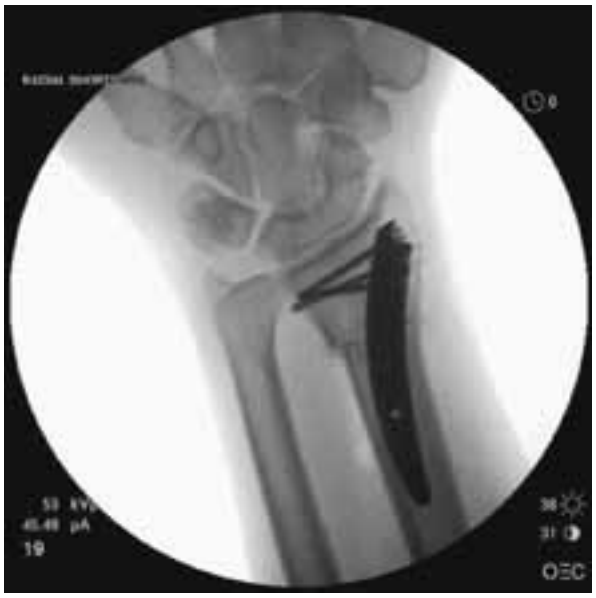

Figure 10A. PA, lateral and AP views.

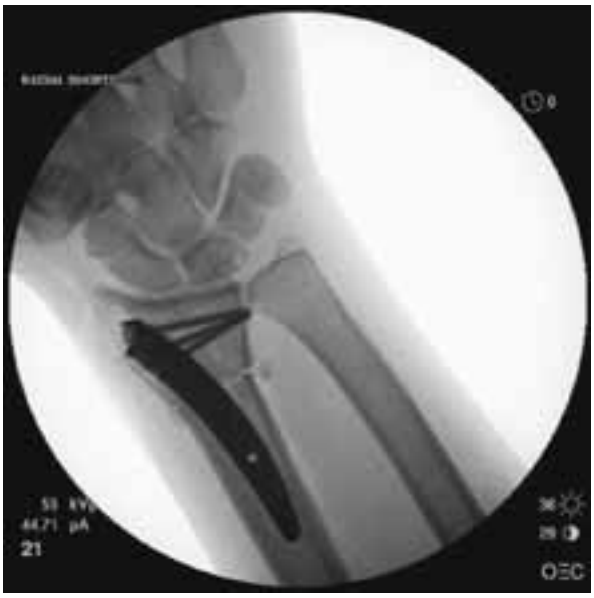

Figure 10C

zero profile implant that eliminates soft-tissue irritation and in the case of the distal radius, tendon impingement and the potential attritional tendon ruptures. The decreased soft tissue dissection ultimately reduces the post-operative swelling allowing for early rehabilitation and return to work.[5]

Other techniques of fixation after a radial shortening osteotomy involve either volar or dorsal plating. The introduction of locking technology has improved the strength of such fixation devices but the common complications associated with such constructs have not been eliminated. Dorsal plating can commonly be complicated by prominent hardware or plate irritation, resulting in hardware removal in up to 30% in some case series.[6] Volar plating like dorsal plates can commonly cause soft-tissue irritation, particularly with the volar tendon structures also necessitating repeat surgery for hardware removal.[7] Both approaches entail more soft tissue dissection leading to an increased amount of post-operative swelling which can ultimately impair the post-operative rehabilitation.[6, 7]

The advantage of this technique with the Micronail includes less soft tissue trauma with a locking fixed angle device that allows for earlier motion while maintaining stability at the osteotomy site.[4, 5] This technique has become our preferred method for radial shortening osteotomy. Long-term studies are needed to further evaluate this procedure. It is our belief that intramedullary fixation provides the patient

with certain advantages such as an earlier return to range of motion, earlier return to activities of daily living and less dependence on post operative immobilization without compromising union of the osteotomy.

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Technique

Fractures of the Distal Radius: Temple University Treatment Algorithm

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Introduction

Fractures of the distal radius represent one of the most common orthopaedic injuries, accounting for approximately 17% of annual emergency room visits later presenting to orthopaedic surgeons for definitive management.[4, 5, 10] Over the past twenty years, our approach to management has changed owing to a better understanding of fracture patterns, clinical outcome measures, and advances in technology. Expectations after fracture treatment for rapid return to functional levels, with good to excellent outcomes are the norm, and the ability to remain independent in activities of daily living have become a high priority among the elderly population.

Proper management of fractures of the distal radius can minimize potential complications. These complications include but are not limited to non-union, malunion, post-treatment joint stiffness of the radio-carpal and distal radio-ulnar joints, as well as post-traumatic arthrosis. Both acute and chronic soft tissue complications can also occur including: acute and late onset median neuropathy, adhesions or ruptures of the flexor and/or extensor tendons, pain dysfunction syndromes, and hand/digital stiffness resulting in a loss of grip strength. Injuries associated with distal radial fractures include ulnar styloid fractures, carpal bone fractures, and triangular fibrocartilage complex (TFCC) tears.

Proper management also requires knowledge of normal distal radius anatomy and biomechanics, as well as indicators of fracture stability, which will help guide treatment. A treatment algorithm has been developed to highlight critical decision points that will influence treatment methods best employed to achieve optimal outcomes.

Anatomy and Biomechanics

The anatomy of the distal radius is designed to allow motion in three planes (flexion/extension, pronation/supination and radial/ulnar deviation) and, along with the distal ulna and TFCC, bear the load which is transmitted from the hand through the forearm to the elbow. At the wrist, the distal radius bears 80% of the load which is transmitted to the third ray and capitate. This load is shared equally between the scaphoid and lunate fossae.[2, 11] In the sagittal plane, this load is born along the volar cortex, reflected in the thickened cortex that creates the "calcar" of the distal radius (Figure 1). Fracture stability is linked to integrity of this cortical line. Once this volar cortex is restored, radial length will be restored.

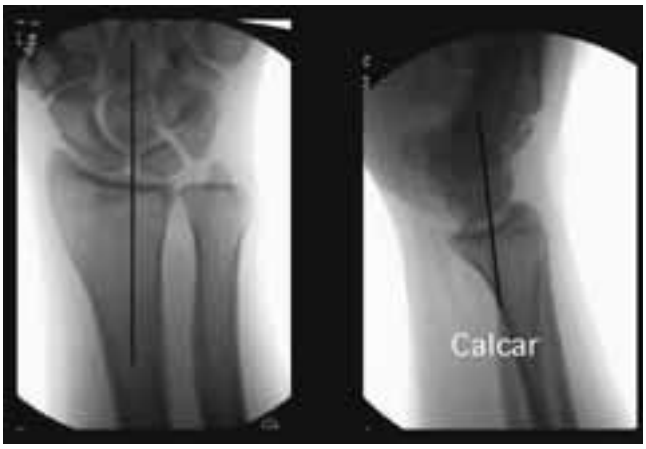

Figure 1. AP and lateral of the wrist demonstrating the weight bearing axis.

Classification

Fractures of the distal radius have been categorized and classified by many different methods over the years. These include but are not limited to fracture eponyms, the Frykman classification (Table 1),[3] the Melone classification (Table 2),[9] the Universal classification (Table 3)[8] and the AO classification (Table 4).[1] While all have value, a system that differentiates stable from unstable fracture patterns serves the treating physician best in terms of guiding appropriate management.

Table 1. Frykman Classification System[3]

	Ulnar Styloid Fracture	
Radial Fracture	**Absent**	**Present**
Extra-articular	I	II
Intra-articular — Radiocarpal Joint	III	IV
Intra-articular — DRUJ	V	VI
Intra-articular — Both	VII	VIII

Table 2. Melone Classification System[9]

Type	Fracture Pattern
I	Minimal displacement, stable, no comminution
II	Displacement of the medial complex, comminuted, unstable
III	Displacement of medial complex and radial shaft, unstable
IV	Split of medial complex into dorsal and volar fragments Soft tissue damage and stripping

Table 3. Universal Classification System[8]

Type	Fracture Pattern
I	Extra-articular, nondisplaced
II	Extra-articular, displaced
III	Intra-articular, nondisplaced
IV	Intra-articular, displaced

Table 4. AO Classification System[1]

23-A	Extra-articular
23-A1	Ulna, Radius Intact
23-A2	Radius, simple and impacted
23-A3	Radius, comminuted
23-B	**Partial Articular**
23-B1	Radius, sagittal split
23-B2	Radius, coronal split with a dorsal rim fragment
23-B3	Radius, coronal split with a volar rim fragment
23-C	**Complete Articular Radial Fracture**
23-C1	Simple articular and metaphyseal
23-C2	Simple articular and comminuted metaphyseal
23-C3	Comminuted articular

Mechanism of Injury

The most common mechanism of injury is the "fall on the outstretched hand." As force impacts the thenar and hypothenar eminences with the wrist in 40–90° of dorsiflexion, failure will result on the tension side (volar cortex) and the force will propagate across the distal radius and dissipate on the compression side (dorsal cortex). This frequently results in dorsal comminution.[7] The energy absorbed at failure can have a dramatic range and affects the degree of comminution, while the position of the carpus at impact will affect the intra-articular component.

Outcomes

Various studies helped to determine range of motion and grip strength depending on anatomic relationships after healing. Acceptable outcomes have been based on radiographic parameters and include restoration of radial length to within 5 mm, radial inclination to 15°, volar tilt to neutral ideally (but it can range from 15 degrees dorsal tilt to 20 degrees volar tilt), and restoration of the articular congruity to within 1–2 mm.[6, 12, 13] These parameters help to predict the development of radiographic degenerative changes but do not clearly reflect long term functional outcomes.

Treatment Option/Decision Making

Stable fractures can be managed with immobilization alone, but unstable fractures require additional intervention. This intervention can range from a closed reduction with cast application to direct or combined open approaches and internal fixation systems to achieve fracture stability. The surgical approach to the distal radius should be dictated by the fracture characteristics, with restoration of volar cortical contact and articular congruity being the ultimate treatment goal. A familiarity with three surgical exposures, the trans-FCR volar approach, the dorsal approach, and the radial approach will allow access to the entire distal radius — from the volar aspect of the distal radio-ulnar joint (DRUJ) to the dorsal aspect of the carpus. The trans-FCR approach accesses the volar surface, from the critical volar ulnar corner to the volar aspect of the first extensor compartment. A dorsal approach through the floor of the third extensor compartment grants access to the distal radius from the second extensor compartment to the dorsal aspect of the DRUJ. The radial approach accesses the distal radius from beneath the first extensor compartment to the ulnar side of the second dorsal compartment. The utilization of these approaches, alone or in combination, provides complete access to the distal radius allowing precise anatomic restoration and adequate internal fixation (Figure 2).

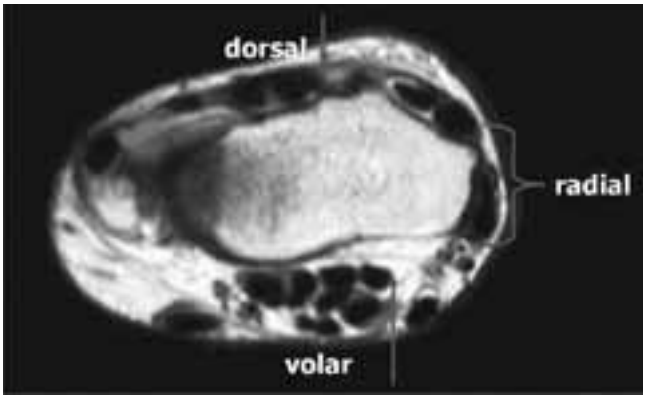

Figure 2. Locations of the radius that can be visualized through the surgical approaches.

The Temple University algorithm (Figure 3) looks at three critical factors of the unstable distal radius fracture to help determine the surgical approach necessary for adequate internal fixation. First, determine if the volar cortex (the "calcar") is reducible. An irreducible volar cortex necessitates a volar approach to help restore this critical portion of load transmitting anatomy. Second, determine if the articular surface is involved and needs restoration. Depending on the location of the articular pieces, the approach should allow the best visualization for anatomic restoration of the joint surface. Finally, determine if structural bone graft is required

to fill a deficit within the dorsal metaphysis. Answering these three questions will determine the choice of approach(es) to the distal radius, allowing exposure for reduction and internal fixation of the unstable fracture pattern.

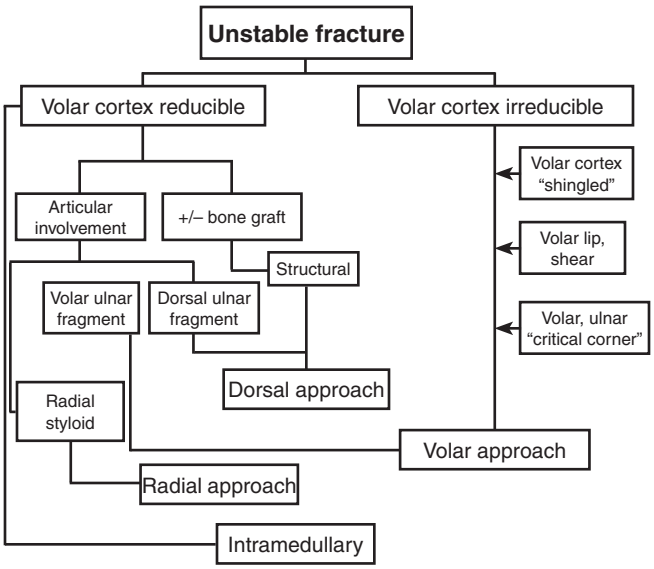

Figure 3. The Temple Algorithm

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Technique

The Sports Physical: Asset or Liability?

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Abstract

Preparticipation physical examinations are required at all levels of competitive athletics. Many different types of physicians perform these evaluations, but there is no consensus on the requirements for the exams. A basic form is proposed to include the critical components for these tests. A major concern is to try to avoid sudden death in an athlete; although not totally preventable, critical questions in the history and an appropriate physical examination can significantly reduce this risk. Careful review of previous athletic injuries and proper physical evaluation are important parameters to include for successful screenings.

One of the major changes in the practice of medicine over the last decade has been in the approach to the treatment of injuries and diseases. Preventive medicine has taken over a major role in our practices. As physicians, we are now not only involved in the healing aspects of medicine but are just as aggressively engaged in prevention of an injury or illness from occurring. Nowhere has this been more evident than in the field of Sports Medicine in relating to preseason sports physicals. These sports physicals are required by most schools starting in grade school and extending into the professional level. The frequency of performing these screenings varies; some school districts require them before each sports season, some colleges require them once yearly. The American Heart Association's original recommendation was for a comprehensive history and physical exam at the time of original sports participation and interim histories and blood pressure checks at yearly intervals. They also stated that high school exams could be done every two years.[3] Most schools test more frequently than these suggestions. As greater importance is placed on these physicals, we must stop and think whether these exams can really prevent serious injury or not. It is often difficult to pick up cardiac abnormalities on physical examination.[6] Different physicians identify different abnormalities and referral cases vary. The pre season sports physical can be paramount to these schools, to the athlete and their family, and to society in general in helping to avoid sometimes preventable tragedies and to allow athletes of all ages to safely participate in their chosen sport.

As a physician, you may ask do I want to entertain the possibility of performing sports physicals in my area? What are the advantages and the disadvantages? The first advantage, especially if you are practicing in a new area, is to introduce yourself to a large group of potential patients. Not just the athlete, but also their family can judge your competence and professionalism during the exam.

This is a great time to educate these patients with handouts or brochures on exercises, asthma, or blood pressure problems. This exposure to the community is a wonderful practice-building tool for you or the institution that you represent. It is also the best time to screen out any athletes with serious underlying musculoskeletal, cardiac, or other medical issues that could potentially harm them if they compete in strenuous athletics. The disadvantages are minimal and are the same liability issues that you would deal with in treating or advising any patient in today's litigious society. You must be precise, accurate, and keep clear records of the examination. It is crucial that you have experience and expertise in all the areas you are examining. If you are not sure, then have other specialists present during the screenings. Even if you do everything right, other forces and issues may intrude on your medical judgment, such as in the tragic Hank Gathers case.[9]

The sports physical can be performed by many different medical providers; however, it is generally not cost effective to perform these screenings in the office, except for an individual older athlete; it seems to be more advantageous for the physician to perform a mass screening at the school. This is done using a TEAM approach. You could have a physician (general practioner, cardiologist, pediatrician, or orthopedist) doing the physical exam, followed by an athletic trainer performing a flexibility (hamstring and groin) test, as well as getting their height and weight, followed by a nurse obtaining their blood pressure and pulse and visual exam. Often larger schools and colleges have multiple specialists available to handle their particular area of expertise. While the physical exam is important, the history taking is more important in these screening sessions. Many schools require parents to fill in this data; obviously, this requirement changes with age. One must be aware; however, how many college students are truly unaware of their family's medical history. Critical questions to be asked include history of underlying asthma, diabetes, or previous surgeries. Other issues concerning head injuries, loss of consciousness, contacts, medicines, and allergy history are all important. The most crucial questions involve heart history; these are the warning signs of possible sudden unexplained death during strenuous exercise. Any history of chest pain during exercise, syncopal epi-

sodes, shortness of breath on exertion, heart murmur or hypertension is ominous. Family history of premature or sudden death needs further investigation (Table 1). Remember to document the answers to all of these questions as this documentation is as important as the reason for the physical itself.

Table 1. Critical Questions for Heart Disease

1. History of chest pain during exercise
2. History of syncope (fainting)/shortness of breath with exercise
3. History of heart murmur
4. History of hypertension
5. Family history of premature/sudden death

Many studies have shown that most preseason screenings do not cover all these issues. Pfister showed that 40% of college screenings do not cover the American Heart Association's 12 item screening requirements and Gomez stated that only 17% of high school screenings included 3 questions covering exercise related symptoms, previous history of heart murmur or hypertension, and family history of heart attack or sudden death.[4, 5]

The purpose of these preparticipation screenings is to pick up potentially harmful medical problems and correct them prior to clearing the athlete to play. The question arises on when to refer; this obviously varies depending on the training of the screening physician. It is common sense that it's always better to ask for another opinion if one is not sure. If the nurse obtains an abnormal blood pressure measurement, the best guide would be to repeat the test in a half hour and possibly have another examiner perform the second check. Remember to always use a larger cuff for the larger athlete. Be on the lookout for loud heart murmurs, as soft innocent murmurs are common and require no treatment. O'Connor found that grade 3 or louder murmurs, holosystolic or diastolic murmurs, split S2 sounds, and accentuation of a murmur with squat to stand or Valsalva maneuvers were significant and required further investigation.[8] Wheezing with no prior treatment can also be a red flag and will need further work up. If the response was affirmative to any heart questions, this will also require further study, with possible EKG or echocardiogram.[6, 7] There is also the possibility of chronic findings such as back pain, shoulder instability or patellar symptomatology which may need further workup. In keeping all of these possibilities in mind, it is most advantageous to have a network set up prior to the physicals to include primary care physicians, cardiologists, pulmonologists and orthopedists in the area to whom you could refer possible problems.

Most physical examinations, especially in the younger student-athletes, are fortunately negative. The problem is the rare, but devastating sudden death of a competitive athlete. This is a personal tragedy with a significant impact; it is usually caused by previously unsuspected cardiovascular disease. The reason this occurs is not known, but the premise is that intense athletic training is likely to increase risk for sudden cardiac death in athletes with underlying structural heart disease. In younger (<35 year old) athletes, congenital cardiac malformations, e.g., hypertrophic cardiomyopathy and coronary artery anomalies, are often implicated.[1, 2] In older athletes, the underlying process is usually atherosclerotic coronary artery disease. As mentioned above, the risks are quite small. There are estimated to be 4 million high school athletes in grades 9–12; 500,000 college students participate in sports; 5000 professional athletes involved; and an unknown number of youth, middle school, and masters athletes who all are at potential risk. The latest estimates are 1:100,000–1:150,000 athletic field deaths per year. The risks are higher in males and higher in older athletes.[2]

What are the legal considerations of screening physicals?[9] A physician who clears an athlete to participate is not legally liable if death occurs to that athlete because of an undiscovered cardiovascular condition. As long as an adequate screening exam is documented, the plaintiff must prove that the physician deviated from the usual standard of care in performing the screening.[2] There are **NO** universally accepted standards for screening — the consensus from the American Heart Association is careful personal and family history coupled with a good physical exam is the best method for doing these preseason screening exams.

In summary, preseason physicals are common and required at all levels of participation. A whole gamut of physicians perform these tests without one universal template for all. We feel a team approach is the best way to accomplish these in a timely fashion. The history is crucial, especially those questions dealing with the heart. Any abnormal physical findings need attention prior to participation. A sample form used at our institution is attached and may be helpful for your use (Appendix A). The risk of sudden death is low, but catastrophic when it occurs. This risk can be lowered, not eliminated, with improved screening methods.

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Appendix A

Temple University School of Medicine
Department of Orthopaedic Surgery

Preparticipation Physical Examination

History

Date _____

Name _____ Sex _____ Age _____ Date of Birth _____

Grade _____ Sport _____ _____ _____

Personal physician _____ Address _____ Phone Number _____

Explain "Yes" answers below:	Yes	No
1. Have you ever been hospitalized?	☐	☐
Have you ever had surgery?	☐	☐
2. Are you presently taking any medications or pills?	☐	☐
3. Do you have any allergies (medicine, bees or other stinging insects)?	☐	☐
4. Have you ever passed out during or after exercise?	☐	☐
Have you ever been dizzy during or after exercise?	☐	☐
Have you ever had chest pain during or after exercise?	☐	☐
Do you tire more quickly than your friends during exercise?	☐	☐
Have you ever had high blood pressure?	☐	☐
Have you ever been told that you have a heart murmur?	☐	☐
Have you ever had racing of your heart or skipped heartbeats?	☐	☐
Has anyone in your family died of heart problems or a sudden death before age 50?	☐	☐
5. Do you have any skin problems (itching, rashes, acne)?	☐	☐
6. Have you ever had a head injury?	☐	☐
Have you ever been knocked out or unconscious?	☐	☐
Have you ever had a seizure?	☐	☐
Have you ever had a stinger, burner or pinched nerve?	☐	☐
7. Have you ever had heat or muscle cramps?	☐	☐
Have you ever been dizzy or passed out in the heat?	☐	☐
8. Do you have trouble breathing or do you cough during or after activity?	☐	☐
9. Do you use any special equipment (pads, braces, neck rolls, mouth guard, eye guards, etc.)?	☐	☐
10. Have you had any problems with your eyes or vision?	☐	☐
Do you wear glasses or contacts or protective eye wear?	☐	☐
11. Have you ever sprained/strained, dislocated, fractured, broken or had repeated swelling or other injuries of any bones or joints?	☐	☐
☐ Head ☐ Shoulder ☐ Thigh ☐ Neck ☐ Elbow ☐ Knee ☐ Chest		
☐ Forearm ☐ Shin/calf ☐ Back ☐ Wrist ☐ Ankle ☐ Hip ☐ Hand. . . . ☐ Foot		
12. Have you had any other medical problems (infectious mononucleosis, diabetes, etc.)?	☐	☐
13. Have **you had a medical problem or injury since your last evaluation?**	☐	☐

14. When was your last tetanus shot? _____
When was your last measles immunization? _____
15. When was your first menstrual period? _____
When was your last menstrual period? _____
What was the longest time between your periods last year? _____

Explain "Yes" answers:

_____

_____

_____

I hereby state that, to the best of my knowledge, my answers to the above questions are correct.

Date _____

Signature of athlete _____

Physical Examination

Date _____

Name _____ Age _____ Date of Birth _____

Height _____ Weight _____ BP _____/_____ Pulse _____

Vision R 20/_____ L 20/_____ Corrected: Y N Pupils _____

	Normal	Abnormal Findings	Initials
Cardiopulmonary			
Pulses			
Heart			
Lungs			
Tanner Stage	1	2 3 4 5	
Skin			
Abdominal			
Genitalia			
Musculoskeletal			
Neck			
Shoulder			
Elbow			
Wrist			
Hand			
Back			
Knee			
Ankle			
Foot			
Other			

Clearance:

A. Cleared

B. Cleared after completing evaluation/rehabilitation for:

C. Not cleared for: ☐ Collision
☐ Contact
☐ Noncontact ------Strenuous -------Moderately Strenuous -------Nonstrenuous

Due to _____

Recommendation _____

_____

Name of Physician _____ Date _____

Signature of Physician _____

Address of Physician _____

_____ Phone _____

Abstract

The Relationship Between Concussion History and Post-Concussion Neuropsychological Performance and Symptoms in Collegiate Athletes

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Investigation performed at Department of Kinesiology, College of Health Professions, Temple University, Philadelphia, PA

Purpose

Athletes who participate in sports are at an inherent risk for sustaining concussions. Research examining the long-term consequences of sport-related concussion has been inconsistent in demonstrating lingering neuropsychological *decrements* that may be associated with a previous history of concussion. The purpose of this study was to determine the relationship between concussion history and post-concussion neuropsychological performance and symptoms in collegiate athletes.

Methods

The study group consisted of 57 concussed intercollegiate athletes. Thirty-six had no history of a prior concussion while 21 had a history of two or more prior concussions. The intervention consisted of a baseline neuropsychological test. Subjects who sustain a concussion were administered two follow-up tests at one and five days post-injury. The independent variables were history of concussion (no history of concussion, two or more concussions) and time (baseline, one day post-concussion, five days post-concussion). All subjects were administer an Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) neuropsychological test battery which measured verbal memory, visual memory, reaction time, processing speed, and 22 concussion symptom inventory.

Results

Results revealed a significant within-subjects effect (time) on ImPACT performance, a between-subjects multivariate effect of group, and a significant group-by-time interaction ($p = .034$). Athletes with a previous history of concussion performed significantly worse on verbal memory ($p = .01$) and reaction time ($p = .023$) when compared to athletes who did not report a previous concussion at five days post-concussion. There were no significant group differences five days post-injury on visual memory ($p = .167$), motor processing speed ($p = .179$), and total concussion symptoms ($p = .09$).

Conclusion

The results of neuropsychological testing indicating that concussed collegiate athletes with a previous history of two or more concussions recovered more slowly than athletes without a previous history of concussion.

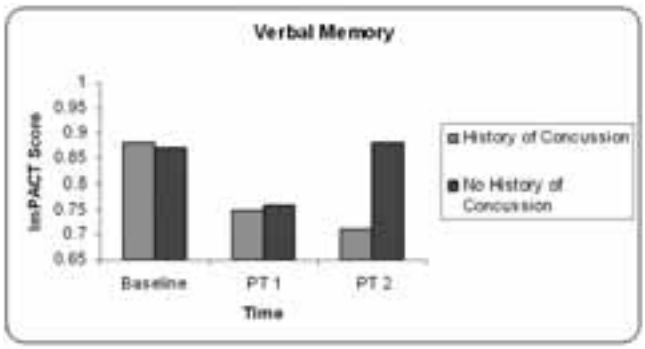

Figure 1. Verbal Memory Composite Scores

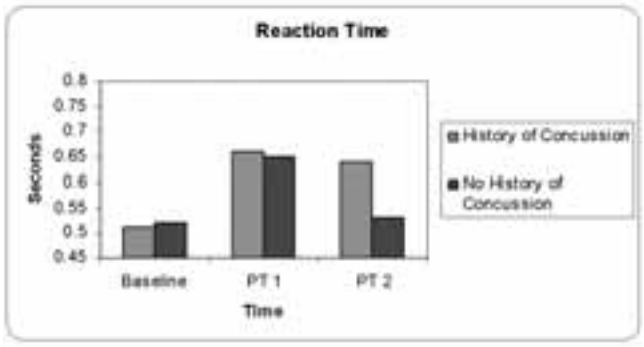

Figure 2. Reaction Time Composite Scores

Abstract

Gender Differences on Neuromuscular Control of the Hip: Implications for Knee Joint Stability and Non-Contact Anterior Cruciate Ligament Injury

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Purpose

The purpose of this study was to determine if gender differences existed in hip muscle strength and activation as well as lower extremity kinetics during a functional landing task.

Methods

42 healthy, NCAA Division I collegiate and club sport male and female athletes (21 males, 21.1 + 1.7 years, height = 181.2 + 8.9 cm, mass = 85.3 + 21.3 kg; and 21 females, 19.7 + 1.5 years, height = 171.1 + 8.6 cm, mass = 65.2 + 7.3 kg) participated in the study. A post-test only design was used. The independent variable was gender. Dependent variables were hip muscle strength, electromyographic (EMG), and kinetic variables. All data were collected in a research laboratory. Isometric strength and strength ratios for the hip extensors, flexors, abductors, adductors, and lateral and medial rotators, preparatory (150ms) and reactive (250ms) EMG area and co-activation area for the gluteus maximus, biceps femoris, gluteus medius, iliopsoas, rectus femoris and adductor longus, and peak vertical ground reaction force, anterior shear force and lower extremity rate of loading were collected on a one-legged standing broad jump-to-vertical jump maneuver.

Results

Independent t-tests revealed that males had 12% greater isometric hip flexor (t = 2.1, p = .041, males = 5.9 + 1.3, females = 5.2 + .7 N/kg). and 15% greater lateral rotator (t = 2.5, p = .016, males = 5.4 + 1.4, females = 4.6 + .6 N/kg) strength. A one-way MANOVA revealed that females had 12% greater gluteus maximus (F = 7.7, p = .008, females = 14.1 + 1.8, males = 13.0 + 1.9), rectus femoris (F = .41, p = .050, females = 15.2 + 1.9, males = 13.5 + 3.3), and combined co-agonist gluteus maximus and biceps femoris (F = 13.7, p = .001, females = 28.6 + 2.7, males = 25.7 + 2.6) EMG reactive area. No other gender differences were statistically significant.

Conclusions

Based on the lack of gender differences on EMG preparatory muscle activity, females and males prepare for landing with similar muscle activation strategies at the hip driven by feed forward mechanisms of dynamic restraint. However, due to specific muscle weakness of the hip flexors and lateral rotators females require greater reliance on reactive activity of these muscles to attenuate force at ground contact. This may lead to early fatigue and predisposition to increased risk of acute non-contact injury.

Figure 1. Standing Broad Jump-to-Vertical Jump Maneuver

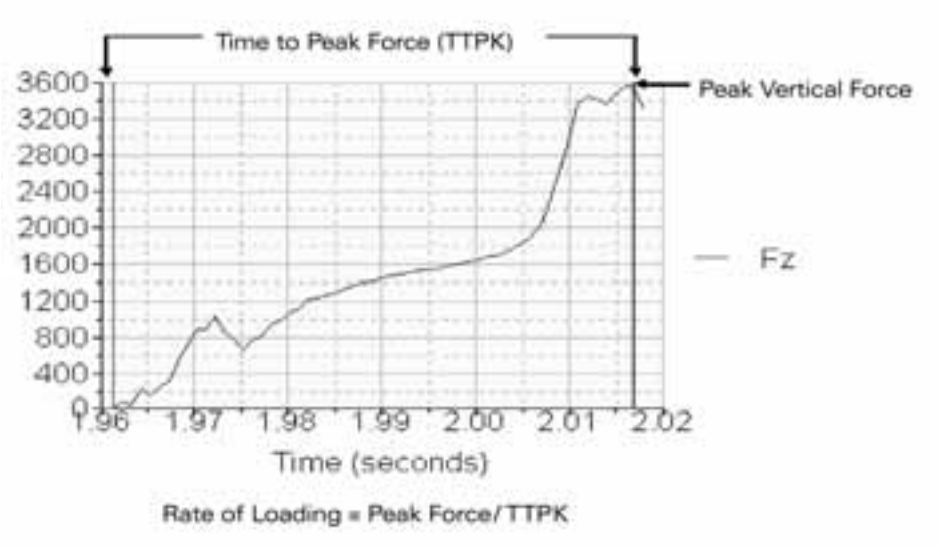

Figure 2. Kinetic Measurements

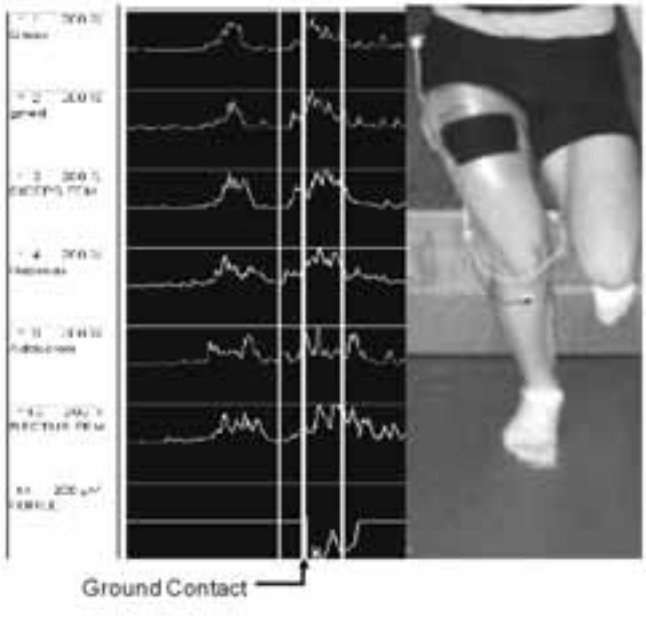

Figure 3. EMG Set Up

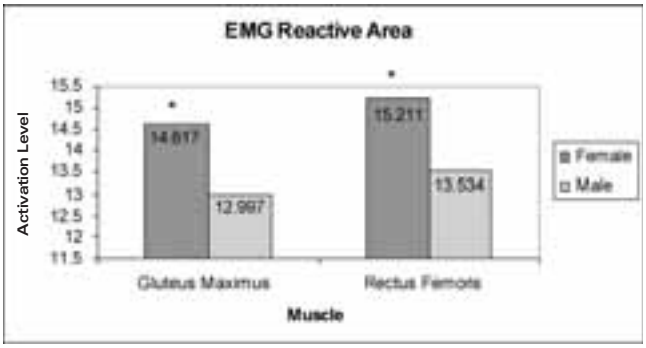

Figure 5. EMG Reactive Area Differences

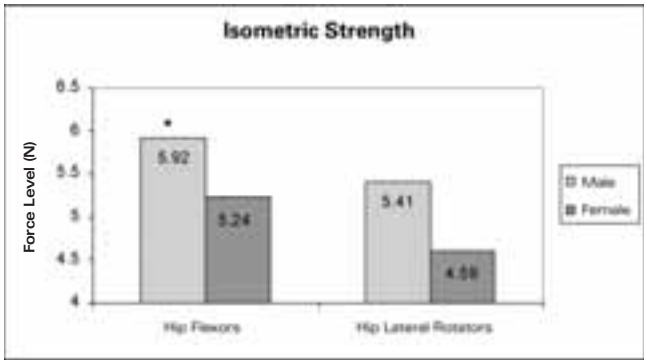

Figure 4. Isometric Strength Differences

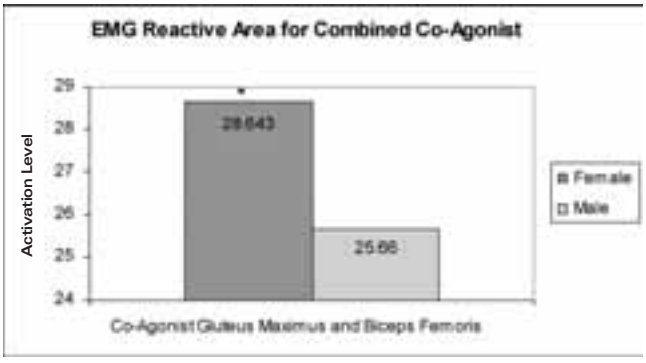

Figure 6. EMG Reactive Area Combined Differences

Abstract

Gender Differences in Head Acceleration During Soccer Heading Wearing Soccer Headgear

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Purpose

Previous research has indicated that female soccer players may be at greater risk of concussion than their male counterparts. Soccer headgear is being marketed for the purpose of reducing head acceleration and risk of concussion. To determine if a gender and/or headgear effect exist in head impact kinematics and dynamic stabilization during soccer heading.

Methods

Forty-four (female N = 29, age = 19.5 ± 1.8yrs, height = 164 ± 9.1cm, mass = 63.2 ± 7.1kg, and males N = 15, age = 20.3 ± 2.9yrs, height = 174 ± 6.7cm, mass = 67.0 ± 9.5kg) soccer players with at least five years of heading experience volunteered for the study. Institutional Review Board approval and participant written informed consent were obtained prior to data collection. The independent variables were gender and headgear (control, Full 90 Select, Headblast). Headgear was a randomized and repeated measure. Participants performed 4 straight (sagittal plane) soccer headers for each headgear condition (12 total). Soccer balls were projected at an initial velocity, angle of projection, and range of 9.83 m/s (22 mph), 40 degrees, and 11m (35 ft), respectively. Head kinematics and superficial neck muscle EMG were determined using a mouthpiece accelerometer (ICC 2,1 = .89; Endevco Corp., San Jaun Capistrano, CA) and the Noraxon Telemyo System (ICC 2,1 = .90; Noraxon USA, Scottsdale, AZ), respectively. The main outcome measures included peak resultant linear head acceleration (g), Head Injury Criteria (HIC_{36}), and neck muscle (left and right SCM and trapezius I) peak (%MVIC) and area (%MVIC x ms) activity. Statistical analyses were performed using SPSS 14.0 and included MANOVA, ANOVA, and appropriate follow-up and post hoc tests ($p \leq .05$).

Results

Females significantly increased, $t(1,28) = -2.33$, $p = .027$, in head acceleration when wearing the Full90 Select (mean = 65.5 ± 16.0g) versus the control (mean = 60.5 ± 12.4g), whereas males significantly decreased , $t(1,14) = -2.74$, $p = .016$, in head acceleration when wearing the Full 90 Select (mean = 45.5 ± 17.5g) versus the control condition (mean = 54.7 ± 13.4g). There was also a trend ($p = .069$) toward a similar gender by headgear interaction effect for the HIC_{36} data. No significant differences were identified in preparatory or reactive neck muscle activity.

Conclusion

Soccer headgear amplified the gender difference in head acceleration during the soccer heading and should not be used as a means of head injury prevention for female soccer players. Females reportedly exhibit lower stability qualities (i.e., head mass, neck girth and muscle strength) than males. With no gender differences in neck muscle activation strategies (e.g., greater pre-activation) to compensate, females were at greater risk of head injury.

Figure 1. Soccer headgear. The Full 90 Select (left) and the Headblast headgear (right).

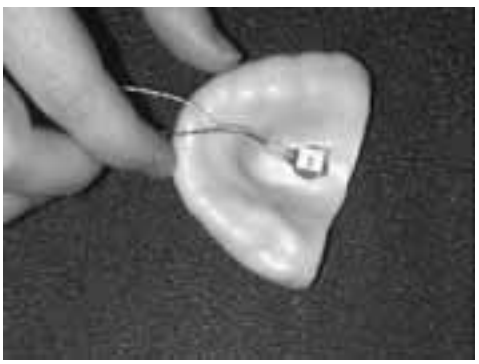

Figure 2. Custom-made mouthpiece with tri-axial accelerometer.

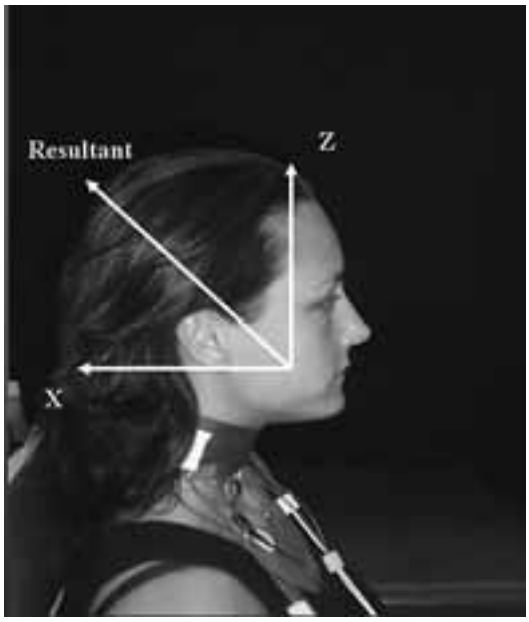

Figure 3. EMG set-up and peak head impact resultant acceleration vector.

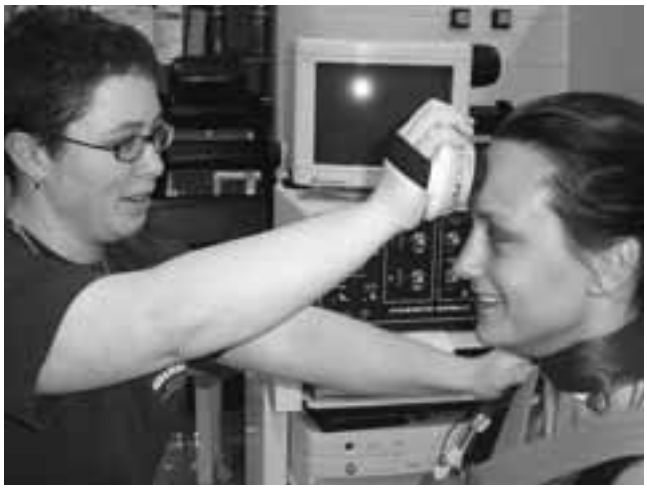

Figure 4. Isometric strength for neck flexion using HHD.

Figure 5. JUGS soccer machine.

Figure 6. Functional heading task.

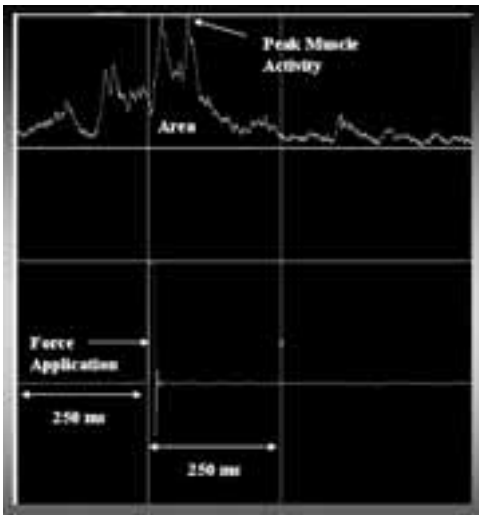

Figure 7. EMG assessment of neck muscle preparatory and reactivity.

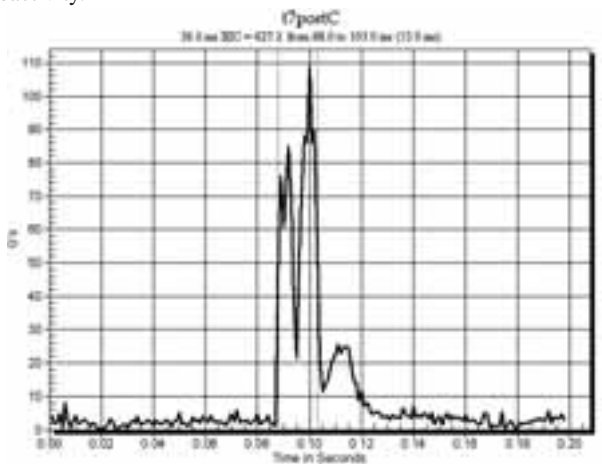

Figure 8. Acceleration-time curve and HIC_{36} value.

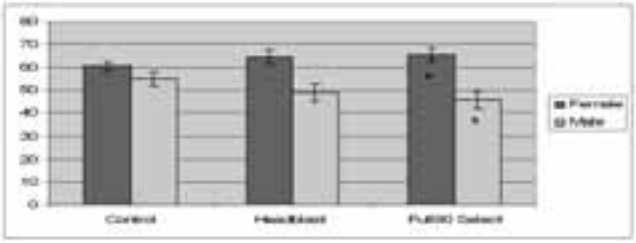

Figure 9. Results

Abstract

Effect of Plyometric Neck Muscle Training on Head-Neck Segment Kinematics and Dynamic Stabilization During Soccer Heading: A Pilot Study

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Purpose

Brain injury is directly related to head acceleration experienced during head impact. Neck muscle strengthening has been purported to reduce head acceleration. Isometric and isotonic neck muscle training have elicited strength gains, yet no protocol has educed head kinematic changes during a functional task. This study investigated the effect of an 8-week plyometric neck muscle training program on head-neck kinematics during soccer heading.

Methods

Twenty division I female soccer players (Experimental group N = 13, age = 18.9 ± 0.64 yrs, height = 166 ± 5.6 cm, body mass 62.7 ± 4.4 kg, head mass = 5.1 ± 0.35 kg; Control group N = 7, age = 19.5 ± 0.54 yrs, height = 165 ± 8.2 cm, body mass 64.8 ± 5.5 kg, head mass = 5.3 ± 0.46 kg). The independent variables were group (experimental vs. control) and time (pre-training vs. post-training). The experimental group performed an 8-week training program in conjunction with their off-season conditioning. The training consisted of three sets of ten repetitions (1 rep/sec) using resistive tubing, and tubing resistance was increased every two weeks. The control group performed no neck exercises. Head-neck isometric strength was assessed pre and post training using a hand-held dynamometer (ICC 2,1 = .96). Neck muscle preparatory and reactive activity (i.e., left and right SCM and Trapezius) and resultant head acceleration were measured pre and post training during 4 soccer headers using a mouthpiece accelerometer (ICC 2,1 = .89) and the Noraxon Telemyo EMG system (ICC 2,1 = .90).

Results

There was a significant effect for time when assessing isometric strength for flexion, $F(1,19) = 5.7$, $p = .027$, left rotation, $F(1,19) = 14.6$, $p = .001$, and right rotation, $F(1,19) = 10.7$, $p = .004$. Subjects exhibited 21, 32, and 29% greater flexor, left and right rotator strength, respectively, during the post-test versus the pre-test. No other significant differences existed. There was a trend, $F(1,19) = 1.7$, $p = .210$, power = .234, toward a significant group by time interaction for head acceleration The experimental group head acceleration was reduced 4% relative to a slight increase in the control group over time.

Conclusion

This pilot study is the first to assess the effect of plyometric neck muscle exercise on head-neck stability during a functional task. Although neck muscle strength gains were elicited, muscle activation strategies and head acceleration were not altered significantly in our sample. Future research will assess neck muscle training effects in a larger sample of soccer players (at greater training intensities and durations), other athletes, and physically active non-athletes for the purpose of head injury prevention.

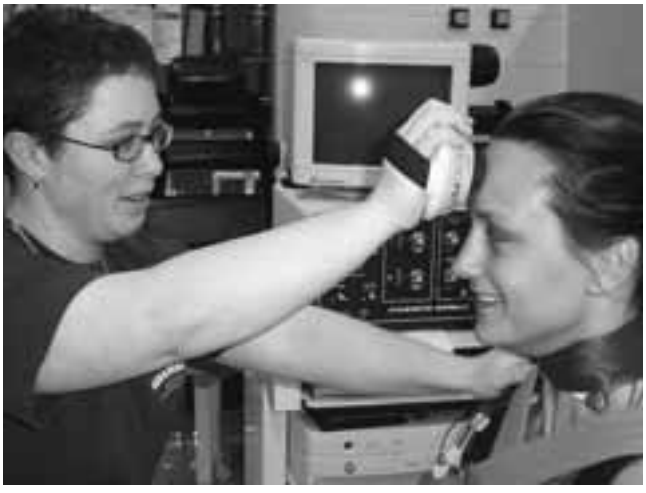

Figure 1. Isometric strength for neck flexion using HHD.

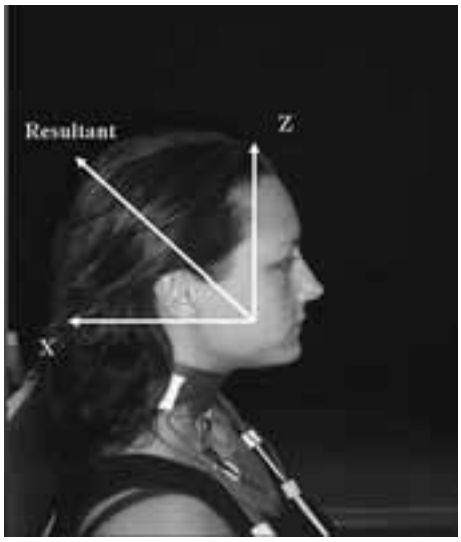

Figure 2. EMG set-up and peak head impact resultant acceleration vector.

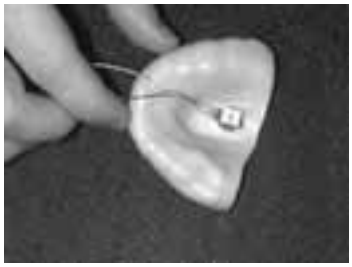

Figure 3. Custom-made mouthpiece with tri-axial accelerometer.

Figure 4. JUGS soccer machine.

Figure 5. Resistive tubing.

Figure 6. Functional heading task.

Figure 7. Rotational plyometric exercises with resistive tubing.

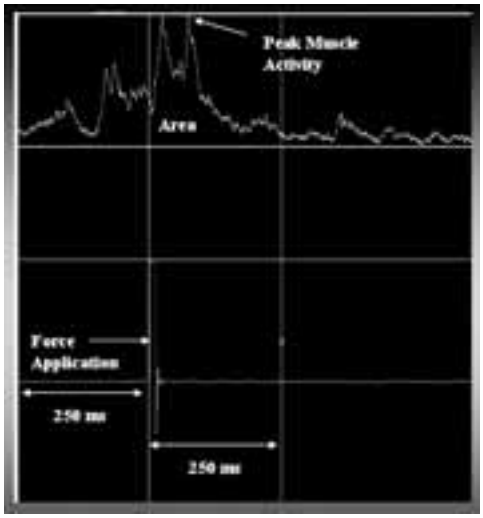

Figure 8. EMG assessment of neck muscle preparatory and reactivity.

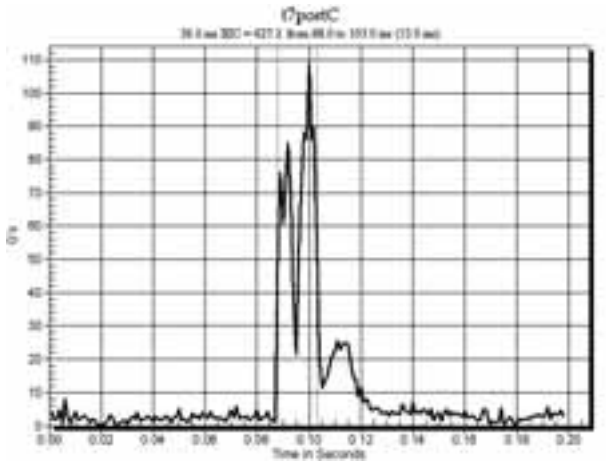

Figure 9. Acceleration-time curve and HIC_{36} value.

Abstract

Functional and Radiographic Results Following Radial Head Replacement

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Purpose

Radial head fractures account for up to one-third of all fractures around the elbow joint. Approximately 5%–10% of elbow dislocations include radial head fractures, which can be extremely disabling. Treatment options for fractures involving the radial head include excision of fragments, open reduction and internal fixation, or radial head replacement with an implant. Each fracture obviously needs to be considered separately but patients with displaced or comminuted radial head fractures unamendable to fixation with ORIF and involving more than one-third of the radial head should have a radial head implant placed with an absolute indication in those with suspected MCL or interosseous membrane injury. The purpose of this study is to document the functional and radiographic outcomes of patients who underwent radial head replacements for comminuted radial head fractures.

Methods

We conducted a retrospective review of the results of twenty-five consecutive radial head arthroplasties by the single author (JT) over a five year period between 1999 and 2005. The same approach was used in all patients consisting of Kocher interval between anconeus and EDC. Implants consisting of vitallium from a single company were used. Evaluation of function was performed by phone interview and involved questions specific activities of daily living, return to work, and subjective satisfaction questions. In addition, DASH forms were sent to willing participants for standardization and validation of our phone evaluation. Available radiographs were also evaluated to monitor any radiographic evidence of suboptimal results including heterotopic ossification or malalignment of the prosthesis.

Results

Analysis of the data received from phone interviews shows 42% of subjects were male with the dominant arm being involved in 42% of fractures. Questions regarding subjective pain showed 67% of patients had pain at rest with an average score of 3/10 (0–8), 58% complained of some pain with flexion, and 42% complained of pain in extension or supination. Functionally, 58% of patients could brush their hair without any problems and feed themselves, only 42% could care for their perineum with the affected hand, 92% could push off from a chair without problems, and 38% could open doors without any problems. X-ray evaluation was available in only 25% of patients with an average of one follow-up xray at 7 weeks. No radiographs showed any evidence of heterotopic ossification, synostosis, nor malalignment.

Conclusions

Radial head fractures account for a large percentage of fractures around the elbow and should be considered for anyone with a traumatic mechanism and elbow pain. Radial head fractures often occur concomitantly with elbow dislocations and may cause severe disability. The three primary stabilizers of the elbow include the bony articulation between the ulna and humerus, the medial collateral ligament and the lateral ulnar collateral ligament. The secondary stabilizers include the radial head, the common extensor and flexor muscles, as well as the capsule. However, when the medial collateral ligament and/or interosseous membrane become compromised, the radial head confers the primary stability to valgus stress and longitudinal compression. Treatment options for fractures involving the radial head include excision of fragments, open reduction and internal fixation, or radial head replacement with an implant. Restoring the native anatomy with ORIF is likely the best option for those minimally displaced fractures where stable constructs can be achieved as cadaver studies have shown native radiocapitellar congruence and size provides superior stability when compared to an implant. Unfortunately, some radial head fractures involve high energy trauma resulting in severely comminuted fractures not amendable to plate and screw fixation. Controversy still resides in whether these severe injuries are best treated with fragment excision or radial head arthroplasty. Fragment excision can be considered if the fractured segment blocks rotation, does not articulate with the lesser sigmoid notch, and accounts for less than one-third of the radial head. Otherwise, functional patients who have severe comminution or fractures involving more of the radial head or neck should be seriously considered for radial head arthroplasty with an absolute indication in those with suspected MCL or interosseous membrane injury. Our study shows all patients undergoing radial head replacement were satisfied with their outcome, however, the majority of patients do still have some residual low level of pain (avg. 3/10) both with activity and even at rest. Surprisingly, a large number of our patients were not able to perform some basic activities of daily living but were still satisfied with the function attributable to the radial head replacement. DASH scores were returned in 50% of patients who were willing to send them back. 2/3 of these patients were mostly satisfied (7/10) with the results of surgery and had an average DASH score of 42. 1/3 of these patients rated their success as somewhat satisfied (4/10) and this corresponded with a DASH score of 56.

Abstract

Performance Analysis and Morphology of Unused Versus Reprocessed Drill Bits

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Purpose

Many hospitals use reprocessed orthopaedic instruments, including drill bits and saw blades, as a cost saving effort. However, the performance and safety of these reprocessed instruments has not been reported in the literature despite clinical suggestions that their performance may be inferior and safety is questionable. The purpose of this study was to compare reprocessed with unused drill bits by microscopic examination as well as biomechanical performance testing.

Methods

Reprocessed drill bits (2.5mm x 110mm) from two different companies (method I and II) were compared with similar unused drill bits. In method I the effect of the number of reprocessing (1, 2, and 3 times) was also investigated. A total of 15 drill bits were tested in 5 groups of 3. The morphology of the tip of the drill bits were studied using a digital microscope at 100X magnification. The biomechanical performance of the drill bits was quantified using a custom-made apparatus by which a pneumatic drill at 750 rpm was pressed vertically into artificial bone specimens with 60 N constant force. The specimens were 52 mm-thick test blocks (Sawbones, WA) made of a solid rigid polyurethane foam block sandwiched between two 6 mm-thick sheets of e-glass-filled epoxy representing the human cancellous and cortical bones respectively. The time histories of the applied vertical force and the displacement of the drill bit were recorded and based on these measurements the drilling time (the time for the drill to pass through the specimen) was determined.

Results

The drilling time (11.5 s for the unused drill bits) was 1 to 3 seconds longer when reprocessed drill bits were used. A strong correlation was found between the drilling time and the chisel edge (the length of the transverse sharp edge at the tip of the drill). The chisel edge (0.7 mm in the unused drill bits) was reduced up to 10% in method I and was increased by 4% in method II. Variability in the geometric and mechanical measurements was significantly higher in the reprocessed drill bits.

Conclusions

The chisel edge was identified as the dominant factor in determining the biomechanical performance of the drill bit. The longer drilling time at constant force measured for the reprocessed drill bits may explain why they seem to be dull in clinical practice. In order to prevent potential thermal bone necrosis and penetrating soft tissue injury, we suggest that current reprocessing techniques be improved accordingly.

Reprint

Early Radiographic Differentiation of Infantile Tibia Vara from Physiologic Bowing Using the Femoral-Tibial Ratio (FTR)

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Jon Davids, MD,[2] Richard Davidson, MD[1]

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Study Conducted at Shriners Hospitals for Children, Philadelphia, Pennsylvania, USA

Abstract

We hypothesize that the ratio of the femoral to tibial metaphyseal-diaphyseal angles (femoral-tibial ratio, or FTR) will more accurately differentiate physiologic bowing from infantile tibial vara than the tibial metaphyseal-diaphyseal angle (TMDA). The purpose of this study is threefold: (1) determine the false-negative and false-positive error of the FTR and TMDA; (2) determine to the effect of rotation on the FTR and TMDA; and (3) determine the reliability of the FTR and TMDA measurements. An FTR less than 1 resulted in a false-negative error of 10% and a false-positive error of 7%, whereas a TMDA greater than 13° resulted in a false-negative error of 23% and a false-positive error of 10%. The difference between internal and external rotation was not significant (p=0.91), whereas it was for the TMDA (p=0.002). The FTR was found to have good interobserver and intraobserver reliability (0.78 and 0.98, respectively).

Key Words. Infantile tibia vara, Blount's disease, femoral metaphyseal-diaphyseal angle, physiologic bowing.

Introduction

It has long been recognized that children under the age of two years have varus alignment of the lower extremities. Salenius and Vankka,[12] in a study of tibiofemoral angles, showed that there is a pronounced varus angle prior to age one that changes to valgus between eighteen months and three years of age. The difficulty arises when distinguishing physiologic bowing from infantile tibia vara (Blount's disease), a pathologic process that affects the proximal medial tibia and often leads to progressive deformity.[1, 3] Early diagnosis is important for treatment, in that early brace treatment may prevent the need for surgical correction.[10, 11, 13, 15]

Unfortunately, the early differentiation between physiologic bowing and infantile tibia vara may be difficult.[8] The average tibiofemoral angle at presentation for the two groups has been shown to be similar.[2, 8] Additionally, radiographic changes of infantile tibial vara as described by Langenskiöld and Riska[7] often do not appear until 24 months of age and may appear as late as 36 months.

Levine and Drennan,[8] in an attempt to better differentiate physiologic bowing from infantile tibia vara, developed the tibial metaphyseal-diaphyseal angle (TMDA). Kline, et al.[6] observed that there is little femoral varus in infantile tibia vara. O'Neill and MacEwen[9] measured the femoral metaphyseal-diaphyseal angle (FMDA) and TMDA and found that children with a greater TMDA than FMDA were more likely to have a residual varus deformity. In the current study, we hypothesized that the ratio of the FMDA to TMDA (femoral-tibial ratio [FTR]) will more accurately differentiate physiologic bowing from infantile tibial vara.

The purpose of this study is threefold: (1) To determine the false-negative and false-positive error of the FTR for differentiating physiologic bowing from infantile tibial vara and compare this with the more commonly used TMDA; (2) to determine to the effect of rotation on the TMDA and FTR; and (3) to determine the reliability (intraobserver and interobserver error) in measurement of the TMDA and FTR.

Materials and Methods

This study is a retrospective review of all patients evaluated for genu varum at our institution from 1985 to 1995. Our inclusion criteria were adapted from Feldman and Schoenecker.[2] To be included in the study, patients had to have initial AP (patella forward) long leg radiographs taken before the age of 3 years; a tibiofemoral angle >10° of varus; no evidence of congenital, metabolic or traumatic etiology; and sufficient follow-up to determine the final diagnosis.

The diagnosis of physiologic bowing required the resolution (either clinically or radiographically) of the genu varum without treatment. The diagnosis of infantile tibia vara

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(Blount's disease) required persistence or worsening genu varum with radiographic changes consistent with Blount's disease, including radiolucency, sclerosis, and apparent fragmentation of the medial portion of the proximal tibia. Of 317 patients at the primary institution evaluated for genu varum, 46 met the study criteria, and an additional 23 with infantile tibia vara were solicited from other hospitals. A total of 69 patients (119 limbs) were studied, 38 patients with physiologic bowing and 31 with infantile tibia vara. The most common reasons for not meeting study criteria included: no radiographs; radiographs obtained after the age of 3 years; a tibiofemoral angle of <10°; or lack of follow-up. The demographics are listed in Table 1.

Table 1. Demographics

	Infantile Tibial Vara	Physiologic Bowing
Number of patients	31	38
Number of limbs	52	67
Bilateral (%)	68%	72%
Age at diagnosis (months)	28 ± 4	21 ± 4

Radiographic Measurements. All patients included in the study obtained long leg anteroposterior (AP) radiographs of the lower extremity with the patella forward. The following measurements were made from these radiographs: the tibiofemoral angle (TFA) as described by Salenius and Vankka;[12] the tibial metaphyseal-diaphyseal angle (TMDA) as described by Levine and Drennan;[8] the femoral metaphyseal-diaphyseal angle (FMDA) as adapted from O'Neill and MacEwen;[9] and the ratio of the FMDA to TMDA, referred to as the femoral-tibial ratio (FTR). The FTR equals the FMDA divided by the TMDA (Figure 1).

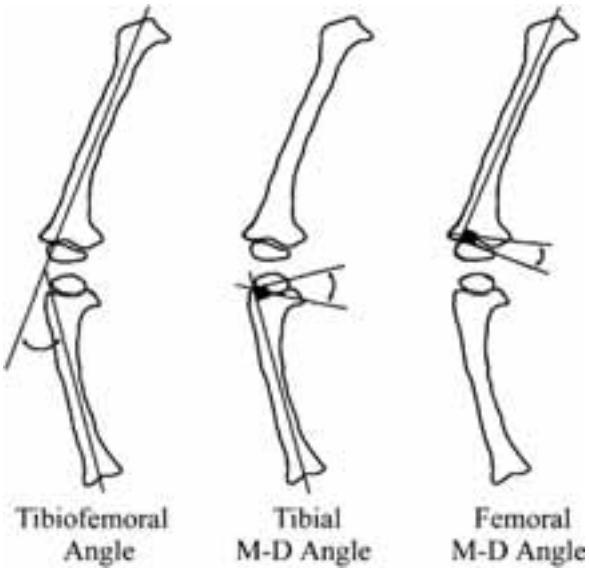

Figure 1. Schematic representation of the tibiofemoral angle (TFA), femoral metaphyseal-diaphyseal angle (FMDA), and tibial metaphyseal-diaphyseal angle (TMDA). The femoral-tibial ratio (FTR) = FMDA/TMDA.

The mean and standard deviation for the FMDA, TMDA and FTR were determined for each group (infantile tibia vara and physiologic bowing). Statistical significance was determined by the t-test, with $p < 0.01$ being considered significant.

False Negative and False Positive Error. The false-negative error is defined as the probability of a person with infantile tibia vara testing negative by the given radiographic criteria. The false-positive error is the probability of a person with physiologic bowing testing positive for infantile tibia vara.[2] The false-negative and false-positive errors were determined for all values of the TMDA and FTR. The cut-off value was determined by selecting the value of TMDA and FTR that produced the lowest combination of false-negative and false-positive errors.

The interval that would predict the correct diagnosis with risk of a false-positive or false-negative error less than 5% was determined for both the TMDA and FTR.[2]

Effects of Rotation. The effects of rotation on the TMDA and FTR were assessed by retrospectively evaluating 13 patients (25 limbs) that had AP radiographs of the lower extremity in both internal and external rotation on the same day. The values for the TMDA and FTR were compared in both internal and external rotation. The degree of rotation was not recorded. Significance was determined using a paired t-test, with $p < 0.01$ considered significant.

Intraobserver and Interobserver Error Measurement. To assess the intraobserver measurement error, one orthopaedic surgical resident measured the TMDA, the FMDA and the FTR from 10 standard AP weight bearing radiographs of the lower extremity three times in blinded fashion. To assess the interobserver measurement error, four orthopaedic residents measured the TMDA, FMDA and FTR on 10 long-leg AP radiographs of the lower extremity. A repeated measures of analysis of variance was carried out followed by the calculation of the intraclass correlation coefficient (ICC) for each measurement variable in each of the two designs. The ICC is a reliability coefficient that is calculated using variance estimates obtained through the analysis of variance. It reflects both the degree of correspondence and the agreement among ratings. For statistical purposes, if the TMDA was recorded as zero, it was given the value of one (to prevent the FTR from being infinite).

Results

Radiographic Measurements. The results of the radiographic measurements for the TFA, TMDA, FMDA, and FTR for physiologic bowing and infantile tibia vara are listed in Table 2. The mean values for each measurement were significantly different between the two groups ($P < 0.01$).

False Negative and False Positive Error. A TMDA >13° or an FTR <1 were prognostic for infantile tibia vara. These values were determined as producing the lowest combination of false-negative and false-positive errors (see Table 3).

Table 2. The Tibial Metaphyseal-Diaphyseal Angle (TMDA), Femoral Metaphyseal-Diaphyseal Angle (FMDA), and Ratio of the Femoral to Tibial Metaphyseal-Diaphyseal Angles (FTR) for Infantile Tibial Vara and Physiologic Bowing

	Infantile Tibial Vara	Physiologic Bowing
TFA (degrees)*	28 ± 11°	22 ± 8°
TMDA (degrees)*	17 ± 4°	8.4 ± 4°
FMDA (degrees)*	7.4 ± 5°	14 ± 5°
FTR*	0.48 ± 0.4	2.6 ± 3

*Represents a significant difference between infantile tibial vara disease and physiologic bowing $p < 0.01$.

Table 3. False Negative and False Positive Values for Varying Cutoff Values of TMDA and FTR

TMDA Value	False Negative	False Positive	FTR Value	False Negative	False Positive
Blount >9	0%	39%	Blount <0.7	27%	1%
Blount >10	10%	30%	Blount <0.8	15%	3%
Blount >11	10%	28%	Blount <0.9	12%	6%
Blount >12	13%	22%	Blount <1.0*	10%	7%
Blount >13*	23%	10%	Blount <1.1	8%	21%
Blount >14	31%	7%	Blount <1.2	8%	27%
Blount >15	48%	1%	Blount <1.3	6%	30%
Blount >16	52%	0%	Blount <1.4	4%	46%

*Lowest combination of false-negative and false-positive errors

Use of the TMDA >13° resulted in a false-negative error of 23% and a false-positive error of 10%. Use of the FTR <1 resulted in a false-negative error of 10% and a false-positive error of 7%.

Only when the TMDA was <9° or >16° did the risk of a false-negative or false-positive error fall below 5%. Therefore a TMDA between 9 and 16 is a "gray zone," and 42% of the patients in our study fell within this "gray zone." When the FTR was ≤0.7 or ≥1.4, the risk of a false-negative or false-positive fell below 5%. Therefore a FTR between 0.7 and 1.4 is also a "gray zone," into which 24% of our patients fell.

Effects of Rotation. The mean TMDA changed by 3.2° and the FTR changed by 0.1 between internal and external rotation. The difference in the FTR was not significant between internal and external rotation ($p = 0.91$), whereas it was for both the TFA ($p < 0.01$) and the TMDA ($p < 0.01$). The TFA, TMDA, and FTR for both internal and external rotation are listed in Table 4.

Table 4. The Tibiofemoral Angle, TMD Angle and FTR for Both Internal and External Rotation

	Internal Rotation	External Rotation	P value
TFA*	16.0 ± 9.8°	23.5 ± 8.1°	0.0004
TMDA*	6.3 ± 4.2°	9.5 ± 3.7°	0.002
FTR	2.4 ± 2.6	2.3 ± 3.9	0.91

*Represents a significant difference between internally and externally rotated values $p < 0.01$.

Intraobserver and Interobserver Error. The intraclass correlation coefficient (ICC) for the intraobserver and interobserver testing is shown in Table 5, demonstrating good intraobserver and interobserver reliability for both the TMDA and the FTR.

Table 5. Intraobserver and Interobserver Error Measurements (Intraclass Correlation Coefficient)

	Intraobserver ICC	Interobserver ICC
FMDA	0.94	0.92
TMDA	0.99	0.79
TFR	0.98	0.78

Discussion

In this study we found that the FTR, defined as the ratio of the FMDA to the TMDA, better differentiates infantile tibia vara (Blount's disease) from physiologic bowing than does the TMDA alone. The FTR results in lower false-negative and false-positive errors, with less rotational effect.

In an effort to differentiate physiologic bowing from infantile tibia vara, several clinical radiographic and parameters have been used. Langenskiöld and Riska[7] defined radiographic bony changes and developed a classification system, although these changes are usually seen only after 24 months of age. The TFA has been shown to vary little between the two groups and is therefore a poor predictor of infantile tibia vara.[2] Levine and Drennan[8] introduced the TMDA as a means to identify infantile tibia vara. They found that only one of 30 patients with a TFA >11° did not develop infantile tibia vara, and only 3 of 58 patients with a TFA <11° had radiographic changes consistent with infantile tibia vara. The false-negative rate was 9% and false-positive rate was 2%. Feldman and Schoenecker[2] showed a similar false negative rate of 9% but a much higher false positive rate of 33% when 11° was used as the cut-off value. Our results, using a TMDA of ≥11° as the criteria for infantile tibia vara, were similar to Feldman and Schoenecker's.[2] We had a 10% false-negative rate and a 30% false-positive rate. The discrepancy between the studies may be due to a difference in the age of the children. Levine and Drennan's[8] criteria included only children over 18 months, whereas Feldman and Schoenecker[2] (as with our study) included children from birth to age 3.

Feldman and Schoenecker[2] found that only when the TMDA was 9° or less, or 16° or more, did the risk of a false-negative or false-positive error become less than 5%. Unfortunately, a large percentage of their patients fell into this "gray zone" (greater than one-third, as estimated from their histogram). Our "gray zone" (value of TMDA where the false-negative and false-positive rates are >5%) was also 9°

or less, or 16° or more. A total of 42% of our patients fell into this "gray zone". When the FTR was ≤0.7 or ≥1.4, the risk of a false-negative or false-positive fell below 5%. Therefore a FTR between 0.7 and 1.4 is also a "gray zone," which included only 24% of our patients.

Rotation appears to have less of an effect on the FTR than the TMDA. Henderson, et al.[5] reported the effect of rotation (neutral, 25° internal and external rotation) in one patient. They found a small (2.8°) but significant difference between the neutral and rotated measurements of the TMDA. Stricker & Faustgen14 examined the effect of rotation on the TMDA in 17 patients (33 limbs) and found a similar difference of 1.4° with 30° of external rotation. We also found a small (3.2°) but significant difference in the TMDA with rotation, but no statistically significant difference in the FTR with rotation.

The variability of these measurements have been previously reported. Henderson and Kemp[4] reported a 1.4° intraobserver error for measurement of the TMDA and a 2° interobserver error. Stricker & Faustgen14 also reported a low intraobserver error (0.8°) for the TMDA. We found a high intraclass correlation for the FMDA, TMDA and FTR (Table 5).

One of the shortcomings of this study is that the infantile tibia vara and physiologic bowing groups differ by age at the time of initial evaluation. We attempted to evaluate this by calculating the false-negative and false-positive error for only those children 2 years of age and older at the time of initial radiographs. This included all patients with infantile tibia vara, but only 7 (18%) of the children with physiologic bowing. The mean age was 27 months for the children with physiologic bowing as compared to 28 months for those with infantile tibia vara. The false-negative error was unchanged and the false-positive error was 8% using the FTR and 21% using the TMDA.

Conclusion

In this study we found the FTR to be a more accurate predictor for differentiating infantile tibia vara from physiologic bowing, than the TMDA. The FTR results in lower false-negative and false-positive error than the TMDA, with less rotational effect.

Acknowledgements

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Reprint

National Athletic Trainers' Association Position Statement: Head-Down Contact and Position Spearing in Tackle Football

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Objective. To present recommendations that decrease the risk of cervical spine fractures and dislocations in football players.

Background. Axial loading of the cervical spine resulting from head-down contact is the primary cause of spinal cord injuries. Keeping the head up and initiating contact with the shoulder or chest decreases the risk of these injuries. The 1976 rule changes resulted in a dramatic decrease in catastrophic cervical spine injuries. However, the helmet-contact rules are rarely enforced and head-down contact still occurs frequently. Our recommendations are directed toward decreasing the incidence of head-down contact.

Recommendations. Educate players, coaches, and officials that unintentional and intentional head-down contact can result in catastrophic injuries. Increase the time tacklers, ball carriers, and blockers spend practicing correct contact techniques. Improve the enforcement and understanding of the existing helmet-contact penalties.

Key Words. catastrophic injuries, cervical spine, head injuries, injury prevention, neck injuries, paralysis, quadriplegia.

Catastrophic cervical spine injuries (CSI) resulting in quadriplegia (paralysis of all four extremities) are among the most devastating in all of sport. In football the primary mechanism for these injuries is axial loading that occurs, whether intentional or unintentional, as a result of head-down contact and spearing. Head first contact also increases the risk of concussion and closed head injury. In 1976, the National Collegiate Athletic Association (NCAA) and the National Federation of State High School Associations (NFSHSA) changed their football rules to broaden the concept of spearing to include any deliberate use of the helmet as the initial point of contact against an opponent. They did this in an effort to reduce the incidence of catastrophic CSI.

Subsequent data on the occurrence of quadriplegia in organized football dramatically demonstrated that the NFSHSA and NCAA rule changes were successful. The incidence has remained at a relatively low level, with a mild increase at the end of the 1980s (Figure 1). However, in spite of this accomplishment, head-down contact still occurs frequently. The helmet-contact penalties also are not enforced adequately. Clearly, a reduction in the incidence of head-down contact and increased enforcement of the existing rules will further reduce the risk of both paralytic and non-paralytic injuries.

The purpose of this position statement is to 1) provide scientifically proven concepts and recommendations to minimize the risk of catastrophic CSI in football; 2) clarify that head-down contact and spearing pose a risk to all positional players regardless of intent; 3) establish the value and necessity of ongoing educational practices for players, coaches, and officials regarding dangerous and proper playing techniques; 4) emphasize that increasing safety is dependent on the participation of sports medicine professionals, coaches, players, officials, administrators, and governing bodies.

Recommendations

The National Athletic Trainer's Association (NATA) recommends the following regarding head-down contact and spearing in football. These recommendations should be considered by sports medicine professionals, coaches, players, officials, administrators, and governing bodies who work with athletes at risk for cervical spine injuries.

Practices and Concepts

1. Axial loading is the primary mechanism for catastrophic CSI. Head-down contact, defined as initiating contact with the top or crown of the helmet, is the only technique that results in axial loading.

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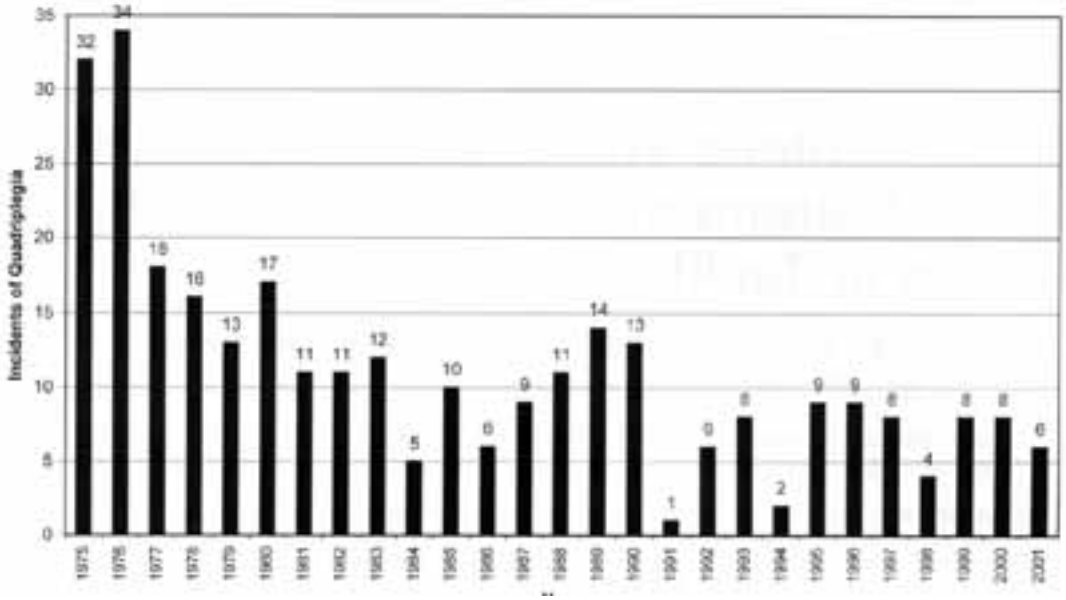

Figure 1. Incidence of quadriplegia in high school and college athletes. Data from the National Football Head and Neck Injury Registry (1976–1991) and the National Center for Catastrophic Sports Injury Research (1992–present).[1–4]

2. Spearing is the intentional use of a head-down contact technique. Unintentional head-down contact is the inadvertent dropping of the head just before contact. Both head-down techniques are dangerous and may result in axial loading of the cervical spine and catastrophic injury (Figure 2).

3. Catastrophic CSI resulting from axial loading is neither caused nor prevented by players' equipment.

4. Injuries that occur as a result of head-down contact are technique related and are preventable to the extent that head-down contact is preventable.

5. Attempts to determine a player's intent regarding intentional or unintentional head-down contact are subjective. Therefore, coaching, officiating and playing techniques must focus on decreasing all head-down contact, regardless of intent.

6. Catastrophic CSI occurs most often to defensive players. However, all players are at risk. Ball carriers and blockers have also become quadriplegics by lowering their heads at contact. Expanding the concept of head-down contact beyond tackler spearing and the "intentional attempt to punish an opponent" will decrease the risk of serious injury players in other positions.

7. As emphasized in the college and high school rulebooks, making contact with the shoulder or chest while keeping the head up greatly reduces the risk of serious head and neck injury. With the head-up, the player can see when and how impact is about to occur and can prepare the neck musculature for impact. Even if inadvertent head-first contact is made, then force is absorbed by the neck musculature, the intervertebral discs, and the cervical facet joints. This is the safest contact technique.

Figure 2. Head-down contact poses significant risks of catastrophic cervical spine injury. This defensive back (dark jersey) sustained fractures of his 4th, 5th, and 6th cervical vertebrae. The hit resulted in quadriplegia.

8. Each time a player initiates contact with his head-down he risks paralysis. Therefore, increased attention to the frequency of head-down contact occurring in games and practices is needed. It is a reasonable conclusion that a reduction in the cause (head-down contact) will further reduce the effect (catastrophic CSI).

9. Data collection on all catastrophic CSIs is important. Attention to the number of nonparalytic cervical spine fractures and dislocations is needed as each incident has the potential for paralysis. These data are less reliable and harder to obtain than data for paralytic injuries. Both injury types require diligent reporting to the National Center for Catastrophic Sports Injury Research (mailing address: CB 8700, 204 Fetzer Gymnasium University of North Carolina, Chapel Hill, NC 27599-8700. Email: mueller@email.unc.edu).

Rules and Officiating

10. Officials should enforce the existing rules to further reduce the incidence of head-down contact. A clear discrepancy exists between the incidence of head-down/head-first contact and the level of enforcement of the helmet-contact penalties. Stricter officiating would bring more awareness to coaches and players about the effects of head-down contact.

11. The current annual education programs for all officials should emphasize the purpose of the helmet-contact rules and the dangers associated with head-down/head-first contact. Emphasis should be on the fact that the primary purpose of the helmet-contact penalties is to protect the athlete who leads with his head. Although the technique is dangerous to both players, it is the athlete who initiates head-down contact who risks permanent quadriplegia.

12. Not all head-first contacts that result in serious injury are intentional. A major area of concern for officials remains application of the penalties to athletes who unintentionally initiate contact with their helmets. Athletic governing bodies should address this issue in order to improve penalty enforcement.

13. Athletic governing bodies should coordinate a protocol to document and quantify all penalties called through their organizations. This will determine the enforcement level of the helmet-contact penalties.

14. The athletic governing bodies should periodically survey their football officials regarding their interpretations and perceptions of the helmet-contact rules. Existing rules and comments need to specifically include the ball carrier in the application of these penalties.

15. Those preparing the football rule books should consider revising the wording "blocking and tackling techniques" with "contact techniques" (or similar). This revised wording would then include all positional players and all types of contact.

16. A task force of athletic trainers, coaches, team physicians, officials, and league administrators should be developed at all levels of play to monitor rule enforcement and the frequency of head-down contact by an annual, random review of game films.

Education and Coaching

17. The athlete should know, understand and appreciate the risk of making head-down contact, regardless of intent. Formal team educational sessions (conducted by the athletic trainer and team physician or both with the support of the coaching staff) should be held at least twice per season. One session should be conducted before contact begins and the other at the midpoint of the season. Recommended topics to include are: mechanisms of head and neck injuries, related rules and penalties, the incidence of catastrophic injury, the severity and prognosis of these injuries and the safest contact positions. The use of videos such as *Prevent Paralysis: Don't Hit With Your Head,*[5] *See What You Hit,*[6] or the prevention portion of *Spine Injury Management*[7] should be mandatory (Table 1). The use of supplemental media and materials are strongly recommended.

Table 1. Available Videos

Title	Available From
Prevent Paralysis: Don't Hit With Your Head[5]	Dick Lester, Riddell Inc E-Mail: dlester@riddellsports.com Cost: Free
See What You Hit[6]	The Spine in Sports Foundation www.spineinsports.org Cost: Free
Spine Injury Management[7]	Human Kinetics www.humankinetics.com Cost: $39.95

18. Correct contact technique should be taught at the earliest organized level. Pop Warner, Midget, and Pee Wee football leagues should perpetually emphasize the importance of coaching and teaching heads-up football.

19. It is crucial that educational programs extend to the television, radio and print media for both local and national affiliates regarding the dangers of head-down contact and the reasons for the helmet-contact rules. This will promote awareness of these issues and provide extended education to viewers, listeners and readers.

20. Initiating contact with the shoulder/chest while keeping the head up is the safest way to play football. The game can be played as aggressively with this technique with much less risk of serious injury (Figure 3). However, it is a technique that must be learned. To be learned it must be practiced extensively. Athletes who still drop their head just before contact require additional practice time. It is imperative for coaches to teach, demonstrate, and practice this technique throughout the year for all position players. Tacklers, ball carriers and blockers must receive enough practice time until it is instinctive to keep the head-up.

21. Initiating contact with the face mask is a rules violation and must not be taught. If the athlete uses poor technique by lowering his head, he places himself in the head-down position and at risk of serious injury.

Figure 3. Initiating contact with the shoulder while keeping the head up reduces the risk of catastrophic injury, as demonstrated by the blocker and potential tackler.

22. Every coaching staff must display and implement a clear philosophy regarding the reduction of head-down contact. The head coach should clearly convey this philosophy to the assistant coaches and the entire team, and pursue an enforcement policy during practice. A player's technique must be corrected anytime he is observed lowering his head at contact. Coaches should also use weekly game film reviews to provide players feedback about their head positions.

23. Athletes should have a year-round supervised neck-strengthening program with appropriate equipment and techniques. Although the role of strength training is secondary to correcting contact technique in axial-loading injury prevention, it provides the strength and endurance required to maintain the neck in extension. It also provides protection against cervical nerve root neuropraxia (burners).

24. Schools, responsible administrators, and the sports medicine team should recognize cyclic turnover in coaches and establish programs that educate new and re-educate existing coaches to appropriate teaching and practicing methods. This will provide a documented and consistent approach to the prevention of these injuries.

History and Background

In 1931 the American Football Coaches Association (AFCA) compiled the first Football Fatality Report.[8] By 1962, its findings caused the American Medical Association Committee on Medical Aspects of Sports to host a national conference on head protection for athletes.[8] The conference convened the principal authorities of that era in what was emerging as "sports medicine" to discuss the current issues adhering to changes in the football helmet and the advent of the football face mask. The focus was the rapidly rising fatality rate among high school and college football players suffering from closed head injuries. Football authorities were divided as to whether the new protective headgear was good for the sport.

Into the 1970s, opinion was more prevalent than scientific data in addressing these problems. The American Medical Association Committee arrived at a collective expert opinion and encouraged pragmatic scholarly attentions to the health and safety issues within sport. Among the recommendations resulting from the 1962 conference were condemning the practice of spearing and the need for research to develop standards for football helmets.[9] Initially, spearing was defined by rule as "intentionally and maliciously striking the opponent with one's helmet after the opponent had been downed."

After the 1962 conference, Blyth from the University of North Carolina assumed the data collection for the Fatality Report of the American Football Coaches Association.[10] Helmet manufacturers began to sponsor research on impact standards for helmets, and high school and college rules committees confirmed that spearing was an illegal form of football contact after the whistle.

American Medical Association Position Statement

The practice of teaching "face into the numbers" was growing in the 1960s as the helmets evolved and coaches felt that players could therefore better withstand the use of the helmeted head.[8, 11, 12] "Face into the numbers" was increasingly popular because it allowed the blocker or tackler to keep his eyes forward, neck "bulled" and to move with the opponent, without having the intent to spear.[8] In essence, coaches considered using the helmet as the primary point of contact as a superior technique.

In 1967, the American Medical Association Committee on Medical Aspects of Sports declared, in a groundbreaking position statement, its opinion that most spearing was unintentional and non-malicious, i.e., "inadvertent".[11] It identified the flaw with teaching "face-into-the-numbers" contact. Athletes do not always execute with precision, and the tendency to duck the head at contact is natural. This position statement was adopted by the National Federation of State High School Associations (NFSHSA) as a joint statement in 1968.

Football Helmet Standards

In spite of this timely recognition of unsafe head position, the annual football fatality data reports revealed a continued rise in frequency during the 1960s.[10] Although it was reported that the risk of death from football did not exceed the actuarial risk of death among males of that age in non-football activities,[13] the need for helmet design standards became more and more evident.

Consequently, the helmet manufacturers agreed in 1969 to pool their resources through a newly devised interdisciplinary National Operating Committee for Safety In Athletic Equipment (NOCSAE).[8] This committee was charged with the development of consensus standards for helmets in football by an independent investigator. Hodgson, from Wayne State University, was selected as the investigator because of

his extensive research in this area.[14] A safety standard was achieved in 1973, and the first helmets were tested on the NOCSAE standard in 1974.[12] The NOCSAE standards went into effect for colleges in 1978 and high schools in 1980.[15] It was commonly understood that the helmets being produced and used by the mid-1970s met the NOCSAE, and standards and all helmets being worn were, in fact, associated with the same low rates of clinical concussions.[16]

The increase in head injury fatalities throughout the 1960s and early 1970s was attributed to the introduction of hard-shell helmets and face masks in the early 1960s, which resulted in playing techniques that increased exposure of the head to contact.[1, 8] Helmet standards and head injuries received football's priority attention during this time.[8] Similar attention to serious neck injuries in the 1960s was lacking because the incidence of nonfatal quadriplegia was not being tracked and therefore was an unknown.

Catastrophic Injury Data

The Annual Football Fatality Report was the only ongoing source of data into the 1970s. Schneider[17] included serious neck injuries in his landmark survey of catastrophic injuries in football in the early 1960s. But it was not until the mid 1970s that 2 concurrent and independent studies by Clarke,[18] and Torg et al.[19, 20] again examined quadriplegia. These data revealed the increased incidence of paralyzed football players.

The total number of head and neck injuries from 1971 to 1975[19, 20] was calculated and retrospectively compared with the data from 1959 to 1963 compiled by Schneider.[17] The number of intracranial hemorrhages and deaths had decreased by 66% and 42%, respectively. This suggested the new helmet standards had been effective in minimizing serious head injuries. However, the number of cervical spine fractures, subluxations, and dislocations had increased by 204% and the number of athletes with cervical quadriplegia had increased by 116%.

Clarke and Torg led the proponents of the spearing rule changes that were implemented by the NFSHSA and NCAA in 1976. These rule changes preceded the publication of their data.[18–20] The purpose of the rule changes was to protect the spearer, whether inadvertent or intentional, from neurotrama.[5, 8, 11, 12, 15, 21–25] On the basis of these data, it was concluded that improved protective capabilities of the polycarbonate helmets accounted for a decrease in head injuries, but encouraged playing techniques that used the top or crown of the helmet as the initial point of contact and put the cervical spine at risk.[1]

The results of the 1976 rule change are an example of one of the most successful injury interventions in sport (Figures 1 and 4). In the first year after the rule change the number of injuries resulting in quadriplegia in high school and college decreased by 53%.[1] By 1984, the number dropped by 87%. Other than increases in 1988, 1989, and 1990 to the low teens, these cases have remained in the single digits through

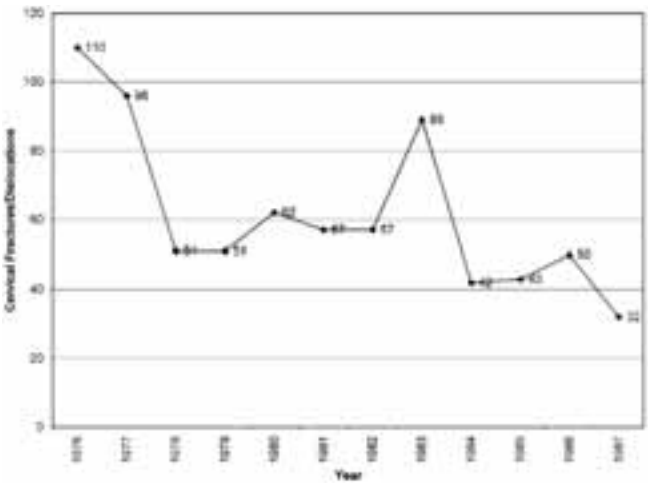

Figure 4. Incidence of cervical fractures and dislocations in high school athletes. Data from the National Football Head and Neck Injury Registry.

the most recent years of available data. This decrease is attributed to the rule change and to improved coaching techniques at the high school and college levels.[8, 12, 15, 19, 23, 24, 26–34]

In order to track nonfatal catastrophic injuries, Torg et al.[1] established the National Football Head and Neck Injury Registry in 1975, which collected data on CSIs through the early 1990s. In 1977, the NCAA initiated funding for a National Survey of Catastrophic Injuries directed by Mueller and Blyth.[2–4] In 1982, this project was expanded to include all sports and renamed the National Center for Catastrophic Sports Injury Research. Both projects used similar methods of collecting data. These sources included coaches, school administrators, medical personnel, athletic organizations, a national newspaper-clipping service and professional associates. The collection of these data was crucial in preventing catastrophic injuries.[12]

In 1987, a joint endeavor was initiated between the National Center for Catastrophic Sports Injury Research and the section on Sports Medicine of the American Association of Neurological Surgeons. As a result, Cantu became responsible for monitoring the collected medical data.[2] This project continues to collect data on these injuries.

Mechanism of Injury

In the early 1970s, several theories existed regarding the mechanisms of CSIs and quadriplegia. The theories of hyperflexion and hyperextension, based on post injury radiographs, were considered 2 primary causes.[1] Forced hyperflexion was considered a primary cause of severe CSI in football and other sports.[1, 35–57] Hyperextension and the concept of the posterior rim of the helmet acting as a guillotine also received attention as an injury mechanism.[58–62] Both of these injury mechanisms received acceptance throughout the 1970s.

In contrast to these early theories, Torg et al.[19, 20] determined that most cases of permanent quadriplegia occurring

between 1971 and 1975 were due to head-down contact or direct compression to the cervical spine. This resulted from the player initiating contact with the top of his helmet. The direct compression or axial loading concept eventually replaced the numerous other inaccurate, theoretical mechanisms of CSI.

The identification of an accurate mechanism of injury was vitally important to the prevention of these injuries.[12, 30] This allowed the development of a precise plan to reduce the incidence of quadriplegia.[8] Axial loading is now accepted as the primary cause of cervical spine fracture and dislocation in football. Numerous studies have supported the role of axial loading[20, 63–97] in catastrophic CSI and refuted the role of hyperflexion and hyperextension in these injuries.[1, 19, 23, 24, 30–32, 63–65, 68, 72, 94, 98, 99]

Axial Loading. In the course of contact activity, such as football, the cervical spine is repeatedly exposed to dangerous energy inputs.[93] Fortunately, most forces are dissipated by controlled spinal motion through the cervical paravertebral muscles, eccentric contractions and intervertebral discs.[19] However, the vertebrae, intervertebral discs and supporting ligamentous structures can be injured when contact occurs on the top or crown of the helmet with the head, neck, and trunk positioned in such a way that forces are transmitted along the vertical axis of the cervical spine. In this situation the cervical spine can assume the characteristics of a segmented column. With the neck in the neutral position, the cervical spine is extended as a result of normal cervical lordosis (Figure 5). When the neck is flexed to 30 degrees, the cervical spine becomes straight. When a force is applied to the vertex, the energy is transmitted along the longitudinal axis of the cervical spine and is no longer dissipated by the paravertebral muscles. This results in the cervical spine being compressed between the abruptly decelerated head and the force of the oncoming trunk.[65] Essentially, the head is stopped, the trunk keeps moving, and the spine is crushed between the two. When the maximum vertical compression is reached, the cervical spine fails in a flexion mode, with a fracture, subluxation, or facet dislocation resulting.[63] In the laboratory, fracture or dislocation has occurred with less than 150 ft-lb of kinetic energy.[28]

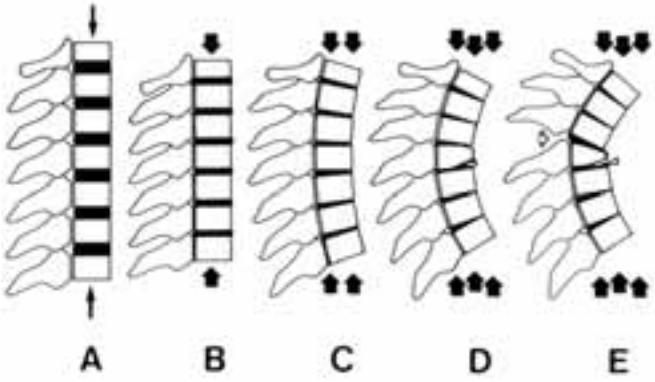

Figure 5. (A) Axial loading of the cervical spine (B) first results in compressive deformation of the intervetebral discs. As the energy input continues and maximum compressive deformation is reached, angular deformation and buckling occur (C). The spine fails in a flexion mode, with resulting fracture, dislocation, or subluxation (D and E).

Distribution of Serious Injuries

Defensive football players receive the majority of fatalities and catastrophic CSI, accounting for approximately 4 times those of offensive players.[2–4, 12, 15, 19, 20, 23] Tackling is the leading cause, followed by being tackled and then blocking.[2–4, 12, 15] By0 position, defensive backs and special teams players are at the greatest risk[2, 3, 12, 15, 16, 19, 23] with ball carrier positions, linebackers and defensive lineman having the next highest incidences of serious injury.[2, 12, 15]

Each time a player initiates contact with his head-down, he risks quadriplegia.[2, 15, 19, 22–25, 27–33, 97, 100–106] Each time an athlete initiates contact head first, he increases the risk of concussion.[22, 29, 101–103, 107, 108] Although catastrophic injuries have occurred to position players at varying rates, mechanism of injury does not discriminate by position or intent.[103, 105, 107, 109] Head-down contact poses a risk to every player who employs this technique.[22, 103–105]

Incidence of Head-Down Contact

According to Hodgson and Thomas,[28] the number of paralyzed players does not accurately identify the risk of hitting with the head down. Because of the decrease in catastrophic injuries since the 1976 rule changes, it is often assumed that head-down contact rarely occurs. Two authors have examined the incidence of head-down contact in the 1990s:[22, 101, 103] twice on film in slow motion and once in live situations. Selected data appear in Tables 2 and 3.

One study compared the incidence of head-down contact between tacklers and ball carriers before and after the rule change on the high school level.[103] No significant change

Table 2. Percentage of Plays Involving at Least 1 Head-Down Contact Between Tacklers and Ball Carriers During a 1990 High School Season[103]

Play	%
All plays	25
Running plays	37
Kick returns	38
Pass plays	7

Table 3. Percentage of Plays Involving Head-Down Contact by High School and College Tacklers or Ball Carriers

Position	%
Tacklers, film (1990)[103]	26
Tacklers, live (1993)101	6
College tacklers, live (1993)[101]	8
Ball carriers, film (1990)[103]	16
Ball carriers, film (1989)[19]	20

was seen in the incidence of head-down contact between the seasons. Approximately 20 head-down contacts occurred per team in a single game. There was 1 head-down contact for every 1.8 kick returns Special teams players have been among the leading position players associated with catastrophic injuries. Considering that kicking plays account for only about 7% of the plays involving a ball carrier, this play is probably the most dangerous play in football.

Ball-carrier spearing (Figure 6) is interesting in that defensive players were 4 times more likely to hit with their head down when tackling a head-down ball carrier. It is possible that a head-down ball carrier influences a tackler to "get lower" and use a similar technique.[22, 103] This coincides well with Drakes'[101, 102] finding that tacklers were 3 times more likely to make head-down contact when tackling below the waist.

Figure 6. Ball-carrier head-down contact, and often overlooked danger, increases the risk of head and neck injuries.

During the 1990 season, 200 head-down contacts occurred during one team's season, and an estimated 2.8 million head-down contacts took place nationally between tacklers and ball carriers on the high school level. This translated into approximately 1 case of quadriplegia for every 251,000 head-down contacts. Based upon these numbers, a high school should have 1 case of quadriplegia for every 11,000 games.[103] Although these numbers are rough estimates at best, they demonstrate the room for additional improvement in decreasing the incidence of spearing and head-down contact.

Rules and Officiating

The current helmet-contact rules for high school and college are shown in Tables 4 and 5, respectively. In 1976, the high school rule change defined butt-blocking and face tackling and made them illegal. It was also a "point of emphasis" that coaches could no longer teach "face in the numbers" as a contact technique.[112] On the collegiate level, the rules were adapted to make "deliberate" use of the helmet illegal. Also, the rule book included a "Coaching Ethics" statement from the American Football Coaches Association that the helmet cannot be used as a primary point of contact in the teaching of blocking and tackling.[113] Since 1976, 2 significant changes to the helmet-contact rules have been made. First, in the mid 1980s the spearing penalty was lessened from an automatic ejection to a 15 yard penalty. Second, in the early 1990s, the NCAA made the face mask an official part of the helmet.

Table 4. Helmet-Contact Rules and Selected Comments from the 2002 National Federation of State High School Associations' *Official Football Rules*[110]

Rules
1. Spearing is the intentional use of the helmet in an attempt to punish an opponent.
2. Face tackling is driving the face mask, frontal area, or top of the helmet directly into the runner.
3. Butt blocking is a technique involving a blow driven directly into an opponent with the face mask, frontal area, or top of the helmet as the primary point of contact either in close line play or in the open field.
4. Illegal personal contact occurs when a player intentionally uses his helmet to butt or ram an opponent.
Points of Emphasis
1. Illegal acts such as spearing, face tackling, and butt blocking should always be penalized.
2. Coaches have the responsibility to teach the proper technique of blocking and tackling. Officials have the responsibility to penalize all illegal contact.
Shared Responsibility and Football-Helmet Warning Statement
1. The rules against butting, ramming, or spearing the opponent with the helmeted head are there to protect the helmeted person as well the opponent being hit. The athlete who does not comply with these rules is a candidate for catastrophic injury.
2. The teaching of the blocking/tackling techniques which keep the helmeted head from receiving the brunt of the impact is now required by rule and coaching ethics.

Table 5. Helmet-Contact Rules and Comments in the National Collegiate Athletic Association's *2001 Football Rules and Interpretations*[111]

Rules
1. Spearing is the deliberate use of the helmet (including the face mask) in an attempt to punish an opponent.
2. No player intentionally shall strike a runner with the crown or top of the helmet.
3. No player intentionally shall use his helmet (including the face mask) to butt or ram an opponent.
Points of Emphasis
1. The NCAA Rules Committee is strongly opposed to tackling and blocking techniques that are potentially dangerous for both the tackler/blocker and the opponent.
2. Coaches are reminded to instruct their players not to initiate contact with any part of their helmets, including the face mask.
Coaching Ethics
The following are unethical practices:
1. Using the football helmet as a weapon. The helmet is for the protection of the players.
2. Spearing. Players, coaches, and officials should emphasize the elimination of spearing.

Although the rule change is credited with reducing catastrophic injuries, the role officials have played by enforcing these rules is questionable (Table 6). To illustrate this, in 2001 college officials called 1 spearing penalty in every 73 games and 1 butting or ramming penalty in every 156 games. No spearing penalties were called in 12 of the 20 major Division 1 conferences.[115] During the 1992 NCAA season, officials called 55 spearing penalties (1 in every 21 games) and 16 related to butting or ramming.[115]

Table 6. Selected 2001 National Collegiate Athletic Association Penalty-Enforcement Data from Major Division 1 Conferences[114]

Penalty Type	No. Called
Total penalties	20 837
Holding	3347
Face mask	945
Spearing	17
Butting or ramming	8

On the high school level, officials called an estimated 1 spearing penalty in every 20 games.[116] During one team's high school season, no spearing penalties were called.[20] This appears to be the norm, rather than the exception. These data contradict the NFSHSA recommendation that infractions involving a safety issue should always be enforced.[110, 117] At this level of enforcement it is doubtful whether actual penalties have decreased the incidence of head-down contact or the mechanism of injury.[116] If illegal helmet contact is not penalized, the message is sent that the technique is acceptable.[118] Adequate enforcement of the rules will clearly further reduce the risk of catastrophic injuries.[4, 12, 15, 21–23, 29–31, 101, 102, 106, 115]

Surveys of football officials have revealed many inconsistencies with regard to the helmet-contact penalties. Football officials may not have a uniform understanding of these rules. Fifty percent of New Jersey officials felt that all head-first contact was illegal.[116] Thirty two percent felt that the rules were difficult to interpret.[116] Another 38% were unsure whether the rules were written in a way that allowed easy enforcement.[116] A survey of college officials found similar results regarding the wording of the rules.[115] A large number of high school and college officials believed that determining an athlete's intent made the rules difficult to enforce.

The helmet-contact penalties are unique in football because they are the only action penalties that penalize a player for his own protection.[105, 109] However, many officials and coaches erroneously perceive the primary purpose of the penalties as protecting the athlete who gets hit.[105, 109, 115, 116] This is reflected by one group's findings that nearly one third of high school players did not know that it was illegal to tackle with the top of the helmet or run over an opponent head first.[119]

Despite the intention of the 1976 rule change to address unintentional or inadvertent spearing, the primary rule still has an association with the "intentional attempt to punish". The wording of the helmet-contact rules does imply the need for intent.[116] On the college level the rules do not address unintentional head-down contact at all. High school rules do address head-down contact through the penalties for face tackling and butt blocking; however, these rules exclude mention of the ball carrier. Although rules do exist at the high school level, officials may enforce them even less than they enforce the spearing penalty.[116, 120] Football's objective should be to alter athlete behavior to eliminate head-down contact, not merely to discourage it.[121]

An appropriate inquiry, which cannot be answered, is, "How many of the approximately 200+ hits resulting in paralysis were flagged at the time of contact?" Although a penalty flag on a play that involves a head or neck injury cannot prevent that injury, it may prevent one later on.[120] In reviewing the video *Prevent Paralysis: Don't Hit With Your Head,* football officials did not feel the rules allowed them to penalize the majority of the hits demonstrated on this film that resulted in quadriplegia.[122] A "litmus test" for the enforcement of the helmet-contact rules is their application to actual hits that have resulted in paralysis. There is no better definition of the type of contact that we have to eliminate.

Safest Contact Position

Initiating contact with the shoulder while keeping the head up is the safest contact position.[2–4, 11, 12, 15, 22, 28, 49, 103, 110, 123] With the head up, the athlete can see when and how impact is about to occur and can prepare the neck musculature for impact. This information applies to all positional players, including ball carriers. The game can be played as aggressively with this technique with much less risk of serious injury. Tacklers can still "unload" a big hit and ball carriers can still break tackles.[105, 109]

Conversely, with the head down, the athlete does not have the advantage of good vision and preparing for the instant of contact. He is likely to receive the full force of the impact on the head instead of the shoulders, chest, or arm. He is more apt to hit low on the opponent's body (including the opponent's hard driving knees), and exposes his cervical spine to impact in its most vulnerable position.[11] Albright et al.[124] found that college and high school players had sufficient nonfatal CSIs to warrant concern over the teaching of head-butting techniques.

Coaches have expressed that they have taught players to tackle correctly, but the players still have a tendency to lower their heads just before contact.[15, 28] It seems that players have learned to approach contact with their head up, but they need to maintain this position during contact.[103–105, 109] It is instinctive for players to protect their eyes and face from injury by lowering their heads at impact.[22, 103–105, 109] Coaches must spend enough practice time to overcome this instinct. Players who drop their heads at the last instant are demonstrating that they need additional practice time with correct contact

techniques in game-like situations. In addition to teaching correct contact in the beginning of the season, coaches should put specific emphasis on this 3 more times throughout the season.[21, 22, 104]

The "See What You Hit" concept has gained popularity in recent years. It is intended to teach athletes to keep their heads up and can be an effective tool. However, caution is required to ensure that coaches and athletes do not misinterpret this slogan as support for initiating contact with the face mask.

Strengthening the neck musculature is an accepted part of neck-injury prevention.[15, 29, 49, 97, 100] Although such strengthening cannot prevent axial loading in the head-down position, it can help athletes keep the head up during contact. Athletes should have access to some type of neck-strengthening equipment, and ideally, the program should be year round. If this is not possible, then adequate time (4 to 6 weeks before the season begins) should be allowed for strength gains. During the season, athletes should continue to lift at least 1 day per week to maintain their strength levels.[125]

Litigation

The occurrence of a catastrophic head or neck injury is characteristically accompanied by litigation.[126–140] The proliferation of litigation for these injuries began in the 1980s. Multi-million dollar verdicts are now common. Of the $45.8 million awarded in verdicts between 1970 and 1985, $38.7 million was awarded between 1980 and 1985.[126, 127] Ironically, the litigation in football is inversely proportional to the injury statistics. During the period when there was a drastic decrease in catastrophic injuries, litigation increased.[126] Any allegation of any fault can have devastating financial consequences for school districts, coaches, medical personnel and equipment manufacturers.

The increase in litigation had serious effects on the football helmet industry. Between 1975 and 1985, 11 of 14 football helmet manufacturers left the marketplace.[126] This exit from the marketplace was due to the cost of defending product liability claims[126] and not from shortcomings regarding the NOCSAE helmet standards. Dramatic increases in liability insurance premiums followed the increase in litigation. At that point, many helmet manufacturers became self-insured or accepted the risk of being underinsured. Approximately 40% of the helmet price was set aside for product liability.[126] Litigation will continue, and medical practitioners will have to determine, as the helmet manufacturers die, if they can afford to work in athletics.[126] For these individuals and others, the implications of the increase in the number of athletic injury lawsuits are obvious. The chance of being named in a lawsuit is significantly increased, regardless of fault or their role in the injury.[126, 127, 139]

Many steps can be taken to decrease the risk of catastrophic injuries and being found at fault for these injuries.[104, 123, 127, 140] A top priority is to ensure players know, understand, and appreciate the risks of making head-first contact in football.[8, 104, 140] The videos *Prevent Paralysis: Don't Hit with Your Head*[5] and *See What You Hit*[6] and the prevention section of the *Spine Injury Management*[7] are excellent education tools. Parents of high school players should also be given the opportunity to view at least one of these videos. Coaches have a responsibility to spend adequate time teaching and practicing correct contact techniques with all position players. Everyone associated with football has a moral and legal responsibility to do all in their power to attempt to eliminate head-down contact from the sport.[104, 105, 109, 140]

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Lectureship

The Howard H. Steel Lecture at the Philadelphia Orthopaedic Society

Presented by:

WALLACE B. LEHMAN, MD

Chief Emeritus and Fellowship Director
Wallace B. Lehman, M.D. Center for Pediatric Orthopaedic Surgery
Hospital for Joint Diseases/New York University, New York City

Members of the Philadelphia Orthopaedic Society as well as orthopaedic residents from the Philadelphia programs enjoyed a lecture by Dr. Wallace B. Lehman on the management of clubfeet at the Hospital for Joint Diseases in New York. The Department of Orthopaedic Surgery of Temple University proudly sponsored this event as the annual Howard H. Steel Lecture. Dr. Steel, Emeritus Chief of Staff of the Shriners Hospital for Children, is a renowned pediatric orthopaedic surgeon and an icon of the Philadelphia orthopaedic community.

Dr. Lehman recounted the long history of surgical and nonsurgical management of the clubfoot deformity and Hospital for Joint Diseases experience. He emphasized the eventual acceptance of early and deliberate nonoperative management utilizing the Ponseti method of casting and percutaneous Achilles tendon lengthening. Attention to detail is critical with serial casting. Dr. Lehman gave his unique perspective on the evolution of our current treatment of this condition and the importance of mothers' support groups. The support groups help mothers cope with the ongoing treatments for their child and improve communication and understanding for the families.

S. Rehman, MD

Dr. Lehman in his clinic.

Lectureship

The John Royal Moore Lecture at the Philadelphia Orthopaedic Society

Presented by:

ROBERT B. ANDERSON, MD

"Foot and Ankle Injuries in the Athlete"

The John Royal Moore lecture at the Philadelphia Orthopaedic Society was held on March 12, 2007 at the Bell Tower building in Philadelphia. Our guest lecturer was Robert B. Anderson, MD, who is a practicing foot and ankle surgeon from Carolinas Medical Center. Dr. Anderson systematically approached the most common foot and ankle problems in the athlete, discussing not only the epidemiology, pathology, and diagnosis, but also the current treatment options. Topics included the "turf toe," Jones fracture, Lisfranc dislocations and variants, and the stress fracture of the navicular. Dr. Anderson also addressed the changing role of the new artificial field and the inability of current cleats to "release" from the deeper linings of the turf — leading to increased injuries of the foot and ankle in the professional athlete.

The lecture was well attended, and post presentation questions were thought provoking and challenging.

Kristofer S. Matullo, MD

Drs. Thoder, Anderson, and Torg

Lectureship

The John Lachman Lecture at the Pennsylvania Orthopaedic Society

Presented by:

MICHAEL A. SMERCONISH

"Fifteen Points of Current Interest"

The Third Annual John Lachman Lecture was presented by Michael A. Smerconish, well-known radio talk show host, *Philadelphia Inquirer* columnist, and author of "Flying Blind" and "Muzzled." In addition, he is a frequent guest on several of the nationally televised news commentary shows. In view of the fact that most orthopaedic surgeons are primarily pre-occupied with such matters as the anterior cruciate ligament, total joint arthroplasty, and tort reform, it seemed appropriate to indulge in matters of public policy and in the arena, Smerconish excels.

The venue for the talk was the Friday luncheon at the fall meeting of the Pennsylvania Orthopaedic Society, before a capacity audience in the ballroom at the Top of the Bellevue. Smerconish opined on fifteen topics: 1) find and kill Bin Laden; 2) profile when screening for terrorists; 3) torture the bad guys; 4) implement all the recommendations of the 9/11 Commission; 5) articulate an exit strategy for Iraq; 6) our borders need to be closed; 7) find an accommodation for same sex couples; 8) sell Plan B over the counter to those 18 and over; 9) fund and do embryonic stem cell research; 10) term limits, two for senate, six for the house; 11) stop trying to regulate campaign finance; 12) raise retirement age from 65 to 70; 13) end the estate tax; 14) on global warming, err on the side of precaution; and 15) guns are the symptom, single parent households are the problem that needs to be addressed.

Smerconish's talk was well delivered, well received, and reflected most favorably on the John Lachman Society!

Joe Torg, MD

Kelly, Flynn, Smerconish, Torg, and Pymer

Lectureship

The Howard H. Steel Pediatric Orthopaedic Seminar and OITE Review at the Shriners Hospital for Children, Philadelphia

On October 14, 2006, the Shriners Hospitals for Children, Philadelphia presented the Howard H. Steel Pediatric Orthopaedic Seminar and OITE Review. Panelists included Dr. Richard S. Davidson, Associate Clinical Professor of Orthopedics at the Hospital of the University of Pennsylvania, Dr. Richard H. Gross, Professor of Orthopedics at the Medical University of South Carolina, and Dr. James W. Roach, Professor of Orthopedics at the University of Utah. Dr. Peter D. Pizzutillo, Professor of Orthopedics at Drexel University College of Medicine moderated this well-attended event for residents, fellows, community physicians, and academic faculty.

This seminar provided an update on the latest techniques in diagnosis and management of a number of controversial topics in pediatric orthopaedics. The panel of experts went over case studies which involved spine, hip, upper and lower extremity deformities and fractures. Audience participation made the discussion quite vibrant as different approaches and strategies were shared during this question and answer period.

The afternoon session included a comprehensive review for residents taking the Orthopaedic Intraining Examination. Important pearls and facts were shared in this high-yield overview of the major topics in pediatric orthopedics in preparation for the upcoming OITE. Key topics related to lower extremity and upper extremity disorders, pediatric trauma, and pediatric spinal deformity were reviewed. All who attended this conference found it both interactive and informative.

Simon Chao, MD

Departmental News

Faculty

Temple University Department of Orthopaedic Surgery & Sports Medicine

Chairman

Joseph Thoder, MD, *The John W. Lachman Professor*

Professors

William DeLong, MD
Ray Moyer, MD, *The Howard H. Steel Professor*
Joseph Torg, MD
F. Todd Wetzel, MD

Associate Professors

John Kelly, IV, MD, *Vice-Chairman*
Pekka Mooar, MD
Albert Weiss, MD

Assistant Professors

Easwaran Balasubramanian, MD
Kristine Fortuna, MD
Stanley Michael, MD
Saqib Rehman, MD
Bruce Vanett, MD

Emeritus Professors

Philip Alburger, MD
Edward Resnick, MD

Adjunct Faculty — Philadelphia Shriners Hospital

Randal Betz, MD, *Chief of Staff*
Linda D'Andrea, MD
James Guille, MD
Scott Kozin, MD
G. Dean MacEwen, MD
James McCarthy, MD, *Assistant Chief of Staff*
Amer Samdani, MD
Howard Steel, MD, *Emeritus Chief of Staff*

Adjunct Faculty — Abington Memorial Hospital

David Junkin, MD, *Chief of Orthopaedics*
Shyam Brahmabhatt, MD
David Craft, MD
Greg Galant, MD
Michael Gratch, MD
Moody Kwok, MD
Guy Lee, MD
Thomas Peff, MD
Jeffrey Rubin, MD
Andrew Star, MD
T. Robert Takei, MD
John Wolf, MD

Temple University Hospital
Department of Orthopaedic Surgery and Sports Medicine
Faculty 2006

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John W. Lachman Professor
Chairman
Hand & Upper Extremity
General Orthopaedics

Philip Alburger, MD
Pediatric Orthopaedics

Eswarian Balasubramanian, MD
Joint Reconstruction
General Orthopaedics

William DeLong, MD
Orthopaedic Trauma
Sports Medicine
General Orthopaedics

Kristine Fortuna, MD
Pediatric Orthopaedics

John Kelly, IV, MD
Vice Chairman
Sports Medicine
General Orthopaedics

Stanley Michael, MD
Sports Medicine
Joint Reconstruction
General Orthopaedics

Pekka Mooar, MD
Sports Medicine
Joint Reconstruction
General Orthopaedics

Ray Moyer, MD
Sports Medicine

Saqib Rehman, MD
Orthopaedic Trauma
General Orthopaedics

Edward Resnick, MD
General Orthopaedics
Pain Management

Joseph Torg, MD
Sports Medicine

Bruce Vanett, MD
Joint Reconstruction
General Orthopaedics

F. Todd Wetzel, MD
Spine Surgery

Albert Weiss, MD
Hand & Upper Extremity
General Orthopaedics

Update from the Office of Clinical Trials and Research Support

The Office of Clinical Trials and Research Support was founded in 2004 under the direction of Pekka A. Mooar, MD. Supported by the School of Medicine Office of Clinical Trials, Joanne Donnelly was hired as a full time study coordinator.

The establishment of this program represents a commitment to enhance resident and faculty research by providing logistical support for grant writing, budget development, statistical support, IRB document submissions, and manuscript and exhibit preparation.

A statistical support program was developed through collaboration with Jeffrey Lidicker and Dr. Alan J. Izenman at the Center for Statistical and Information Science. This program allows residents and students to develop statistically valid prospective clinical trials that will have the greatest chance of being accepted for presentation and publication.

The program works with Arleen Wallen and Craig Pfister in the Office of Clinical Trials to develop budgets, informed consents and IRB submissions. Mr. Pfister also serves as an industry liaison to bring clinical trials to the attention of interested faculty members.

Ms. Donnelly and Dr. Mooar direct summer medical student research. Students receive training in research ethics and research design. The summer experience lasts 8 weeks. Last summer there were three students and this year there will be nine students supported by work study funds from the Orthopaedic Department and the Office of the Dean of the School of Medicine. Topics are developed throughout the year in collaboration with our faculty.

2006 student summer project program:

Andrew Walker: Pilot study for osteoporosis screening in orthopaedic patients; is there a social economic divide? (Dr. Mooar)

Micah Cohen: Retrospective study of the effect of BMI on knee osteotomies performed for osteoarthritis. (Dr. Mooar)

Michael Herbst: Development of an online data base to track surgical outcomes for injuries to the knee and shoulder. (Dr. Kelly)

2007 student summer proposed projects:

Dr. Torg: Prevention strategies for catastrophic injuries resulting from athletic activity.

Dr. Rehman: Incidence of neurologic injury in gunshot extremity fractures.

Dr. Rehman: Development of an orthopaedic trauma outcomes database.

Dr. Balasubramanian: One year follow up of patients with mobile-bearing total knee:functional assessment.

Dr. Balasubramanian: Efficacy of adjunctive intra-operative heparin use during total hip and total knee arthroplasty in the prevention of thromboembolic disease.

Dr. Kelly: Differences in stability using “Moyer Technique” for graft placement in allograft vs autograft in ACL reconstruction of the knee.

Dr. Mooar: Open MRI imaging of Patella femoral tracking under load.

Dr. Mooar, Dr. Moyer: DVT surveillance in knee ligament reconstruction.

2007 student summer proposed projects *(continued)*

Dr. Mooar: Pain management and complications in the elderly undergoing orthopaedic surgery.

Dr. Thoder: Pearls and Pitfalls of two-incision reconstruction of distal biceps avulsions.

2006 Federal Grant Submissions Via our Office

NIH RO1
"Role of T cells in the Initiation of Osteoarthritis"
PI: Christopher Palatsoucas (Microbiology & Immunology). Collaborating departments: Orthopaedics (Mooar), Medicine (Myers)

NIH R03
"Enhancement of Fracture Healing in a Rat Head Injury Model"
PI: Rehman. Collaborating Departments: Orthopaedics (DeLong), Anatomy & Cell Biology (Arango-Hisijara, Popoff, Safadi)

U.S. Dept. of Defense
"Enhancement of Fracture Healing in a Rat Head Injury Model"
PI: Rehman. Collaborating Departments: Orthopaedics (DeLong), Anatomy & Cell Biology (Arango-Hisijara, Popoff, Safadi)

U.S. Dept. of Defense
"Analysis of high vs. low pressure irrigation techniques in open fracture management"
PI: Rehman. Collaborating Departments: Orthopaedics (DeLong), Pathology (Khurana, Truant), Statistics (Lidicker).

U.S. Dept. of Defense
"Treatment guidelines in the management of open fractures"
PI: Rehman.

Medical Student Orthopaedic Interest Group

Ms. Donnelly coordinates resident faculty participation in the Temple Medical Student Orthopaedic Interest Group. Last year there were 32 students attending the lectures and this year there are 66.

Dr. Torg: Riding the Crest with Sports Medicine

Dr. Matullo: Things you need to know to get into a residency in Orthopaedic Surgery

Dr. Rehman: Life as an orthopaedic surgeon (Babcock Surgical Society/Ortho Interest Group panel discussion)

Dr. Junkin: How to make your preclinical years count

Exhibit Preparation by our Office

2007 AAOS EXHIBIT
Joseph Thoder, Matthew Reish, Kristofer Matullo, Pekka Mooar, Leonard DAddesi
"Surgical Decision Making for Unstable Fractures of the Distal Radius: The Temple University Algorithm"

Current Industry-Sponsored Clinical Trials

"Pain Management via PCEA for Patients Undergoing Total Knee Arthroplasty." Pfizer Pharmaceuticals 2006 PI: Dr. Mooar, Orthopaedics, Dr. David Kim, Anesthesia

"Acute Hip Pain Management Using Oral Analgesic Medication for Patients Undergoing Total Hip Replacement Surgery." Johnson & Johnson Pharmaceuticals 2007 PI: Dr. Mooar

"Fractures of the Distal Radius: Comparison of Operative Fixation with the Micronail and Volar Plating." Wright Medical Inc. 2006 PI: Dr. Rehman

"Reducing Risk for the Development of Ibuprofen-Associated Upper-Gastrointestinal Ulcers in Orthopaedic Patients Requiring NSAID Therapy." Horizon Therapeutics 2007 PI: Dr. Mooar, Orthopaedics, Dr. Benjamin Krevsky, Gastroenterology

"DVT Prophylaxis with an Oral Medication in Patients Undergoing Elective Total Hip Replacement Surgery." Bristol-Myers Squibb Pharmaceuticals 2007 PI: Dr. Mooar, Orthopaedics, Dr. John Blebea, Vascular Surgery

Current Investigator-Initiated Prospective Clinical Studies

Arthroscopic "Mumford" of distal pole of patella for refractory patellar tendonitis: a preliminary report. PI: John D. Kelly, IV, MD, Temple IRB #10876

Analysis of osteoactivin in human fracture hematoma. PI: Saqib Rehman, MD, in collaboration with Drs. Steven Popoff and Fayez Safadi, Dept. of Anatomy & Cell Biology Temple IRB#10757

Current Resident Research Projects Coordinated by the Office

An Electromyographic Assessment of Three Special Tests for Subscapularis Tears
David Yucha, MD, PGY-V Temple IRB#4695

Minimally Invasive Posterior Lumbar Fusion-Clinical and Radiographic Results and Complications in 75 Consecutive Patients
Simon Chao, MD, PGY-III Temple IRB#10654

Low-Velocity Intra-Articular Gun Shot Wounds of the Knee — Clinical Review of Initial Treatment
David Junkin, MD, PGY-V Temple IRB#10712

14 Year Results of a High Density Polyethylene Prosthetic Anterior Cruciate Ligament Reconstruction
Robert Purchase, MD, PGY-V Temple IRB#10830

Analysis of Osteoactivin in Human Fracture Hematoma
Carlos Moreyra, MD, PGY-III Temple IRB #10757

Proximal Row Carpectomy: Clinical Evaluation of a Novel Surgical Technique
Kristofer Matullo, MD, PGY-IV

First Carpometacarpal Arthroscopy: A Review of Treatment Options and Introduction of a Limited Incision Technique
Kristofer Matullo, MD, PGY-IV

Low Back Pain and Unrecognized Cobb Syndrome in a Child Resulting in Paraplegia
Kristofer Matullo, MD, PGY-IV

An Electromyographic Assessment of the "Bear Hug" — A New Exam for the Evaluation of the Subscapularis Muscle
Simon Chao, MD, PGYIII

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Department of Orthopaedic Surgery and Sports Medicine
House Staff 2006–2007

Leonard D'Addesi, MD
PGY-5

David Junkin, MD
PGY-5

Robert Purchase, MD
PGY-5

David Yucha, MD
PGY-5

Wade Andrews, MD
PGY-4

Kristofer Matullo, MD
PGY-4

Joseph Morreale, MD
PGY-4

William Pfaff, MD
PGY-4

Simon Chao, MD
PGY-3

Neil MacIntyre, MD
PGY-3

Carlos Morerya, MD
PGY-3

Alyssa Schaffer, MD
PGY-3

Abtin Foroohar, MD
PGY-2

John Parron, MD
PGY-2

Allen Tham, MD
PGY-2

Ian Duncan, MD
PGY-1

Brian George, MD
PGY-1

Gbolabo Sokunbi, MD
PGY-1

Christopher Kestner, MD
PGY-1

Department of Orthopaedics Graduating Residents

Leonard L. D'Addesi, MD

Leonard grew up in south Philadelphia. He attended Villanova University where he earned a Bachelors Degree in Mechanical Engineering, followed by a Masters Degree in Biomedical Engineering from Drexel University. Lenny completed his medical school training at Temple University. He is married with three children. He is going on to complete a Hand/Upper Extremity Fellowship at the Philadelphia Hand Center at Thomas Jefferson University Hospital.

David M. Junkin, MD

David grew up outside Philadelphia in Huntingdon Valley. He attended Bucknell University and attained a Bachelors Degree in Biology followed by a Masters Degree in Medical Sciences from Allegheny University. He attended Temple University Medical School. Dave is married with two children. He is completing a sports fellowship at University of Kentucky with Darren Johnson.

Robert J. Purchase, MD

Robert grew up in Erie, Pennsylvania and attended Washington and Jefferson College where he attained a Bachelors Degree in Biology. He continued his training at Temple University Medical School. Rob is married with two children. He will be completing a sports fellowship at California Pacific Medical Center.

David T. Yucha, MD

David grew up in Central Pennsylvania. He attended the University of Pennsylvania where he attained a Bachelors Degree in History, followed by a Masters Degree in Biology from Saint Joseph's University. He attended Temple University Medical School and graduated AOA. David is married with two children. He is completing a sports medicine fellowship at the Rothman Institute, Thomas Jefferson Hospital, in Philadelphia.

Alumni Day: Purchase, Junkin, Yucha, D'Addesi

The Junkins

Christmas Party: D'Addesi, Purchase and wives

Christmas Party: Yucha, Junkin and family

Journal Club: Purchase, Junkin, D'Addesi

Junkin, Purchase, Yucha, D'Addesi

Alumni Day 2006

A day on the greens

The Chiefs

Reunion dinner

Catching up

Meeting old friends

Food and laughs

Christmas Party 2006

We look good

PGY-3s and the Chief

Thoder, Kelly, Vanett

We can dance

Who stopped the music?

The girls

Instructions to Authors

Editorial Philosophy

The purpose of the *Temple University Journal of Orthopaedic Surgery and Sports Medicine (TUJOSM)* is to publish clinical and basic science research performed by all departments of Temple University that relate to orthopaedic surgery and sports medicine. As such, TUJOSM will consider for publication any original clinical or basic science research, review article, case report, and technical or clinical tips. All clinical studies, including retrospective reviews, require IRB approval.

Editorial Review Process

All submissions will be sent to select members of our peer review board for formal review.

Manuscript Requirements

Manuscripts are not to exceed 15 double spaced type-written pages and/or 5000 words (minus figures/tables/pictures). The Manuscript should contain the following elements: Title page, Abstract, Body, References, and Tables/Legends. Pages should be numbered consecutively starting from the title page.

(1) Title Page — The first page should contain the article's title, authors & degrees, institutional affiliations, conflict of interest statement, and contact information of the corresponding author (name, address, fax, email address).

(2) Abstract — The second page should be a one-paragraph abstract less than 200 words concisely stating the objective, methods, results, and conclusion of the article.

(3) Body — Should be divided into, if applicable, Introduction, Materials & Methods, Results, Discussion, and Acknowledgements. Tables and figures (in JPEG format) with their headings/captions should be listed consecutively on separate pages at the end of the body, not continuous within the text.

(4) References — Should be listed following the format utilized by JBJS. For example: Smith, JH, Doe, JD. Fixation of unstable intertrochanteric femur fractures. *J Bone Joint Surg Am.* 2002;84:3553–58.

(5) Each page should have continuous line numbers placed, as well as the first author's name, date submitted and page number in the footer.

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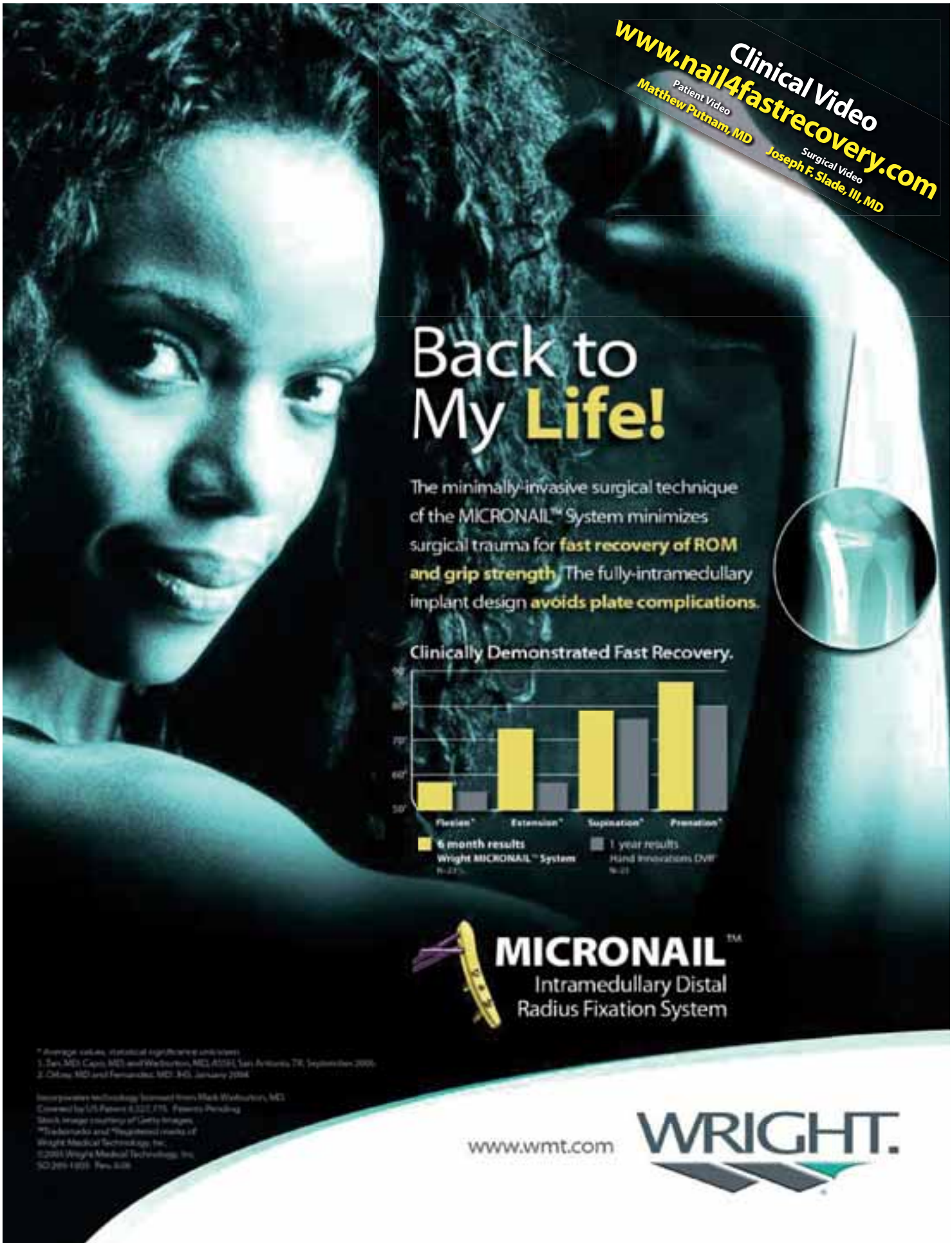
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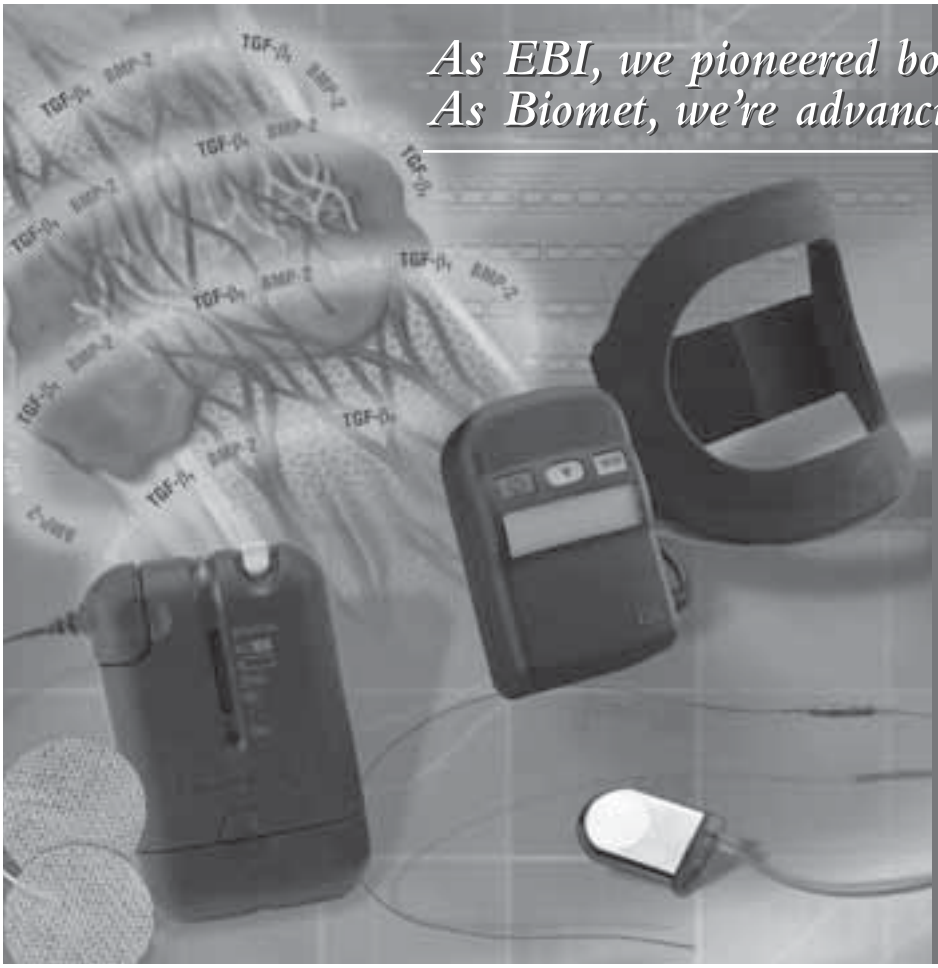

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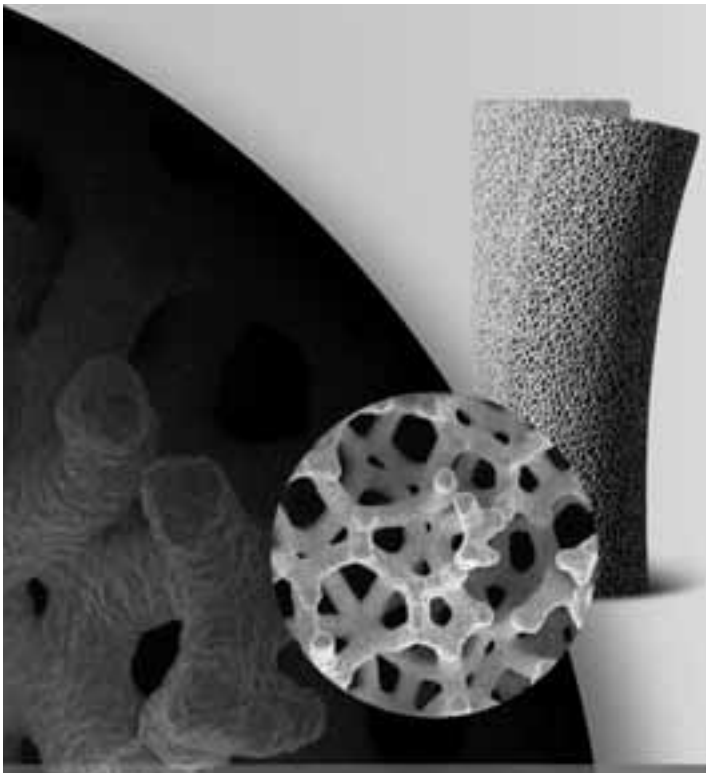

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For exceptional ingrowth, *Trabecular Metal* is the best thing next to bone. Its bone-like physical and mechanical properties contribute to extensive bone infiltration.[1,2]

- **High Porosity** – 80% porosity allows 2-3 times greater bone ingrowth compared to conventional porous coatings and double the interface shear strength.[1]
- **Compressive Strength Similar to Bone** – supports physiologic loading without a solid metal substrate.[2]
- **Stiffness Similar to Bone** – low stiffness facilitates load transfer and helps minimize stress shielding.[2]
- **High Friction** – bone interfacing struts generate a friction coefficient that is 76% greater than a sintered bead coating, providing increased initial stability.[3]

Trabecular Metal™ – The Best Thing Next To Bone

Zimmer Trabecular Metal Technology
10 YEARS
A Decade of Clinical Success!

Ask your Zimmer associate about the Monoblock Acetabular Cup System, Augmentation Patella with *Trabecular Metal*, or the *Trabecular Metal* Monoblock Tibial Component, or visit us at www.zimmer.com.

[1] Bobyn JD, Stackpool GJ, Hacking SA, et al. Characteristics of bone ingrowth and interface mechanics of a new porous tantalum biomaterial. J Bone Joint Surg Br. 1999; 81-B; 5: 907-914.

[2] Bobyn JD, Hacking SA, Chan SP, et al. Characterization of a new porous tantalum biomaterial for reconstructive orthopaedics. Scientific Exhibit, Proc of AAOS; 1999; Anaheim, CA.

[3] Fitzpatrick D, Ahn P, Brown T, et al. Friction coefficients of porous tantalum and cancellous and cortical bone. Presented at the 21st Ann Amer Soc Biomechanics; 1997; Clemson, SC.

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