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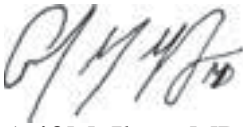
Letter from the Editors

We are proud to present the first edition of the *Temple University Journal of Orthopaedic Surgery and Sports Medicine.* This journal was inspired by our chairman, Joseph J. Thoder, MD, and it strives to uphold the "Tradition of Excellence" begun by Drs. John Royal Moore, John Lachman, and Michael Clancy. Our charge was to construct a high quality journal that would encompass a broad spectrum of musculoskeletal pathology, engineering, and basic science. The content is representative of the spirit, insight, and diligence of Temple University. In such, it contains the peer-reviewed works of the faculty and residents of the Temple Orthopaedic community, both past and present. Along with these scientific endeavors, we offer some insight into our world by providing a snapshot of the lives of our residents, alumni, and staff.

First, we would like to say thank you for being chosen to be the first editors of the *Journal.* It was truly an honor and a privilege. A work of this size is not possible without the help of many individuals. We would like to thank Synthes for their generous grant to begin the process, the faculty for their guidance and support, the editorial board for their insight, and the authors for allowing us to share their work. Two people deserve particular attention: first, our Chairman, Dr. Joseph Thoder, whose embrace of this initiative propelled it forward; and second, Dr. Joseph Torg, whose personal commitment and direction led to the journal's completion. We hope that the Journal will grow in both quality and breadth with each coming year.

In closing, we invite you to peruse the inaugural issue of the *Temple University Journal of Orthopaedic Surgery and Sports Medicine,* and thank you for allowing us to share with you some of our academic endeavors.

Sincerely,



Asif M. Ilyas, MD

Kristofer S. Matullo, MD

Letter from the Chairman

Over the past five years, the Department has grown in its "vision" of what Temple Orthopaedics should provide to its clinical and academic communities. Our faculty has grown in number and includes the entire spectrum of orthopaedic surgeons from the young and energetic physicians to the wise and grizzled veterans. In all we have added seven new faculty members over the past three years. Our staff now numbers sixteen, and will continue to grow over the next few years as our programs expand. These programs include re-emphasis on our already strong Sports Medicine services, being the team physician to the Philadelphia Flyers and the Philadelphia Phantoms; as well as the Temple University Department of Athletics, Philadelphia University and numerous Philadelphia and Suburban High Schools. In addition, we are developing a regional referral center for orthopaedic trauma, and creating centers of excellence in both Spine Surgery and the Upper Extremity.

We are fortunate to have the full support of the Temple University Health Center and the School of Medicine in realizing the new "vision" for Temple Orthopaedics.

The Temple University Health System has expanded its services to include not only Temple University Hospital but also Northeastern and Jeanes Hospitals where the Department of Orthopaedics has a prominent role in providing clinical services and developing clinical programs both locally and regionally.

Temple University School of Medicine is in the final stages of planning for a brand new state of the art medical school on North Broad Street. This new facility along with new collaborative projects with the Department of Anatomy will further enhance our research opportunities. In order to better coordinate this research effort, the Department has recently hired a full time research coordinator to help orchestrate grant proposals, clinical trials and IRB protocols. The Department is committed to increasing our research effort to reflect the quality of our academic activity. The newly formed *John Lachman Research Fund* has raised over $100,000 in the past year designated for academic programs and resident research projects, including the publication of this *Journal.*

The Department of Orthopaedics Surgery and Sports Medicine here at Temple University takes great pride in its tradition of excellence in clinical service, education and research. This tradition began in the1930's with our first chairman, John Royal Moore, MD and has continued through his successors, John W. Lachman, MD and Michael Clancy, MD. I am privileged to have the charge of carrying on this tradition through the *Bone and Joint Decade,* and beyond. This *Journal* is a tangible representation of the new "vision" of Temple Orthopaedics and Sports Medicine, and would not have been possible without the efforts of several key members of the Department. In particular, I would like to recognize Drs. Asif Ilyas and Kris Matullo for their tireless efforts to be sure manuscripts were compiled, deadlines were met, and papers were edited. And, Dr. Joseph Torg for overseeing the publishing process.

The Department of Orthopaedic Surgery and Sports Medicine at Temple University School of Medicine is proud to present you with the inaugural issue of our *Journal.*

Joseph J. Thoder, MD
Chairman, Department of Orthopaedic Surgery & Sports Medicine
Temple University School of Medicine

Foreword from the Dean

Inauguration of a new journal is a proud moment for it represents the culmination of concept and effort from many sources. In this case, the idea emanated from Drs. Thoder and Torg, the work was conducted by Drs. Ilyas and Matullo and the original research, commentaries, etc., were produced from many authors.

Temple has a brilliant history as a school, but none shines more than that of the Department of Orthopaedic Surgery. As a Temple medical student from 1969 to 1973, I had the pleasure to undertake a clerkship with Dr. Torg as he initiated the field of Sports Medicine. He had the self-assurance and clinical rapport that only a true master surgeon can bring to the patient. I was also drawn to the Department chairman, Dr. John Lachman, who was a gentleman to the students, both a taskmaster and teacher for the residents, and a kind and careful physician with his patients. These individuals are but a sampling of the wonderful legacy for a department which exhibits the same greatness today. Temple is "on the move," recruiting outstanding faculty (>250 since 2002), building new facilities (a new medical school groundbreaking in Fall 2006) and creating new knowledge to improve patient care. This inauguration is truly a proud moment for an outstanding department of faculty, residents and staff. Temple salutes your accomplishments.

John M. Daly, MD
Dean, Temple University School of Medicine
Professor, Department of Surgery
Temple University

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 purpose of this communication is to answer two questions: 1) who is John Lachman? and 2) what is the structure of the Society and how will it accomplish its mission?

"Who is John Lachman?" A graduate of both Temple University and its School of Medicine, he also completed his orthopedic residency at that institution under John Royal Moore, MD, the first professor and chairman of the Department of Orthopaedic Surgery. Remaining on the faculty at Temple following his residency, Dr. Lachman succeeded Dr. Moore in 1965 as professor and chairman. An apolitical person, thoroughly at ease with himself and his role in life as he sees it, his relative obscurity had been self-imposed. He has completely dedicated his life to serving two purposes: God and orthopedics. A devout Catholic, he attends mass daily and is a strong supporter of his church. In the conduct of his professional life, he has been totally devoted to excellence in orthopedics and to the education of medical students and residents. John Lachman is an extraordinary individual, both from the standpoint of his superior intellect and his pervasive personal qualities. By his residents, colleagues and friends he is loved, respected, admired and affectionately referred to as "Latch".

A proponent of the concept of meticulous attention to detail, early in his career he noticed that certain patients with torn anterior cruciate ligaments demonstrated passive anterior subluxation of the proximal tibia in relationship to the femur apparent while lying supine. Exploring this observation further, he demonstrated that anterior cruciate ligament insufficiency was determined easily by stressing the knee in extension rather than in the manner of the classic drawer test. My first exposure to this observation occurred as a resident when presenting a patient to the chief on rounds. Upon turning back the covers, he simply looked at the patient's knee and stated, "He has a torn anterior cruciate ligament." The patient's anterior drawer test was unremarkable, however, surgical findings were that of a complete tear of the anterior cruciate ligament. Thus, a clinical "pearl" had been simply passed on as he had done hundreds if not thousands of times before to his students and residents.

To put things in historical perspective, in 1970, the sports medicine mavens and self-appointed knee experts, with the exception of John Fegan and the late John Marshall, virtually denied the existence of the anterior cruciate ligament. Other than acknowledging its presence in the knee joint and its role as a part of O'Donoghue's "unholy triad", the fact that it tore either as an isolated structure or was associated with injury to the menisci was both unrecognized and denied. A review of the literature of this period reveals unequivocally that those who were writing on knee injuries and knee surgery not only denied, but demeaned the importance of the structure.

Using this simple maneuver of stressing for cruciate laxity with the knee slightly flexed, it soon became apparent that not only did the anterior cruciate ligament tear both in an isolated manner or in conjunction with a meniscal injury, but also that this phenomenon occurred frequently. A paper entitled "Clinical Diagnosis of Anterior Cruciate Ligament Instability in the Athlete" was presented before the annual meeting of the American Orthopaedic Society for Sports Medicine in 1976 in New Orleans. In addition to describing the Lachman test as a procedure preferable and much more reliable than the classic anterior

drawer test, it also reported the frequency of injury to the cruciate ligament associated with injuries to other structures. Specifically, reporting on anatomic lesions observed in surgery in 250 consecutive knees in a population of recreational and collegiate athletes, 172 (69%) had tears of the anterior cruciate ligament, 200 (80%) had tears of the medial meniscus, and 62 (25%) had tears of the lateral meniscus. Again, a review of the literature will clearly indicate that this study, subsequently published in the *American Journal of Sports Medicine* in April 1976, was the first to identify the frequent association of anterior cruciate ligament and meniscal lesions.

Again, considering the orthopedic/sports medicine mind-set of this period, it is interesting to note that the critique of the presentation indicated that it was the consensus of the audience that the paper "had no clinical relevance." However, the Lachman test has not only withstood the test of time, but is generally recognized as the most sensitive clinical test for the determination of anterior cruciate ligament integrity. It is clear that both the sign and the term "The Lachman Test" are firmly engraved in contemporary orthopedic vocabulary. As long as young men and women continue to tear their anterior cruciate ligaments, the eponym will prevail and John Lachman will be memorialized for his contributions as a mentor, teacher and clinician.

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 the 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 the Executive Committee, working committees of the Society, or the Chairman and will require the approval of either 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.

CHARTER MEMBERS OF THE JOHN LACHMAN SOCIETY

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John Casey, MD
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John Van Orden, MD
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F. Todd Wetzel, MD
Gerald Williams, MD
John Wolf, MD

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.

RESIDENTS DISSERTATIONS: The Temple University Residents Research Presentations sponsored by The John Lachman Society were held on May 14,2005. Victor Hsu was the winner of the $500 first place award and plaque for his presentation of "Elastic Instability and Orthopedic Injury". Ammar Anbari won $300 and took the second place award for his presentation on "C5-C6 Foraminal Stenosis and the Burner Phenomena". Neil MacIntyre took third place and $100 for his presentation on "Prophylactic Knee Bracing in College Lineman." For reasons to be explained, it was the opinion of all who attended, including Dr. Lachman that this was a sentinel event for the residency program.

THE *TEMPLE UNIVERSITY JOURNAL OF ORTHOPAEDICS AND SPORTS MEDICINE:* In view of the quality of the residents research presentations and their interest in becoming involved in clinical research Joe Thoder expressed his desire that these papers, as well as the academic activities of the department be committed to a print and/or digital format. In response, The John Lachman Society, utilizing the resources of The John Lachman Orthopedic Research Fund, will sponsor this endeavor. Asif Ilyas and Kris Matullo will serve as Editors-in-Chief, Joe Torg will serve as the Managing Editor, Albie Weiss will be the Business Manager and Pekka Mooar and Saqib Rehman will chair the editorial review committee.

SYNTHES AWARD: Synthes has awarded The John Lachman Orthopedic Research Fund $25,000 to support the research and academic activities of the Department of Orthopaedic Surgery.

RESIDENT RESEARCH SUPPORT: The following resident research projects 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 Orthopaedic Injury".
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'Addesi, 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".

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

ETHICS COMMITTEE: The JLS Ethics Committee was charged to develop a position statement for publication dealing with the Pennsylvania statute requiring physicians to report possibly impaired drivers to the Pennsylvania Department of Transportation. The Ethics Committee reports that the position paper on "Legislating Breach of Confidentiality in Medical Settings" has been accepted by the American Journal of Orthopedics for publication.

TEMPLE SHRINERS ALUMNI RELATIONS: To be emphasized, it is the intention of The John Lachman Society to function in a cooperative and complimentary manner with the Temple Shriners Alumni. It is expected that former Temple Shriners residents who are members of The John Lachman Society will maintain their membership and support of the Alumni.

RESIDENTS MENTORING PROGRAM: We are happy to report that the Society's resident mentoring program has played a significant role in five of our residents obtaining excellent fellowships. Manish Patel is currently with Buddy Savoie in Jackson, Mississippi. Ammar Anbari is at Rush Presbyterian Hospital in Chicago with Bernie Bach. Next year Matt Reish will go to Long Beach, California with Doug Jackson, Victor Hsu to the San Diego Spine Center with Behrooz Akbarnia, and Asif Ilyas to the Massachusetts General Hospital to do a Hand Surgery fellowship with Jessie Jupiter and David Ring. All five are great fellowships.

JLS ANNUAL MEETING: The first annual meeting of The John Lachman Society was held Saturday, November 19, 2005 at the Philadelphia Country Club. John Kelly, President, gave an overview of the successful academic and research activities supported by The John Lachman Orthopedic Research Fund. He emphasized that these programs plus the current and potential funding of the residents and fellows clinical research is having a positive impact on their attitudes and enthusiasm. Joe Thoder, Department Chairman, pointed out that the current economic environment in the medical school and the health sciences system preclude these entities from providing the funds necessary to conduct an academic residency program. He emphasized the important role that The John Lachman Research Fund will play in the education of residents and emphasized that the program has his full support.

The slate of Officers and Directors of The John Lachman Orthopedic Research Fund was proposed and unanimously approved. They included:

John Kelly, Director and President
Philip Alburger, Director and Vice President
Albert Weiss, Director and Treasurer
Joe Torg, Director and Secretary
Ray Moyer, Director
Joseph Thoder, Director
David Junkin, Director
Kevin Kolmer, Director

It was suggested that consideration be given to a separate campaign to raise funds for renovating and equipping the current orthopedic conference room with a state-of-the-art audiovisual system. Joe Thoder agreed that this would be a most welcome project. The possibility of having the Temple Shriners Alumni Association join in this endeavor was suggested.

Latch in his Teaching Mode

The John Lachman Society supporting the John Lachman Orthopaedic Research Fund

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.

Joe Torg, MD
Secretary, The John Lachman Society

Commentary

Legislating Breach of Confidentiality in Medical Settings

ALBERT A. WEISS, MD & JOSEPH S. TORG, MD

In the Spring of 2004, a resident of Pennsylvania had his driver's license revoked, based on his physician's report to the State Department of Motor Vehicles of the physician's perception of impairment of his patient's ability to safely operate a motor vehicle, submitted in accordance with Pennsylvania law. That perception, in turn, was based on the voluntary provision of information by the patient regarding his own alcohol consumption — part of the physician's appropriate inquiry into his patient's social and drug history. That the physician was diligent in seeking the information, and that the patient was responding honestly in his own best interests, is not in question. What is in question is the physician's conduct in reporting information the patient had reason to believe was being revealed in confidence, and the deduction by the physician that his patient was unable to safely operate a motor vehicle, admitting to significant alcohol consumption. It is beyond the scope or purpose of this paper to consider either the patient's blood-alcohol levels based on the quantity of alcohol described, or the relevance of the temporal relationship between the consumption and the vehicle operation.

Subsequently, Philadelphia newspapers were inundated with editorial comments, and published many over the ensuing two weeks, with varying perspectives of appall and support, both for the physician and the patient.

The Pennsylvania statute[1] requires "All physicians . . . report . . . every person over 15 years of age diagnosed as having any specified disorder," defined as "mental or physical disability affecting the ability of a person to drive safely." The statute grants the physician "immunity from civil and criminal liability . . . brought by any person . . . for providing the information. . . ." The Pennsylvania courts have repeatedly ruled that parties injured by an impaired driver who was not reported by a physician cannot automatically collect damages from the physician based solely on the physician's failure to comply, although no law exists which specifically protects physicians from action on this basis. Furthermore, Commonwealth Court has ruled that the PA Department of Transportation may not be sued for incorrectly failing to revoke a driver's license under these provisions.[2]

In 1993, Pennsylvania physicians reported approximately 21,000 possibly impaired drivers, of whom nearly 6,000 had their licenses revoked.[3] It is not clear who, in the Pennsylvania Department of Transportation, had the wisdom to determine which of the 21,000 deserved revocation. Of those 6,000 who had their licenses revoked, roughly 125 were attributed to "loss/impairment of limb,"[3] a figure particularly relevant to Orthopedic Surgeons. Considering the incidence of fracture and other orthopedic disorders that incapacitate a driver, it is clear that this represents a largely under-reported population, and failure of compliance with the law. Of note, the law does not distinguish between permanent and temporary impairments.

In question is the ethical dilemma of a physician's responsibility in two regards: the safety of the general public; and the confidentiality of information concerning an individual. Not to be considered, although relevant, are the potential legal conflicts between the Pennsylvania statute and Federal HIPPA regulations, and the potential for exposure to liability by the physician through actions by the "exposed" patient, or an injured third party.

Physicians in the 20th century have accepted a compromise in the seal of confidentiality by communicating diagnostic and treatment information, which could be confidential, to third party payers. Further compromise is required by worker compensation insurers who demand even more comprehensive reporting in exchange for financial responsibility. It is assumed, although not necessarily practiced, that there is an implied consent by the patient to divulge the required information in exchange for the provision of insurance coverage for their care.

Government regulations have required reporting of certain communicable diseases without patient consent. As ethical questions arose concerning the need to advise sexual partners of the presence of sexually transmitted diseases (STD), the privacy rights of patients with potentially lethal and untreatable STDs, like HIV, took on their own subset of government regulations and reporting requirements, in general, sensitive to the needs of the individuals' right to confidentiality as a priority over the welfare of the general public.

Society has come to accept these transgressions on the individual's right to expect confidentiality. Therefore, is the requirement to report medical conditions that impair the ability of someone to drive a motor vehicle simply an extension of these exceptions? Is the ascension of another reason to violate the confidentiality an indication that government is on the potentially slippery slope, which, unchecked, can undermine the entire confidentiality paradigm?

The American Medical Association has adopted nine principles of medical ethics[4] which reflect rather than clarify the dilemma. Principle IV states, "A physician shall respect the rights of patients . . . and shall safeguard patient confidences. . . ."[4] Principle VII states, "A physician shall recognize a responsibility to participate in activities contributing

to the improvement of the community and the betterment of public health."[4] Finally, Principle III offers a concept, which may help resolve the conflict, stating, "A physician shall respect the law and also **recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.**"[4]

Medicine, law, clergy, and the press have relied upon a privileged confidentiality in relations with those they serve. This privilege is offered to allow the individual the opportunity to benefit from the advise of their confidant without fear of exposure. Although acknowledged by each profession as having individual circumstances in which society as a whole would benefit from a breach of that confidence, the larger good is felt to be served by the assurance of confidence in the absolute. This begs the question: **Is the public safety served by encouraging an impaired patient to hide from his physician that impairment in order to avoid repercussion?**

It is curious that no legislation exists compelling an attorney to reveal to the government an issue about his client, which represents a real, perceived, or potential threat to society. The legal code of ethics[5] requires that an attorney "shall" maintain a confidence, unless he perceives a real threat to "life or significant property" without defining significant, in which case he "may" break the confidence.

Priests have repeatedly used the shield of the confidence of the confessional in withholding information on inquiries from police or the courts. Often described as "inviolable," the Code of Cannon Law[6] clearly limits this level of inviolability to confessions from "the faithful," suggesting that a priest counseling a non-Catholic may not be held to the same standard of confidence. Yet, Pennsylvania has legislated an exception to the confidentiality with physicians, which centuries of medical practice indicated a compelling reason to honor.

Often, ethical dilemmas arise in which the choice compromises an otherwise protected principle. It behooves the decision-maker to judge on the basis of the greatest good — a term sufficiently vague to allow bad decisions. However, many such decisions can be resolved by a third option, not necessarily self-evident, which forces no compromise in either principle. Great efforts should be expended to seek such a solution. In the case of the Pennsylvania legislation, none was sought or found.

Confidentiality has been identified elsewhere as a strong contributor to positive outcomes for society. For example, when the search for an effective means of developing systems to reduce risks of aviation crashes was hampered by the "threat of embarrassment and professional sanction for possible violation of a rule,"[7] the Aviation Reporting System held at its core that "by law, by contract, and by practice, its confidentiality is absolute. . . . The continued improvement in outcomes speaks for itself."[7]

Within this context, harm is done to the patient who loses the ability to reveal to his physician a piece of information, which may be highly significant to his overall well being, because of fear of repercussion. As well, harm is done to the physician who is hampered in his ability to effectively diagnose, treat, or counsel his patient, based on inadequate or false information. In that the burden to society, socially and financially, from alcoholism is recognized as substantial, and that physicians are pivotal in addressing the disease from the perspective of treatment and counseling, it follows that society is harmed by interfering with an effective means of addressing this problem. The good done by removing dangerous drivers from the road through this means may be overshadowed by the societal harm done by this law, without regards to the issue of ethics.

The conflict itself suggests the need for another solution which both satisfies societal needs, while preserving the useful principle of confidentiality. **The burden of reporting motor vehicle operation impairment to the authorities could be transferred to the patient or the parent, in the case of a minor, under the advice of the physician.** The physician could be required to document in his records that he discussed the issue with the patient, and could further provide the patient with the tools, such as phone number, address, or e-mail, for self-reporting. This satisfies three goals: it maintains confidentiality; it relieves the state of the unacceptable situation in which it allows impaired drivers known to their physicians to have no check until caught; finally, it places a responsibility for behavior on the owner of the behavior, where it belongs, and encourages the driver with behavioral change without a punitive component.

The study of ethics does not give answers. It provides the tools for developing answers to issues of moral consequence. This paper addressed a public policy that created an unnecessary breach of confidentiality to satisfy a public safety concern. The resolution proposed addresses both of the ethical principles of public safety and confidentiality, and fosters a positive mechanism for change, rather than a punitive one.

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Commentary

Vioxx and Merck: What Did They Know and When Did They Know It?

JOSEPH S. TORG, MD

On February 18, 2005 a panel of experts advised the Food and Drug Administration (FDA) against banning Vioxx and other Cox-2-inhibitors. Clearly a reasonable recommendation, however, from a cause and effect standpoint, it is my view that Merck, the panel of experts, and the FDA just don't get it.

It has been with a profound sense of dismay that I have watched the development of the current Vioxx fiasco. On the basis of a personal anecdotal experience, it is clear that Merck has grossly mishandled what was, in my view, an entirely preventable situation. To understand its genesis, one must necessarily answer the now all too familiar question of "what did they know and when did they know it?" and perhaps most important, what did they do and what did they not do?

Let me explain. In the course of thirty-five years practicing clinical orthopedic surgery in several academic institutions in Philadelphia, I have had an opportunity to examine, treat, and follow many thousands of patients suffering from what can be euphemistically described as the ravages of the beat up, broken down, middle aged deterioration syndrome. Low back pain at 35, rotator cuff problems at 45, a painful, stiff neck at 55, and hip and knee discomforts thereafter. Earlier treatment options were limited to heat, cold, rest and aspirin. Aspirin, available for pennies a pill, fell into disfavor because of its use occasionally resulting in irritation and bleeding from the stomach lining, a problem also associated with the later generation anti-inflammatory drugs.

Cortisone, the mother of all anti-inflammatories is associated with too many side effects to be used in the treatment of complaints associated with the normal course of muscle, tendon and joint attrition. Non-steroidal anti-inflammatory drugs or NSAIDs, the next generation agents came into vogue because of the perception of their having less side effects than aspirin.

In 1999 the Federal Drug Administration (FDA) approved the marketing of a class of drugs known as Cox-2-inhibitors. The selling feature of these agents was a marked decrease in stomach irritation and bleeding. And with FDA approval, Merck embarked on a massive and inordinately expensive marketing campaign that, in my view, was nothing less than obscene. Millions of dollars were spent on full page newspaper and prime time television advertisements. Thousands of "detail" people were hired to stalk and harass physicians in an attempt to persuade them to prescribe Vioxx. Also, there was a distribution to the profession of a myriad of trinkets: clocks, pens, clipboards, hats, refrigerator magnets to mention a few, emblazed with the Vioxx logo. This successful marketing effort resulted in Merck reaping 2.5 billion dollars in annual sales at the time of the recall. However, the marketing expense surely contributed to the inordinate expense for the patient, each Vioxx pill costing $2.50. While Merck and the pharmaceutical industry attribute the high cost of prescription drugs to research and development, unappreciated is the fact that it is marketing that drives up the retail price.

Let us return to the question of what did they know, when did they know it, and what did they do or not do about it? Late in 2000, I prescribed Vioxx for eight patients, took their blood pressures, and six of the eight developed high blood pressure. Merck representatives were notified regarding concern over this observation and information was requested about known complications of the drug. This seemed pertinent in that Merck publications did not describe hypertension as a possible adverse reaction. In response to my report and inquiries, Merck provided three responses: two on March 16, 2001 and one on July 17, 2001, by Jeffrey Melin, Associate Director of Medical Services. A correspondence dated March 16, 2001 reported on a study published in *The New England Journal of Medicine* on November 23, 2000 stating "hypertension was reported in 8.5% of patients treated with Vioxx, 50mg once daily" and that 0.6% of these patients discontinued the drug because of hypertension. Another correspondence dated July 17, 2001 reported that "17% of the patients treated with Vioxx 25mg once daily experienced an increase in systolic blood pressure" although the range of that increase was not delineated. It can therefore be concluded that early in 2001 high blood pressure was described as a side effect of Vioxx and that the company continued to distribute information circulars that did not list this problem as an adverse reaction. And to my knowledge, Merck never advised that blood pressures should be monitored in patients taking Vioxx.

To give Merck the benefit of the doubt, one must assume that they did not recognize the well established correlation between high blood pressure and stroke, nor between high blood pressure and heart disease. It will be interesting to see if the FDA recognizes the correlation.

On September 30, 2004, Merck announced a voluntary worldwide recall of Vioxx. This decision was based on the observation that patients taking Vioxx for longer than eighteen months in a clinical trial were twice as likely to have a stroke or heart attack as patients taking a placebo. To be

noted, the incidence of these adverse reactions was quite low: 1.5% for Vioxx and 0.75% for the placebo. To this orthopedic surgeon, the cause and effect sequence is obvious. A susceptible patient takes Vioxx and becomes hypertensive. If on the drug for a prolonged period of time, the recognized effects of high blood pressure will, in some patients, result in a stroke or heart disease. Again, to give Merck the benefit of the doubt, it appears that they have not recognized the causative correlation between Vioxx, hypertension, and stroke or heart disease. If the investigators who identified these complications had taken, as they should have, the patients' blood pressures, this information could either prove or disprove this theory.

The solution to the Vioxx conundrum: monitor the patient's blood pressure. If hypertension develops, discontinue the drug.

The only certainty in this situation is that the plaintiff attorneys, circling about like the class action vultures that they are, will be the ones to benefit from Merck's failure to understand the correlation between the drug, high blood pressure, and the complications.

Original Research

Intradiskal Electrothermal Therapy to Treat Discogenic Low Back Pain: Two-Year Follow-Up of a Multi-Center Prospective Cohort Study

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Abstract

Background: Options for the treatment of low back pain due to degenerative disc disease include non-operative care (physiotherapy and pharmacotherapy), arthrodesis or more recently disc replacement. In an effort to expand the treatment armamentarium, attention has been devoted to the development of minimally invasive alternatives. Intradiscal Electrothermal Therapy (IDET) is such a treatment, whose efficacy remains controversial. The purpose of this study was to determine the clinical success rate of IDET across multiple treatment centers, in a patient population who otherwise would have been candidates for arthrodesis. Standard outcome measures, Visual analog Scale (VAS), and the American Academy of Orthopaedic Surgeons Lumbar Spine Instrument (AAOS/LSI) were administered pre-treatment and at post-treatment intervals. Data were analyzed with respect to confounding variables based on demographic data, and outcomes.

Results: At twenty-four months post-procedure, VAS decreased from a mean of 6.3 ± 2.2 to 3.7 ± to 2.9 (P<.001), and significant improvement (P<.05) was noted in 6 of 8 scales on the AAOS/LSI. Both patient groups showed significant improvements in Short Form 36 scales, specifically Bodily Pain (BP), and Physical Function (PF). When the patients who went on to fusion were looked at as a separate subgroup, no significant differences in outcomes were noted. In terms of patient satisfaction, 78.6% of (44 of 56) responded "definitely or probably yes" as to whether they would choose the same treatment again. No particular demographic characteristics were found to predict a good outcome, or predict response to IDET *per se.* The sub-group that failed IDET and went on to fusion exhibited a similar rate of improvement in all outcome scales, but reported a significantly lower satisfaction rate.

Conclusion: These data are suggestive but not conclusive treatment of efficacy, due predominantly to limitations in study design.

Introduction

Degenerative disc disease is a common cause of low back pain. As a rule, treatment for non-radicular, axial pain is non-operative, despite the fact that the optimal conservative regimen remains has not been defined. In cases refractory to non-operative treatment, arthrodesis has been suggested, but considerable variability in outcomes has been reported.[1–6] Disc or nuclear replacement has also been proposed as a treatment for single level symptomatic degenerative disc disease.[7–12] This has the theoretical advantages of motion preservation lowering the risk of adjacent segment degeneration. A relative disadvantage of disc or nuclear replacement is the magnitude of the surgery, at least equivalent to most conventional open fusion techniques. In an attempt to refine treatment, and to increase the number of options in the treatment armamentarium, Intradiscal Electrothermal Therapy (IDET) was developed.[15]

Thermal energy has been shown to shrink collagen fibers in peripheral joint capsules, cauterize and disrupt granulation tissue, and cause nociceptive cell death.[13–14] A spinal catheter (ORATEC Interventions, Menlo Park, California) was developed in an effort to deliver thermal energy intradiscally and realize potential clinical benefits from thermal effects on tissue stabilization and neural ablation.[16] Clinical reports, have suggested a therapeutic effect in patients with low back pain caused by degenerative disc disease, as confirmed by provocative discography.[15–19]

The present study was designed to assess the potential therapeutic effects of IDET on discogenic low back pain. The study was designed to select patients who otherwise would have been candidates for fusion.

Materials and Methods

Six centers participated in the study. Inclusion criteria were: three or more months of low back pain, with or without non-radicular referred pain, low back pain greater than referred pain, failure of conservative care (active physical therapy, non-steroidal anti-inflammatory medications), and MRI consistent with degenerative disc disease. Exclusion

criteria were: previous surgery at the treated level, disc prolapse with significant radicular pain, spondylolisthesis, kyphosis, pregnancy, confounding systemic disease, spinal infection, tumor, or psychosis. Symptoms were confirmed by provocative discography; patients were candidates for study entry with a maximum of two positive and concordant levels, with negative rostral and caudal controls, where appropriate.

A variety of demographic data were gathered. A standard history and physical examination were conducted including a detailed neurological examination. Additional exclusionary criteria were weakness, or signs of nerve root irritation, as suggested by the presence of root tension signs.[20] Standard outcome instruments were used — a 10 cm Visual Analog Scale (VAS) and the American Academy of Orthopaedic Surgery Lumbar Spine Instrument (AAOS/LSI) which includes the Short Form 36 (SF-36), and additional outcome questions. Patients were asked to complete these before the procedure, and at 2-year follow-up.

IDET was performed in a standard manner under conscious sedation, using biplanar fluoroscopy. Catheter position was monitored during the procedure with the use of 1 of 3 described temperature protocols left to the discretion of the treating physician. Protocol I entailed a maximum catheter temperature of 80° centigrade, Protocol II, 85° temperature maximum, and Protocol III, 90° maximum. Patients were monitored during the procedure with VAS scores. In no case did the procedure have to be terminated.

Results

74 patients were originally enrolled. Of the 74, three were excluded. One patient was excluded due to mental health issues requiring treatment, and one underwent implantation of a subarachnoid infusion system for pain control. In this instance, the intervention was indicated for pain other than that for which the IDET was recommended, and study participation was terminated. One patient was improperly enrolled initially. Complete 24-month follow-up data were available on 56 of 71(78.8%).

Of the initial 71 patients — 53 were treated with one time IDET only. Of this cohort, 41 (77.4%) completed the 2-year follow-up assessment. Three patients underwent repeat IDET; two of them completed the 2-year follow-up for a final IDET only cohort of 43 (Table 1). 15 patients who reported unacceptable pain relief post IDET underwent fusion; 13 completed the 2-year follow-up protocol. Outcomes were reviewed for the study group as a whole, as well as for sub-groups — most notably, the group that failed IDET and went on to fusion, (n=13).

Table 1

		Overall	IDET	Fusion	P-Value
Number Treated	1	67.9%	63.4%	75.0%	.7320
	2	32.1%	36.4%	25.0%	
Disks Treated (1st Treatment)	L5/S1	32.8%	34.1%	33.3%	.6786
	L4/L5	25.9%	20.5%	41.7%	
	L4/L5 & L5/S1	19.0%	22.7%	8.3%	
	L3/L4 & L4/L5	8.6%	9.1%	8.3%	
	L3/4	5.2%	4.5%	0.0%	
	L3/L4 & L5/S1	3.4%	2.3%	8.3%	
	L2/3	3.4%	4.5%	0.0%	
	L2/L3 & L4/L5	1.7%	2.3%	0.0%	

Two patients who completed follow-up assessment received a second IDET. The treated disks for these 2 patients are given below.

First Treatment	Second Treatment
L2/3 (right approach)	L2/3 (left approach)
L5/S1	L5/S1 and L4/5

Disks treated: A total of 58 procedures in 56 patients were performed. There were no significant differences between groups.

There were no significant differences in demographic characteristics between the fusion sub-group and the remainder. All patients had disabling LBP for at least three months, with 75% having had symptoms for 12 or more months. 49% had never smoked cigarettes, and 26.4% were former smokers. Most patients had attended college, or completed college (79.3%). 70.7% were married and 55.3% did not attribute their back pain to any trauma or injury. Of the 12.5% who underwent previous back surgery, none had previous surgery at the levels to be treated with IDET. Most patients had central back pain (86.2%), with approximately half having paracentral pain and lower numbers of patients having non-radicular distal pain. All patients had 1 or 2 levels treated with single level treatment being the most common (67.9% vs. 32.1%). Levels treated are as noted in Table 1. Thirty-seven patients underwent the 90 degree maximum temperature protocol; fourteen, the 85 degree maximum protocol; and five the 80 degree maximum protocol.

Outcome data: Mean VAS preoperatively was 6.3 (+/–2.2). At 24-months mean VAS were 3.7 (+/–2.9) (P<.0001). Raw data from the SF 36, adapted from the AAOS/LSI for the entire study cohort, are shown on Table 2. Pooled data from all patients show significant improvements in 6 of 8 scales (all scales except Role Emotional (RE), and General Health (GH)). According to Deyo, et al.[21] changes in SF-36 scales of 7 points or more are considered to be significant. Such significant changes were noted in Bodily Pain (BP), and Physical Function (PF) scale. An overall improvement in quality of life is suggested by significant improvements in Role Physical (RP), Social Functioning (SF), Vitality (VT), and Mental Health (MH) scales.

Table 2

N=56	Baseline Mean	N-56 Per	24 Months Mean	Std	Change Mean	Per	T-test P-value
VAS	6.3	2.2	3.7	2.9	2.6	2.6	<.0001
PF	36.7	22.6	59.7	31.2	23.0	27.1	<.0001
BP	25.8	14.9	48.3	28.7	22.5	24.7	<.0001
RP	18.3	32.1	48.1	45.1	29.8	44.6	<.0001
RE	64.7	42.0	66.0	44.5	1.3	50.3	.8548
SF	48.6	27.3	66.2	33.0	17.6	27.8	<.0001
VT	39.6	21.3	53.3	24.8	13.7	26.3	.0004
GH	71.7	18.1	68.0	23.7	–3.6	19.5	.1799
MH	62.7	18.9	69.2	21.9	6.5	22.1	.0360
Neurogenic Symptoms	57.7	31.7	72.7	31.0	14.7	28.2	.0004
Pain/Disability	44.9	16.6	62.3	25.9	18.0	20.4	<.0001

Outcomes: The mean outcomes, pre-treatment at 24-months are noted. A significant improvement in pain as a result of treatment is suggested by significant improvement in VAS, bodily pain scale. Additionally, significant improvement in physical function (PF was noted), as well as improvements in overall quality of life (QCL) as demonstrated by significant improvements in roll physical (RP), social function (SF), vitality (VT) and mental health ((MH) scales of the SF-36.

Outcome data for IDET only ("IDET") and fusion subgroups are shown in Table 3. Mean VAS in IDET declined from 5.9 pre-treatment to 3.4 post-treatment (P<.0001). The mean VAS in the fusion subgroup declined from 7.4 to 4.4 (P=.0034). There were no significant differences in VAS and SF-36 scale scores between the IDET and fusion subgroups. All patients showed significant improvement in neurogenic symptoms based on the AAOS Neurogenic Symptom Scale.

Table 3

		Baseline Mean	Std	24 Months Mean	Std	Change Mean	Std	Overtime P-value
VAS	IDET	5.9	2.3	3.4	2.8	2.4	2.4	<.0001
	Fusion	7.4	1.4	4.4	2.9	3.0	3.0	.0034
	Groups	.0241		.2703		.4832		
SF 36 Data								
PF	IDET	38.7	23.6	61.3	31.1	22.7	24.8	<.0001
	Fusion	30.4	18.9	54.7	32.1	24.3	34.5	.0259
	Groups	.2529		.5056		.8532		
BP	IDET	27.1	15.6	50.1	28.6	23.0	24.4	<.0001
	Fusion	21.5	12.1	42.4	29.4	20.8	26.7	.0157
	Groups	.2447		.4006		.7826		
RP	IDET	21.3	35.1	50.6	47.2	29.4	45.6	.0002
	Fusion	8.3	16.2	39.6	37.6	31.3	42.8	.2080
	Groups	.2254		.4621		.8998		
RE	IDET	62.5	42.8	65.8	45.6	3.3	53.8	.6973
	Fusion	72.2	39.8	66.7	42.6	–5.6	37.1	.6147
	Groups	.4869		.9553		.5218		
SF	IDET	51.0		68.9	32.2	18.0	26.2	<.0001
	Fusion	41.3	30.8	57.7	35.1	16.3	33.6	.1050
	Groups	.2754		.2901		.8551		
VT	IDET	41.3	20.4	55.4	25.6	14.0	25.0	.0010
	Fusion	34.2	24.0	46.9	21.9	12.7	31.0	.1665
	Groups	.3010		.2906		.8743		
GH	IDET	74.0	17.2	71.1	24.0	–2.7	19.3	.3767
	Fusion	65.1	20.0	58.7	21.3	–6.5	20.7	.2820
	Groups	.1342		.1011		.5547		
MH	IDET	62.8	18.0	68.8	22.8	6.0	2.0	.0757
	Fusion	62.5	22.0	70.5	19.3	8.0	26.1	.2904
	Groups	.9624		.8145		.7851		
Additional Scales								
Neurogenic Symptoms	IDET	59.3	33.9	73.5	32.5	13.8	29.2	.0050
	Fusion	52.6	23.7	70.0	26.5	17.4	25.8	.0313
	Groups	.5060		.7263		.6867		
Pain/Disability	IDET	47.1	17.7	63.4	26.1	17.1	18.1	<.0001
	Fusion	38.0	10.4	58.9	26.1	20.8	26.7	.0158
	Groups	.0889		.5887		.5726		

Outcomes between and within groups: Groups were analyzed internally, to determine whether significant differences occurred as a result of treatment with the groups; the analysis was then repeated for differences between groups. Both groups, IDET only (I), N=43, and fusion (II), N=13, demonstrated significant improvement in pain at 24-month follow-up based on VAS, and significant scale changes in BP. Both groups demonstrated significant improvement in physical function at 24-months, as well as similar improvement in overall QOL (RF, SF, AND VT).

Patient Satisfaction (Table 4): Patients were asked, "if you could go back in time and make the decision again, would you chose the same treatment for your musculoskeletal conditions/problem?" Overall, 78.6% answered "definitely yes or probably yes". In the sub groups, 60.5% of the IDET sub group answered "definitely yes" vs. 23.1%, of the fusion subgroup, P=0.0048). In the remainder of the responses — "probably yes," "completely uncertain," "probably not," and "definitely not" — there were no differences between subgroups.

Table 4

	Overall	IDET	Fusion	P-value
Definitely yes	51.8%	60.5%	23.1%	.0048
Probably yes	26.8%	23.3%	38.5%	
Completely uncertain	5.4%	0.0%	23.1%	
Probably not	7.1%	9.3%	0.0%	
Definitely	8.9%	7.0%	15.4%	

Patient Satisfaction (24 months f/u): In the "definitely yes" category, the results were significantly better in Group I (IDET only) as compared to Group II (fusion). Overall, 83.8% of patients in Group I responded that they would definitely or probably choose the same treatment again, vs. 61.6% of the fusion group.

Work Tolerance: Overall work tolerances improved. A higher percentage of IDET sub group were able to perform 75% to 100% of their jobs at 24-months post treatment, but this difference was not statistically significant. The use of pain medication also improved. There was no significant effect between subgroups for medication usage. Patients who used no medications at all were comparatively rare.

Technical Factors: Several morphologic characteristics were analyzed with respect outcome presence of osteophytes, presence of high intensity zone (HIZ) on MRI,[22] presence of Modic changes,[23] Adams-Hutton discogram grade,[24] or full vs. partial thickness tear. None were significant (P = .0569). Additionally, there were no significant differences in outcome according to which of the three temperature protocols were utilized.

Table 5. Overall

	IDET Pre-treatment	IDET 24 Months Post-treatment
Perform 100% of job	18.5%	40.7%
Perform 75% of job	25.9%	29.6%
Perform 50% of job	13.0%	5.6%
Perform 25% of job	9.3%	3.7%
Cannot perform any part of job	33.3%	20.4%

Sub Groups

	IDET Pre-treatment	IDET 24 Months Post-treatment	Fusion Pre-treatment	Fusion 24 Months Post-treatment
Perform 100% of job	22.0%	45.2%	7.7%	25.0%
Perform 75% of job	29.3%	31.0%	15.4%	25.0%
Perform 50% of job	0.0%	4.8%	7.7%	8.3%
Perform 25% of job	7.3%	2.4%	15.4%	8.3%
Cannot perform any part of job	26.8%	16.7%	53.9%	33.3%

For analysis purposes the above data was collapsed into the following categories.

	IDET Pre-treatment	IDET 24 Months Post-treatment	Fusion Pre-treatment	Fusion 24 Months Post-treatment
Perform 75%–100% of job	51.3%	76.2%	23.1%	50.0%
Perform 50% or less of job	48.7%	23.8%	76.9%	50.0%

Work Tolerance: A repeated measures analysis was used to test for differences over time and/or by group. It was found that both groups improved over time (p=0.0013). However, both times the IDET group was more likely to be working at 75%–100% of their job (p=0.0409).

Discussion

The rates of clinical improvement reported in the present study are similar to those reported in the literature.[1–6, 15–18] All of these studies suffer from the same study design flaw — retrospective cohort. In the current prospective study, patients, including those who failed IDET and went on to fusion, demonstrated significant improvement in BP and PF scales on the SF-36. Significant improvement in quality of life was suggested by improvement in the RP, SF, and VT scales as well. Significant improvements in pain driven scales (BP and PF) and QOL scales were also noted.

Patient satisfaction was greater in those that did not go to fusion. Patients who responded "definitely yes" as to whether or not they would choose the same treatment again were heavily weighted toward the IDET group (60.5% vs. 23.1% P = .0048). It is certainly intuitively plausible that a significant negative bias coupled with enhanced expectations for treatment outcome was present in the patients who elected to undergo fusion. This bias is further suggested by the findings in one particular categorical outcome response: the "completely uncertain" response was given by none in IDET only

subgroup and by 23.1% of fusion patients. It is tempting to infer that this may be explained by the minimal morbidity of IDET as compared to fusion. However, if the categories of "definitely yes and probably yes" are combined, there is no statistically significant difference between these subgroups (83.8%, v 61.6%). These data aside, the design of this study precludes any meaningful comparison of the effectiveness of IDET, as opposed to fusion, for the treatment of discogenic low back pain. Further limitations include sample size.

Interestingly, whether or not Intradiscal therapy, such as IDET, or fusion are reasonable therapeutic approaches for discogenic pain remains surprisingly unclear. The bulk of retrospective case controlled studies suggest a therapeutic effect for both. There remains however, a glaring paucity of rigorous data regarding treatment efficacy for either approach; to date, only two randomized prospective studies have attempted to compare fusion directly to non-operative care as a method of treating back pain presumably due to degenerative lumbar disease and only one similarly designed report regarding IDET has appeared.

Regarding fusion, Fritzell et al.[25] reported the results of a randomized prospective study-comparing operative vs. non-operative treatment for chronic low back pain. This study reported 2-year follow-up on 294 LBP patients who were randomized into four treatment groups, three operative and one non-operative. Selection of fusion levels was based on the presence of degenerative changes on Computed Tomography (CT) or MRI. Clinical results were significantly better in the surgical group. Back pain was reduced in the surgical group by 33% (vs. 7% in the non-surgical group, P=.0002). 63% of the surgical group rated themselves as "much better, or "better", vs. 29% in the non-surgical group (P<.0001). The return to work rate was also higher than the surgical group, although absolute numbers were low in both groups (36% vs. 13%, P=.002). Brox et al.[26] compared the results of treatment of back pain from degenerative lumbar disc disease between two treatment groups. 64 patients were randomly assigned either instrumented posterior fusion with postoperative physical therapy or to cognitive intervention and exercise. At one year follow up, similar improvements in the Oswestry Disability Index (OD1) were reported by both groups with success rates, as reported by an independent observer of 70% and 76% in the surgical and cognitive-exercises groups, respectively. Both of these studies are well designed, but both may be criticized for surgical selection criteria, and in the case of the study of Fritzell et al., considerable variability within treatment groups. The study of Brox et al. appears to have less variance within treatment groups, but the same criticism for surgical selection applies: both studies selected patients for fusion on the basis of degenerative disc disease as demonstrated on imaging techniques (planar films, Magnetic Resonance Imaging, and Computed Tomography) which are widely acknowledged to be highly sensitive and highly nonspecific.[27] As such, the diagnosis of back pain due to degenerative lumbar disc disease itself, as well as selection criteria for levels to be fused are questionable in both of these studies.

The six month outcomes of a randomized double-blind placebo controlled trial of IDET vs. a sham discal intervention were presented by Pauza et al.[28] In this study, the preliminary results of 64 patients were reported. 32 of 37 assigned to the IDET group complied with study protocol and were included in the final data analysis; corresponding numbers in the Sham group were 24 of 27. Mean improvements in pain, disability and depression were significantly greater in the IDET group. 40% of patients reported greater than 50% improvement in pain; 50% of patients experienced no appreciable benefit. While rates of improvement in the study of Pauza et al.[28] also suggest significant differences between active and sham groups, absolute rates of improvement are also disappointingly low.

Despite the fact that the current data suggest a therapeutic effect from IDET, the precise mechanism of that presumptive effect remains unknown. Thermal energy has been shown to induce tissue shrinkage in cadaver and animal models and has been applied to treat peripheral joint instability.[13, 14, 29, 30] Several studies have evaluated the histology and stability of the lumbar spine after IDET. Lee et al[31] assessed spinal stability non-destructively in 5 IDET treated cadaver specimens. They found that increasing preloads resulted in a decrease in the motion of the spinal segment in all planes of testing. There was no significant difference in the stability of the lumbar spine before and after treatment with IDET. Based on these data, the authors concluded that the IDET did not destabilize the spinal motion segment *in vitro.* Shah, et al.[32] studied the histology of cadaver discs following IDET. Light microscopy of the posterior aspect of the anulus revealed denaturation, shrinkage, and coalescence of collagen. Neither the anterior anulus — an internal control — nor the end plates showed evidence of damage. Electron microscopy revealed extensive collagen disorganization, decreased collagen quantity, fibril shrinkage and chondrocyte damage. Temperature mapping showed parallel changes in temperature at the level of the probe and the posterior anulus. The authors concluded that IDET raised temperature sufficiently to induce collagen denaturation.

These findings are in contrast to those of Kleinstueck et al.[33] In this study, IDET was performed on 19 fresh frozen lumbar cadaver experiments. A pattern of increased motion and decreased stiffness was observed following treatment. This suggests an effect on collagen; however, histology revealed no apparent alteration of the anular architecture around the catheter sites in IDET treated discs. Additionally, the measured disc temperatures ranged from 37° to 65°C (despite use of the 90°C. 17 minute protocol). These temperatures were highest within 1mm of the catheter and declined to near body temperature of 10–15 mm. The apparent discrepancy between this study and others[30, 31] may be due to the model used to support the cadaver spine, a 37°C water bath. While the data are internally consistent, the water bath may

have acted as a heat sink, preventing anular heating. Any of these studies, however suffer from the limitations of *in vitro* models. Clearly, an *in vivo* model is required so that the adaptive processes of healing can be taken into account to determine both the biologic and histologic consequences of the procedure.

The current study was designed to study outcomes post IDET across several centers in a standardized patient group. The finding that 13 patients failed IDET and went on to fusion provided an opportunity for coincidental subgroup study. It is interesting that the rates of self-reported improvement in pain, function, and quality of life are similar in both subgroups. Although patient satisfaction was slightly (although not to an accepted level of statistical experience) higher in the IDET only subgroup, it should not be inferred to mean that patients are more likely to be pleased with the amount of pain relief obtained only if it could be achieved with a less momentous treatment; clearly the patients who did not respond to IDET and went onto fusion would not be expected to have been as satisfied as they would have been had IDET met their expectations.

On the basis of these data, a convincing recommendation for IDET to treat discogenic low back pain cannot be made. In a study such as this, without a *bona fide* control group, the effect of intervention cannot be excluded. The only manner in which this can be controlled is in a randomized prospective study.[34, 35] To date, two studies, as discussed above, have attempted to do this with fusion;[25, 26] neither has convincingly demonstrated efficacy compared to non-operative care. A preliminary attempt has been made with IDET.[27] While this study did report somewhat better results in the active lesion group, absolute rates of clinical improvement were low.

Additional basic science studies, focusing on *in vivo* models as well as longer-term randomized prospective blinded clinical studies are required. Until such data are available the proper role of IDET, if any, in the clinical armamentarium cannot be defined.

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Original Research

C5-C6 Foraminal Stenosis as Possible Explanation for Manifestation of Burner Phenomenon

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Abstract

Background: An anatomical dissection study was performed to determine the relative dimensions of the C5-T1 nerve roots and foramina to investigate a predisposition to compression burner syndrome. Fourteen cadavers were used in the study. The inner and outer foraminal and nerve heights and widths for levels C5-T1 were measured. Functional reserve values (Foramen Height – Nerve Height or Foramen Width – Nerve Width) were calculated on the inner and outer sides of the intervertebral foramina.

Results: The inner height functional reserve for C5 and C6 was 0.15 +/– 0.17 cm and 0.13 +/– 0.17 cm respectively while the functional reserve for levels C7-T1 was 0.30 +/– 0.07 cm. The inner width functional reserve for C5 and C6 was 0.02 +/– 0.06 cm and 0.04 +/– 0.14 cm respectively while the functional reserve for levels C7-T1 was 0.13 +/– 0.17 cm. The outer height functional reserve for C5 and C6 was 0.29 +/– 0.25 cm and 0.16 +/– 0.21 cm respectively while the functional reserve for levels C7-T1 was 0.17 +/– 0.14 cm. The outer width functional reserve for C5 and C6 was 0.07 +/– 0.19 cm and 0.04 +/– 0.12 cm respectively while the functional reserve for levels C7-T1 was 0.20 +/– 0.16 cm. C5 and C6 inner height functional reserve values were significantly lower than the remaining levels (p(C5 vs. C7-T1) = 0.005 and p(C6 vs. C7-T1) = 0.004). Moreover, C5 and C6 inner width functional reserve values were significantly lower than the remaining levels (p(C5 vs. C7-T1) = 0.012 and p(C6 vs. C7-T1) = 0.002). The outer measurements did not exhibit significant differences.

Conclusion: There is less functional reserve for the C5-C6 nerve roots on the inner side of the intervertebral foramina, thereby rendering them more at risk for compression. This should help explain the preponderance of the C5-C6 symptoms seen clinically.

Introduction

Injuries to the brachial plexus are commonly seen with motorcycle and motor vehicle accidents, penetrating wounds to the shoulder, obstetric traumas and athletic injuries. Injuries are characterized as either brachial plexus *traction* or *compression* injury. *Traction* is the most common injury to the brachial plexus, and usually results from the distraction of the head away from the ipsilateral shoulder.[7] If the arm is abducted at the moment of injury, the lower brachial plexus roots suffer the greatest stress, while if the arm is adducted, the upper roots sustain the most traction.[7] *Compression* injuries of the plexus are caused chiefly by compression of the nerve roots or dorsal root ganglia at the intervertebral foramina.[7] Extension and/or axial compression of the cervical spine, with concomitant foraminal constriction, is a common means of evoking compression plexus injuries.

Burner syndrome is a common injury in contact sports such as football. It usually occurs after an athlete's head or shoulder collides with another player or against the ground. This leads to either a traction injury of the ipsilateral side[10] or a compression injury of the ipsilateral brachial plexus nerve roots (extension-compression burner). The athlete experiences immediate pain, burning, or tingling that originates in the neck and radiates down the arm as far as the fingers. Furthermore, weakness is commonly observed clinically a few days later, especially in the C5-C6 distribution (e.g. spinati, deltoid, and biceps).[10]

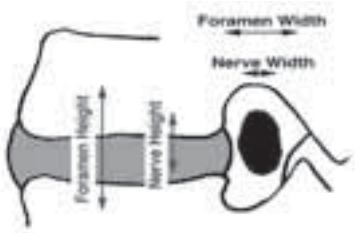
Since the C5-C6 level approximates the axis of rotation of the subaxial spine, extension or lateral bending would be expected to produce significant foraminal narrowing at this level.[6] However, no study to date has examined the anatomic features of the brachial plexus nerve roots and their respective intervertebral foramina as predisposing factors to compression injuries. We were interested in answering the following question: Are there inherent anatomic differences in the C5/C6 brachial plexus levels compared to the remaining levels that promote compression injuries of their nerve roots? We undertook an anatomical dissection study to determine the relative dimensions of the nerve roots and the intervertebral foramina of the brachial plexus in order to investigate a possible predisposition to compression injuries at the C5-C6 foramina, thus explaining the preponderance of C5-C6 deficits seen clinically.

Material and Methods

Fourteen cadavers were used in the study. All cadavers were embalmed prior to dissection. The cadavers had an average age of 78 years at the time of death, and consisted of 7 men and 7 women. After removing the skin, cervical spines were cut mid-sagittally and the left cervical spine nerve roots

and brachial plexus nerves were carefully dissected. The right side was discarded. Before removing the nerve roots from their foramina, a digital caliper (Mitutoyo Corporation, Kanagawa, Japan) was used to measure the nerve heights and widths for each brachial plexus level, both on the inner and outer sides of its intervertebral foramen. Height was defined as the cephalic-caudal dimension, and width was defined as the anterior-posterior dimension. Subsequently, the nerves were carefully removed from the intervertebral foramina, and the foraminal widths and heights were measured on both inner and outer sides. Therefore, for every level of the brachial plexus, eight values were measured: outer nerve height, outer nerve width, outer foramen height, outer foramen width, inner nerve height, inner nerve width, inner foramen height and inner foramen width.

We defined a new value called "functional reserve" as the difference between a given foraminal dimension and the respective nerve dimension, (i.e., functional reserve = foramen dimension – nerve dimension). This value reflected the average amount of space the nerve has within its foramen. For example, to calculate the functional reserve of C5 nerve height on the inner side of the foramen, we subtracted the height of the nerve on the inner side of the C5 level from the height of the foramen on the inner side (Figure 1). Functional reserve values for C7, C8 and T1 were averaged into one value. Paired t-tests were performed to compare functional reserve of C5 and C6 to the average functional reserve of C7-T1 for the inner and outer heights and widths.



Functional Reserve = Foramen Dimension – Nerve Dimension

Figure 1. Functional reserve.

Results

The ***inner height functional reserve*** (inner foramen height minus inner nerve height) for levels C5 and C6 were 0.15 ± 0.17 cm and 0.13 ± 0.17 cm respectively, and the average inner height functional reserve for levels C7-T1 was 0.30 ± 0.07 cm.

The ***inner width functional reserve*** (inner foramen width minus inner nerve width) for levels C5 and C6 were 0.02 ± 0.06 cm and 0.04 ± 0.14 cm respectively, and the average inner width functional reserve for levels C7-T1 was 0.13 ± 0.17 cm.

The ***outer height functional reserve*** (outer foramen height minus outer nerve height) for levels C5 and C6 were 0.29 ± 0.25 cm and 0.16 ± 0.21 cm respectively, and the average outer height functional reserve for levels C7-T1 was 0.17 ± 0.14 cm.

The ***outer width functional reserve*** (outer foramen width minus outer nerve width) for levels C5 and C6 were 0.07 ± 0.19 cm and 0.04 ± 0.12 cm respectively, and the average outer width functional reserve for levels C7-T1 was 0.20 ± 0.16 cm.

Paired t-tests performed using an alpha value of 0.05 confirmed statistical significance when comparing both C5 and C6 inner heights and widths to their respective C7-T1 dimensions. Both C5 and C6 ***inner height*** functional reserve values were significantly lower than the remaining levels C7-T1, with p(C5 vs. C7-T1) = 0.005 and p(C6 vs. C7-T1) = 0.004. Likewise, both C5 and C6 ***inner width*** functional reserve values were significantly lower than the remaining levels, with p(C5 vs. C7-T1) = 0.012 and p(C6 vs. C7-T1) = 0.002.

Conversely, the ***outer height*** functional reserve values were not significantly different when comparing C5 or C6 to the remaining levels. The ***outer width*** functional reserve was not significantly different when comparing C5 to C7-T1, but was different when comparing C6 to C7-T1, with p(C6 vs. C7-T1) = 0.014.

Discussion

The "burner" syndrome, also known as "stinger," receives its name from the symptoms experienced by the athlete after injury. The incidence of burners in tackle sports such as football, rugby, wrestling and soccer is high. Defensive football players have the highest incidence among athletes.[3] About half of football players surveyed in two different studies reported having experienced at least one burner.[10] Most of these occurrences go unnoticed,[2] and only 10% of the cases get examined by a team physician.[10] The athlete feels a sudden sharp burning sensation radiating down the arm, but this sensation does not correspond to any specific dermatomal distribution.[3] This burning feeling disappears in minutes, and a motor exam is often normal soon after injury. True clinical weakness can usually be elicited a few days after the injury.[3]

Traction injuries are common in burners and appear more often in high school and college football players with acute symptoms. Compression injuries, the focus of our study, are more common in college and professional players with chronic or recurrent burner syndromes.[8] Compression usually affects the nerve roots and/or the dorsal root ganglia within their intervertebral foramina.[10] It commonly occurs after hyperextension or hyperextension with lateral flexion of the neck. Intrinsic factors predisposing to compression burners include cervical disc disease or developmental cervical stenosis.[8] Patients who sustain a compression burner often have a positive Spurling's test.[10] This test involves reproducing symptoms by applying axial pressure to the

head, forcing the neck into extension and ipsilateral lateral flexion.

Some studies have attempted to identify athletes predisposed to sustaining a burner.[1] Kelly et al. investigated the relationship between the Torg-Pavlov ratio and the occurrence of burners in athletes and concluded that patients with a low Torg-Pavlov ratio were more likely to develop burners.[4, 5] Meyer et al. confirmed the above conclusion and reported that players with a ratio of less than 0.8 had three times higher incidence of extension-compression burners than those with a ratio higher than 0.8.[9]

While the Torg-Pavlov ratio can screen the athletes with more risk of developing extension-compression burners, no study to date has investigated why patients predisposed to burners are more likely to present with C5-C6 nerve distribution weakness. In our study, the C5 and C6 nerve roots appear to occupy more relative space within their respective foramina on the inner side of the intervertebral foramina than the remaining levels of the brachial plexus. The inner foraminal diameter represented the minimum foraminal diameter. Therefore, there is less functional reserve for the C5 and C6 nerve roots, which render them more at risk for nerve root or dorsal root ganglion compression injuries. This, in addition to the C5-C6 level approximating the apex of bending of the subaxial spine, helps to explain the predominant C5-C6 symptoms experienced by burner patients.

A weakness of the study was the average age of the cadaveric specimens, 78 years old, and the inherent degenerative changes that have taken place by that age. In addition, the average age of patients experiencing burners is significantly less than the age of our specimens. Interpretation of our findings must be considered in this light.

In this anatomical study, we have demonstrated that C5 and C6 have less reserve space within their inner foramina than the remaining levels of the brachial plexus. This may explain partially the preponderance of the C5 and C6 nerve distribution muscle weakness observed clinically.

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Original Research

Prophylactic Knee Bracing of Offensive and Defensive Linemen in College Football

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Abstract

Background: Knee injuries in high school, college, and professional sports are common and can significantly jeopardize an athlete's career. Over the past few decades the concept of prophylactic knee-bracing has evolved to prevent or lessen the incidence of these hazardous injuries. Unfortunately, the scientific community has been unable to reach a consensus over the efficacy of brace prophylaxis. After a series of significant knee injuries to offensive and defensive football linemen of Temple University, a Division I program, mandatory prophylactic knee-bracing was instituted for all lineman.

Results: Our study prospectively examined the incidence of knee injuries for three years after institution of mandatory prophylactic knee-bracing and compared it retrospectively to the three years prior. A statistically significant decrease in the incidence of these injuries was found after the institution of bracing. However, there was not a statistical significant decrease in the severity of all knee injuries.

Conclusion: This data further supports the movement to promote prophylactic knee-bracing in football linemen.

Introduction

Knee injuries in high school, college, and professional football are common and can significantly jeopardize an athlete's career. They result in lost practice/game time, substantial financial consequences, and can end an athlete's career. With these devastating possibilities in mind, the concept of prophylactic knee bracing (PKB) has evolved as a method to prevent and/or lessen the incidence of knee injuries. Since the concepts inception in the 1970's, the scientific community has had much difficulty in arriving at a consensus to the efficacy of bracing. Anderson et al. proposed the first use of the prophylactic knee brace in 1979.[1] They demonstrated prevention of re-injury to the medial collateral ligament (MCL) in nine players who used a lateral upright brace (Anderson Knee Stabilizer) after their injury. It was therefore extrapolated that a knee brace could also have the ability to prophylactically prevent new knee injuries from occurring. Over the next 24 years several epidemiological studies have both proven and disproven the efficacy of the PKB.[2–4, 7–11]

During the preseason of the 1999 Temple college football season, a member of the Division I Big East Conference, a third degree MCL and ACL injury occurred to one of Temple's offensive linemen. This incident spurred the coaches on the recommendation of the team orthopaedists to institute the mandatory use of prophylactic knee bracing during all practices by the offensive and defensive lineman. The goal of our study was to prospectively assess whether the newly instituted use of PKB's in practice over the course of the next three seasons would result in a decrease in the number of knee injuries to linemen as compared to the previous three seasons.

Materials and Methods

Beginning in 1999, offensive and defensive linemen on Temple University's football team were required to wear a knee brace during all practices. The brace was a custom molded double sided, dual hinged Breg® (Vista, California) X2K knee brace worn bilaterally. The players were allowed to shed the brace during game time. To eliminate bias, all injuries were triaged and recorded by a single trainer, author D.S. In addition, all injuries were diagnosed and treated by a single attending Orthopaedic surgeon, author R.M., from the Department of Orthopaedic Surgery and Sports Medicine of Temple University. The study was continued prospectively for three consecutive seasons; 2001, 2002, and 2003. The year 2000 was excluded because of variability in the enforcement of the knee brace. The control was designated to be the preceding 3 seasons (1997, 1998, and 1999) where prophylactic knee bracing was not used through retrospective review of the records and charts. The coaching staff and practice regiments remained the same during all six seasons. Each lineman was required to wear the knee braces bilaterally. The device was attached to the thigh and lower leg with Velcro® and was placed beneath the pants (Figure 1). New braces were issued when the original was lost or damaged. Each time a knee injury resulted in lost playing time, the injury was recorded. The same Orthopaedic attending surgeon, R.M., diagnosed the injury. All of the knee injuries during this period were limited to ligamentous ones involving the Anterior Cruciate (ACL) and/or the Medical Collateral Ligaments. Players were excluded from the data set if they had an injury prior to 1999 or if they were a re-injury during 2001–2003. An injury was included in the study if resulted in lost game playing time. Statistical analysis was then performed on the data set.

Figure 1. Breg X2K brace.

Results

Incidence/Exposure of Knee Injury

Year	Total # Linemen	Total # Knee Injuries	Total # Playing Hours*
Pre-Bracing (1997–99)	66	12	331
Post-Bracing (2001–03)	69	5	327

*Average of one season over the three season intervals, including practice and game time.

p-value is 0.041, statistically significant, by Chi-Square testing.

Type of Knee Injury

	ACL Injury	MCL Injury
Pre-Bracing (1997–99)	3	9
Post-Bracing (2001–03)	1	4

p-value is 0.293, not statistically significant, by Chi-Square testing.

Discussion

Over the past twenty-four years there has been a debate as to whether or not Prophylactic knee braces have decreased the incidence of knee injuries. Several studies are in favor of their use.[2–4, 10–11] In 1990, Sitler et al.[2] studied 1396 West Point cadets who were required to play 8-man tackle football. This prospective study lasted 2 years, each player without regard to position, was randomized to the brace or non-brace group, and adherence was mandatory. They showed a statistically significant decrease in knee injuries, including MCL sprains, and a trend towards less severe injury if one were to occur (3.40 injuries per 1000 athletes if non-braced and 1.50 per 1000 if braced). In 1994, a 3-year prospective multi-institutional study of 987 healthy college football players was conducted by Albright et al. and the Big Ten Sports Medicine committee.[3, 4] They noticed that offensive and defensive linemen had incurred the most MCL injuries and were thereafter both more likely to wear a brace. Controlling for position, they showed a consistent but not significant trend of lower injury rates in the braced versus non-braced athlete. More importantly they were able to contradict a study by Teitz et al.[5] which showed an increase in injuries rates while wearing a brace, by showing that PKB's did not increase the rate of knee injuries. Several other studies were inconclusive.[7–9] In 1987, Garrick et al.[6] reviewed six studies relating PKB's and MCL injury and they were unable to arrive at any conclusions. Two studies showed a statistically significant lower number of MCL injuries,[10, 11] two reported decreasing trends of MCL injuries,[7, 8] and two reported increases in knee injuries while wearing a brace.[5, 9] Surrogate limb models were used in the late 80's and early 90's in hopes to develop a reproducible model that could test the strain on knee ligaments. France et al.[12] used spring loaded cables (based on cadaver ligament strain averages) to simulate the strain place on ligaments when a knee is subjected to lateral valgus forces. They determined a "critical" impact velocity caused 7 mm of medial opening, which correlates to the initiation of MCL sprain, and a "rupture" impact velocity caused 15 mm of medial joint opening, which correlates to the rupture of the MCL. They concluded that a brace could be effective if it were able to disperse the force of the lateral blow to the thigh and calf. The most effective braces in their study were stiffer and less likely to strike the knee joint during a lateral blow. Moreover, they stated that PKB's were able to provide the most protection when the outside force was a large mass at a slow velocity against an extended knee. Along these lines, Paulos et al.[13] using a surrogate limb, were able to show that a PKB would protect both the MCL and the ACL against a lateral blow. They noted improved protection when the hinge of the brace did not come into contact with the knee (i.e. stiffer braces displace force to thigh and tibia). Albright et al.[14] reviewed several surrogate limb studies and concluded that PKB's most likely reduce MCL strain in a fully extended surrogate knee about 20–30%. They go on further to suggest that the greatest amount of protection may be produced by a custom fitted dual-upright brace (much like the Bregg X2K used in this study).

Over time, several rule changes in football, such as banning the "crackback block," have been implemented to protect the players from unnecessary injuries to the knee.[14] Moreover, the use of arthroscopic surgery has increased dramatically over the past twenty years. Knee injuries, which once posed an end to one's career, can now be managed with minimally invasive and highly effective treatments.

The question to brace or not to brace also has financial consequences. The Bregg X2K costs approximately $300 per brace, thus costing $600 per lineman. This does not fac-

tor in the time needed to outfit the brace, train the players on how to wear them properly, and cost of maintenance. For a team with twenty practicing linemen, the cost is approximately $12,000 per season. This can be a significant financial burden on athletic programs with smaller budgets.

The medical legal aspect must also be considered. With significant evidence-based studies showing the efficacy of bracing, would a program be liable for negligence if they failed to outfit their entire team or at least position players with a brace that represents the "standard of care"?

With this in mind it is imperative that a well run and properly designed study be performed. The Temple University football team was ideal for this purpose. Over the six-year period, it was shown that the number of knee injuries leading to loss of playing time dropped from twelve to four in players who played in lineman positions. Since it was noted that the majority of injuries occurred during practice, limiting brace use to practice demonstrated a consistent decrease in injury incidence and severity and was not associated with a reciprocal increase in game time injury.

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Original Research

The Subscapularis Footprint: An Anatomical Study of the Subscapularis Tendon Insertion

Leonard D'Addesi, MD, Ammar Anbari, MD, Matthew W. Reish, MD, John D. Kelly, IV, MD

Abstract

Background: Arthroscopic repair of the subscapularis tendon has become more prevalent in recent years. Incomplete tears of the subscapularis tendon insertion have not been described well in the literature. Tears of the subscapularis insertion can be measured arthroscopically if the size of the average subscapularis tendon insertion is known. This anatomical study was performed to measure the dimensions and describe the anatomy of the subscapularis footprint.

Methods: Six male and six female shoulders were dissected down to the insertion of the subscapularis tendon. The insertion was demarcated, the tendon was detached, and the dimensions of the insertion site were measured.

Results: The footprint is the insertion of the subscapularis tendon onto the lesser tubercle. The shape of the footprint was characterized as that resembling a human ear. The insertion is broad proximal and tapered distally, and has a straight medial border that is almost parallel to the longitudinal axis of the humerus. The total average height of the footprint was 25.8 mm (+/–3.2 mm). The total average width was 18.1 mm (+/–1.6 mm). The average male height was 26.7 mm (range 22 to 32) and width 18.3 mm (range 16 to 21). The average female height was 24.8 mm (range 22 to 29) and width 17.8 mm (range 15 to 19). Although the male footprint was slightly larger than the female, these were not statistically different (p=0.18 and 0.31 for height and width, respectively).

Conclusions: An anatomic study was performed to determine the size of the footprint of the subscapularis tendon. We found the average height of the footprint to be 25.8 mm and average width to be 18.1 mm.

Introduction

Tears of the subscapularis tendon are becoming increasingly recognized as surgeons gain proficiency in arthroscopic shoulder surgery. Traditionally, subscapularis tears have been repaired using open techniques,[1–6] but more recently, there has been much interest in arthroscopic visualization techniques and repair.[3, 7–15]

Subscapularis tears have been classified as partial-thickness/partial-length, full-thickness/partial-length, full-thickness/complete-length with no retraction, and full-thickness/complete-length with retraction.[8, 11] The optimal critical tear size requiring surgical repair has yet to be determined.[15] Comparative studies of different sized tears are still missing.[6] Full-thickness and partial-thickness tears of isolated and combined subscapularis tears have been reported.[1–6, 8, 10–12, 15] The size of the tear, based on the cephalocaudal length of the insertion of the subscapularis tendon has not been well described. Some reports of open repairs have quantified the size of the tear by thirds,[3, 5, 6] however, arthroscopic measurements of the size of the tear are lacking.

The size of the subscapularis tear can be calculated during arthroscopy using a mathematical model,[9, 13] which is based on the known size of the footprint. There is a limited description in the literature regarding the average size of the footprint. Tierney et al.[16] (abstract publication) measured the average height of the subscapularis insertion to be 40.35 mm. Burkhart and Tehrany[9] (unpublished data) reported an average size of 2.5 cm. Pearsall et al.[17] found the overall height of the tendinous insertion to be 31.3 mm.

We performed this anatomical study to better describe the detailed anatomy of the subscapularis footprint. This will aid in calculating the percentage of the subscapularis that is torn during arthroscopy. The goal is to characterize the anatomy of the tendon insertion, its dimensions and shape, so that the surgeon can achieve a more anatomic repair.

Materials and Methods

We examined the subscapularis footprint in 14 embalmed cadaveric shoulders. Two were discarded because they had a subscapularis tear. Of the remaining twelve, there were six male and six female shoulders. The average age of the male shoulders was 76 years (range 63 to 83), and the female shoulder 84 years (range 74 to 92). All of the female shoulders were left-sided. There were three left male shoulders and three right male shoulders. The shoulders were dissected grossly down to the conjoint tendon excising the elbow flexor musculature and anterior deltoid. The conjoint tendon was detached from the coracoid and the subscapularis muscle was identified. The biceps tendon lying in the bicipital groove was identified as a guide to the lateral border of the lesser tubercle. The perimeter of the tendinous insertion of the subscapularis tendon was determined on the lesser tubercle and demarcated. The subscapularis tendon was then detached sharply from the lesser tubercle so that the footprint could be measured. Measurements consisted of the cephalo-caudal (height) distance of the tendinous portion of the insertion, and the maximum medio-lateral (width) distance of the footprint. This maximum width corresponded to an area in the proximal two-thirds of the footprint.

Results

The total average height of the footprint was 25.8 mm. The total average width was 18.1 mm. The average male height was 26.7 mm (range 22 to 32) and width 18.3 mm (range 16 to 21). The average female height was 24.8 mm (range 22 to 29) and width 17.8 mm (range 15 to 19). Although the male footprint was slightly larger than the female, these were not statistically different (p=0.18 and 0.31 for height and width, respectively). The shape of the footprint was inspected as well (Figure 1). We found the medial border to be a straight edge and aligned almost parallel to the longitudinal axis of the humerus. The overall shape resembled that of a human ear. The footprint was broad proximally and tapered distally.

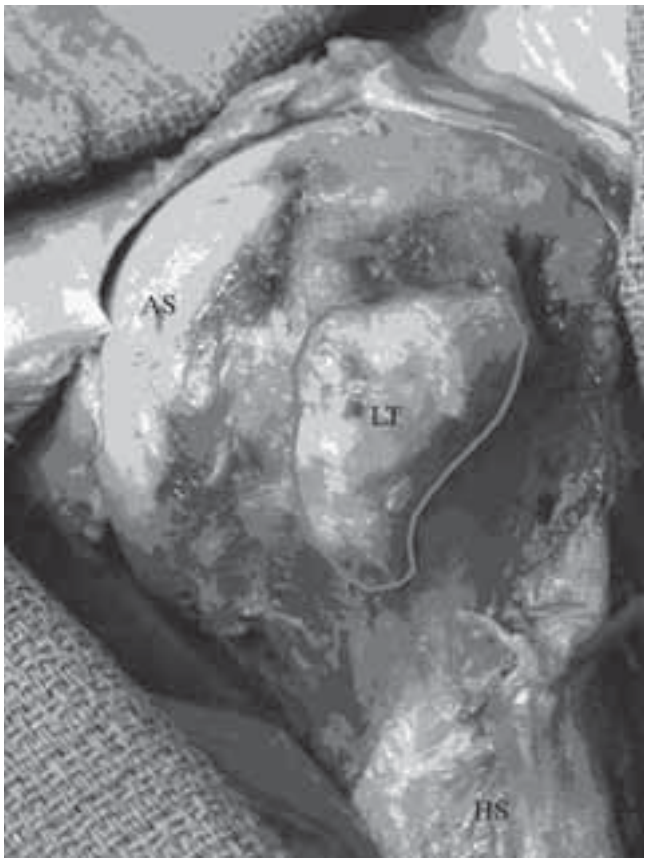

Figure 1: The lesser tubercle of the proximal humerus is the site of insertion of the subscapularis tendon. The footprint was defined as the grossly tendinous insertion of the subscapularis tendon (red line). The arrow denotes the bicipital groove, which abuts the lateral border of the lesser tubercle. The insertion was broad proximally and tapered distally. The shape resembles that of a human ear. Measurements were taken of this tendinous portion of the insertion, which averaged 25.8 mm in height and 18.1 mm at the maximum width. AS, articular surface of the humeral head; LT, lesser tubercle; HS, humeral shaft.

Discussion

The subscapularis tendon inserts onto the lesser tubercle. The subscapularis footprint is the description of the tendon insertion into the lesser tubercle. The proximal two-thirds of the insertion is tendinous and the inferior one-third is more muscular.[18]

The subscapularis can be injured in falls on an outstretched arm, with forceful hyperextension or external rotation with the arm adducted, during an anterior dislocation of the shoulder, and from coracoid impingement.[1, 19–21] Repairs have been performed using open[1–6] and arthroscopic techniques.[3, 9–12, 15] Isolated, complete tears of the subscapularis insertion have been studied.[1–3] Partial-thickness, incomplete tears are being increasingly recognized arthroscopically, since partial tears are articular-sided and may not be visualized with traditional open techniques.[9, 15] The prevalence of partial-thickness tears has been found to be between 13–19%.[8, 12]

The literature is scarce with information on partial tears. Several reports describe the length of the tear by separating the height of the tendon into thirds, but these have been mainly open techniques.[3, 5, 6] Deutsch et al.[3] arthroscopically debrided one patient with a partial-thickness tear, and Kim et al.[15] arthroscopically repaired isolated articular-sided partial-thickness tears of the subscapularis that were at least 5 mm in width, whereas smaller lesions were debrided. It is unclear what percentage of the length of the tendon insertion these tears represented. Other arthroscopic studies classify the tear as partial-thickness, full-thickness or complete,[5, 8, 10–12] without reporting the size of the tear. The description of an incomplete tear is meaningful when one knows how much of the overall height of the tendon insertion the tear represents. There may be an optimal critical tear size that requires surgery,[15] but this has not been determined since studies on different sized tears are lacking.[6]

A mathematical model can be used to calculate the size of the tear during arthroscopy. The bare area of the footprint, as would be seen in full-thickness tears, is measured and divided by the known average height of the footprint.[9, 13] Quantifying the percentage of the subscapularis tear can only be achieved by knowing the overall height dimension of the footprint. Our goal was to measure the total height of the subscapularis footprint (insertion of the tendinous portion of the subscapularis) to aid the surgeon in calculating the size of the tear during arthroscopy. The average height of the tendinous insertion of the subscapularis in this study was 25.8 mm. Thus, 13 mm of exposed footprint will roughly correlate with a 50% tear of the subscapularis tendon.

Studies describing the height of the subscapularis footprint are wanting. Tierney et al.[16] (abstract publication) measured 20 fresh frozen cadaver shoulders and found the average length of the footprint to be 40.35 mm (range 35–55) and width 19.58 mm (range 15–25). The authors do not

report whether these measurements are of the entire insertion including the muscular portion, or only the tendinous portion. Comparison is difficult since this report lacks detail. Burkhart and Tehrany[9] briefly reported unpublished data on the height of the footprint. Nineteen cadaver shoulders were used. The superior to inferior length of the subscapularis footprint was found to be 2.5 cm (range 1.5–3). The authors did not give a description of what was measured, so again, comparison is difficult. Pearsall et al.[17] measured 10 cadaver shoulders and found the total cephalad-caudad height of the tendinous insertion to average 31.3 mm (+/–9.6 mm). Assessing the causes as to the difference in average values is difficult because Pearsall's study focused on the dimensions of the intra-articular subscapularis tendon. It is unclear why this is a greater than 5 mm difference in average height.

There were several limitations in our study. First, our cadaver population was older than the average patient with rotator cuff injuries. We do not believe this impacted our results, since all the study shoulders had no subscapularis tears, and their respective footprints were intact. Secondly, our sample size was small, but was within the range of specimens used in other studies.[9, 16, 17]

Future research should be aimed at expanding the number of specimens measured while simultaneously performing a biomechanical analysis of different sized subscapularis tears to determine if there exists a critical size tear necessary for repair. Physical exam, MRI and arthroscopic findings should also be correlated with patient outcomes on repair of different sized tears.

We performed an anatomic study to determine the dimensions of the footprint of the subscapularis tendon. We found the average height of the footprint to be 25.8 mm and average width to be 18.1 mm. The shape of the footprint was characterized as that resembling a human ear. The medial border of the tendinous insertion is almost parallel to the longitudinal axis of the humerus. The insertion is broad proximal and tapered distally.

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Original Research

Elastic Instability, Columnar Buckling, and Non-Contact Anterior Cruciate Ligament Ruptures: A Preliminary Report

VICTOR HSU, MD, DAVID STEARNE, PHD, JOSEPH TORG, MD

Introduction

Classically, we have learned mechanical constructs usually fail in one of five ways: tension, compression, shear, torsion, and fatigue or cyclic failure.[1] The objective of this paper is to introduce elastic instability, another mechanism of failure rarely recognized in orthopaedic literature. Burstein first acknowledged this mechanism in 1970 when he described dislocations of the PIP joint in basketball players.[2] The basketball struck the outstretched tip of the long finger loading the digit and causing a buckling of the finger and dorsal dislocation (Fig. 1).

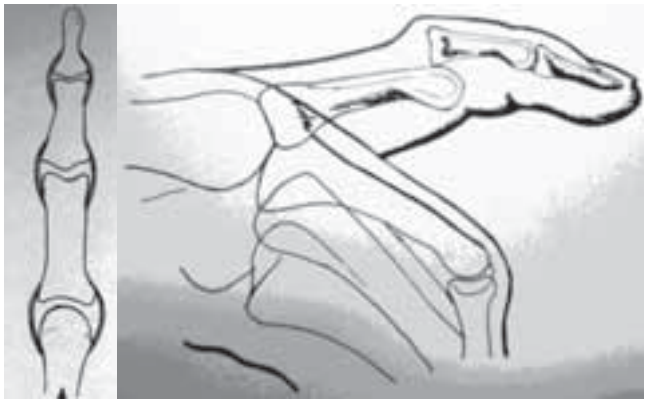

Figure 1. Diagramatic representation of a PIP dislocation due to an axial energy input which occurred when the end of the digit was struck with a basketball. (Orthopaedic Biomechanics: The Application of Engineering in Musculoskeletal System. Frankel V.H. and Burstein A.H. Lea and Febiger, Philadelphia, 1970.)

Elastic instability is a basic tenet in biomechanics and engineering. Classically it is illustrated as slender column buckling under an axial load resulting in a lateral deformation without fracture. An example is when a straw is placed between two fingers and gentle pressure applied on either side resulting in a lateral bowing of the straw (Fig. 2).

Figure 2. Axial loading of a column, in this case a drinking straw, resulting in lateral deformation without failure.

Our colleagues in engineering have long recognized the importance of understanding Elastic Instability in structural supports. Neathery explained its importance by stating: "The buckling of structures is not something we observe everyday — fortunately. The reason we rarely see buckling failure is that when they do occur they are usually catastrophic. Catastrophic failures are unpleasant and limit one's opportunity to exercise his design talent. We, therefore, expend considerable effort assuring that buckling does not occur."[1] Columns can be divided into two separate categories. Unsegmented columns are those made of one solid material and are seen in the facades of buildings. Analogous structures in the body include long tubular bones such as the femur or radius. Segmented columns are those made up of smaller pieces stacked on each other forming a tubular support. This type of configuration is seen in the cervical spine. Both columns exhibit similar properties under axial loads and obey the same laws governing columns (Fig. 3).

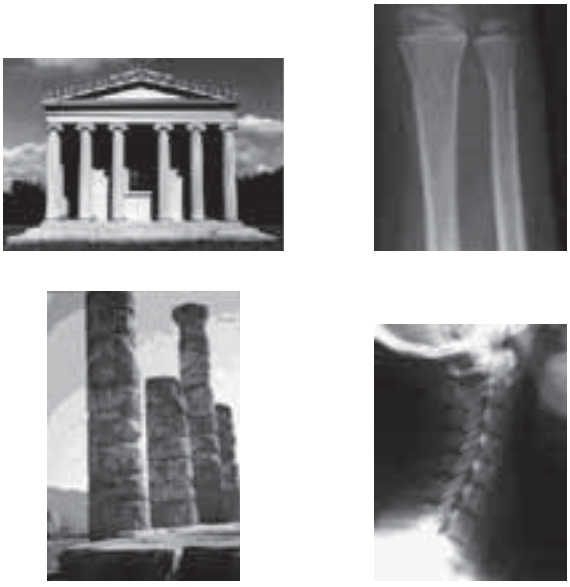

Figure 3. Unsegmented versus segmented columns.

Under smaller axial forces, a column remains straight and is able to support the load and resist small lateral deflections. As load increases, the ability to resist lateral displacement decreases until a force is reached where the axial load is so large that the structure can no longer resist any lateral deflection and deforms laterally. The force at which this occurs is

called the critical buckling load. The critical buckling load lies on the elastic portion of the stress-strain curve and while the force is enough to distort the original shape, it is not enough to disrupt the internal structure leading to fracture of the material. Using the straw analogy, as the thumb and index finger apply more pressure the straw begins to deform, but if the force is released, the straw regains its initial shape.

In the 18th century, the Swiss mathematician Leonard Euler (1707–1783) derived a formula to calculate the buckling load. The force is directly proportional to the modulus of elasticity and moment of inertia, which is the distribution of mass around the center of the structure and inversely related to the square of the effective length of the column. Thus, we see that columns that are less stiff, have a distribution of mass near the center, and are longer have lower critical buckling loads.

$$\mathbf{Pcr = \pi^2 EI/L^2}$$

Pcr = Critical Buckling Load

E = Modulus of Elasticity

I = Moment of Least Inertia

L = Buckling Length

The bending moment experienced by a column is determined by multiplying the load by the lever arm, which is the lateral displacement of the column (Fig. 4). This is usually catastrophic because as the critical buckling load is achieved, the force is not usually released but continues to be applied leading to larger deforming moments, ultimately resulting in structural failure. Once again using the straw analogy, the straw begins straight and bends when the critical buckling load is achieved; at this point if the force is relieved, the straw should revert back to its original shape. However, as the force is maintained, the straw continues to deform until finally it fails (Fig. 5).

Figure 4. The bending moment and lateral displacement. The bending moment at any point (X) is the product of lateral displacement (Y) and the load. As the lateral displacement increases, the propensity for further buckling increases.

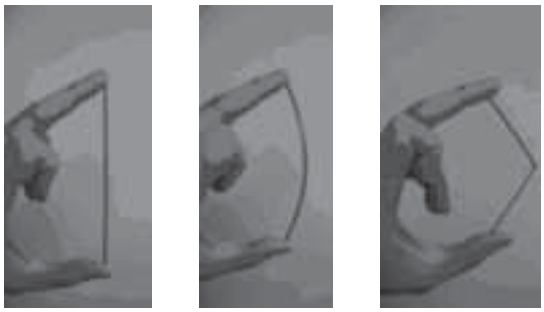

Figure 5. Critical buckling load.

Columnar buckling has been cited in the past as previously mentioned by Burstein when describing PIP dislocations. As Torg demonstrated, when the spine is flexed 30 or so degrees, the cervical spine forms a segmented column and football players who tackle that way are at risk for buckling of the vertebrae resulting in catastrophic fracture or dislocations.[3] We believe non-contact anterior cruciate ligament ruptures are also caused by elastic instability.

When the knee is in the extended position, the relatively flat distal femur and proximal tibia form a two-piece segmented column. The same can be assumed even when the knee is in a low degree of flexion as the knee is still in the glide phase of knee kinematics. When this configuration is maintained and minimal angular displacement occurs, the muscles about the knee are unable to absorb energy. As an axial force is transmitted through the bony segmented column, the critical buckling load is achieved which causes angular shear force at the junction of the femur and tibia. Because of posterior tibial slope, the buckling accentuates anterior translation of the tibia resulting in rupture of the anterior cruciate ligament. With knee flexion the muscles in the anterior and posterior compartments are able to absorb energy, protecting the bony and ligamentous structures and decreasing the force seen at the joint. The purpose of this study is to bring attention to the relationship between elastic instability and orthopaedic injuries, specifically non-contact anterior cruciate ligament injuries and how the position of the knee is crucial in regards to energy absorption.

Materials and Methods

Our pilot study consisted of two phases. The first consisted of evaluating seven videos showing real time footage of non-contact anterior cruciate ligament injuries in soccer and basketball participants. Mechanisms in all seven episodes revealed a knee in the "at risk" position. We defined the "at risk" (extended) position to be a knee in extension or near extension at heel strike resulting in a valgus stress.

The second limb of our study was a force plate analysis where subjects were asked to perform drop-landings from two different heights with their knees flexed and then with their knees extended to re-create the "at risk" position. The preliminary results represent a sample population of two

subjects jumping from a height of 20 and 45 cm. Ground reaction forces were measured in Newtons (N) by the force plate analysis. The rate of loading was also measured by dividing the force (N) by duration of jump (sec).

Results

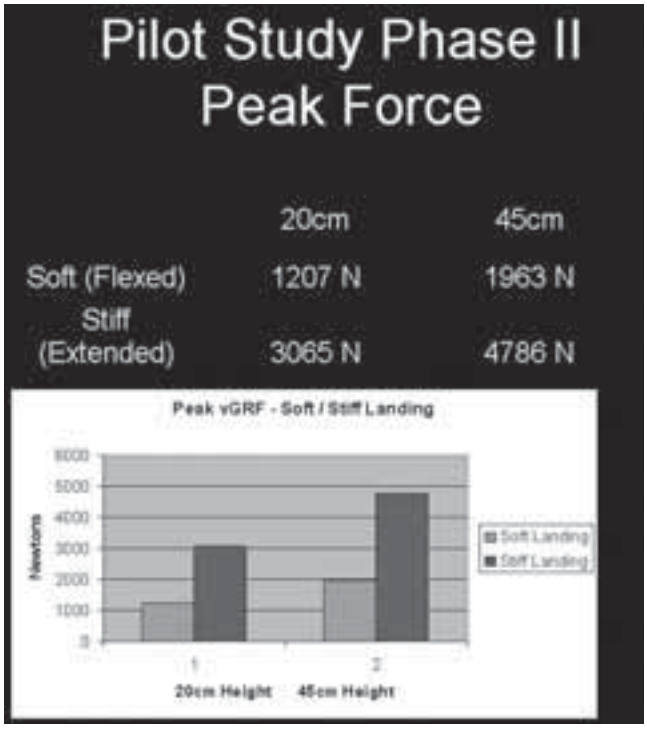

Figure 6. Peak forces.

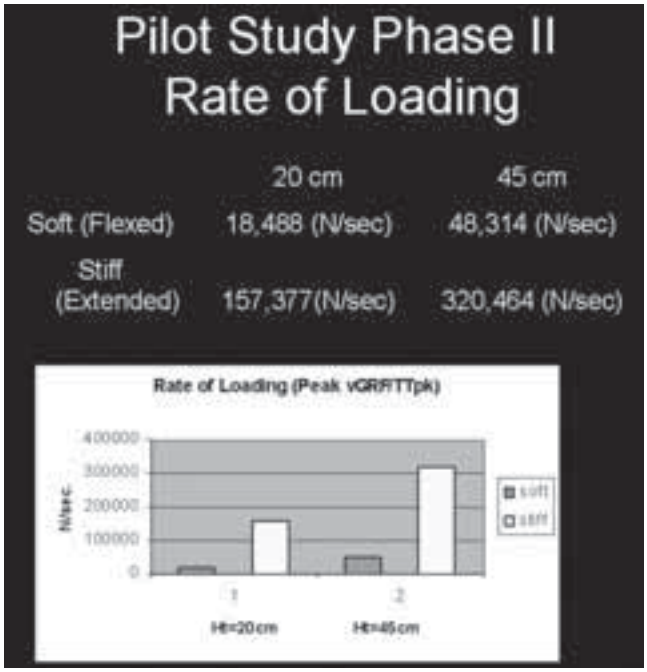

Figure 7. Rate of loading.

Discussion

The ground reactive forces for the extended "at risk" position was almost 2.5 times greater than the flexed knee position from both heights (Fig. 6). In addition, the rate of loading was about eight times greater in the "at risk" position (Fig. 7).

The clinical relevance of these observations is recognizing the role of axial load, elastic instability, and columnar buckling as a mechanism of ACL injuries and other orthopaedic injuries. Stiff landings with the knee "at risk" occur when the knee is in a relatively extended position and result in almost 2.5 times more energy absorption by the bony column. The information points to a dramatic increase in energy transmitted through the extended bony column and clinically it is reasonable that a large step in preventing ACL injuries is dependant on effective neuromuscular and proprioceptive training in strategies of landing, cutting, and deceleration maneuvers. Furthermore, this lends biomechanical evidence to the observed phenomenon that instruction in "soft landing" techniques result in a diminution of non-contact anterior cruciate injuries.

This was a preliminary pilot study with a small sample size, and its findings must be interpreted in light of this. But, we feel that there is consequence to this and intend to build upon this pilot.

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Original Research

Low Anterior-Inferior 5 O'Clock Portal for Shoulder Arthroscopy

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Abstract

Background: Current arthroscopic techniques for repair of avulsions of the anteroinferior glenoid labrum (Bankhart lesions) involve anterior working portals that produce an acute angle of approach to the area of injury causing difficulty in tissue fixation. An anterior-inferior portal, from the 5 o'clock position, yields a perpendicular approach to the glenoid at the critical area of capsulolabral detachment in a Bankart lesion, facilitating direct insertion of fixation devices. Secondly, in order to restore an effective labral concavity, suture anchors must be placed from a lateral direction to avoid skiving off the articular surface. The goal of the study was to assess the safety and describe the technique of creating a 5 o'clock portal using an outside-in technique while reproducing clinical conditions as closely as possible.

Results: Ten cadaveric shoulders were positioned in simulated lateral decubitus position (five with humerii maximally adducted and five in 30 degrees abduction) and an arthroscope was introduced through a standard posterior portal. Then, 3 and 5 o'clock anterior portals were created using an outside-in technique through which Steinman pins were inserted into the edge of the glenoid. When inserting the Steinman pins, the investigator brought his hands laterally, placing both at approximately a 40 degree angle off the face of the glenoid. Anterior dissection was then performed. Angle between pins was recorded (mean 17 degrees) and 5 o'clock pin's relationship to the conjoined tendon was recorded. Distances were measured from the 5 o'clock pin to the musculocutaneous nerve (9.5–36 mm, mean 20.48 mm), axillary nerve (15.5–50 mm, mean 34.57 mm) and cephalic vein (0–29 mm, mean 12.574 mm) in several different arm positions. Statistically significant findings included the distances from cephalic vein, musculocutaneous, and axillary nerves at 0 and 30 degrees of abduction were greater than the distance at 70 degrees of abduction. Also of statistical significance, the distance from the cephalic vein to the 5 o'clock pin was greater when arm was placed in 0 degrees abduction at the time of portal placement than when the arm was in 30 degrees abduction.

Conclusion: The 5 o'clock anteroinferior portal can be established in cadaver shoulders without significant risk to major nerves if the arm is placed in relative adduction at the time of portal placement. Rotation, flexion, and extension had no significant effect on the distance from the portal site to the nerves. The cephalic vein, however, appeared in jeopardy during portal placement in abduction with vein violation seen in one specimen. Also all specimens in which the portal was created in 30 degrees abduction had transgression of the conjoined tendon. In light of these findings, 5 o'clock portal creation in the lateral decubitus position must be exercised with caution.

Introduction

Surgical treatment of glenohumeral instability often involves repair of the anteroinferior glenoid labrum and glenohumeral ligament. If the glenoid fossa is described as the face of a clock, coordinates for orientation and location of pathologic lesions are defined. The classic Bankart lesion involves an avulsion of the anteroinferior capsulolabral complex from the glenoid and, in a right shoulder, extends from 3 to 6 o'clock along the anteroinferior glenoid rim (Figure 1).

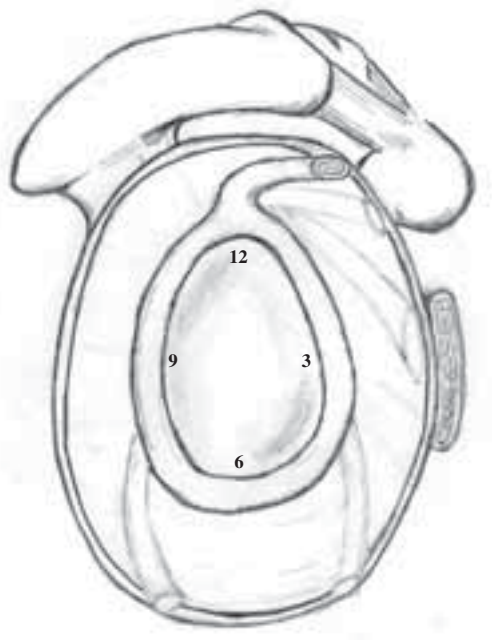

Figure 1. When the face of the glenoid of a right shoulder is given coordinates of a clock face, the Bankart lesion extends from 3 to 6 o'clock.

Unfortunately, the current arthroscopic repair techniques employ anterior working portals that are somewhat remote from the area of interest, creating often difficult access to the anteroinferior labrum. Matthews et al. illustrated an anterior portal in the triangle defined by the biceps tendon, glenoid rim, and humeral head at about 2 o'clock position. Wolf defined an anterior-inferior portal located at the superior border of the subscapularis tendon at the 3 o'clock position.[7] Utilization of these portals results in an acute angle of approach to inferior labral areas of injury, causing difficulty in tissue access (Figure 2).

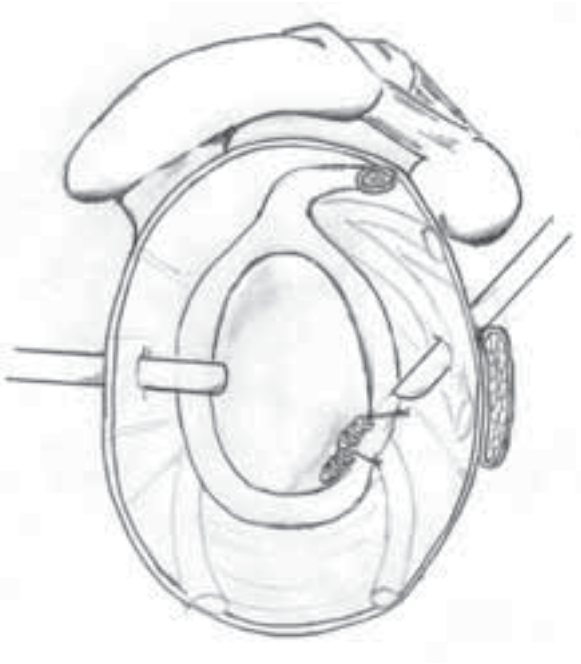

Figure 2. The acute angle of approach to the Bankart lesion from the standard 3 o'clock portal can lead to suboptimal fixation.

This angle of approach also causes intraosseous fixation devices to be inserted at a tangential angle to the glenoid rim resulting in suboptimal fixation of the capsulolabral complex[1] and, conceivably, increased failure rates.[2] An anterior-inferior portal, from the 5 o'clock position, approaches the glenoid rim at a right angle to the critical area of capsulolabral detachment in a Bankart lesion, facilitating direct insertion of fixation devices (Figure 3).

Previous investigators[1, 6] have described the relationship of this low portal to neighboring neurovascular structures. However, these studies were done either in the "beach chair" position (Pearsall et al.)[6] or employed an "inside out" method of portal creation (Davidson et al.).[1] The goal of the study was to assess the safety and describe the technique of creating a 5 o'clock portal using an outside-in technique in the lateral decubitus position, which will allow direct perpendicular access to the anteroinferior glenoid for Bankart lesion repair.

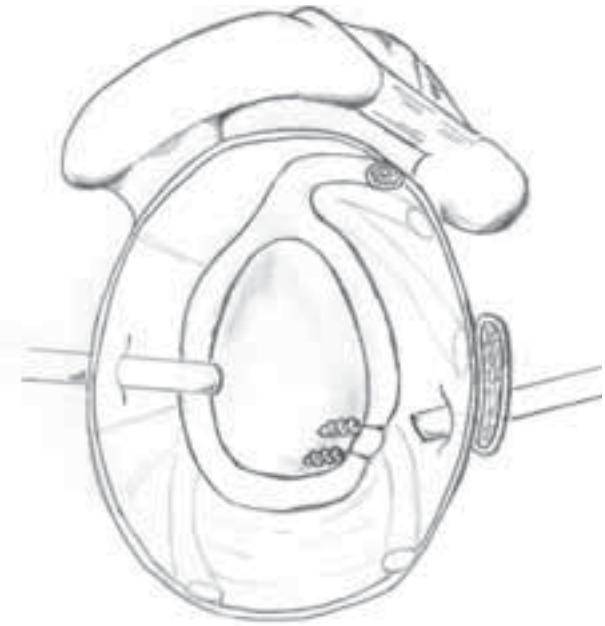

Figure 3. Approaching the Bankart lesion through a 5 o'clock portal offers a more direct, almost perpendicular approach to the lesion.

Materials and Methods

Five matched pairs of fresh frozen shoulders were used in this study. A cadaveric shoulder holder was designed in which the scapula was mounted and positioned to simulate lateral decubitus position. The humerus was not constrained and afforded free range of motion. A Steinman pin was placed into the humeral shaft and attached to a scapula mounted external fixator in order to control humeral abduction.

The arthroscope was introduced through a standard posterior portal, 2 cm inferior and 1 cm medial to the posterolateral aspect of the acromion. An 18 gauge spinal needle was used to localize the 3 o'clock portal just proximal to the subscapularis in an outside-in fashion. Once location of the spinal needle on the glenoid was confirmed by the arthroscope, a Steinman pin was inserted into the edge of the articular surface and left in place. Then, the 5 o'clock portal site was located using an 18 gauge spinal needle inserted in an outside-in fashion through the subscapularis tendon. A Steinman pin was then drilled into the glenoid at the edge of the articular surface and left in place (Figure 4).

In creating both 3 and 5 o'clock portals, the investigator brought his hands laterally when inserting Steinman pins, placing both at approximately a 35 degree angle off the face of the glenoid. Both pins were placed perpendicular to the edge of the articular surface. Half of the specimens had the Steinman pins placed with the humerus maximally adducted; the other specimens were placed with the arm in 30 degrees abduction. All humerii were positioned in 0 degrees forward flexion at the time of pin placement.

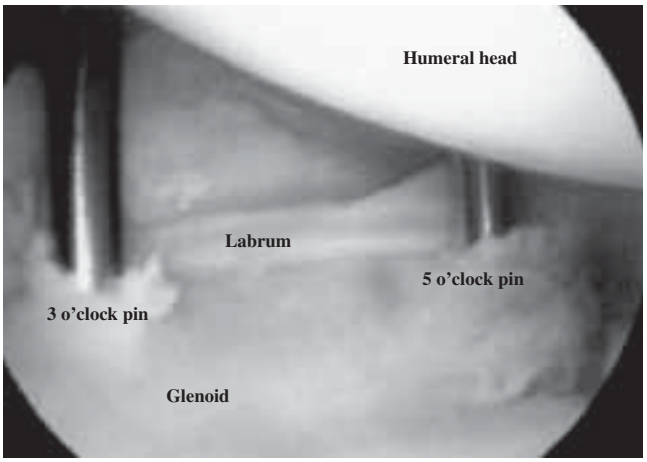

Figure 4. Steinman pins were inserted into the glenoid through 3 and 5 o'clock portals.

After both pins were placed, the arthroscope and cannula were removed from the joint, and anterior dissection was undertaken. Skin and subcutaneous tissues were incised and retracted out of the way. First, the angle between the pins was measured (Figure 5). Next, the distance was measured from the 5 o'clock pin to the cephalic vein (Figure 6). The deltopectoral interval was utilized to expose the deeper structures and both pin's relationship to the conjoined tendon was recorded. The distance from the 5 o'clock pin to the superior edge of the subscapularis tendon was also determined.

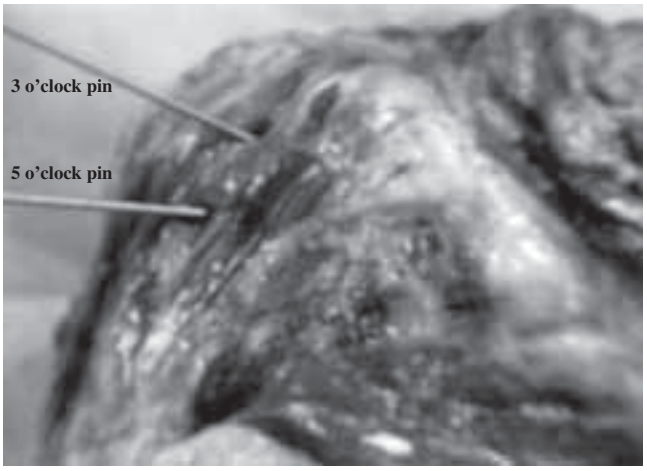

Figure 5. Superficial dissection of cadaveric shoulder specimens. At this point in the dissection, the angle between the pins marking 3 and 5 o'clock portals was measured.

The relationship of the musculocutaneous and axillary nerves to the 5 o'clock pin were measured at several different arm positions (Figure 7). First, the distances were measured at 0, 30, and 70 degrees abduction. The humerus was then placed in 30 degrees abduction and measurements were taken with maximum internal rotation, maximum external rotation, 30 degrees flexion, and 30 degrees extension. The humerus was returned to 0 degrees abduction and measurements were again taken with maximum internal rotation, maximum external rotation, 30 degrees flexion, and 30 degrees extension. Two independent observers recorded each measurement.

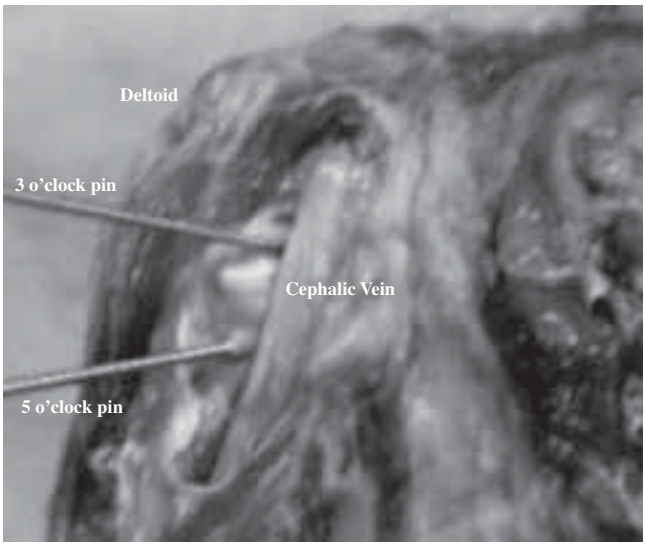

Figure 6. Distance was measured from both the 3 and 5 o'clock pins to the edge of the cephalic vein.

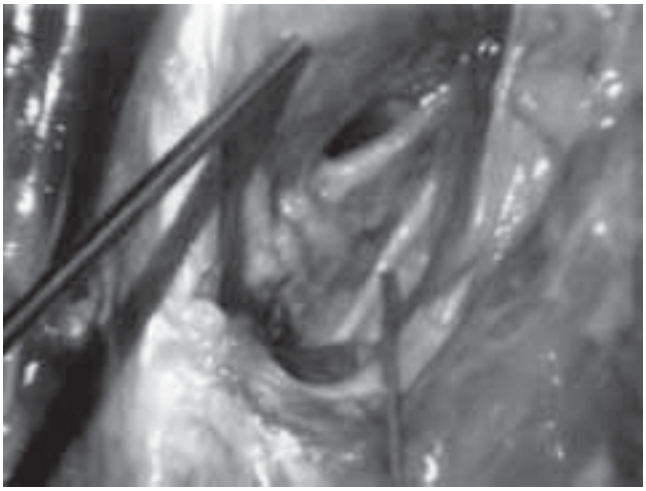

Figure 7.

Results

The angle between the pins ranged between 10–22 degrees with a mean of 17 degrees. Arm position (adduction vs 30 degrees abduction) during pin insertion had no effect on this angle.

With the humerus at 0 degrees abduction at the time of pin placement, all 5 o'clock pins were found to be lateral to the deltopectoral interval. With the arm in 30 degrees abduction

during portal placement, the relationship was less consistent with 3 lateral, 1 medial, and 1 through the interval. With the humerus abducted to 30 degrees during placement, all 5 pins pierced the conjoined tendon, with one pin resting 16 mm medial to the lateral tendon border. The distance from the 5 o'clock Steinman pin to the superior border of the subscapularis tendon ranged from 7–18 mm and averaged 13.6 mm.

The distances to the musculocutaneous and axillary nerves were measured in all positions. Statistical analysis included an ANOVA with repeated measures. Posthoc analysis consisted of dependent t-tests with Bonferroni correction with an Alpha level of 0.05. No statistical significance was shown for flexion, extension, maximum internal rotation, or maximum external rotation at either 0 or 30 degrees abduction. There was a statistically significant difference for both arm position at time of pin insertion (0 versus 30 degrees abduction) and for degree of abduction during measurements (0, 30, or 70 degrees).

The range of distances from the musculocutaneous nerve was 9.5–36 mm, with a cumulative mean value combining all positions was 20.48 ± 7.91 mm. Of statistical significance, the distance from musculocutaneous nerve at both 0 and 30 degrees of abduction was greater than the distance at 70 degrees of abduction. However, there was no statistical significance when the arm was placed in maximum internal or external rotation, flexion, extension or in arm position at pin insertion (Table 1).

The range of distances from the axillary nerve to 5 o'clock pin was 15.5–50 mm, with a cumulative mean value combining all positions was 34.574 ± 9.345 mm. Of statistical significance, the distance from axillary nerve at both 0 and 30 degrees of abduction was greater than that at 70 degrees of abduction. Again, there was no statistical significance when the arm was placed in maximum internal or external rotation, flexion, and extension or in arm position at pin insertion (Table 2).

The distances to the cephalic vein (CV) were measured in all positions. Statistical analysis included an ANOVA with repeated measures. Posthoc analysis consisted of dependent t-tests with Bonferroni correction with an Alpha level of 0.05 (Table 3). The 5 o'clock pin was lateral to the CV in all specimens except one, where the CV was draped laterally over the pin, resting on the pin (inserted with arm positioned at 30 degrees abduction). The distance from the pin ranged from 0–29 mm. The cumulative mean value combining all positions was 12.574 ± 9.6 mm. Statistically significant findings included the distance from the CV at 0 and 30 degrees abduction was greater than the distance at 70 degrees of abduction. Also of statistical significance, the distance from CV to the 5 o'clock pin was greater when arm was placed in 0 degrees abduction at the time of pin placement than when the arm was in 30 degrees abduction. However, there was no statistical significance when the arm was placed maximum internal or external rotation, flexion or extension.

Table 1. Distance from the 5 O'clock Pin to the Musculocutaneous Nerve in Multiple Arm Postitions (ab = Abduction, MI = Maximal Internal Rotation, ME = Maximal External Rotation)

Humeral Abduction at Time of Pin Insertion (Below)	0.0	30 ab	70 ab	0 ab, MI	30 ab, MI	0 ab, ME	30 ab, ME	0 ab, 30 flex	30 ab, 30 flex	0 ab, 30 ext	30 ab, 30 ext
Average 0	21.0	20.0	16.6	20.0	20.9	22.1	20.6	21.1	21.3	19.8	19.0
Std Dev 0	8.59	9.68	6.61	9.33	8.98	10.04	9.13	10.49	11.86	9.43	6.70
Average 30	23.6	22.5	18.6	20.8	23.4	24.3	24.0	24.8	22.9	22.5	19.9
Std Dev 30	7.21	8.86	8.81	7.78	9.17	7.79	9.17	7.54	7.00	9.22	9.95
Average all	22.4	21.4	17.7	20.4	22.3	23.3	22.5	23.2	22.2	21.3	19.5
Std Dev all	7.45	8.72	7.50	7.94	8.61	8.33	8.75	8.57	8.84	8.83	8.16

Table 2. Distance from the 5 O'clock Pin to the Axillary Nerve in Multiple Arm Postitions

Humeral Abduction at Time of Pin Insertion (Below)	0.0	30 ab	70 ab	0 ab, MI	30 ab, MI	0 ab, ME	30 ab, ME	0 ab, 30 flex	30 ab, 30 flex	0 ab, 30 ext	30 ab, 30 ext
Average 0	33.3	31.6	30.0	33.1	30.4	34.0	32.5	32.5	30.8	32.0	31.1
Std Dev 0	14.54	13.75	13.27	16.00	13.75	13.57	12.77	13.58	13.60	15.94	14.60
Average 30	40.9	37.4	32.5	40.5	38.5	42.7	36.9	40.2	34.4	42.1	38.5
Std Dev 30	4.17	3.80	2.85	4.64	3.59	3.11	2.38	3.21	3.34	4.04	4.08
Average all	37.5	34.8	31.4	37.2	34.9	38.8	34.9	36.8	32.8	37.6	35.2
Std Dev all	10.21	9.35	8.48	11.04	9.78	9.74	8.33	9.53	8.87	11.48	10.17

Table 3. Distance from the 5 O'clock Pin to the Cephalic Nerve in Multiple Arm Postitions

Humeral Abduction at Time of Pin Insertion (Below)	0.0	30 ab	70 ab	0 ab, MI	30 ab, MI	0 ab, ME	30 ab, ME	0 ab, 30 flex	30 ab, 30 flex	0 ab, 30 ext	30 ab, 30 ext
Average 0	22.9	22.3	20.3	22.0	22.1	21.4	20.6	22.8	22.3	21.0	19.8
Std Dev 0	2.87	2.53	3.66	3.14	2.95	2.95	4.33	4.73	6.06	0.71	0.96
Average 30	6.7	6.0	2.9	6.3	4.6	6.2	5.1	5.6	5.5	6.5	5.6
Std Dev 30	6.16	5.24	2.70	5.70	3.96	4.86	3.73	6.07	5.39	4.83	4.63
Average all	13.9	13.2	10.6	13.3	12.4	12.9	12.0	13.2	12.9	12.9	11.9
Std Dev all	9.73	9.46	9.61	9.40	9.82	8.89	9.00	10.42	10.31	8.38	8.17

Discussion

Davidson and Tibone described the 5 o'clock portal for shoulder instrumentation using a cadaver model placed in the lateral decubitus position. They reported that this portal could be safely created; however, portal creation was performed in maximal humeral adduction and in an "inside out" fashion employing a Wissinger rod.[1] Pearsall et al defined the safety of a 5 o'clock portal created in "outside in" fashion and stated that the cephalic vein was endangered using this approach. However, these investigators employed the modified "beach chair" position with humeral adduction during creation of their portals. Furthermore, portal placement was tangential to the glenoid surface.[6]

In this study, we sought to investigate the safety of this portal creation while reproducing clinical conditions as closely as possible. The lateral decubitus position was chosen because it is the senior author's (JDK) contention that this is the favored position for most shoulder arthroscopists for instability surgery. Secondly, it is becoming increasingly evident that suture anchors should be placed on the articular surface, 1–2 mm from the glenoid rim, in order to restore an effective labral "bumper".[3, 4] In order to accomplish this, suture anchors must be placed from a lateral direction to avoid skiving off the articular surface. Therefore, in this study, we attempted to simulate the conditions seen during performance of an arthroscopic Bankhart: lateral decubitus position, 30 degrees humeral abduction and a portal that afforded approximately 35 degrees angulation to the articular surface. With these conditions, the 5 o'clock anteroinferior portal can be established in cadaver shoulders without significant risk to major nerves. The minimum distance to the musculocutaneous nerve was 9.5 mm and the minimum distance to the axillary nerve was 15.5 mm. Abduction of the humerus moves the nerves closer to the portal site. Therefore, the arm should be placed in relative adduction at the time of this low portal placement. Rotation, flexion, and extension had no significant effect on the distance from the portal site to the nerves. The cephalic vein, however, appeared in jeopardy during portal placement in abduction with vein penetration seen in one specimen. Similarly, the conjoined tendon was imperiled with creation of this low portal. In fact, all specimens in which the portal was created in 30 degrees abduction had transgression of the conjoined tendon. The clinical significance of conjoined tendon violation is not certain, but an 8.25 mm cannula placed in this location could conceivably compromise tendon integrity or even violate a proximally located musculocutaneous nerve. Injury to the conjoined tendon can be mitigated by maximally adducting the humerus and using blunt dilators to both displace the cephalic vein and lessen tendon trauma.

One of the limitations of this study was the small sample size. Furthermore, the placement of cadaveric humerii in fixed abduction did not truly mimic the clinical state of longitudinal traction. It has been the senior authors' experience and that of Davidson et al. that longitudinal traction can obscure the starting point for this low portal.

Conclusion

The anteroinferior 5 o'clock portal position facilitates perpendicular approach to glenoid bone for fixation of Bankart lesion. Using an outside-in technique with the arm maximally adducted, in the lateral decubitus position, the portal can be established at a safe distance from vital nervous structures. However, both the cephalic vein and conjoined tendon are jeopardized when portal placement is performed in 30 degrees or greater of abduction. Both the musculocutaneous and axillary nerves can be safely avoided with "outside in" creation of this portal; however, increasing abduction brings these structures, as well as the conjoined tendon and cephalic vein, closer to the portal tract. Flexion, extension, and rotation had no significant effect on relationship to vital structures.

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Original Research

Posterior Rotator Interval and the Suprascapular Nerve: An Anatomic Study

Ammar Anbari, MD, Asif M. Ilyas, MD, Alan Zonno, MD, John D. Kelly IV, MD

Abstract

Background: Massive, contracted, immobile rotator cuff tears have two types: longitudinal and crescent-shaped. Crescent contracted tears are wider and are more difficult to repair. They often require both an anterior and a posterior interval slide to provide lateral mobility of the muscles. However, the posterior interval release places the suprascapular nerve at risk. In our anatomic study, we attempted to answer the following questions: How does one find the posterior interval in a massive rotator cuff tear? How does one avoid transgression of the suprascapular nerve?

Results: The right posterior interval angle measured 60.6 +/– 7.3, and the left measured 56.9 +/– 8.3. This converts to about 2 o'clock in left and 10 o'clock in right shoulders. The right scapular spine angle measured 70.6 +/– 8.1, and the left measured 71.3 +/– 7.8. This converts to about 2:20 o'clock in left and 9:40 o'clock in right shoulders. The closest branch of the suprascapular nerve at risk was located near the spinoglenoid notch. The right suprascapular nerve distance measured 1.9 +/– 0.2 cm, and the left distance measured 1.8 +/– 0.2 cm. No statistically significant differences were found between sides.

Conclusion: In our study, the interval extended from about 2 o'clock to 2:20 for left shoulders and 10 o'clock back to 9:40 for right shoulders. The closest suprascapular nerve branch encountered was located on the scapular spine about 2 cm medial and inferior to the glenoid rim.

Introduction

Advances in shoulder arthroscopy have made it possible to treat some of the more challenging types of rotator cuff tears. By using different portals and newly designed instruments, some of the tears which were treated open in the past are now repaired with arthroscopic techniques.

Four types of rotator cuff tears have been defined:[1] crescent-shaped, U-shaped, L-shaped and massive contracted immobile tears. The first three types represent over 90% of rotator cuff tears and are readily repaired without extensive rotator cuff mobilization. The fourth type, i.e. the massive, contracted and immobile tears accounts for 9.6% of tears and requires mobilization of the cuff muscles to achieve an adequate repair.[1] These massive tears are further divided into 2 subtypes: longitudinal and crescent-shaped. While it may be possible to perform an anterior interval slide and mobilize a longitudinal tear by 1–2 cm, the crescent-shaped tears may require both an anterior and posterior interval release. A posterior release may provide as much as 3 cm of lateral mobility of the supraspinatus and allows superior and lateral mobility of the infraspinatus.

The anatomy of the anterior rotator interval has been well defined previously and its location is marked by specific structures.[2] The posterior rotator interval, on the other hand, has not been studied as thoroughly and its location is not defined by specific structures. It simply represents the point where the supraspinatus and infraspinatus converge to insert on the greater tuberosity. When performing a posterior interval release, care has to be taken not to damage the suprascapular nerve which lies medial to the glenoid rim.

We performed an anatomic dissection study to answer two questions: 1) How does one best find the posterior rotator interval arthroscopically in a crescenteric massive rotator cuff tear? 2) How does one avoid transgression of the suprascapular nerve during the interval release?

Materials and Methods

Sixteen embalmed cadaveric shoulders (8 right sided and 8 left sided) with an average age of 69 years were used in this study. Selected cadavers had no previous surgery on their shoulders. The skin, subcutaneous tissue and the deltoid were dissected off the shoulders. Using a 1 inch osteotome, the acromion was removed as well. Next, using an osteotome, the greater and lesser tuberosities were taken off the remainder of the humeral head keeping their muscular attachment intact. A mid shaft humerus osteotomy was made and the proximal humerus was removed leaving the greater and lesser tuberosities and the glenoid intact and thereby maintaining the origin of the intervals at the glenoid.

At this point, a line was drawn on the glenoid from the 12 o'clock position though the bare area to the 6 o'clock position. With careful dissection, the posterior rotator interval between the supraspinatus and infraspinatus was identified and followed to the glenoid rim. A probe was inserted through the interval into the glenoid. A line was drawn from the point of entry of the probe to the middle of the 12–6 o'clock line. The angle formed between the 12–6 o'clock line and above line was measured (Figure 1). This angle represented the location of the interval at the glenoid rim.

The supraspinatus and infraspinatus were both peeled off further. The posterior interval was then followed from the glenoid medially to the scapular spine. Another line was drawn from the spine to the middle of the 12–6 o'clock line. The angle formed was also measured. This angle represented the medial extension of the rotator interval.

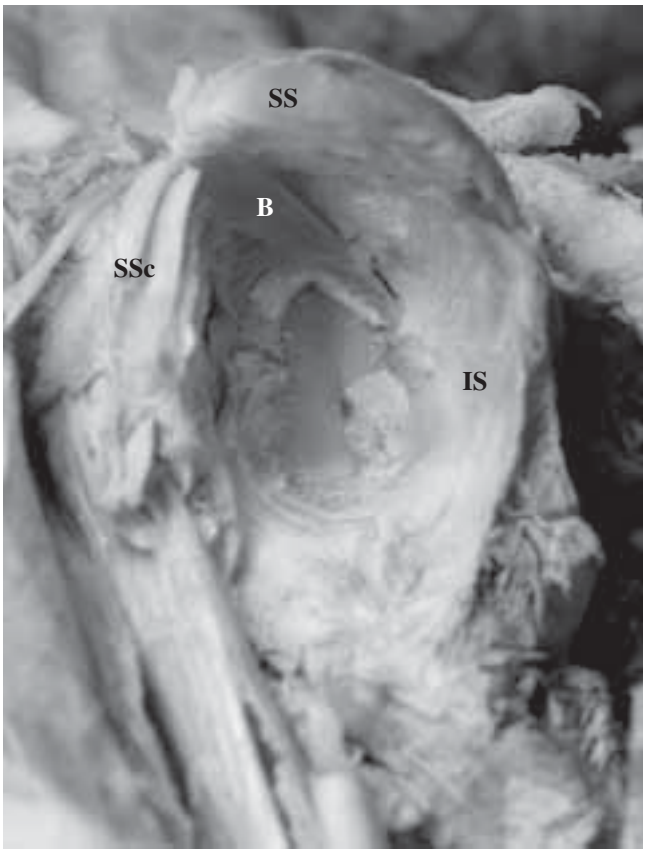

Figure 1. Dissected view of the glenoid. SSc — subscapularis, B — biceps tendon, SS — supraspinatus, IS — infraspinatus.

With further dissection, the suprascapular nerve was exposed. The closest branch along the path of the rotator interval was identified. The distance between the location of the interval on the glenoid rim and the nerve was measured (Figure 2).

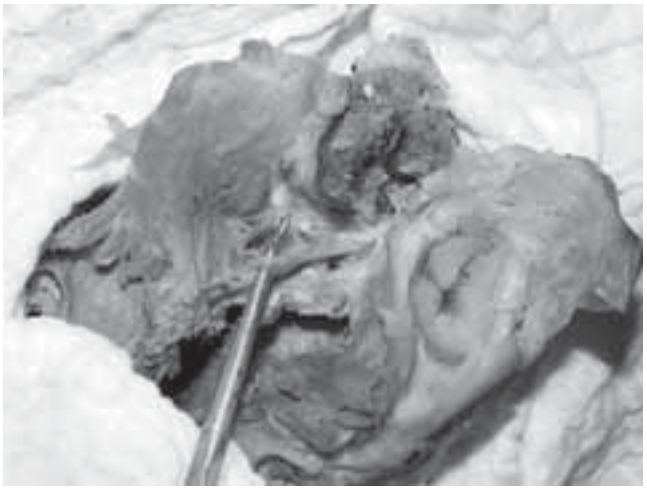

Figure 2. The dissected suprascapular nerve (on the probe).

Results

The first angle recorded, representing the lateral extent of the posterior rotator interval, measured 60.6 +/– 7.3 degrees on the right side and 56.9 +/– 8.3 degrees on the left. No statistically significant difference was found between right and left shoulders. This angle approximately converts to about 2 o'clock in left and 10 o'clock in right shoulders.

The closest branch of the suprascapular nerve to the posterior rotator interval was located near the spinoglenoid notch supplying the infraspinatus. The right suprascapular nerve distance from the lateral extent of the interval measured 1.9 +/– 0.2 cm, and the left measured 1.8 +/– 0.2 cm. No statistically significant differences were found between sides.

Discussion

Massive, contracted, and immobile rotator cuff tears have traditionally been repaired using open surgical techniques. When an arthroscopic repair was attempted for these tears, an anterior interval release with debridement was employed as described by Tauro.[2] However, in massive, immobile crescent-shaped tears, this method is not sufficient, and a double interval release (anterior and posterior) may be necessary.[1] The additional posterior release not only provides further mobilization of the supraspinatus muscle, but also allows the infraspinatus and teres minor to be mobilized and repaired in a biomechanically favorable position.[1] This can lead to a better surgical outcome in terms of range of motion, strength and pain control. However, extensive posterior release places the suprascapular nerve at risk for injury.

Warner et al. studied the anatomy and course of the suprascapular nerve in cadaveric shoulders.[4] The suprascapular nerve motor branches to the infraspinatus were about 2.1 +/– 0.5 cm from the posterior glenoid. These results were similar to our results. In our study, we found the shortest distance from the posterior interval on the glenoid to the suprascapular nerve branch to be 1.8–1.9 cm.

The anatomy and histology of the posterior rotator interval was studied by Miller et al.[3] The interval was found to be consistent in every cadaver studied with a length averaging 77.8 mm. The interval was made of loose fibroadipose tissue with synovial lining similar to that found in the shoulder capsule.

In our study, we performed an anatomic study to define the path of the posterior rotator interval from the glenoid to the point where the closest branch of the suprascapular nerve was encountered on the scapular spine. Following our findings, the optimal posterior interval release is performed with the arthroscope placed in the lateral portal. The soft tissues should be lifted away from the glenoid. Using a punch, the interval release should be performed by starting at the 2 o'clock position for left shoulders (10 o'clock for right

shoulders) and progressing in a slight medial-inferior direction towards the 2:20 position (9:40 for right shoulders). To avoid transgressing the suprascapular nerve, a release should be limited to under 2 cm.

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Original Research

Cross-Talk Between BMP-2 and Osteoactivin in Osteoblast Function

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Abstract

Introduction: Our laboratory previously showed that osteoactivin (OA) is a novel, osteoblast-related glycoprotein that plays a role in osteoblast differentiation and function. The purpose of this study was to examine the regulation of OA expression by BMP-2 and the role OA plays as a downstream mediator of BMP-2 effects in osteoblast function.

Materials and Methods: Using primary osteoblast cultures, we tested different doses of BMP-2 on the regulation of OA expression during osteoblast development. Regulation of OA expression by BMP-2 was examined using real time-PCR and Western blot analysis, in cultures treated with 50 ng/ml of BMP-2 and terminated after 7, 14 and 21 days. To test whether Smad-1 signaling is responsible for BMP-2 regulation of OA expression, osteoblast cultures were transfected with Smad1 siRNA, treated with 50 ng/ml of BMP-2 and analyzed by Western blot. To examine the role of OA as a downstream mediator of BMP-2 effects on osteoblast differentiation and matrix mineralization, osteoblast cultures were transfected with OA antisense oligonucleotides and treated with 50 ng/ml of BMP-2 and analyzed by Western blot. Alkaline phosphatase activity, module formation and matrix mineralization were also measured.

Results: BMP-2 treatment increased OA mRNA and protein expression in a dose-dependent manner and this upregulation was blocked in Smad1 siRNA transfected cultures. Cultures transfected with OA antisense oligonucleotides and treated with BMP-2 showed a reduction of OA expression associated with a significant reduction in early and late differentiation markers induced by BMP-2. Therefore, OA acts, at least in part, as a downstream mediator of BMP-2 effects on osteoblast differentiation and matrix mineralization.

Conclusions: Our findings suggest that BMP-2 regulates OA expression through the Smad1 signaling pathway. Our data also emphasize that OA protein acts as a downstream mediator of BMP-2 effects on osteoblast differentiation and function.

Introduction

Osteoactivin (OA) is a novel factor that was initially identified from studies using an animal model of Osteopetrosis, the *(op)* mutation in rats. Using the technique of mRNA differential display, the expression of OA cDNA was highly up-regulated in *op* compared to normal bone.[1] OA has high homology to human gpnmb (glycoprotein nmb),[2] and mouse DC-HIL (dendritic cell-associated, heparan sulfate proteoglycan-integrin ligand).[3] It has two isoforms, one is transmembrane type I with a MW of 65 kDa and the other is a secreted glycoprotein with MW of 115kDa.[1] OA was found to be highly expressed in various malignant tumors such as in glioma,[4] and hepatocellular carcinoma.[5] It has been shown that over-expression of OA in glioma cell lines,[6] as well as in hepatoma cell lines,[5] permits tumor invasiveness. The OA protein has been found to modulate osteoblast differentiation and function *in vitro* by stimulating osteoblast differentiation markers, including alkaline phosphatase activity, nodule formation, osteocalcin production, and matrix mineralization, without affecting cell proliferation or viability.[7]

Bone morphogenetic proteins (BMPs) are secreted growth factors, which form a subgroup of the transforming growth factor (TGF-β) superfamily based on amino acid homology of a highly conserved seven-cysteine domain in the carboxy-terminal region of the proteins.[8, 9] BMPs were originally known by their ability to induce ectopic bone and cartilage formation *in vivo,*[10] but recently it became evident that BMPs also act as multifunctional regulators in morphogenesis during development in vertebrates.[11] BMP dimers initiate signaling by binding to both type I and type II serine/threonine kinase receptors and the phosphorylation of type I receptors upon ligand binding.[12] Receptor-regulated Smads (R-Smads) (Smad 1, 5, 8) are activated by type I receptors (BMPR-IA or BMPR-IB),[13] associate with Smad4, and translocate to the nucleus, where they interact with transcription factors to regulate the transcription of target genes.

It is known that BMP proteins initiate the cascade of endochondral bone formation, where mesenchymal stem cells differentiate into chondrocytes which lay down cartilage that is replaced by bone tissue.[4] BMPs can also act as local factors in the regulation of osteoblast differentiation.[15] Several BMP knockout experiments in mice have contributed to elucidate the role of BMPs in bone formation and development. For example, BMP-2 deficient mice had amnion/chorion malformation and defects in cardiac development, and died during embryonic development.[16] A number of studies have shown that BMP-2, -3, -4, and -7 can up-regulate differentiation markers of the mature osteoblast, including short term

such as alkaline phosphatase (ALP) activity, and long term surrogates such as osteocalcin expression.[17] In addition, studies have demonstrated increased expression of osteoblast markers in pluripotent mesenchymal stem cell cultures after stimulation with BMP-2, suggesting that BMPs may regulate specific differentiation pathways in uncommitted cells.[18]

The similarity in the temporal expression patterns of BMP-2 and OA during osteoblast differentiation and the fact that both of these factors play a role in osteoblast function *in vitro* led us to examine the relationship between BMP2 and OA in osteoblasts. In this study, we were interested in determining whether BMP-2 regulates the expression of OA through the Smad-1 signaling pathway, and whether OA acts as a downstream mediator of BMP-2 effects on osteoblast development and function.

Materials and Methods

Antibodies

Anti-OA antibody (0.59 mg/ml) was raised against peptide sequence between amino acids 551-568 of the rat OA protein. This peptide was selected based on its potential antigenicity and screened using protein database to assure lack of homology. Chickens were immunized and the precipitated crude IgY was purified by affinity chromatography on Sepharose 4B derivatised with the antigen (immunizing peptide) (AstraZeneca, UK). Anti-Smad1 rabbit antibody (1.0 mg/ml) was purchased from Upstate (Lake Placid, NY). Anti-BMP-2 mouse antibody (0.5 mg/ml) was purchased from R&D system (Minneapolis, MN). HRP-conjugated donkey anti-chicken secondary antibody (0.8 mg/ml), HRP-conjugated goat antirabbit secondary antibody and (0.8 mg/ml) and HRP-conjugated goat anti-mouse antibody (0.8 mg/ml) were purchased from Jackson Immnunoresearch (West Grove, PA).

Primary Osteoblast Culture

Neonatal rat pups (1–4 days) were decapitated; their heads were swabbed with 70% ethanol. After a midline incision, the calvaria were isolated and placed in a Petri dish with 20 ml isolation media [phosphate buffered saline (PBS) + 1% penicillin/streptomycin + Hank's media (Sigma-Aldrich, St. Louis, MO)]. After removal of the dura, each calverium was cut along the sagittal and coronal sutures and all pieces transferred to another Petri dish with 20 ml isolation media before being cut into smaller pieces. The pieces were then transferred into a 50 ml siliconized Ehrlenmeyer flask with digest media (PBS + 0.1% collagenase P + 0.25% trypsin). The flask was placed in a shaker bath at 37°C for 5 minutes. After discarding the supernatant, 10 ml of digestion media was added and the pieces were again cut vigorously for about 5 minutes. The bone pieces were incubated for 15 minutes at 37°C. The supernatant was then filtered through 200 μm mesh metal screen filter (Fisher Scientific-Millipore filter and screen), split into 2 tubes, each with 5 ml washing media (Hank's media + 1% penicillin/streptomycin + 10% fetal bovine serum), and centrifuged for 5 minutes at 1200 rpm at 4°C. The supernatant was transferred into 2 tubes as in the first digestion. The same procedure was repeated again for the third digestion. The cell pellets were re-solubilized into 5 ml of fresh washing media. Fifty μl were removed and added to another 50 μl of Trypan blue, and the cells counted using a hemacytometer. Cells were then plated in 100 mm Petri dish at a density of 500,000 cells with 10 ml initial plating medium (EMEM (Mediatech-Cellgro, AK) + 1% penicillin/streptomycin + 10% fetal bovine serum) and incubated at 37°C with 5% CO_2. To induce osteoblast differentiation, cells were treated with 10 mM β glycerol phosphate + 25 μg/ml ascorbic acid on day 3 and every time the culture media was changed.

Treatment with Recombinant BMP-2

Primary osteoblasts were cultured in 6-well plates at a density of 50,000 cells/well, rinsed with Hank's medium and treated with different doses (10, 25, 50, 100 and 200 ng/ml) of recombinant BMP-2 (Sigma-Aldrich, St. Louis, MO) depending on the experiment protocol for 24 hours in serum free condition before assessment of OA protein expression by Western blot analysis.

RNA Isolaion

Cell cultures were harvested and frozen at -80oC. Cells were homogenized in Trizol, separated into organic and aqueous layer by chloroform, and RNA was recovered from the aqueous layer by isopropyl alcohol precipitation. Pellets were washed with 70% ethanol to clean RNA from DNA contamination. Concentration of RNA were calculated using spectrophotometer and RNA integrity was checked on a 1% agarose/paraformaldehyde minigel stained with ethidium bromide.

RT-PCR Analysis

RT-PCR analysis for OA and G3PDH were performed as follow. Two μg of total RNA isolated from the cell layer were reverse transcribed to cDNA at 42°C for 50 minutes in a volume of 20 μl containing the following components: 1x first strand buffer (5x = 250 mM Tris, pH = 8.3, 375 mM KCl and 15 mM MGCl2), 0.5 mM dNTP mix, 10 mM dithiothreitol (DTT), 0.5 ug oligo (dT) and 20 U Superscript II (RNase H free reverse transcriptase) (Invitrogen). The reaction was stopped at 70°C for 15 minutes, and 1 U RNase H was added to the mixture followed by incubation at 37°C for another 10 minutes to degrade the RNA. One μl aliquots of the generated cDNA was amplified in 50 μl of PCR reaction mixture containing 1 nM primers, 10 μl 10x Advantage buffer, 10 nM dNTP mix, 1 μl DMSO and 1 μl Advantage polymerase mix (Clonetech). The primers for OA were sense; 5' CCAGAAGAATGACCGGAACTCG 3' and antisense 5' CAGGCTTCCGTGGTAGTGG 3'. These primers were

designed from the 5' end of the protein coding region starting at position 729 bp from the ATG starting codon to position 1280. The primers for G3PDH were sense; 5' ACCACAGTCCATGCCATCAC 3' and antisense 5' TCCACCACCCTGTTGCTGTA 3'. PCR were performed using Perkin Elmer GeneAmp PCR System 9600 (Perkin-Elmer). PCR parameters for OA were: denaturation at 94°C for 3 minutes, followed b 25 cycles of 94°C for 20 seconds, 62°C for 20 seconds and 68°C for 20 seconds; with final extension step at 68°C for 7 minutes. The expected OA PCR product was 552 bp. PCR parameters for G3PDH were: denaturation at 94°C for 3 minutes, followed by 35 cycles of 94°C for 45 seconds, 60°C for 2 minutes and 72°C for 2 minutes; with final extension step at 72°C for 7 minutes. The expected G3PDH PCR product was 452 bp. The PCR products were analyzed by 1% agarose gel electrophoresis stained with ethidium bromide. A 100 bp ladder was used as a M.W marker (Invitrogen).

Real-Time PCR Analysis

Total RNA was isolated as described above. CDNA was prepared using TaqMan Reverse Transcription Kit (applied Biosystem, Foster City, CA). PCR was performed on ABI PRISM 7700 (Applied Biosystem) usinf the Cyber Green method. Primers for OA and G3PDH, as internal control, are described above.

Transfection of OA Antisense Oligonucleotide

Primary osteoblasts were cultured in 6-well plates as described above. When cells reached subconfluence (60%, day 2 in culture), they were transfected with different doses (0.25, 0.5 and 1 μM) of OA antisense depending on the experimental protocol or 0.5 μM sense oligonucleotides using Lipofectamine 2000 (Invitrogen, Carlsbad, CA). For the effect of OA antisense on mineralization (day 21), on day 14, cultures were treated with a second dose of OA antisense. The sequence of OA antisense oligonucleotides is 5'-CCCTAGTCCCATCCACCAGG-3' and the sequence of sense oligonucleotides is 5'-GGGCGTCTCTGAAAGGTA-ACG-3'. The sequence of OA antisense oligo was analyzed by Blast search. No homologies other than OA were found in the database. Transection efficiency was determined using BLOCK-iT fluorescent oligonucleotides (Invitrogen, Carlsbad, CA). The primary osteoblast cultures were transfected with fluorescent oligo as described above, and then transfection efficiency was evaluated by counting fluorescent labeled cells versus total number of cells. A transfection efficiency of 65% was reproducibly achieved.

Transfection of Smad1 siRNA

Primary osteoblasts were cultured in 6-well plate as described above and when cultures reached 60% confluence, they were transfected with different doses (10–100 nM) of Smad1 siRNA (Santa Cruz, CA) depending on the experimental protocol or 50 nM non-silencing siRNA (Santa Cruz, CA) using Lipofectamine 2000 (Invitrogen, Carlsbad, CA) as a vehicle. After 4 hours of transfection, 10% serum + EMEM were added to transfection medium and the cells were incubated at 37°C in CO_2 for 24 hours until cells were ready for transgene expression. Transfection efficiency was determined to be between 70–80%.

Protein Isolation

After culture termination, cells were rinsed with 10 ml ice cold PBS, then trypsinized with 0.25% trypsin and 2 mM EDTA in Hank's medium for 10 minutes. Cell layers were harvested and centrifuged at 4°C for 10 minutes at 1200 rpm. Cells were lysed in 500 μl of RIPA buffer consisting of (50 mM Tris HCL; pH 7.5; 135 mM NaCl; 1% Triton X-100; 1% sodium deoxycholate; 2 mM EDTA; 50 mM NaF; 2 mM sodium orthovanadate; 10 μg/ml aprotinin; 10 μg/ml leupeptin and 1 mM PMSF). Samples were centrifuged at 14,000 rpm for 15 minutes at 4°C and total protein concentration was measured using bicinchoninic acid (BCA) protein assay (Pierce, Rockford, IL).

Western Blot Analysis

Twenty to 40 μg of total proteins isolated from primary osteoblast cultures were mixed with 2X sample buffer and heated at 100°C for 5 minutes to denature the proteins. Samples were subjected to 10% SDSPAGE in 1x TGS (0.25 M Tris, 1.92 M glycine and 1.0% SDS in ddH2O, pH 8.6) (Biorad, Hercules, CA) at 100 mv for one hour. Gel was then transferred to PVDF membrane by semi-dry transfer apparatus (Biorad, Hercules, CA) at 15 mv for one hour at room temperature. The blot was incubated in blocking buffer (5% skim milk + 1% bovine serum albumin) for one hour at room temperature. Primary antibody was added to blocking buffer overnight at 4°C. The next day, the blot was washed 5 times in 1X TTBS (Tris buffered saline + 0.1% Tween 20) (Biorad, Hercules, CA), 5 minutes each, on a shaker. The blot was then incubated with HRP-conjugated secondary antibody, for one hour at room temperature. The blot was washed in TTBS for 5 times, 5 minutes each time. Protein was visualized suing ECL kit (Pierce, Rockford, IL) and signals were detected using XL-exposure films.

Alkaline Phosphatase Histochemistry

Primary osteoblasts were cultured in 12-well plates. Alkaline Phosphatase (ALP) staining was performed on day 14 using ALP staining kit (Sigma-Aldrich, Louis, MO). Briefly, cells were fixed with citrate-acetone-formaldehyde fixative for one minute then rinsed with dH_2O. Alkaline dye mixture was added to the cells and incubated at room temperature for 15 minutes with protection from direct light. Cells were then rinsed with dH2O for 2 minutes before counterstaining with hematoxylin for 2 minutes. Cells were then rinsed with dH_2O and allowed to air dry before evaluating with E600 Nikon inverted microscope.

von Kossa Staining of Mineralized Nodules

Von Kossa staining was used to stain mineralized matrix on day 21 of culture. Cells were rinsed twice with Hank's Balanced Salt solution, and then fixed with 2% paraformaldehyde solution for 10 minutes at room temperature. The cells were then rinsed with dH_2O and stained with 3% silver nitrate solution; cells were exposed to direct sunlight for one hour. Cells were then rinsed with dH_2O and fixed by adding 5% sodium thiosulfate for 2 minutes. Cells were counterstained with 1% fast green then rinsed with dH_2O and allowed to air dry. Cells were then evaluated using E600 Nikon inverted microscope.

MTT Cell Viability Assay

Primary osteoblasts were plated at 24-well plates at a density of 12,400 cells per well for 5 days and 24 hours prior to termination. MTT (3-[4,5-Dimethylthiazole-2-yl]-2,5-diphenyltetrazolium bromide), (Sigma-Aldrich, Louis, MO), (5 mg/ml) substrate was added (100 µl/well) and cells were incubated for 4 hours in 37°C CO_2. After the incubation period, solubilizer (20% SDS and 50% DMF) was added (250 µl/well) and plates were rocked at room temperature overnight to solubilize the formazen crystals. The next day, 100 µl aliquots were transferred into 96-well plate and samples were read on an ELISA plate reader at 570 nm.

Image Analysis

Pictures for stained or non-stained mineralized nodules were taken from different fields using E600 Nikon inverted microscope. Images were analyzed using BIOQUANT 98 (Bioquant Image Analysis Corporation, Nashville, TN) image analysis software. Nodule number was computed using the object count feature. The size of the nodules was computed using the area measurement feature combined with the irregular region of interest (ROI) option of the BIOQUANT program. Nodule mineralization was computed using the videocount area array option. Videocount area is defined as the number of pixels in a field that meet a user-defined color threshold of staining multiplied by the area of a pixel at the selected magnification. In this case, color thresholds were selected based on mean level of von Kossa staining. Percent area fractions of von Kossa staining were calculated by dividing the videocount area containing pixels at or above the defined threshold by the videocount area of total number of pixels in the entire field, and multiplying by 100. This determination was made at 4 different locations per well, 3 wells per group.

Statistical Analysis

For multiple group comparison, analysis of variance (ANOVA) was used to evaluate the effect of one variable on multiple independent groups. In the event of a significant group effect, individual pairs of means were compared using Newman-Keuls post hoc test. A p value ≤0.05 was considered statistically significant. Group means + standard error of the mean (SEM) are plotted in graphs.

Results

Induction of OA Expression by BMP-2

It has been reported previously that osteoblasts in culture undergo three different stages of differentiation, proliferation (1–7 days), matrix maturation (7–14) and matrix mineralization (14–21).[19, 20] We first examined the endogenous level of BMP-2 in osteoblasts during different stages of development in culture using Western blot analysis. BMP-2 expression levels were markedly elevated during the third week (the stage of matrix mineralization) compared to first and second weeks of culture (the stages of cell proliferation and matrix maturation, respectively) (Fig. 1A). Densitometric analysis shows a greater than 2-fold increase in BMP-2 expression level during third week of culture (Fig. 1B). Endogenous OA protein expression was also examined

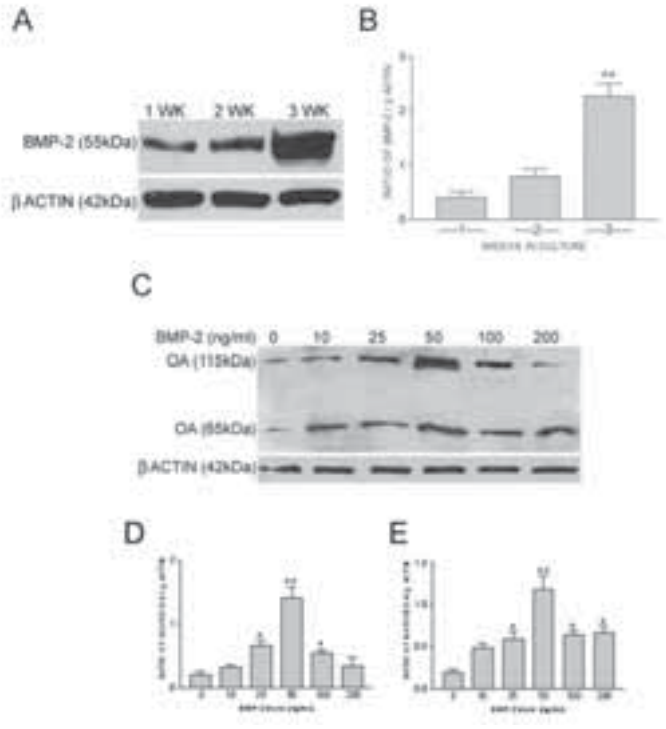

Figure 1. BMP-2 Induces OA Expression in Osteoblast Culture. (A-B) Primary osteoblasts were cultured and terminated at the end of 1, 2, or 3 weeks of culture. (A) Immunoblot shows endogenous level of BMP-2 expression reaching a maximum at 3 weeks (3 WK) of culture. β-actin was used as a loading control. (B) Densitometry of 3 immunoblots quantifying percent of BMP-2 expression as a ratio of β-actin. Data presented as mean + SEM. ** = $p < 0.01$ compared to the first week in culture. (C-D) Primary osteoblasts were cultured for 5 days before switching to serum free media containing different doses of BMP-2 (0–200 ng/ml) for 24 hours before termination. (C) Immunoblot shows that OA expression reached maximum in cultures treated with 50 ng/ml BMP-2. β-actin was used as a loading control. (D-E) Densitometry of 3 immunoblots quantifying percent of the glycosylated (D) and transmembrane (E) isoforms of OA protein expression over β-actin. Data presented as mean + SEM. * = $p < 0.05$ and ** = $p < 0.01$ compared to untreated, control cultures.

during osteoblast development and also showing maximal expression levels during osteoblast matrix mineralization stage at 3 weeks[7] and Fig. 2. These data imply that BMP-2 and OA proteins play a role in regulating osteoblast terminal differentiation, i.e. (matrix mineralization).

To examine a possible relationship between BMP-2 and OA proteins and whether BMP-2 may induce OA expression *in vitro,* primary osteoblast cultures were treated with different doses (0-200 ng/ml) of BMP-2 for 24 hours in serum-free media and OA expression was assessed by real-time PCR (data not shown) and Western blot analyses. Levels of the transmembrane (65kDa) and the glycosylated (115kDa) OA isoforms were gradually increased in a dose-dependent manner, reaching maximum at 50 ng/ml of BMP-2 before declining again (Fig. 1C). Densitometric analysis shows 4–5-fold increase in glycosylated and transmembrane OA isoforms, compared to untreated control (Fig. 1D and E). These data indicate that 50 ng/ml of BMP-2 can induce OA expression *in vitro.* Similar results were obtained using quantitative PCR (data not shown).

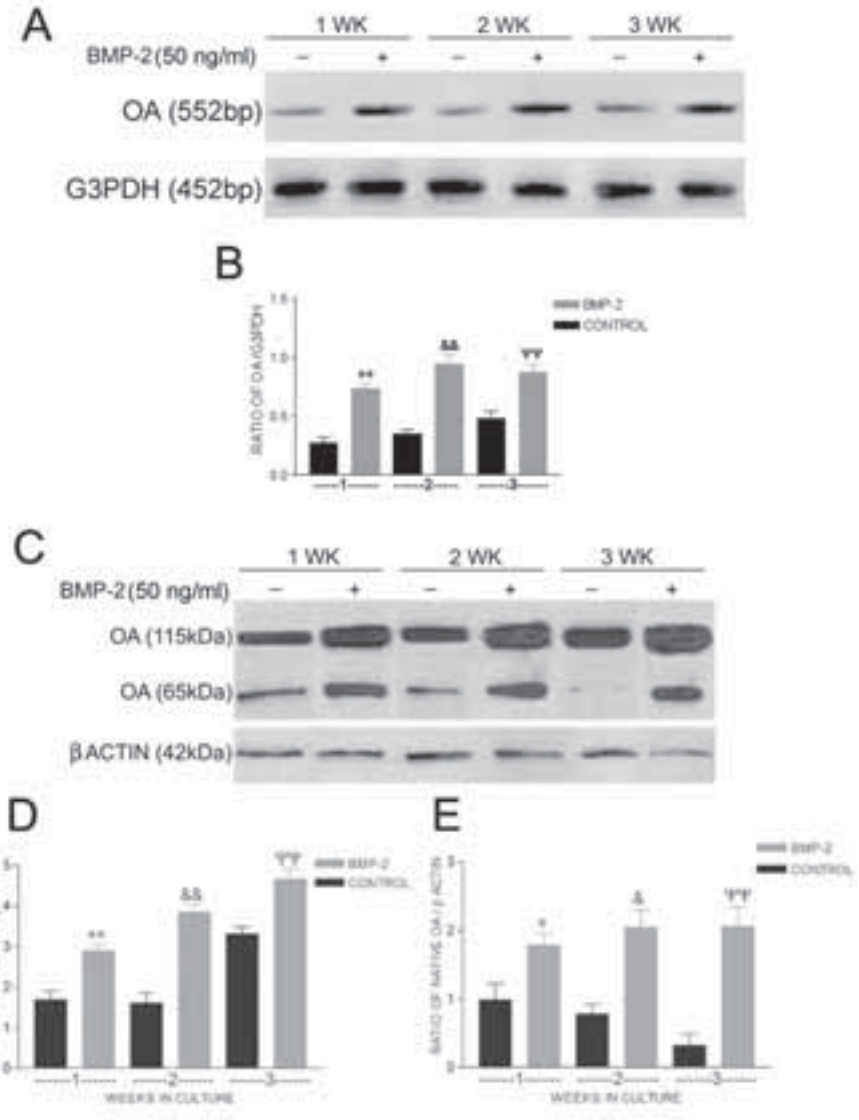

Figure 2. Regulation of OA Expression by BMP-2 during Osteoblast Differentiation. Primary osteoblasts were cultured and switched to serum free media with (+) or without (–) 50 ng/ml BMP-2 for 24 hours before termination at the end of 1, 2, or 3 weeks of culture. (A) RT-PCR analysis shows that OA mRNA is increased in BMP-2 treated cultures when compared to untreated controls. G3PDH was used as loading control. (B) Quantitative real-time PCR of RNA colleted at different time points (1, 2, 3 WK) in cultures with (+) and without (β) BMP-2 treatment. Data are presented as the ratio of OA to G3PDH. BMP-2 significantly increased OA mRNA levels at all time points examined. Data presented as mean + SEM. ** = $p < 0.05$, && = $p < 0.01$ and ψψ = $p< 0.01$ when compared to untreated controls of each time points. (C) Immunoblot shows up-regulation of two OA isoforms (115kDa and 65kDa) in BMP-2 treated cultures compared to untreated controls. β-actin was used as a loading control. (D-E) Densitometry of 3 immunoblots quantifying percent of the glycosylated (glyco) (115kDa) (C), and the transmembrane isoforms (65kDa) (E) of OA protein as a ratio of β-actin. Data presented as mean + SEM. * = $p < 0.05$ and ** = $p < 0.01$ when compared to untreated 1 Wk cultures. & = $p < 0.05$ and && = $p < 0.01$ when compared to untreated 2 Wk cultures. ψψ = $p < 0.01$ when compared to untreated 3 Wk cultures.

BMP-2 Regulates OA Expression During Osteoblast Differentiation

To examine the regulation of OA mRNA and protein expression by BMP-2 during osteoblast differentiation *in vitro,* osteoblasts were treated with 50 ng/ml of BMP-2 for 24 hours before termination of the culture at 3 time points during their course of development (1, 2 and 3 weeks). Semi-quantitative RTPCR and quantitative real-time PCR analyses were performed showing that expression of OA mRNA levels was increased following BMP-2 treatment at 1, 2 and 3 weeks compared to untreated control (Fig. 2A and B). Using Western blot analysis, expression of OA glycosylated and transmembrane isoforms (MW 116kDa and 65kDa, respectively) was increased after BMP-2 treatment during each stage of development compared to untreated controls (Fig. 2C). Densitometric analysis showed that expression of both the glycosylated (115kDa) and transmembrane (65kDa) OA isoforms were significantly increased after BMP-2 treatment (Fig. 2D and E). Collectively, these data demonstrate that BMP-2 up-regulates OA mRNA and protein expression during different stages of osteoblast differentiation in culture. These data also show that endogenous levels of the glycosylated OA isoform increased while the transmembrane OA isoform decreased as the cultures terminally differentiated (Fig 2C, D and E).

BMP-2 Regulates OA Expression Through the Smad1 Signaling Pathway

To examine the mechanism by which BMP-2 regulates OA expression in osteoblast cultures, Smad1 siRNA oligonucleotides were used to down-regulate Smad1 expression in culture. To select the appropriate dose of Smad1 siRNA, osteoblasts were transfected with different doses of Smad1 siRNA. Smad1 expression (Fig. 3A) was not inhibited at 25 nM of Smad1 siRNA, while 50 nM and 100 nM doses significantly inhibited Smad1 level expression, compared to untreated controls (data not shown) or controls transfected with 50 nM of non-silencing siRNA; both controls showed similar results. Densitometric analysis showed a greater than 50% reduction of Smad1 expression in cultures transfected with 50 nM, and a greater than 90% reduction in cultures transfected with 100 nM of Smad1 siRNA, compared to controls (Fig. 3B). To select the appropriate non-toxic dose of Smad1 siRNA, the same doses were used in cell viability MTT assays. Smad1 siRNA at doses of 25 and 50 nM showed no inhibition in cell viability, while a dose of 100 nM siRNA had significant reduction of cell viability (Fig. 3C). These data suggest that Smad1 siRNA at a dose of 50 nM is the most appropriate for down-regulating Smad-1 expression without affecting cell viability. To assess whether the regulation of OA expression by BMP-2 is mediated through Smad1 in cultured osteoblasts, cultures at 60% confluence were transfected with 50 nM of Smad1 siRNA and then received BMP-2 treatment at 50 ng/ml in the last 24 hours of a 5-day culture period. Smad1 and OA protein expression (Fig. 3D) were up-regulated in BMP-2 treated cultures compared to untreated controls (data not shown) or cultures transfected with 50 nM non-silencing siRNA. Inhibition of Smad1 expression by Smad1 siRNA showed an inhibition of OA expression in primary osteoblast cultures. BMP-2 treatment of Smad1 siRNA transfected cultures resulted in levels of Smad1 and OA protein expression that were similar to control levels (Fig. 3D). Quantitative analysis showed that BMP-2 treatment alone caused a significant increase in Smad-1 and OA protein expression (Fig. 3E and F). There was a significant reduction in Smad1 and OA expression, in Smad1 siRNA transfected cultures compared to controls (Fig. 3E and F), while BMP-2 treatment of these cultures resulted in Smad1 and OA expression levels similar to the control cultures. Collectively, these data suggest that BMP-2 regulates OA expression, in part, through the Smad1 signaling pathway.

Down-regulation of OA Expression by OA Antisense Oligonucleotides

To examine the role of the OA protein in regulation of osteoblast differentiation with BMP-2, OA antisense oligonucleotides were used to down-regulate OA expression in osteoblast cultures. For assessment of transfection efficiency in primary osteoblasts, cells were cultured for 2 days, transfected with 50 nM fluorescent-tagged scrambled oligonucleotides using Lipofectamine 2000 for 4 hours. Cells with a green fluorescent signal (Fig. 4B) were counted and it was determined that ~65% of osteoblasts were transfected (Fig. 4C).

To select the appropriate dose of the OA antisense oligonucleotides, primary osteoblasts were transfected with different doses of OA antisense and assessed by Western blot analysis. OA expression was not inhibited by the 0.25 μM dose, while 0.5 μM and 1 μM dose resulted in a robust inhibition of OA expression, compared to untreated controls (data not shown) or controls transfected with 0.5 μM OA sense oligonucleotides (Fig. 4D and E). The OA expression levels were similar in untransfected cultures and cultures transfected with 0.5 μM OA sense oligonucleotides. To select the appropriate non-toxic dose of OA antisense oligonucleotides, MTT assay for cell viability was performed using 0.25, 0.5 and 1 μM of OA antisense (Fig. 4F) and both 0.25 and 0.5 μM doses of OA antisense had no effect, the but 1 μM dose of OA antisense showed a significant reduction in cell viability. Therefore, a dose of 0.5 μM OA anitsense was used for all subsequent experiments.

To examine if OA antisense maintains inhibition of OA expression during terminal osteoblast differentiation (3 weeks in culture), cells at 60% confluence (day 2 in culture) were transfected with 0.5 μM OA antisense and treated at day 14 with a second dose of OA antisense. OA expression was assessed on day 21 by Western blot analysis and was found to be dramatically down-regulated compared to control cultures transfected with OA sense oligonucleotides

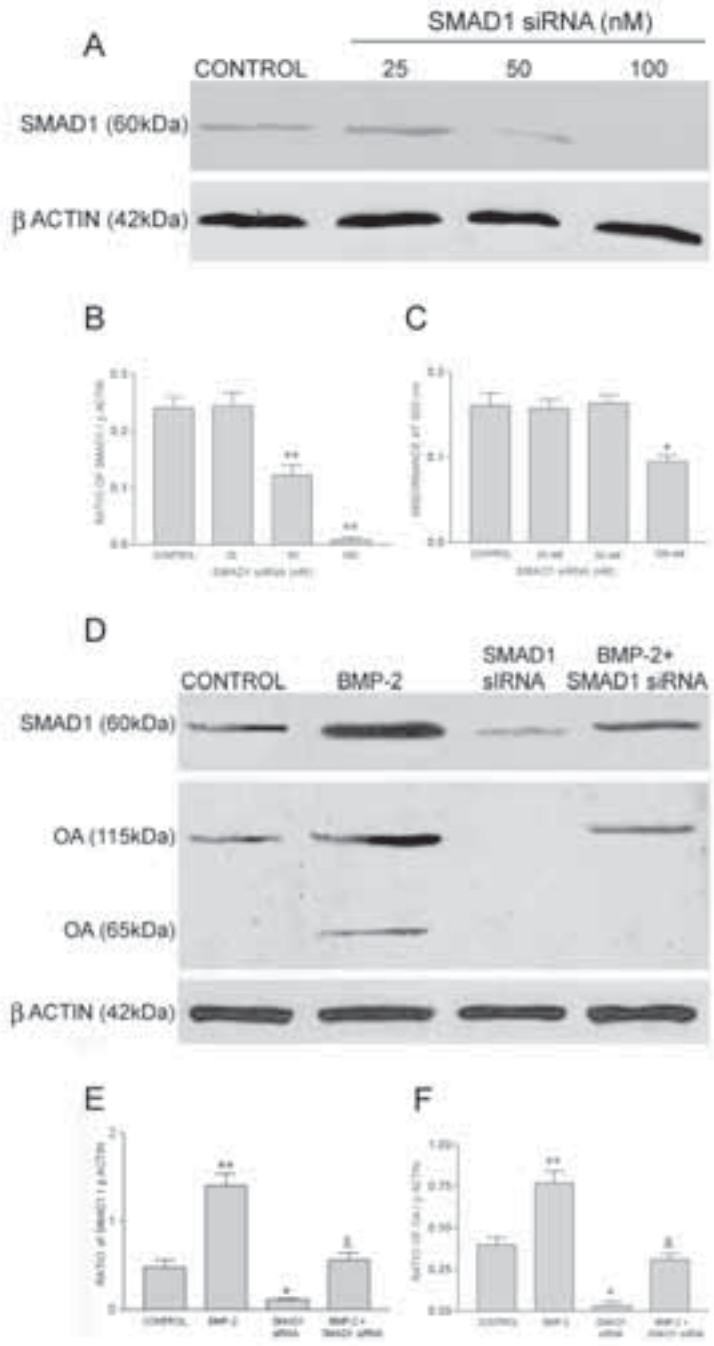

Figure 3. Smad1 Inhibition Down-regulates OA Expression. (A) Primary osteoblasts were cultured, and then transfected with different doses of Smad1 siRNA (0-100 nM). (A) Immunoblot shows Smad1 expression was inhibited by 50 nM and 100 nM doses of Smad1 siRNA compared to control untreated (not shown) or cultures treated with non-silencing siRNA. β-actin was used as a loading control. (B) Densitometry of 3 independent immunoblots quantifying percent of Smad1 expression as a ratio of β-actin. (C) MTT assay for Smad1 siRNA at different doses. Primary osteoblasts were cultured and transfected with different doses of Smad1 siRNA and MTT assay for cell viability was performed. Non-silencing siRNA (control) and Smad1 siRNA (50nM dose) had no significant effect on osteoblast viability. Data presented as mean + SEM. * = $p < 0.05$ and ** = $p < 0.01$ when compared to control. (D-F) Primary osteoblasts were cultured then transfected with 50 nM Smad1 siRNA before switching to serum free media with 50 ng/ml BMP-2 for 24 hours prior to termination. (D) BMP-2 treatment significantly increased Smad-1 and OA expression when compared to non-silencing siRNA tranfected cultures. Smad-1 siRNA transfected cultures showed inhibition of both Smad-1 and OA protein expression levels when compared to non-silencing siRNA transfected control cultures. The Smad1and OA expression levels induced by BMP-2 was blocked by Smad1 siRNA compared to BMP-2 treated cultures and to levels comparable to nonsilencing siRNA transfected control cultures. β-actin was used as a loading control. (E-F) Densitometry of 3 independent immunoblots quantifying percent of Smad1 (E) or glyco-OA (F) protein expression as a ratio of β-actin. Data presented as mean + SEM. * = $p < 0.05$ and ** = $p < 0.01$ when compared to non-silencing siRNA transfected controls; & = $p < 0.05$ when compared to BMP-2 treated cultures.

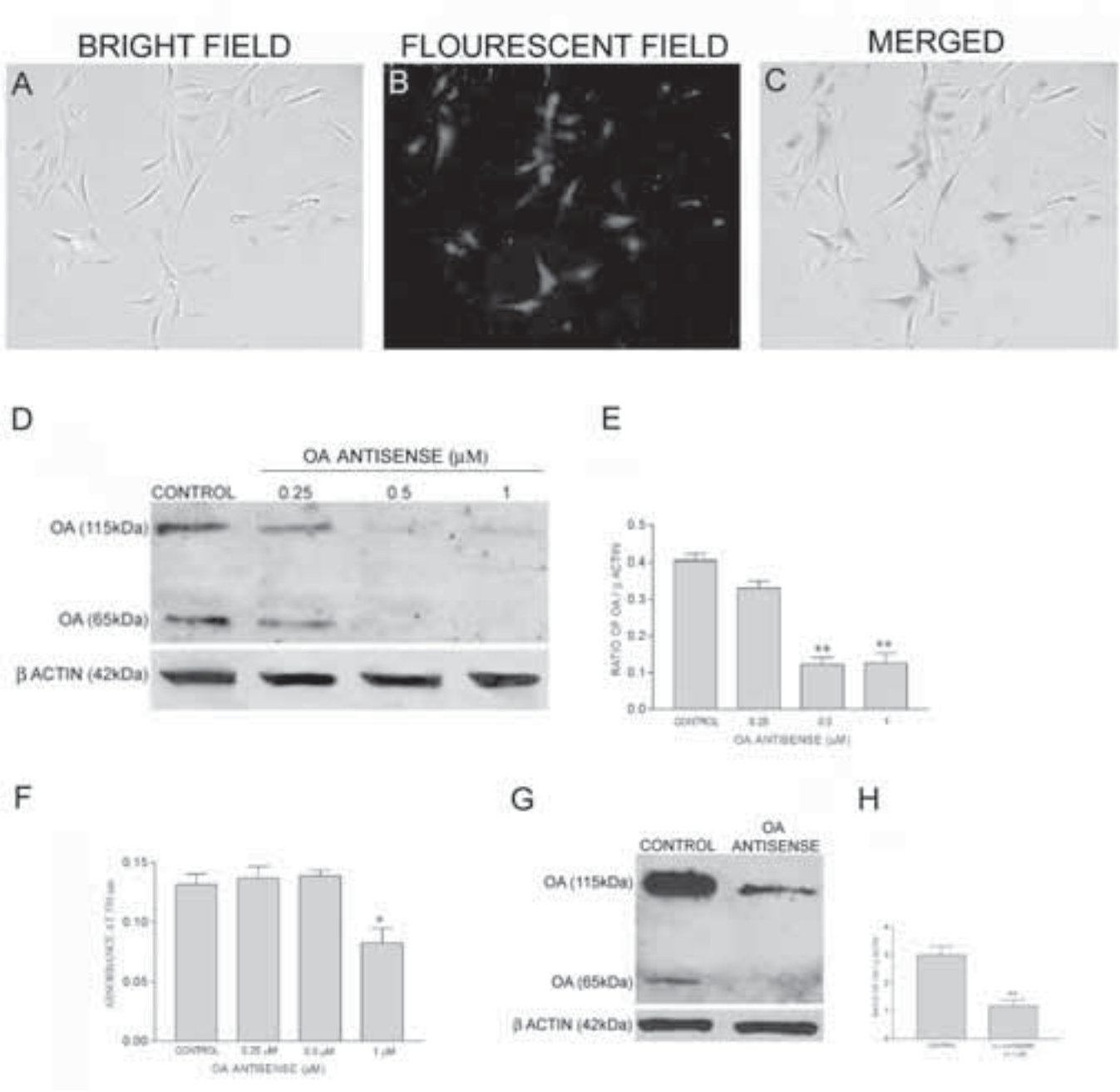

Figure 4. Down-regulation of OA Expression with OA Antisense Oligonucleotides. (A-C) Primary osteoblasts were cultured and transfected with 50 nM fluorescent oligonucleotides for 4 hours. Oligo uptake was assessed and transfection efficiency determined to be ~65%. Magnification: 300x. (D-E) Primary osteoblasts were cultured and transfected with 0.25, 0.5, or 1 μM of OA antisense. (D) Immunoblot shows both isoforms of OA expression were greatly inhibited by 0.5 μM and 1 μM doses of OA antisense when compared to OA sense transfected control cultures. β-actin was used as a loading control. (E) Densitometry of 3 immunoblots quantifying percent of the glycosylated isoform of OA expression as a ratio of β-actin. Data presented as mean + SEM (** = $p < 0.01$ when compared to sense transfected control). (F) An MTT assay for cell viability was performed after 5 days of culture. Control represents cultures transfected with 0.5 μM OA sense. 0.25 μM and 0.5 μM of OA antisense had no significant effect on osteoblast viability. Data presented as mean + SEM. * = $p < 0.05$ when compared to sense transfected control. (G-H) Primary osteoblasts were cultured, transfected with 0.5 μM OA antisense, treated at day 14 with a second dose of OA antisense and then terminated at day 21. (G) Immunoblot shows inhibition of both isoforms of OA, compared to OA sense transfected controls. β-actin was used as loading control. (H) Densitometry of 3 immunoblots quantifying percent of glycosylated OA expression as a ratio of β-actin. Data presented as mean + SEM. ** = $p < 0.01$ compared to control.

(Fig. 4G). Quantification showed a greater than 70% reduction of OA expression level in OA antisense transfected osteoblasts compared to sense transfected controls (Fig. 4H). These data suggest that 0.5 μM of OA antisense is the most appropriate dose for oligotransfection resulting in significant down-regulation of OA expression without affecting cell viability.

Down-regulation of OA Expression Inhibits BMP-2-Induced Early Osteoblast Differentiation

To examine whether OA is a downstream mediator of BMP-2 effects on early osteoblast differentiation in culture, primary osteoblasts were treated with either BMP-2 alone or transfected with 0.5 μM of OA antisense, and then treated with 50 ng/ml of BMP-2 for 24 hours before culture termination at day 5 (data not shown) and 14. By Western blot analysis, OA expression was highly up-regulated in BMP-2 treated cultures, as shown above, compared to cultures transfected with OA sense. The increased in OA expression induced by BMP-2 was blocked in OA antisense transfected cultures (Fig. 5A). Densitometric analysis showed that BMP-2 treatment alone significantly increased OA expression, while BMP-2 treatment of OA antisense transfected cultures demonstrated OA levels that were comparable to sense transfected, control cultures (Fig. 5B).

To determine whether the effects of BMP-2 on alkaline phosphates (ALP) activity is OAdependent, primary osteoblasts were cultured, transfected with 0.5 μM of OA antisense and treated with 50 ng/ml of BMP-2 for 24 hours before termination on day 14. BMP-2 treatment alone greatly enhanced ALP staining (Fig. 5D) compared to OA sense trasnfected control cultures (Fig. 5C). In contrast, ALP staining was reduced to control levels in OA antisense transfected cultures treated with BMP-2 (Fig. 5E).

The percent of area fraction of ALP staining was measured and calculated for each condition using computerized bioquantification (BIOQUANT) software. BMP-2 treated cultures showed a significant increase in percent ALP area fraction staining (Fig. 5G) compared to controls. In contrast, the increase in area fraction of ALP staining induced by BMP-2 was decreased in OA antisense transfected cultures resulted in a percent ALP area fraction similar to control levels (Fig. 5G). These data suggest that OA can act, at least in part, as a downstream mediator of BMP-2 effects on early osteoblast differentiation and that the induction of ALP activity by BMP-2 is OA-dependent.

Down-regulation of OA Expression Inhibits BMP-2-Induced Nodule Formation and Mineralization

To examine whether down-regulation of OA expression by OA antisense has any effect on BMP-2-induced ostoeblast nodule formation and matrix mineralization, primary osteoblasts were either treated with BMP-2 alone, or transfected with 0.5 μM of OA antisense at day 2 in culture, treated with second dose of OA antisense at day 14 then treated with BMP-2 and terminated at day 21 for the measurement of OA expression, nodule formation and matrix mineralization. By Western blot analysis, OA expression was upregulated by BMP-2 treatment compared to cultures transfected with OA sense oligonucleotides (Fig. 6A). This increase in OA expression induced by BMP-2 was inhibited in OA antisense transfected cultures (Fig. 6A and B).

Von Kossa staining was used to stain minerals. Cultures treated with BMP-2 (Fig. 6D) showed larger areas of mineral staining compared to OA sense transfected control cultures (Fig. 6C), while cultures transfected with OA antisense and treated with BMP-2 showed a reduction in mineralization compared to BMP-2 treatment alone (compare Fig. 6E with 6D).

Mineralization (percent of area fraction of von Kossa), nodule size (area) and nodule number were measured and calculated for each condition using BIOQUANT software. BMP-2 treated cultures showed a 3-fold increase in percent von Kossa area fraction staining (Fig. 6F), as well as significant increases in nodule size and number (Table 1) compared to OA sense tranasfected controls cultures. The treatment of OA antisense transfected cultures with BMP-2 resulted in a percent von Kossa area fraction staining comparable to OA sense transfected control cultures (Fig. 6G). These data clearly demonstrate that OA acts, at least in part, as a downstream mediator of BMP-2-induced nodule formation and matrix mineralization in primary osteoblasts culture.

Table 1.

Condition	Nodule Count in 100 mm dish Mean ± SEM (% Control)	Average Nodule Size (μm) Mean ± SEM (% Control)
Control	34 ± 2.3 (100)	34 ± 0.25 (100)
BMP-2	45 ± 3.7** (134)	5.4 ± 0.14** (152)
BMP-2 + OA antisense	28 ± 2.4* (84)	3.1 ± 0.3* (93)

Nodule count and average nodule size in each condition were quantified. Numbers represent mean ± SEM of 3 independent experiments. * = $p < 0.05$ when compared to BMP-2 treated cultures, ** = $p < 0.01$ when compared to OA sense transfected controls. Control = 100%.

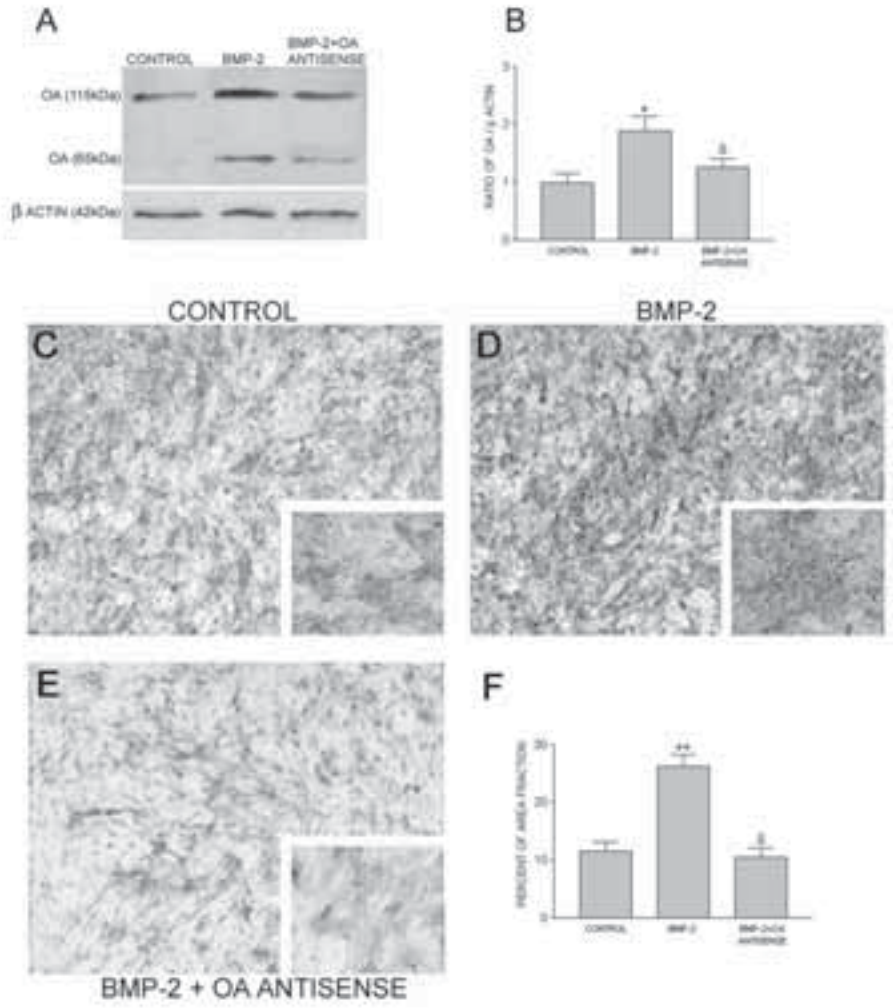

Figure 5. Down-regulation of OA Expression Inhibits BMP-2-Induced Alkaline Phosphatase Activity (A-B) Primary osteoblasts were cultured, treated with BMP-2 alone or transfected with OA antisense (0.5 μM) before switching to serum free media with 50 ng/ml of BMP-2 for 24 hours prior to termination at day 5 (data not shown) and 14. (A) Immunoblot shows BMP-2 treatment increased OA expression and this increase was blocked with OA anti-sense to levels comparable to sense transfected controls. β-actin was used as a loading control. (B) Densitometry of 3 independent immunoblots quantifying percent of glyco-OA expression over β-actin. Data presented as mean + SEM. * = $p < 0.05$ when compared to OA sense transfetced controls; & = $p < 0.05$ when compared to BMP-2 treated cultures. (C-F) Primary osteoblasts were cultured, treated as above and terminated for alkaline phosphatase (ALP) staining at day 14. Photomicrographs of alkaline phosphatase staining (red). BMP-2 treatment (D) shows intense staining for ALP compared to sense transfected controls (C). OA anitsense transfected cells treated with BMP-2 shows less alkaline phosphatase staining compared to BMP-2 treated cultures and to levels comparable to OA sense transfected cultures (E). Low power photomicrograph magnification: 60x; inset magnification: 300x. (F) Bioquant analysis of 3 independent experiments quantifying percent area fraction of the field occupied by ALP staining. Data presented as mean + SEM (** = $p < 0.01$ when compared to sense transfected controls, & = $p < 0.05$ when compared to BMP-2 treated cultures).

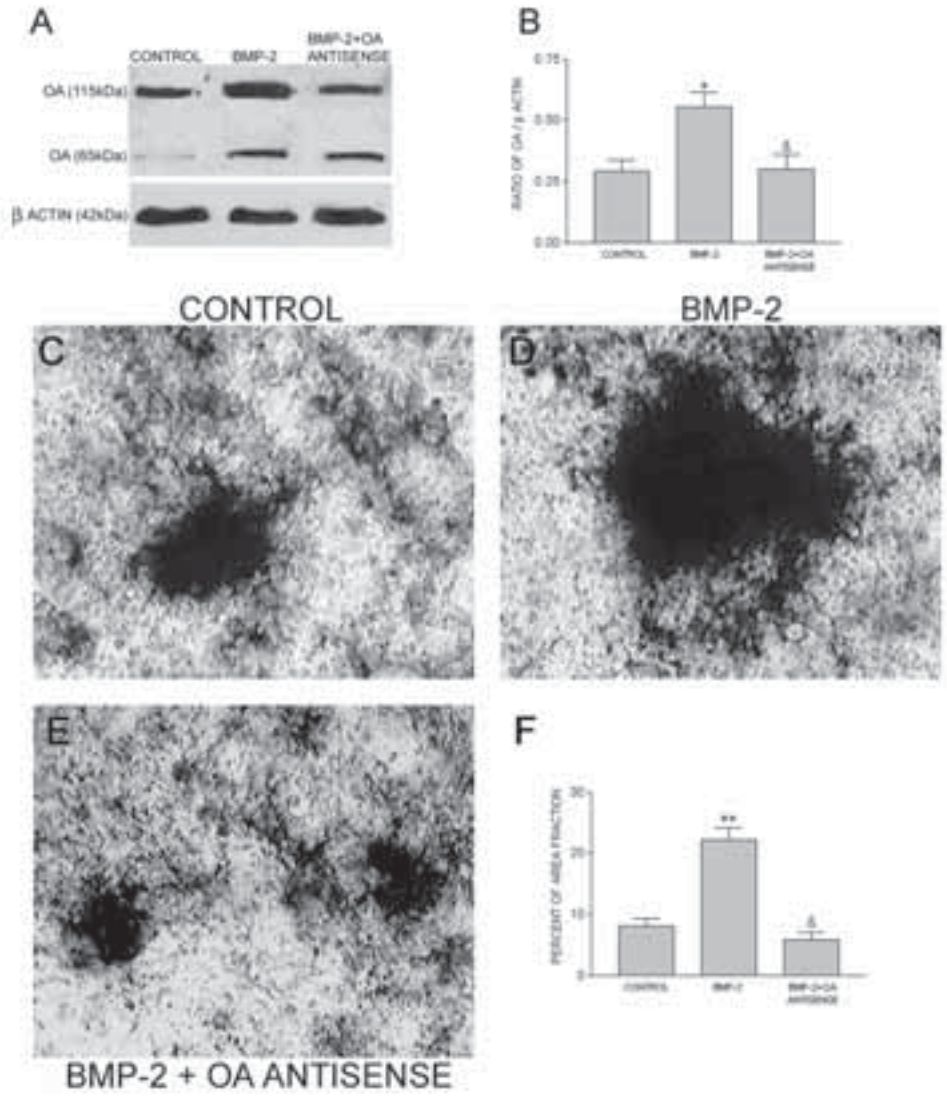

Figure 6. Down-regulation of OA Expression Inhibits BMP-2-Induced Matrix Mineralization. Primary osteoblasts were cultured, treated with BMP-2 alone or transfected with OA antisense (0.5 μM) and treated with a second dose of OA antisense at day 14 in culture, before switching to serum free media with 50 ng/ml BMP-2 for 24 hours prior termination and von Kossa staining at day 21. (A) Immunoblot shows upregulation of OA expression by BMP-2 treatment. Treatment with BMP-2 + OA antisense resulted in inhibition of this up-regulation to levels comparable to OA sense transfected controls. β-actin was used as a loading control. (B) Densitometry of 3 independent immunoblots quantifying percent of the glycosylated OA isoform expression as a ratio of β-actin. Data presented as mean + SEM (* = $p < 0.05$ when compared to OA sense transfected controls, & = $p < 0.05$ when compared to BMP-2 treated cultures). (C-E) Photomicrographs of von kossa staining (black). BMP-2 treatment (D) shows intense staining for minerals compared to OA sense transfected controls (C). OA transfected cells treated with BMP-2 shows less von kossa staining levels compared to BMP-2 treated cultures and similar to OA sense transfected cultures (E). Low power photomicrograph magnification: 60x. (F) Bioquant analysis of 3 independent experiments quantifying percent area fraction of the field occupied by von Kossa staining, an indicator of mineralization. Data presented as mean + SEM (** = $p < 0.01$ when compared to sense transfected controls, & = $p < 0.01$ when compared to BMP-2 treated cultures).

Discussion

As previously described, OA is homologous to other family members of trans-membrane proteins such as GPNMB,[2] DC-HIL,[3] PMEL17 and HGFIN (human growth factor inducible neurokinin).[21] These family members play a role in differentiation of multiple cell types, such as DC-HIL in dendritic cells,[3] PMEL17 in melanocytes[22] and HGFIN in differentiation of lymphohematopoietic stem cells.[23] In this report we examined the regulation of OA expression by BMP-2 during osteoblast differentiation and whether OA modulates BMP-2 effects on early and terminal osteoblast differentiation. The stages of osteoblast development have been well characterized in numerous previous studies. Primary osteoblasts *in vitro* undergo three distinct stages beginning with cell proliferation (days 0–7), followed by nodule formation, collagen deposition and matrix maturation (days 7–14), and ending with osteoblast differentiation and matrix mineralization (days 14–21).[19, 20] The fact that OA and BMP-2 have similar pattern of expression during osteoblast development in culture (see Fig. 1 and 2) and both of these factors have been shown to exhibit an overlapping effects in regulating osteoblast differentiation and function.[7, 11] Data presented in this report suggest a relationship between OA and BMP-2 in regulating osteoblast function.

The OA protein has two isoforms, one is secreted (glycosylated at 115kDa) and one is transmembrane (native at 65kDa).[1] As primary osteoblasts develop in culture, the secreted isoform of OA reaches its highest level during the terminal differentiation of osteoblasts, while the trans-membrane isoform reaches its lowest levels during terminal differentiation (3 weeks in culture). In support of our findings, another group reported similar findings for DC-HIL, the mouse ortholog of osteoactivin.[3] They showed that in SX52, a long-term mouse dentritic cell line, DC-HIL is detected in both the cytosolic fraction that represents the secreted isoform and the membranous fraction that represents the transmembrane isoform. They also showed that the transmembrane isoform of DC-HIL mediates the adhesion of SVEC, mouse vascular endothelial cell line. Our group has previously shown that neutralizing the secreted isoform of OA using an anti-osteoactivin antibody inhibited osteoblast differentiation as evidenced by decreasing alkaline phosphatase activity, nodule formation, osteocalcin production and matrix mineralization.[7] Thus, these findings indicate possible dual roles for both isoforms of OA during osteoblast differentiation, an adhesion role for the transmembrane isoform and a differentiation role for the transmembrane isoform. However more experiments are warranted to explore these possibilities.

The results in this study also showed that expression of both isoforms of OA are upregulated by BMP-2 in a dose-dependent manner, reaching maximum levels at 50 ng/ml of BMP-2. The fact that BMP- 2 up-regulates the expression of OA isoforms during the different stages of osteoblast development in culture suggests that OA may play a role as a downstream mediator of BMP-2 during osteoblast development *in vitro.* Other factors have been reported to be regulated by BMP-2 and are a key regulators of osteoblast differentiation including, BIG-3,[24] Runx-2[25] and Osterix.[26] It has been well documented that BMP stimulation of osteoblast cell differentiation is mediated by heterotetrameric serine/threonine kinase receptors and the downstream transcription factors Smad1,-5,-8.[27] We showed here that OA is regulated by BMP-2 and this regulation is mediated through the Smad-1 signaling pathway. Smad1 is an essential intracellular component that is specifically phosphorylated by BMP receptors and translocated into the nucleus upon ligand stimulation.[28] Phosphorylation of Smad1 involves serines in the carboxy-terminal motif. These residues are phosphorylated directly by a BMP type I receptor *in vitro.* Mutation of these carboxy-terminal serines prevents Smad1 association with the related protein, accumulation in the nucleus, and gain of transcriptional activity.[29] Transgenic mice expressing the Smad1 domain, termed Smad1C, show increased skeletal bone mineral density compared to their littermates. Bone histomorphometric analysis of transgenic mouse tibiae showed that Smad1C significantly increases trabecular bone area and length of trabecular surface covered with osteoid, and up-regulates several osteoblast-related genes in cultured osteoblasts derived from Smad1C transgenic mouse.[30] Targeted deletion of the Smad1 gene results in early embryonic lethality due to failure of the allantois to fuse to the chorion.[31, 32] In conclusion, our results demonstrate that BMP-2 signaling plays an important role in the regulation of OA expression. By close analysis of the OA promoter, multiple Smad1 binding motifs (CAGAC)[33] have been identified. These motifs are located in tandem, 1442 base pairs upstream from the ATG starting codon (data not shown). Further analysis of the OA promoter by generating deletion constructs of the Smad1 binding motifs will clearly demonstrate the regulation of OA expression by BMP-2. Our study indicates that Smad1 inhibition in osteoblast cultures by Smad1 siRNA at a dose of 50 nM inhibited OA expression, a result that suggests regulation of OA expression by BMP-2 is mediated by Smad-1 signaling.

We were also interested to examine whether the effects of BMP-2 on early and late osteoblast differentiation are OA dependent. In order to test this possibility, we used an antisense approach that has been shown previously to be effective in blocking different factors in osteoblast and other cell types (Galindo et al., 2005, Aubin paper 2001). Using an OA antisense oligonucleotides, we were able to inhibit OA expression significantly in cultures terminated at day 14 and 21. We have also shown in a separate study that modulation of OA expression using the antisense approach leads to decreased early osteoblast differentiation associated with decreased nodule formation and ALP activity at day 14 and osteoblast mineralization and osteocalcin expression at day 21.[36] We have also previously shown that neutralizing the constitutively secreted OA protein in primary osteoblast cul-

tures with anti-OA antibody inhibited osteoblast differentiation.[7] In this study, we demonstrated that short-term treatment of osteoblast cultures with BMP-2 under serum-free condition increased osteoblast alkaline phosphatase (ALP) production (day 14), nodule formation and mineralization (day 21). Similar results were reported by Hay and colleagues,[37] where short-term treatment with BMP-2 in human neonatal primary osteoblasts stimulated cell differentiation markers. However, when OA expression was blocked in our cultures, BMP-2-induced early and late markers of differentiation were decreased to levels comparable to control. These data suggest that BMP-2-induced osteoblast function is, at least in part, OA dependent.

The mechanism whereby OA acts downstream of BMP-2 effects on osteoblast function is not fully understood. Our findings suggest that OA production is required for BMP-2-mediated osteoblast maturation and mineralization in primary osteoblast cultures. One possible mechanism is that a downregulation of OA expression inhibits osteoblast differentiation markers indirectly. The inhibition of OA expression could activate some BMP-2 antagonistic/inhibitory regulatory pathways that influence osteoblast differentiation. For example, the soluble BMP-2 antagonists such as, noggin, chordin, chordinlike, cerebrus and gremlin bind BMP-s in the extracellular space and mask receptor binding interfaces for BMP type I and type II receptors.[38, 39] Another alternative mechanism whereby OA acts downstream mediator of BMP-2 effects on osteoblast maturation and terminal differentiation could be explained by the fact that the inhibitory effects of OA antisense oligonucleotides could decrease the expression/phosphorylation of regulatory Smads (1,5 and 8) or increase the expression/phosphorylation of inhibitory Smads (Smad 6 and 7). The latter Smads inhibit signaling by either interacting with phosphorylated BMP type I receptors to prevent activation of receptor-activated Smads,[40–42] or though competition to prevent formation of the receptor-activated Smad/co-Smad complex.[43] Data from our laboratory showed that transfection of primary osteoblasts with OA antisense oligonucleotides showed a dramatic reduction in the amount and the phosphorylation levels of Smad1,5,8 and an increase in the amount and phosphorylation levels of Smad-7 (un-published observations), suggesting that OA acts, at least in part, as downstream mediator of BMP-2 actions on osteoblasts through modulating regulatory (Smad 1,5,8) and inhibitory (Smad 7) signaling molecules (Fig. 7).

Another possibility is that down-regulation of osteoactivin

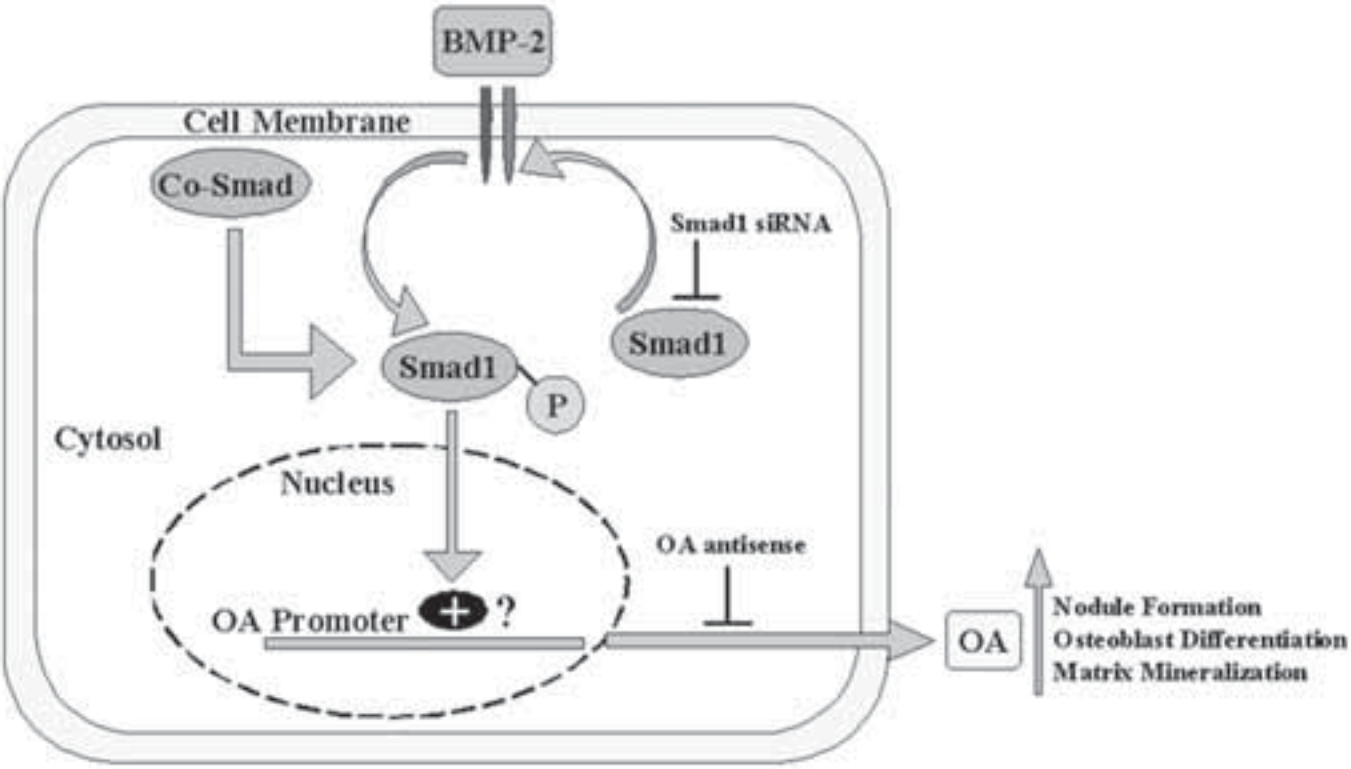

Figure 7. Schematic Diagram of the Relationship between BMP-2 and Osteoactivin in Osteoblasts BMP-2 dimers bind to serine/threonine receptors and induce phosphorylation (P) of Smad1. P-Smad1 binds to Smad4 in multimeric complex then translocates into nucleus. P-Smad1 might activate the transcription of OA gene through binding to Smad1 response elements in the OA promoter. The secreted OA protein induces alkaline phosphatase activity, nodule formation and matrix mineralization. Smad1 siRNA inhibits the expression OA through inhibition of Smad1 expression. OA antisense inhibits OA expression that resulting in blocking BMP-2-induced alkaline phosphatase activity, nodule formation and matrix mineralization.

expression might stimulate other intracellular molecules, such as Smurf1 and Smurf2 (Smad ubiquinitation regulatory factors), which selectively target activated type I receptors and Smad proteins for degradation.[44–46] Several transcription factors, such as Runx-2,[47] and growth factors, such as Wnt3a,[48] modulates at least partially, the effects of BMP-2 on osteoblast differentiation and function. Similar results were presented where connective tissue growth factor (CTGF), a factor that plays a role in osteoblast differentiation *in vitro* and *in vivo.*[19] CTGF expression is regulated by TGF-β and acts as a downstream mediator of TGF-β-induced effects such as matrix production and differentiation of osteoblasts and other cell types.[49, 50]

In this study, we have examined OA expression and its regulation by BMP-2 and have explored regulatory interaction between these two proteins. We also showed that the effects of BMP-2 on ALP activity, nodule formation and matrix mineralization in osteoblasts are partially mediated through OA protein. Further dissection of the relationship between BMP-2 and OA in osteoblasts will lead a better understanding of the role of OA in osteoblast differentiation *in vitro* and bone formation *in vivo.*

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Original Research

Gender Differences in Head-Neck Segment Dynamic Stabilization During Head Acceleration

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Abstract

Purpose: Recent epidemiological research has revealed that gender differences exist in concussion incidence but no study has investigated why females may be at greater risk of concussion. Our purpose was to determine if gender differences existed in head-neck segment kinematic and neuromuscular control variables responses to an external force application with and without neck muscle pre-activation.

Methods: Forty (20 females and 20 males) physically active volunteers participated in the study. The independent variables were gender (female vs. male), force application (known vs. unknown), and force direction (forced flexion vs. forced extension). The dependent variables were kinematic and EMG variables, head-neck segment stiffness (pounds per degrees), and head-neck segment flexor and extensor isometric strength (pounds). Statistical analyses consisted of multiple multivariate and univariate analyses of variance, follow-up univariate analyses of variance and t-tests. Alpha level was set at $p \leq .05$.

Results: Gender differences existed in head-neck segment dynamic stabilization during head angular acceleration. Females exhibited significantly greater head-neck segment peak angular acceleration (50%) and displacement (39%) than males despite initiating muscle activity significantly earlier (SCM only) and using a greater percentage of their maximum head-neck segment muscle activity (79% peak activity and 117% muscle activity area). The head-neck segment angular acceleration differences may be because females exhibited significantly less isometric strength (49%), neck girth (30%), and head mass (43%), resulting in lower levels of head-neck segment stiffness (29%).

Conclusion: For our subject demographic, the results of this study revealed gender differences in head-neck segment dynamic stabilization during head acceleration in response to an external force application. Females exhibited significantly greater head-neck segment peak angular acceleration and displacement than males despite initiating muscle activity earlier (SCM only) and using a greater percentage of their maximum head-neck segment muscle activity.

Introduction

Acute and long-term concussion sequela are produced by acceleration or deceleration of the freely moving head.[10, 12] Based on athlete exposure epidemiological data in the late 1990s, females in high school and college had a higher incidence of concussions compared to their male counterparts (e.g., soccer, baseball and softball, and basketball).[8, 23] The reason for the higher rate of concussions among females in non-contact sports is unknown[18] but may be due to differences in head-neck segment mass, dynamic stabilization (i.e., muscle activity, timing, strength and stiffness qualities) compared to males.[26]

Females have less head-neck segment mass versus males[22] which results in a greater risk of deleterious segment angular acceleration[16] and concussion during standardized force application.[26] Dynamic joint stabilization is defined as the ability of the myotendon unit to absorb external loads and minimize excessive joint movement.[17] Two primary dynamic stabilizers of the head and neck are the sternocleidomastoid (SCM) and trapezius I.[29] The timing and amount of activity of these muscles in response or prior to the application of an external force should reduce the resultant head acceleration.[2, 13, 15, 31] Although there have been no reported gender differences in muscle activity amount during head accelerations, previous authors reported faster neck muscle reflex times in females during low speed rear-end car impacts.[4] Insufficient muscle strength could also predispose individuals to concussion because they would not be able to create the internal (muscle) forces necessary to counter the external forces that result in head acceleration.[16] Head-neck segment muscle strength gender differences have been reported for college-aged volunteers with males having 30 to 40% more isometric cervical flexor and extensor strength.[9] The differences were attributed to neck girth (muscle mass) and not muscle function.

Contraction of these primary stabilizing muscles and greater girth also increases muscle and joint stiffness.[19, 32] Neck muscle contraction prior to external head loading increases resistance to movement[24] and should enhance an athlete's ability to absorb external forces.[2, 28, 32] In a comparison of one female and seven males (18–23 years old), however, Reid et al. (1981) reported that neck muscle resistance to impact was less in the female under various loads and

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conditions (e.g., muscles relaxed, muscles pre-activated).[24] This is consistent with other authors who reported lower stiffness values at other joints in females compared to males.[3, 21, 30]

Although previous studies have identified gender differences in anthropometric, neuromuscular, and strength variables, no research has examined if these differences make females more susceptible to greater head acceleration. The purpose of this study was to determine if gender differences exist in kinematic and dynamic stabilization variables responses to an external force applied to the head.

Methods

Research Design. The study consisted of a three-factor research design with repeated measures. The independent variables were gender (female vs. male), force application (known vs. unknown), and force direction (forced flexion, trapezius I, vs. forced extension, SCM). The dependent variables consisted of anthropometric, kinematic (peak angular acceleration and angular displacement), EMG (muscle activity peak, area, and onset), stiffness, and isometric strength. An external force applicator was used to apply an external force to the head-neck segment (Fig. 1).

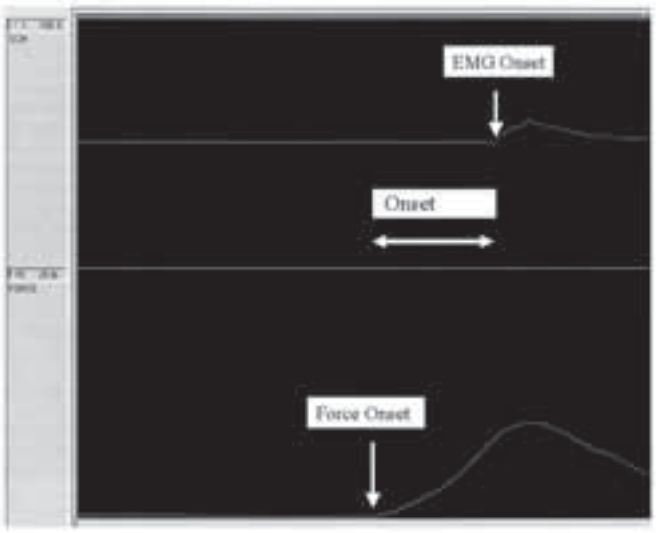

Figure 1. EMG Trace Example. This is an example of a typical EMG trace illustrating SCM onset latency during an unknown force application trial for forced extension.

Participants. Forty physically active males (N = 20, age = 26.3 + 4.3 years, height = 177.1 ± 6.1 cm, mass = 84.5 ± 11.8 kg) and females (N = 20, age = 24.2 ± 4.1 years, height = 165.3 ± 5.3 cm, mass = 59.0 ± 5.1 kg) participated in the study. Physically active was defined as performing a minimum of 30 min of exercise five or more times a week. One female did not participate in the force application trials due to a scheduling conflict. Also, EMG and stiffness data for 3 subjects (2 males and 1 female) were omitted because of improper set-up with the load cell during one day of testing. Potential participants were excluded from the study if they had a history of neurological disorder (e.g., seizures), prior cervical spine or head injury (i.e., concussion), or participated in a neck-strengthening program in the 6 months prior to data collection. Institutional Review Board approval as well as participant written informed consent and consent to videotape were obtained prior to data collection.

Instrumentation — Head-Neck Segment Anthropometric Assessments. Participant height, weight, head-neck segment length, and neck girth were assessed. Participant weight was measured in pounds and converted to body mass (kilograms). Body mass was multiplied by the gender specific head-neck segment to total body mass percentage (male = 8.26% and female = 8.20%) to determine head-neck segment mass.[22] Head-neck segment length was measured with a metric tape measure from the seventh cervical vertebrae spinous process to the top of the head with the participant looking at an object at eye level. Neck girth was measured with a metric tape measure just above the thyroid cartilage. The investigator's intra-tester reliabilities for the anthropometric measurements were intraclass correlation coefficients (ICC; 2,1) of .99 (height), .99 (weight), .98 (head-neck segment length), and .99 (neck girth).

Head-Neck Segment Kinematic Assessment. The PEAK Motus Motion Analysis System (Peak Performance Technologies, Inc., Englewood, CO) was used to collect two-dimensional kinematic data. Video was collected at 60 Hz, and reflective markers were used to aid in the digitizing process. The head-neck and torso segments[11] were used to determine head-neck flexion and extension peak angular displacements (deg) and accelerations (deg/s2). Raw video data were auto-digitized, filtered (fourth order, zero lag Butterworth filter with a 6 Hz cutoff), and analyzed using the PEAK Motus software, version 6.1 (Peak Performance Technology's, Inc., Englewood, CO). Intra-tester measurement reliability of this instrument has been reported to be an ICC of .98.[27]

Head-Neck Segment EMG Assessment. The Noraxon Telemyo System (Noraxon USA, Scottsdale, AZ) was used to assess the EMG activity of the SCM and trapezius I muscles. They were chosen because of their importance as superficial muscles that help to control head-neck flexion and extension and utilization in previous head and neck research.[2, 32] The skin over the right SCM and trapezius I muscles was shaved, lightly abraded, and cleaned with 70% alcohol. Ten-millimeter diameter self-adhesive silver/silver-chloride bipolar surface electrodes (Multi Bio Sensors Inc., El Paso, TX) were placed on the skin 10 mm apart and parallel to the fiber orientation of the underlying muscle. The resistance between the paired electrodes was less than 2 kΩ and verified with a standard digital multi-meter (model 982017, Sears, Roebuck & Company, Hoffman Estates, IL). Placement of the electrodes was identified by palpating the mid-length of the muscle's contractile component during an isometric contraction. A reference electrode was positioned on the skin over the right clavicle. Signals from the muscle

leads were passed to a battery operated 8-channel FM transmitter worn by the participant. The signal was amplified (gain 1,000) with a single-ended amplifier (impedance > 10 MΩ) and filtered with a 4th order Butterworth filter (10 to 500 Hz) and common mode rejection ratio of 130 db at direct current (minimum 85 db across entire frequency of 10 to 500 Hz). An antenna receiver (Antennex, Inc., Glendale, IL) with a 6th order filter (gain 2, total gain 2,000) further amplified the signal. The analog signal was converted to a digital signal by an analog-to-digital converter card (Keithley KPC-MCIA 12A1-C, Keithley Instruments, Inc., Cleveland, OH) and was stored in the MyoResearch Software, version 2.02 (Noraxon USA, Scottsdale, AZ). The raw digital signal (for MVC and trials) was sampled at a rate of 960 Hz, rectified, and smoothed using a root mean square algorithm over a 20 ms moving window. All analyses were performed on processed EMG data during a 250 ms time period after force application. This time period was chosen because pilot data of reactive muscle activity revealed the greatest activity within 250 ms following force application.

Data collected with the EMG equipment were used to determine peak muscle activity (%), muscle activity area (% • ms), and muscle onset latency (ms). Peak muscle activity was defined as the highest amplitude of the smoothed EMG data during one trial. Muscle activity area was defined as the product of the sum of the amplitudes of EMG activity and the total time of the trial (250 ms). The data for the two muscle activities were normalized to a peak value obtained during a MVC for each muscle. Subjects maintained a neutral head position (i.e., head up and eyes facing forward, looking straight ahead) during the maximum voluntary contraction assessment. Muscle onset latency was defined as the time between force application and the first upswing of myoelectric activity from baseline[5] and measured only during the unknown force application trials (Fig. 1). The primary investigator's intra-tester measurement reliabilities for the EMG dependent variables were ICC of .92 (peak activity), .87 (muscle activity area), and .72 (onset latency).

Head-Neck Segment Stiffness Assessment. A tension force load cell (model ELFS-T3, Entran Devices, Inc., Fairfield, NJ) and the Peak Motus Motion Analysis System were used to assess head-neck segment stiffness. Tension force was assessed during the entire trial using the load cell that was inserted in-line with the external force applicator's pulley cord and headgear (Fig. 2). Head-neck segment angular displacement was assessed using the Peak Motus Motion Analysis System. Tension force and head-neck segment displacement data were synchronized during each trial using the time of force application as the event that initiated data collection. The peak force during each trial was used to mark the end of the data collection. This enabled a force-angular displacement line to be created. Head-neck segment stiffness was determined from the slope of the line[3] that was defined as the change in force (pounds) over the change in angular position (degrees)[19] (Fig. 3). The reliability for the amount of force applied using the tension load cell was an ICC of .98 (force application).

Head-Neck Segment Isometric Strength Assessment. The Microfet Hand-Held Dynamometer (Hoggan Health Industries, Inc., West Draper, UT) was used to assess head-neck segment isometric flexor and extensor muscle strength. Flexor strength was assessed with the dynamometer placed in the center of the participant's forehead. Extensor strength was assessed with the dynamometer placed just above the participant's external occipital protuberance. The participant applied maximum force against the dynamometer for 3 s during each trial and rested for 30 s between trials. The primary investigator's intra-tester measurement reliability for this instrument was an ICC of .96.

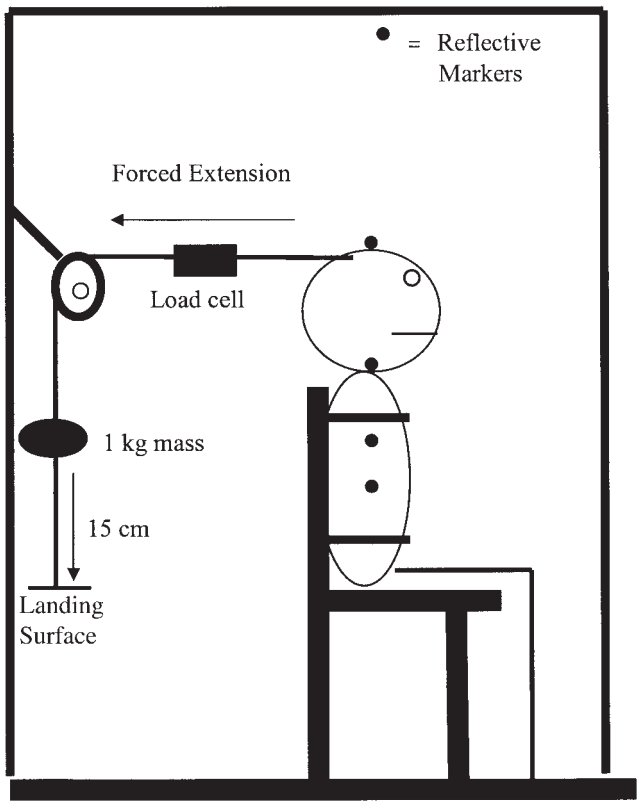

Figure 2. External Force Applicator. The external force applicator was designed and constructed by the investigator. The applicator consists of a metal outer frame, headgear, two cords with plastic stoppers, and two pulleys (only forced extension illustrated). Cords from the front or back of the headgear (Strength Systems Inc., Jefferson, LA) wrap around the pulleys and connect to plastic stoppers (landing surfaces) at the end of the cord opposite the headgear. The plastic stopper enables a 1-kg mass to be dropped from a predetermined height of 15 cm creating a load of approximately 11 lbs (50 N). The load is verified during testing using a tension force load cell (model ELFS-T3, Entran Devices, Inc., Fairfield, NJ). The pulleys in front and back of the seated participant are used to cause head-neck segment flexion and extension, respectively. The heights of the pulleys are modified so that each force is applied at 90 deg to the head-neck segment of the participant during testing. This is verified by visual inspection.

Potential participants met with the primary investigator and the purpose and procedures of the study were explained. Prior to testing, participants performed a neck warm up consisting of neck rotations (15 s clockwise and 15 s counter clockwise) and neck stretching (two repetitions each of 15 s for flexion and extension). After the participants were fitted with the headgear and reflective markers, they were seated within the external force applicator (Fig. 2) and instructed to sit with their head up and eyes facing forward, looking straight ahead. The thorax and pelvis of the participants were positioned against the back of a chair and stabilized with a Velcro (Velcro USA, Manchester, NH) strap to minimize extraneous body movements.[24] Video was also used to ensure no trunk movement during the trials. Participants performed three flexion and extension maximum voluntary isometric contractions. Participants then had their eyes and ears covered with modified goggles and earphones,[24] respectively, to ensure no visual or auditory feedback during testing. The external force applicator's pulley was then attached to the headgear and the 1-kg mass was placed on the landing surface for 10 s to allow participants to become accustomed to the amount of the external load. The 1-kg mass was then dropped 15 cm to apply the external load to the head-neck segment (Fig. 1) causing forced flexion (i.e., trapezius I eccentric tension) or forced extension (i.e., SCM eccentric tension). The amount of force applied was not normalized to bodyweight to simulate real life situations (e.g., soccer heading) where forces applied to the head are not related to bodyweight.

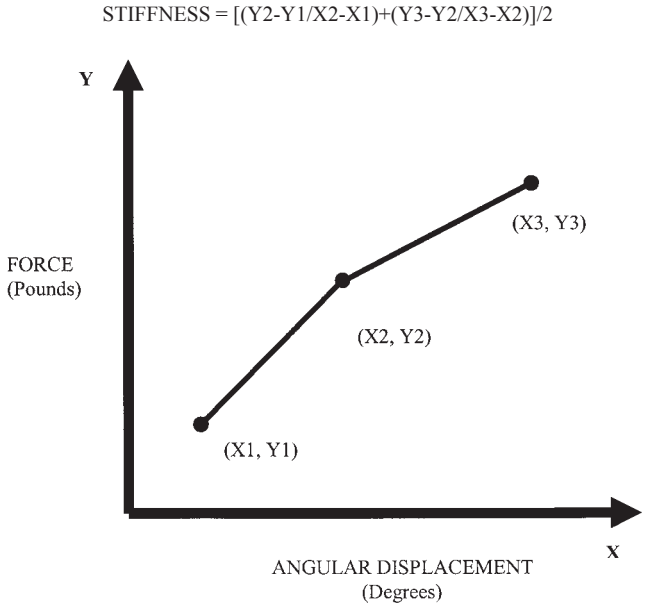

Figure 3. Stiffness Calculation Example. This is an example of a force-displacement line created from a trial with three force (Y1, Y2, and Y3) and three displacement (X1, X2, and X3) data points. These points create two lines and stiffness was determined from the average slope of the lines.

Trials were performed with and without participants' knowledge of force application to simulate sport situations. Three known force application trials occurred first followed by three unknown force application trials for each direction. Direction of force application was randomized. During trials with participants' knowledge, they were instructed to prepare (pre-activate) their neck muscles for the external force application.[24, 31] There was a 3 s count down and then the mass was dropped. During trials without participant knowledge, participants were instructed to relax their neck muscles and then resist as soon as they felt a tug.[24, 31] They were told that the force would be applied at some point during the next 30 s. The duration before the drops was random and ranged from 5 to 25 sec. Also, unknown trials were removed if muscle pre-activation occurred. The average of three trials was used as the criterion measure for all analyses.

Data Analysis. Data were analyzed using descriptive and inferential statistics. Statistical analyses consisted of multiple multivariate and univariate analyses of variance, follow-up univariate analyses of variance and t-tests with Bonferroni correction. Alpha level was set at $p < .05$.

Potential covariates in the kinematic, EMG, head-neck segment stiffness and isometric strength analyses were head-neck segment mass (kilograms) and length (centimeters) as well as neck girth (centimeters). Covariates were correlated a priori with the appropriate dependent variables. A correlation value $r \geq .60$ was used as the criterion for inclusion as a covariate. Neck girth and head mass were correlated ($r > .60$) with head-neck segment isometric flexion and extension strength. They were not statistically significant ($p > .05$) in the ANCOVA model, however, and were therefore not included in the analysis. None of the potential covariates met this criterion for any other dependent variable and were not utilized. The SPSS for Windows, Version 11.5, statistical program (SPSS, Inc., Chicago, IL) was used for data analysis.

Results

Head-Neck Segment Anthropometric Assessments (Table 1). T-tests revealed significant gender differences in head-neck segment mass and neck girth. Females exhibited 43 and 30% less head-neck segment mass and neck girth than males, respectively. There were no significant gender differences in head-neck segment length.

Table 1. Participant Anthropometric Measurements by Gender

	Head-Neck Length (cm)		Head-Neck Mass (kg)		Neck Girth (cm)	
Gender	*M*	*SD*	*M*	*SD*	*M*	*SD*
Male	24.3	1.4	6.9	0.9*	40.7	3.3*
Female	23.9	2.2	4.8	0.4	31.3	2.6

*Note. Significant gender difference ($p \leq .05$). Male $N = 20$ and female $N = 20$. Head-neck segment length ($t(1,38) = -.76$, $p = .453$); head-neck segment mass ($t(1,38) = -9.02$, $p = .000$); and neck girth ($t(1,38) = 10.13$, $p = .000$).

Head-Neck Segment Kinematics (Table 2). A 2 x 2 x 2 MANOVA revealed a significant gender x force application interaction effect. The follow-up ANOVAs revealed a significant interaction for angular acceleration ($F(1,37) = 4.72$, $p = .036$), but not for angular displacement ($F(1,37) = 1.15$, $p = .290$). Post hoc analysis for the two-factor interaction revealed a significant difference ($t(1, 38) = –2.8$, $p = .007$) between the known and unknown force applications for males. Specifically, males exhibited 25% less angular acceleration during the known trials versus the unknown trials. There was no significant difference ($t(1, 36) = –0.28$, $p = .778$) in females between the known and unknown force application conditions. Also, there were significant differences between genders during the known ($t(1, 37) = 5.97$, $p < .001$) and unknown ($t(1, 37) = 3.3$, $p = .001$) force applications. Specifically, females had 70 and 31% more head-neck segment angular acceleration than males during the known and unknown conditions, respectively.

Table 2. Means and Standard Deviations for Head-Neck Segment Kinematic Data

			Angular Acceleration (Deg/s²)		Angular Displacement (Deg)	
Gender	**Knowledge**	**Direction**	***M***	***SD***	***M***	***SD***
Male	Known	Forced Flexion	995.2	368.2	6.3	1.1
		Forced Extension	1103.9	516.5	6.8	1.6
	Unknown	Forced Flexion	1275.2	502.4	10.6	2.5
		Forced Extension	1497.3	602.8	11.0	2.1
Female	Known	Forced Flexion	1503.9	516.5	9.2	1.5
		Forced Extension	2072.1	673.2	9.7	2.4
	Unknown	Forced Flexion	1717.4	607.9	13.8	3.3
		Forced Extension	1868.0	514.7	15.4	2.9

Note. Male $N = 20$ and female $N = 19$. $MANOVA_{gender\ x\ force\ application\ x\ force\ direction}$ $F(2, 36) = 1.79$, $p = .181$; $MANOVA_{gender\ x\ force\ application}$ $F(2, 36) = 3.31$, $p = .048$; $MANOVA_{gender\ x\ force\ direction}$ $F(2, 36) = 1.15$, $p = .234$; $MANOVA_{force\ application\ x\ force\ direction}$ $F(2, 36) = 0.84$, $p = .440$; $MANOVA_{gender}$ $F(2, 36) = 33.6$, $p < .001$; $MANOVA_{force\ application}$ $F(2, 36) = 65.18$, $p < .001$; $MANOVA_{force\ direction}$ $F(2, 36) = 10.35$, $p < .001$

The 2 x 2 x 2 MANOVA also revealed a significant gender main effect and the follow-up ANOVAs indicated a main effect for angular displacement ($F(1,37) = 43.02$, $p < .001$). Results of the gender main effect revealed that head-neck segment angular displacement was 39% greater in females compared to males.

Peak Muscle Activity and Muscle Activity Area (Table 3). A 2 x 2 x 2 MANOVA revealed no significant interaction effects, but did show a significant main effect for gender. The follow-up ANOVAs revealed a significant gender effect for peak muscle activity ($F(1, 34) = 10.22$, $p = .003$) and muscle activity area ($F(1, 34) = 14.28$, $p < .001$) only. Females exhibited 79% more peak muscle activity and 117% more muscle activity area compared to males.

Table 3. Means and Standard Deviations for Head-Neck Segment Muscle Activity

			Peak (%)		Area (% • ms)	
Gender	**Knowledge**	**Direction**	***M***	***SD***	***M***	***SD***
Male	Known	Forced Flexion	31.6	19.2	3.4	1.9
		Forced Extension	29.2	24.6	3.6	3.3
	Unknown	Forced Flexion	35.4	22.7	3.1	1.9
		Forced Extension	29.2	18.0	2.9	2.2
Female	Known	Forced Flexion	56.9	33.7	6.9	4.8
		Forced Extension	54.9	28.2	7.5	4.2
	Unknown	Forced Flexion	52.8	36.4	6.4	5.1
		Forced Extension	64.3	33.7	7.3	3.9

Note. Male $N = 18$ and female $N = 18$. $MANOVA_{gender\ x\ force\ application\ x\ force\ direction}$ $F(2, 33) = 2.05$, $p = .145$; $MANOVA_{gender\ x\ force\ application}$ $F(2, 33) = 0.04$, $p = .960$; $MANOVA_{gender\ x\ force\ direction}$ $F(2, 33) = 0.60$, p = .556; $MANOVA_{force\ application\ x\ force\ direction}$ $F(2, 33) = 0.74$, $p = .486$; $MANOVA_{gender}$ $F(2, 33) = 7.37$, $p = .002$; $MANOVA_{force\ application}$ $F(2, 33) = 4.88$, $p = .014$; $MANOVA_{force\ direction}$ $F(2, 33) = 2.76$, $p = .078$.

Muscle Onset Latency (Table 4). A 2 x 2 ANOVA revealed a significant gender x force direction interaction. Post hoc independent t-tests indicated a significant difference ($t(1, 36) = –3.32$, $p = .002$) between genders during forced extension but not during forced flexion ($t(1, 36) = .692$, $p = .494$). Muscle onset latency was 29% faster for females versus males for the Sternocleidomastiod and only 9% faster in the trapezius.

Table 4. Means and Standard Deviations for Muscle Onset Latency

		Muscle Onset Latency (ms)	
Gender	**Force Direction**	***M***	***SD***
Male	Forced Flexion	42.0	17.6
	Forced Extension	47.8	16.1
Female	Forced Flexion	38.2	15.5
	Forced Extension	33.8	8.5

Note. Male $N = 18$ and female $N = 18$. $ANOVA_{gender\ x\ force\ direction}$ $F(1,34) = 4.86$, $p = .034$; $ANOVA_{gender}$ $F(1, 34) = 4.66$, $p = .038$; $ANOVA_{force\ direction}$ $F(1, 34) = .017$, $p = .896$.

Head-Neck Segment Stiffness (Table 5). A 2 x 2 x 2 ANOVA revealed a significant main effect for gender. The significant gender main effect indicated that the females exhibited 29% less stiffness than the males.

Table 5. Means and Standard Deviations for Head-Neck Segment Stiffness

Gender	Knowledge	Direction	Stiffness (lbs/deg) *M*	*SD*
Male	Known	Forced Flexion	1.29	.39
		Forced Extension	1.75	.55
	Unknown	Forced Flexion	1.34	.66
		Forced Extension	1.26	.40
Female	Known	Forced Flexion	.92	.43
		Forced Extension	1.21	.55
	Unknown	Forced Flexion	.88	.47
		Forced Extension	1.00	.48

Note. Male $N = 18$ and female $N = 18$. $ANOVA_{\text{gender x force application x force direction}}$ $F(1, 34) = 1.72$, $p = .198$; ANOVAgender x force application $F(1, 34) = 0.48$, $p = .493$; $ANOVA_{\text{gender x force direction}}$ $F(1, 34) = .931$, $p = .931$; $ANOVA_{\text{force application x force direction}}$ $F(1, 34) = 7.08$, $p = .012$, $ANOVA_{\text{gender}}$ $F(1, 34) = 12.85$, $p = .001$; $ANOVA_{\text{force application}}$ $F(1, 34) = 6.50$, $p = .015$; $ANOVA_{\text{force direction}}$ $F(1, 34) = 6.59$, $p = .015$.

Isometric Head-Neck Segment Muscle Strength (Table 6). A 2 x 2 ANOVA revealed significant main effects for gender. Females exhibited 49% less isometric neck muscle strength than males. Participants exhibited 30% more isometric strength during forced flexion than during forced extension.

Table 6. Means and Standard Deviations for Head-Neck Segment Flexor and Extensor Isometric Strength

Gender	Force Direction	Isometric Strength (lbs) *M*	*SD*
Male	Forced Flexion	57.4	11.7
	Forced Extension	42.7	7.3
Female	Forced Flexion	31.2	7.5
	Forced Extension	19.7	4.9

Note. Forced flexion = extensor strength and forced extension = flexor strength. Male $N = 20$ and female N = 20. $ANOVA_{\text{gender x force direction}}$ $F(1, 38) = 1.91$, $p = .174$; $ANOVA_{\text{gender}}$ $F(1,38) = 114.78$, $p < .001$; $ANOVA_{\text{force direction}}$ $F(1,38) = 115.8$, $p < .001$.

Discussion

The results of this study revealed gender differences in head-neck segment dynamic stabilization during head acceleration in response to an external force application. Females exhibited significantly greater head-neck segment peak angular acceleration and displacement than males despite initiating muscle activity earlier (SCM only) and using a greater percentage of their maximum head-neck segment muscle activity. In addition, knowledge of force application had no effect in limiting head-neck segment angular acceleration for females, whereas, it resulted in a significant reduction for males. These findings are similar to that of previous research involving low speed rear-end car collisions[15] and indicate gender differences in the ability of physically active individuals to use their dynamic stabilizers for protection against head injury. The reason for the greater head-neck segment angular acceleration in females may be related to their lower levels of strength, neck girth, and head mass, resulting in less head-neck segment stiffness compared to males.

Females exhibited 29% less stiffness and almost 50% less isometric strength than males, regardless of force direction. Previous researchers reported similar stiffness[24] and isometric strength[9, 25] gender differences which they attributed to the amount of muscle tissue. In the present study females had 23% less neck girth than males. Because more tissue correlates positively with greater joint resistance to motion,[6] females should perform head-neck segment resistance training to increase neck girth.

In the present study, female head-neck segment mass was 43% less than males and may have contributed to the significant gender differences in acceleration. Risk of concussion is directly related to the amount of head acceleration (linear and angular) during force application.[10] Following Newton's Law of Acceleration, for a given force application less head mass correlates with greater head acceleration[16] and risk of concussion.[1, 26] Schneider and Zernicke[26] reported an increased risk of head injury during soccer headings for individuals with a lower head mass to soccer ball mass ratio (i.e., women and children).

Force application knowledge trials were not randomized in this study (known then unknown trials) in order to enhance protection for the subjects during this novel task involving the head and neck. This design could have caused a learning effect allowing subjects to be more prepared for the unknown force application trials. We believe this effect was reduced because any unknown trials with muscle pre-activity were not included in any analysis. Also, this design elicited a significant gender by force application knowledge interaction for angular acceleration as well as main effects (EMG and stiffness not discussed).

The gender differences in kinematics cannot be attributed to improper muscle function. The EMG data indicated that females had significantly muscle activity than males regardless of force application or force direction. Since EMG data were collected over a 250 ms window beyond initiation of force application, this indicates a combination of greater reflex and voluntary muscle activities.[13] Greater head angular acceleration and displacement should stimulate a higher percentage of mechanoreceptors in the muscular (i.e., muscle spindle) and articular tissues (i.e., paciniform corpuscles) of the cervical spine leading to a greater activation of motor neurons and more reflex muscle firing.[14]

Females also had a significantly faster onset latency time in the sternocleidomastiod than males during the unknown

condition. This result is similar to previous research[4] and may be related to the greater acceleration values for females during forced extension. The unknown forced extension trials for females elicited the greatest peak angular accelerations. Greater angular acceleration indicates a faster initial movement of the head after force application. Head movement is monitored by the vestibular apparatus of the inner ear[13] and causes a vestibulocollic reflex to the appropriate stabilizing head-neck segment muscles (e.g., SCM). The present study results indicate that the gender and condition with the greatest amount of head-neck angular acceleration yielded the fastest muscle reflex.

Brault et al.[4] reported significantly faster SCM muscle onset latency time in females compared to males during low speed rear-end collision testing, but no gender differences in peak SCM muscle activity. The present study's results indicated faster SCM onset latency times and a larger amount of muscle activity for females. The difference in results of the two studies may be related to method of force application. Our subjects had to stabilize their heads with an added load applied directly to it, whereas the previous studies subjects needed only to control their own head mass. Therefore, time of muscle firing would be similar, yet more muscle activity would be required of our subjects.

The muscle onset latency times (SCM reflex times of 40 ms) were longer compared to those of previous research.[13] The differences are attributed to the method of assessing the reflex. Ito et al.[13] reported SCM reflex times of 24 ms but tested participants positioned supine with their heads supported in a sling and their neck muscles relaxed. Their heads were then released causing an immediate acceleration due to gravity. In the present study, force was applied via a pulley attached to headgear that was worn by participants seated in a chair. The time from force application to head movement, when loading of the pulley occurred, would have resulted in the longer muscle onset latency time.

The amount of force used (and resultant angular accelerations elicited) in this study was by design lower than what is thought to cause a concussion.[10] However, with the relatively low applied force gender differences in head-neck segment angular acceleration were still evoked. These differences may be because females exhibited significantly less head-neck segment isometric strength, neck girth, head mass, resulting in lower levels of stiffness. This is important because angular acceleration is directly related to concussion.[10] Although these subjects were not athletes the results suggest that females may be at greater risk of concussion in sports with greater loads being applied to the head (e.g., soccer, >180N).[2] Future research should examine other populations and the effect of head-neck resistance training on kinematic and EMG responses to force application for the purpose of concussion prevention.

Conclusion

For our subject demographic, the results of this study revealed gender differences in head-neck segment dynamic stabilization during head acceleration in response to an external force application. Females exhibited significantly greater head-neck segment peak angular acceleration and displacement than males despite initiating muscle activity earlier (SCM only) and using a greater percentage of their maximum head-neck segment muscle activity.

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We would like to thank Jack Reed for his help in the construction of the external force applicator.

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Kappa Delta Paper

The Pathomechanics, Pathophysiology, and Prevention of Reversible and Irreversible Cervical Spinal Cord Injury: Results of a Thirty-Year Clinical Experience

Recipient of the 2004 Orthopaedic Research Society Kappa Delta Award

JOSEPH S. TORG, MD, HELENE PAVLOV, MD, ALBERT BURSTEIN, PHD, LAWRENCE THIBAULT, SCD

Athletic traumas to the cervical spine resulting in cord injury are infrequent but potentially catastrophic events. Recognition of the problems associated with such injuries has led to a series of field, clinical and basic research studies conducted over the past thirty years that have answered basic questions regarding the pathomechanics and pathophysiology of cervical cord injury. The clinical relevance of this experience is three-fold. First, identification of axial energy inputs resulting in failure and buckling of a segmented column, a previously unappreciated mechanism, has been translated into injury prevention measures. Second, clarification of both the pathomechanics and pathophysiology of cervical spine and cord trauma resulting from athletic activity has provided guidelines to assist clinical management decisions. Third, correlation of the clinical manifestations cord trauma to with deformation of in vitro axonal injury model both explains both the variable response to injury and supports the case for spinal cord resuscitation.

Mechanisms of Injury

Injuries resulting in spinal cord trauma have been associated with football, water sports, gymnastics, wrestling, rugby, trampolining, and ice hockey. Traditionally, hyperflexion and hyperextension have been implicated as the primary mechanisms in cervical spine injuries based on post-injury radiograph interpretation. In 1972, Schneider[1] reported a series of cervical spine injuries occurring in tackle football that he attributed to striking of the head with a knee, acute cervical hyperextension, tackling by the face guard, forced hyperflexion, and head butting. He and others[2–23] concluded that the most serious cervical injuries in football and other sports occurred as a result of forced hyperflexion. Hyperextension has also received attention as a mechanism leading to cervical spinal cord injury.[24–29] While some authors recognized axial loading as a possible mechanism for cervical athletic injuries,[30–41] it was not generally accepted as the predominant mechanism in cervical spine injury producing cord damage prior to 1975.

Axial Loading

The National Football Head and Neck Injury Registry (NFHNIR), established in 1975, collected data on over 1,300 cervical spine injuries.[42–44] The criteria for inclusion in the Registry were injuries requiring hospitalization for greater than seventy-two hours; those that required operations; and fractures, subluxations, or dislocations resulting in neurologic injury or death. Data was collected from the athlete, parent, and school officials, radiographs, medical records, and when available, analysis of game films or videotapes. The total number of head and neck injuries was calculated retrospectively from 1971 to 1975 and compared with the data compiled by Schneider[45] in a similar study during the period 1959 to 1963. The results indicated both intracranial hemorrhages and deaths due to intracranial injuries had decreased by 66 percent and 42 percent, respectively, while the number of cervical spine fractures, subluxations, and dislocations had increased 204 percent, and the number of cases of cervical quadriplegia had increased 116 percent. The majority of the permanent cervical quadriplegias occurring between 1971 and 1975 were determined to be due to so-called "spearing" or direct compression when the player had made initial contact with the top of his helmet (Fig. 1).

Figure 1. A college defensive back (dark jersey) is shown ramming an opposing ball carrier with his head, resulting in severe axial loading of his cervical spine. The defensive player suffered fractures of C4, C5, and C6 and was rendered quadriplegic. (From *Am. J. Sports Med.,* 21:640–649, 1993.)

Documentation of axial loading as the responsible mechanism of injury in the production of catastrophic football cervical spine injuries was obtained from stop-frame kinetic analysis of sixty game films and video tapes of actual injuries resulting in permanent quadriplegia.[44] The mechanism of injury was determined in 85 percent of the cases, and in all instances, it was axial loading.

Based on these findings, it was concluded that the improved protective capabilities of modern helmets accounted for the decrease in head injuries, however, it led to the development of playing techniques that used the top or crown of the helmet as the initial point of contact, placing the cervical spine at risk.

Modification of Playing Techniques

In the course of a contact activity, such as tackle football, the cervical spine is repeatedly exposed to potentially injurious energy inputs.[46] Fortunately, most energy inputs are dissipated by controlled spinal motion through the cervical paravertebral muscles and the intervertebral discs.[47] With the neck in a neutral position, the cervical spine is actually extended due to the normal cervical lordosis. When the neck is flexed to thirty degrees, the cervical spine becomes straight. In this situation, the cervical spine assumes the physical characteristics of a segmented column, motion is precluded in response to axially directed impacts, and the forces are directly transmitted to the spinal structures. This results in the cervical spine being compressed between the abruptly decelerated head and the force of the oncoming trunk.[48] When the maximum vertical compression is reached, the cervical spine fails and buckles in a flexion mode with fracture, subluxation, or facet(s) dislocation occurring (Fig. 2). This observation is consistent with the accepted mechanical engineering principles of elastic instability and buckling or failure of a segmented column.

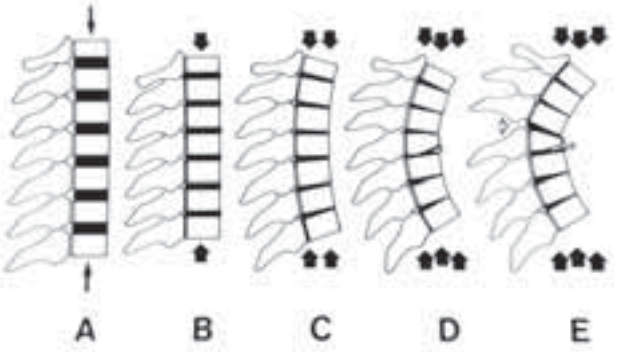


Figure 2. Biomechanically, the straightened cervical spine responds to axial loading forces like a segmented column. (A and B) axial loading of the cervical spine first results in compressive deformation of the intervertebral disks. (C) As the energy input continues and maximum compressive deformation is reached, angular deformation and buckling occur. (D and E) The spine fails in a flexion mode, with resulting fracture, subluxation, or dislocation. Compressive deformation leading to failure, with a resultant fracture, dislocation, or subluxation occurs in as little as 8.4 msec. (From *Am. J. Sports Med.,* 18:50–57, 1990.)

In 1976, in response to these reports, the National Collegiate Athletic Association (NCAA) banned "spearing" (intentionally striking an opponent with the crown of the helmet) and tackling techniques using the helmet as the initial point of contact. Similar rules changes were also enacted at the high school level.[42, 43, 48] As a result of these changes, fractures, subluxations, and dislocations of the cervical spine declined dramatically between 1976 and 1987. In 1976 the injury rates for these conditions were 7.72/100,000 and 30.66/100,000 for high school and college athletes, respectively; they decreased to 2.31/100,000 and 10.66/100,000, respectively, by 1987. Cervical spine injuries resulting in quadriplegia consistently declined. In 1976, the injury rate for quadriplegia was 2.24/100,000 at the high school level and 10.66/100,000 at the college level. In 1977, one year following the rule changes the injury rate for quadriplegia decreased to 1.30/100,000, and 2.66/100,000, for high school and college athletes, respectively. By 1984, the injury rates had decreased to 0.40/100,000 for high schools, and to 0/100,000 for colleges (Fig. 3). Numerous biomechanical studies subsequently further supported the axial loading theory.[50–58]

Yearly Incidence of Permanent Quadriplegia Due to Football, 1975–1995

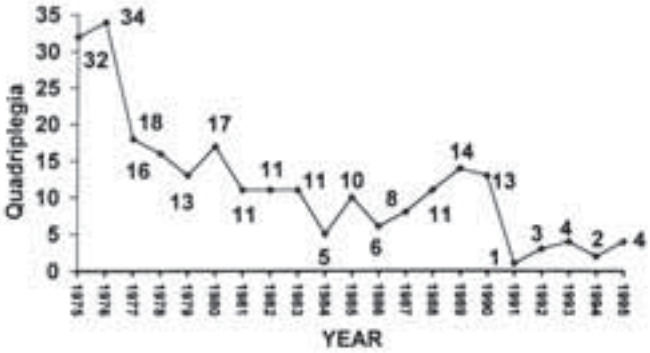


Figure 3. The effect of the 1976 rule changes banning spearing and head impact playing techniques was dramatic, and a sustained decrease in the occurrence of permanent cervical quadriplegia occurred. (From *J. Bone Joint Surg.,* 84-A:112–122, 2002.)

Pathomechanics/Pathophysiology of Cervical Spine Cord Trauma and Guide to Management

Injuries at the C3-C4 Level

Injuries at the C3-C4 level are rare and infrequently reported.[24, 59, 60] Injuries at this level accounted for only twenty-five (2.4 percent) of the NFHNIR documented 1,062 injuries, occurring between 1971 to 1988.[61, 62] Axial loading was again the predominant injury mechanism. The specific injuries were acute intervertebral disc herniation, anterior subluxation, unilateral and, bilateral facet dislocation, and C4 vertebral fracture.

Injuries to the middle cervical segment are unique in that these lesions generally do not involve fracture and more

favorable results were observed with prompt reduction of unilateral and bilateral facet dislocations. In two cases of unilateral facet dislocation reduced within three hours of injury, marked neurologic recovery occurred compared with the remaining patients, treated with delayed open reduction or closed skeletal traction who remained quadriplegic. In four in whom a bilateral facet dislocation was reduced successfully with either closed or open methods had no neurologic recovery, but all four patients survived. The three patients who did not have a successful reduction died.[59, 60]

The Axial Load Teardrop Fracture

Schneider and Kahn[63] were the first to describe a triangular fracture fragment at the antero-inferior corner of a cervical vertebral body as a *teardrop* fracture. Their description was made on the basis of analysis of lateral roentgenograms and concluded that the fracture was caused by acute flexion. The terms *acute flexion* and *teardrop*[39] have been recognized and accepted as the terms for vertebral body fractures with an antero-inferior corner fracture fragment. Others have described these fractures as burst[64] or compression fractures, or have used the terms *flexion teardrop*[65] and *burst* interchangeably. Because of the inconsistency in both terminology and injury mechanism, the neurologic sequelae of each of the fracture patterns had not been clarified.

Utilizing data from the NFHNIR fifty-five patients with fifty-eight *teardrop fractures* of C4, 5 and 6 were analyzed.[69] In fifty-one of the fifty-five patients, axial compression was determined to be the mechanism of injury. Radiographically, only six patients had an isolated antero-inferior vertebral body fracture; in forty-nine patients, there was in addition, a sagittal vertebral body fracture and fractures through the lamina. In this series, five of the six patients with an isolated antero-inferior corner fracture had no serious neurologic sequelae; one patient had posterior element fractures of the subjacent vertebra and was quadriplegic. Of the forty-nine patients with a documented three-part, two-plane injury, forty-four (90 percent) were quadriplegic.

These results identified two fracture patterns associated with the antero-inferior corner fracture (teardrop) fragment; the isolated fracture that is not associated with permanent neurologic sequelae, and the three-part, two-plane fracture in which there is a sagittal vertebral body fracture and fracture of the posterior neural arch, are usually associated with permanent neurologic sequelae. The mechanism of injury for both fracture patterns is axial loading.

Spear Tackler's Spine

Spear tackler's spine is a clinical entity that constitutes an absolute contraindication to participation in tackle football and other collision activities that expose the cervical spine to axial energy inputs.[70] A subset of football players were identified who demonstrated: 1) developmental narrowing of the cervical canal (canal-vertebral body ratio < 0.8); 2) straightening or reversal of the normal cervical lordosis; 3) post-traumatic radiographic abnormalities; and 4) video documentation of using spear tackling techniques; and 5) a history of cervical cord, nerve root or plexus neurapraxia.

Fifteen cases of spear tackler's spine were identified by the NFHNIR between 1987–1990 meeting these criteria.[70] Eleven had complete neurologic recovery from their injuries and four patients had permanent neurologic deficits. Permanent neurologic injury occurred as the result of axial loading of a persistently straightened cervical spine from use of head-impact playing techniques. On the basis of these observations, it was concluded that individuals who possess the aforementioned characteristics of spear tackler's spine be precluded from participation in activities that expose the cervical spine to axially directed energy inputs.

Cervical Cord Neurapraxia (CCN)

We have described neurapraxia of the cervical spinal cord with transient quadriplegia from both NFHNIR and clinical practice data.[71–73] The prevalence of cervical cord neurapraxia has been estimated at 7 per 10,000 football participants. It involves an athlete who sustains an acute transient neurologic episode of cervical spinal cord origin associated with sensory changes of burning pain, numbness, tingling or loss of sensation with or without motor changes of weakness or complete paralysis. The episode is transient, with complete recovery usually occurring in ten to fifteen minutes, although recovery may take up to two days. The cervical area is pain free at the time of injury and there is complete return of motor function and full range of motion.

In athletes with diminution of the anteroposterior diameter of the spinal canal, the cord can, on forced hyperextension or hyperflexion, be compressed causing transient motor and sensory manifestations described as the "pincer mechanisms" by Penning.[74]

To determine which athletes had a decreased anteroposterior diameter of the spinal canal, an objective measurement was devised comparing the spinal canal sagittal diameter to that of the vertebral body on the lateral radiograph[75] (Fig. 4). There is normally a one-to-one relationship. A spinal canal-vertebral body ratio of 0.80 or less was recorded at one or more levels in all patients who experienced cervical cord neurapraxia.[72]

To be noted, Herzog et al.[76] studying a cohort of professional football players, pointed out that although the canal-vertebral body ratio has a high sensitivity for detecting cervical spinal stenosis, it has a poor positive predictive value.

Questions regarding both the relationship of cervical sagittal spinal canal size and injury as well as the reliability of the canal-vertebral body ratio as an indicator of stenosis arose.[77, 78]

Matsuura et al.[79] comparing computerized tomographic parameters in the cervical spine of cord-injured patients with those of normal controls concluded that the intrinsic dimensions and shape of the cervical spinal canal may contribute a predisposition to cord injury.

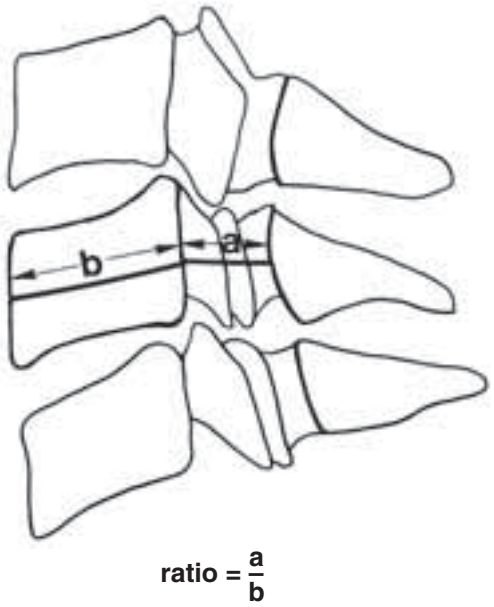


Figure 4. The spinal canal/vertebral body ratio is the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the corresponding spinolaminar line (A) divided by the anteroposterior width of the vertebral body (B). (From *J. Bone Joint Surg.,* 68-A:1354–1370, 1986.)

To address the issues of canal size and cord injury, and to determine the relationship between a developmentally narrowed cervical spinal canal and reversible and irreversible injury, a classification system and an epidemiologic study was conducted. A classification system of CCN[80] was developed based on clinical, radiographic, and magnetic resonance imaging data. CCN was classified according to the degree of neurologic deficit ranging from complete paralysis to only sensory deficit; graded according to length of neurologic symptoms; and defined by the anatomic distribution of the neurologic symptoms. The epidemiologic study was performed with use of various cohorts of football players as well as a large control group.[80] Cohort I and II — 227 college and ninety-seven professional football players who were asymptomatic without transient cervical neurapraxia. Cohort III — forty-five high school, college, and professional football players who had at least one episode of transient cervical neurapraxia. Cohort IV — seventy-seven high school or college football players who were permanently quadriplegic. Cohort V — 105 control non-athlete male patients without history of cervical spine injury or symptoms. The findings of this study demonstrated that 1) a ratio of 0.80 or less had a high sensitivity (93 percent) for transient neurapraxia 2) none of the seventy-seven quadriplegic individuals (Cohort IV) had had an episode of transient neurapraxia of the spinal cord before the catastrophic injury; none of the forty-five high school, college, and professional players who had had an episode of transient neurapraxia (Cohort III) became quadriplegic despite narrowing of the cervical spinal canal. The data provided evidence that the occurrence of transient cervical neurapraxia and an injury associated with quadriplegia are unrelated (Fig. 5). It was concluded that developmental narrowing of the cervical spinal canal in the absence of instability is neither a harbinger of nor a predisposing factor for permanent neurological injury.

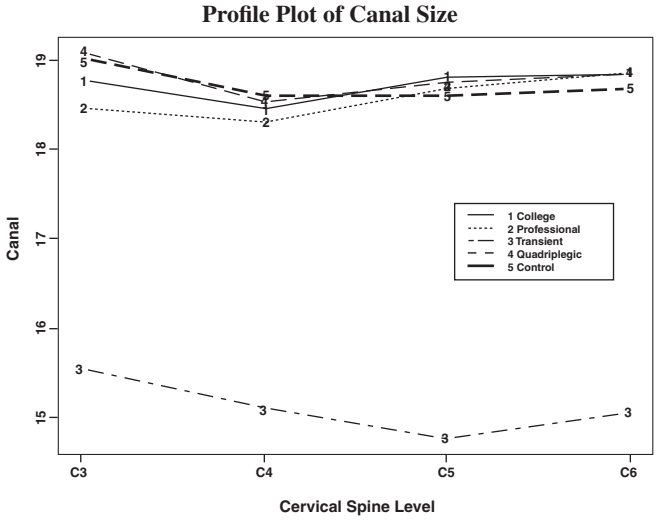


Figure 5. Profile plot of the mean diameter of the spinal canal measured in millimeters demonstrating a significantly smaller value for Cohort III (transient neurapraxia) compared with all the other cohorts ($p < 0.05$). No significant difference was found among Cohorts I, II, IV, V. (From *J. Bone Joint Surg.,* 78-A:1308–1314, 1996.)

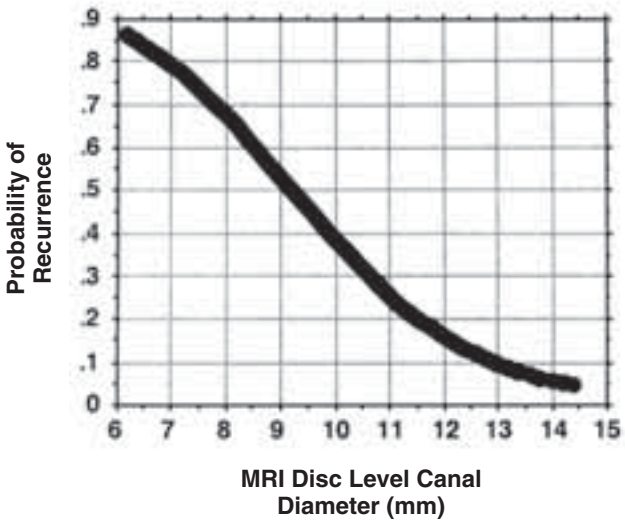
On the basis of available data, it appears that developmental narrowing of the cervical spinal canal without associated instability does not predispose an individual to permanent neurological injury. The major factor in the occurrence of cervical quadriplegia in football is a playing technique in which the head is used as the primary point of contact, with an axial energy input to, and subsequent failure of, the cervical spine. Although a controversial issue and one in which many spine surgeons would disagree, we believe that CCN should not preclude an athlete from participation in contact sports.[81–84]

To analyze the relationship of the spinal cord to the spinal canal, a computerized system was developed to analyze the magnetic resonance images using a graphics digitizer pad with a resolution of 0.01 mm. The disc-level canal diameter was measured as the shortest distance between the intervertebral disc and the bony posterior elements to quantify spondylolytic narrowing. The cord diameter was determined by measuring it's transverse diameter. Graphic plots were constructed using logistic regression analysis of the percentage risk of recurrence versus the disc-level canal diameter and the spinal canal-vertebral body ratio (Fig. 6a & 6b). The overall average recurrence rate for those who returned to football was 56 percent. Specific risk of recurrence is inversely correlated canal size e.g. the smaller the canal, the greater the risk, and is clearly predictable. Individuals who experience uncomplicated CCN are not at risk of incurring

permanent neurological sequelae, rather, the problem is recurrence of subsequent transient episodes.

A clear understanding of the pathomechanics of cervical spine injury combined with a description of those specific injury patterns have aided in attempts to establish criteria for return to activity.[81, 85, 86]

A



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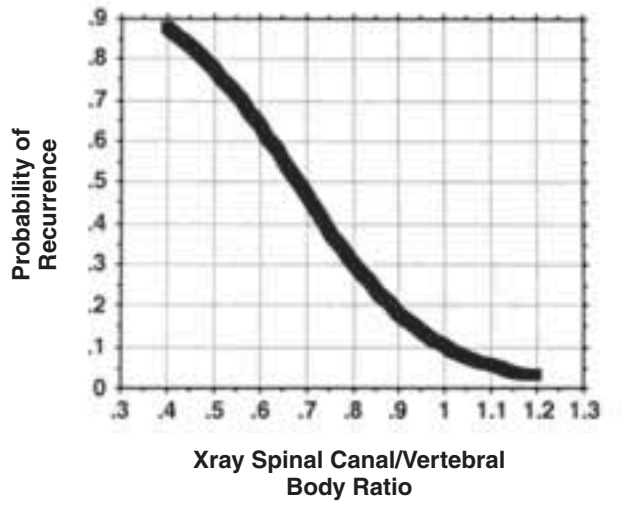

Figure 6. (A and B) Graphs developed using logistic regression analysis in which the risk of recurrence can be plotted as a function of the disc level diameter measured on MRI (upper) and the SC/VB ratio calculated on the basis of a radiograph (lower). The construction of these plots is based on the result that increased risk of recurrence is inversely correlated with canal diameter. Future CCN patients can be counseled regarding their individual risk of recurrence based on the particular size of their spinal canal. (From *J. Neurosurgery,* 87:843–850, 1997.)

Pathophysiology of Cervical Cord Injury as it Relates to the Principles of Cord Resuscitation

Athletic injuries to the cervical spine have resulted in reversible, incompletely reversible, and irreversible neurologic deficits.[59, 60, 70, 72, 87]

A possible explanation for the variable response to injury has been obtained from the study of the histochemical responses of an in vitro axon injury model to mechanical deformation.[88] The spinal cord is considered an element with a low modulus of rigidity in which compressive macroscopic deformations result in local elongation. With axial elongation of the cord, all elements experience stretch. With extension or flexion, the tension in the cord will vary across the diameter. Highly localized loading, such as shearing from subluxation, or focal compression, result in elongation in the direction of the long axis of the cord.

The giant axon of the squid was used as the tissue model to determine the effects of high strain and uniaxial tension to various degrees of stretch in concert with the neurophysiologic changes. The effects of mechanical deformation of the axon membrane leads to an alteration in membrane permeability which allows calcium to flow into the cell and results in membrane depolarization. These experiments demonstrated that the degree of mechanical injury to the axon influences the magnitude of the calcium insult and the time course of the recovery phase. A low rate of deformation produces a small reversible depolarization; the axon responds to the increased intracellular calcium by pumping it extracellularly without residual deficit. As the rate of loading increased, the magnitude of the depolarization and the recovery time to the original resting potential increase in a nonlinear fashion; the axon may or may not fully recover depending on the ability of the cell to pump calcium. With a large influx of calcium, intracellular calcium pumps may be overwhelmed resulting in irreversible injury. The excess intracellular calcium results in accumulation of proteins intracellularly. The resulting increased osmotic pressure causes the cell to swell and eventually rupture (Fig 7). In addition to the immediate and direct effect of mechanical deformation on the cytosolic calcium concentration within the axon, it has been shown that high strain rate elongation of isolated venous specimens elicits a spontaneous constriction. This mechanically induced vasospasm alters blood flow in various regions as a function of the level of vessel stretch. Ultimately, the outcome for the neural tissue will depend synergistically on the level of calcium introduced into the cytosol and the degree to which the metabolic machinery of the cell may be compromised by regional reduction in blood flow.[88] The clinical evidence of varying degrees of recovery to cervical spine injury correlate with the in vitro axon model.

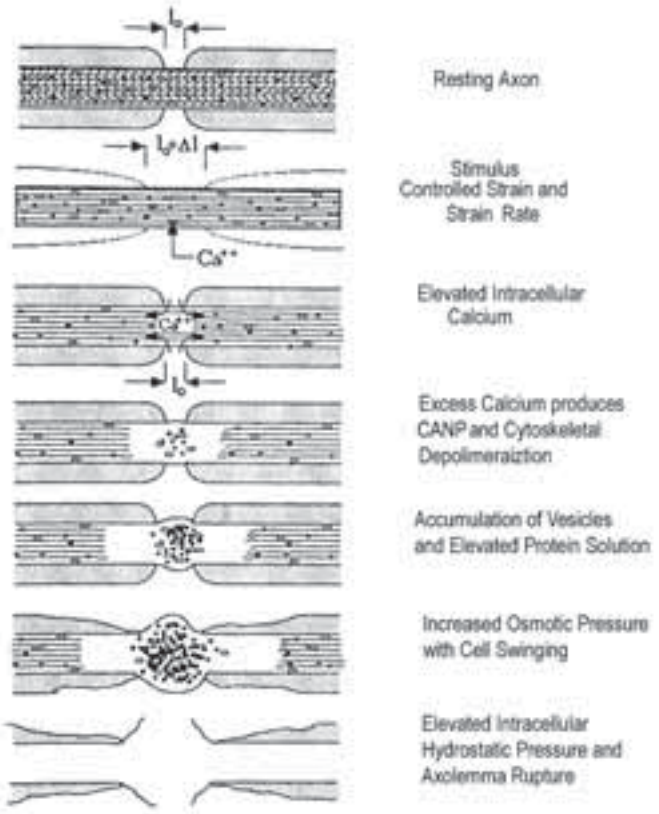

Figure 7. Schematic representation of the effects of elevated intracellular calcium concentration on cell viability. Specifically, elevated cytosolic-free calcium in excess of 50 micromolar will result in calcium activated neutral protease (CANP) that can damage protein structures of the cell. (From *Clin. Orthop. Rel. Res.,* 321:259–269, 1995.)

Clinical Correlation

Cord neurapraxia and transient quadriplegia, a completely reversible cord lesion, are associated with developmental narrowing of the cervical spinal canal. Cord deformation occurs rapidly and is attributable to a hyperflexion or hyperextension mechanism. Disruption of cell membrane permeability leads to a small increase in intracellular calcium, but spinal stability and cell anatomy is not disturbed, and the deleterious effect of local anoxia secondary to venous spasm do not impede recovery of axonal function.

Incomplete cord reversibility is often associated with instability whereby the cord undergoes maximal elastic deformation. It is proposed that lack of full recovery is attributable to prolonged duration of deformity with local anoxia inhibiting cell membrane function and a reduction of intracellular calcium concentrations.

Irreversible cord injury with permanent quadriplegia results from an axial load mechanism, which causes a fracture or dislocation that renders the spine markedly unstable. The cord undergoes functional plastic deformation with anatomic disruption of axonal integrity.

The literature supports the concept that acute spinal cord injury with concomitant subluxation and dislocation should be reduced promptly.[59, 82, 87, 89–92] Similar to irreversible neurologic sequelae following closed head injuries, it is the secondary injury phenomena, cerebral hypoxia and ischemia due to swelling, that is the major problem. It is well established in the neurosurgery literature that the release of excitotoxic substances, cell membrane depolarization, rise in intracellular calcium concentration, and increased intracellular hydrostatic pressure results in increase neuronal pressure and rupture.[83, 88] It is proposed with regard to permanent neurologic sequelae that the same pathophysiologic mechanistic phenomena occurs in acute spinal cord trauma. It is the secondary injury phenomenon to the cord caused by edema, hypoxia, and aberration of cell membrane potential that is largely responsible for resultant neurologic deficit. Admittedly, based, in part, on clinical observations lacking scientific format, the concept of spinal cord resuscitation has been proposed for consideration as an attempt to reverse the secondary injury phenomena to obtain maximum neurologic recovery. Such measures include support of both respiratory and hemodynamic function to facilitate spinal cord perfusion, prompt relief of cord defamation, administration of intravenous corticosteroids as recommended by Bracken,[93] and early spinal stabilization.

Conclusions

The major contributions of this work have been: 1) implementation of an effective, nationally recognized injury prevention program generated from within the orthopaedic community; 2) the attempt to correlate the observed clinical pathomechanics with cellular pathophysiology in instances of cervical spine and cord trauma; and 3) initiate recognition of the importance of appropriate early interventional treatment regimens for catastrophic cervical cord injuries.

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Case Reports

Post-Operative Anterior Cruciate Ligament Allograft Infections: Report of Two Cases

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Introduction

The anterior cruciate ligament (ACL) is commonly injured, with an estimated 95,000 ACL injuries occurring per year with 50,000 resulting reconstructions.[3] While there have been studies describing post-operative complications in ACL reconstructions; perhaps the most devastating is joint infection,[6] and the treatment of these infections is still controversial. Most authors recommend treating the infected joint with intravenous antibiotics, joint irrigation, and graft retention, progressing to graft excision in cases involving resistant organisms within the joint or infection recalcitrant to treatment.[6]

In cases of allograft reconstruction infection, the graft itself must be considered a source of contamination with organisms such as: *Clostridium Sordellii, Pseudomonas aeruginosa, Staphylococcus aureus, Enterococcus faecalis, Citrobacter werkmanii, Citrobacter youngae, Klegsiella oxytoca,* and *Hafnia alvei.*[1, 2] This report will present two cases of ACL allograft reconstructions in which the patients developed post-operative joint infection, two of which involved grafts which tested culture positive intra-operatively.

Unfortunately, infection in allogenic graft reconstruction surgery, while uncommon, still exists. Different techniques to limit the risk of infection, including freeze-dried ethylene oxide-treated allografts, have either failed to reduce the infection rate or led to mechanical compromise of the graft itself.[5, 8] The challenge is to find a new procedure to help reduce the risk of infection without materially damaging the graft's integrity or ability to be remodeled within the knee joint.

Within our institution, we had two allograft infections within a three-month period. We present these cases here.

Case Report One

AS is a 19 year old male who ruptured his ACL while playing basketball in June of 2000. Physical exam revealed a moderate knee effusion, 10 degree extension loss, lateral femoral condyle tenderness, and a positive Lachman's test. MRI confirmed an ACL tear, and the patient was scheduled for allograft bone-patellar-bone reconstruction.

Intra-operatively, a partial medial meniscus tear was debrided, a full thickness lateral meniscal tear was repaired and the ACL was reconstructed with a bone-patellar-bone allograft (CryoLife, Atlanta, Georgia) affixed with a cannulated bio-interference screw (Arthrex, Naples, California). Intraoperative cultures of the graft were negative. Post-operatively, the patient did well until he sustained another injury in September of 2001 while playing basketball. He was scheduled for an arthroscopic ACL revision with allograft and extra-articular augmentation.

Intra-operatively, the patient had a partial lateral meniscus tear debrided and an ACL rupture, which was reconstructed with a bone-patellar-bone allograft (CryoLife) and extra-articular augmentation with an ITB sling. Intra-operative cultures were taken. He was discharged home on post-operative day one with three days of oral cephalexin, analgesia, and in a locked hinged knee brace with weight bearing as tolerated in extension.

The patient returned on post-operative day four with a large hemarthrosis and a temperature of 104 degrees. The final results of the intra-operative cultures were available and demonstrated broadly sensitive *Escherichia coli.* He was admitted and brought urgently to the operating room early on post-operative day five for an arthroscopic irrigation and debridement, and was started on intravenous ciprofloxacin and clindamycin. Intra-operative cultures grew *E. coli.* Four days later, the patient underwent another irrigation and debridement and two small catheters were placed. Antibiotics were continued. Cultures again grew *E. coli.* The patient underwent a third irrigation of the knee and removal of the allograft. The extra-articular ITB sling did not appear to be involved. A PICC line was inserted and the patient was placed on intravenous antibiotics for a total of six weeks. The allograft was sent for culture and grew *E. coli,* pathology demonstrated necrotic fibrous tissue with acute inflammation and bone fragments demonstrating possible osteomyelitis. The patient did well on antibiotics, was symptom free, eventually had a normal CBC and ESR and therefore underwent an autogenic bone-patellar-bone reconstruction of his ACL four months later. The patient went on to full recovery without further complications.

Case Report Two

DD arrived in May of 2001 as a level one trauma having been struck on his motorcycle by a car. His many injuries included multiple brain hemorrhages, scrotal injuries, a right pelvic fracture, and ruptures of his ACL, PCL, and MCL of his right knee without fracture. Angiography of his lower extremity revealed no vascular injury. The patient was placed in an external fixator for his pelvic injury and a knee immobilizer with no weight bearing. The patient was discharged

from the hospital 25 days later to a skilled nursing facility.

The patient was scheduled for surgery three weeks after removal of his pelvic external fixator when the wounds from the pin sites and skin abrasions on his anterior knees had healed. The patient underwent ACL, LCL, and PCL reconstructions with one bone-patellar-bone allograft (Southern Transplant Service, Metairie, LA) and two achilles allografts (Community Blood Center — Community Tissue Center, Philadelphia). The patient tolerated the procedure well and was discharged home on cephalexin for five days. Operative cultures from the grafts and the knee were taken, which subsequently grew methicillin resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas.* Two weeks postoperatively, the patient complained of increasing pain and swelling in his right knee. Aspiration was performed and revealed purulent fluid which again was culture positive for MRSA and *Pseudomonas.*

The patient was urgently taken back to the operating room. Upon opening the portals, purulent fluid was immediately noted. Necrotic tissue was debrided from the PCL, a purulent pocket was was drained from the Anterolateral aspect of the lateral portal, and the LCL allograft was found detached from the fibula. The knee joint and incisions were debrided and irrigated, and three drains were placed. Cultures again grew MRSA and *Pseudomonas aeruginosa;* the patient was placed on vancomycin. The following day, the knee was again debrided and irrigated with antibiotic saline. The ACL and PCL grafts were removed and vancomycin antibiotic beads were placed within the knee. Two drains were inserted. A PICC line was placed and the patient was started on cefepime, minocycline, and rifampin for eight weeks.

The antibiotic beads were removed one week later. During the next two months, the patient finished his intravenous antibiotics, his PICC line was removed and his laboratory values normalized. Five months after the initial reconstruction, the patient underwent reconstruction of his PCL, LCL, and ACL with achilles allografts (Community Blood Center — Community Tissue Services). The patient was given 600 mg of clindamycin prophylactically. Intra-operative cultures were negative. Antibiotics were continued at home for three weeks. The patient's post-operative course was complicated with minor wound dehiscence over the tibial incision which healed with local wound care. At one year from the index knee surgery, the patient was free of any clinical signs of infection.

Discussion

Within our institution, we had two allograft infections within a three-month period, and intra-operative cultures yielding growth in both. Although the second case had extensive injuries, we believe his knee infection was due to contaminated graft tissue. While this contamination can be discovered with intra-operative cultures in most cases, some contaminated tissues may yield falsely negative culture results.[9] Grafts with a presumably lower bacterial contamination "burden" or with less virulent bacterial contaminants, may present in delayed fashion.

Controversy still exists with respect to management of infected knees following allograft reconstructions. Since these tissues are avascular, they are in essence foreign bodies. Some investigators state that appropriate antibiotics along with serial irrigation and debridement may be sufficient to eradicate the infection with graft retention,[6] however, it must be realized that under certain circumstances during active infection, damage is done to surrounding tissues by host defenses and phagocytes.[7]

Tissue banks decontaminate their soft tissue grafts. However, as these cases demonstrate, there are times when the decontaminating techniques are not without failure. New methods must be developed to reduce the risk while not compromising the strength or mechanical integrity of the graft itself. Standard doses of radiation at 28 kGy have been shown to decrease the allograft strength to 64% of its native form and increase the stiffness,[4] freeze drying can lead to some change in the tissue properties if not carefully rehydrated, and ethylene oxide has been shown to lead to inflammatory reactions.[5, 8] Clearly, sterilization of allograft tissue must be more carefully checked before the tissue is deemed safe for implantation. The importance of intra-operative cultures cannot be understated, since tracking infections, and their source, will likely minimize future infection events.

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Technique

The Use of a Modified Humeral Locking Plate for Tibio-Talar Fusion

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Introduction

Painful ankle arthritis is a debilitating condition that severely affects the quality of life for thousands of patients each year. Tibiotalar arthrodesis is well accepted as the mainstay of surgical treatment for end stage arthritis of the ankle.[1, 2] Treatment success is highly dependent on proper surgical technique as well as stable fixation. The position of 5 degrees valgus, neutral flexion, and minimal external rotation is generally accepted as the standard of care. Over the past century, over forty different procedures have been developed to perform successful arthrodesis. Such procedures include but are not limited to the Blair and modified Blair procedure, external fixation compression, chevron technique, Ilizarov, triangular prism bone grafting, and various fixation techniques.[1–7] Different techniques have proven useful in particular situations or patients. Current literature reports ankle fusion failure rates ranging from zero to thirty six percent.[8–12] At this time, ankle replacement has not yet been proven as a superior method of treatment, particularly for post-traumatic arthritis.[13]

As internal fixation techniques have improved, their use in various fusion procedures has appeared, particularly in spinal fusion. Locked plating has become extremely popular for the treatment of fractures, particularly in the periarticular zone. The use of a locked plate for ankle fusion is a logical step in light of the recent success of locked plating for fractures. Locked plating provides fixed angle stabilization in a user-friendly device. We describe the use of a modified proximal humeral locked plate for tibiotalar fusion. Our goal was to provide stable fixation at the tibiotalar joint that would allow for a successful joint fusion.

Case

Our patient is a 38-year-old male who sustained an open right bi-malleolar ankle fracture in addition to an anterior-posterior compression pelvic injury, a left tibia fracture, and a right distal femur fracture. He was initially treated at an outside institution and underwent open reduction internal fixation of the ankle and syndesmotic repair at that time with vacuum assisted closure of the medial wound. He was transferred to our care for further treatment, which included surgical treatment of his other injuries and delayed wound closure of his open ankle wound. His syndesmotic screws were removed three months later and the medial malleolar screws were removed five months after the index injury. By ten months after the initial injury, radiographs demonstrated severe arthritis of the tibiotalar joint (Figure 1).

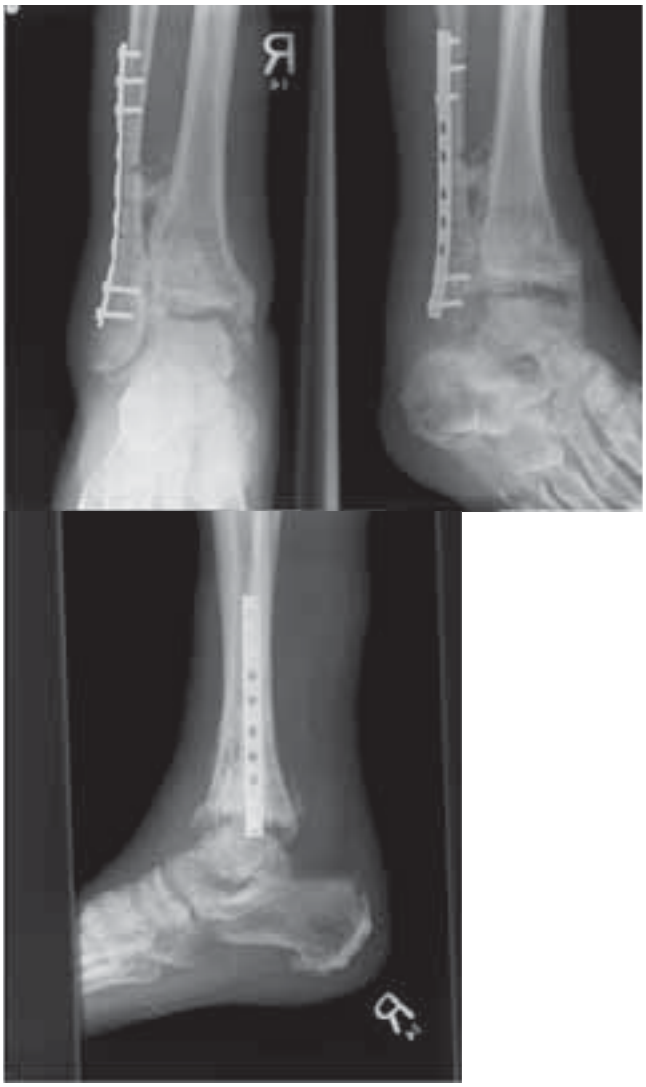


Figure 1. AP, mortise, and lateral views of the ankle ten months after initial injury demonstrating severe arthritic changes at the ankle joint with evidence of osteomyelitis.

The patient was diagnosed with osteomyelitis of the distal tibia and talus along with post-traumatic arthritis of the ankle joint. Further debridement of the medial wound, tibia, and talus was performed twice for persistent drainage. He continued to have painful arthritis of the ankle. Fourteen months after his initial injury, he underwent an arthrodesis of the

right ankle using a modified proximal humerus locking plate with placement of allograft bone and OP-1 (BMP-7, Stryker, Mahwah, New Jersey. The plate was trimmed to size with the assistance of the hospital machine shop and was subsequently cleaned and sterilized pre-operatively (Figure 2). The total tourniquet time was 93 minutes and estimated blood loss was 650 cc. Immediate post-operative x-rays are shown in Figure 3. Successful fusion was noted by six months post-fusion, as shown in radiographs in Figure 4. His ankle pain had improved considerably but due to intermittent wound drainage, hardware removal was performed at eleven months post-fusion. Post-operative x-rays and CT scans confirmed a successful radiographic fusion as shown in Figure 5. At the 30 month follow-up visit, he had no signs of acute or chronic infection and had returned to work without significant complaints.

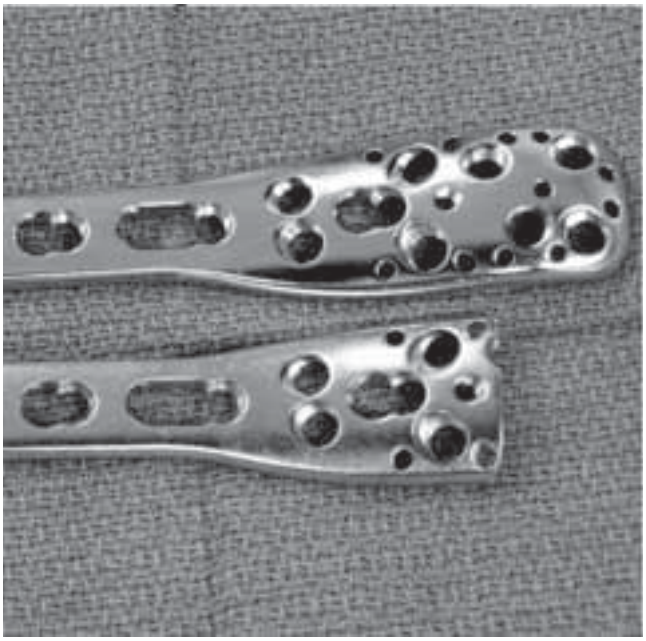


Figure 2. The Synthes 3.5mm proximal humeral locking plate: unaltered plate (top) and modified plate for ankle fusion (bottom).

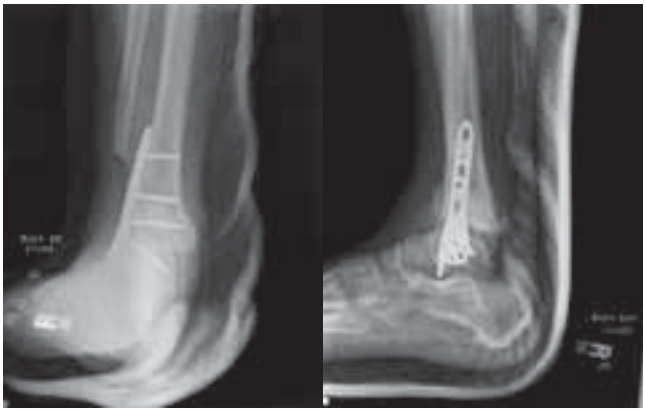


Figure 3. AP and lateral views of the ankle immediately postoperatively after removal of hardware and ankle fusion with modified proximal humeral locking plate.

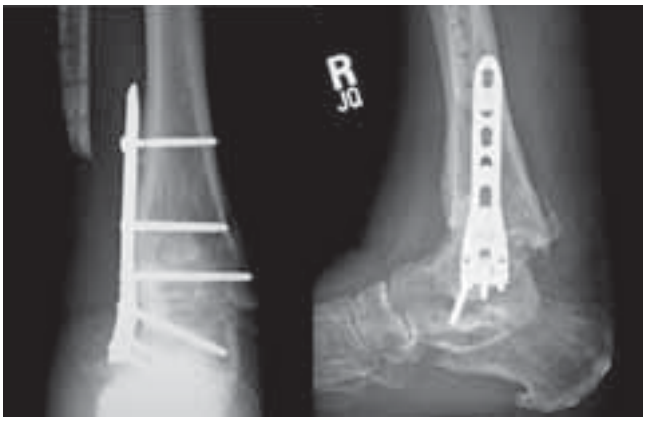
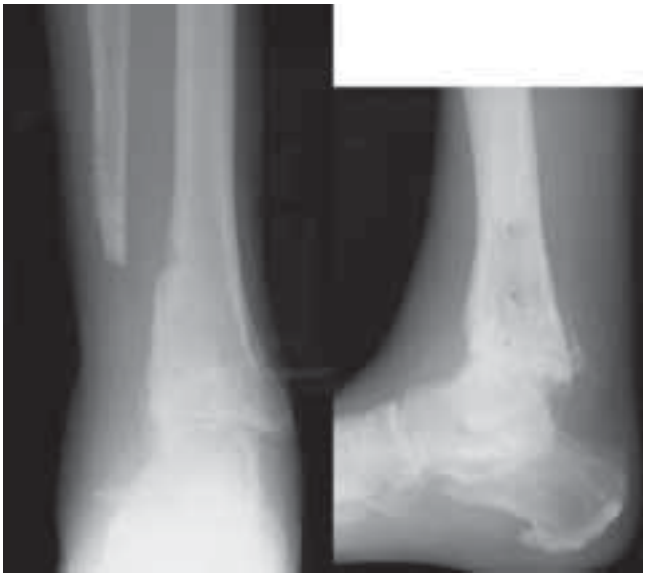


Figure 4. AP and lateral views of the ankle six months after successful ankle fusion.



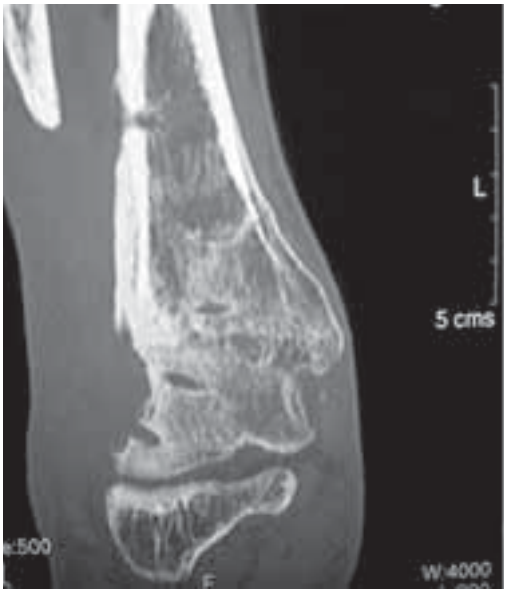


Figure 5. AP and lateral radiographs and coronal re-formatted CT scan image eleven months after fusion demonstrating successful radiographic fusion. Multiple screw holes demonstrate previous screw position in the tibia and talus.

Discussion

At this point, ankle replacement surgery has not proven to be an improvement over ankle fusion for treatment of post-traumatic arthritis.[13] Ankle fusion remains the mainstay procedure for treatment of end stage arthritis of the ankle joint. Nevertheless, ankle fusion rates using current techniques can arguably be improved.

The use of a locked plate as a rigid device can help to decrease strain at a fusion or fracture site and theoretically improve fusion rates. Locked plates are helpful in situations of bone loss and nonunions in which perfectly apposing bone ends often cannot be created. This creates a high-strain environment at the fusion site which requires absolute stability to allow bone formation.[14] In the peri-articular region or any location in which sufficient bone is not available for multiple standard screw fixation, fixed angle fixation is a potential solution.[15] Cervical spinal fusion has been successfully instrumented with locking plates for several years before locking plates were available for the appendicular skeleton.[16–18]

Blade plates have long served as a useful device for providing fixed angle stabilization. Chiodo et al. demonstrated the biomechanical superiority of blade plate fixation over intramedullary nail stabilization for tibiotalocalcaneal arthrodesis.[19] Though this study addressed tibiotalocalcaneal fusions rather than only tibiotalar fusions, their results are not surprising. Intramedullary nail stabilization affords relative stability rather than absolute stability in the form of a blade plate.

Morgan et al. described the use of the AO cannulated 90 degree blade plate for use in salvage of failed fixation of distal tibial fractures.[6] In this study, patients with tibial plafond fractures and metaphyseal delayed unions who developed post traumatic arthritis were treated successfully with tibiotalar fusion using the blade plate. Their technique involved a posterior approach to the nonunion and the ankle joint for fusion.

The use of blade plates is considered to be technically demanding and offers less options compared with the use of the newer locked plates. Therefore, we can expect to see more expanded indications for the use of locked plates. In the case of ankle fusion, it appeared to us that the Synthes 3.5mm proximal humeral locked plate, which provides multi-angle locked fixation across the joint, could serve as an ankle fusion implant if shortened appropriately. Modification of available hardware to adapt to special situations is a long-practiced technique in orthopaedic surgery, particularly in trauma.[20, 21] In fact, these methods are often the precursors of commercially-available modified devices to suit particular situations. We used our hospital machine shop to cut the end of the humeral locked plate off. This modified device was particularly suited to be used on the lateral aspect of the distal tibia and talus. As shown in our example, this creates an excellent fixed angle device with multiple locked screws. The Synthes 3.5mm proximal humeral locked plate is available in the locking small fragment set and is therefore available to most orthopaedic surgeons.

We feel that locked plating has a place as a device for arthrodesis procedures. If this gains more acceptance, improving the existing plates to match the anatomy of the ankle joint would be a useful step.

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Technique

Intramedullary Nailing for the Treatment of Fractures and Malunions of the Distal Radius

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The distal radius fracture is a common orthopaedic injury. In the United States, 17% of all emergency room visits are due to wrist injuries.[2, 3] In 1992, McMurtry et al reported that distal radial fractures account for one sixth of all fractures seen in the emergency department. A bimodal age distribution exists with peaks occurring at ages 5–14 and 60–69.[4] Wrist fractures occur in older postmenopausal women, with a female-to-male ratio of 4:1.[5] Many of these fractures are treated closed with some requiring manipulation. Some fall into an unacceptable reduction thus necessitating operative reduction. Though external fixation with or without pins can be done, open reduction and internal fixation is sometimes necessary. This can be accomplished via volar and/or dorsal plating techniques. Dorsal plating can result in symptomatic hardware and may require late plate removal. In order to circumvent problems with dorsal plating, volar plates were introduced. The surgical approach for the application of a volar plate is not without morbidity and requires significant periosteal stripping. This has resulted in evolution of distal radial intramedullary nails and the concept of "zero profile" hardware. We have been using this device to treat acute fractures of the distal radius and have found it particularly useful in treating older, malunited fractures of the distal radius.

The goal of this report is to describe a technique of intramedullary fixation for distal radius fractures that are malunited in a dorsally angulated, shortened, and rotated position.

Operative Technique

The patient is placed supine with the extremity on a hand table. A mini fluoroscopic machine is positioned to be used before and during the case. It is crucial to have quality AP and lateral x-rays of the injured wrist as well as contralateral films for comparison. An attempt at closed reduction is performed if indicated (Figure 1). The case is performed under tourniquet. A dorsal approach is carried out from just ulnar to Lister's tubercle extending proximally over the radial shaft. The length and location of the incision can be marked by placing the intramedullary device on the wrist and taking an x-ray. This will allow the surgeon to minimize the length of the incision and soft tissue dissection required to lock the device proximally.

Sharp dissection is carried out through skin only. Blunt dissection is then performed to the level of the extensor retinaculum. EPL is identified and its sheath incised obliquely in line with the muscle and tendon. The EPL tendon is transposed. The ridge between the 3rd and 4th compartment (Figure 2A) is dissected from the distal radius and the hypertrophied tissue resected. The fracture site is exposed and evaluated.

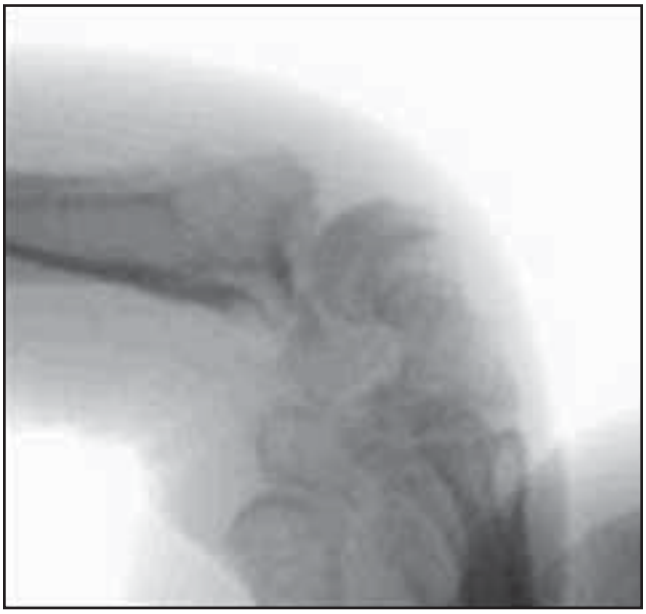


Figure 1. Attempted closed reduction.

For malunited fractures, osteotomy of the fracture site is required to mobilize the fragments. This is performed using a freer elevator and osteotome dorsally and moving in both a radial and ulnar direction. This allows for manipulation of the fragments and a controlled osteotomy. At this point, if shingling of the volar cortex is noted on x-ray, this can be addressed through the fracture site with a freer using a levering technique.

Acceptable restoration of radial length, inclination, angulation and articular congruity is assessed and confirmed on PA, lateral and articular views using x-ray. While maintaining the reduction with an osteotome or manually, a size .062" k-wire from the Micronail set (Wright Medical, Memphis, TN) is placed in the dorsal 4-5 interval (Figure 2B) to hold the reduction by engaging the volar cortex of the shaft. Reduction can again be assessed radiographically after pin insertion (Figure 3). It is crucial to obtain deformity correction prior to proceeding to the next step.

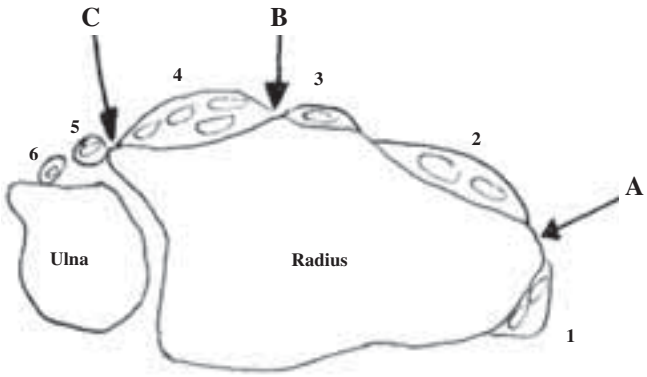


Figure 2. Dorsal intervals for (A) styloid incision; (B) osteotomy dissection; (C) 4-5 pin placement.

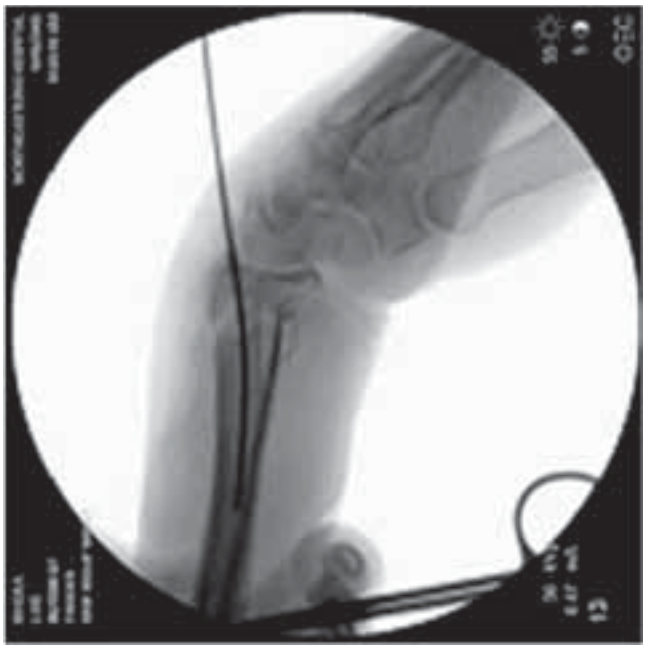
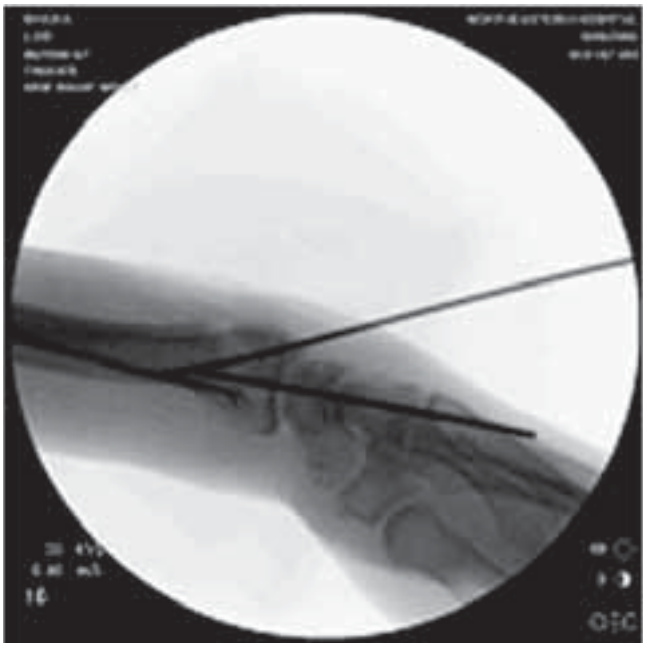


Figure 3. Provisional Fixation with dorsal 4th-5th interval pin.

Attention is then turned to the radial side of the distal radius. A 3-4cm incision is made over the radial styloid and blunt dissection is carried out down to the interval between the 1st and 2nd compartment (Figure 2C). Branches of the radial sensory nerve are maintained in the dorsal flap and protected at all times. Sharp dissection is used to subperiosteally expose the styloid while preserving the two dorsal compartments. Another .062" k-wire is placed into the styloid across the fracture site and into the ulnar cortex of the radius at approximately a 45 degree angle from the longitudinal axis of the radial shaft (Figure 4).

4A



4B

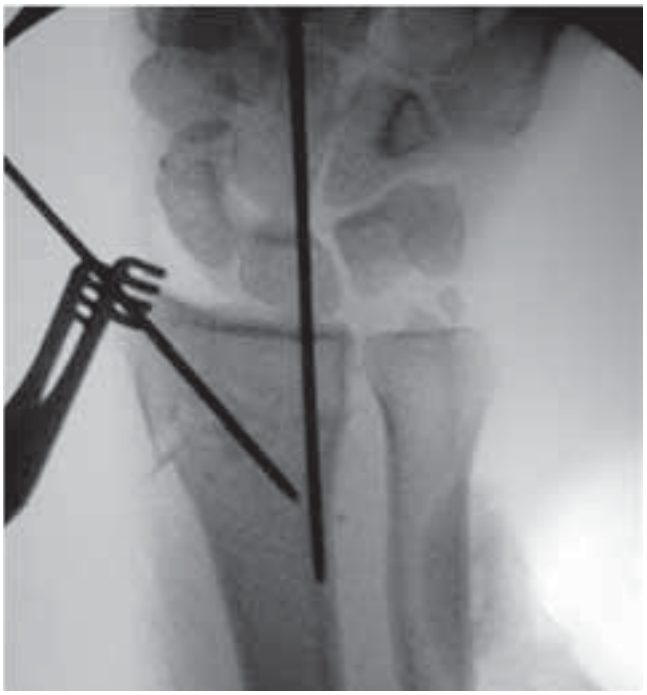


Figure 4A, B. Styloid Pin inserted centered on the lateral view.

Nail insertion is performed through the styloid in the interval between the 1st and 2nd compartment. It is crucial that the k-wire is placed into the styloid centered on the lateral projection and at the tip of the styloid on the AP x-ray. Once this is confirmed by x-ray, a cannulated drill opens the styloid. Bone debris is removed and a rongeur is used to remove a small amount of bone at the proximal portion of

this starting hole to allow for broaching and proper seating of the implant. The radius is then entered using a starting awl placed across the fracture site hugging the radial cortex of the proximal fragment. The depth of the implant can be checked using a k-wire through a hole in the awl to ensure that there will be subchondral placement of the distal locking screws (Figure 6). The radius is then broached to the appropriate size to allow for canal fill. X-ray is used to confirm placement of the broach in both views.

The implant of appropriate size is chosen and is located in the set (Figure 5). It is placed into the broached path through the styloid. A locking jig is used for the placement of the distal three locking screws, which are subchondral and splay slightly volarly and dorsally. These holes are drilled as soft tissue is protected with retractors. Length of screws is determined by the calibrated drill bit or depth gauge. The distal screws lock into the implant and are intramedullary. The optimal placement for these locking screws is subchondral and can be checked by passing a wire through the locking jig. It is crucial to be subchondral to optimize the support of the metaphysis by these fixed angle screws. Do not violate the DRUJ. Proximal locking is performed by using a radiolucent locking jig over the dorsum of the distal forearm. The incision is made just ulnar to the muscle bellies of the 1st compartment and exposure is achieved by blunt dissection. Proximal locking screws are inserted using drill and screw sleeves and are bicortical nonlocking screws.

Prior to proximal lock insertion, the dorsal defect (Figure 7) can be addressed with bone graft. It is our preference to use a combination of structural graft augmented with osteoinductive material to fill the void (Figure 8). Irrigation should be performed prior to grafting and wound closure is standard. Dressings and a volar plaster splint are applied prior to deflation of the tourniquet.

5A

5B

Figure 5A, B. Micronail (Wright Medical, Memphis TN).

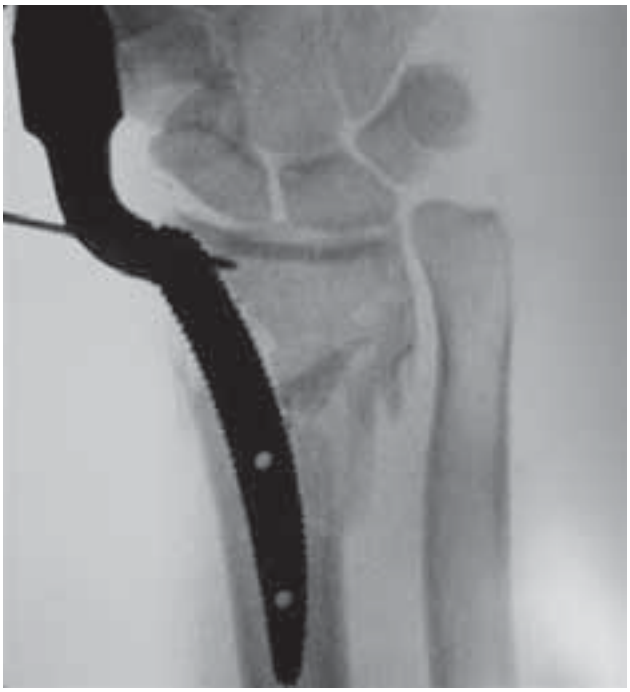


Figure 6. Broach in place with k-wire to show placement of distal locking screw.

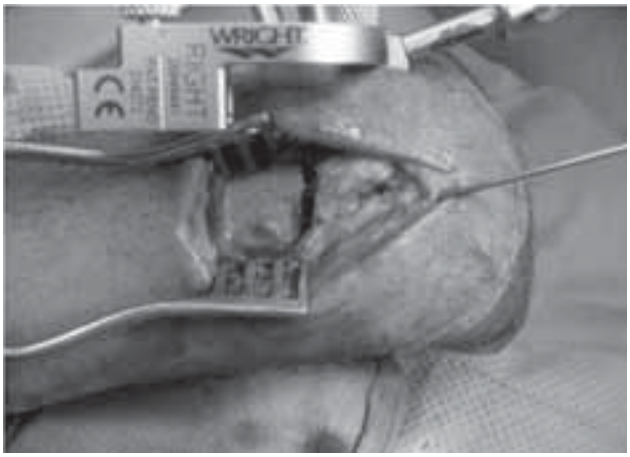


Figure 7. Dorsal defect prior to grafting with wrist flexed.

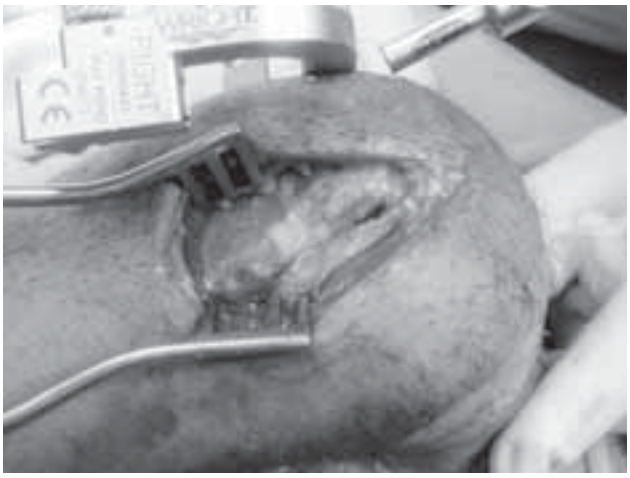


Figure 8. Dorsal defect with bone graft applied.

Case Examples

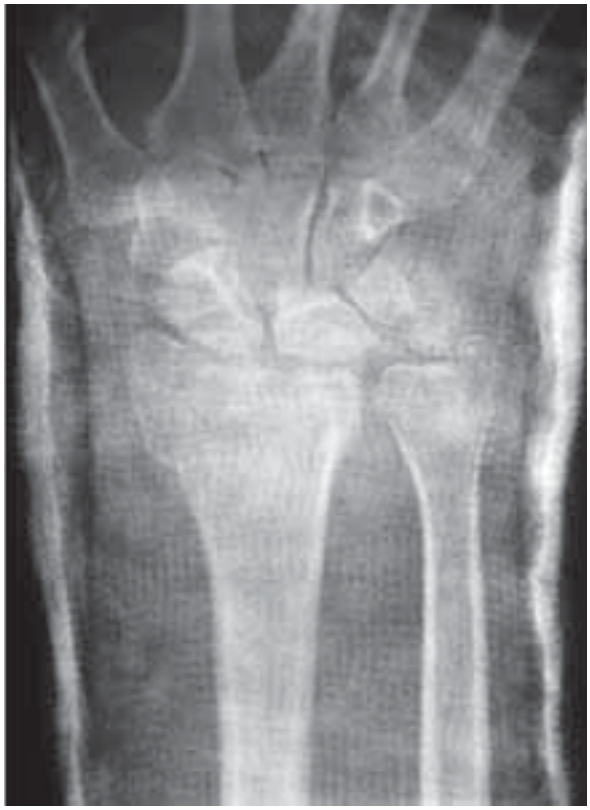
Case 1

A 45-year-old right hand dominant male fell off a ladder onto his dominant hand. He was treated in the emergency room with a splint. He was seen in the office approximately two weeks post injury where a closed reduction was performed and a long arm cast was placed. Although post-reduction films showed an acceptable reduction, at 1 week follow up the fracture had collapsed.

Preoperatively, radial inclination was 11 degrees, radial length was 6 mm, and dorsal angulation was 23 degrees (Figure 9A, B). The patient was scheduled for malunion corrective osteotomy, open reduction and internal fixation of the distal radius. Operative intervention was performed at approximately 4 weeks post injury. This case showed unacceptable parameters, specifically dorsal angulation even after being held in extreme hyperflexion in a closed reduction maneuver (Figure 1). A dorsal approach was chosen to address the dorsal angulation as well as the large dorsal gap that would be created from reduction. Once the osteotomy was performed the fracture was held provisionally with a 4-5 interval pin and a styloid pin (Figure 3, 4A, B). The corrected position of the distal radius was held with the pins and the technique for insertion of the Micronail was begun. The nail was inserted and locked distally. The dorsal defect could be seen (Figure 7) and directly bone grafted prior to placing the proximal locks (Figure 8).

Proximal locks were placed through the dorsal incision. Final PA and lateral x-rays are shown in Figure 10A & B. Postoperative radiographs show a radial inclination of 16 degrees, volar tilt of 7 degrees and radial height of 10 mm. Clinical intraoperative photos are shown in Figure 11A & B.

9A



9B

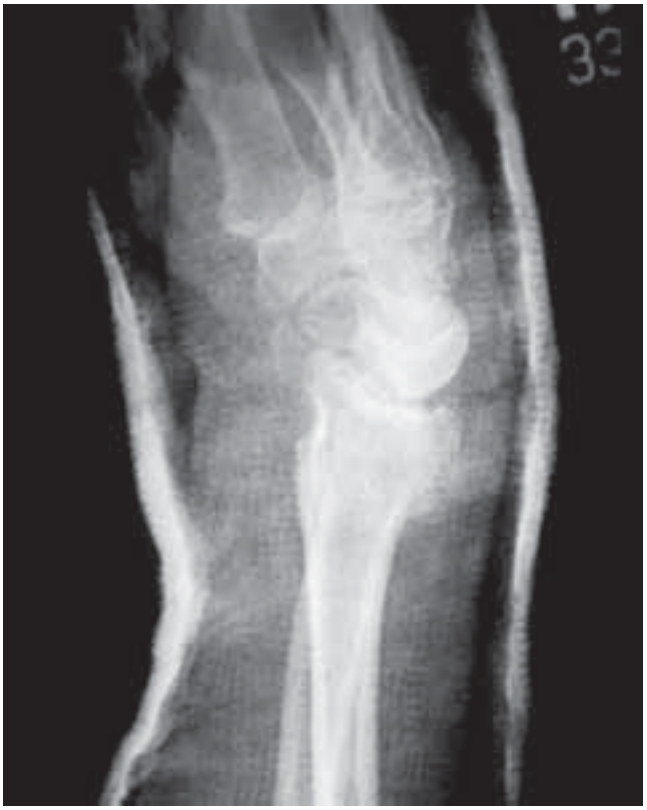


Figure 9A, B. AP and lateral x-ray of preoperative malaligned fracture in cast.

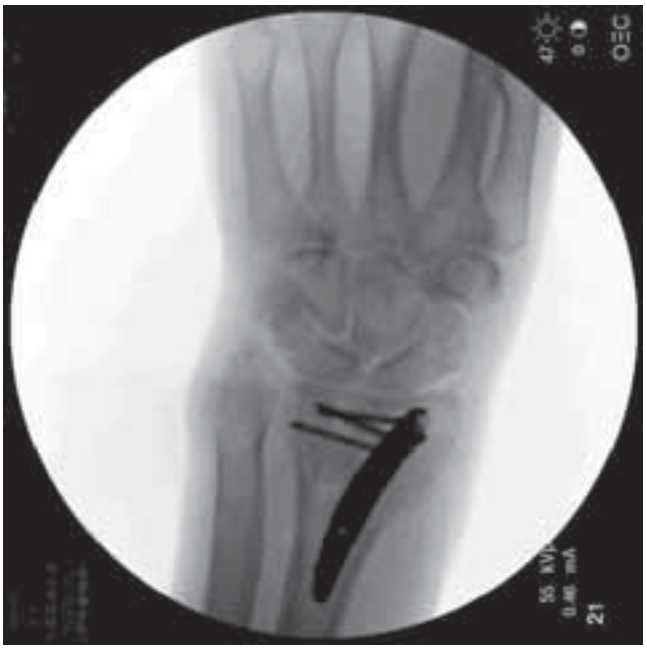


Figure 10A. Postop PA x-ray.

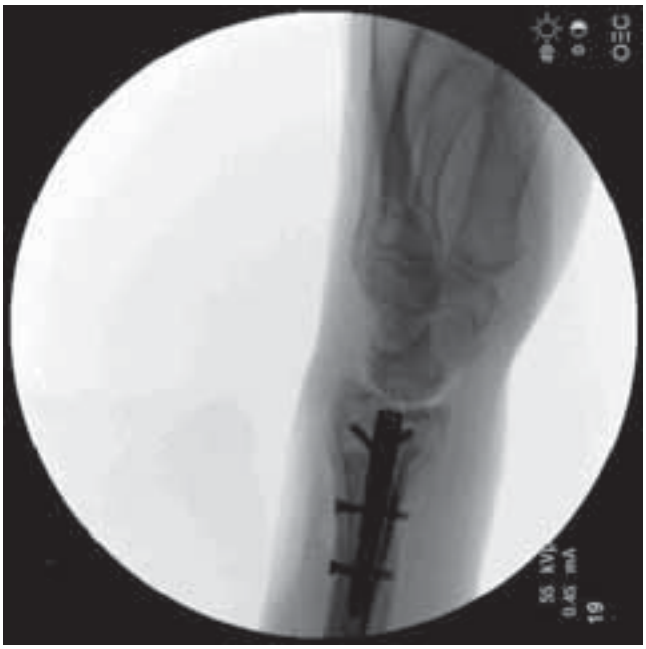
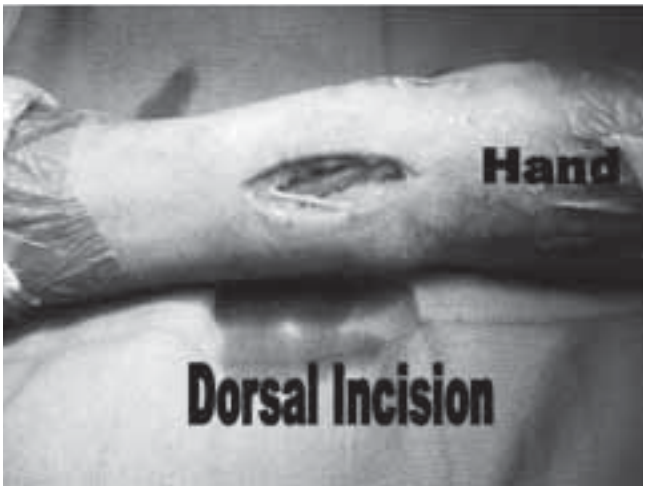


Figure 10B. Postop lateral x-ray.

11A



11B

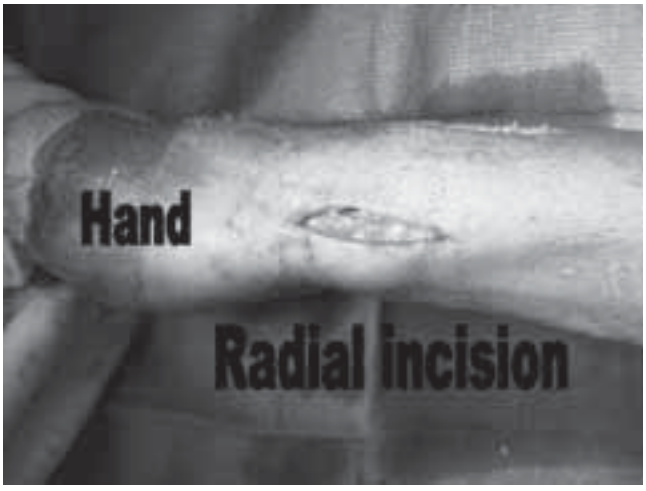


Figure 11A, B. Clinical photos of dorsal and radial incisions.

Case 2

A 24-year-old right hand dominant female injured her distal radius of her dominant hand. She was seen in an emergency room where she was casted and instructed to follow up with her orthopaedist. She presented four weeks post injury to our institution. She was seen in the office and noted to be nontender to palpation at the distal radius with a gross deformity with radial deviation and mild dorsal translation. Rotation of the forearm showed an arc of only 50 degrees. Preoperative x-rays show dorsal angulation of 7 degrees, radial inclination of 6 degrees and radial length of <5 mm. In addition, her distal ulna is dorsally translated consistent with shortening and incongruity of her DRUJ. This was felt to contribute to her overall dysfunction. She was scheduled for the operating room and consented for osteotomy, open reduction internal fixation, and bone grafting. While the deformity of Case 2 is less severe than that in Case 1, the rotational deficit supported the decision to correct the deformity.

Preoperative films in a cast (Figure 12A, B) and intraoperative films (Figure 13A, B) are shown. The fracture did not move upon attempted closed reduction. Therefore a dorsal approach and osteotomy was selected and carried out. Radiographic assessment of the restoration of radial inclination, length, and volar tilt was performed and pictured in Figures 14A & B. A radial approach was then used for nail insertion and the defect was grafted prior to locking proximally. 6 week postoperative radiographs are shown in Figure 15A & B. X-rays show volar tilt restored back to 9 degrees with radial inclination of 19 degrees, and a radial height back to 9 mm.

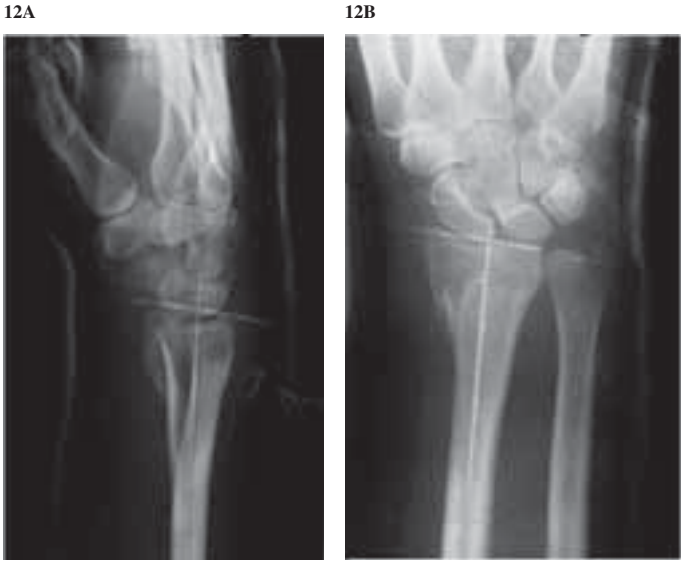


Figure 12A, B. Preoperative x-rays in cast.

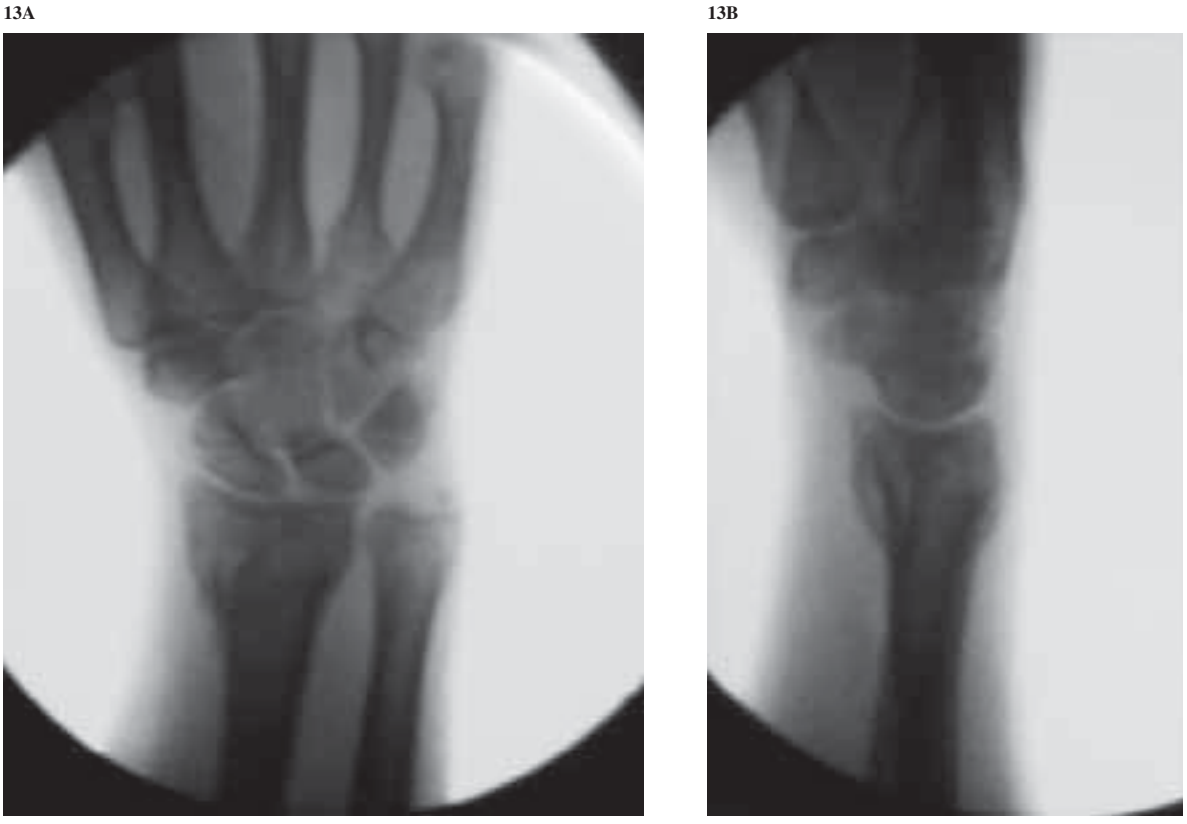
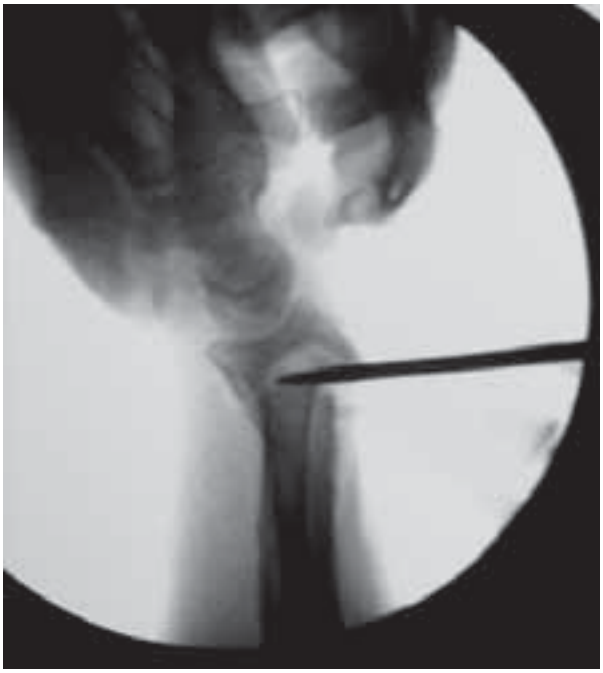


Figure 13A, B. Intraoperative x-rays.

14A



14B

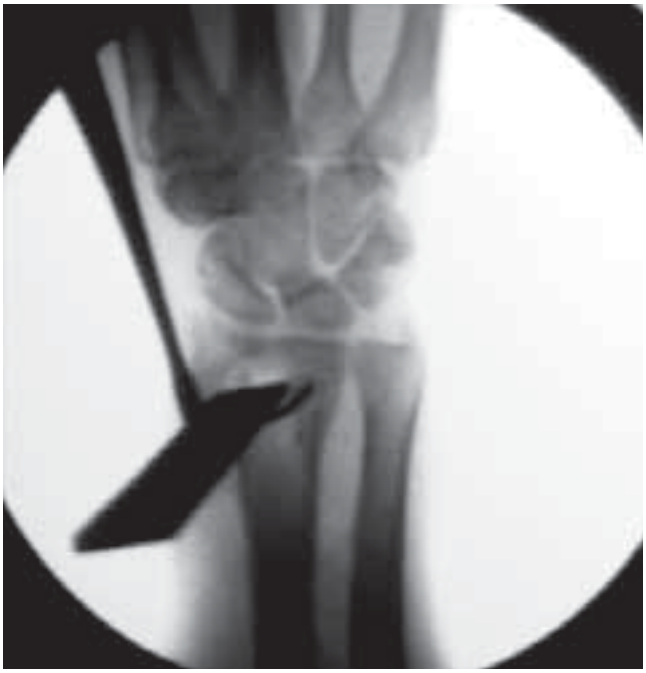
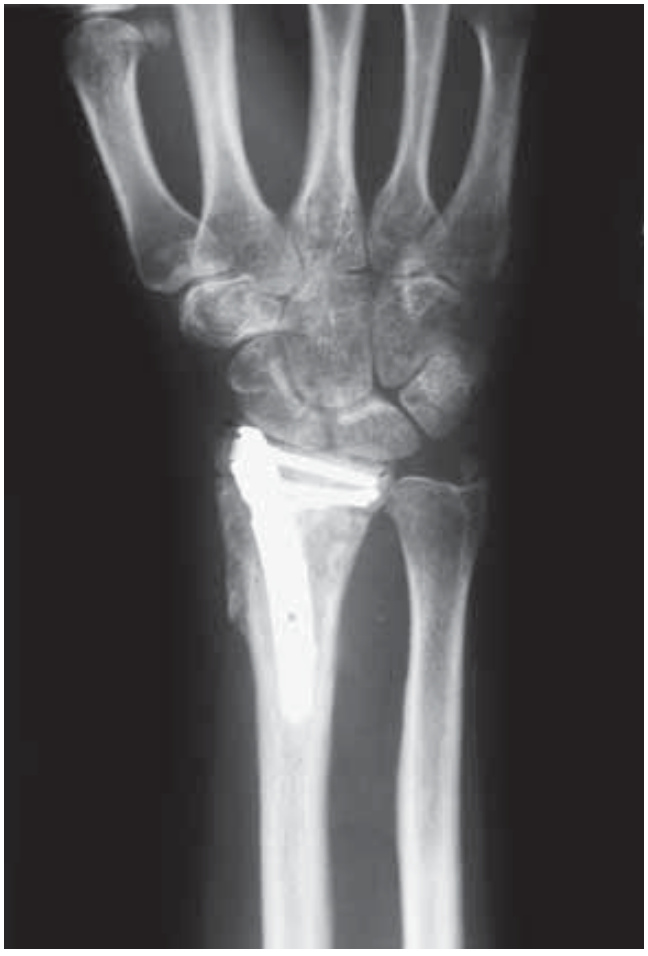


Figure 14A, B. Corrective osteotomy.

15A



15B

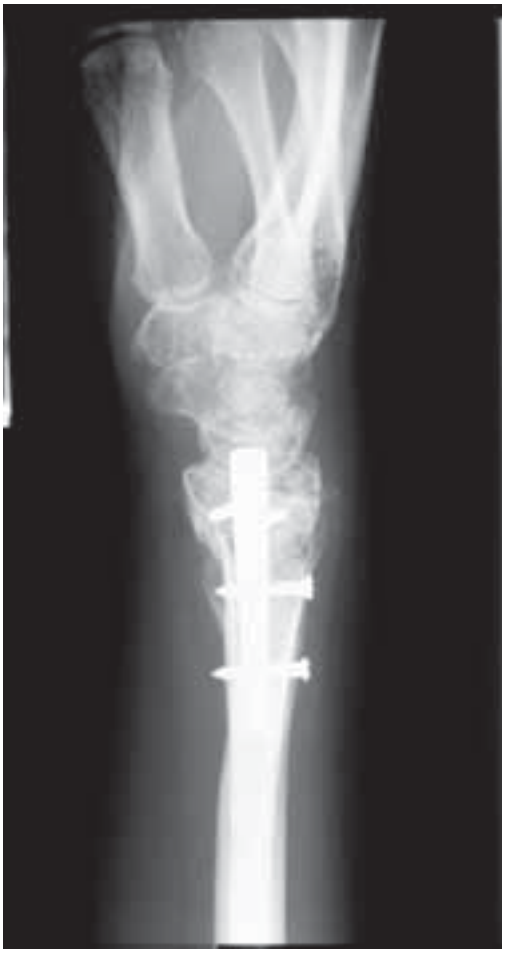


Figure 15A, B. Six weeks post-op AP & lateral x-rays.

Discussion

Intramedullary fixation using the Micronail is indicated for displaced extraarticular fractures and simple displaced intraarticular fractures of the distal radius. Pitfalls of the device were mentioned and include the potential of splitting the radial styloid and broaching the ulnar or radial column too aggressively resulting in a breach of the intact cortex. Distal screws must be subchondral while remaining extraarticular. Xrays must be critically evaluated to ensure proper rotation and screw placement. The entry site must be centered in the radial styloid as seen on the lateral radiograph to ensure that there is no translation or angulation of the distal fragment.

Options for malunion and corrective osteotomies of the distal radius are the use of pins, external fixation, and plating from the volar, dorsal or radial side.[6] Intramedullary fixation for the distal radius is a new technique and quite different from the alternatives of more traditional techniques. The Micronail intramedullary radial fixation device potentially eliminates several problems that are inherent with plating, pins and external fixation. It may eliminate associated complications of extrinsic tightness, wrist capsular stiffness, osteopenia, and radial sensory nerve problems.[7] Fixation does not cross the carpus and therefore allows for motion of the hand and wrist.

Plating of the distal radius using dorsal plating technique can result in tendon irritation, adhesions, attrition, and rupture. Even with the lower profile dorsal plate, symptomatic or prominent hardware may require an additional surgery for removal of the hardware. Several series have reported up to 30% rate of plate removal.[8] Soft tissue dissection can result in significant postoperative swelling and difficulty mobilizing the wrist, hand, and fingers. This can lead to stiffness and necessitate a prolonged period of rehabilitation. Volar plating also may necessitate plate removal and in one study 18% of volar plates needed to be removed due to irritation.[9] A similar approach to the malunion is performed as is done with dorsal plating but less overall exposure is needed than when a plate is applied.

The advantages of using this technique are the ability to perform a dorsal osteotomy and address the potential dorsal defect directly. There is less surgical trauma than there would be with use of dorsal plating. An intramedullary implant gives the ultimate low profile and in fact gives a "zero profile" result with no continued irritation of surrounding structures. Less soft tissue trauma means less hand and finger swelling and subsequent quicker return of motion.

The biomechanical advantage of a locking fixed angle device is subchondral support which is well documented in the literature. This fixation allows for earlier motion while maintaining stability of the fracture. This leads to the ability of patients to return to work more quickly.

Pitfalls of this technique range from reduction, radius preparation, nail insertion and, locking. Malreduction or improper deformity correction can result in residual pain, deformity and continued loss of function. The osteotomy must correct the deformity and reduction must be maintained during broaching, nail insertion and locking. The starting hole in the radial styloid is crucial. If this hole lies too volar or dorsal, the distal fragment will angulate and translate as the nail is inserted and engages the proximal fragment (similar to what happens when nailing a proximal tibia fracture). When using the awl and broach, the proper position of these is to hug the radial cortex. If these instruments are directed in an excessively ulnar direction, the ulnar cortex of the radius can be violated. In addition, if the instruments are aimed too radially, they can exit the fracture site into the soft tissue or splinter the radial cortex.

Malrotation of the nail may occur if forearm and wrist positioning is not closely monitored. If this is not recognized, the volarly or dorsally angled distal screws can potentially breach the volar or dorsal cortex of the distal fragment. In addition, the distal locking screws must be subchondral and extraarticular with respect to the radiocarpal and distal radioulnar joint. These should have a firm endpoint as with any locking screw as they seat into the nail. Finally, the proximal screws should be inserted through an incision that ensures that they are seated in the dorsal cortex of the radius with no soft tissue interposition. Avoidance of countersinking the proximal screws is also important especially in osteopenic bone.

This technique for malunion takedown, osteotomy, and mobilization of the fracture fragments has long been effective.[6] The ability to apply bone graft directly through the defect is crucial. The application of a "no profile" implant while maintaining the benefits of locking technology and rigid fixation results in a solid construct that can allow for reliable healing and quicker recovery and return to work.

At our institution intramedullary nailing of the distal radius for several applications has been employed with success. This technique has become our preferred method of fixation for malaligned late presenting fractures as well as for corrective osteotomies for malunions of the distal radius.

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Lectureship

The John Lachman Lecture at the Pennsylvania Orthopaedic Society

JOHN A. BERGFELD, MD

Director of Sports Medicine, Cleveland Clinic

"Should the Team Doctor Pay to Play?"

In keeping with the Lachman principle of integrity, the John Lachman Society has developed an annual lectureship to discuss timely issues dealing with ethics and the practice of orthopedic surgery.

The First John Lachman Lecture was given by David Apple, MD, Medical Director of the Shepard Spine Center in Atlanta, Georgia. Dr. Apple's presentation entitled "The Ethical Practice of Orthopedic Surgery" was a basic primer dealing with the accepted principles.

Dr. Bergfeld's 2005 presentation concerned the recent practice of orthopedic groups and/or hospitals paying professional teams to provide physician and medical services. Thus the title "Should the Team Doctor Pay to Play?".

Dr. Bergfeld, the past President of both the American College of Sports Medicine and the American Orthopedic Society for Sports Medicine, has served as team physician for the Cleveland Browns for the past 25 years. Internationally recognized for his numerous accomplishments and impeccable integrity, he is extremely well qualified to discuss this issue.

The problem confronting Dr. Bergfeld was that the doctor appointed team physician, as part of a multi-million dollar marketing contract, might not be the best qualified individual for the job. Also, with patient care involved and potential for conflict of interest, clear ethical implications exist.

Bergfeld's answer to the question of should the team physician pay to be the team physician was an emphatic "absolutely not". However, he pointed out that the sports venue provides a very effective marketing tool. But all involved must separate the marketing contract from the medical contract.

Accepting the realities of the current marketing/medical environment, Bergfeld defined the following principles that the team physician must follow:

1) Be ethical;
2) Be properly trained;
3) Be willing to walk away from situations where he/she feels compromised;
4) Be aware of financial arrangements;
5) Separate medical reimbursement from marketing contract;
6) Be careful how marketing people promote you;
7) Be willing to make full disclosure.

Dr. Bergfeld's presentation was well attended, well received, and greatly appreciated.

The John Lachman Society is pleased and proud that the Lachman Lectureship is now a permanent part of the Annual Meeting of the Pennsylvania Orthopaedic Society.

J. Torg, MD

Dr. John A. Bergfeld

Lectureship

The Howard H. Steel Lecture at the Philadelphia Orthopaedic Society

SCOTT J. MUBARAK, MD

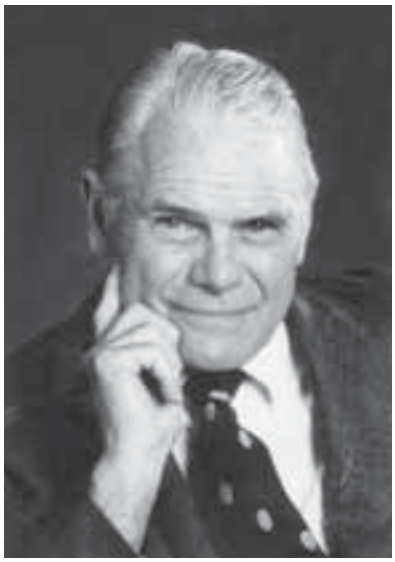
Director, Orthopaedic Institute at Childrens Hospital of San Diego
Clinical Professor, University of California, San Diego

"Acute Compartment Syndromes: When Is It Too Late?"

The Department of Orthopaedic Surgery at Temple University Hospital proudly hosted the annual Steel lecture at the Philadelphia Orthopaedic Society on Sept 12, 2005. Howard H. Steel, MD, PhD, is the emeritus Chief of Staff of Shriner's Hospital for Children. He has been a pillar of the orthopaedic community in Philadelphia as well as on the international stage for several decades. His publications and contributions are too numerous to recount. Simply put he is an icon of orthopaedics in Philadelphia and in the world.

The lecture in Dr. Steel's honor was given by Dr. Scott Mubarak. He discussed Acute Compartment Syndrome. He spoke about the pathogenesis and pathophysiology of Compartment Syndrome as well as the evolution of monitoring techniques. He also elaborated on the surgical indications and techniques. He closed by highlighting the increasing relationship of hip spica casting in children and the increasing risk of developing a compartment syndrome.

A. Ilyas, MD



Dr. Howard H. Steel

Lectureship

Philadelphia Pediatric Symposium at the Shriners Hospital for Children, Philadelphia

On December 10, 2005, Shriners Hospital for Children, an Orthopaedic based hospital providing free medical care for its patients hosted the annual Pediatric Symposium in December. Its goals are to provide a forum for the discussion of current pediatric orthopaedic topics, case presentations, and "hot topics." Throughout the day, many important issues, such as Legg-Calve-Perthes, slipped capital femoral epiphysis, pediatric trauma, and Blount's disease are discussed with a focus on etiology, pathology, diagnosis and treatment. The day provided a good review of the current thoughts from leaders in their field.

This year, the symposium was moderated by Dr. James McCarthy, who introduced the panel of Drs. Dennis Wenger, Vernon Tolo, and Kay Wilkins. Dr. Howard Steel also made his appearance to add a few experienced comments and answer questions regarding his famous "Mary Poppins" case.

The format of the day consisted of case discussions that illustrated the critical points of history, physical examination, and diagnosis of important pediatric disease processes. At each step, there was a panel discussion regarding their recommendations, at times illustrating the myriad of options, once again reinforcing that medicine is an art form with many different approaches to the same problem. There were many questions from the audience and eager discussion of what was done in their practice. Following the cases, a lunch was provided by Shriners Hospital, which was well attended that allowed further informal discussions with the panelists and other community members.

K. Matullo, MD

Shriners Hospital for Children, Philadelphia.

Lectureship

The John Royal Moore Lecture at the Philadelphia Orthopaedic Society

SCOTT P. STEINMANN, MD

Assistant Professor of Orthopaedics, Mayo Clinic College of Medicine

"Arthroscopic Treatment of Elbow Arthritis"

The Philadelphia Orthopaedic Society had the pleasure of hosting Dr. Scott Steinmann on February 13, 2006 for the annual John Royal Moore Lecture. Dr. Steinmann comes from the Mayo Clinic in Rochester, Minnesota and specializes in arthroscopic shoulder and elbow surgery, brachial plexus injuries, vascularized bone grafting, and reconstructive microsurgery. We had the distinct honor of hearing a dynamic lecture on the Arthroscopic Treatment of Elbow Arthritis.

Dr. Steinmann presented his work and clinical experience with elbow arthroscopy for the treatment of various conditions including osteoarthritis, removal of foreign bodies, inflammatory disease, radial head fractures, ulnar nerve decompression, release of contractures, and lateral epicondylitis. He discussed the indications for arthroscopy, various techniques of portal placement and debridement, and the many advantages and limitations for this developing procedure. He illustrated some of the dangers involved with this procedure including damage to the surrounding nerves, compartment syndrome, and septic arthritis. Dr. Steinmann balanced out his talk by stating that an open approach to the elbow is still the more well-studied and perhaps more cost effective way to treat certain elbow disorders. However, as research continues in the field of elbow arthroscopy, we may soon see expanding indications and better results for this evolving procedure.

The lecture generated some intellectual and lively discussion about the role of arthroscopy in the treatment of some elbow pathology. Dr. Torg inquired about the available outcome data regarding the use of arthroscopic surgery, and its potential efficacy versus the open treatment of certain elbow disorders. Dr. Resnick inquired about the role of arthroscopy for the treatment of lateral epicondylitis, an extraarticular disorder, with an intra-articular treatment. Dr. Steinmann concluded his discussion by citing the developing clinical data on the indications for this procedure and was extremely excited about the growing potential of arthroscopic surgery for the treatment of elbow disorders.

S. Chao, MD

Drs. Resnick, Torg, Steinmann, Thoder, and Shaffer.

Departmental News

Faculty

Temple University Department of Orthopaedic Surgery & Sports Medicine

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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
Gene Shaffer, MD
Bruce Vanett, MD

Emeritus Professors

Philip Alburger, MD
Edward Resnick, MD

Adjunct Faculty — Philadelphia Shriners Hospital

Randal Betz, MD, *Chief of Staff*
Howard Steel, MD, *Emeritus Chief of Staff*
James McCarthy, MD, *Assistant Chief of Staff*
G. Dean MacEwen, MD
Scott Kozin, MD
Linda D'Andrea, MD

Adjunct Faculty — Abington Memorial Hospital

David Junkin, MD, *Chief of Orthopaedics*
Jeffrey Rubin, MD
Michael Gratch, MD
Andrew Star, MD
David Craft, MD
John Wolf, MD
T. Robert Takei, MD
Guy Lee, MD
Moody Kwok, MD
Shyam Brahmabhatt, MD
Thomas Peff, MD

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General Orthopaedics

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General Orthopaedics

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Joint Reconstruction
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Saqib Rehman, MD
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Edward Resnick, MD
General Orthopaedics
Pain Management

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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

Report from the Residency Director

The last year has been a busy one for the Orthopaedic residency at Temple. In addition to the increasing research effort, the residents have continued with ever-busier clinical duties, organizational responsibilities, and educational activities. On the clinical side, the residents cover Hand, Joint Reconstruction, Sports Medicine, Spine, Foot and Ankle, and Trauma at Temple University Hospital. They also treat children at Temple Children's and Shriners Hospital for Children, as well as rotate to Northeastern Hospital, Jeanes Hospital, and Abington Memorial Hospital. Patient volume remains high at each of these institutions. Organizationally, the residents must track all of their surgical cases and record their duty hours in separate Internet databases. At Temple, they maintain and constantly update a complete database on all patients treated, a practice started by William Delong, MD, Director of Trauma, that now includes thousands of patients. The database not only guides and organizes day-to-day care on the floors, but also is accessible for research and follow-up. The resident's educational activities continue to center around the daily morning conference and reading schedule. The explosion of new books, journals, technique guides, knowledge updates, videos, review courses and lectures from the American Academy of Orthopaedic Surgeons makes education and learning ever more challenging. One of the most popular educational events on our calendar is the monthly Journal Club. Excellent participation by attending staff and residents along with lively discussion and debate over the articles has made this happen.

Orthopaedic Surgery continues to be a popular residency choice among graduating medical students. Last year, we had over 400 applicants for the 4 residency slots at Temple. We continue to have strong, well-rounded, diverse residency classes selected from this motivated pool. Our interview day this past January saw candidates from across the country. When they finish, Temple residents generally seek fellowship training, although as many as one in four will enter the military or practice general orthopaedics. Last year, two of the graduating senior residents entered fellowships in Sports medicine; one in Biloxi, Mississippi, and one in Chicago, while one began serving his military commitment in South Carolina.

In 2006, we will graduate five residents, instead of the usual four. Dr. Chandra Reddy joined us in 2003 with the Class of 2005, and after finishing up some requirements for the Boards, will be finishing this year. He is planning on remaining in the area for work, as his wife is a resident in the Anesthesia Department at Temple. The four other senior residents all have plans for next year. Dr. Paul Codjoe plans to enter private practice in New Jersey, hopefully giving him time to train and compete for his native Ghana in the next Summer Olympics in weightlifting. Dr. Victor Hsu has taken a Spine Fellowship in San Diego, CA, with Behrooz Akbarnia. He plans to try out for the local USBL team, attempting to become the first Asian, nearsighted, loose-shouldered, and beaten-once by Asif Ilyas player in the league. Speaking of Dr. Ilyas, he'll be doing a Hand and Upper Extremity fellowship next year. His only athletic endeavor is the day he beat Victor at one-on-one basketball. Dr. Matthew Reish rounds out the Class of 2006, and he will be traveling to Long Beach, CA to work on his golf game and study Sports Medicine with Doug Jackson.

Gene Shaffer, MD

Temple University Department of Orthopaedic Surgery and Sports Medicine House Staff 2006

Paul Codjoe, MD
PGY-5

Victor Hsu, MD
PGY-5

Asif Ilyas, MD
PGY-5

Matthew Reish, MD
PGY-5

Leonard D'Addesi, MD
PGY-4

David Junkin, MD
PGY-4

Robert Purchase, MD
PGY-4

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PGY-4

Wade Andrews, MD
PGY-3

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PGY-3

Joseph Morreale, MD
PGY-3

William Pfaff, MD
PGY-3

Simon Chao, MD
PGY-2

Neil MacIntyre, MD
PGY-2

Carlos Morerya, MD
PGY-2

Alyssa Schaffer, MD
PGY-2

"The Chiefs"

Victor, Lenny, and Paul at the end of year party.

"Happy Birthday, Eddie" (80th!!)

Dr. Torg's all smiles at a Journal Club.

Kel "prosletyzing"

The chiefs having a smoke.

The Class of 2007: Dave, Rob, Dave, and Lenny

The Class of 2008: Wade, Kris, Bill, and Joe

Kris, Joe, and Dave getting ready for the alumni meeting.

Terri, Jen, Matt, and Latch

Victor making a move on Matt at the Shrine Bowl.

Shrine Bowl 2005 — The Class of 2006 remains undefeated.

The Class of 2009: Neil, Carlos, Simon, and Alyssa

Ragball Game 2005

The Class of 2007 with their wives.

The Class of 2008 with their wives.

Department of Orthopaedic Surgery
Graduating House Staff 2006

Paul Codjoe, MD

Paul grew up in Ghana and migrated to the United States while in High School. He attended Rutgers University where he earned a B.A. in Biology. He went on to medical school at UMDNJ – Robert Wood Johnson. He is married with two children. After residency he plans to go in to Private Practice.

Victor Hsu, MD

Victor grew up on the Main Line. He attended the University of Pennsylvania where he earned a B.A. in the Biologic Basis of Behavior. After college he spent two years on Wall Street as an Analyst. He later went to medical school at Temple University, where he graduated as a member of *Alpha Omega Alpha.* He is married with one child. He is going on to a fellowship with Dr. Behrooz Akbarnia at the San Diego Center for Spinal Disorders in Spine Surgery.

Asif Ilyas, MD

Asif grew up outside Philadelphia in the Lehigh Valley. He attended Wilkes University where he earned a B.S. in Biology and graduated *Summa Cum Laude.* He went on to medical school at the MCP-Hahnemann School of Medicine, where he graduated as a member of *Alpha Omega Alpha.* He is married with two children. He is going on to work with Drs. Jupiter, Ring, and Lee at the Massachusetts General Hospital as a fellow in Hand & Upper Extremity Surgery.

Matthew Reish, MD

Matt grew up in Lewisburg, Pennsylvania. He attended Dickinson College and earned a B.A. in History. After college he spent two years as an Analyst for Vanguard. He later went on to medical school at Temple University, where he graduated as a member of *Alpha Omega Alpha.* He is married with three children. He is going on to do a Sports Medicine fellowship with Dr. Doug Jackson at the Southern California Institute of Sports Medicine.

Alumni Day — May 13, 2005

Instruction to Authors

Editorial Philosophy

The *Temple University Journal of Orthopaedic Surgery and Sports Medicine* is structured to provide a review of different Orthopaedic disorders, treatments, and techniques. As such, the *Journal* will consider for publication any original clinical or basic science research, review articles, case reports, and technical or clinical tips.

Editorial Review Process

All submissions will be sent to expert consultants for review.

Manuscript Requirements

Manuscripts are not to exceed 15 double spaced type-written pages and/or 5000 words. 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 third 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, into 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 the *Journal of Bone & Joint Surgery.*

(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.

Submission Checklist

- Three hard copies
- Two CD's labeled with the author's last name and manuscript title

Disclaimer: **This journal contains manuscripts that are considered interpersonal communications and extended abstracts and not formalized papers unless otherwise noted.**

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