Temple University Journal of Orthopaedic Surgery & Sports Medicine

John Michael Daly, MD

Volume 8 Spring 2013

A John Lachman Society Publication

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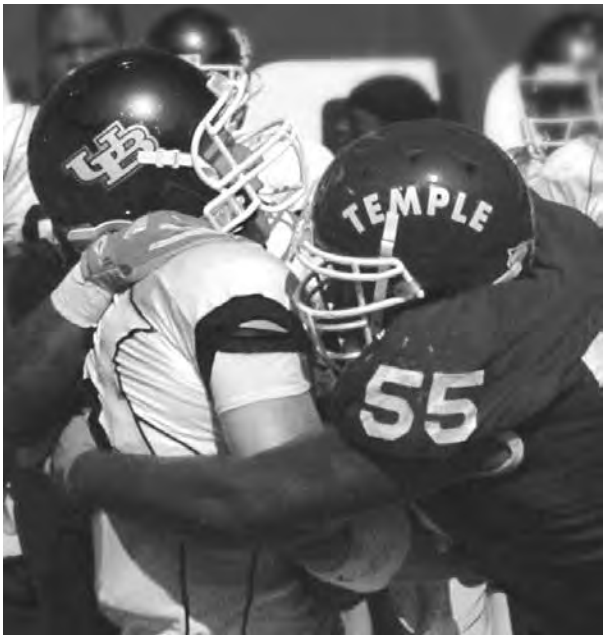
The Temple University Concussion and Athletic Neurotrauma Program

Cerebral concussion, traumatic brain injury, transient spinal cord paralysis and brachial plexus injuries are potentially serious insults to the nervous system that are associated with contact athletic injuries. In accord with the principle that the management and return-to-play decisions should only be made by a qualified professional, Temple University has established its **Concussion and Athletic Neurotrauma Program.**

Temple's experienced, multidisciplinary faculty is well-suited to evaluate and manage athletic-induced neurotrauma, utilizing the latest imaging capabilities, neurocognitive **ImPACT™** testing and clinically established **return-to-play** protocols.

Utilizing the facilities of Temple University Hospital, Temple Orthopaedics & Sports Medicine satellite offices, Temple Medical School faculty and in concert with the Shriners Hospitals for Children in Philadelphia, this program is designed to provide the necessary experience to meet the needs of team and family physicians, athletic trainers, athletic administrators, coaches, parents and, most importantly — the athletes.



Proper tackling technique protects both head and cervical spine.

Research Goals

Current understanding of cerebral concussion and athletic-induced traumatic brain injury is limited to a variety of descriptive classifications and epidemiologic patterns. Lacking is an application of the known underlying pathophysiology to clinical management practice with particular regard to injury prevention. Clearly, much is not known and there are many questions to be answered regarding athletically-induced neurotrauma. The goal of this program is to bring this issue to the same meaningful conclusion that Temple physicians achieved with paralytic spinal cord injuries 35 years ago.

Temple University Hospital

Clinical Program

Athletes sustaining impact injuries and experiencing any of the following signs or symptoms should be evaluated and, if indicated, managed by a physician experienced with athletic injuries to the head, spine and brachial plexus:

Central Nervous System

- Loss of consciousness
- Confusion
- Dazed appearance
- Forgetfulness
- Unsteady movements
- Slow cognition
- Personality changes
- Retrograde/antegrade amnesia
- Headache
- Dizziness
- Nausea or vomiting
- Altered sense of well-being

Spinal Cord

- Four extremity paresthesias (numbness)
- Four extremity weakness
- Four extremity transient paralysis

Brachial Plexus

- "Stinger" lasting more than 20 minutes
- "Stinger" with persistent weakness
- Recurrent "stingers"

The neurotrauma team consists of orthopaedic sports medicine specialists, neurologists, neurosurgeons, neurophysiologists, physiatrists and biostatisticians.

ATHLETES REQUIRING EVALUATION AND/OR MANAGEMENT CAN BE SEEN AT TWO OF TEMPLE'S CLINICAL SITES:

Temple University Hospital

3509 N. Broad Street
5th Floor Boyer Pavilion
Philadelphia, PA 19140
215-707-2111

Temple Orthopaedics & Sports Medicine Satellite Office

414 Commerce Drive
Fort Washington, PA 19034
215-641-0700

E-mail us at: concussion@tuhs.temple.edu
Website: www.templeconcussion.com

Temple Orthopaedics & Sports Medicine

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How to Reach Us

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Department of Orthopaedics, 6th Floor Outpatient Building
Philadelphia, PA 19140
Telephone: (215) 707-3411
Fax: (215) 707-7976

Letter from the Chairman

It is my pleasure to present this year's edition of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine.* Over the years, the *Journal* has evolved to represent the collaborative efforts of the Department's basic science and clinical research activity, contributions from the summer medical student research program, editorials and updates on the Department's extracurricular events.

The Temple University Health System continues to enhance its reputation of scholarly and clinical excellence at both Temple University Hospital and the Temple University School of Medicine. The concept of "Broad Street and Beyond" is coming to fruition, and the Department of Orthopaedic Surgery and Sports Medicine continues to play a prominent role in that vision. In the past year, we have partnered with other departments and TUHS to expand and improve our satellite presence: in Northeast Philadelphia in conjunction with Temple ReadyCare, in Oaks, PA along with Pulmonary Medicine, and in the coming year, a multispecialty facility in Fort Washington, PA.

That being said, the main campus on North Broad Street is a hub of academic, research and clinical excellence. The cornerstone of that campus is the Medical Education and Research Building, otherwise known as the newest medical school. It is that edifice that personifies the man to whom this edition of the *Journal* is dedicated, John Daly, MD, FACS. His tireless energy and dedication to the project have left him with a well deserved tangible representation of his term as Dean and leader of the Medical School.

As always, I wish to thank those organizations and people who make this all possible. The Temple/Shriners Alumni Association and the John Lachman Society members for their continued support of resident education, faculty members Saqib Rehman, MD and Eric Kropf, MD for the resident contributions and Joseph Torg, MD and Pekka Mooar, MD for the medical student research contribution; and Joanne Donnelly for keeping order and direction among those diverse entities. Congratulations to co-editors Matt Kleiner (PGY5) and Rick Tosti (PGY3) as well as Scott Barbash (PGY4) and Colin Mansfield (PGY2) for a job well done. With that said, we proudly present to you Volume 8 of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine.*

Joseph J. Thoder, MD
Professor and Chairman
Department of Orthopaedics and Sports Medicine
Temple University School of Medicine

Letter from the Editor-in-Chief

It has been quite a year for Temple Orthopaedics! Thanks to the hard work of several of our residents and attendings, we have continued to produce high-quality research that has garnered a great deal of national attention. At the 2012 American Academy of Orthopaedic Surgeons in San Francisco, our department had a total of three poster presentations, a scientific exhibit and two podium presentations, including "Best Presentation" awarded to Rick Tosti by the Hand and Wrist Committee for his presentation on "Is Antibiotic Prophylaxis Necessary in Elective Soft Tissue Hand Surgery?". In addition, faculty member Saqib Rehman, in collaboration with former Program Director Asif Ilyas, published a textbook, *Contemporary Surgical Management of Fractures and Complications.* Furthermore, Temple had several national publications in peer-reviewed journals over the past year — some of which will be featured in this year's *Temple Journal.*

Volume 8 of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine* is dedicated to John Daly, MD. Dr. Daly is a graduate of Temple University School of Medicine in 1973 and an accomplished surgical oncologist. He served as the Dean of Temple's medical school for eight and a half years before retiring from the position in 2011.

I have a lot of people to thank. First, I have to thank John Fowler for paving the way as the ultimate editor-in-chief and proponent of the *Temple Journal.* I would like to thank my co-editor-in-chief, Rick Tosti, as well as my fellow editors, Scott Barbash and Colin Mansfield, for their hard work and commitment to making Volume 8 come to fruition. I would also like thank faculty advisors Joseph Torg and Saqib Rehman for their tireless dedication and availability in making the *Journal* a rousing success. Finally, I would like to recognize Joanne Donnelly from the Office of Clinical Trials as well as Program Director Milo Sewards and Chairman Joseph Thoder for their continuous and unconditional support of all of our research efforts.

As an outgoing fifth year resident, I am overcome with a sense of pride in Temple Orthopaedics. In addition to the clinical prowess for which Temple has always been known, we have continued to grow as a leader in academics. I have no doubt that my fellow residents and attendings, with whom I have had the privilege to train, have repeatedly put me in positions where I can not only succeed, but thrive as a clinician and surgeon. Most importantly, over the past five years, I have forged friendships and relationships that will undoubtedly last a lifetime. The people at Temple have and always will be its greatest strength and I am extremely proud to have been a part of it. I believe the best way to reach your destination is to remember all the help you got along the way. For me, Temple will always be home and the foundation of my career success.

Thank you.

Matthew T. Kleiner, MD
Co-Editor-in-Chief
Class of 2013

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 also provides 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 is 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.

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

THE JOHN LACHMAN SOCIETY MEMBERSHIP — NOVEMBER 27, 2011

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Johnny C. Benjamin, Jr.
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Gene Shaffer, MD
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Michael Sitler, PhD
Gary Smith, MD
Charles Springer, MD
John Stelmach, MD
Edward J. Stolarski, MD
Zigmund Strzelecki, MD
Robert Sutherland, MD
Jay Talsania, MD

*Deceased

(Continued on next page)

Joseph Thoder, MD
Joseph Torg, MD
Joseph Trubia, MD
Warren T. Vance, MD
Bruce Vanett, MD
John Van Orden, MD
John B. Webber, MD
Paul Weidner, MD
Albert Weiss, MD
F. Todd Wetzel, MD
Eric Williams, MD
Gerald, Williams, MD
John Wolf, MD
Steven Wolf, MD
Thomas Yucha, MD

At the annual meeting of the board of directors of the John Lachman Orthopedic Research Fund, the following officers were re-elected for a one-year term:

President: J. Milo Sewards, MD
First Vice President: Phil Alburger, MD
Second Vice President: Eric Lebby, MD
Treasurer: Albie Weiss, MD
Secretary: Joe Torg, MD

The summer medical school intern program continues to be a most successful program. This past summer, 15 sophomore medical students participated in the program. In addition to a number of the students producing manuscripts suitable for publication in the *Journal,* it became evident that the major value of this program is that in view of the curriculum changes no longer requiring students to rotate through orthopedics, those students interested have an opportunity to interface with our department. Clearly, this has become a major avenue of acquainting students to the residency program.

Once again, the John Lachman Society published and distributed the *Temple University Journal of Orthopaedic Surgery & Sports Medicine,* Volume 7. Eighteen hundred copies of the *Journal* have been distributed as follows: a) active faculty of the Temple University School of Medicine, b) orthopedic surgeons who are alumni of Temple University School of Medicine, c) members of the John Lachman Society, d) department chairman and residency directors of all orthopedic programs throughout the United States, and e) fellowship directors to all orthopedic programs throughout the United States.

Academic support for resident travel to meetings by the John Lachman Orthopedic Research Fund during the period January 1, 2012 through December 31, 2012, involved 15 residents who have attended either formal courses or national meetings.

The Eighth Annual John Lachman Lecture was presented by J. Milo Sewards, MD at the annual meeting of the Pennsylvania Orthopaedic Society this past fall which was held in Pittsburgh. A decorated combat surgeon, Dr. Sewards presented his experience with advance management techniques of orthopedic injuries resulting from blast trauma. His talk was excellent and well received except for the few non-medical personnel in attendance who experienced syncope episodes at the sight of gross war trauma.

The John Lachman Society web page can be entered at www.johnlachmansociety.org.

In keeping with the request of the director of the residency program, the John Lachman Orthopedic Research Fund is committed to a $2,500 year expenditure for texts and other educational materials.

The John Lachman Society, through the John Lachman Orthopedic Research Fund and working in close cooperation with the Temple-Shriners' Alumni group, continues its mission to support and enhance both the academic program of the department and the orthopedic residency program.

Joe Torg, MD
Secretary

Report from the Residency Director

It is an honor for me to write the residency program director's introduction to the current edition of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine.* The *Journal* has been a tremendous source of pride for the department and residency, and it continues to bolster the program's reputation for clinical excellence. I would like to thank this year's editors, Matt Kleiner and Rick Tosti, as well as our faculty editors, Joe Torg and Saqib Rehman. I cannot sufficiently describe the amount of work that they all put into ensuring the successful publication of the *Journal* each year. Joe Torg, Pekka Mooar and Joanne Donnelly also deserve recognition for their stewardship of the summer research program, which is the source of an increasing number of manuscripts published in the *Journal.*

Interest in the Residency Program continues to grow. This past year, we received over 600 applications for our four PGY-1 positions. Also, changes are on the horizon again with respect to the guidelines by which we train the residents. Beginning with the next academic year, interns will become fully integrated into the Orthopaedic program instead of being General Surgery interns. While they will still get experience in various General Surgery rotations, they will each spend six months in Orthopaedics. During that time, they will complete a curriculum in surgical skills, using simulation ranging from the familiar suture boards to the use of our space in the anatomy lab in the medical school. All of your contributions to the Lachman Society are very much appreciated as we expect to be requesting grants from the society to support these teaching efforts.

Thanks to all of my colleagues on the faculty at Temple as well as our affiliate institutions, and the supporting members of the John Lachman Society, I continue to have the privilege of directing a strong residency program that improves each year.

J. Milo Sewards, MD

Update from the Department of Orthopaedic Surgery and Sports Medicine Office of Clinical Trials and Research Support

The Office of Clinical Trials and Research Support was established in 2004, under the direction of Pekka A. Mooar, MD and supported by the School of Medicine's Office of Clinical Research Administration, with Ms. Joanne Donnelly as the full-time research and program coordinator.

We have been on this exciting journey for the past nine years, and achieved the goals originally set forth: establishing a stream of clinical trials for all interested attending surgeons creating direction support and encouragement to the residents in their pursuit of investigator initiated research, fostering collaborative projects with Pathology, Microbiology, Radiology, Anesthesia and continuing our work in the Basic Science departments of Anatomy and Cell Biology.

We have also established and maintained the popular Temple Medical Student Summer Orthopaedic Research Program (TSSORP) under the leadership of Dr. Torg and supported by Dr. Mooar and myself. This program provides any Temple medical student interested in Orthopaedics with an eight-week summer program on how to implement a research study with a hands-on approach. Students have an orientation which includes our research librarian and statistician. Students come away knowing how to think of a research topic, perform the literature search, submit the necessary documents to the Temple IRB for approval, data mine from our new electronic medical record system and, most of all, begin the writing process. We meet with the students on Tuesday and Wednesday mornings to go over their projects and offer guidance and assistance. Students are also encouraged to go to the OR and see as many cases as they wish during this time, as well as attend office hours. After this program, students have a keen insight into Orthopaedics from a truly unique prospective. Students also get to be first author on their research projects published in the TUJOSSM journal.

I am happy to report that this summer we will host 12 Temple Medical Students into the program. Below is a list of projects that we will undertake:

2012 Summer Medical Student Research Projects:

See Journal under "Medical Student Research Projects"

2013 Summer Medical Student Research Projects:

Dr. Torg *(4 projects)*
- Reliability of Neurocognitive Testing: A Pilot Study *(continued from last year)*
- Performance Enhancing Drugs and Morality: A Commentary
- Conflict of Interest: A Real or Imagined Problem in the Practice of Orthopaedics
- Pre-Disposing Factors Responsible for Post-Concussion Sequalae

Dr. Mooar *(1 project)*
- Is TNT (Toradol, Neurontin,Tylenol) an Effective Post-Op TKA, THA Pain Management Program?

Dr. Weiss *(1 project)*
- Financing of Orthopaedic Graduate Medical Education: The Role of Non-Profits in the Development of Extramural Funding *(continued from last year)*

Dr. Rehman *(3 projects)*
- Development of a Clinical Treatment Guideline for the Orthopaedic Management of Open Long Bone Fractures at TUH
- Development of a Clinical Treatment Guideline for the Orthopaedic Management of Femoral Shaft Fractures at TUH
- Development of a Clinical Treatment Guideline for the Orthopaedic Management of Proximal Tibial and Tibial Shaft Fractures at TUH

Dr. Kropf *(1 project)*
- Tunnel Enlargement Socket: Aperture Fixation vs Tight Rope Fixation in ACL Reconstruction

Current Industry-Sponsored Clinical Trials Drug or Device:

Smith and Nephew
(TRUST) Trial to Evaluate Ultrasound in the Treatment of Tibia Fractures
Saqib Rehman, MD, Principal Investigator; Alyssa Schaffer, MD, Sub-Investigator; J. Milo Sewards, MD, Sub-Investigator, Phase IV Device. Enrollment completed, 21 subjects. Study closed to enrollment.

Baxter Healthcare
A Prospective Randomized Controlled Single-blinded Multicenter Study to Evaluate the Efficacy and Safety of Floseal® for Hemostasis in Primary Unilateral Total Knee Arthroplasty (TKA)
Pekka Mooar, MD, Principal Investigator, Phase IV Device. Enrollment completed, 3 subjects.

Stryker
(INSITE) Intramedullary Nail Versus Sliding Hip Screw Intertrochanteric Evaluation: A Multi-Center Randomized Controlled Trial of Intramedullary Nail Versus Sliding Hip Screw in the Management of Intertrochanteric Fractures of the Hip
Saqib Rehman, MD, Principal Investigator; Bruce Vanett, MD, Sub-Investigator; Christopher Haydel, MD, Sub-Investigator, Phase IV Device. Enrolling 7 subjects to date.

EMSI
The Electrostim Medical Services, Inc. (EMSI) Bone Growth Stimulator (BGS) Clinical Study for the Treatment of Long Bone Fractures Acquired Secondary to Trauma Where Serial Radiographs Taken at Least 90 Days Apart Have Shown No Visible Progressive Signs of Healing
Pekka Mooar, MD, Principal Investigator, Phase IV Device (starting March 2013).

Department of Defense
Assessment of Severe Extremity Wound Bioburden at the Time of Definitive Wound Closure or Coverage: Correlation with Subsequent Post-Closure Deep Wound Infection (Bioburden Study)
Saqib Rehman, MD, Principal Investigator. Prospective cohort observational study (March 2013).

Current Investigator and Resident Initiated Studies Coordinated by the Office:

The Prevalence of Methicillin Resistant Staph Auresus (MRSA) Colonization Among Resident Physicians at an Urban Teaching Hospital. *Renewed until 13-Jan-2014.*
Alyssa Schaffer, MD, Principal Investigator (IRB Approval #13721)

Comparison of Autograft and Allograft ACL Reconstruction: Long Term Histologic Analysis. *Renewed until 13-Jan-2014.*
Eric J. Kropf, MD, Principal Investigator (IRB Approval #13719)

S. Ali, S. Huebner, F. Groshek, A. Schaffer. The Floating Fat Sign of Trauma. *Canadian Association of Radiologists Journal.* Accepted for publication.

S. Popinchalk, A. Schaffer. Physical Examination of Upper Extremity Compressive Neuropathies. *Orthopedic Clinics of North America* 2012;43(4):417–430.

Joanne Donnelly

John M. Daly, MD
Dean of Temple University School of Medicine 2002–2011

Dedication

John Michael Daly, MD

JOSEPH TORG, MD

As suggested by Joe Thoder, Department Chair, and unanimously and enthusiastically agreed upon by the editorial board, this volume of the *Temple University Journal* of *Orthopaedic Surgery & Sports Medicine* is dedicated to John M. Daly, Dean of the Medical School from 2002 to 2011. And what a dean he was: surgeon extraordinaire, internationally renowned academic animal, leader of men, visionary and role model for the ages. Such platitudes clearly describe the man but first let us examine his short comings.

I have known John Daly for the past 38 years. Our first encounter occurred when he was a medical student at Temple on an orthopedic rotation and I was a young faculty member. As a student, he appeared capable, enthusiastic, and destined for a career as an orthopedic surgeon. But as we all know, he rejected or was rejected by orthopedics and pursued a general surgery residency. What happened? Was he not physically capable of managing large bone and joint problems? Was he too intellectually challenged to handle the concepts of orthopedic principles and practice? Did he lack the eye-hand coordination and cognitive skills to deal with the arthroscope and other advanced orthopedic devices?

The house that Daly built

John Daly, a general surgeon! Such a disappointment! But he did recover as an itinerant surgeon at the University of Texas School of Medicine, Cornell University Medical College, the Weill Medical College of Cornell University, and the University of Pennsylvania, where our paths crossed once again.

And let me share another Daly vignette that occurred when we were both on the staff at Penn back in the 80s. While vacationing at the Jersey shore, I was asked by a friend to see a patient who had been admitted to a local hospital and was scheduled for a "diagnostic laparotomy" because of what was undiagnosed abdominal pain. Having spent the first year of my orthopedic residency at Temple on general surgery and being somewhat vaguely familiar with inter-abdominal problems, opening the belly without a diagnosis just didn't seem cool. So I called my good friend John Daly, explained my concern and arrangements were made to transfer the patient to Penn. And my good friend John Daly then, on the basis of his extraordinary clinical acumen and experience, diagnosed acute mumps pancreatitis, initiated appropriate conservative, non-surgical management that resulted in a complete recovery without sequela. What made the experience more meaningful was that surgical intervention would have most certainly resulted in disaster. And my good friend John Daly — what a guy — clinician, diagnostician, academician — the whole enchilada.

But let us examine John Daly in a more serious vein. I can attest to the fact that he is a man of faith, has been a committed husband and father, a most supportive friend of Temple Orthopaedics and the School of Medicine and a great a humanitarian. With an unassuming demeanor, he maintains a low profile, avoids rhetorical bombast and self promotion, and is modest almost to a fault.

And did he recover as a general surgeon! During his travels, he produced 250 publications in peer review journals, amassed a total of $2,831,000 as principal investigator in 20 research grants, and was awarded honorary doctorate degrees from both the University of Glasgow and the University of Dublin — a classic example of Irish cohesiveness.

But let's talk about his really important contributions. John Daly's tenure as Dean of the Medical School was initiated by the threat of the loss of the academic accreditation of the medical school. And with diligence and foresight, he initiated a transformital program that involved the academic curriculum, faculty acquisition and, of course, the design and construction of the new Medical Education and Research Building, the signature accomplishment of a magnificent career. And today, the MERB is euphemistically referred to as "Daly's finest erection."

Commentary

Electronic Medical Records: An Epic Disaster?

JOSEPH TORG, MD

What can we say about electronic medical records? First is to recognized that they are potentially preferable to the traditional medical records which are non-potable, segmented, often incomplete and subject to the varies of unintelligible physician handwriting.

So now we have electronic medical records which we have been told will vastly improve patient care, implement cost-effective measures that will dramatically reduce costs, and facilitate practice models so as to maximize physician efficiency and effectiveness. Available evidence clearly indicates that just the opposite is occurring. Let me explain.

According to a recent report by the Rand Corporation published in the January 2013 edition of *Health Affairs,* electronic health records have not produced a decrease in health care costs but have rather seen an increase of $800,000,000,000 (that's $800 billion!) since their first report in 2005. This is in contradistinction to their initial prediction that the widespread use of EMRs could save the health care system $81 billion annually. So what happened? First, Rand admits that their earlier estimate was "overly optimistic" as was the Congressional Budget Office overstatement of project savings. Second, Congress and the Obama administration spent billions of dollars in federal stimulus money to encourage doctors and hospitals to pay for the installation of electronic record systems. And lastly, critical analyses of current systems clearly indicate that they are much more oriented to increasing provider billings than in improving medical care or cost effectiveness. As reported by Reed Abelson and Julie Creswell recently in the *New York Times,* "there is increasing concern that electronic records have actually added to costs by making it easier to charge more for some services." They also point out that RAND's 2005 report was paid for by a group of companies "that have profited by developing and selling electronic records systems to physician practices and hospitals. To be noted, one of these companies, Cerner Corporation, revenues has nearly tripled from $1 billion in 2005 when his report was released to a projected $3 billion in 2013.

In a report on how the growth of electronic medical records eases the path to inflated medical billing, Fred Schulte points out that in the rush to get the program off the ground, federal officials failed to impose adequate controls over billing software despite warnings from "several prominent medical fraud authorities." As a result Medicare, regulators now acknowledge they are struggling with a surge of aggressive and expensive billings by doctors and hospitals linked to the rapid proliferation of billing software and electronic medical records. And it has been acknowledged that billing abuse (fraud) has taken a back seat to steps to entice the medical community to embrace the new technology. This, of course, is in keeping with the thinking that two wrongs make a right.

The recent report of Daniel R. Levinson, Inspector General of the Department of Health and Human Services entitled "Early Assessment Finds that CMS Faces Obstacles in Overseeing the Medicare EMR Incentive Program" is clearly indicative of the conundrum that the rush to implement EMR has resulted. Predicated on the "new talk" concept of "meaningful use" in a 150-page manual on electronic medical records, "meaningful use" remains an enigma. However, it presumably has not been determined "whether professionals' and hospitals' self-reported meaningful use information met meaningful use measure criteria." But CMS does not verify the accuracy of professionals' or hospitals' self-reported meaningful use information prior to payment or as well as post-payment audits. As well, the report concludes that electronic health record technology is not sufficient to verify self-reported information and may not always be accurate.

Dr. Anne Marie Valinoti has clearly expressed my views in her recent article published in the *Wall Street Journal* where she point out that "it seems that this is all about taking care of the chart as opposed to taking care of the patient . . . in that demonstrating meaningful use of EMR may be getting in the way of meaningful encounters with our patients." "It's hard to be both stenographer and empathetic listener at the same time."

It is my view that electronic medical record systems have been introduced without adequate "clinical trials" by a governmental bureaucracy intent on controlling a large segment of the economy with a "damn the torpedoes — full speed ahead" mentality. I would further suggest that the major burden of implementation has been thrust upon the physician now saddled with the collection of worthless data by an inordinately complicated and time-consuming medical records system. It is generally agreed by users of the system that EMR decreases physician efficiency by 25% and this impression has been substantiated by system implementation of decreasing patient scheduling loads by 25%. Of course, the ready explanation of system implementers is that this is a function of the learning curve and familiarity with

the system will resolve the increased time expenditure phenomenon. I submit that the inordinate time-consuming factors are systematic and unrelated to user familiarity.

User familiarity will not obviate the labyrinth of diagnostic "bullets" and billing-orientated quires to justify level of service.

User familiarity will not obviate the multiple log-in/password issues compounded by the absurd, short automatic bureaucratic log out times presumably imposed to protect patient confidentiality. What happened to the user ID card swipe system available for other systems including later Epic editions?

User familiarity will not obviate the time necessary for physicians to perform the functions of a transcriptionist and edit their dictations or typewritten documentation.

User familiarity will not obviate the delay in the incorporation of radiology images within the electronic medical record.

User familiarity will not obviate screens that require repetitive selection of facility, location, or service.

User familiarity will not obviate network and printer server failures that limit access and functionality to the electronic medical record system.

So there you have it — a politically motivated, inordinately expensive, user unfriendly system that has yet to demonstrate an ability to improve the quality of patient care.

Commentary

EMR Is the Way to Go

Saqib Rehman, MD

EMR is the way to go. Rather than fight it, we should work to optimize it.

Imagine that you go to your bank to make a withdrawal, they pull out your paper record file, realize something is missing, then make their best estimate at what your balance is. Or you make an airline reservation with a travel agent who doesn't use computers and has to put everything in a paper file and issue a paper ticket. Or you take your car to the dealer to be serviced, but they can't find that paper file on your car to know when your last oil change and tire servicing was. Of course, these situations would be considered unacceptable to most Americans in 2013. So how can it possibly be acceptable to keep medical records in paper charts, especially knowing what we know as physicians? When just about everything in our world is being computerized for obvious reasons of efficiency, space-saving, accuracy, and accessibility, how can we possibly cling to paper medical records?

As a practicing physician, I will readily admit that some aspects of seeing patients in the office is better without EMR. Dictating into a Dictaphone on the fly as I go from patient to patient is certainly faster than having to type or use Dragon software. But that's about where the advantages end.

How does a health system-wide EMR help me? Let me count just some of the ways:

1. Progress notes: If I need to look up my old notes in a paper chart, I'm at the mercy of whoever has arranged the papers in the chart (and God forbid it had to be recently pulled out and photocopied for legal or other purposes). Obviously not an issue in EMR.

2. Lab results and imaging reports might be in there if I look, but if it's not there, that could just mean that it fell out of the chart.

3. Prescriptions: When did I last prescribe mobic for this patient? Who knows? I would have to go through all those carbon copy scripts that are in that little envelope in the chart, hoping that they are all there.

4. Stolen Rx pads? It's happened to me. Not a problem when you are eprescribing. I thought Dr. Torg wrote a paper on the benefits of eprescribing . . .

5. No more chart multiplicity: With paper charts, there would be multiple charts in different locations with different things in them. And I would not even consider trying to find notes on this patient from other physicians, which are also in their own paper charts sitting on a shelf in yet another office.

6. Drug checker: My EMR can tell me if there are unsafe drug-drug interactions. (Previously, I had to pull out my smartphone, open Epocrates, and run a check myself.)

7. Reports easy to find: Need that EMG report or other report that other departments in your hospital do a crappy job in providing to you? Your staff looks bad, and you look bad as your patient sits waiting inexplicably as you try to get the report. With EMR, it's now right there — no waiting around.

8. More thorough documentation for billing purposes. With macros and shortcuts, I can make sure that those compliance people are not on my case about underdocumentation of history that was collected and examination that was done.

9. Better documentation of patient phone calls/requests. With paper charts, every method that we tried didn't seem to work. With EMR, there is a clear, consistent, and accurate method that is properly documented.

10. It's a better research tool. Our particular EMR is not quite there yet, but the accuracy, consistency, and accessibility of electronic data far surpasses having to sift through boxes of charts with no way to scan through data other than to leaf over every page. We have been literally unable to do certain research projects because of the resources needed to do massive chart reviews on crappy paper charts with useless (often illegible) information in them. That being said, our particular EMR, and most EMRs, are not optimized for research purposes. This has been somewhat of a disappointment, but we are working to improve this by integrating our EMR data with third party solutions for research data registries and searchable data warehousing.

11. Accessibility: For the obsessive doctor, EMR allows them to check records on patients from multiple offices, from their home, and even from their smartphone. For the doctor who is not quite obsessive, but simply wants the information there when they ask for it, how can you go back to "waiting for the paper chart" or going by memory about a patient's condition. Taking this a step further, the increased accessibility appeases to the obsessive patients, who want to have access to at least a part of their medical record. Many EMRs (including ours) allow patients to do this. This, in turn, also helps researchers reach out to patients to conduct questionnaire surveys.

Good medicine and careful documentation go hand in hand. Our profession would not have made significant

advances in patient care without attention to accurate medical documentation. If you are a doctor who doesn't ever look in the chart, then EMR is clearly an obstacle to efficient clinic hours. But I would argue that that is just not good medicine. Just as the American pubic, on the whole, will be demanding that their physicians have accurate documentation of their medical history, we as physicians should recognize that EMR, for many of the reasons I have outlined above, is simply better medicine. Clearly, EMR is in its infancy compared with electronic data management in many industries. But it is the future, and is not going away. Rather than oppose it, we should be looking for ways to improve it, particularly with regards to research optimization.

Distinguished Alumnus

John A. Bergfeld, MD

This issue of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine* marks the third Editorial Board's selection of a Distinguished Alumnus who has graduated from the Temple University School of Medicine and/or completed his or her Orthopedic residency at the Temple University Hospital. The purpose of this selection is to acknowledge the accomplishments of individuals who have excelled in their academic, scientific, and/or humanitarian endeavors. The acknowledgement will be made when appropriate and not necessarily on an annual basis.

It is a distinct pleasure for the editorial board to recognize John Bergfeld, a graduate of the Temple University School of Medicine, for numerous service and academic accomplishes over his 40-year career. He is a graduate of Bucknell University where he excelled as a hard-hitting fullback on the football team. He then entered Temple and achieved two major accomplishments: met his wife Wilma and received a Doctor of Medicine degree.

After completing his residency, he served in the Navy as Chief of Orthopaedics of the United States Naval Academy, and Naval Hospital, Annapolis, MD and aboard the USS Dubuque (1970–1973) with rank of Commander MC USNR.

John has served as a Board member of the American Academy of Orthopaedic Surgeons, 1997–2003; President of the American College of Sports Medicine, 1984–1985; President of the American Orthopaedic Society for Sports Medicine, 1992–1993, President of the International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine, 2005–2007; and Chairman, International Anterior Cruciate Ligament Study Group.

He has served for 26 years as Team Physician for the Cleveland Browns of the NFL as well as for the Cleveland Cavaliers of the NBA from 1986–2001. He also has been physician to the Cleveland Ballet, 1976–1990; Baldwin Wallace College, 1996–present; and the Cleveland Metropolitan Schools, 1976–present. He presently serves as consultant to the Cleveland Browns and Cavaliers and has presented multiple endowed lectureships, both in the USA and internationally.

As the Head of the Section of Sports Medicine at the Cleveland Clinic from 1976 to 2002, he has trained over 50 post graduate fellows (known as the Warthog Society), several of which are physicians for professional teams, Division I colleges and chairmen of their Departments of Orthopaedics. He has published over 90 articles for international/national publications, books and chapters.

John's academic, research, and teaching endeavors have covered the spectrum of orthopedic sports medicine. However, he is somewhat of an exception in his appreciation of the fact that the anterior cruciate ligament is not the only structure in the knee. Clearly, his interest and work dealing with both the function and problems of the posterior cruciate ligament have been exceptional. We, therefore, find it appropriate to republish his paper "A Biomechanical Comparison of Posterior Cruciate Ligament Reconstruction Techniques," previously published in the *American Journal of Sports Medicine.*

Clearly, John Bergfeld's many contributions to orthopedic surgery and orthopedic sports medicine have reflected extremely well on himself, his associated institutions, and the Temple University School of Medicine. The editorial board is delighted and honored to recognize John as a Distinguished Alumnus.

Joe Torg, MD

Distinguished Alumni Paper

A Biomechanical Comparison of Posterior Cruciate Ligament Reconstruction Techniques

JOHN A. BERGFELD, MD, DAVID R. MCALLISTER, MD, RICHARD D. PARKER, MD, ANTONIO D.C. VALDEVIT, MSC, HELEN E. KAMBIC, MS

The Cleveland Clinic Foundation, Cleveland, Ohio

Abstract

Most posterior cruciate ligament reconstruction techniques use both tibial and femoral bone tunnels for graft placement. Because of the acute angle the graft must make to gain entrance into the tibial tunnel, abnormal stresses are placed on the graft that could lead to graft failure. An alternative technique for posterior cruciate ligament reconstruction involves placement of the bone plug from the graft anatomically on the back of the tibia (inlay), preventing formation of an acute angle at the tibial attachment site. We used six pairs of human cadaver knees to compare the biomechanical properties of these two techniques. One knee from each pair underwent tunnel reconstruction while the other knee underwent inlay reconstruction. There was significantly less anterior-posterior laxity in the inlay group when compared with the tunnel group from 30° to 90° of knee flexion and after repetitive loading at 90° of knee flexion. Evaluation of the grafts revealed evidence of mechanical degradation in the tunnel group but not in the inlay group. The inlay technique resulted in less posterior translation with less graft degradation than did the tunnel technique for posterior cruciate ligament reconstruction.

The PCL is the primary restraint to posterior tibial translation in the intact knee. It has been reported that 5% to 20% of all ligament injuries to the knee involve the PCL.[4, 9, 13, 14, 23, 29] Posterior cruciate ligament injuries have been divided into those that are isolated and those that are combined with other injuries. Posterior cruciate ligament injuries that occur with other major knee ligament injuries appear to fare better with surgical reconstruction rather than nonoperative management for symptoms of instability. The appropriate treatment for isolated PCL injuries is less obvious because instability is often asymptomatic. Although the natural history of these injuries is unclear, there is evidence that certain PCL injuries will lead to instability, pain, and osteoarthritis of the knee.[6]

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Several studies have reported good results with nonoperative treatment for isolated PCL injuries,[11, 12, 16, 26, 32] while other studies have shown poorer long-term results that deteriorate with time.[8, 21] Increased articular contact pressures have been demonstrated in the medial and patellofemoral compartments after sectioning of the PCL in cadaver knees,[31] which probably accounts for the increased incidence of degenerative changes observed in these compartments in patients with a PCL-deficient knee. Furthermore, damage to the articular cartilage of these compartments may be underestimated when viewed by conventional radiographs.[8] Although several clinical studies have documented degenerative changes in the medial compartment and patellofemoral joint after isolated PCL injuries, the severity of these changes does not appear to be related to the amount of abnormal posterior laxity.[6]

Thus, the indications for PCL reconstruction are not clear-cut, and whether PCL reconstruction will alter the natural history of the PCL-deficient knee is currently unknown. Clinical results after PCL reconstruction have not been as predictable as those after ACL reconstruction.[1] Current techniques for PCL reconstruction in general have yielded inconsistent results and do not appear to eliminate abnormal posterior laxity in the knee.[22] It is currently unclear whether PCL reconstruction techniques can restore normal knee laxity *in vivo* and thus alter the natural history of the PCL-deficient knee.

Most PCL reconstruction techniques use both tibial and femoral bone tunnels for graft placement (Fig. 1). Grafts placed through the tibial bone tunnel are required to make at least a 90° turn at the posterior aspect of the tibia. Placing this degree of bend in the tendon graft at the tibial tunnel creates increased internal tendon pressures.[30] In addition, the acute angle that the graft must make on the posterior tibia may predispose the graft to "saw" on the tibia and further degrade its mechanical integrity. Furthermore, the normal anatomy of the PCL is not reproduced by the tunnel technique in which the graft must enter the joint through a tibial drill hole. This drill hole entry site is difficult to locate properly, especially if the procedure is performed arthroscopically. These factors may contribute to the variable clinical results after PCL reconstruction.

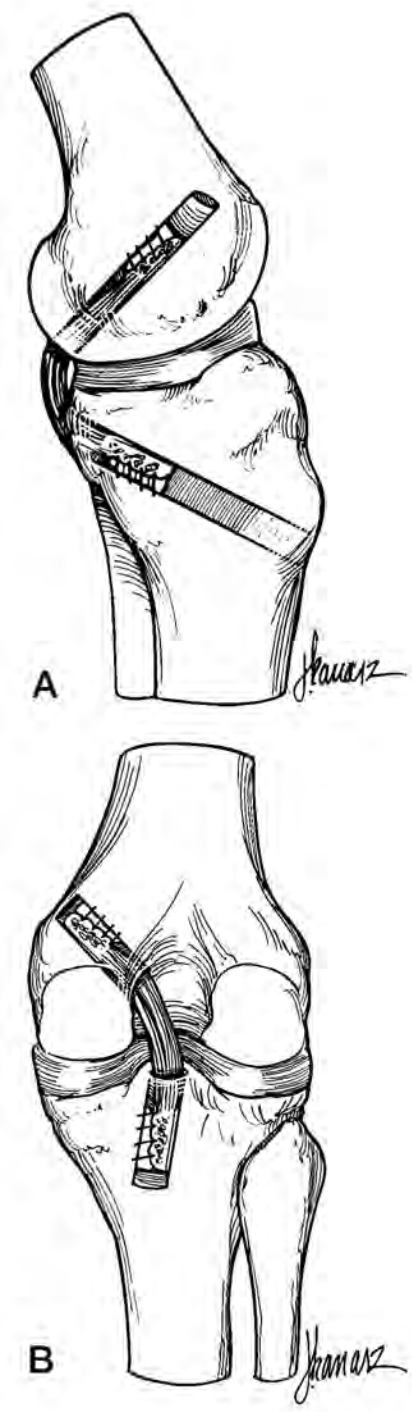

Figure 1. Lateral **(A)** and AP **(B)** views of the tunnel technique of PCL reconstruction.

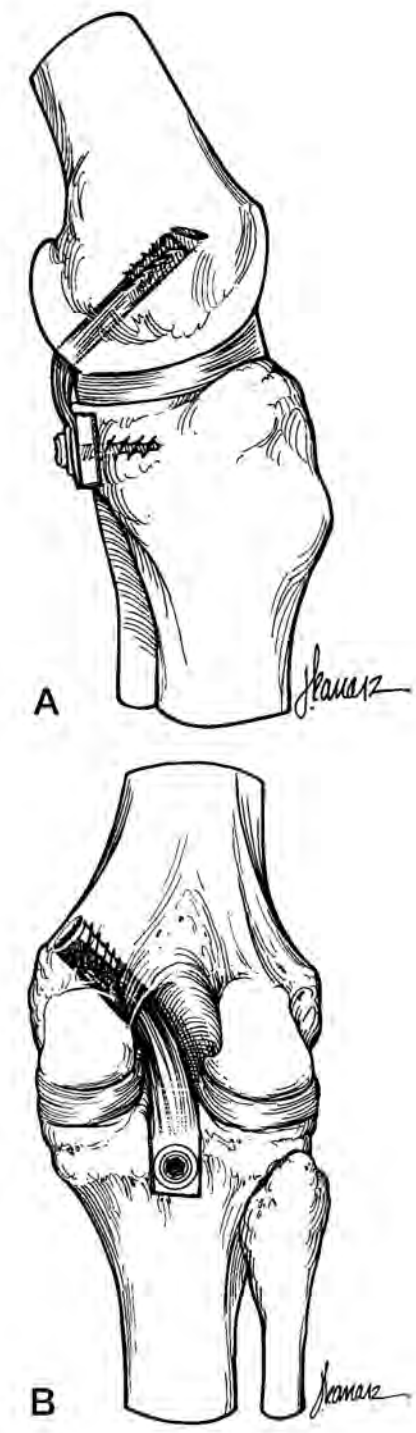

Figure 2. Lateral **(A)** and AP **(B)** views of the inlay technique of PCL reconstruction.

Most of these factors could be avoided if the orientation of the tibial attachment site of the graft could be altered. The anatomic tibial inlay technique of PCL reconstruction uses the same femoral bone tunnel but employs direct screw attachment of the bone plug from the graft to the proximal posterior tibia (Fig. 2). This technique more closely replicates the anatomic insertion site of the PCL on the proximal posterior tibia and avoids the excessive bending of the graft to gain entrance into the tibial bone tunnel that is necessary in the tunnel PCL reconstruction technique.

Jakob and Rüegsegger[20] as well as Berg[3] have previously described this technique of PCL reconstruction. Although they have advocated this technique for its theoretical advantages regarding graft fiber orientation, there have been no

substantial clinical or basic research studies to validate this concept. The purpose of this study was to compare the anterior-posterior (AP) stability provided by the inlay technique and the tunnel technique of PCL reconstruction. Specifically, we compared AP laxity at four positions of knee flexion and three positions of tibial rotation for the tunnel and inlay techniques of PCL reconstruction.

Materials and Methods

Six pairs of fresh-frozen cadaver knees were used in this study. Donors were all men with a mean age of 65 (±9) years. Only knees without evidence of knee abnormalities, including prior knee surgery, abnormal knee ligament laxity, or significant degenerative joint disease, were included. Specimens were amputated transfemorally and transtibially.

Specimen Preparation and Instrumentation

The knees were thawed, and the tibia and femur were scraped clean of soft tissue to within 10 cm of the joint line. Skin, subcutaneous fat, and muscle around the knee joint were excised, leaving only the joint capsule, ligaments, popliteus muscle, and extensor mechanism. The portion of the fibula distal to the fibular neck was resected, and the proximal tibiofibular joint was stabilized with a 4.5-mm cortical screw. The proximal end of the femur and distal end of the tibia were potted in aluminum tubes with polymethyl methacrylate. Transfixion pins within the tubing were used to eliminate rotation at the polymethyl methacrylate-tube interface. A custom-made testing apparatus with six degrees of freedom was constructed, similar to that described by Fleming et al.[15] A Bionix 858 MTS with Testar software (MTS Systems Corporation, Eden Prairie, Minnesota) was used to apply shear loads to the knee while displacement was measured. The potted femoral end of the specimen was clamped into a yoke attached to the load ram of the MTS, which applied an AP shear force to the knee. The epicondylar axis of the knee joint was placed at the pivot axis of the femoral yoke. The knee joint flexion angle was adjusted with the yoke and locked at the desired flexion angle before testing. A bearing system allowed free motion of varus-valgus angulation. The potted tibial end was mounted in a clamp housed within a bearing mechanism that allowed axial rotation and could be locked if desired. The tibial bearing mechanism was mounted on the plate of an X-Y table, allowing free translation of the tibia in the coronal plane (Fig. 3).

Anterior-posterior displacement was measured as the amount of translation of the tibia relative to the femur in the midsagittal plane. An AP ramp load of 150 N was applied to the femur and measured by the MTS load cell. The linear variable displacement transducer of the MTS measured the amount of displacement of the femur. Tibial rotation was locked during AP testing. Neutral rotation was defined as midway between tibial rotations produced by 5 N•m of internal and external tibial torque, as previously described by Markolf et al.[24] Torque was measured with a calibrated torque wrench that was applied to the tibial pot with the tibial clamp unlocked, allowing free rotation. Positions of internal and external rotation were defined as those resulting from application of 5 N•m of internal and external tibial torque. The yoke of the testing apparatus held the knee in various amounts of knee flexion while an AP force was applied with the MTS machine.

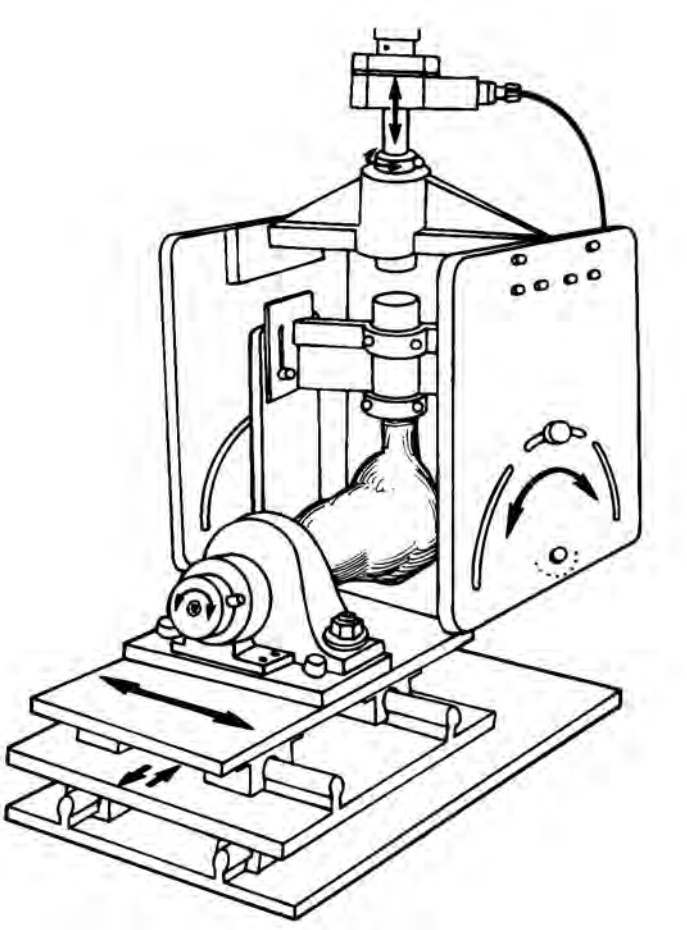

Figure 3. The custom-made testing apparatus allowing six degrees of freedom.

Testing Procedures

The intact knees were mounted on the custom-made apparatus and subjected to AP testing at 0°, 30°, 60°, and 90° of knee flexion with the tibia locked in neutral, internal, and external rotation. A 150-N AP force was applied at a rate of 0.2 Hz to the femoral yoke at the level of the joint line. Tibial rotation was allowed between tests when the knee flexion angle was being changed; neutral rotation was redefined at each new knee flexion angle. Tibial rotation was locked at various knee flexion angles while varus-valgus angulation was unconstrained and the tibia was allowed to translate in the coronal plane during AP loading. Under each testing condition, the knee was loaded six times. The first three loading cycles were used to precondition the knee, while the

latter three loading cycles were used for data collection. In all cases the load-deformation curve became reproducible after two loading cycles.

Anterior-posterior knee laxity was defined as the AP knee displacement of the tibia that occurred relative to the femur between the limits of 150-N anterior and 150-N posterior loads. This convention was chosen to load the ACL and used as a reference. Because the ACL was not transected, it is reasonable that the reference for displacement measurement was an anteriorly applied load to the tibia that engaged the ACL. These tests were performed using the load-defined feedback loop option on the MTS system. The neutral position for AP translation was defined as the inflection point identified during anterior loading, representing loading of the ACL. This zero-load position of the MTS was maintained while changing the knee flexion angle and tibial rotation between testing cycles.

PCL Sectioning

After laxity data had been obtained from the intact knees, a posterior arthrotomy was made, and the PCL and meniscofemoral ligaments were transected. The meniscofemoral ligaments were transected because they are closely associated with the PCL and have been shown to contribute to posterior knee stability.[19] The arthrotomy was repaired, and AP loading was repeated under the previously listed testing conditions. Because the specimen was not removed from the testing jig between the loading conditions, the zero-load neutral position could be maintained throughout the testing sequence.

PCL Reconstruction

One of the knees from each pair was randomly assigned to the tunnel group and underwent a tibial tunnel PCL reconstruction technique. The contralateral knee was assigned to the inlay group, which had the PCL reconstructed using the anatomic tibial inlay technique, involving direct screw attachment of the graft bone plug to the proximal tibia. Autogenous central one-third bonepatellar tendon-bone grafts with 30-mm bone plugs were harvested and used as the graft material in both groups. In both groups, a 10-mm femoral tunnel was made that entered the knee in the distal anterior footprint of the native PCL in the region of the isometric point, as previously described.[5, 7,17] The bone tunnel was made using a drill and a PCL femoral drill guide (Acufex, Mansfield, Massachusetts). In both groups, the posterior capsule was opened to expose the proximal posterior tibia.

In the tunnel group, a drill and the PCL tibial drill guide were used to make a tibial tunnel that exited the proximal posterior tibia approximately 1 cm distal to the joint line, in the center of the tibial insertion of the PCL. The tibial side of the graft was secured with a 9 x 20 mm interference screw (Acufex). In all cases, the bone plug was placed with the patellar tendon fibers positioned anteriorly, while the screw was placed in the posterior aspect of the tunnel, between the bone plug and the posterior tunnel wall. The bone plug was placed 5 mm inferior to the opening of the tibial tunnel in all cases.

In the knees in the inlay group, a bone trough was created with osteotomes at the insertion site of the PCL on the posterior proximal tibia. The tibial side of the graft was secured flush to the posterior surface of the proximal tibia with a 4.5-mm bicortical screw (Synthes, Paoli, Pennsylvania) and a washer and a nut. This method of fixation differs slightly from that used clinically in that a 6.5-mm cancellous screw and washer are used for patients. This slight modification was necessary to achieve stability in the older osteoporotic specimens used in this study.

The grafts were tensioned in a manner similar to that described by Pearsall et al.[27] In both groups, the tibial side of the graft was secured first, and the femoral side of the graft was tensioned to 89 N with the knee flexed 90°. An anterior force of 156 N was applied to the proximal tibia, simulating an anterior drawer maneuver, and a 9 x 20 mm femoral interference screw (Acufex) was placed to secure the femoral side of the graft. Because of the osteoporotic nature of the specimens, fixation of the grafts to the tibia and femur was augmented with polymethyl methacrylate at the bony interfaces between the graft bone plug and the bone tunnel or trough. Laxity testing was repeated under the same conditions as previously described.

Effect of Repetitive Loading

After completion of mechanical testing of the PCL-reconstructed knees (72 cycles), the knees from both groups were tested again with a 150-N AP force with the knee flexed at 90° and in neutral tibial rotation to determine the effects of repetitive loading.

Graft Evaluation

At the conclusion of mechanical testing, the grafts were removed from the knees of both groups and inspected for defects. The grafts were evaluated for thinning in the region of the proximal posterior tibia by gross inspection and quantified with relative optical density measurement using BioQuant software (R & M Biometrics, Nashville, Tennessee). Standard red-green-blue (RGB) measurements were made with the resultant density determined by the square root of $R^2 + G^2 + B^2$.

Statistical Analysis

The data consisted of translational measurements (in millimeters) from the same knee under three different loading conditions (intact, PCL cut, and PCL reconstruction) measured at several different angles. This introduced a correlation structure between the observations obtained from the same knee. Therefore, repeated-measures analysis of variance was used for statistical analysis. Pair-wise comparisons between different conditions were performed using the Bonferroni adjustment for multiple comparisons. All modeling

was performed using SAS software (SAS Institute Inc., Cary, North Carolina) running on a Sun Sparc workstation (Sun Microsystems, Inc., Palo Alto, California). A post hoc power analysis was performed on laxity measurements obtained at 90° of knee flexion. A Student's paired *t*-test was used to determine statistical significance in relative optical density measurements between the tunnel and the inlay grafts.

Results

Neutral Tibial Rotation

With the tibia in neutral rotation, the mean AP laxity in the inlay-reconstructed knees was significantly less than in the PCL-intact state at all knee flexion angles except 0° (Fig. 4A). In the tunnel group, the mean laxity after reconstruction was greater than in the intact knee at all flexion angles, but the differences were not significant (Fig. 4B). Mean laxity measurements after reconstruction in the inlay group were significantly less than those in the tunnel group at 30°, 60°, and 90° of knee flexion (Fig. 5).

Internal Tibial Rotation

With the tibia in internal rotation, AP laxity in the inlay-reconstructed knees was not significantly different from that in the intact state at any flexion angle tested (Fig. 6A). In the tunnel group, the mean laxity in the reconstructed state was greater than that in the intact state at all flexion angles, but this increase was significant only at 90° (Fig. 6B).

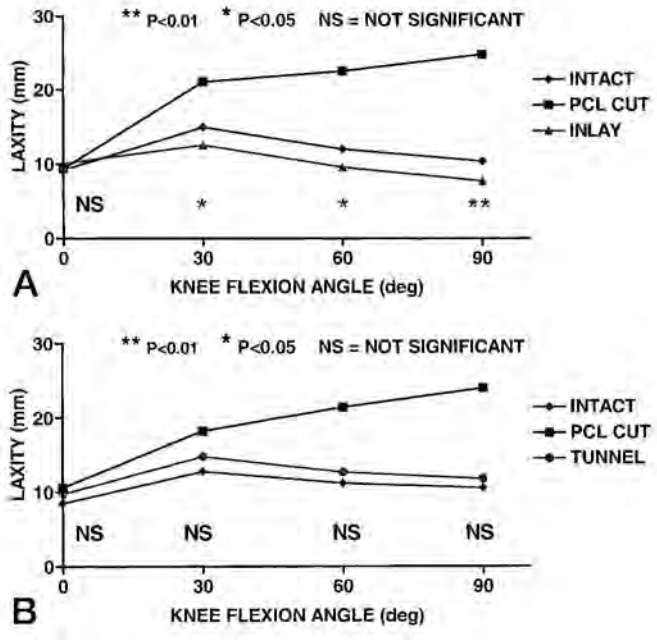

Figure 4. (A) Knee laxity measurements at four flexion angles with the tibia in neutral rotation in the group with the inlay PCL reconstruction. The reconstruction measurements were compared with those in the same knees with the PCL intact and cut. **(B)** Knee laxity measurements in the tunnel-reconstruction group compared with the PCL intact and cut states for the same knees.

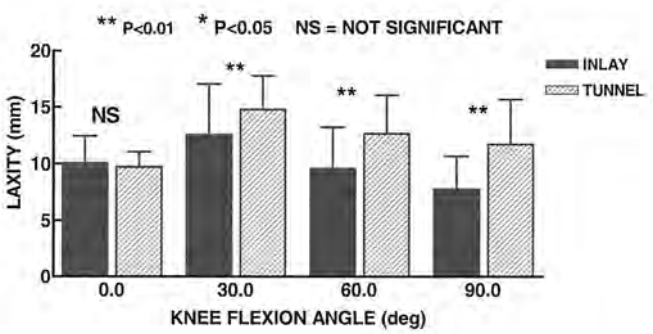

Figure 5. Knee laxity at 90° of knee flexion with neutral tibial rotation: a comparison of inlay and tunnel PCL reconstruction techniques.

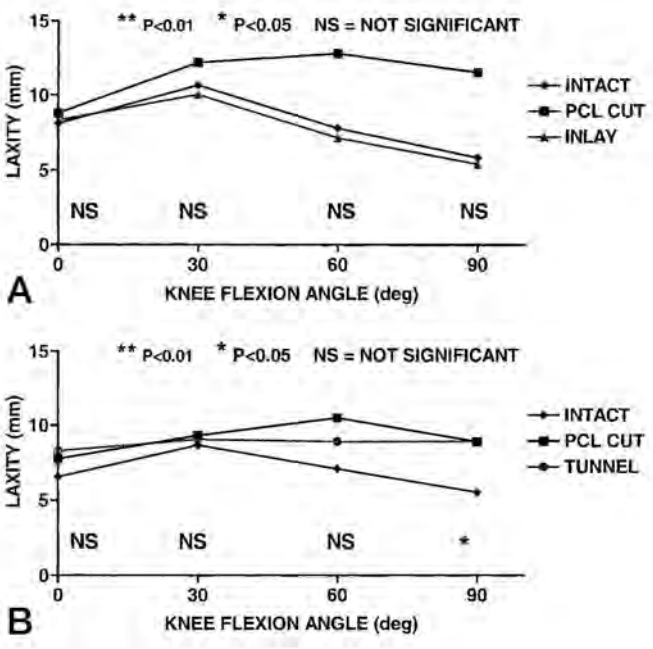

Figure 6. (A) Knee laxity measurements at four flexion angles with internal tibial rotation in the inlay PCL reconstruction group. The reconstruction measurements were compared with those in the same knees with the PCL intact and cut. **(B)** Knee laxity measurements in the tunnel-reconstruction group compared with the PCL intact and cut states for the same knees.

External Tibial Rotation

With the tibia in external rotation, mean laxity after inlay PCL reconstruction was less than that in the intact state at all flexion angles tested, but the differences were not significant (Fig. 7A). Mean laxity after tunnel PCL reconstruction was greater than in the intact state, but, again, the differences were not statistically significant at any flexion angle tested (Fig. 7B).

Statistical Analysis

A pair-wise comparison of laxity measurements at 90° of knee flexion is shown in Table 1. A post hoc power analysis at the 5% significance level revealed that a 2-mm difference in laxity could be detected at 68% power and that a 3-mm difference in laxity could be detected at 83% power.

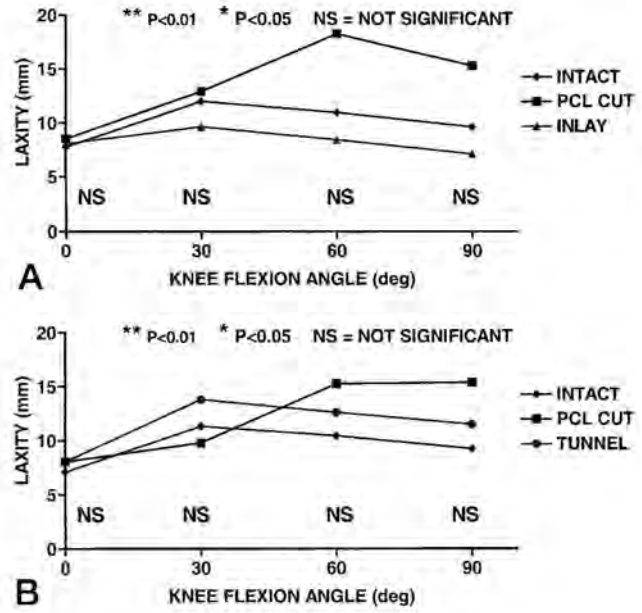

Figure 7. (A) Knee laxity measurements at four flexion angles with external tibial rotation in the inlay PCL reconstruction group. The reconstruction measurements were compared with those in the same knees with the PCL intact and cut. **(B)** Knee laxity measurements in the tunnel-reconstruction group compared with the PCL intact and cut states for the same knees.

Table 1. Pair-wise Comparison of Laxity Measurements at 90° of Knee Flexion for Inlay Versus Tunnel PCL Reconstruction

Condition	Difference (mm)	Standard Error	*P* Value
Neutral rotation	3.8	0.79	<0.001
Internal rotation	3.4	1.34	0.06
External rotation	4.8	1.76	0.04

Effects of Repetitive Loading

After 72 loading cycles, both groups had increased laxity compared with the amount of laxity after initial fixation. However, the final laxity measurements in the inlay group were not significantly different from those in the intact state, while those in the tunnel group were significantly more than in the intact state (Fig. 8).

Graft Evaluation

The grafts from the tunnel group had appreciable thinning and fraying at the site of maximal graft curvature around the proximal posterior tibia, whereas the grafts from the inlay group had no appreciable defects (Fig. 9). The thinning of all grafts was quantified by relative optical density measurements, and a significant difference ($P < 0.05$) was found between the groups (inlay group, 49 relative units; tunnel group, 117 relative units).

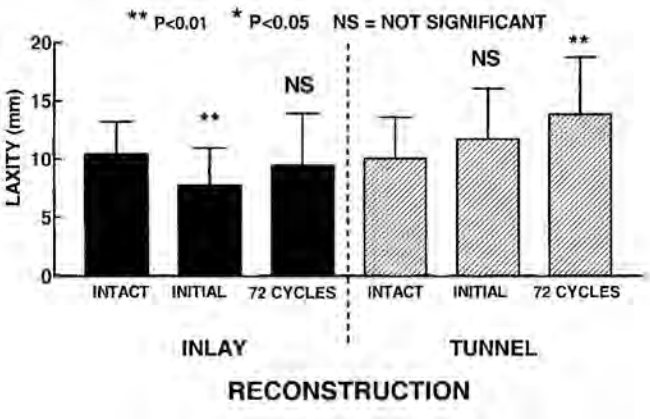

Figure 8. The effects of repetitive loading (72 cycles) at 90° of knee flexion with neutral tibial rotation. Laxity in the intact knee is compared with laxity immediately after reconstruction (initial) and after 72 loading cycles.

Figure 9. Transilluminated grafts from a matched pair of knees after the completion of mechanical testing. The graft on the left is from a knee that underwent the inlay reconstruction while the graft on the right is from the contralateral knee that underwent tunnel reconstruction.

Discussion

The PCL has been the subject of numerous recent investigations. Many basic science studies have advanced our understanding of the kinematics of the PCL-deficient knee and the PCL-reconstructed knee. Previous studies have documented the importance of femoral tunnel placement and

have shown that nonisometric positioning of the graft best corrects abnormal posterior laxity.[2, 5, 10, 17] We agree with the findings of Bomberg et al.,[5] Burns et al.,[7] and Galloway et al.[17] that support making the femoral tunnel in the distal footprint of the native PCL. These studies have contributed to a growing knowledge base and have aided surgeons in better understanding the PCL. However, all of the biomechanical studies to date have used a cadaver knee model with a tibial tunnel technique of PCL reconstruction. A normal laxity pattern has been achieved after a tunnel technique of PCL reconstruction in cadaver knees with isolated PCL deficiency.[24, 25] However, clinical results after this type of reconstruction have been less conclusive.[22] More recent biomechanical studies have investigated the results of double-bundle PCL reconstruction. Race and Amis[28] reported that only a doublebundle reconstruction restored normal knee laxity across the full range of knee motion. Harner et al.[18] found that a double-bundle reconstruction better approximated both normal knee laxity and normal PCL force.

In our study, the grafts were tensioned on the femoral side after securing the tibial side of the graft to the proximal tibia. This convention was chosen so that the grafts in both groups could be tensioned similarly. In addition, this technique has been shown to result in higher tension on the intraarticular portion of the graft when compared with tensioning on the tibial side of the graft.[24] Both reconstruction techniques restored function of the anterolateral bundle of the PCL and significantly reduced abnormal posterior laxity in the PCL-deficient knee. With neutral tibial rotation, the inlay technique of PCL reconstruction resulted in less posterior laxity than the tunnel reconstruction after initial fixation. With internal tibial rotation, the laxity pattern of the inlay group better approximated the intact knee laxity than did the tunnel group. With external tibial rotation, both reconstruction techniques replicated the intact knee laxity. After 72 loading cycles, the laxity of both reconstructions increased, suggesting that both the inlay and tunnel grafts stretched. However, after repetitive loading the inlay reconstruction still approximated intact knee laxity, while in the tunnel group laxity was significantly increased. Although grafts applied with both reconstructive techniques appear to stretch out with repetitive loading, the inlay reconstruction appears to result in less laxity for a given graft tension. This suggests that a lower graft force is required in the inlay group to restore a given laxity.

There was visible evidence of mechanical degradation of the grafts in the tunnel group and no evidence of mechanical degradation in the inlay group. This degradation may be one of the causes of clinical failure after tunnel reconstruction. Another possible cause of tunnel technique failure could be inconsistency in the placement of the tunnel outlet in the posterior tibia. Accurate tunnel placement may be difficult, especially when done arthroscopically. Our study represents a "best-case scenario" for placement of the tibial tunnel, with the drill guide placed in the center of the PCL insertion through an open incision. Consistent placement of the drill guide with arthroscopic techniques may not be so accurate.

Although we have demonstrated that the inlay reconstruction better restrains posterior translation in the PCL-deficient knee, there are some limitations to our study. One weakness of this study was that the same graft pre-tension load was used for all specimens. The selected load of 89 N was adequate to restore normal AP laxity in the tunnel group after initial fixation. However, this same load appears to have overconstrained the knees in the inlay reconstruction group after initial fixation. The 156-N anterior drawer load applied during tensioning may be higher than what could be consistently obtained manually in the operating room. Perhaps less graft tension or less anterior drawer force should be used to correct laxity to normal at the time of initial fixation when performing the inlay PCL reconstruction. Maybe overconstraint should be the goal at the time of surgery to account for stretch of the graft that could occur with repetitive loading. These issues are currently unresolved.

This study used paired cadaver knees with one knee undergoing one type of reconstruction and the other knee undergoing the other type of reconstruction. Although the differences in laxity in the intact and PCL-deficient states were not significant between sides, the comparisons are less statistically powerful because the same knee did not undergo both PCL reconstruction techniques. In addition, static shear loads were applied to the knee during testing. Although this type of loading has been traditionally used for knee laxity testing, this may not represent physiologic loading conditions.

Thinning and fraying of the grafts were identified in the tunnel reconstruction group but not in the inlay group. Although we find it interesting that grafts from the inlay group did not appear to be thinned, the mean age of the specimens was 68 years of age. Perhaps grafts from a younger cadaveric population would demonstrate less mechanical degradation.

Another problem in this study was that we did not observe a statistically significant increase in laxity at 0° of knee flexion in either group after transection of the PCL. This is contrary to what has been reported in another study[24] and suggests a methodological error. The testing jig that we constructed aligned the knee according to the angle made by the tibia and femur and did not take into account individual variability among the test specimens. Many cadaver knees from older donors have slight flexion contractures. Forcibly extending the knee to 0° probably was beyond the normal physiologic limit in some specimens. This leads to tightening of the capsule and other secondary restraints, resulting in less laxity.

Further studies are needed to clarify the natural history of the PCL-deficient knee and to determine whether this natural history can be altered with PCL reconstruction. If abnormal laxity cannot be adequately corrected and maintained, PCL reconstruction will probably not alter the natural history of

the PCL-deficient knee. Current techniques of PCL reconstruction appear to be inadequate.[22] The anatomic tibial inlay technique of PCL reconstruction may better correct abnormal posterior laxity and could potentially result in lower graft forces. Further study is needed to clarify these issues.

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Temple Pearls

Use of the Articulating Tensioning Device ("Push Pull Device")

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So when do you use that thing you learned about in the AO course? It's that little device in your Synthes basic instrument set that provisionally goes on the end of the plate and is used to either compress or distract your fracture. Sounds good, but the problem is that it requires you to extend your incision quite a bit longer than planned. And with dynamic compression plates, when do you really need it? Here are the two uses and some situations where you might want to pull that "ATD" out.

Compression

Keep in mind that with the advent of locking plates, dynamic compression with oval holes has been somewhat compromised with those combi-hole plates, and left out altogether with many other plate designs. Certain fracture patterns like subtrochanteric femur fractures, and to a lesser degree, humeral and tibial shaft fractures, really require good compression, if you are plating them, to heal properly and avoid nonunion. And you often can't get enough compression with eccentric drilling and dynamic compression built into the plate design. So don't be afraid to secure your plate on the neutral side, apply that ATD to the end of the plate, hold your fracture in place with a clamp, and compress the fracture with that ATD!

Distraction

We've all been in a fracture case when you just can't get the fracture distracted enough manually to get it reduced, and it's not really amenable to using the large femoral distractor. Proximal posteromedial tibia plateau fractures are a good example. You think you are going to get it reduced, it needs a plate, but you just can't get it to go those last few millimeters or so. Just put either the plate you plan to use for definitive fixation, or maybe a ⅓ tubular plate, put some screws in on one side of the fracture, then put the ATD on the other end of the plate and distract it until it reduces.

Final Tips for Using the ATD

1. Make sure that your ATD works with the screws (3.5 and/or 4.5) that you are using.
2. Be careful with screw placement in the ATD itself as errant technique here can leave a stress riser just beyond the end of the plate.
3. Make sure your fracture is well aligned, and held in place with a clamp before compressing.
4. Make sure that the plate is lightly clamped to the bone on the ATD side before compressing or distracting (you only have one point of fixation on that side, and the fracture can sometimes angulate or plate can go off bone).

Temple Pearls

How I Apply a Cast — Tips and Techniques

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For any limb, you need to have a good assistant to hold the limb. Even for a cast on a limb that's not grossly unstable, the part to be casted still needs to be stabilized in some way in order to not be working on "a moving target." Ask an assistant to steady and maintain "functional position." In the case of a short leg cast, a metal stand to support the ankle will do. In the example of a short cast, the ankle should be held that 90° or as close as possible.

I almost always use a stockinette to protect the patient from the cast rubbing at its edges. I cut a small section out on the anterior aspect of the ankle to prevent irritating wrinkles from forming here. For a short arm cast, cut a thumb hole with a small tab of stockinette based distally to be able to fold back over the loop of cast through the thenar web space to protect the thumb from rubbing on the cast (especially with fiberglass).

I use standard cotton Webril. The rule of thumb here is "little Webril, big plaster" in choosing the size to apply. For a short leg cast, use either or four inch and three inch. I usually roll the Webril on from top to bottom trying to place a uniform layer from top to bottom overlapping by about 50%. Usually, two or three thicknesses is enough. Be careful not to apply too much padding as this is unnecessary and results in a cast NOT securing the limb. I will usually apply an extra double thickness of Webril at the prominences such as the malleoli, the achilles, and the heel. Also, an extra thickness or two provides a nice cushion at the ends and edges of the cast. Roll the Webril on without wrinkles accomplishing this either by stretching the Webril to prevent wrinkles or by tearing it and apply it at a different angle at the next lower level. I try not to lift the Webril off the patient as lifting the padding tends to allow you to pull the Webril too tight at one edge, thereby creating bands that can be constricting.

Once the Webril is in place, I usually use fiberglass casting tape. Despite the fact that it is more expensive for each roll, fiberglass is certainly lighter and stronger. This allows you to use fewer rolls at first and you will find you encounter fewer times that the cast will fail needing a new application. Fiberglass is also less messy. (You DO have to be careful you don't get it on you hands, clothing, or office equipment and furniture, however. The only way to remove it is by an acetone scrub.) For a short leg cast in an average-sized patient, you usually need to use two four-inch rolls of fiberglass and one three-inch roll. I still use fairly cold water as this allows me to work a little more slowly and carefully. This is a big advantage because fiberglass certainly dries fast enough that you do not have to wait long for it to set. Also, using cold water, you can apply the cast at a much more uniform and smooth thickness. In essence, it gives you more control when you use cold water.

Again, I start at the top to let gravity help me — overlapping the fiberglass material by 50% as I come down and I try to put a uniform thickness throughout the cast. Do not lift the fiberglass roll off the patient since by lifting the roll off the intended site with the casting material, you can make one edge tight causing little spirals of pressure points. I keep the material against the patient's limb to minimize the chance of causing a pressure point and also by doing so makes for a nice uniform thickness of the cast. I go from top to bottom covering by 50% and, if I reached the bottom, I start back up again. The second roll is started at this "initial finish point" and I continue back up toward the knee. Again, applying the cast in this fashion allows a nice uniform thickness which translates into the sleekest, lightest, but strongest finished product. Now, before I reach the top of the cast as I am rolling, I turn down the stockinette over the previously applied layer and I can cover the stockinette and seal it with the second roll of fiberglass as I coming up. At this point, I rub the fiberglass to smooth and integrate the layers and, in doing this, I also create a mold as needed for holding the fracture in position.

Before the fiberglass sets (hardens), I use a pair of dedicated fiberglass cutting scissors and I cut out a portion over the dorsum of the toes to allow the toes to move freely (if the fracture allows). This creates a little plantar plate distally; I then fold the stockinette back over the fiberglass. I then add a three-inch roll of fiberglass starting distally and working up the foot and ankle. At this point, I also will "Cadillac" the cast with a roll of color if the patient requests this. I have tried to stock a variety of two-inch colors and use one (or occasionally two) to finish off the cast. Patients often feel a little better about being in a cast if they can smile at a color. Truth be told, I may add a little thickness to the heel and around the ankle as I finish by a rolling the three-inch up the leg if necessary. If it's a nonweightbearing cast, I don't think any further additional material is necessary. If it is a weightbearing cast, sometimes there is still a little equinus deformity despite trying to reduce it to 90 degrees; in this instance, usually you can use a two-inch or three-inch roll of the casting material to bunch up and add some height to the heel attaching, of course, with the last few feet of that roll. This balances the ankle for ambulation and avoids pushing the

patient into "back knee" as they walk. Their knee should always slightly flex as they progress forward with weight-bearing.

I always warn the patient about the signs of a tight cast: excessive, unremitting PAIN and the feeling of a VERY tight cast. Also, inform the patient — and relatives — about what to do if there is any rubbing of the cast causing sores, ulcers, blisters, or any significant irritation. You must tell them of the consequences of a cast problem and that if any of these occur — especially a tight cast — that they are emergencies that need to be treated as such by immediate, right now, attention — either loosening or removing the cast.

To loosen a cast, you need to cut ALL layers. Whether you bivalve or univalve the cast, the Webril also needs to be split so that any circular dressing is released completely (even sticky, blood soaked gauze can form constrictions). Bivalving a cast and removing the anterior portion of the cast, cutting all the Webril so it is not circular and holding the resultant splint in place with loosely wrapped Ace wraps is the safest method when possible. I never wrap Kling or Kerlex gauze under a cast.

If you still want to use plaster (which is clearly less expensive but messy, weaker, and heavier), place a towel or cloth on the floor. For a short leg cast, I use three six-inch rolls (or two six-inch rolls and a four-inch roll depending on the patient's size). Again, use cold water as it gives you better control especially when you are first beginning to apply casts. It will take longer to dry. Rubbing "like you love it" will result in a much better, stronger cast as well as will look better and have less chance of causing any irritation from irregularities in the cast (see above discussion re: Webril and fiberglass application).

Apply the Webril as directed above. Then to apply the cast, set the plaster in the water (cold or cool water) and wait for the bubbles to stop forming; squeezing the two ends of the rolls to prevent a "banana." Now apply the plaster directly on the patient (don't lift roll) and cross 50% of the layer above. Do not tuck; just roll it and the plaster would gently fold into itself and, again, create a much smoother, more uniformly layered cast. Making tucks takes time and also slows your application time. So you can put on as good a cast as possible, give yourself as much time by using cold water and don't waste time on tucks. Apply enough rolls of the plaster to secure the limb. Don't worry about rubbing the cast until all the rolls are applied; this will save time and assure that the plaster will be molded into one single (stronger) layer. Then begin rubbing to compress the layers together and smooth the plaster. Always rub it in the opposite direction you placed the plaster on so that the edges won't wrinkle (if the last roll is from top/down, rub up; if distal to proximal, rub downward). As the plaster begins to set, the plaster gets to the "cardboard stage." Now the plaster has become clearly a little more tacky and less slippery. It is at this point, if needed, you can apply your molds using the thenar eminences to apply the mold. Don't keep your hands in one spot; gently move them a little to prevent an excessive pressure point. After the plaster hardens enough to stop applying a mold, trim the edges of the cast and apply a sealing roll as needed.

Be sure to again inform the patient and relatives about cast problems (one of the true orthopedic emergencies) and give them some tips on cast care. It will take 24 to 48 hours for the plaster to dry (and reach maximum hardness) so keep the patient from walking on the cast for two days.

To remove a cast, remember that the cast saw vibrates so the soft Webril padding — and your skill — all serve to keep you from cutting the patient. Wet Webril or blood-soaked Webril makes it easier to cut or burn the patient. Also listen to the patient — if he or she protests too much, reevaluate whether you may be actually injuring them. Usually you are not, but be sure. Hold the saw with one hand and use the thumb as a prop against the cast to steady and guide your cutting. Remember, the saw blade is circular so you can use this to your advantage. After the initial gentle, careful "plunge," move the saw steadily distally; not plunging multiple times instead of moving distally makes it less likely you will come against the patient's skin. This technique allows the incline of the saw blade to always serve as a "plunge guide." Make your cuts away from bony prominences — and away from neurovascular structures — whenever possible. Pop the cast open with the spreaders and then cut the stockinette and Webril. Remove the cast. Patients should wash, not scrub skin at first and use cream or lotion on skin for a while. Splint the limb if/as necessary for awhile after removal of the cast.

Original Research

Connective Tissue Growth Factor (CTGF) Regulates BMP Signaling During Osteoblast Differentiation

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Abstract

Connective tissue growth factor (CTGF) and bone morphogenetic protein (BMP)-2 are both produced and secreted by osteoblasts. Both proteins have been shown to have independent effects in regulating osteoblast proliferation, maturation and mineralization. However, how these two proteins interact during osteoblast differentiation remains unknown. In this study, we compared the differentiation of KO and WT osteoblasts to investigate the effects of CTGF and BMP-2 on osteoblast development and function *in vitro.* In cultures not stimulated with BMP-2, the absence of endogenous CTGF did not affect osteoblast maturation and mineralization. There was no significant difference in alkaline phosphatase (ALP) activity and staining, alizarin red staining or mRNA expression of runt related transcription factor 2 *(Runx2)* and osteocalcin *(Oc).* Interestingly, in WT and KO osteoblast cultures stimulated with BMP-2, KO osteoblasts exhibited enhanced osteoblast differentiation. There was a significant increase in the number and size of mineralized nodules, as well as *Runx2* and *Oc* mRNA expression in KO osteoblast cultures. The increase in osteoblast differentiation was accompanied by increased protein levels of phosphorylated Smad 1/5/8 and mRNA expression levels of bone morphogenetic protein receptor Ib. The present findings demonstrate, through functional studies, a novel function of endogenous CTGF in regulating osteoblast development and function by inhibiting BMP signaling.

Introduction

Osteoblast differentiation is a complex process, which involves the commitment of mesenchymal cells to osteoblasts followed by synthesis and deposition of bone matrix proteins. The development of pre-osteoblasts to matrix-secreting osteoblasts encompasses three phases: 1) proliferation, 2) maturation and 3) mineralization.[1] These phases require the appropriate expression of several osteoblast markers, including runt-related transcription factor 2 *(Runx2),* osterix, alkaline phosphatase *(Alp),* and osteocalcin *(Oc)* to ensure proper osteoblast development and function. This process is highly regulated by local factors such as bone morphogenetic proteins (BMPs). BMP-2 has been widely studied and is known to induce bone formation *in vivo* and promote osteoblast differentiation *in vitro.*[2–4] The mechanism by which BMP-2 promotes osteogenesis is well understood. BMP-2 exerts its effects through the canonical Smad pathway involving Smads 1, 5, and 8, which are phosphorylated by serine/threonine kinase receptors composed of type I and II components. Once activated, Smads 1, 5, and 8 form a complex with Smad 4, which translocates to the nucleus and induces the transcription of target genes, such as *Runx2.*[5, 6]

In addition to BMP-2, connective tissue growth factor (CTGF) has been shown to regulate osteogenesis. CTGF knockout (KO) mice exhibit skeletal dysmorphisms due to defects in chondrogenesis and extracellular matrix production.[7] Recently, our lab did an in-depth characterization of CTGF KO mice and found these mice to exhibit numerous site-specific defects in the axial, appendicular, and craniofacial skeleton.[8] CTGF is expressed and secreted by osteoblasts during bone formation and fracture healing. *In vitro* studies have shown that CTGF promotes the proliferation and differentiation of osteoblasts.[9, 10] These studies support a role for CTGF as a regulator in osteoblast development and function, but how CTGF regulates such processes remain unknown.

CTGF is a matricellular protein that interacts with several growth factors, including BMPs. Studies revealed that CTGF interacts with BMP-2 and BMP-4 through its von-Willebrand type C and C-terminal domains.[11, 12] More specifically, when CTGF interacts with BMP-4, CTGF sequesters BMP-4 thereby preventing the ligand from binding to its cognate receptor, BMPR-1a, thus inhibiting BMP signaling.[11] Despite these findings, it remains unclear how CTGF and BMP-2 interact to regulate osteoblast differentiation. In this study, we utilized primary osteoblasts derived from calvaria of E18.5 CTGF wild-type (WT) and KO embryos to investigate the interaction of CTGF and BMP-2 during osteoblast differentiation.

Material and Methods

Source of Animals

CTGF heterozygous mice ($CTGF^{+/LacZ}$) were used as breeders to obtain CTGF KO ($CTGF^{LacZ/LacZ}$) mice as previously described.[13] Genotype was determined as previously described.[13] All animals were maintained and used according to the principles in the NIH Guide for the Care and Use of Laboratory Animals (U.S. Department of Health and Human Services, Publ. No. 86-23, 1985) and guidelines established by the IACUC of Temple University.

Primary Osteoblast Cell Culture

Primary cells were isolated from parietal calvaria of embryonic day 18.5 CTGF wild-type (WT) and knockout (KO) embryos from which the cranial sutures were removed to reduce non-osteoblast cell contamination. Calvaria pieces were placed in digestion media consisting of 0.1% Collagenase (Sigma)/2.5% trypsin and subjected to a series of digestions of 5, 15, 30, 20, 15, and 15 minutes at 37°C. The purpose of the first digestion is to remove non-osteoblastic cells and this digestion was discarded. The osteoblast cell population was obtained from the remaining digestions. Cells were plated in 100 mm dishes (Corning) at 5 x 10^5 cells/plate in Alpha Minimal Essential Medium (α-MEM; Mediatech) supplemented with 10% fetal bovine serum (FBS; HyClone). The cells were incubated at 37°C with 5% CO_2 with a change of media every three days until they reached 80% confluence. For experiments, cells were cultured in osteogenic media containing 50 μg/ml ascorbic acid (Sigma) and 10 mM β-glycerophosphate (Sigma) in addition to α-MEM/10% FBS to stimulate osteoblast differentiation.

BMP-2 Stimulation

Recombinant BMP-2 (R&D Systems) was reconstituted to a concentration of 10 μg/ml in sterile 4 mM HCl containing 0.1% BSA and stored at –20°C. Cells were treated with a standard concentration of 100 ng/ml of BMP-2 every three days during osteoblast differentiation. For evaluating levels of phosphorylated Smad1/5/8, WT and KO cells were serum starved for 24 hours then stimulated with BMP-2 for 5–60 minutes. For evaluating BMP receptor levels, WT and KO cells were stimulated with BMP-2 for zero and eight hours.

Cell Proliferation and Spreading

Cell number was determined using the CyQUANT® NF Cell Proliferation Assay Kit (Molecular Probes) according to the manufacturer's protocol. Briefly, CTGF WT and KO osteoblasts were plated at 4 x 10^3 cells/well in a 96 well plate (Falcon) in α-MEM/10% FBS. On Days 1, 3, and 7, media was aspirated and replaced with DNA binding dye solution. Cells were incubated at 37°C for one hour and samples were measured using a Wallac 1420 fluorometer. Cell number was calculated based on a standard curve generated for primary osteoblasts. Phase contrast images of cells were taken on Days 1, 3, and 7 with the Nikon Eclipse TE300 inverted microscope. For cell spreading experiments, eight-chamber glass slides (Lab-Tek II) were coated with 1% Bovine Serum Albumin (BSA) (Sigma) or 2 μg/ml of Fibronectin (FN) and incubated at room temperature under sterile conditions overnight. WT and KO osteoblasts were plated at 2 x 10^3 cells/chamber and incubated for eight hours at 37°C. Cells were fixed and stained for actin using the Actin Cytoskeleton and Focal Adhesion Staining Kit (Millipore). Cells were imaged on a Nikon Eclipse E1000 fluorescence microscope, and images were digitized for cell area measurements using Image J software.

Alkaline Phosphatase Staining and Activity

CTGF WT and KO cells were plated in a 48-well plate (Corning) at 1.1 x 10^4 cells/well in osteogenic media, which was changed every three days. The osteoblast cultures were stopped at Day 14 to evaluate the production of alkaline phosphatase. Alkaline phosphatase staining was performed using the Leukocyte Alkaline Phosphatase Kit (Sigma). Cells were washed with 1X HBSS and fixed in a citrate buffer containing acetone and formaldehyde. Following fixation, cells were washed with ddH_2O and incubated with a staining solution consisting of equal parts of sodium nitrite solution, FRV-alkaline solution and napthol AS-BI alkaline solution for 25 minutes at room temperature. Cells were washed with ddH_2O and allowed to air dry. Wells were observed and images taken using the Nikon Eclipse TE300 inverted microscope. Quantification of alkaline phosphatase production was determined using Quantichrom™ Alkaline Phosphatase Assay Kit (BioAssays Systems). Cells were lysed in ddH_2O water containing 0.2% Triton X-100 for 20 minutes at room temperature. Cell lysates were collected and the assay was carried out according to the manufacturer's protocol. Alkaline phosphatase activity was normalized to total protein content. Total protein content was determined using BCA protein assay kit (Pierce) on identical cultures.

Alizarin Red Staining

CTGF WT and KO osteoblast cultures were stopped at Day 21 to evaluate mineralization. For alizarin red staining, cells were washed in 1X HBSS and fixed in 10% paraformaldehyde for 15 minutes at room temperature. Cells were washed with 1X PBS and stained with 40 mM Alizarin Red S (Sigma) for 15 minutes at room temperature. Cells were washed with ddH_2O and allowed to air dry. Wells were observed and mages taken using the Nikon Eclipse TE300 inverted microscope.

RNA Isolation and Quantitative PCR

Total RNA was isolated from CTGF WT and KO osteoblasts at Days 7, 14, and 21. To evaluate changes in BMP receptor levels, CTGF WT and KO osteoblasts were serum starved for 24 hours, stimulated with BMP-2 (100 ng/ml),

and RNA was collected at zero and eight hours. RNA was isolated from cell cultures using Trizol reagent (Invitrogen) according to the manufacturer's directions. RNA was purified using the RNeasy Mini Kit (Qiagen) and treated with DNase using the RNase-Free DNase Kit (Qiagen). RNA quality and quantity was determined using spectrophotometry and the integrity of all samples was confirmed using 1% formaldehyde-agarose gel. For cDNA synthesis, 1 μg of cDNA was transcribed from total RNA using SuperScript® First-Strand Synthesis System (Invitrogen). Gene expression for runt-related transcription factor 2 *(Runx2),* osteocalcin *(Oc),* bone morphogenetic protein receptor Ia *(BMPR-Ia),* and bone morphogenetic protein receptor Ib *(BMPR-Ib)* was determined by qPCR, as described previously.[8]

Protein Isolation and Western Blotting

WT and KO cell monolayers were washed in cold 1X PBS and lysed in 1X RIPA (Millipore) for one hour at 4°C. Cell lysates were centrifuged and the resultant supernatants were used for determination of protein concentration using BCA Protein Assay Kit (Pierce). Western Blot was performed as previously described.[9] The membrane was blocked with 5% BSA/1X PBS-Tween20 for one hour and incubated with the following primary antibodies: anti-rabbit phospho-Smad (p-Smad) 1/5/8 (1:1000; Cell Signaling) and anti-rabbit actin (1:1000; Sigma) overnight at 4°C. The membrane was washed with 1X PBS-Tween20 and incubated with horseradish-peroxidase conjugated donkey anti-rabbit (1:10,000; Jackson Immunoresearch) for one hour at room temperature. The membrane was washed again, incubated with SuperSignal West Pico Chemiluminescent Substrate (Thermo Scientific), and exposed to film. Quantification of the bands was done using Image J software.

Statistical Analysis

Unpaired Student's t test was used to determine whether the absence of CTGF had any effect on quantitative parameters related to osteoblast proliferation, maturation, and mineralization compared to WT. Data are expressed as ± SEM. A P-value <0.05 was considered statistically significant.

Results

CTGF KO Osteoblasts Display Reduced Cell Spreading But Increased Cell Proliferation During Early Differentiation

To determine whether the absence of CTGF affects osteoblast proliferation, we examined the proliferative capacity of WT and KO osteoblasts at Days 1, 3, and 7. At Day 1 after plating, there was no significant difference in cell number between WT and KO osteoblasts (Fig. 1A, B: top panel). However, by Day 3, KO osteoblasts showed increased proliferation compared to WT osteoblasts (Fig. 1A). Interestingly, a representative phase contrast image showed a population of KO osteoblasts, which appeared smaller, suggesting reduced cell spreading, whereas the WT osteoblasts appeared larger, suggesting increased cell spreading (Fig. 1B: middle panel). This change in KO cell behavior was more apparent in Day 7 cultures. By Day 7, the WT osteoblasts reached confluence and therefore stopped proliferating, whereas the KO osteoblasts still continued to proliferate due to their smaller cell shape. The phase contrast image at this time point displayed a clear difference in cell shape between WT and KO osteoblasts (Fig. 1A, B: bottom panel). To confirm that the KO osteoblasts spread less than the WT osteoblasts, we plated the cells on BSA and FN and measured the cell area after immunofluorescence staining for actin. When plated on either substrate, KO osteoblasts exhibited reduced cell spreading compared to WT osteoblasts (Fig. 1C and 1D). Collectively, these data demonstrate that the reduced spreading of KO osteoblasts allows these cells to continue to proliferate for a greater period of time to reach a confluent state.

CTGF KO Osteoblasts Display Normal Maturation and Mineralization **In Vitro**

To investigate the role of CTGF during osteoblast differentiation, we cultured primary WT and KO osteoblasts for a period of 21 days. We evaluated osteoblast maturation at Day 14 by ALP staining and activity, and there was no difference in osteoblast maturation between WT and KO cultures (Fig. 2A: left panel and B). Next, we examined osteoblast mineralization at Day 21 by Alizarin red staining. In both WT and KO cultures, osteoblasts started to aggregate, which is demonstrated by an increase in Alizarin red staining at sites of the cell aggregations (arrows). There was no delay in osteoblast mineralization in the KO cultures, which were similar in appearance to the WT cultures (Fig. 2A: right panel). *Runx2* is an essential transcription factor for osteoblast differentiation and is known to up-regulate other critical genes important for later stages of osteoblast differentiation, such as *Oc*. Therefore, we measured *Runx2* mRNA expression in WT and KO cultures at Day 7. Although the KO cultures showed an increase in *Runx2,* it was not significant (Fig. 2C). In addition, there was no difference in *Oc* mRNA expression between WT and KO cultures (Fig. 2D). These data demonstrate that osteoblasts can differentiate normally in culture in the absence of endogenous CTGF production (KO osteoblasts).

CTGF KO Osteoblasts Exhibit Enhanced Differentiation in Response to BMP-2

BMP-2 is a well-known osteoinductive agent both *in vivo* and *in vitro*.[2–4] CTGF interacts with BMPs,[11, 12] but it is unknown how CTGF and BMP-2 interact during osteoblast differentiation. To examine this interaction, we treated osteoblast cultures with rBMP-2 for 21 days. Interestingly, ALP staining at Day 14 revealed the presence of nodules in the KO cultures, which were not seen in the WT cultures (Fig. 3A: left panel). There was no difference in ALP activity between WT and KO cultures. Alizarin red staining at Day

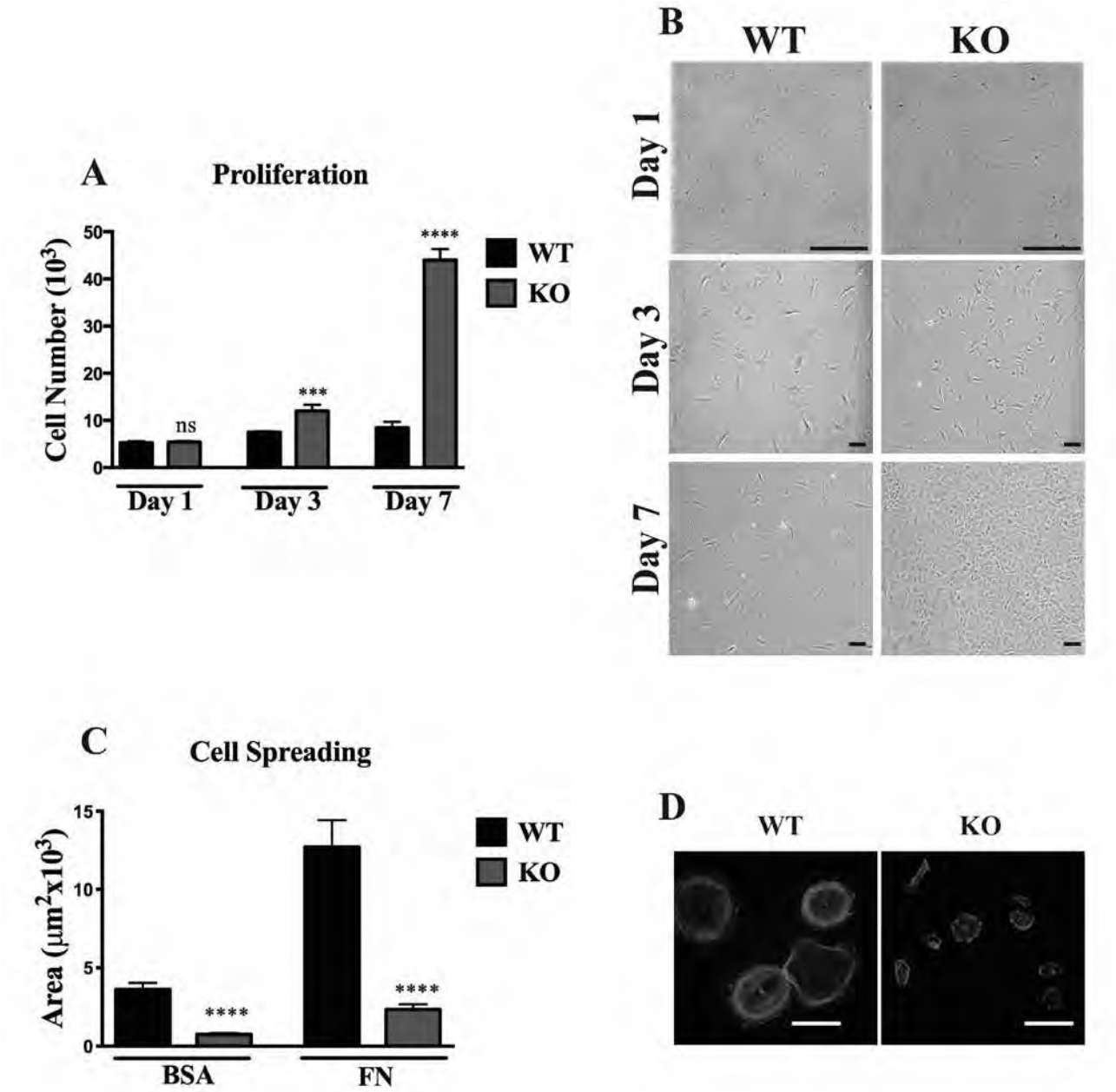

Figure 1. CTGF KO osteoblasts display increased proliferation. (A) The proliferation of CTGF WT and KO osteoblasts was assessed on Days 1, 3 and 7. Cell numbers were based on fluorescence at 520 nm. **(B)** Phase contrast images of cells at Day 1, Day 3, and Day 7. **(C)** WT and KO osteoblasts were plated on BSA and FN and cell area (spreading) was measured eight hours later. **(D)** Immunofluorescence staining for actin in WT and KO cells plated on FN. ***P < 0.001; ****P < 0.0001. Scale bar: 50 μm (Day 1); 10 μm (Days 3 and 7); 100 μm (actin staining). Abbreviations include bovine serum albumin (BSA) and fibronectin (FN).

21 showed the presence of small, mineralized nodules in WT cultures, whereas the KO cultures displayed a greater number of nodules, which fused with one another to form larger mineralized nodules (Fig. 3A: right panel). In addition, *Runx2* and *Oc* mRNA expression levels were significantly up-regulated in CTGF KO cultures (Fig. 3D). These data demonstrate that osteoblast differentiation is accelerated and enhanced in response to exogenous BMP-2 in KO compared to WT cultures.

Increased BMP signaling in CTGF KO osteoblasts

Smads 1, 5, and 8 are important mediators in the BMP signaling pathway.[5] To investigate whether the enhanced osteoblast differentiation seen in the CTGF KO cultures is attributed to an increase in p-Smad 1/5/8 levels, we treated WT and KO osteoblasts with BMP-2 for 5 to 60 minutes. p-Smad 1/5/8 protein levels increased with BMP-2 stimulation in both WT and KO osteoblasts, yet p-SMAD 1/5/8 levels were greater in KO compared to WT osteoblasts at all

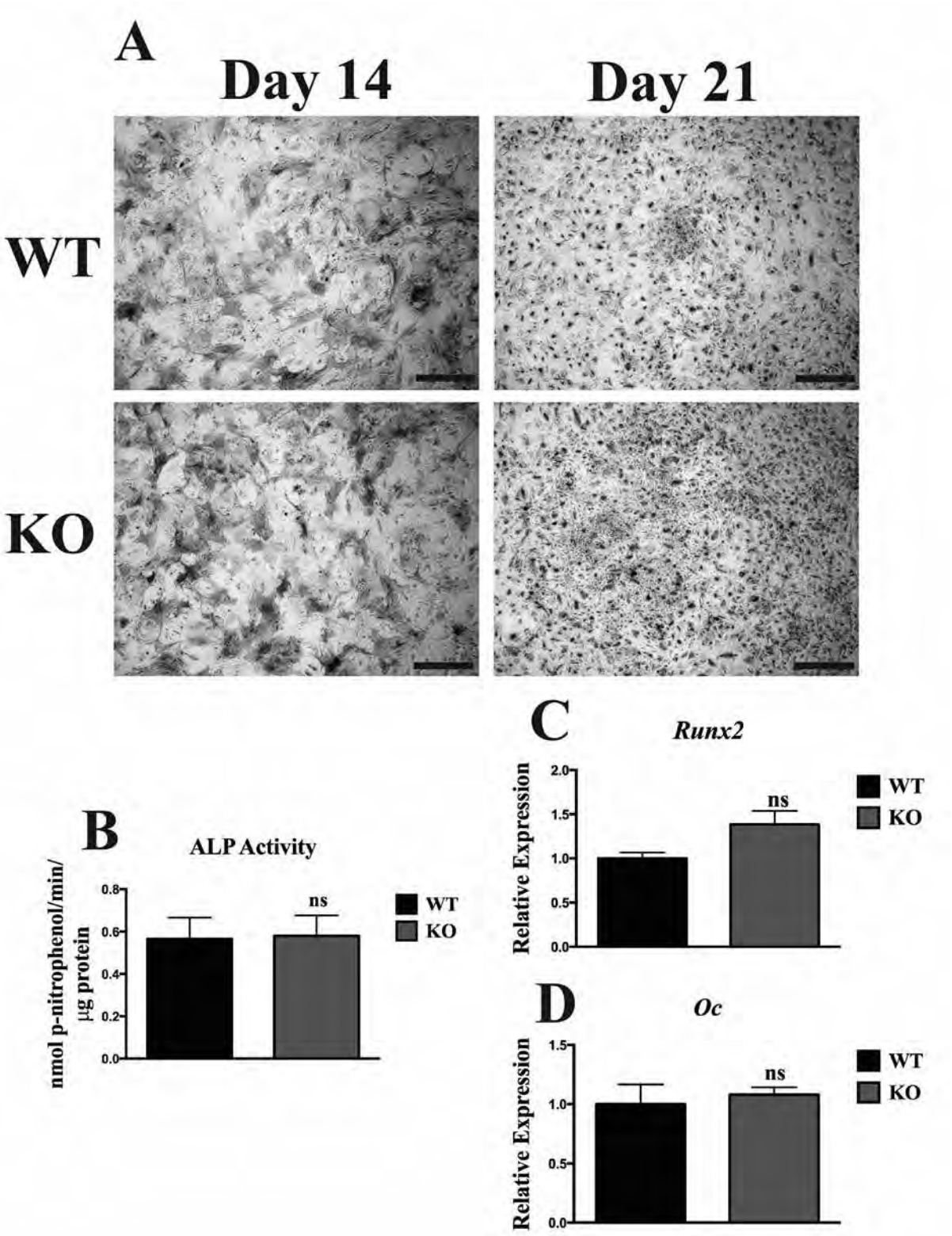

Figure 2. CTGF KO osteoblasts exhibit normal differentiation *in vitro.* (A) ALP staining at Day 14 of osteoblast culture (left panel) and Alizarin Red S staining at Day 21 (right panel). **(B)** ALP activity quantified at Day 14 of culture. **(C** and **D)** mRNA gene expression of *Runx2* at Day 7 of culture **(C)** and *Oc* at Day 21 **(D).** Scale bar: 50 μm. Abbreviations include alkaline phosphatase (ALP), runt-related transcription factor 2 *(Runx2)* and osteocalcin *(Oc).*

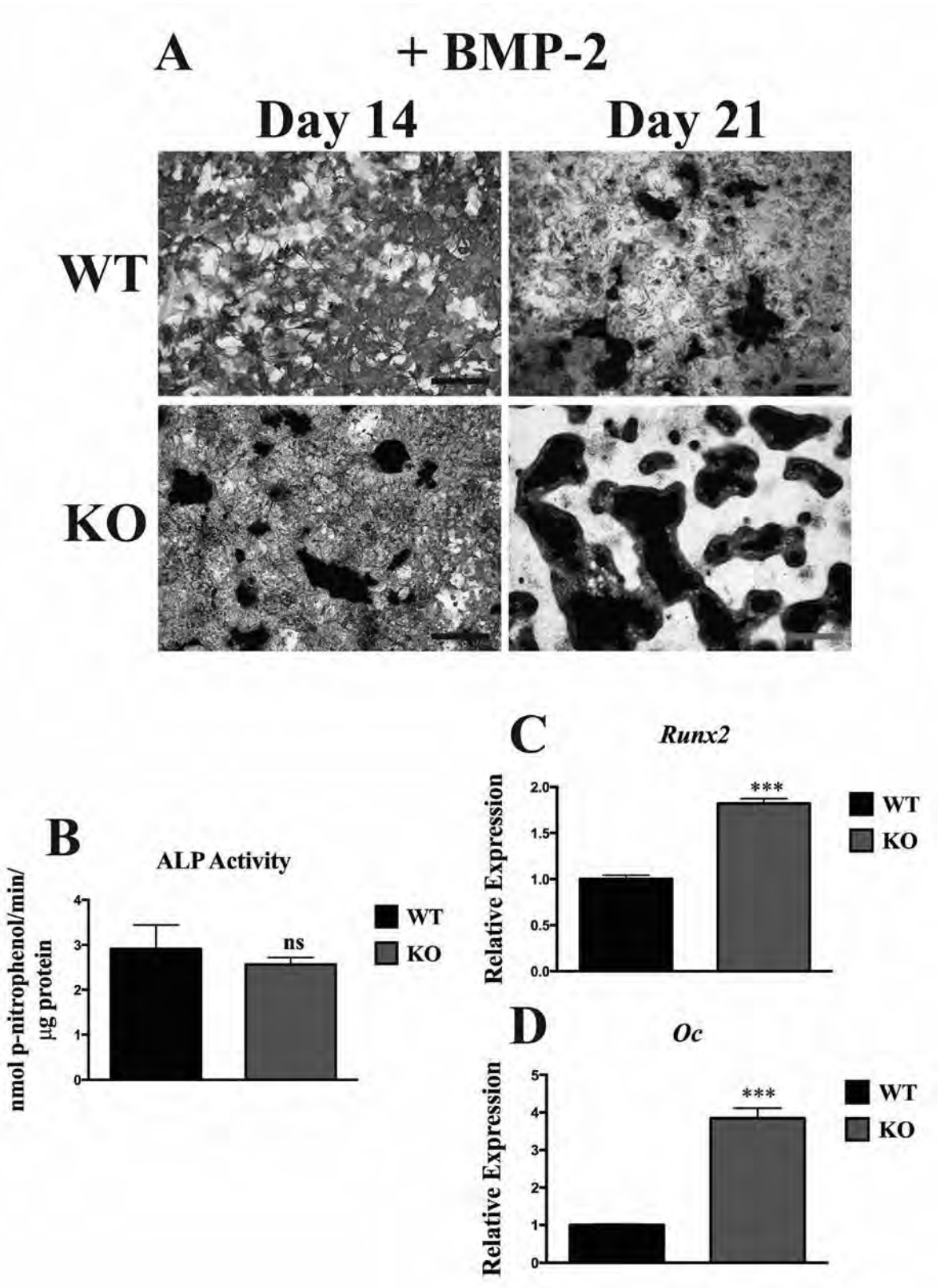

Figure 3. CTGF KO osteoblasts exhibit enhanced maturation and mineralization in the presence of BMP-2. (A) ALP staining at Day 14 of osteoblast culture (left panel) and Alizarin Red S staining at Day 21 (right panel). **(B)** ALP Activity was measured at Day 14 of culture. **(C** and **D)** mRNA gene expression of *Runx2* at Day 7 **(C)** and *Oc* at Day 21 **(D).** ***P < 0.001. Scale bar: 50 μm. Abbreviations include alkaline phosphatase (ALP), runt-related transcription factor 2 *(Runx2),* and osteocalcin *(Oc).*

time points (Fig. 4A). Since the BMP-2 ligand/receptor complex activates Smads 1, 5, and 8, we investigated the expression of signaling receptors, *BMPR-Ia* and *BMPR-Ib.* We stimulated WT and KO cultures with BMP-2 for zero and eight hours, and evaluated changes in mRNA expression of BMP receptors. In un-stimulated conditions there were no significant differences in *BMPR-Ia* and *BMPR-Ib* expression between WT and KO osteoblasts. However, upon stimulation with BMP-2, *BMPR-Ia* levels decreased 0.5- and 0.75-fold in WT and KO osteoblasts, respectively (Fig. 4B). The observation that *BMPR-Ia* was down-regulated upon BMP-2 stimulation suggests that this receptor does not play a critical role in BMP-2-induced osteoblast differentiation. When WT and KO osteoblasts were treated with BMP-2 and assessed for *BMPR-Ib* expression levels, *BMPR-Ib* levels increased by 10- and 35-fold in WT and KO osteoblasts, respectively. Interestingly, at eight hours, KO osteoblasts displayed an increased expression of *BMPR-Ib* compared to WT osteoblasts (Fig. 4C). These data demonstrate that KO osteoblasts exhibit increased signaling in response to BMP-2, and suggest that BMPR-Ib is the prominent receptor required for BMP-induced signaling in osteoblast cultures.

Discussion

CTGF interacts with numerous growth factors and matrix proteins to regulate various cellular processes such as migration, adhesion, proliferation, and differentiation.[14] These processes are important for proper osteoblast development and function. Previous studies have shown that CTGF interacts with BMP-2 and BMP-4. One group demonstrated that CTGF interacts with BMP-4 and prevents BMP-4 from binding to its receptor, BMPR-1a, thereby inhibiting the action of BMP-4 during embryonic patterning.[11] Another group demonstrated that CTGF interacts with BMP-2 and together, these proteins can modulate chondrocyte proliferation and differentiation.[12] Both proteins are produced and secreted by osteoblasts, and have independent effects in regulating osteoblast proliferation, maturation and mineralization. However, the interaction between CTGF and BMP-2 has yet to be explored during osteoblast differentiation. In this study, we compared the differentiation of KO and WT osteoblasts to investigate the effects of CTGF and BMP-2 on osteoblast development and function *in vitro.*

During the first phase of osteoblast differentiation, the cells actively proliferate in culture and express cell cycle and cell growth regulated genes. When cells come into contact with each other, they undergo cell contact inhibition, which initiates arrest of cell growth.[15] Our data shows that WT osteoblasts spread more, indicated by a significantly larger average cell area, than KO osteoblasts (Fig. 1C, D). Therefore, WT osteoblasts reach confluence and cell contact inhibition, earlier than KO osteoblasts. As a result, the KO osteoblasts continue to proliferate for a longer period of time until they achieve a confluent state (Fig. 1A, B). We believe this is one possible explanation for the significantly higher cell numbers in KO compared to WT osteoblast cultures at Days 3 and 7. To fully understand the proliferative behavior of WT and KO osteoblasts, future studies will involve FACS analysis to compare cell cycle markers, such as cyclins and cyclin-dependent kinases.

The second and third phases of osteoblast differentiation are matrix production/maturation and mineralization, respectively. Following cell cycle arrest, the osteoblasts undergo a series of morphological changes and temporally express *Alp, type I collagen,* and *osteocalcin,* all of which are necessary for the synthesis of a mineralized bone matrix.[15] We evaluated these two phases in osteoblast cultures derived from CTGF WT and KO embryos. Under normal osteogenic culture conditions that were not stimulated with exogenous BMP-2, there was no difference in ALP staining and activity, alizarin red staining, or the mRNA expression of osteoblast markers when comparing WT and KO osteoblast cultures (Fig. 2). These results are contradictory to what has been previously published regarding the differentiation of osteoblasts derived from CTGF KO mice. This previous study showed a significant reduction in the differentiation of CTGF KO compared to WT osteoblasts.[10] One possible explanation for the difference in our results from those previously published may be related to cell plating densities used in the two studies. The authors of the previous study used a significantly lower cell plating density that was used in this study. Interestingly, when we plated WT and KO osteoblasts at half the normal cell density which is still higher than that used in the Kawaki et al. study, the KO osteoblasts showed less ALP activity at Day 14 than WT, but by Day 21 the levels of ALP activity in WT and KO cultures were comparable (data not shown). Another possible reason for the discrepancy may be related to the time points that were chosen to evaluate osteoblast differentiation in the previous study. Our results suggest that CTGF is not essential for osteoblast differentiation under unstimulated conditions.

However, when we treated WT and KO osteoblast cultures with BMP-2, the KO osteoblasts exhibited a markedly accelerated differentiation. It is well-documented that *Runx2* is required for osteoblast differentiation.[16, 17] This essential transcription factor is expressed throughout osteoblast differentiation and it up-regulates several downstream genes necessary for later stages of osteoblast differentiation, such as *type I collagen* and *Oc.*[18, 19] Interestingly, studies have shown that BMPs up-regulate *Runx2* mRNA expression and inhibition of BMP signaling disrupts the ability of *Runx2* to stimulate osteoblast differentiation *in vitro.*[6] Our data shows that upon stimulation with BMP-2, *Runx2* mRNA expression levels were significantly up-regulated in KO osteoblast cultures, which resulted in enhanced osteoblast maturation and mineralization compared to WT osteoblast cultures. It is important to note that when we measured ALP activity in these cultures, even though the results were not significant, the KO osteoblasts showed a decrease in activity (Fig. 3B).

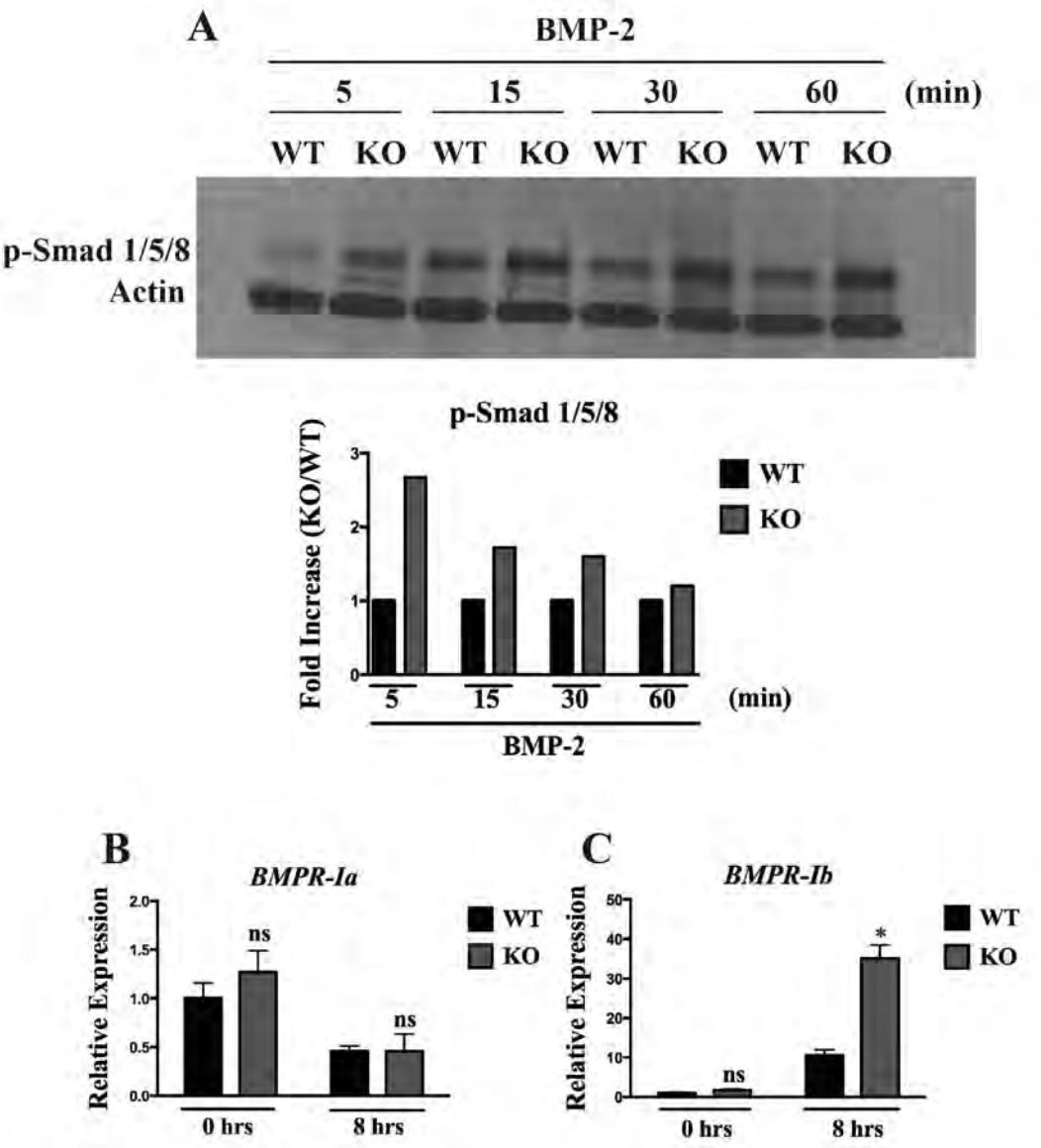

Figure 4. BMP signaling is increased in CTGF KO osteoblasts. Representative Western blot analysis using 15 μg total protein from WT and KO osteoblast cultures treated with BMP-2 for the indicated time points. **(A)** p-Smad 1/5/8 levels and quantification. p-Smad 1/5/8 expression levels were normalized to actin and data are expressed as fold increase compared to WT cultures at each timepoint. **(B)** mRNA expression levels of BMPR-Ia in the absence and presence of BMP-2. **(C)** mRNA expression levels of BMPR-Ib in the absence and presence of BMP-2. *P < 0.05. Abbreviations include bone morphogenetic protein receptor-Ia (BMPR-Ia) and bone morphogenetic protein receptor-Ib (BMPR-Ib).

A possible explanation for this decrease is at this time point the osteoblasts are forming mineralized nodules, and it is known that cellular levels of *Alp* mRNA decreases as the cultures progress into the mineralization phase.[15]

Since BMP-2 signals through the canonical Smad pathway, we evaluated levels of p-Smad 1/5/8 in WT and KO osteoblast cultures. We show that KO osteoblasts have increased levels of p-Smad 1/5/8, suggesting increased BMP signaling (Fig. 4A). Previous studies have shown that BMPR-1b plays a significant role in osteoblast differentiation. Primary osteoblasts derived from transgenic mice overexpressing a truncated dominant negative BMPR-1b under the type I collagen promoter exhibited impaired osteoblast differentiation due to inhibition of BMP signaling.[20, 21] We show that BMPR-1b is significantly up-regulated after treatment with BMP-2 in KO osteoblast cultures compared to WT osteoblast cultures. In contrast, addition of BMP-2 into WT and KO osteoblast cultures decreased the expression of BMPR-1a. This finding is consistent with a previous study, in which BMP-2 treatment of 2T3 cells, an osteoblast cell line, resulted in decreased expression of BMPR-1a during osteoblast differentiation.[20] Taken together, these findings suggest that the absence of endogenous CTGF results in an increase in BMP signaling through BMPR-1b, which activates Smads 1, 5, and 8, resulting in up-regulation of *Runx2* levels thereby accelerating osteoblast differentiation.

In conclusion, our findings are the first to demonstrate, through functional studies, a novel function of endogenous CTGF in regulating osteoblast development and function by inhibiting BMP signaling. The findings are consistent with earlier studies suggesting that CTGF can act to inhibit BMP-2 and BMP-4 by preventing the ligands from binding to its cognate receptor. Based on the observations made in this study, endogenous CTGF appears to inhibit the effects of BMP-2 on osteoblast differentiation. In the CTGF KO osteoblasts where this inhibition is absent, BMP-2 has an enhanced differentiation effect via increased Smad signaling presumably through the BMPR-1b. This results in increased transcription of *Runx2* and other downstream factors required for osteoblast differentiation. This is a novel mechanism that warrants further investigation. How does endogenous production of CTGF regulate BMP-2 bioavailability and/or receptor levels? Based on our results, one would predict that use of the monoclonal anti-CTGF antibody, FG-3019, alone or in conjunction with BMP-2 treatment, may enhance bone formation in certain clinical scenarios (see Lambi and Popoff in this edition).

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Medical Student Research Project

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Spectrum of Vitamin D Deficiency in an Orthopaedic Outpatient Setting

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Abstract

Background: Vitamin D, otherwise known as the "sunshine vitamin," is vital to the mineralization of healthy bone. The primary objective of this retrospective study is to examine the prevalence of vitamin D deficiency among symptomatic orthopaedic outpatients. The secondary objective is to identify risk factors that predispose individuals to becoming vitamin D deficient. Understanding these risk factors may offer valuable insight to the prevention and reversal of the vitamin D deficient state that plays a critical role in the development of osteomalacia, fracture, neuromuscular pain and extra-skeletal disease.

Methods: A retrospective review was conducted on 70 symptomatic outpatients seen at the orthopaedic clinic at Temple University Hospital between January and May of 2010. All patients with a vitamin D 25-hydroxy (25-OHD) test ordered were included. Correlations between serum 25-OHD concentration and patient age, gender, BMI, ethnicity and presenting orthopaedic symptom were evaluated for significance. A univariate and multivariate analysis of an ordinal logistic regression was performed.

Results: Vitamin D sufficiency (25-OHD ≥34 ng/mL) was present in only 11% of the orthopaedic outpatients. Thirty-one percent were deficient (25-OHD 11–20 ng/mL) and 29% were severely deficient (25-OHD ≤11 ng/mL). Vitamin D deficiency was significantly associated with younger age (P = 0.0177, OR = 0.964, CI 0.936–0.994), African-American ethnicity (P = 0.0171, OR = 6.245, CI 1.385–28.151), and chondromalacia of the patella (P = 0.0283, OR = 4.352, CI 1.169–16.208). Vitamin D status did not significantly differ between men and women (P = 0.5475) and did not significantly correlate with BMI (P = 0.205). In a multivariate model, only age and ethnicity were significant independent predictors of vitamin D deficiency.

Conclusions: Our findings suggest that vitamin D deficiency is very prevalent among orthopaedic outpatients. We propose that individual risk can be predicted using an algorithm based on age and ethnicity alone.

Introduction

Vitamin D is well known for its role in developing healthy bones. A century ago, it was discovered that this "sunshine vitamin" maintains the homeostasis between calcium and phosphorus, which is vital to bone mineralization. Classically, its deficiency state presents as rickets in children and osteomalacia in adults. It can also present with neuromuscular pain syndromes, muscle weakness and fractures secondary to decreased bone mineral density.[1, 2]

Over the last decade, however, vitamin D has been receiving new praise. Recent studies have revealed its cardioprotective effects in heart failure, anti-proliferative effects in cancer and immunomodulatory effects in multiple sclerosis, psoriasis, rheumatoid arthritis, inflammatory bowel disease and type I diabetes.[3] In light of this new understanding, the potential extra-skeletal manifestations of an undiagnosed vitamin D deficiency are now of great concern.

The primary objective of our study is to determine the prevalence of vitamin D deficiency among symptomatic orthopaedic outpatients in North Philadelphia, PA. Our secondary objective is to identify risk factors that can predict a vitamin D deficiency. Higher levels of melanin seen in darker pigmented individuals act as a natural sunscreen, limiting cutaneous production of vitamin D. Thus, certain minority populations are predisposed to deficiency.[4] We hypothesize that the spectrum of vitamin D deficiency in this predominantly African-American community with musculoskeletal complaints will show suboptimal concentrations of vitamin D.

Materials and Methods

A retrospective review was performed on 70 outpatients seen at the orthopaedic clinic at Temple University Hospital between January and May of 2010. All patients presenting with musculoskeletal symptoms who had a 25-hydroxy (25-OHD) test documented in their medical record were included in this study. If multiple studies were performed, only the first 25-OHD concentration was included. Patients receiving vitamin D supplementation at the time the 25-OHD concentration was measured were excluded.

Our primary outcome was to identify how many patients were vitamin D sufficient, insufficient, deficient and severely

deficient. Vitamin D status was determined by serum concentrations of total 25-OHD, which represents the sum of its two naturally occurring forms, ergocalciferol (25-OHD_2) and cholecalciferol (25-OHD_3). Total 25-OHD is a superior indicator of vitamin D status compared to its biologically active metabolite 1,25-$(OH)_2D_3$ calcitriol because its longer half-life more accurately reflects vitamin D stores obtained from sunlight and diet over longer periods.[5] 25-OHD concentrations were measured using a Liquid Chromatography/Tandem Mass Spectrometry assay. There is currently no universal consensus as to what level of 25-OHD constitutes a deficiency.[2] The values we selected are based on our own orthopaedic practice (Table 1).

Table 1. Classification of Vitamin D Status by 25-OHD Concentration[a]

Vitamin D Status	25-OHD Concentration
Sufficient	≥34 ng/mL
Insufficient	21–33 ng/mL
Deficient	11–20 ng/mL
Severely Deficient	≤10 ng/mL

[a]25-OHD = 25-hydroxyvitamin D

Our secondary outcome was to determine the association between vitamin D deficiency and patient characteristics that included age, gender, body mass index (BMI), ethnicity and presenting orthopaedic symptom. BMI was calculated using heights and weights documented within a year of the 25-OHD test. Ethnicity was self-reported on the patient intake form. Orthopaedic symptom was identified by the International Classification of Disease (ICD-9) diagnosis corresponding to the office visit the 25-OH test was ordered. There were 12 categories of diagnosis including osteoarthritis, chondromalacia of the patella, loose body in knee, joint pain, rheumatism, dorsopathy, osteopathy and chondropathy, fracture, sprain, dislocation, contusion and surgical complication. In most cases, multiple diagnoses were recorded.

All demographic and clinical variables were analyzed statistically for a correlation with low levels of vitamin D at onset. Both univariate and multivariate analyses of an ordinal logistic regression were used to calculate odds ratios (OR) with 95% confidence intervals (CI). All P values less than 0.05 were considered significant.

Results

Vitamin D sufficiency (25-OHD ≥34 ng/mL) was present in only 11% of the orthopaedic population. Thirty-one percent were deficient (25-OHD level 11–20 ng/ml) and 29% were severely deficient (25-OHD ≤11 ng/mL).

Vitamin D deficiency was significantly associated with younger age (P = 0.0177), African-American ethnicity (P = 0.0171) and chondromalacia of the patella (P = 0.0283) on univariate analysis (Table 2). Older patients were 4% less likely to be deficient than younger patients (OR = 0.964, CI 0.936–0.994). African-American patients were six times more likely to be deficient (OR = 6.245, CI 1.385–28.151), and patients diagnosed with chondromalacia of the patella were four times more likely to be deficient (OR = 4.352, CI 1.169–16.208). Vitamin D status did not significantly differ between men and women (P = 0.5475) and did not significantly correlate with BMI (P = 0.205). In a multivariate model, only age (P = 0.018, OR = 0.964, CI 0.936–0.994) and African-American ethnicity (P = 0.0153, OR = 6.541, CI 1.433–29.857) were significant independent predictors of vitamin D deficiency.

Table 2. Univariate Analysis of Risk Factors for Vitamin D Deficiency[a]

Risk Factor	P value[b]	Odds Ratio	95% Confidence Limits	
Age	**0.0177**	**0.964**	**0.936**	**0.994**
Gender	0.5475	1.365	0.495	3.763
African-American	**0.0171**	**6.245**	**1.385**	**28.151**
Caucasian	0.1443	0.248	0.038	1.612
Body Mass Index	0.2050	1.044	0.977	1.115
Osteoarthritis	0.9009	0.944	0.382	2.331
Chondromalacia Patellae	**0.0283**	**4.352**	**1.169**	**16.208**
Loose Body	0.4841	0.473	0.058	3.854
Joint Pain	0.1003	2.214	0.858	5.724
Rheumatism	0.7124	0.823	0.293	2.313
Osteopathy	1.0000	1.000	0.264	3.782
Chondropathy	0.1800	2.564	0.647	10.158
Fracture	0.9863	0.990	0.309	3.168
Sprain	0.1612	0.333	0.071	1.551
Dislocation	0.1149	2.470	0.803	7.598
Contusion	0.0871	0.088	0.005	1.425
Surgical Complication	0.1443	0.248	0.038	1.612

[a]Outcome measured was vitamin D deficiency
[b]P value of <0.05 is significant

Using the multivariate model, we formulated an algorithm that predicts individual vitamin D status (Table 3). Probability of deficiency can be calculated by substituting the patient's age and ethnicity ("1" if African-American, "0" if not African-American) into the equation. For example, a 50-year-old African-American will have a 6.27% chance of being vitamin D sufficient, a 24.7% chance of being insufficient, a 37% chance of being deficient and 32% chance of being severely deficient. Therefore, the probability that this middle-aged African-American patient has sub-optimal levels of vitamin D exceeds 90%.

Discussion

The purpose of our study was to describe the spectrum of vitamin D deficiency among orthopaedic outpatients at Temple University Hospital in North Philadelphia, PA. This patient population represents a specific high risk group, namely African Americans living in an urban community. We found African-American status to be a statistically significant predictor of vitamin D deficiency on both univariate and multivariate analysis.

Previous studies looking at this high-risk population have drawn similar conclusions. In a study conducted at Fox

Table 3. Individual Prediction Algorithm for Vitamin D Status

Probability of being Sufficient (PROB S)[a]	$\frac{e^{-2.647 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}{1 + e^{-2.647 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}$
Probability of being Insufficient (PROB I)	$\frac{e^{-.7447 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}{1 + e^{-.7447 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}$ – (PROB S)
Probability of being Deficient (PROB D)	$\frac{e^{0.8116 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}{1 + e^{0.8116 + .0364\,[\mathbf{AGE}] - 1.878\,[\mathbf{AA}]}}$ – (PROB I) – (PROB S)
Probability of being Severely Deficient (PROB SD)	1 – (PROB D) – (PROB I) – (PROB S)

[a]For each patient, substitute their age into **[AGE]** and 1 into **[AA]** if they are African American or 0 into **[AA]** if not African-American.

Chase Cancer Center on adult African-American men living in North Philadelphia, 61% were found to have 25-OHD levels below 15 ng/mL, with a mean of 13.7 ng/mL.[6] Data from the National Health and Nutrition Examination Survey (NHANES III) demonstrated a significantly higher prevalence of vitamin D deficiency in minority populations when compared to white populations.[7] In a study published by the Mayo Clinic at an inner-city health center in Minneapolis, deficiency (25-OHD <20 ng/mL) was seen in 100% of African-American patients and 93% of patients presenting with persistent musculoskeletal pain.[8] Similar data has been published for minority pediatric populations living in low-income, urban areas.[9, 10]

Older age significantly decreased individual risk for being vitamin D deficient on univariate and multivariate analysis. The Mayo Clinic has described similar findings.[8] Other studies, in contrast, report a high prevalence of vitamin D deficiency in the elderly due to reduced sunlight exposure, decreased efficiency of cutaneous production, poor nutritional intake and decreased renal metabolism.[11]

Chondromalacia of patella was a statistically significant predictor for vitamin D deficiency on univariate analysis. To our knowledge, this relationship has not been published in other studies. Chondromalacia of patella is characterized by anterior knee pain secondary to soft-tissue swelling of the patellar cartilage of unclear etiology.[12] Surgery remains the current standard of treatment, yet some patients do not experience complete resolution of symptoms. Our findings suggest a potential therapeutic role for vitamin D supplementation in these patients.

Gender and BMI were not found to be significantly related to a vitamin D deficient state. Women, especially postmenopausal women, are at high risk for becoming deficient.[7, 13] The relationship between vitamin D deficiency and determinants of obesity such as BMI, waist circumference and metabolic syndrome remain controversial.[14, 15]

There are some limitations of our study. First, the ICD-9 diagnosis might not reflect prior or chronic orthopaedic issues of the patient. Second, we only included patients seen from January through May of 2010. Data collection year-round could improve our study as 25-OHD concentrations exhibit seasonal variation.[16] Third, we did not exclude other patient populations considered at risk for vitamin D deficiency including those with chronic kidney disease, liver disease, hyperthyroidism, HIV and burn injury.[1]

Our study is unique in that we have developed an algorithm that predicts where an individual would fall on the spectrum of vitamin D deficiency using age and ethnicity as variables. The potential value of this study to be used as a screening tool in outpatient clinics serving inner-city populations is profound. Vitamin supplementation is safe and relatively inexpensive — a cost-effective way to prevent and reverse a deficiency state that has been implicated in the pathogenesis of disease affecting almost every organ system in the body. We hope our simple algorithm can be adopted and utilized by the medical community in order to enhance awareness to this rising condition and potentially identify symptomatic individuals at risk.

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Medical Student Research Project

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Complications of FiberWire Fixation of Achilles Tendon Ruptures: A Case Series

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Abstract

Background: Temple University Hospital successfully repairs many Achilles tendon ruptures surgically. However, we feel obligated to report two unusual cases where patients experienced suture and skin breakdown after otherwise successful surgeries. In both cases, FiberWire sutures were used. The purpose of this study is to document these two cases and to conduct a systematic literature review.

Methods: Two patient charts were reviewed with their consent and IRB approval. A systematic literature review was conducted using MEDLINE and Cochrane Database.

Results: After surgical repair, both patients developed late wound dehiscence and purulent drainage from the wound site. In one case (Case 2), the tendon repair completely broke down. There was an excessive amount of hypergranulation tissue in both cases, possibly indicating a foreign body reaction.

Conclusions: FiberWire is commonly used in many orthopedic procedures, but to our knowledge, complications have only been seen in Achilles tendon repair. Our current hypothesis is that the superficial locations of FiberWire sutures in Achilles tendon repair may contribute to wound breakdown based on our findings and published literature.[18] We hope that our article prompts additional reporting of FiberWire-associated complications in Achilles tendon repair as well as the pathophysiology of FiberWire breakdown.

Introduction

Acute Achilles tendon rupture is a common injury in adults. The estimated incidence is 5.5–9 ruptures per 100,000 people in North American adults,[1] is most common in males in the third and fourth decade, and has a high prevalence during sporting activities.[1] Diagnosis is usually readily apparent by physical exam including decreased plantar flexion strength, a palpable gap at the site of the rupture, and a positive Thompson test.[1–3] Occasionally, an MRI may be needed to aid in the diagnosis. There still exists some controversy in the literature whether non-operative or operative treatment is most effective for the treatment of this injury. The goal of treatment is to restore normal strength, full ankle range of motion, normal function, and minimize complications of weakness, re-rupture, infection, and disability.[4–8] The most current meta-analysis of 14 randomized controlled trials favored operative intervention as the re-rupture rate was significantly lower with surgical treatment.[9] Surgical intervention does allow accelerated rehabilitation, earlier weight bearing, and earlier return to work as compared to traditional casting protocols.[10–13]

While there is no universal agreement as to what suture is best for fixing Achilles tendon ruptures, most American surgeons prefer braided, non-absorbable sutures.[14] These include braided polyethylene (Ethibond®) and braided polyblend polyethylene sutures (FiberWire®, Orthocord®, Ultrabraid®). Laboratory experiments have demonstrated that FiberWire suture has superior tensile strength over Ethibond.[15–17] FiberWire (Arthrex, USA) was one of the first high tensile strength sutures developed and has been used extensively by orthopedic surgeons for a variety of orthopedic procedures including rotator cuff repairs, quadriceps and patellar tendon repairs, and Achilles tendon repairs. Its core is made of ultra-high molecular weight polyethylene, surrounded by a braided polyester jacket, and it is then externally coated with silicon.[18] Although surgical treatment has many advantages as noted above, there are also potential complications as with all operations. The major risks associated with Achilles tendon repair center on deep infection and skin healing problems, accentuated by the superficial position of the tendon beneath the skin and lack of soft tissue coverage.[4–8] Most tendon ruptures occur in the hypovascular zone 2 to 6 centimeters above the tendon insertion site on the calcaneus.[2] This site has very little subcutaneous coverage and may explain some of the reasons for the complications that are seen after operative repair. Previous laboratory studies have documented higher bacterial adherence in braided non-absorbable sutures than in nonbraided sutures.[19] Some studies have shown higher rates of bacterial ingrowth with FiberWire compared to Ethibond;[19] others have shown chronic inflammation *in vivo* with FiberWire.[18] The purpose

of this study is to present two cases of delayed infection after Achilles tendon repair with FiberWire. This report may raise awareness in the orthopedic community of potential issues with this suture material.

Materials and Methods

After obtaining IRB approval, patient charts for the two cases were collected retrospectively with their consent. The entire history, operative notes, postoperative visits, and pathology reports after reoperation were all analyzed. A literature review using MEDLINE and the Cochrane Library was also conducted.

Case Series

One patient (Case 1), a 24-year-old male, ruptured his right Achilles tendon and had a surgical repair in California. He had no immediate problems, but nine months postoperatively, he presented to the clinic complaining of pain and wound problems. Physical exam then showed no palpable gap in the tendon and a negative Thompson's sign, but he did exhibit wound breakdown with excessive granulation tissue. He was subsequently taken to surgery where suture remnants were removed, cultures were taken, and a cast was applied. Wound cultures grew Methicillin resistant staphylococcus aureus and appropriate IV, then oral, antibiotics were prescribed. Local wound care and cast changes led to slow, but eventual wound healing. Range of motion and strength returned over 2–3 months and the patient eventually returned to full function.

The second patient (Case 2) was a 47-year-old male who ruptured his right Achilles tendon stepping off a street curb. He had the classic physical findings of a palpable gap, positive Thompson sign, and weakness of plantar flexion strength. He had no associated medical diseases such as previous steroid injections, use of fluoroquinolone antibiotics, or preexisting foot or ankle structural abnormalities.[2, 3] He did have poorly controlled hypertension, which was stabilized preoperatively. He elected to undergo operative repair and this procedure was done at Temple University Hospital using #2 FiberWire sutures with a Krackow type suture repair. He was casted postop in equinus and then started on our standard postop rehabilitation program at the two-week mark, when the cast was removed and replaced with a CAM boot with a variable angle ankle joint. He had no postop wound problems and was walking in shoes with a silicone rubber heel insert without difficulty. Three months postop, he returned to the clinic with pain, swelling, and drainage from his posterior heel wound. Unfortunately, his blood pressure then was significantly elevated and could not be adequately controlled and thus, only a limited incision and drainage could be done under local anesthesia with sedation. Wound cultures grew MRSA and appropriate antibiotics were administered. The wound did not heal and he underwent successful split thickness skin grafting to the wound. He did regain full ankle motion and strength over several months and did eventually return to work.

Discussion

Postoperative infection is a known complication of all operative procedures, but the delayed onset of these infections and the severity of the complications in our patients made these cases notable. Neither of our patients had contributory medical conditions, both were compliant with the postop rehabilitation regimen, and both developed significant problems more than three months after their operative procedures.

There is a paucity of literature documenting the complications of FiberWire suture in otherwise successful surgeries. A systematic literature search using MEDLINE and the Cochrane Library yielded only one case series.[18] Five Army soldiers, who underwent transtibial or transfemoral amputations after severe trauma, developed wound problems in their stumps after fascial closure with FiberWire sutures. These complications included sinus tract formation, clear wound drainage, and amorphous tissue formation around the sutures. Pathologic examination of the suture material and granulation tissue revealed multinucleated giant cells that had engulfed the silicone outer coating of the sutures. One culture grew MRSA. The sutures were removed and replaced with Ethibond sutures and the wounds healed.[19] The common symptoms in the previously mentioned case series and ours included delayed wound healing and chronic inflammatory reaction. Both of our wounds became infected with MRSA. Removal of the suture remnants, IV antibiotics, and aggressive local wound care did lead to eventual healing in all patients.

FiberWire is used in a variety of orthopedic procedures due to its increased strength and lower rate of breakage.[15–17] We have not frequently seen these severe healing problems in shoulder, elbow, or quadriceps tendon repairs. We, therefore, feel that the superficial position of the Achilles tendon repair may contribute to an enhanced inflammatory response. Also, the giant cell inflammatory response seen in response to the silicone outer covering of FiberWire has been seen before with silicone spacers used in radial head and great toe implants in sensitive patients. We do not know why certain patients are more likely to develop these hypersensitivity reactions and this issue needs further investigation. These issues, however, do point out the need to reevaluate the use of FiberWire sutures in Achilles tendon repairs and to consider possible use of less reactive sutures in this area.

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Original Research

Nerve and Tendon Injury with Percutaneous Fibular Pinning: A Cadaveric Study

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Abstract

Objective: The purposes of this study were to measure the average distance from a percutaneous pin in each quadrant of the distal fibula to the sural nerve and nearest peroneal tendon, and define the safe zone for pin placement as would be used during surgery.

Method: Ten fresh-frozen cadavers underwent percutaneous pin fixation into four quadrants of the distal fibula. The sural nerve and peroneal tendon were identified as they coursed around the lateral ankle. Distances from the K-wire in each quadrant to the anatomic structure of interest were measured.

Results: Average distances (mm) from the K-wire to the sural nerve in the anterolateral, anteromedial, posterolateral, and posteromedial quadrants were 19.1 ± 8.9 (range, 5.1–35.5), 12.8 ± 8.2 (range, 0.3–27.8), 12.6 ± 6.8 (range, 3.0–27.8), and 5.9 ± 5.5 (range, 0.1–19.9), respectively. Average distances from the K-wire to the nearest peroneal tendon in the anterolateral, anteromedial, posterolateral, and posteromedial quadrants were 15.7 ± 4.4 (range, 9.5–23.1), 11.9 ± 5.2 (range, 3.2–21.7), 6.3 ± 3.9 (range, 0.1–14.4), and 1.0 ± 1.6 (range, 0–5.6), respectively.

Conclusions: Percutaneous pinning of distal fibula fractures is a successful treatment option with minimal complications. Our anatomical study found the safe zone of percutaneous pin placement to be in the anterolateral quadrant. The sural nerve can be as close as 5.1 mm and the peroneal tendons as near as 15.7 mm. In contrast, the posteromedial quadrant was associated with the greatest risk of injury to both the sural nerve and peroneal tendons.

Introduction

Distal tibia and fibular fractures are frequently treated with open reduction and internal fixation of the tibia and fibula, with fibular fixation typically consisting of plate and screw fixation.[1] With tibial pilon fractures and some extra-articular distal tibia fracture patterns, the fibular fracture often does not involve the ankle joint, and the surgeon has to decide whether or not fibular fixation is needed.[2] Unfortunately, soft tissue concerns with these injuries may preclude fibular plating, or lead to wound complications when plating is done through usual extensile incisions. Closed reduction and percutaneous pinning is a surgical treatment option for displaced distal fibular fractures and segmental distal fibular fractures.[3–5] Percutaneous pinning is a successful treatment method for elderly patients and those with multiple medical comorbidities who are prone to wound complications and osteoporotic bone such as diabetics and chronic steroid users.[5–8] The percutaneous technique avoids significant stripping of the soft tissue envelope and provides lateral column stabilization. In the absence of fibular fixation in concomitant distal tibia fractures, there is destabilization of the lateral column depending on fracture configuration and associated ankle injuries. Further, the fibula fracture has the potential to affect tibial length, alignment, and rotation.[2, 3] This ultimately can affect functional outcome, tibial malunion rate in combined tibia-fibula fracture, and post-traumatic ankle arthrosis.[9, 10]

The goals of fibular pinning or an intramedullary rod are to improve bending stability of the tibia and fibula fractures, and to some degree, maintain fibular length. Because this is often done as a percutaneous method, the peroneal tendons and sural nerve are at risk for injury. Furthermore, we have noted that the incidence of painful implants is not insignificant in our practice, potentially in part due to irritation of nearby anatomic structures. The proximity of percutaneously-placed fibular pins to the nearby structures, namely the peroneal tendons and sural nerve, has not been described. The purpose of this anatomic study is to measure the distance from the percutaneous pin in the distal fibula to these anatomic structures and define the safe zone for pin placement correlated with fluoroscopic images as would be used during surgery.

Materials and Methods

Ten fresh-frozen cadaver lower extremities with an average age of 78 years (range, 57–95 years) were dissected after percutaneous pinning of the distal fibulas. All procedures were performed on left fibulas from six female and four male cadavers in the prone position and ankles in the resting equinus position. Plantar flexion angles were measured with a

goniometer for each cadaver and averaged 33.8 degrees (range, 17–51 degrees). No cadavers had evidence of lower extremity bony disease or trauma. Under mini C-arm fluoroscopic guidance, four Kirschner wires (K-wires) (1.1 and 2.0 mm) were inserted with a Small Battery Drive drill (Synthes, West Chester, PA).

One orthopaedic trauma-trained fellow performed all percutaneous pin placements. K-wires were inserted through the skin 1 to 2 cm distal to the tip of the lateral malleolus and directed towards and parallel to the intramedullary canal as in standard approach for fibular intramedullary fixation. One K-wire was placed into each of four different quadrants of the distal fibula and verified by anteroposterior and lateral fluoroscopic imaging. The four quadrants as viewed from the axial plane were defined as anteromedial, anterolateral, posteromedial, and posterolateral (Figures 1 and 2). A chief-level orthopaedic surgery resident dissected the lateral ankle after all four pins were inserted. The dissection and percutaneous pinning was supervised by one orthopaedic trauma fellowship-trained attending.

Each cadaveric dissection was performed without disrupting local anatomy and included identification of the sural nerve and its branches (SN), and the peroneal tendons (PT). Using a single incision, the PT were dissected from approximately 5 cm proximal to the lateral malleolus to an area several centimeters distal to the K-wires along the lateral border of the foot. The shortest distance from the center of each K-wire to the closest section of the sural nerve, whether a branch or the nerve proper, was measured with a Brown & Sharpe caliper (Dial-Cal Metric Model no. 599-579-14; North Kingstown, RI). Distances to the nearest tenth of a millimeter were recorded. The same procedure was repeated for PT, measuring the shortest distance between each K-wire and the nearest tendon. K-wires that penetrated the sural nerve or peroneal tendons were given a distance of 0 mm and wires that abutted against these structures were recorded as 0.1 mm. All measurements were performed and recorded by three different individuals. Measurements were then averaged and the range, standard deviation, and variance were calculated.

Results

The sural nerve and peroneal tendons were identified in all cadavers. The distances of the K-wire to these anatomical structures are summarized in Tables 1 and 2. Two of the 10 K-wires in the posteromedial quadrant (PM-Q) were found to be abutting the sural nerve. In four of the 10 specimens, the PM-Q K-wire was found to be piercing the peroneal tendons and in three separate specimens abutting the tendons as they curved anteriorly around the distal fibula. The PM-Q K-wire was an average distance of 5.9 mm (range, 0.1 [abutting the nerve]–19.0 mm; SD 5.52) from the sural nerve and an average of 0.96 mm (range, 0 [piercing the tendon]–5.6 mm; SD 1.61) from the peroneal tendons.

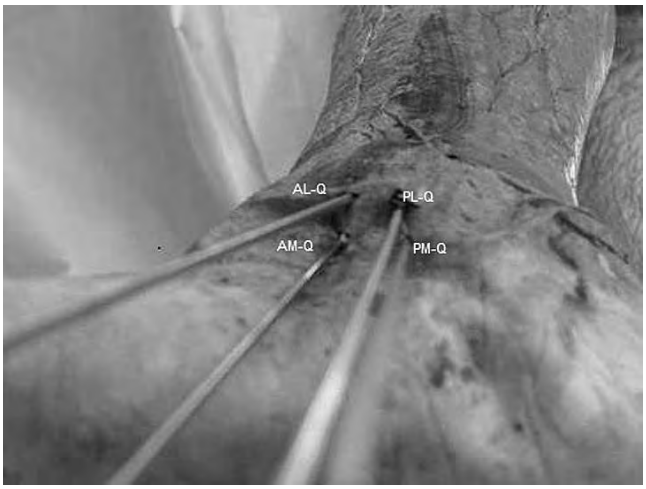


Figure 1. Axial view of K-wire placement in distal fibula of left cadaver leg. AL-Q = anterolateral quadrant; AM-Q = anteromedial quadrant; PL-Q = posterolateral quadrant; PM-Q = posteromedial quadrant.

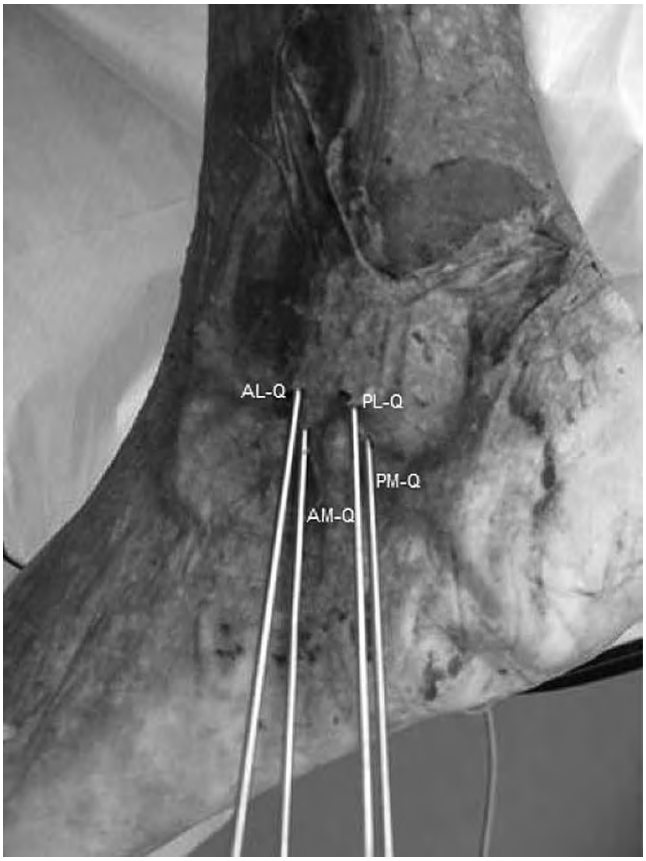


Figure 2. Lateral view of K-wire placement in resting plantar flexion.

In one of 10 cadavers, the K-wire abutted the peroneal tendons during insertion into the posterolateral quadrant (PL-Q); the average distance between PL-Q K-wire and the tendons was 6.3 mm (range, 0.1–14.4 mm; SD 3.9). In one cadaver, the PL-Q K-wire was only 3 mm from a branch of the sural nerve and the average distance from wire to sural nerve was 12.56 mm (range, 3–27.8; SD 6.82).

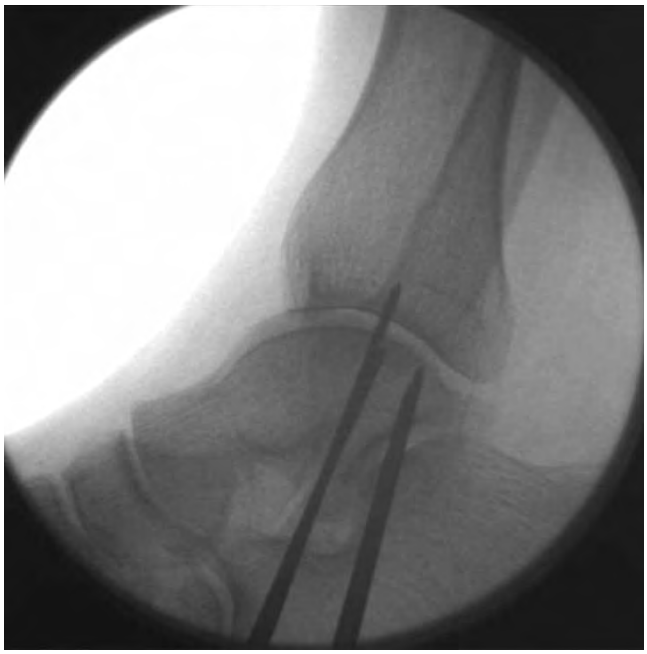


Figure 3. Lateral fluoroscopic image of K-wires after insertion into distal fibula.

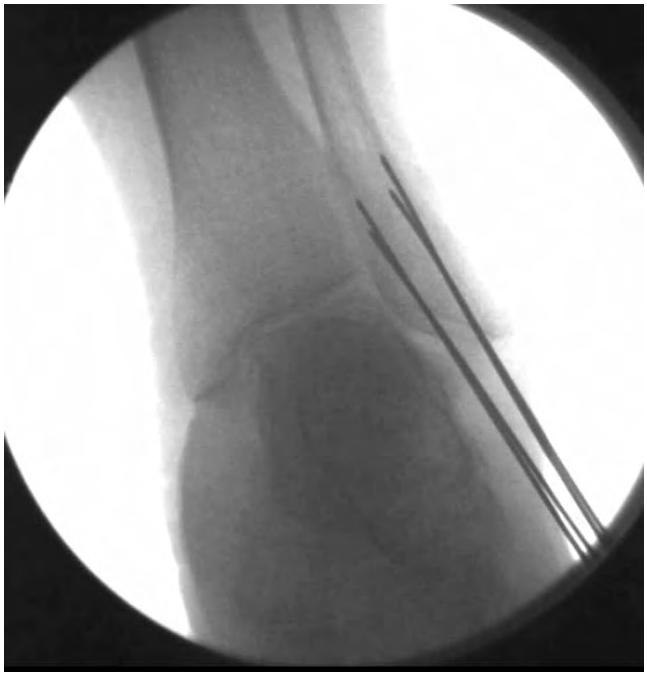


Figure 4. Anteroposterior fluoroscopic image of left ankle showing K-wires in four quadrants.

No K-wires in the anteromedial quadrant (AM-Q) contacted the nerve or tendons, although in one cadaver the wire was only 0.3 mm from a branch of the sural nerve. The average distance between AM-Q K-wire and nerve was 12.82 mm (range, 0.3–27.8; SD 8.22). K-wires were an average distance of 11.92 mm (range, 3.2–21.7 mm; SD 5.2) from the peroneal tendons.

Table 1. Distances and Injury to Sural Nerve After K-wire Placement into Four Quadrants of Distal Fibula

Quadrant	Avg.	Min–Max	SD	Variance	# Pierced or Abutted	% Injured
Anterolateral	19.1	5.1–35.5	8.9	80.1	0	0
Anteromedial	12.8	0.3–27.8	8.2	67.6	0	0
Posterolateral	12.6	3.0–27.8	6.8	46.5	0	0
Posteromedial	5.9	0.1–19.0	5.5	30.5	2	20

Distances are in millimeters.

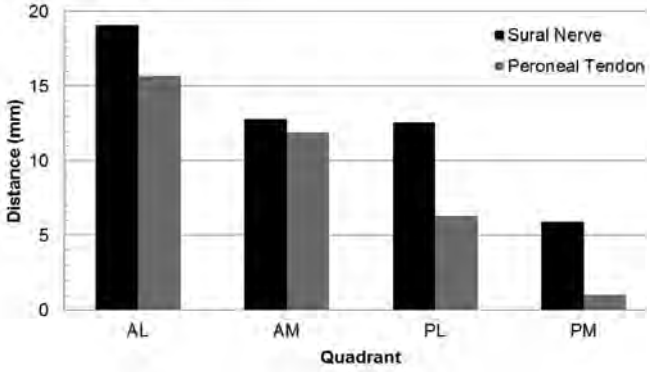
Table 2. Distances and Injury to Peroneal Tendon After K-wire Placement into Four Quadrants of Distal Fibula

Quadrant	Avg.	Min–Max	SD	Variance	# Pierced or Abutted	% Injured
Anterolateral	15.7	9.5–23.1	4.4	19.1	0	0
Anteromedial	11.9	3.2–21.7	5.2	27.0	0	0
Posterolateral	6.3	0.1–14.4	3.9	15.5	1	10
Posteromedial	1.0	0–5.6	1.6	2.6	7	70

Distances are in millimeters.

The anterolateral quadrant (AL-Q) K-wires were the furthest from the sural nerve with an average distance of 19.1 mm (range, 5.1–35.5 mm; SD 8.95). Distances measured in this quadrant exhibited the greatest variability among quadrants. Similarly, no tendons were abutted or pierced when K-wires were inserted in this quadrant (average distance, 15.65 mm; range, 9.5–23.1 mm; SD 4.37).

Figure 5. Average Distance from K-Wire in Each Quadrant of Distal Fibula to Sural Nerve and Nearest Peroneal Tendon



AL = anterolateral, AM = anteromedial, PL = posterolateral, PM = posteromedial.

Discussion

The fundamental advantage of fibula fixation in certain isolated fibula fractures and in combined distal tibia-fibula fractures is well established.[10] Intramedullary fibula fixation is a successful surgical option in patients with potential for poor wound healing, soft tissue damage, osteoporotic bone, segmental or fragility fractures, open fractures and elderly patients.[5] Percutaneous pinning of the fibula is a more minimally invasive option than open reduction internal fixation, with limited incisions and less prominent implants. Pub-

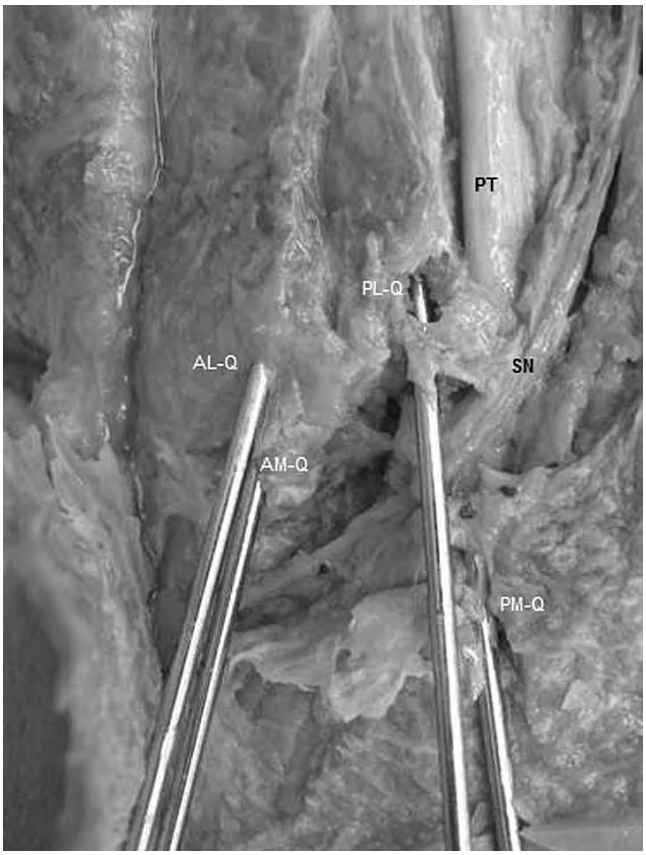


Figure 6. Dissected cadaver specimen with K-wires in each quadrant and identification of SN and PT.

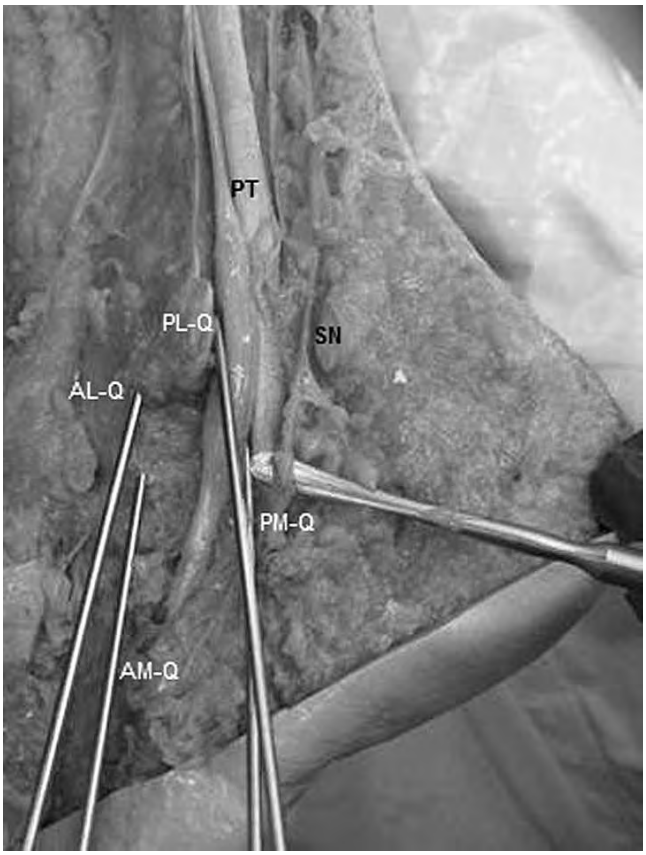


Figure 7. Dissected specimen with K-wires in all four quadrants and identification of SN and PT. Note proximity of posterolateral and posteromedial K-wires to nerve and tendon.

lished studies for intramedullary fibular fracture fixation describes the starting point to be at the distal tip of the lateral malleolus or 2 mm medial to the distal tip.[1, 4–6, 11] However, the starting point in relation to anatomic structures has not been described. The rate and morbidity of sural nerve and peroneal tendon injury and irritation from intramedullary fibula fixation has also not been previously reported.

Indications for intramedullary fibula fixation include segmental or axially stable fractures, open fractures, fragility fractures, patients with potential healing problems or soft tissue damage and any displaced ankle fracture that involves the lateral malleolus.[3–6]

Our cadaveric study looked at pin placement starting at the distal tip of the lateral malleolus in four different quadrants — anteromedial, anterolateral, posteromedial and posterolateral. The posteromedial quadrant starting point was the most dangerous with regard to injury of the peroneal tendons and/or sural nerve. Twenty percent (2/10) of cadavers had pins that abutted the sural nerve and 70 percent (7/10) cadavers had pins that pierced or abutted the peroneal tendons. The posterolateral quadrant was the second riskiest location for intramedullary wire placement. Ten percent (1/10) of cadavers had pin placement next to the peroneal tendon and there was no injury to the sural nerve with an average distance of 12.6 mm.

Our anatomic study found the safe zone for the starting point in percutaneous pinning of the distal fibula to be in the anterolateral quadrant of the lateral malleolus. The ideal pin orientation is parallel to the medullary canal. No cadavers had damage to the sural nerve or peroneal tendons in this group. The sural nerve was as close as 5.1 mm from the pin insertion point with an average distance of 19.1 mm. The peroneal tendons were as close as 9.5 mm from instrumentation with an average proximity of 15.7 mm.

Open fibular plating can be complicated by wound complications, particularly in high energy injuries, as well as by late complaints of painful implants. Wound complications from closed distal fibula fractures treated with open reduction internal fixation occur at a rate as high as 17.5%.[12, 13] Lee et al. compared pinning of the fibula with a Knowles pin to open reduction internal fixation. No patients treated with the pin had wound complications. The tubular plate fixation group had a wound complication rate of 13.3%.[11] There was no statistical difference in the rate of elective implant removal. No patients with the pin complained of pain due to the instrumentation; however 24 out of 45 patients opted for removal. Forty percent (12 out of 30 patients) of the plate fixation group complained of painful screws or plates and 18 of them elected for removal.[11] Brown et al. had a symptomatic plate/screw rate of 31% in 126 patients treated by open

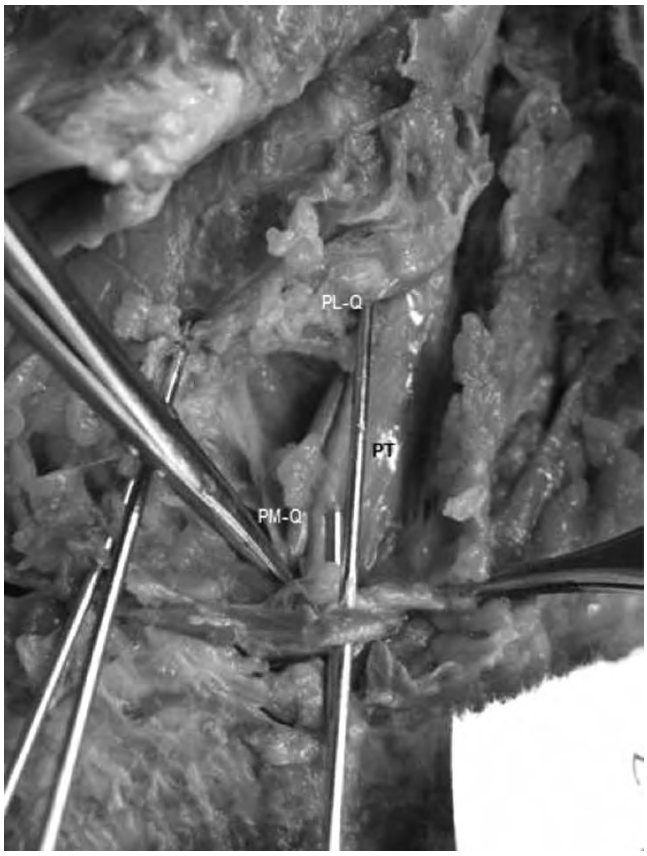


Figure 8. Dissected cadaver ankle specimen with pins in four quadrants and identification of SN and PT. Posteromedial quadrant pin is piercing peroneal tendons and abutting the sural nerve. Posterolateral quadrant pin is abutting the peroneal tendon.

plating.[14] Bugler et al. reviewed 105 unstable ankle fractures treated with the Acumed fibular nail. The average patient age was 64.8 years and 76% had significant medical comorbidities. The lateral wound infection rate was 4.7% (five patients) and 15% (16 patients) required removal of the nail or screws.[4] The locking screws were removed in nine patients and the fibular nail in seven patients.[4] Appleton et al. treated 37 patients with unstable ankle fractures with a fibula intramedullary nail. The average patient age was 67 years and all of them had a medical comorbidity that compromised the skin on the lateral ankle; such as diabetes or chronic steroid use. One patient had a post-operative superficial infection of the lateral ankle that required irrigation and debridement in the operating room and a week of antibiotics.[5] The patients average Olerud and Molander score was 87 out of 100 and 85% had ankle range of motion that was within 90% of the uninjured ankle at final follow-up.[5]

Rajeev et al. performed a retrospective review of 24 patients aged 71 to 91 years with fragility fractures and treated with intramedullary fibula fixation reported no wound complications, no non-unions or deep infections.[6] The average union rate for fibula intramedullary fixation was 8.7 weeks.[6] Pritchett studied functional outcome in Rush rods vs. plate osteosynthesis. The study had good to fair functional outcomes in 88% of patients that underwent rush rods fixation compared to 76% of patients treated with open reduction internal fixation.[7] The Rush rod group reached full weight bearing six weeks earlier than the open reduction internal fixation group.[7]

Although these several studies cited above indicate the utility of intramedullary fibular pinning as a treatment option for certain fractures, very little mention is made of the risks of nerve or tendon injury in their series. The clinical significance of smooth pin penetration of the sural nerve or peroneal tendons is not well described, but we believe that it is safe to assume that this could contribute to unexplained pain postoperatively. Larger diameter implants such as locked intramedullary nails marketed for distal fibular fixation do not provide any surgical technique suggestions for avoiding nerve or tendon injury. It is possible that careful evaluation in unexplained complaints of pain or limited motion postoperatively could possibly point to inadvertent injury as an etiology.

Weaknesses of our study involve the limitations with fixed (not fresh) cadaveric specimens. The ankles were in a fixed equinus position and unable to be manipulated during the percutaneous pinning. Therefore, our study was limited in observing what ankle position, in regards to the degree of plantarflexion and inversion, puts the anatomic structures furthest from the starting point. Another weakness of our study is the smaller instrumentation size. We used smaller diameter K-wires (1.1 and 2.0 mm) than the cannulated drills and reamers (range from 3.1–6.1 mm) used in the Acumed and Biomet fibular nail technique.[4, 6, 8] Therefore, the safety zone for percutaneous pinning with a K-wire is theoretically larger than the safety zone for a 4.5 mm diameter rod. Furthermore, our cadaver study has no clinical follow-up. The possible complications of Kirschner wire migration, pin site infection and loss of reduction could not be addressed.

Percutaneous pinning of distal fibula fractures is a successful treatment option with minimal complications. Our anatomical study found the safe zone of percutaneous pin placement to be in the anterolateral quadrant. The sural nerve can be as close as 5.1 mm and the peroneal tendons as near as 15.7 mm.

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Original Research

Does Intraoperative Fluoroscopy Improve Component Position During Anterior Hip Arthroplasty?

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Abstract

Objective: The goal of this retrospective review is to determine if fluoroscopic guidance improves acetabular cup abduction and anteversion alignment during anterior total hip arthroplasty.

Method: A single-center, case-control study of 199 patients (fluoroscopy group = 98, non-fluoroscopy group = 101) undergoing anterior hip arthroplasty. Acetabular cup abduction and anteversion angles were measured and compared between groups.

Results: The fluoroscopy group had mean abduction and anteversion angles of 43 and 23 degrees, respectively. The non-fluoroscopy group had mean abduction and anteversion angles of 46 and 23 degrees, respectively. Difference in acetabular abduction angle between groups was significant but radiographic anteversion was not significantly different.

Conclusions: A significantly higher percentage of acetabular cup abduction angles were in the safe zone in the fluoroscopy group. The percentage of cups in combined anteversion and abduction safe zones was higher in the fluoroscopy group. Use of fluoroscopy is not required for proper anteversion placement of acetabular components but may increase combined safe zone placement.

Introduction

In 1947, Robert Judet performed the first hip arthroplasty through an anterior approach at Hospital Raymond Poincare outside Paris. He originally named this the "Hueter" approach, which may have been a reference to Hueter Volkmann's method for drainage of a hip infected with tuberculosis.[20] Today, it is commonly referred to as the Smith-Peterson approach. Over the years, this technique has been modified to allow exposure of the femur and pelvis with less soft trauma by avoiding release of any of the surrounding muscles from their bony attachments.

The direct anterior approach to total hip arthroplasty (THA) is a less invasive technique with the advantages of reduced soft tissue trauma, lower dislocation rate, and earlier improvement in function as compared to the posterior and anterolateral approaches.[22] This can be performed with or without C-arm fluoroscopic guidance, but potential benefits of an image intensifier include improved component positioning, longer implant survival, less wear, and better range of motion.[25]

Acetabular orientation is critically important in the outcome of hip arthroplasty. Component positioning is related to impingement, stability, wear rates, and survivorship.[23] Poor cup positioning results in limited hip range of motion and undesired impingement. Kummer et al. suggest acetabular inclination between 35° and 45° and anteversion less than 20° for optimal hip range of motion.[13] Increases in linear polyethylene wear of 40% occur with abduction angles of ≥45°.[28] Avoiding malposition of components is crucial in reducing the occurrence of aseptic loosening and the incidence of THA revisions which is expected to double in the next 20 years, reaching an estimated 96,700.[23]

In 1978, Lewinnek et al. described a safe zone for acetabular cup positioning of 15 ± 10° of anteversion and 40 ± 10° of abduction.[14] In their review of 300 THA patients, the difference in dislocation rate was statistically less for subjects within the safe zones versus those outside of the defined safe zones (1.5% vs. 6%). Further studies have supported the importance of proper placement despite the varied consensus on safe zone parameters. In 1990, McCollum and Gray performed a prospective study of 441 THAs and reported a dislocation rate of 1.14%.[21] They concluded that 30–50° of abduction and 20–40° of anteversion prevents dislocation and impingement. Safe zone recommendations vary from little or no anteversion to 40° and depend on method of THA fixation.[6, 10, 14, 21, 24] Presently, it is accepted that ≤45° of acetabular abduction provides optimal stability and wear rates.[18]

To our knowledge, no studies have compared postoperative acetabular component positioning after anterior THA with and without fluoroscopy. Proposed disadvantages of fluoroscopy are increased operative times, radiation exposure to both the surgeon and patient, and field contamination.[4, 29, 34] The goal of this retrospective review is to determine if fluoroscopic guidance improves acetabular cup abduction and anteversion, thus prompting the question, is fluoroscopy necessary?

Methods

A single-center, case-control study of 199 patients who underwent primary THA at a single institution were ran-

domly selected from the primary surgeon's case log. All patients were operated on by a single surgeon (A.S.). Patients for the non-fluoroscopy (NF) group were selected from the 2008 surgical log because the primary surgeon was not yet using fluoroscopy consistently during this time. Subjects for the fluoroscopy (FL) group were selected from the 2011 surgical log because fluoroscopy was routinely used for patients during that year. Fluoroscopy was used intraoperatively during reaming of the acetabulum and socket impaction with adjustments made until components were fully seated in the pelvis. Imaging was not used live to guide insertion. Components were adjusted if determined to be outside the safe zones and the same fluoroscopy procedure was repeated until the surgeon was satisfied with positioning. Safe zones were defined as an anteversion of 15 ± 10° and an abduction angle of 40 ± 10° as described by Lewinnek et al.[14]

Types of implants used in the FL group (n = 98) include Corail Total Hip Systems (n = 5, 5.4%) and Trilock Bone Preservation Systems (n = 87, 94.6%) (DePuy Synthes; Warsaw, IN). In the NF group (n = 101), Corail (n = 53, n = 54.1%), Summit Cementless Hip System (n = 4, 4.1%), and Trilock products (n = 41, 41.8%) were used (DePuy Synthes).

One researcher (a senior-level resident) measured acetabular cup abduction and anteversion angles on six-month postoperative anteroposterior (AP) pelvic radiographs. This was performed using the method described by Widmer and Ing which allows for obtaining angles from an AP pelvic x-ray (Figure 1).[35] Acetabular inclination was determined by drawing a line parallel to the tear drop and another line through the long axis of the acetabular ellipse. Anteversion was measured by dividing the short axis of the ellipse which reflects the cup opening by the total length of the cup and corresponding the ratio to the table or graph in Widmer and Ing's article.

Exclusion criteria were revision arthroplasty, periprosthetic fracture, metal-on-metal prostheses, and insufficient postoperative radiographs that would preclude proper acetabular measurements. After exclusion, 101 NF and 92 FL replacements were included in the study. Demographic data including age, body mass index (BMI), and gender were collected.

Data was combined to provide a mean, range, standard deviation, and p-value for each variable. Analysis of variance (ANOVA) was used to determine differences between variables. A p-value of ≤0.05 was used for statistical significance.

Results

Demographic data is displayed in Table 1. A significant difference ($p \leq 0.05$) in acetabular cup abduction between the FL and NF groups was found (Figure 2). The difference in anteversion angle between the two groups was not significant ($p = 0.875$). The FL group (n = 98) had mean abduction and anteversion angles of 43.4 and 23.1 degrees, respectively. The NF group's (n = 101) mean abduction and anteversion angles were 45.9 and 23.1 degrees, respectively. Eighty-eight percent (n = 86) of acetabular cup abduction angles in the FL group fell within the safe zone (30–50°) versus 72% (n = 73) in the NF group. Eight-six percent (n = 85) of anteversion angles in the FL group fell within the safe zone (15 ± 10°) versus 79% (n = 101) in the NF group.

The number of patients with components in safe zones for both anteversion and abduction were 78 (80%) in the FL group and 64 (63%) in the NF group. There was a trend toward greater combined safe zone placement in patients undergoing anterior THA with intraoperative image guidance.

Discussion

THA is one of the most successful procedures performed by orthopaedic surgeons. Anterior THA is the only approach that uses an internervous plane, which is between the superior and inferior gluteal nerves and the femoral nerve. The goal of this approach is to obtain immediate stability by sparing posterior muscle attachments. Surgical approaches and component positioning affect postoperative stability and function with the anterior approach being associated with earlier return to function, shorter hospital stay, and better perioperative outcome in some studies.[1, 19, 23] Another benefit of the anterior approach is preservation of gluteal muscles in cases of future revision surgery that tend to be more difficult and invasive than primary arthroplasty.[3]

Acetabular component positioning affects patient outcomes after THA. Impingement, pelvic osteolysis, and bearing wear are all associated with suboptimal placement.[7, 12, 23, 33] Patient-related outcomes are also affected by cup positioning. One example is leg length discrepancy (LLD). LLD after THA is a cause of postoperative morbidity due to limping, muscle strain, sciatic nerve palsy, and aseptic loosening.[32] Additionally, it is a common cause of litigation in the United States.[27] Roder compared 478 cases of lengthening and 275 cases of shortening after THA with matched controls.[32] Outcomes were ability to walk greater than 60 minutes, hip pain, limping, and patient satisfaction. Compared to controls, patients with lengthened extremities were more likely to not be able to walk for more than one hour, more likely to limp at follow-up, and more likely to not achieve excellent patient satisfaction scores (OR 1.70, 95% CI 1.28–2.26; OR 2.08, 95% CI 1.55–2.8; and OR 1.67, 95% CI 1.23–2.38; respectively). In the shortened group, the odds ratios for these same parameters were 1.23 (95% CI 0.84–1.81), 2.61 (95% CI 1.78–3.21), and 2.15 (95% CI 1.44–3.21), respectively. LLD is associated with significant patient dissatisfaction and is largely preventable by surgeon technique.

Leg length differences from our patient cohort are not reported although LLD in patients after anterior THA have been reported in the literature. Matta's series of 437 patients

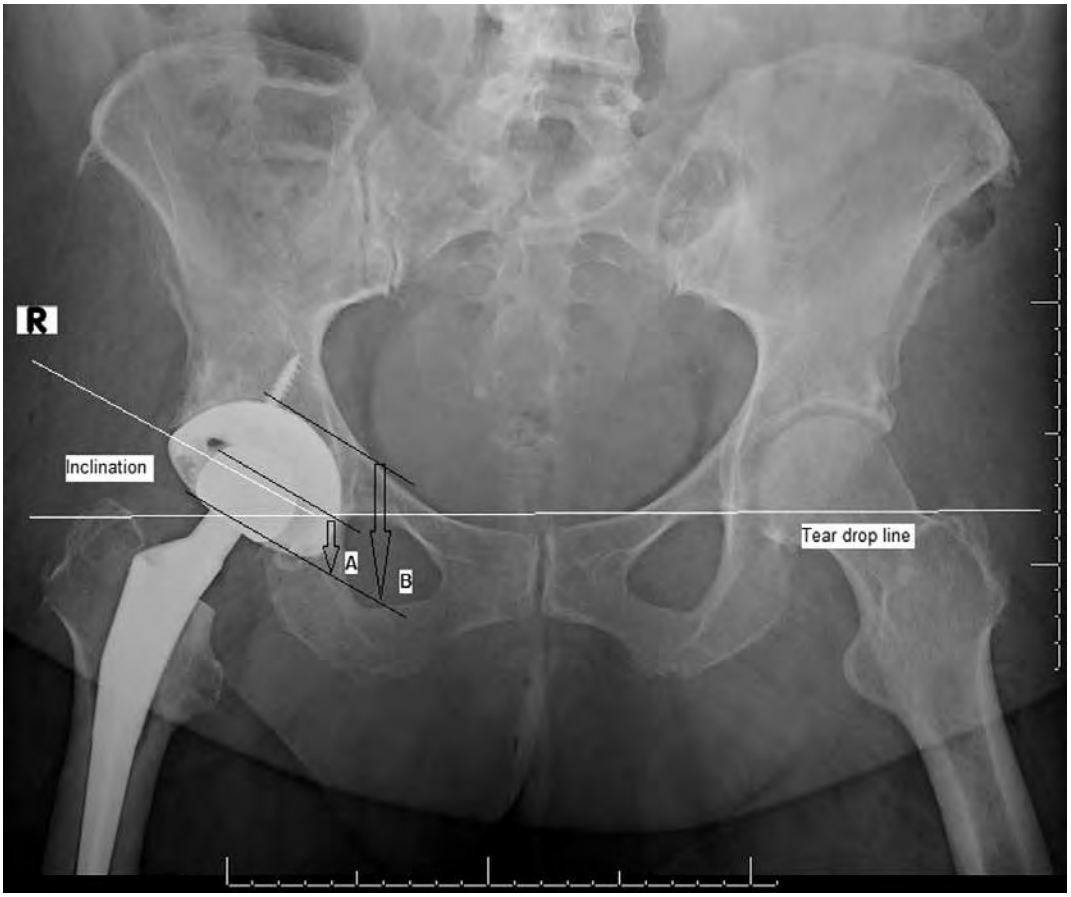

Figure 1. Technique for measuring inclination and anteversion. Inclination is the angle formed by a line parallel to the tear drop and a line running through the long axis of the ellipse. Anteversion is measured by dividing the width of the ellipse **(A)** by the total length of the cup **(B).**

Table 1. Comparison of Demographic Data and Radiographic Measurements for Anterior THA

	Fluoroscopy (n = 98)	Non Fluoroscopy (n = 101)	p-value
Age			0.017
Mean	69	66	
Range	40–91	45–86	
Std. Dev.	9.4	10.3	
BMI			0.005
Mean	28.1	25.8	
Range	18.8–43.2	20.1–42.8	
Std. Dev.	4.9	4.5	
Abduction			0.002
Mean	43.4	45.9	
Range	31–59.9	32.7–61.6	
Std. Dev.	4.9	6.3	
Anteversion			0.876
Mean	23.1	23.1	
Range	17–28	18–29	
Std. Dev.	4.9	2.24	

(494 hips) undergoing fluoroscopic-guided, primary anterior THA showed an average postoperative LLD of 3 ± 2 mm (range, 0–26 mm) and the author reported enhanced accuracy of leg lengths with the help of imaging.[19] Fifty-eight percent (n = 287) of the hips had LLD between 0–2 mm and all four of the patients with greater than 11 mm postoperative leg length inequality had preoperative LLD greater than 15 mm and hip dysplasia. To our knowledge, no studies comparing postoperative LLD between patients receiving anterior THA with and without fluoroscopy exist.

A serious concern is component positioning outside of safe zones. Malpositioning results in impingement and edge loading thereby accelerating polyethylene wear rates in metal-on-polyethylene THAs and increasing serum levels of metal ions after metal-on-metal THA.[7] The clinical implications of wear debris are numerous despite absence of long-term clinical data. Wear debris are thought to be involved in the development of pseudotumors, iliopsoas bursal cystic lesions, vascular compression, loosening, and fractures.[2, 18] Polyzois reviewed several animal studies and found adverse effects of metal ions released from joint arthroplasty materials on several organs including liver, kidney, heart, and nervous systems.[31] Necropsy results from animals showed hepatic necrosis, acute tubular necrosis, impaired bone remodeling after fracture, testicular toxicity, and retinal degeneration. However, the long-term effects in humans are still unclear.

Wear rates in THA are significantly greater with acetabular inclination angles greater than 45°. Little et al. prospectively followed 43 patients after uncemented THA for a

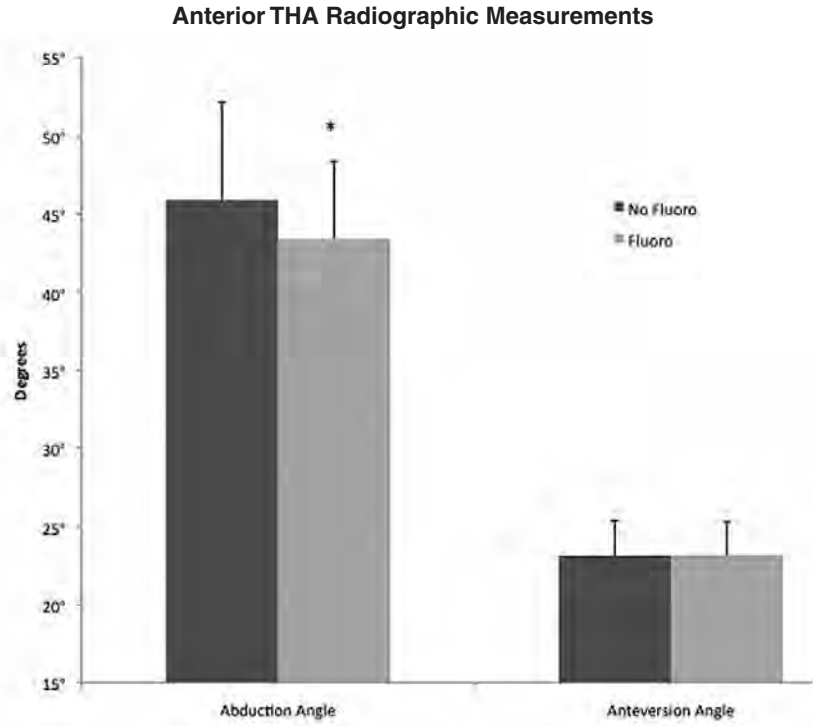

Figure 2. Mean acetabular cup abduction and anteversion angles with and without fluoroscopic guidance. Acetabular cup abduction angles are significantly different (*) between the two groups ($p < 0.05$).

mean of 64 months and found a significantly greater mean wear rate (50%) in those with less than 45° versus greater than 45° of inclination (0.12 mm/year vs. 0.18 mm/year, $p = 0.012$).[15] Volumetric wear in the group with more than 45° inclination was 44% greater and approached statistical significance ($p = 0.17$). Patil and colleagues computed contact stresses during a normal gait cycle using a finite element model and validated their results by comparison with findings from hip wear simulator studies in which acetabular cups were positioned at 45° or 55° of inclination.[28] Forces on the hip joint were scaled to represent loads produced by a 75 kg patient. The authors concluded greater abduction angles result in increased contact stresses and linear wear rates likely due to reduced contact area of the femoral head and increased anteversion decreases contact stresses and wear rates due to greater contact (17.2 vs. 21.7 mg/million cycles; $p < 0.01$). In the clinical arm of the study, 56 patients (60 THAs) were followed for up to five years and linear wear rates were measured on AP radiographs by a technique described by Livermore et al. Cups with greater than 45° inclination had 40% greater mean linear wear than those with less than 45° inclination ($p < 0.0001$).[16]

Acetabular components are involved in greater than 50% of THA revisions.[8] Dislocation after anterior approach to THA is a less likely cause of revision surgery due to its lower dislocation rates as compared to other approaches. Dislocation rates after anterior THA have been reported as low as 0.61% while rates after posterior THA vary from roughly 1% to 11%.[19] In contrast, bone loss remains a common reason for implant failure and results from osteolysis and stress shielding, creating technical difficulties in revision surgery. Schmalzried reviewed 93 patients (113 hips) with an average follow-up of 64 months and found pelvic osteolysis in 17% (19 hips).[33] Osteolysis was significantly associated with cup abduction in excess of 50° ($p < 0.0001$). Kennedy et al. compared a group of patients with mean acetabular inclination of 61.9° against a group with an average of 49.7° and a found significantly higher percentage of patients with pelvic osteolysis in the former group (24% vs. 13%).[12] They concluded that a more horizontal cup position reduces osteolysis.

The anterior approach to THA has seen advancements since its inception by Judet in 1947. Over the years, it has been modified to allow exposure of the femur and pelvis with less soft trauma by avoiding release of any of the surrounding muscles from their bony attachments. In recent years, the use of an image intensifier has become popular because it provides intraoperative assessment of cup and femoral stem positioning and potentially reduces error despite use of alignment rods and patient experience. Surgeon assessment of component positioning may be incorrect. Hassan et al. prospectively measured cup abduction and anteversion in 50 consecutive THAs intraoperatively via alignment guide and postoperatively on radiographs.[11] Goal cup placement was 30–50° of abduction and 5–25° of anteversion. Four surgeons recorded their intraoperative inclination and version values after the alignment guide was fitted into the component. Cup abduction was measured directly from x-rays and version was calculated according to the equation in their study. The authors concluded a 5° (range, 0–20°) and 9° (range, 0–24°) mean error of cup abduction and version, respectively.

Disadvantages include radiation exposure and risk of contamination from the intensifier. In a study by Schuler evaluating fluoroscopy of the hip, knee, and ankle, the hip produced the greatest amount of scatter secondary to tissue density. Reduction in radiation was most affected by reducing fluoroscopy time rather than standing at a distance or using laser targeting.[34] C-arm contamination of the surgical field during anterior THA is largely theoretical as there is a paucity of studies on this topic. Biswas et al. studied sterility of C-arm drapes after spine surgery cases and found that although some degree of contamination was found on all areas of the drape after surgery, the upper portions exhibited the greatest rates of contamination.[4] Another study investigating the timing of C-arm drape contamination found 50% contamination at 20 minutes and a large correlation with lateral position changes.[29]

There are several limitations to our study. First, this is a retrospective analysis in which a single rater was not blinded to the variable of fluoroscopy. Second, the non-fluoroscopic guided THAs were performed earlier in the surgeon's career

when he was less familiar with the anterior approach. The high percentage of acetabular components in safe zones may reflect the surgeon's experience and high case volume. Anterior THA is technically demanding and there is a substantial learning curve which may have accounted for the greater number of cup abduction angles and combined anteversion-abduction angles outside safe zones in the NF group. Since patients were randomly selected, the first several patients may have been included in our analysis at a time when complications are known to reflect the learning curve.[9, 30]

A third limitation is that measurement of cup anteversion is not always precise. In the method described by Widmer and Ing, the anteversion is measured on a plain radiograph of the pelvis centered on the tear drop.[35] Unfortunately, precise determination of component landmarks is obscured by implant materials and the quality of postoperative radiographs is not always optimal. Pelvic tilt may have also affected our measurements.

A fourth limitation of our study is the non-uniform demographics between the FL and NF groups. The two groups were significantly different in age and BMI and it is unclear whether these differences affected our results. A study by Paterno et al. found no correlation between age and BMI and dislocation rates after primary THA.[26] Another study found BMI great than 30, surgical approach (specifically, minimally-invasive surgery), and surgeon volume to be independent predictors of malpositioned cups.[5] However, this study did not specifically address the anterior approach nor did they discuss fluoroscopy. Matta reported accurate and reproducible component positioning using an anterior approach and fluoroscopy for THA in patients with an average age of 64 years regardless of body habitus.[19]

In summary, the anterior approach to THA may be performed with and without fluoroscopy. The advantages of fluoroscopy include intraoperative assessment of component positioning and, according to the results of our study, a significantly higher percentage of acetabular cup abduction angles in the safe zone. Our results also show a trend toward a larger proportion of components in combined abduction and anteversion safe zones when using image guidance. This may reduce wear and increase longevity of the arthroplasty. Our results also support accurate cup anteversion without imaging and this may avoid the drawbacks of fluoroscopy which include radiation exposure and possible field contamination. In the future, we would like to determine the differences in postoperative complications between our groups. Prospective studies investigating component positioning during anterior THA with and without fluoroscopy are still needed.

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Medical Student Research Project

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Incidence of Fracture Displacement and Hemorrhage from Stable Pelvic Injuries Treated Nonoperatively with Early Weight Bearing

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Abstract

Mechanical stability of pelvic ring injuries frequently determines the ability to mobilize a trauma patient safely and comfortably. The assessment of stability can be made on static imaging, dynamic imaging, and clinical examination. With dynamic imaging often difficult in acute trauma patients, we have instituted a pelvic fracture management protocol at our institution that involves repeat static radiographs after weightbearing of presumably stable injuries as well as serial hemoglobin checks to rule out pelvic instability and hemorrhage. We performed a retrospective analysis of our data over 18 months. Nearly half of the patients with presumably stable injuries did not have followup radiographs and were excluded, and one patient did not have followup hemoglobin checks. Of the remaining patients, 26 were evaluated which consisted of LC-1, APC-1, and isolated pubic ramus fractures. Radiographs were assessed for displacement using a previously described method, which demonstrated a maximal displacement of 5.7 mm. No patients required any surgical treatment or change in their nonoperative treatment course. Eight patients required packed red blood cell transfusions, although several of these patients also had other injuries. In conclusion, routine followup weight bearing radiographs may not be required in cases of stable fracture patterns, although serial hemoglobin checks might be helpful, particularly in patients with additional injuries.

Introduction

Most orthopaedic surgeons agree that decision making with pelvic ring injuries centers on an assessment of the stability of the injury. This includes both hemodynamic stability as well as mechanical stability. Although percutaneous treatment of the posterior and anterior pelvic ring has become more popular, there are clearly still a large group of patients who are treated nonoperatively because their injury is assessed to be mechanically stable. But how does a surgeon decide that a pelvic fracture will be stable? Predictions can be made based on static radiographic imaging, dynamic standing radiographs, or dynamic fluoroscopic stress images in the operating room. Mechanical instability of a fracture on a stress view, however, does not necessarily indicate that surgical repair is indicated. Loss of alignment on interval static views, however, is arguably more concerning and indicative of the need for improved mechanical stability (i.e., surgical intervention).

Another concern with pelvic fractures is the potential for hemorrhage and its sequelae. Although fractures with more severe, unstable fracture patterns are more likely to incur clinically significant blood loss requiring intervention, minimally displaced fractures are also reported to cause hemorrhage in certain circumstances, particularly related to noncompliant vasculature in the elderly. Therefore, most surgeons have a heightened clinical suspicion for hemorrhage in patients with pelvic fractures, often requiring admission to the hospital and hemoglobin checks.

At most institutions, pelvic fractures that are stable and without significant associated hemorrhage are typically treated nonoperatively. At our institution, a clinical protocol was established to ensure that, amongst other things, serial hemoglobin tests were checked over the first 24 hours and repeat pelvic radiographs were done after weightbearing on fractures deemed to be nonoperative. There are differing opinions regarding the degree of scrutiny required for these matters, particularly in lateral compression type 1 fractures. The aim of this study is to retrospectively evaluate the incidence of clinically significant hemorrhage and fracture displacement after immediate weightbearing of stable pelvic fractures to help determine the appropriate level of surveillance warranted for stable fracture patterns treated nonoperatively.

Methods

Our Level 1 trauma center instituted a pelvic fracture management protocol in October 2010. All patients with pelvic fractures from blunt and penetrating trauma with the exception of Tile type A avulsion fracture were admitted,

and had serial hemoglobin (every eight hours) checked for 24 hours. Fractures types were grouped into stable pelvic ring fractures, unstable pelvic ring fractures, and unstable sacral fractures. Details for proper initial evaluation and management of the pelvic fracture patient were outlined, the details of which are not given here. Ongoing management guidelines were also provided, which included repeat neurological examination as determined by initial findings, serial hemoglobin checks, serial lactate level assessment until normalized in multiple trauma patients and patients with known pelvic hemorrhage, and repeat radiographic examination after the patient is ambulatory to check for interval displacement. IRB approval was obtained for this particular study. Our orthopaedic database as well as the trauma surgery database (Pennsylvania Trauma Systems Foundation registry) were retrospectively reviewed for all patients treated nonoperatively for a pelvic fracture between October 2010 and April 2012. Inclusion criteria included all patients, 18 years or older, who presented within that timeframe with stable pelvic fractures, including lateral compression type 1, APC type 1, Tile type A, and isolated pubic ramus injuries. Any patient with unstable fracture patterns, including lateral compression types 2 and 3 or APC types 2 and 3, gunshot injuries, or pathologic fractures from tumors were excluded. Pelvic fracture patterns were classified according to the Tile classification system and Young and Burgess classification system.[1, 2] The initial search identified 55 patients. Of these 55 patients, 26 patients had AP pelvic radiographs available at both initial presentation and following advancement to weightbearing status. Serial hemoglobin levels were reviewed for the initial value upon admission and the value following 24 hours.

In order to evaluate fracture displacement using radiographic interpretation, a novel method developed by Soles et al. was applied to AP radiographs both at the time of presentation and following weightbearing.[3] Measurements taken on the initial radiographs were compared to measurements taken on radiographs following advancement to weightbearing status to determine interval fracture displacement.

On the AP radiograph, a plumb line drawn through the midline of the spine and sacrum served as a reference for perpendicular lines drawn from points of interest on either hemipelvis. These points of interest included iliac wing height, sacral height, ischial height, sacral width, and pelvic ring width (Fig. 1). The difference between left and right hemipelvis was recorded as a positive value and taken as a measurement of vertical displacement. These measurements were repeated for all AP radiographs. Interval displacement was determined by comparing values from time of admission to those following weightbearing.

Results

The final analysis included 26 patients admitted between the months of October 2010 and April 2012. The mean age at the time of injury was 55 years, with a range of 20–88 years.

All fractures consisted of stable patterns, including lateral compression type 1, APC type 1, Tile type A, and isolated ramus injuries according to both the Tile as well as the Young and Burgess classification systems.[1, 2] Of the 26 patients included in the study, nine (35%) had unilateral ramus fractures, two (8%) had bilateral rami fractures, five (19%) had intraarticular (acetabular) extension, one (4%) had a pubic symphysis diastasis, and eight (31%) had incomplete sacral fractures.

The mean interval displacements recorded on AP radiographs following advancement to weightbearing status were as follows: iliac wing height, 0.17 ± 2.01; sacral height, 0.02 ± 1.61; ischial height, 0.08 ± 1.40; sacral width, 0.89 ± 2.14; pelvic ring width 0.04 ± 2.67. Interval displacement results were calculated as absolute values. The greatest displacement was observed in pelvic right width, with three patients found to have 5.2, 5.5, and 5.7 mm of additional displacement on early radiographs. However, no patients were determined to require surgical intervention and all patients progressed to clinical union of their pelvic fractures.

Of the 26 patients included in this study, two were found to have pelvic hematomas and eight patients ultimately required transfusion of packed red blood cells. The mean interval change in hemoglobin levels over the first 24 hours of admission for the eight patients that required transfusion was 2.05 ± 2.99. The mean interval change in hemoglobin levels for the 17 patients that did not require a transfusion (one patient did not have follow-up hemoglobin levels available) was 1.68 ± 1.43. Of the two patients found to have pelvic hematomas, one patient had an admitting hemoglobin level of 6.6 and required two units of packed red blood cells. The second patient did not require transfusion.

Among the eight patients requiring transfusion, six (75%) also presented with a non-pelvic fracture, two (25%) presented with an abdominal hematoma, three (38%) presented with an abdominal organ laceration, and one (13%) presented with a pelvic hematoma. Among the 18 patients that did not require a transfusion, six (33%) presented with a non-pelvic fracture, two (11%) presented with an abdominal organ laceration, and one (6%) presented with a pelvic hematoma.

Discussion

Stable pelvic fracture patterns are commonly treated nonoperatively with early weightbearing. Soles et al.[3] reported on 118 patients with LC 1 pattern pelvic fractures that presented at a single Level I Trauma Center. These patients were treated nonoperatively with immediate mobilization and repeat radiographs to monitor for further displacement. Of the 118 patients, only one patient failed nonoperative management and presented radiographically with 5 mm of additional displacement, while the other 117 patients healed

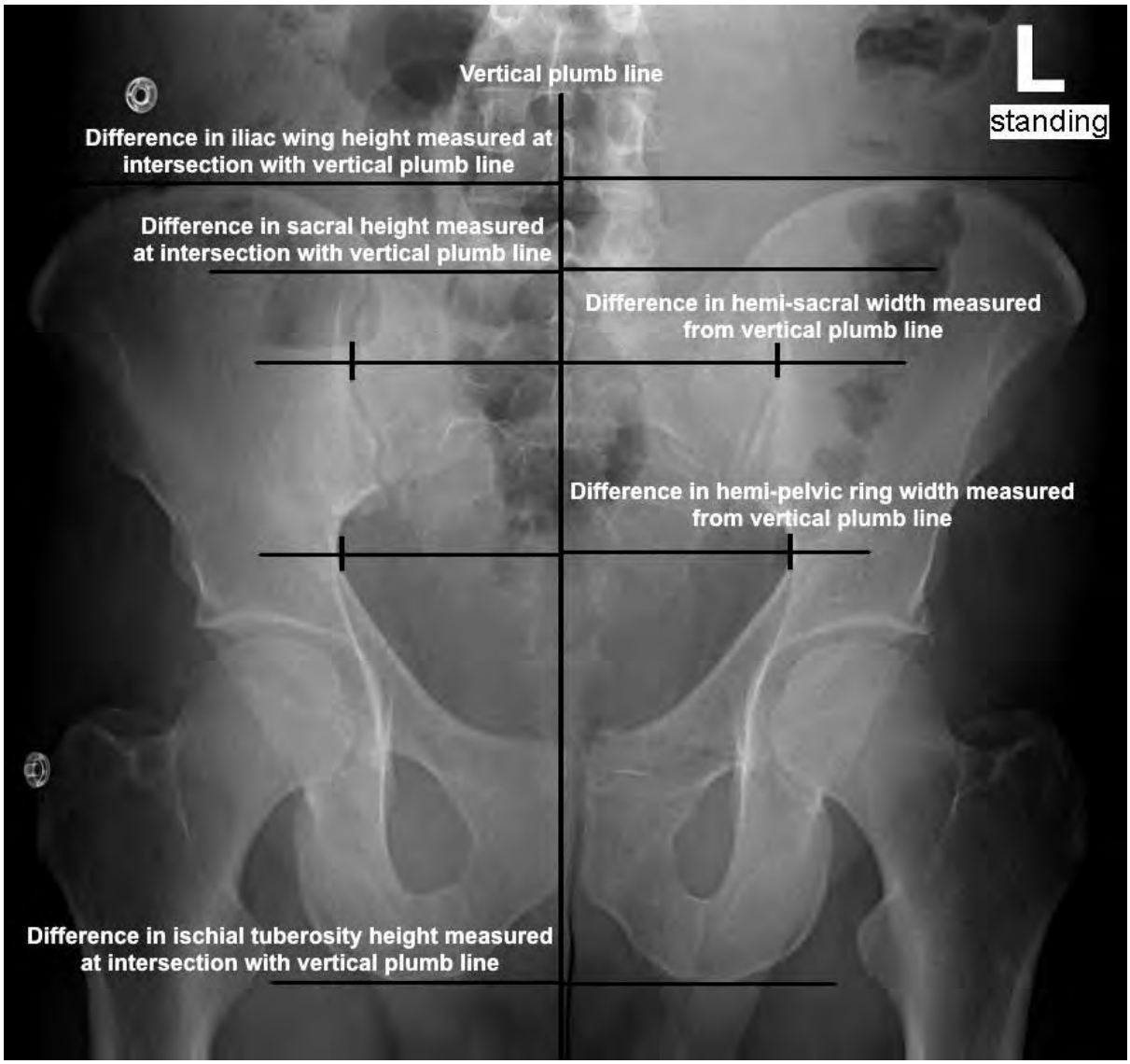

Figure 1. AP pelvis radiograph demonstrating measurement technique. A vertical plumb line was drawn through the midline of the spine and sacrum with perpendicular lines at the level of the iliac wing height, sacral height, ischial tuberosity height, sacral width, and pelvic ring width. The difference in height was recorded.

Table 1. Interval Fracture Displacement

Measurement of Interest	Interval Displacement (mm)
Iliac wing displacement	0.17 ± 2.01
Sacral displacement	0.02 ± 1.61
Ischial displacement	0.08 ± 1.40
Sacral width difference	0.89 ± 2.14
Pelvic ring width difference	0.04 ± 2.67

Table 2. Associated Injuries

Injury	Transfusion	No Transfusion
Non-pelvic fracture	75%	33%
Abdominal hematoma	25%	0%
Abdominal organ laceration	38%	11%

26 patients (eight transfusion, 18 no transfusion)

with minimal additional displacement. The investigators concluded that minimally displaced LC sacral fractures could be treated safely nonoperatively with immediate weightbearing. A similar study by Bruce et al.[4] consisted of 117 patients who presented to two Level I trauma centers with an LC pelvic fracture with less than 5 mm of initial displacement. The patients were treated nonoperatively with mobilization and monitored with serial radiographs. Of the 117 patients, 23 presented radiographically with more than 5 mm of additional displacement. However, none of these 23 patients included incomplete LC sacral fractures with ipsilateral rami fractures, and the investigators concluded that such fracture patterns could be treated nonoperatively. These studies support nonoperative management of minimally dis-

placed LC fractures, but there is still a question of what degree of scrutiny is required with early serial radiographs and hemoglobin checks.

In our study, the majority of our patients showed less than 5 mm additional displacement in all parameters on early radiographs. Only three patients were found to have greater than 5 mm additional displacement in a single parameter, but still went on to clinical union without requiring surgical intervention. Our results suggest that clinically significant displacement does not occur with nonoperative treatment and immediate weightbearing of these stable fracture patterns, as evidenced by early radiographs.

Catastrophic hemorrhage is a concern with pelvic fractures and serial hemoglobin checks are employed by our institution to monitor for such incidences. Two patients were found to have pelvic hematomas, and only one of these patients required transfusion. However, eight patients ultimately required transfusion, suggesting that the majority of hemorrhage seen in these patients is due to associated injuries rather than their pelvic fractures. This is further supported by the higher rates of associated injuries seen among these eight patients. This suggests that the overall clinical picture of the presenting patient taken into consideration with serial hemoglobin checks may provide the best predictive tool for incidences of hemorrhage.

One of the challenges in assessing interval fracture displacement was determining a methodology for radiographic interpretation. Lefaivre et al.[5] determined that a lack of standardization exists in the measurement of radiographic outcomes. In an effort to establish a level of consistency, we chose to model our measurements from a novel method developed by Soles et al.,[3] a technique similar to one used by Bruce et al.,[4] which utilizes a vertical plumb line for reference and multiple points of interest. We have detailed the radiographic measurement technique used in our study to present a reproducible method. However, we acknowledge the variability in radiographic quality and technique as well as the inevitable human error in attempting to standardize each measurement. As Lefaivre et al.[5] have discussed, further investigation in the area of radiographic interpretation is needed to establish a standardized method that is both reproducible and reliable.

Conclusion

In our retrospective analysis of 26 patients with stable pelvic injuries, we found minimal displacement following early weightbearing and a single incidence of pelvic hemorrhage that required transfusion of 2 units of packed red blood cells. This suggests that stable pelvic fracture patterns do not result in significant interval displacement or catastrophic hemorrhage.

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Medical Student Research Project

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The Role of Pharmacological Agents in the Management of Post and Chronic Concussion Syndrome: A Systematic Literature Review

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Introduction

Concussion, or minor traumatic brain injury (mTBI), is a common neurologic problem that has received increasing attention in recent years. The Center for Disease Control and Prevention (CDC) has estimated that 1.7 million patients present each year to the emergency departments with traumatic brain injury (TBI).[1] About 75% of TBIs that occur each year are concussions or other forms of mild traumatic brain injury (mTBI).[1]

The terms concussion and mTBI are used interchangeably and is the most common form of TBI.[2] mTBI is defined by the World Health Organization (WHO) as:

• One or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery.

• Glasgow Coma Score (GCS) of 13–15, 30 minutes post-injury or later upon presentation for healthcare.[3]

Previous review articles discussed pharmacological agents in treating traumatic brain injuries, but the results were either inconclusive or not specific to mild traumatic brain injuries. Comper et al.[3] performed a systematic review for treatments, but they did not find any significant evidence on the efficacy of drug interventions for treating the symptoms of mTBIs. Other review articles covered more severe forms of TBI, but did not report findings specific to mild traumatic brain injuries.[4–6] Therefore, this systematic literature review aims to investigate the use of drug therapies in treating mild traumatic brain injuries.

Methods

Search Strategy

Relevant studies were identified using PubMed. The search strategy included terms related to concussions, mild traumatic brain injuries, and pharmaceuticals. Variations of these terms were included in the search as both free text and Mesh terms. The complete search strategy was as follows:

PubMed
#1 Concussion* or mild traumatic brain injury or mild TBI or mTBI or "Brain Concussion" [Mesh] (8,465 results)
#2 Traumatic Brain Injury or "Brain Injuries" [Mesh] (61,893 results)
#3 Drug Therapy or "Drug Therapy" [Mesh] or pharmacological agent* or pharmaceutical agent* (2,196,844 results)
(#1 or #2) and #3 (6,452 results)

The search was last performed on 6/22/2012.

Reference lists of identified articles were also checked for relevant publications in order to identify additional articles that were not found by the search strategy. There were no restrictions on the year of publication; however, the search was limited to articles in English.

Selection of Studies and Data Extraction

Articles were screened and selected if they included studies that treated concussed patients or patients with mTBI using pharmaceutical agents. The articles were screened and reviewed based on the following inclusion criteria: (1) study must include patients with a concussion or mTBI; (2) patients being studied must have a GCS ≥13.

Study design methodology information was extracted, such as the study design type, study size, pre-treatment GCS, symptom of concussion, study drug, follow-up time, and study results. The study's population demographics were also collected, such as cohort size, mean age, and the number of mTBI patients in the study.

Results

The search strategy identified 6,581 citations with an additional nine studies identified from the hand search of review articles. From the 6,590 citations, the full text of 338 articles was retrieved for further review. Of the 338 articles, 23 studies met the inclusion criteria to be reported in the review. The flow diagram is shown in Figure 1.

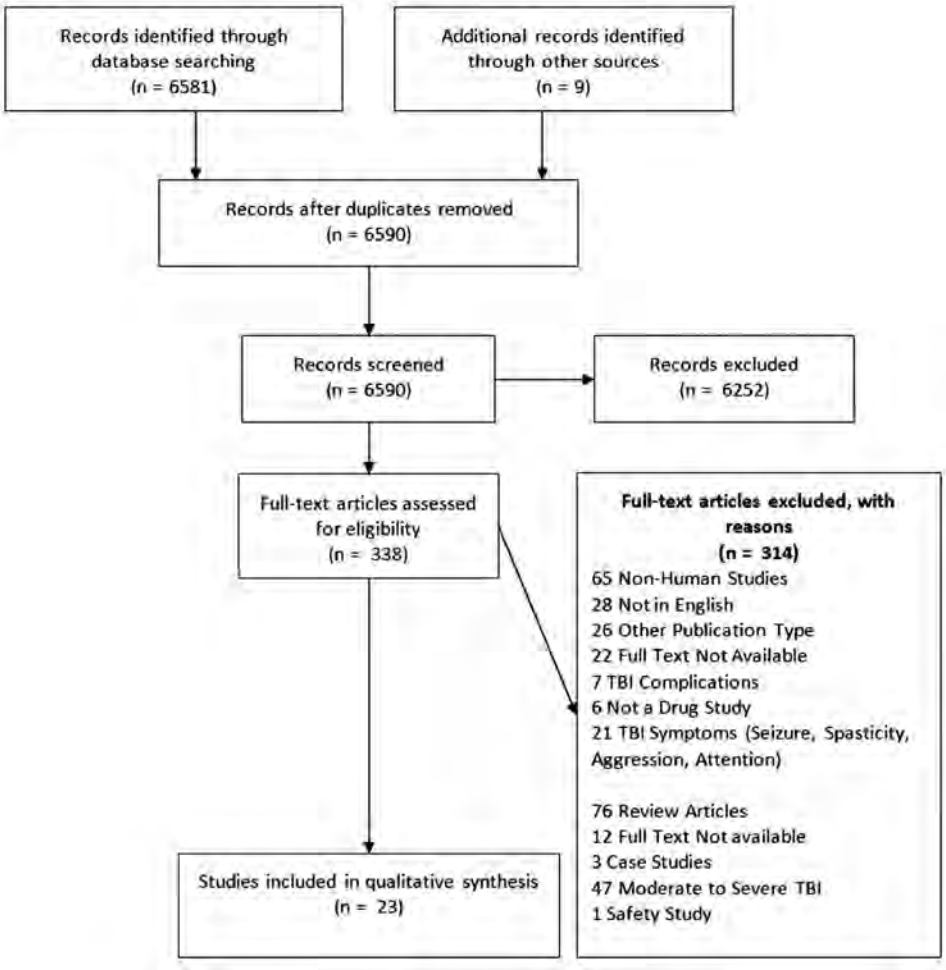

Figure 1. Flow Chart for Identifying Studies

Attention, Cognition, and Memory

Of the identified articles, 14 articles described pharmaceutical treatments with outcomes of attention, cognition, and memory.[7–20] The population characteristics of the studies are described in Table 1. The sample sizes in these studies were small, ranging from 10 to 182 patients. The number of patients with mild TBI is also shown in Table 1, with most studies having only a fraction of their total patients with mTBI. The mean ages of patients from these studies ranged from 11.9 to 51.7, indicating that most of the patients were young. The pre-treatment GCS was not available for all studies, since some studies used different methods to distinguish between mild to moderate TBIs.[11, 13, 17, 20] The length of time from injury to treatment initiation varied from hours to months, with most studies initiating the intervention months after the initial traumatic insult.

Table 2 describes the study design, study drug, the outcome measure, and the results. Eight studies utilized a randomized, double-blind, placebo-controlled trial, while the other six studies used other study designs. The outcomes used to measure the effects of the drug interventions were heterogenous between the studies. The Controlled Oral Word Association Test (COWAT) and the Paced Auditory Serial Addition Test (PASAT) were used in several studies;[11, 12, 15, 16, 20] however, many studies used different outcomes to measure the patient's attention, memory, and cognition.

Dopaminergic Agents

Methylphenidate was investigated in five studies for its effects on attention and cognition in patients with brain injury. Plenger PM et al.[7] showed that there were improvements in attention ($p < 0.03$) and motor performance ($p < 0.05$) when compared to placebo. However, only four patients out of 23 had a mild TBI. Mahalick DM et al.[8] studied children and showed that performance on all tasks of attention and concentration was statistically significant when compared to placebo ($p < 0.04$ to $p < 0.005$), but only two patients in this study had a mTBI. Kaelin DL et al.[9] found a significant improvement in attention with patients using methyl-

Table 1. Population Characteristics of Studies with Outcomes in Attention, Cognition and Memory

Author	Sample Size/ mTBI Sample Size	Mean Age of Population	Pre-treatment GCS	Time Since Injury to Study Treatment
Dopaminergic Agents				
Plenger PM et al., 1996[7]	23/4	Acute phase placebo 26.6 (8.7), acute phase methylphenidate 31.4 (17), 30-day follow-up placebo 22.2 (5.2), 30-day follow-up methylphenidate 28.6 (13.9), 90-day follow-up placebo 21.5 (6.5), 90-day follow-up methylphenidate 25.8 (13.4)	6–15	Enrolled from hospital
Mahalick DM et al., 1998[8]	14/2	10.67	3–15	14.14 Months (mean)
Kaelin DL et al., 1996[9]	10/2	51.7	3–13	19.8 Days (mean)
Whyte J et al., 1997[10]	19/1	30.8	3–14	514.1 Days (mean)
Kraus MF et al., 2005[11]	22/6	TBI patients 36.0 (11.8 (SD)), TBI patients undergoing PET 33.2	GCS not reported (mild, moderate or severe closed head TBI)	63.2 (6–240) Months (mean, range)
Serotonergic Agents				
Fann JR et al., 2001[12]	15/15	41.9 (SD 8.5)	13–15	10.6 Months (mean)
Dopaminergic and Serotogenic Agents				
Lee H et al., 2005[13]	30/Not reported	Methylphenidate group 35.3 (SD 8.0), sertraline group 33.6 (SD 12.3), placebo group 35.5 (SD 7.2)	GCS not Reported (mild to moderate TBI)	Methylphenidate: 34.8 days (mean) Sertraline: 31.9 days (mean) Placebo: 30.0 days (mean)
NMDA Antagonists				
Merchant RE et al., 1999[14]	45/19	14–75 (range)	9–14	<12 Hours
Phospholipid Intermediates				
Levin HS, 1991[15]	14/14	CDP-choline median 25, placebo median 20	15	Recruited bedside once out of post-traumatic amnesia
Cholinergic Agents				
Zhang L et al., 2004[16]	18/Not reported	Group A 33 ± 2 Group B 31 ± 2	Group A: 9.3±1.1 (mean ± SD) Group B: 8.9±1.0 (mean ± SD) 3–15	Group A: 4.6 ± 0.7 months (mean ± SD) Group B: 3.9 ± 0.5 months (mean ± SD)
Kaye NS et al., 2003[17]	10/6	41	GCS not reported (mild to severe TBI)	1.2 Years (mean)
Tenovuo O et al., 2005[18]	111/64	All patients 40 ± 1.3	13–15	71 ± 6.7 Months
Peptide Treatments				
Alvarez XA et al., 2003[19]	20/3	30.1 ± 2.15	3–15	23–1107 Days (range)
Filipova M et al., 1989[20]	17/17	DDAVP mean 41, placebo mean 28	Minor head injury patients	Within 16–40 hours

phenidate, when compared to a natural recovery of rehabilitation. However, due to the small sample size of 10 patients, the study was unable to find statistical significance in many of the tests. Additionally, only two patients included in this study had a mTBI. In the study by Whyte J et al.,[10] methylphenidate had statistically significant differences in the variables of speed and mental processing ($p < 0.001$), but did not have statistically significance in the overall effect over all the variables ($p = 0.46$). In this study, there was only one patient with a mild TBI. Lee H et al.[13] found that methylphenidate improved cognitive function in nine out of 10 cognitive tests ($p < 0.05$ to $p < 0.001$). Methylphenidate was also found to improve daytime sleepiness ($p < 0.01$). In this study, it was unclear how many patients with mild TBI were included, but the patients in the study ranged from mild to moderate TBI.

The use of amantadine was investigated for its effects on attention, memory and behavior. In an open-label trial, Kraus MF et al.[11] found that there was improvement in executive function ($p = 0.02$) with amantadine. However, no significant improvements were found in attention ($p = 0.10$) or memory ($p = 0.44$). Furthermore, only six out of 22 study patients had a mild TBI.

Serotonergic Agents

Sertraline was investigated for its use in cognition and memory. Fann JR et al.[12] found improvements in cognitive function such as cognitive efficiency, psychomotor speed, and flexible thinking ($p < 0.01$ to $p < 0.05$). They also found improvement in recent memory ($p < 0.001$ to $p < 0.002$).

Table 2. Attention, Cognition, and Memory Outcomes of Identified Studies

Author	Study Design	Drug	Measure	Main Result
Dopaminergic Agents				
Plenger PM, et al., 1996[7]	Randomized, double-blind, placebo-controlled study	Methylphenidate	Disability Rating Scale (DRS) and tests of attention, memory, and vigilance	The methylphenidate group was significantly better at 30 days on the DRS ($p < 0.02$), and on tests of attention ($p < 0.03$) and motor performance ($p < 0.05$). No significant differences were noted between groups at 90 days.
Mahalick DM, et al., 1998[8]	Randomized, double-blind, placebo-controlled, cross-over experimental design	Methylphenidate	Measures of attention and concentration: The Gordon Diagnostic System (Model III); the Woodcock-Johnson Psychoeducational Test Battery-Revised	Performance on all tasks of attention and concentration was statistically significant when compared to placebo ($p < 0.04$ to $p < 0.005$) for all analyses.
Kaelin DL, et al., 1996[9]	Prospective multiple baseline design (A-A-B-A) utilized on a consecutive sample of patients.	Methylphenidate	Neuropsychological battery of tests to assess attention: Digit Span Sub-Test of the Wechsler Adult Intelligent Test — Revised; the Mental Control Sub-Test of the Wechsler Memory Test-Revised; Trail Making Parts A and B; the Symbol Search Sub-Test (Part A) of the WISC-III; the Mesulam Verbal Cancellation Test	Use of methylphenidate in acutely brain-injured adults was well tolerated and demonstrated a significant improvement in attention compared to natural recovery in a rehabilitation setting. Methylphenidate also correlated with faster functional recovery as measured by the Disability Rating Scale although the improvement did not achieve statistical significance. Total digit span score, mental control, and symbol search was improved significantly ($p < 0.05$). There were not enough data points to perform a statistical analysis for the other tests.
Whyte J, et al., 1997[10]	Randomized, double-blind, placebo-controlled, repeated crossover design	Methylphendiate	Measures of attention: Sustained arousal task, phasic arousal task, distraction task, choice reaction-time task, behavioral inattention task	Methylphenidate had no significant overall effect across the 22 performance variables ($p = 0.46$). Methylphenidate did have different effects on individual performance variables ($p < 0.001$), specifically to variables of speed and mental processing. Methylphenidate appeared to affect the arousal and speed of mental processing. Methylphenidate appears to increase mental processing speed.
Kraus MF, et al., 2005[11]	Open-label trial	Amantadine	Executive domain: Trail Making Test Part B, Controlled Oral Word Association Test (COWAT) Attention domain: Trail Making Test Part A, Digit Span (from WAIS-R) Memory domain: California Verbal Learning Test (CVLT), Rey Osterreith Complex Figure-immediate and delayed recall	Significant improvements on tests of executive function were observed with treatment ($p = 0.02$). No significant improvements found in attention ($p = 0.10$) or memory ($p = 0.44$).
Serotonergic Agents				
Fann JR, et al., 2001[12]	Non-randomized, single-blind, placebo run-in trial	Sertraline	Depression: HAM-D Neurological tests (cognition and attentional): Digit Span; Digit symbol; Vocabulary; Finger Tapping Test; Trailmaking Test, Parts A and B; Controlled Oral Word Association Test (COWAT); Logical Memory I and II; Visual Reproduction I and II; Buschke Selective Reminding Test (SRT); Benton Visual Retention Test (BVRT); Self-Perception of TBI Severity	Depression scores changed significantly with sertraline, from the baseline mean SD HAM-D score of 25.0 ± 4.36 to a mean of 7.2 ± 5.30 at Week 8 ($P < 0.001$). Vocabulary was unchanged compared to baseline. Simple auditory attention was not significantly different. Digit symbol scores changed significantly ($p < 0.01$), indicating improvement in general cognitive efficiency. Psychomotor efficiency and attention improved on Trailmaking Test Part A ($p < 0.05$). Speed and flexible thinking skills were improved on Trailmaking Test Part B ($p < 0.04$). Recent memory ability was improved as seen in the verbal recent memory tests, as well as the WMS-R Logical Memory Tests ($p < 0.001$ to $p < 0.002$). There were significant improvements in cognitive function, especially in psychomotor speed, cognitive efficiency, flexible thinking, and recent memory ability. Vocabulary unchanged, some improvement in psychomotor speed, improvements in memory ability.

(continued on next page)

Table 2. Attention, Cognition, and Memory Outcomes of Identified Studies *(Continued)*

Author	Study Design	Drug	Measure	Main Result
Dopaminergic and Serotogenic Agents				
Lee H et al., 2005[13]	Randomized, prospective, double-blind, placebo-controlled, comparative drug trial	Methylphenidate, sertraline	Depression: Hamilton Rating Scale for Depression (HAM-D); Beck Depression Inventory (BDI) Rivermead Postconcussion Symptoms Questionnaire (RPQ) for postconcussional symptoms; SmithKline Beecham Quality of Life Scale (SBQoL) for quality of life Seven performance tests: Critical Flicker Fusion Threshold (CFFT), Choice Reaction Time (CRT), Continuous Tracking Task (CTT), Mental Arithmetic Test (MAT), Sternberg Memory Scanning Task (STM), Digit Symbol Substitution Test (DSST) and Mini-Mental State Examination (MMSE) Subjective measures of sleep: Leeds Sleep Evaluation Questionnaire (LSEQ) and daytime sleepiness (Chonnam Epworth Sleepiness Scale (CESS))	Both methylphenidate and sertraline improved depressive symptoms compared to placebo. However, methylphenidate improves cognitive function and maintains daytime alertness. Methylphenidate and sertraline were significantly superior to placebo, as measured by the HAM-D ($p = 0.005$), no significant differences were found by the BDI. Postconcussional symptoms (as measured by the RPQ) were significantly improved in the methylphenidate ($p < 0.001$) and placebo ($p < 0.05$) groups compared to baseline. Postconcussional symptoms for sertraline were not significantly improved. Methylphenidate significantly improved cognitive function in nine out of 10 cognitive tests ($p < 0.05$ to $p < 0.001$) compared to baseline; placebo significantly improved cognitive function in seven out of 10 cognitive tests ($p < 0.05$ to $p < 0.001$); sertraline significantly improved cognitive function in only two out of 10 cognitive tests ($p < 0.05$ to $p < 0.01$). Methylphenidate ($p < 0.01$) and placebo ($p < 0.05$) both significantly reduced daytime sleepiness as measured by the CESS, while sertraline did not. Quality of life was improved for all three drug treatments when compared to baseline (methylphenidate ($p < 0.01$), sertraline ($p < 0.05$), placebo ($p < 0.05$)).
NMDA Antagonists				
Merchant RE et al., 1999[14]	Randomized, double-blind, placebo-controlled study	CP-101,606 (Traxoprodil)	10-item Neurobehavioral Rating Scale; Kurtzke Neurologic Status Evaluation; Galveston Orientation and Amnesia Test (GOAT); National Institutes of Health (NIH) Stroke Scale; a battery of nine neuropsychological tests	CP-101,606 had no psychotropic effects and was well-tolerated in patients who had sustained either a mild or moderate TBI or an atraumatic hemorrhagic stroke. The GCS score showed no statistical significance in the speed of recovery between cohorts. Neurobehavioral rating scale showed improvement in all groups but was not statistically significant between groups. The Kurtzke Neurologic Status Evaluation was not statistically different between groups. No statistical difference was seen with the GOAT, NIH stroke scale, and the battery of nine neuropsychological tests.
Phospholipid Intermediates				
Levin HS, 1999[15]	Randomized, double-blind, placebo-controlled study	Cytidine diphosphoryl choline (CDP-choline)	Memory: verbal recall, spatial memory, recognition memory test Fluency: verbal and design tests Attention: Continuous Performance Test, Paced Auditory Serial Addition Test (PASAT)	Results showed that CDP-Choline produced a greater reduction in post-concussional symptoms than placebo ($p < 0.005$). Analysis of the neuropsychological findings revealed a significantly greater improvement in recognition memory for the CDP-Choline group ($p < 0.02$) whereas the other changes in test performance did not differ between the two groups.

Table 2. Attention, Cognition, and Memory Outcomes of Identified Studies *(Continued)*

Author	Study Design	Drug	Measure	Main Result
Cholinergic Agents				
Zhang L, et al., 2004[16]	Randomized, placebo-controlled, double-blind crossover trial	Donepezil	Memory: Auditory Immediate Index [AII], Visual Immediate Index [VII] from the Wechsler Memory Scale–III Cognition: Paced Auditory Serial Addition Test (PASAT) (measures sustained attention, working memory, and information processing speed)	At week 10, group A taking donepezil had statistically significant benefits to memory when compared to group B taking placebo (AII score $p = 0.002$, VII score $p < 0.001$). After cross-over, scores at week 24 were not statistically significant, indicating that donepezil's effects were carried over. At week 10, the cognition scores were also statistically significant for group A taking donepezil compared to group B taking placebo ($p \leq 0.001$ for all PASAT scores). After cross-over, scores at week 24 were not statistically significant, indicating that donepezil's effects were carried over. Donepezil increased neuropsychologic testing scores in short-term memory and sustained attention in postacute TBI patients.
Kaye NS, et al., 2003[17]	Open-label trial	Donepezil	Clinical Global Improvement (CGI) ratings; symptom focused neuropsychological test battery Global memory scale (GCS); Memory Assessment Scale (MCS)	Overall impression of improved focus, attention, clarity, and thought while on medication. CGI showed improvement. Global memory scale (GCS) and Memory Assessment Scale (MCS) did not improve.
Tenovuo O, et al., 2005[18]	Randomized trial	Donepezil, galantamine and rivastigmine	Subjective description of drug effect	Higher vigilance, better attention and raised general functioning seem to be the most constant and expected effects.
Peptide Treatments				
Alvarez XA, et al., 2003[19]	Open exploratory clinical trial, without a control group	Cerebrolysin	Syndrome Kurztest test (SKT, cognitive improvement test) Glasgow Outcome Scale (GOS)	Significant improvement in cognitive performance was seen, only evident during the first year of brain trauma. A significant improvement in SKT performance was observed after treatment with Cerebrolysin ($p < 0.01$). A significant improvement in GOS scores was observed ($p < 0.05$).
Filipova M, et al., 1989[20]	Randomized, double-blind, placebo-controlled	1-desamino-8-D-arginine-vasopressin (DDAVP)	Mika's Tactile Memory Test; Dichotic Listening; Postcard Recognition Test; Story Memory; Rhythm Pursuing; the Paced Auditory Serial Addition Test (PASAT)	The first and second series of PASAT (an information processing test) was statistically significant ($p < 0.05$ and $p < 0.01$ respectively) compared to placebo. The Story Memory (a test of verbal logical memory) DDAVP was significantly ($p < 0.05$) superior to placebo.

However, Lee H et al.[13] found that sertraline was not as effective as methylphenidate in treating postconcussional symptoms and cognitive function.

NMDA Antagonists

The use of Traxoprodil (CP-101,606) was investigated by Merchant RE et al.[14] in patients with mild to moderate traumatic brain injury. There were no statistically significant differences in the speed of recovery and neurobehavior when compared to placebo.

Phospholipid Intermediates

Levin HS[15] investigated the use of Cytidine diphosphoryl choline (CDP-choline) in 14 patients with mild TBI. Their results showed that there was a greater reduction in postconcussional symptoms when compared to placebo ($p < 0.005$). They also found that improvement in recognition memory ($p < 0.02$). They found no other statistically significant differences in the other tests evaluated in the study.

Cholinergic Agents

Zhang et al.[16] investigated the use of donepezil in patients from mild to severe TBI. This study found that the patients taking donepezil had statistically significant improvements in memory ($p < 0.001$) when compared to placebo after 10 weeks, with effects that carried over even during the cross-over period. Additionally, cognition scores were also statistically significant for patients taking donepezil ($p \leq 0.001$) when compared to placebo, as measured by the Paced Auditory Serial Addition Test (PASAT). Similar to the memory

analysis, the effects of cognitive improvements were carried over during the cross-over period. However, only 18 patients were included in this study, and it was unclear how many of those patients had a milder TBI.

In an open-label trial, Kaye NS et al.[17] investigated the use of donepezil in 10 patients, six of whom had mild TBI. They reported that there was an impression of improved focus, attention, clarity, and thought while on donepezil. The Clinical Global Improvement Ratings in this study showed an improvement. However, there was no statistical analysis to indicate any statistical significance.

Tenovuo et al.[18] also investigated the use of donepezil, along with galantamine and rivastigmine. The outcome used in this trial was the personal subjective experience with one of the study drugs. There was an apparent higher vigilance, attention, and increased functioning of the patients.

Peptide Treatments

Alvarez XA et al.[19] investigated the use of cerebrolysin in 20 patients, three of whom had mild TBI. A significant improvement in cognitive performance was seen by the Syndrome Kurztest Test ($p < 0.01$). Filipova M et al.[20] assessed the effects of 1-desamino-8-D-arginine-vasopressin (DDAVP) on memory in patients with mild TBI. The Paced Auditory Serial Addition Test (PASAT) was found to be statistically significant in both the first and second series of tests ($p < 0.05$ and $p < 0.01$ respectively) compared to placebo. The Story Memory Test was also found to be statistically significant ($p < 0.05$) when compared to placebo.

Depression

From the identified articles, six studies were found that studied the effects of a pharmaceutical agent to treat depression after a TBI.[12, 13, 21–24] The pharmaceutical agents that were investigated to treat depression were dopaminergic or serotonergic agents. All six studies used the Hamilton Rating Scale for Depression (HAM-D) as a measure for depression.

Lee H et al.[13] found both methylphenidate and sertraline improves the HAM-D score ($p = 0.005$) of depressed patients, when compared to placebo. In two articles, Fann JR et al.,[12, 21] described the treatment of sertraline in improving the HAM-D scores ($p < 0.001$) in patients with mild TBIs when compared to baseline scores. However, Ashman TA et al.[22] found no statistically significant improvements in HAM-D scores when comparing sertraline to placebo.

Dinan TG et al.[23] compared the use of amitriptyline in functionally depressed patients to patients who developed symptoms of depression following a mild TBI. It was found that the patients that were functionally depressed responded better to amitriptyline when compared to patients with depression following mTBI (HAM-D, $p < 0.01$).

The use of milnacipran was investigated by Kanetani et al.[24] in 10 patients, seven of whom had mild TBI. In this open label study, it was found that milnacipran statistically significantly improved HAM-D scores when compared to baseline at two weeks ($p = 0.0044$), four weeks ($p = 0.005$), and six weeks ($p = 0.0002$), when compared to baseline.

Amnesia

Two articles were identified that investigated the use of pharmaceutical agents and its effects on post-concussive amnesia symptoms. The use of rosuvastatin was investigated by Tapia-Perez et al.,[25] in which it was reported that there was a reduction in amnesia time with a hazard ratio of 53.76 (95% confidence interval (CI), 1.58–1824.64) when compared to placebo. However, it was unclear how many of the 21 patients had an initial GCS of at least 13. In a retrospective medical record review, Mysiw JW et al.[26] found that neuroleptic use during the acute stage of recovery can increase post-traumatic amnesia by almost seven days ($p = 0.00$).

Fatigue

The use of modafinil to treat post-concussive fatigue was investigated by Jha A et al.[27] in 51 patients, with 13 patients who had mild TBI. It was found that there was no significant difference in treatment with modafinil when compared to placebo over a 10-week period.

Corticosteroids

The death and disability from the use of methylprednisolone in treating patients with TBI was investigated in the Corticosteroid Randomisation After Significant Head Injury (CRASH) Trials.[28, 29] Overall, the study reported a higher relative risk of death within two weeks when treated with corticosteroids, when compared to placebo (RR = 1.18; 95% CI 1.09 to 1.27; $p = 0.0001$).[28] When the endpoint was extended to death after six months of treatment, the relative risk of death was still higher when treated with corticosteroids compared to placebo (RR = 1.15; 95% CI 1.07–1.24; $p = 0.0001$).[29]

In the CRASH trial, 10,008 patients were enrolled, where 3,002 patients reported with a GCS of 13 or above. In this mTBI group, the relative risk of death within two weeks was not statistically significant when compared to placebo (RR = 1.032; 95% CI 0.7322 to 1.4546; $p = 0.8573$).[28] Additionally, the relative risk after six months of treatment was not statistically different when compared to placebo (RR = 1.0808; 95% CI 0.8109 to 1.4405; $p = 0.5961$).[29]

Discussion

The number of studies which investigated the use of drug therapies in patients with mild traumatic brain injuries was limited. Studies which included patients with mild TBI were usually only a fraction of the total sample size, and there usually was no separate analysis for this subgroup population. Most studies investigated the use of drugs in patients with more severe TBI patients. However, this present systematic review shows that there may be promising results if more studies were to be conducted on patients with milder traumatic brain injuries.

The use of methylphenidate should be investigated further in mild traumatic brain injuries. The current available studies show evidence of improvement in patients with mild to severe traumatic brain injuries, but it is unclear if there would be any benefits for patients with milder forms of traumatic brain injury.[7–10, 13] Amantadine was also found to improve cognitive function, but did not show any difference in memory or attention. However, the study's sample size was small and a larger study investigating the use of amantadine in mTBI patients would help confirm these findings.[11]

The use of sertraline in patients with mild traumatic brain injury was found to have improvements in cognition, memory, and depression in the study by Fann et al.[12] However, Lee H et al.[13] found that the cognitive improvements of sertraline were not any better compared to placebo. A double-blind, randomized, placebo-controlled trial on the effects of sertraline on patients with mild TBI would help elucidate these findings.

The NMDA antagonist (Traxoprodil) was found to have no difference in effects on neurobehavioral outcomes or the speed of recovery.[14]

Levin HS et al.[15] investigated the use of CDP-choline in patients with mild traumatic brain injury in which he found greater reductions in post-concussional symptoms and improvements in memory. However, the number of patients in this study was small, and the author suggested a larger follow-up study should be performed. Such a study has yet to be carried out.

Donepezil was found to be beneficial in patients with mild to severe TBI.[16–18] However, these studies did not perform a separate analysis for patients with mild TBI. Therefore, a focused study investigating the use of donepezil in mild TBI patients would help confirm the benefits in attention, cognition, and memory.

Peptide treatments were found to be beneficial in cognition and memory.[19, 20] However, these studies were small and larger studies should be undertaken to confirm the results of these studies in patients with mild TBI.

Several studies investigated the use of pharmacological agents to treat a few symptoms following a mTBI. For treating the symptom of depression, serotonergic agents (sertraline, amitryptylin, milnacipran) and a dopaminergic agent (methylphenidate) were shown to be effective in treating post-concussive depression when measured by HAM-D scores.[12, 13, 21–24] Treating post-traumatic of amnesia with rosuvastatin was shown to reduce the length of the symptom; however, this study had a small sample size with only a fraction of the patients with mTBI.[25] A larger study should be performed with mTBI patients to confirm the effects of rosuvastatin. Modafinil was investigated in treating post-traumatic fatigue and was found to have no statically significant difference when compared to placebo.[27]

The use of corticosteroids in patients was recommended to not be used according to the CRASH trial results.[28, 29] However, when analyzing the mild TBI subgroup separately, there was no statistically significant difference in deaths in the use of corticosteroids at the two-week and six-month endpoints. A careful risk-benefit assessment should be made in advising the use of corticosteroids in patients with mild TBI.

The results of this systematic literature review have several limitations. Although the search was comprehensive, there may be articles that were not included in this search. The absence of these articles could lead to a bias in one drug intervention over another. Additionally, the studies that were included had heterogeneous outcome measures, causing the studies to be less comparable.

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Medical Student Research Project

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Predicators of Complications Associated with External Fixators

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Introduction

External fixation is a surgical procedure used to treat a number of different bone pathologies. The method entails percutaneous insertion of pins or wires into a bone, and subsequent attachment to an external fame, in order to stabilize the bones to allow for healing. The concept of external fixation was first utilized by Hippocrates, although the technique was not popularized until the twentieth century.[1, 2] Numerous developments since then, including notable designs by Swissman Raoul Hoffmann and Russian Gavril Ilizarov, have led to the external fixator designs used today. Such designs include monolateral external fixators, ring external fixators, and hybrid fixators (which combine technology of both monolateral and ring designs.) External fixators are quite versatile in their use. They can be used to reduce open and closed fractures, arthodesis, limb lengthening, and repair of bone deformities. External fixation can be utilized alone as a definitive treatment, or as a temporary treatment to allow soft tissue healing before internal fixation[3] and have been used on bones of the upper and lower extremities, cervical and lumbar spine, mandible, and pelvis.[4–6] The benefits of external fixation include its versatility, relative ease of surgical application, ability to maintain the wound site for better healing, and allowance for early weight-bearing[1, 7] as well causing less disruption of soft tissue and blood supply compared with internal fixation or intramedullary nails, making them better for trauma settings or in patients with compromised healing abilities.[2]

Despite the many of advantages of external fixation, the method is not without potential problems with numerous complications having been described, most notably pin tract infections.[1, 8–11] Recent studies have reported rates of pin tract infections of up to 96.6%, although other studies show lower rates.[10] Other infections, such as chronic osteomyelitis, sepsis, or myiasis, may occur as well.[1, 10–14] Non-union or mal-union of the bones is another potential adverse effect of external fixators, as is development of iatrogenic fractures.[1, 15–18] Another common complication of external fixators is neurovascular damage. Neurovascular damage may manifest itself as a neurological deficit, reflex sympathetic dystrophy, compartment syndrome, deep vein thrombosis, fat embolism, or pseudo-aneurysm.[1, 3, 9, 14, 19–25] Additional complications observed in the literature include bone angulation, contractures, tissue necrosis, and hardware malfunction.[1, 12, 26–28]

Much research has been devoted to understanding the adverse effects of external fixation; however, there is currently a lack of consensus about what additional variables are associated with developing these complications. Most studies dealing with complications have mainly focus on the rates of complications and their treatment recommendations, while excluding mention of what other factors may place a patient at risk. For example, Ahlborg and Josefsson note that they observed such complications as secondary displacement requiring re-reduction, non-unions, additional surgeries due to carpal tunnel, tendon rupture, and reflex sympathetic dystrophy, but the authors do not give comment or explanation beyond the rates of each complication.[29] Other studies state factors that predict complications, but without sufficient evidence to support their claims.[1, 12] Some studies do show predictive factors for complications, but with too limited a scope, i.e. only certain indications for external fixation, certain anatomical locations, certain outcome measurements, or certain types of external fixator devices.[30–32]

This study aims to expand on the research that describes variables associated with complications of external fixation by providing a more comprehensive analysis than is currently found in the literature. Such variables may include patient demographics, injury type, intra- and post-operative care, patient behaviors, and presence of comorbidities. By discovering correlations between one or more variables and development of complications after external fixation, these variables may identified as risk factors that predict a bad outcome after external fixation.

Materials and Methods

After IRB approval for exempt status was obtained, a patient registry database was used to compile a list of patients who underwent surgical application of one or more external fixator to an upper or lower extremity at Temple University Hospital between the dates of January 1, 2006 and May 30, 2012. The CPT code 20690, associated with application of a

uniplanar external fixator, was used. Surgical application of external fixator devices by orthopedic surgeons at Temple University Hospital were included. This search generated an initial list of 153 patients. After removing patients for which adequate medical records could not be obtained, a list of 75 patients were eligible for the study. A data sheet was generated to document all parameters to be studied, as described below. Retrospective review of hospital electronic medical records of eligible patients was performed and findings noted in the data sheet.

Demographic data, defined as age, gender, race, and insurance status, was recorded. Details about the indication for external fixation was noted, including extremity and bone affected, associated injuries, and mechanism of injury. Specification of open or closed fracture was recorded as well, in addition to device manufacturer.

The authors documented preoperative, intraoperative, and postoperative care according to the following parameters. For preoperative care, the time between injury and external fixator application was recorded, as well as whether anticoagulants and antibiotic medications were given during this timeframe. Intraoperative care parameters recorded included time in surgery, tourniquet time, and whether pinholes were pre-drilled or hand-drilled. Also noted were details about the pins used by the surgeon, including their size, position, and number of pins. For postoperative care, the authors recorded length of hospital stay, frequency and agent of pin cleaning, whether physical therapy was given, time in external fixation, secondary treatments after external fixator removal, time between external fixation and secondary treatments, additional surgeries performed, and length of follow up.

Medical history and comorbidities were noted, as documented in subjects' medical records, including presence of diabetes mellitus (complicated or uncomplicated), rheumatoid arthritis, present systemic or local infection, HIV, renal disease, osteoporosis or osteopenia, depression, dementia, chronic vascular insufficiency, coronary artery disease, hypertension, hypothyroidism, lymphedema, or other diseases. MRSA history, history of disease in affected bone or joint, and BMI were also recorded, as were patient behaviors including smoking, and drug use. The authors recorded preoperative glucose levels as well.

Incidences of complications were recorded, as documented in patient charts. Infections were noted, including pin tract infection, osteomyeltis, myiasis, or other infections. The authors also documented incidence of ARDS, tissue necrosis, and purulent pin-site drainage. Bone-related complications were recorded, including nonunion or mal-union, heterotopic ossification, angulation, and iatrogenic fractures. Neurovascular adverse effects were noted, including neurologic deficit, reflex sympathetic dystrophy, compartment syndrome, deep vein thrombosis, fat embolism, or pseudoaneurysm. The authors documented both occurrence of complications at any point during follow up and complications that occurred during specific time frames. The authors defined these time frames as follows: 1) during external fixation; 2) after removal of external fixator before placement of additional treatment method; 3) after removal of external fixator and during subsequent treatment method; 4) after removal of both external fixator and subsequent treatment (or after removal of external fixator without subsequent treatment); 5) up to one week after external fixator application; 6) up to 30 days after external fixator application; 7) up to six months after external fixator application; 8) up to two years after external fixator application or to total follow up time (if follow up exceeds two years).

After data was collected, results were analyzed with the assistance of the Temple Biostatistical Center. Outcome variables were correlated with risk variables to screen for a risk or predictive relationship. The relationships found to have statistical significance ($p < 0.05$) and moderate or strong strength of correlation (Spearman correlation coefficient >0.5) were further studied using odds ratio estimates and slope regression estimates.

Results

In total, 75 uniplanar external fixators were applied to 75 patients (55 male, 20 female), with an average patient age of 43.6 (range 17–82). Fifty-nine patients had documented comorbid conditions at the time of external fixator application, with an average of 2.8 comorbidities per patient (of those who had one or more comorbidity). The most common comorbid conditions were hypertension (21 cases), obesity (BMI >30, 20 cases), asthma (13 cases), dyslipidemia (10 cases), diabetes mellitus (10 cases), and history of injury in currently injured site (seven cases). Twenty five of the external fixators were applied to treat factures in the upper extremity, while 50 were applied to the lower extremity. The most common site of injury was the distal tibia. The most common causes of injury were falls, followed by gunshot wounds and pedestrian-versus-auto accidents. The average time between injury and application of external fixator was 4.5 days, and the average length of hospital stay was 11.6 days. External fixators remained on fracture sites for an average of 51 days (range 2–439 days). The average length of follow-up was 14 months.

Table 1. Overall Comorbid Conditions

Comorbidity	Number of Cases	Average Per Case
Any comorbidity	59	2.8
Hypertension	21	
Obesity (BMI >30)	20	
Asthma	13	
Dyslipidemia	10	
Diabetes mellitus	10	
Prior injury	7	

Of the 75 cases, 59 developed one or more complication during the follow-up period (78.67%). Of those who developed complications, the average number of complications

was 2.4 per patient. The most commonly recorded complications were heterotopic ossification (16 cases), nonunion (15 cases), neurologic deficit (14 cases), tissue necrosis (11 cases), and pin tract infection (nine cases). Other complications of note that occurred in one or more cases included compartment syndrome, purulent pin site drainage, osteomyelitis, lipohemarthrosis, deep vein thrombosis, ARDS, angulation, and iatrogenic fracture.

Table 2. Overall Complications

Complication	Number of Cases	Average Per Case
Any complication	59	2.4
Heterotopic ossification	16	
Nonunion	15	
Neurologic deficit	14	
Tissue necrosis	11	
Pin tract infection	9	

Overall, patients with higher numbers of comorbidities were more likely to develop complications than those with fewer comorbidities. Specifically, for each additional comorbidity that a patient had, the risk of an increased number of complications developing within 90 days of application of external fixator increased by 1.5 times, adjusted for age, sex, and BMI. Furthermore, the number of complications that a patient develops at any time increased by an average of 0.24 for each additional comorbidity that a patient had.

While only three patients developed iatrogenic fractures, this complication was correlated with two specific comorbidities. Patients with a history of osteomyelitis were almost 37 times more likely to develop iatrogenic fractures than those without a history of osteomyelitis (odds ratio estimate 36.958). Similarly, patients with a history of sarcoidosis were almost 140 times more likely to develop iatrogenic fracture than those without a history of sarcoidosis (odds ratio estimate 139.985). Sarcoidosis history was also correlated with an increased incidence of pin tract infection (odds ratio estimate 18.285) and tissue necrosis (odds ratio estimate 13.778). A history of injury in the affected limb was correlated with purulent pin site drainage, with an estimated odds ratio of 16.00.

Discussion

Previous studies have shown very high complication rates associated with external fixation, and our study confirms that complications are quite common with this treatment modality.[1, 10] Our study also indicates that patients' comorbidities play a significant role in predicting complications that may occur during the postoperative period. Regardless of what medical conditions a patient has, more comorbid conditions may lead to more complications after external fixation. This suggests that patients' medical histories should be more closely assessed to determine if they are safe candidates for external fixation. If a patient has significant comorbidities, perhaps an additional treatment modality would be better indicated.

Our study indicates that sarcoidosis, osteomyelitis, and previous injury in the fractured limb may be problematic comorbid conditions for external fixation since they were associated with significant complication rates. This may be explained by the idea that osteomyelitis and other previous bone injuries cause weakened bone tissue that predispose it to fracture or infection when an external fixator is applied. Similarly, the systemic inflammation associated with sarcoidosis may predispose bones to structural damage and infection upon placement of an external fixator. Further research regarding the mechanisms by which these conditions may lead to external fixation complications is needed.

Limitations of the study include the small sample size and low statistical power. Because so many variables were examined with a small number of cases, there may be limitations on the reproducibility of statistical outcomes. For instance, only three patients had comorbid sarcoidosis and only two had history of osteomyelitis, so the calculated odds ratio estimates of these comorbidities associated with specific complications may be overestimates of their true associations. Additionally, all information was collected from electronic medical records, so errors in documentation may have occurred. Future studies, particularly of a prospective design, are warranted in order to more accurately gauge the risk factors of external fixation complications and determine causality.

Conclusion

According to our study, having multiple co-morbidities at the time of external fixator application may be correlated with higher rates of complications. Medical conditions of particular concern include osteomyelitis, sarcoidosis, and previous injury in the limb undergoing external fixation. Further research is needed to assess these risk factors and complications in more detail.

Table 3. Risk Factors for Complications

Variable	Outcome	Odds Ratio Estimate	Slope Regression Coefficient	p Value
Number of comorbidities	Total number of complications		0.2377	0.0307
Number of comorbidities	Number of complications within 90 days	1.492		0.0313
Osteomyelitis history	Iatrogenic fracture	36.958		0.0381
Sarcoidosis	Iatrogenic fracture	139.985		0.0018
Sarcoidosis	Pin tract infection	18.285		0.0240
Sarcoidosis	Tissue necrosis	13.778		0.0398
Prior injury	Purulent pin site drainage	16.000		0.0041

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Disparities in Internet Usage by Orthopaedic Outpatients

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Abstract

The "digital divide," identified in the US, established that internet access has lagged behind among patients of lower income and of certain ethnic groups.

Expansion of internet access over the past decade is analogous to the recent proliferation of smartphones, extending online use to specific patient groups, affecting access to online health information, and potentially revolutionizing this divide. The 28-question survey, completed by 100 orthopaedic outpatients, evaluated associations between patient age, ethnicity, income, or education level, and their access to the internet, use to obtain information about their medical condition, privacy concerns during this online research, and patient use of mobile phones as a primary means of internet access. The internet was used by 57% of orthopaedic outpatients in our urban population, internet access decreased with age, and increased with income and education level, which were consistent findings from similar studies. Despite the inability to identify an association between ethnicity and internet access in this patient population, fewer Latinos sought information about a personal medical condition than did Caucasians or African Americans. Amongst patients who used their mobile phone as a primary method for online access, 74% were African American or Latino, significantly greater than 26% Caucasians. This deviation in online smartphone use in conjunction with the lack of disparities in internet use found between ethnic groups insinuates that mobile phones have provided ethnic minorities with greater internet access, and thus made a probable contribution to the narrowing of the "digital divide" amongst the races in our population of orthopaedic outpatients.

Introduction

The technological revolution triggered by the rapid growth of the internet has provided patients with unprecedented access to medical information.[1] Access to the internet by adults in the US has improved greatly over the past decade, and the internet is becoming the preferred source for patients to acquire health information.[2] According to a survey conducted in 2010, 74% of adults in the US use the internet and 80% of them searched for health-related topics, which equates to 113 million Americans that obtain health information on the internet, or 59% of all adults in the US.[3] Within the field of medicine, it was found that as many as 75% of patients had access to the internet themselves or through a close friend or family member.[4] This widespread use of the internet by patients to access health information has progressively broadened and reshaped the role of physicians and health care providers, while it has the potential to enhance patient satisfaction and participation in health care.[5]

Previous studies have evaluated the use of the internet in the specific clinical setting of orthopaedic outpatient practices. A research group at the University of Michigan found that internet use by orthopaedic patients had increased from 20% in 1998 to 46% in 2003.[6] Of internet users in a study of over 500 orthopaedic patients at the University of Pennsylvania, 47% of patients who accessed the internet reported that they had used the web to retrieve health or medical information.[7] More explicitly related to orthopaedic information, 20% of patients in a community outpatient practice used the internet to research their current orthopaedic diagnosis.[8] A patient, or a surrogate responder for the patient, was more likely to search for the patient's pediatric orthopaedic diagnosis on the internet if the condition was chronic like scoliosis (54%), as compared with a much lower rate (18%) in acute conditions like fractures.[9]

The majority of consumers who searched for health information on the internet began their inquiry by using a search engine, and 58% of people implicated that the information obtained from their search had an impact on a decision regarding treatment of an illness or condition.[10] While health information on the internet has clearly influenced the health decisions made by patients, only one of 57 pediatric orthopaedic patient education articles available on the AAOS and POSNA web sites had the recommended readability grade level.[11] Other prevalent concerns that pertain to health information on the internet, particularly because confidentiality is imperative in maintaining the integrity of a patient's medical care, include the security and privacy of personal information when health information is accessed online. Analysis at

an orthopaedic outpatient clinic found to a statistically significant degree, that patients with lower annual household incomes were more apprehensive about both privacy and security when they went online.[12] The internet has the ability to eliminate barriers to access to information, but only if online material can be accessed, read and understood by many different types of users.[2]

Despite the rapid growth of the internet and the penetration of the internet into the health care industry in recent years, internet access has lagged behind among lower socioeconomic classes and certain groups of ethnic minorities, a phenomenon that is known as the digital divide.[13] This concept was studied in a hand surgery outpatient clinic, which showed that as household income increases, there was a greater likelihood of owning a computer, more time was spent on the internet, and patients more frequently found that information on the internet was trustworthy, secure, and private.[12] A similar study conducted in an elective spinal surgery outpatient population in Ireland showed that increasing age, higher education level, and possession of health insurance were all significantly associated with access to the internet.[14] Although in recent years, the online population has become more representative of the larger US population in terms of race, age, income, and level of education,[15] uncertainty remains as to whether the "digital divide" remains prevalent in access to the internet and in the use of health care information by the orthopaedic outpatient population. Furthermore, the soaring popularity of smartphones in the general US population, illustrated by an increase in adults who own a smartphone from 35% in May 2011 to 46% in February 2012, has changed internet access particularly for groups that have traditionally been on the other side of the "digital divide."[16] This report described that amongst smartphone users, ethnic minorities, less educated individuals, and those with lower household incomes were more likely to identify their mobile phone as the primary source for accessing the internet. It is our suspicion that this phenomenon has improved our own patients' access to the internet, many of whom are ethnic minorities and of low income, perhaps narrowing the "digital divide."

Although disparities between ethnic groups in the general US population have been identified in home computer ownership, work computer access, and internet use,[17] no medical research to date has explored the influence of ethnicity on the internet use by these orthopaedic patients in an urban outpatient setting. This study, therefore, aims to evaluate differences in patient demographics that may influence internet access in urban orthopaedic outpatients, determine if mobile access has narrowed the "digital divide" in our patients, and explore the variation in the patient use of the internet based on their ethnic background.

Materials and Methods

Adult patients aged 18–89 years old who were being treated for an orthopaedic condition at an outpatient orthopaedic clinic of an urban academic medical center between June and August 2010 were given the opportunity to participate in the study. The 103 patients who consented to participate were administered a survey in a private setting with a laptop and secure internet connection. The survey was administered via Surveymonkey.com. Incarcerated persons were excluded from this study. In addition, two patients that completed the survey were under the age of 18 and one patient did not submit a finished survey. These three patients were also excluded and, consequently, the remaining 100 patients were included in our analysis. The institutional review board approved this investigation.

A single questionnaire consisting of 28 questions was used (Appendix 1). Eight questions pertained to the patient's background information and included: age, gender, ethnicity, primary language, annual household income, level of education, type of health insurance, and the condition for which the patient was seeking treatment. The remaining 20 questions were designed to ascertain information from the patients regarding their internet use in the following categories:

1. General internet access: use within the past year, modes of access, average use per day, main reasons for use, and primary search engines used.

2. Internet use pertaining to the patient's health: had the patient used the internet to find out about a personal health condition, did the patient or anyone else use the internet to find out about the condition that the patients was currently visiting the clinic for, and which websites did the patient or the surrogate use to obtain the information about this condition.

3. The influence of health information obtained from the internet on the patient's interaction with the doctor: did the patient plan on discussing what they learned on the internet with their doctor, would the patient mention to the doctor that the question or topic resulted from an internet search, and did they believe that doctors appreciate it when patients use the internet to find out medical information.

4. The patient's experience and opinions of the health information that they found on the internet about their condition: was it helpful, clear, trustworthy, difficult to understand; did it help the patient make a decision about their health, and did the information bring up things that the patient wanted to discuss with their doctor.

5. Internet privacy: did this concern the patient, and did it affect the patient's use of the internet to look up personal health information.

The survey enabled patients to skip or leave questions blank if they desired. The questions regarding the patient's means for accessing the internet, main reasons for using the internet, primary search engines used, and the sites used to obtain health information, allowed patients to select all applicable choices. Five participants reported multiple education levels, and the highest level that they selected was exclusively used in our analysis. Three patients that selected

Appendix 1

THIS IS AN ANONYMOUS SURVEY. YOU MAY SKIP QUESTIONS THAT YOU DO NOT WANT TO ANSWER.

Have you used the internet within the past year?

☐ Yes ☐ No

What is the condition for which you are currently seeking treatment?

☐ Fracture ☐ Arthritis ☐ Deformity
☐ Sprain/strain ☐ Spinal condition ☐ Other

Please select all the ways that you access the internet.

☐ Computer at home ☐ Computer at a library or other public building
☐ Computer at work or school ☐ Mobile phone

On average, how many hours do you spend on the Internet each day? _____ Hours

What are the main reasons for using the internet? You may select more than one answer.

☐ E-mail/Communication ☐ News ☐ Entertainment
☐ Social networking ☐ Shopping ☐ Health/medical information

What search engines do you primarily use? You may select more than one answer.

☐ Google ☐ AOL Search ☐ Ask.com
☐ Yahoo ☐ Bling ☐ Alta-Vista

Have you ever used the internet to find out about a personal health condition?

☐ Yes ☐ No

Did you use the internet to find out information about the condition you are here for today?

☐ Yes ☐ No

Did anyone use the internet to find out information about the condition you are here for today?

☐ Yes ☐ No

Which sites did you or someone else use? Please select all choices that apply.

☐ WebMD ☐ CDC Health Topics A to Z ☐ HealthAtoZ
☐ Health Central ☐ AAOS.org ☐ Other
☐ WrongDiagnosis.com ☐ Wikipedia

Did you or do you plan on discussing anything that you learned in your internet search with your doctor?

☐ Yes ☐ No

Do you think you will mention that the question or topic came up as a result of an internet search?

☐ Yes ☐ No

Do you have concerns about privacy when using the internet?

☐ Yes ☐ No

Does privacy affect your use of the internet to look up personal health information?

☐ Yes ☐ No

What is your current age? _____ Years old

Gender ☐ Male ☐ Female

Ethnicity

☐ African American ☐ Latino/Hispanic ☐ Caucasian
☐ Asian ☐ Native American ☐ Other

Primary language ☐ English ☐ Spanish ☐ Other

Annual household income

☐ <$10,000 ☐ $18,001–35,000 ☐ $50,001–100,000
☐ $10,001–18,000 ☐ $35,001–50,000 ☐ >$100,000

Education level

☐ No high school ☐ GED ☐ Some high school
☐ Some college ☐ High school graduate ☐ College graduate

Health insurance

☐ Medicare ☐ Health Partners ☐ Private
☐ Medicaid ☐ Keystone Health ☐ None

Continued on next page

Appendix 1 ***Continued***

FOR THE FOLLOWING QUESTIONS, PLEASE SELECT THE STATEMENT THAT BEST FITS YOUR EXPERIENCE ABOUT USING THE INTERNET TO FIND INFORMATION ABOUT YOUR MEDICAL CONDITION.

The information was helpful.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

The information was clear.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

The information was trustworthy.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

The information was difficult to understand.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

The information helped me to make a decision about my health.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

The information brought up things that I wanted to discuss with my doctor.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

I think doctors appreciate it when patients use the internet to find out medical information.

☐ Strongly agree ☐ Agree ☐ Neutral ☐ Disagree ☐ Strongly disagree

GED for their level of education were excluded from analysis based on education, and one patient who chose other for their ethnicity was likewise excluded from analysis based on race. Annual household income levels that were originally ascertained in the survey based on levels of less than $10,000, $10,001 to $18,000, $18,001 to $35,000, $35,001 to $50,000, $50,001 to $100,000, and greater than $100,000, were then grouped into three categories of less than $18,000, $18,000 to $50,000, and 50,001 to greater than $100,000. This was done on the basis that results were similar amongst the groups that we combined, which was also noted and performed by Parekh in a study conducted at a hand surgery outpatient clinic.[12] In a similar manner, the age of each patient was grouped into three categories consisting of patients less than 50 years old, patients aged 50 to 65, and those who were older than 65, as Baker had previously done successfully in a study of elective spinal surgery outpatients.[14]

Statistical analysis was performed using the JMP5 statistical software. Differences in the categorical data was assessed using the chi squared test, and differences that were found to have a $p < 0.05$ were considered statistically significant.

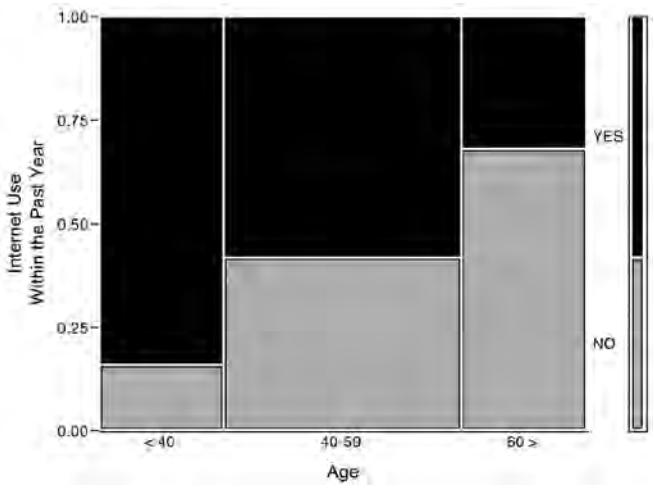

Figure 1. Orthopaedic outpatients who had used the internet within the past year in different age groups of patients.

Results

Internet Access

Of 100 patients attending the orthopaedic outpatient clinic in the urban setting of Philadelphia, 57% had used the internet within the past year. Internet use decreased as age of the patient increased (Figure 1). Younger patients less than 40 years of age were more likely than middle-aged patients of 40 to 50 years old to have used the internet ($p < .001$), and this trend of greater internet use in the younger population was even more striking when compared to use in elderly individuals 60 years or older ($p < .001$). Other demographic variables that were found to significantly influence patient internet use were household income and level of education (Table 1). Patients with an annual household income of less that $18,000 were less likely to have accessed the internet within the past year, as compared to those making between $18,000 and $50,000 ($p < .01$) and those making $50,001 to greater than 100,000 ($p < .05$). An analogous finding was seen in patients that did not graduate high school, who were found to have accessed the internet within the last year significantly less than patients who graduated high school ($p < .05$), attended some college ($p < .001$), and patients who graduated college ($p < .001$). In contrast however, we were unable to demonstrate a statistically significant difference in access to the internet between Caucasian, African-American, and Hispanic patients in our study population.

Table 1. Internet Use Within the Past Year

	No	Yes	p Value	Odds Ratio	95% CI
Age			.001		
<40	16% (4)	84% (21)	—	—	—
40–59	42% (20)	58% (28)	.03	0.27	(.08–0.89)
>60	68% (17)	32% (8)	<.001	0.09	(.02–0.35)
Ethnicity			.66		
Caucasian	37% (9)	63% (15)	—	—	—
African American	41% (21)	49% (30)	.76	1.17	(.43–3.16)
Latino/Hispanic	50% (12)	50% (12)	.38	.60	(.19–1.89)
Annual Household Income			.002		
<$18,000	58% (26)	42% (19)	—	—	—
$18,000–$50,000	21% (6)	79% (22)	.002	5.02	(1.71–14.76)
$50,001–>$100,000	18% (2)	82% (9)	.02	6.16	(1.19–31.82)
Highest Level of Education			<.001		
No high school diploma	77% (20)	23% (6)	—	—	—
High school graduate	50% (18)	50% (18)	.03	3.33	(1.09–10.24)
Some college	10% (2)	90% (18)	<.001	30	(5.36–167.93)
College graduate	13% (2)	87% (13)	<.001	21.67	(3.78–124.19)

Internet Use for Medical Purposes

In the outpatient clinics of our orthopaedics department, 65% of patients had used the internet in the past to find out about a personal medical condition. More explicitly, of these 57 patients, 19% reported that they had used the internet to find information regarding their health condition that was being addressed at the current orthopaedic visit, and in a similar question with 78 responses, 14% of patients stated that someone else had used the internet to obtain this information for the patient. A statistically significant association ($p = .03$) was found in our analysis between patient ethnicity and use of the internet to find information about a personal health condition (Table 2). Hispanic patients were found to have researched their medical condition on the internet less frequently than Caucasian or African American patients (Figure 2). The difference between Hispanics and Caucasians was nearly, but not statistically significant ($p = .09$), however there was definitively less use of the internet by Hispanics to obtain information about a personal health condition, as compared to African American patients ($p < .01$). In contrast, patient internet use to find out about a personal health condition was not significantly different based on age, or annual household income in this urban orthopaedic outpatient population. The final aspect of this survey that pertained to access of the internet for medical information was used to assess the prevalence of privacy concerns when patients searched the internet for information on their medical condition. Of the 52 patients who responded, 62% expressed that they had concerns about privacy. Despite finding that African Americans (67%) and Hispanics (64%) more frequently expressed a concern for privacy on the internet than Caucasians (50%), this was not a statistically significant disparity between the ethnic groups. In the same way, we were also unable to demonstrate a significant association between annual household income and patient outlook on privacy while searching the internet for information about their medical condition.

Table 2. Use of the Internet to Find Out About a Personal Health Condition

	No	Yes	p Value	Odds Ratio	95% CI
Ethnicity			.03		
Latino/Hispanic	67% (8)	33% (4)	—	—	—
Caucasian	33% (5)	67% (10)	.09	.25	(.05–1.25)
African American	23% (7)	77% (23)	.008	.15	(.04–0.66)

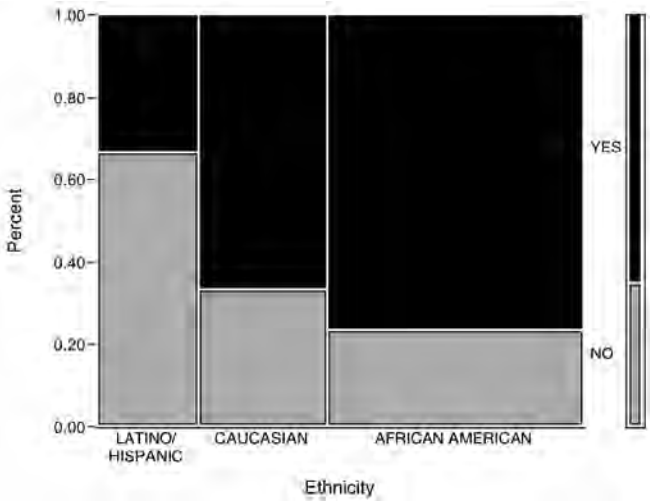

Figure 2. Patients who have used the internet to research a personal medical condition based on patient ethnicity amongst orthopaedic outpatients.

Characteristics of Patient Internet Use

Although part of this study relied on patient characteristics, which primarily included income, ethnicity, and smartphone use to assess disparities in internet access and use amongst orthopaedic outpatients, the survey also examined general elements of internet use in this population as a whole. Patients who attended orthopaedic clinics in this urban setting averaged three hours of internet use per day. Of these 56 responders that addressed their amount of internet use, the vast majority predominantly accessed the internet by

using a computer at home (84%), but there was also a large group of patients (48%) who used a mobile phone as their primary means for internet access (Figure 3). The main reason patients used the internet was for e-mail or communication (85%), while only 33% of the 55 participants that responded to this question listed health or medical information as a main reason for use. In comparison, other main reasons these patients used the internet included 53% for entertainment, 49% for news, and 40% for shopping. Internet use and the process of obtaining information online is frequently initiated through search engines, and an additional evaluation of these 55 patients determined that Google was the most widely used search engine (80%), although the majority of patients used Google, Yahoo (56%), or both.

Discussion

This study was designed to evaluate the level of internet access within a sample of patients attending orthopaedic outpatient clinics of an urban academic medical center and to determine patient demographics that predict characteristics of patient internet use.

The internet was accessed within the past year by 57% (95% CI: 47–66%) of patients in our survey, which was to some extent less than the 2010 national average (74%) of adults using the internet in the US.[3] Highly probable explanations for this observed disparity are the annual household income and education level of individuals living in the urban population of Philadelphia. Our study population contained 39% (95% CI: 30–50%) of patients with an annual household income less than $10,000, and only 7% (95% CI: 3–15%) with an annual household income greater than $100,000. This varies from the 2011 US population assessed by the US Census Bureau to have 8% of citizens with an annual household income less than $10,000 and 21% with greater than $100,000.[18] An additional aspect of our study population that contributed to this discrepancy in internet access was education level, most notably 27% (95% CI: 19–36%) of patients failed to obtain a high school diploma and only 15% (95% CI: 10–24%) had graduated college, as compared to 13% and 37% respectively, which are characteristic of the US population.[19] Because of demographic disparities between our patients population and US citizens in general, including household income and education level, comparing patient internet access found in our study with a similar urban population of orthopaedic outpatients would be of greater analytic utility. In doing this, the 57% of patients that we found to have access to the internet was identical to the percentage reported in a 2004 study of orthopaedic patients at the University of Pennsylvania, which consisted of a survey population that profoundly mirrored ours in many of these aspects. Significant characteristics of internet access by these patients pertain to outpatient orthopaedic care, including the use of the internet to obtain general health or medical information and to research a personal health condition or orthopaedic diagnosis. In 2006, 64% of internet users searched online for a specific disease or medical condition.[10] A similar result from our study demonstrated that 65% (95% CI: 52–76%) of orthopaedic outpatients had at some point used the internet to find out about a personal health condition. There was also marked consistency between the 19% (95% CI: 11–31%) of patients found in our study who used the internet to find information about the orthopaedic condition for which they were currently attending the clinic, and the 21% of orthopaedic outpatients in another Philadelphia practice who used the internet to learn about their orthopaedic condition.[7]

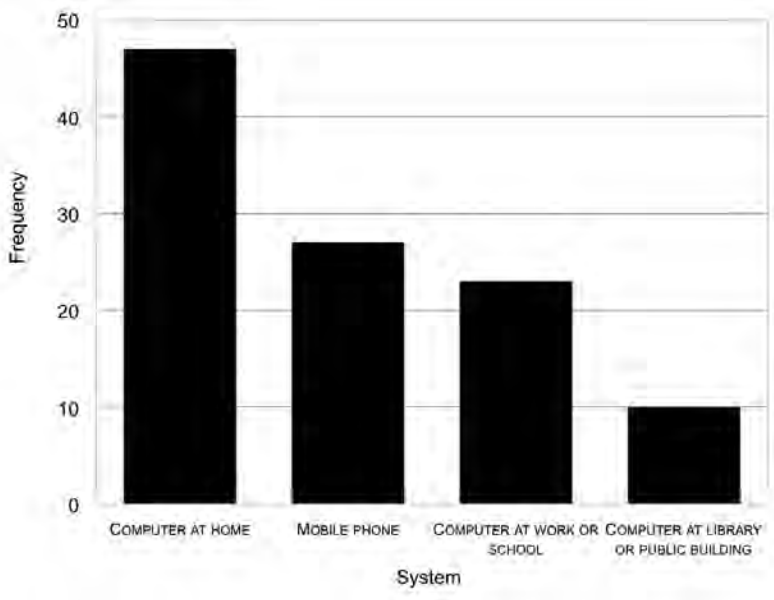

Figure 3. The primary means used by orthopaedic outpatients to access the internet.

Household income, education level, and age had previously been shown to predict internet access in the US population, as well as in surveys of orthopaedic outpatients. Based on the income of US citizens, 57% of people earning less than $70,000 use the internet, as compared with 95% of individuals earning greater than $75,000.[20] This "digital divide" amongst households with annual income inequalities was also depicted in a population of orthopaedic patients, where a statistically significant increase in internet use was associated with increased household incomes in groups earning <$18,000, between $18,000 to $50,000, and $50,000 to greater than $100,000, which was determined by the hours per day patients at an outpatient hand surgery clinic used the internet.[12] Using these same income groupings in our study, we were able to reassert this trend, as a statistically significant increase in patient internet access was identified when annual household income increased. Education level is also a contributing factor to the "digital divide," illustrated in a 2012 survey that showed

43% of US citizens with no high school diploma were internet users, while 71% of high school graduates, 88% of individuals with some college education, and 94% of college graduates used the internet.[16] While the significance that education level has in influencing internet access had been studied in an elective spinal surgery outpatient population in Ireland, the structure of the European education system is considerably different, and no study to date has evaluated if the education level of orthopaedic outpatients predicts their internet access in a US medical survey. Our study in the urban population of Philadelphia illustrated statistically significant greater use of the internet as the level of education achieved by the orthopaedic outpatient increased, which was determined by individually comparing patients with no high school diploma to high school graduates, patients who attended some college, and college graduates. An additional factor contributing to orthopaedic outpatient internet access had been previously discovered between age groups, with a range of 77% use in patients less than 30 years old to 16% use in those greater than 70 years old.[7] This trend of decreased internet use with increased age was confirmed to be statistically significant in our study, where fewer patients aged 40–59 and 60 or older were found independently to have accessed the internet within the past year than patients who were less than 40 years old.

In the setting of an orthopaedic outpatient population, research has not established patient race as a predictor of internet access. The disparity in internet use amongst particular ethnic groups in the US had been characterized in 2011, which found that 77% of Caucasians, 66% of African Americans, and 62% of Latinos go online.[3] In comparison with the general US population, the results from our study found that the percent of orthopaedic outpatients who used the internet within the past year was lower in each ethnic group, with 63% of Caucasians, 49% of African Americans, and 50% of Latinos reporting use. As depicted previously, the annual household incomes in our patient population were substantially lower than the incomes representative of US citizens in general, which conceivably had a critical impact on the lower percentage of orthopaedic outpatients in each ethnic group who were found to have used the internet in our study. Despite the smaller percent of patients with internet access in our study, their disproportionately low household incomes was an improbable explanation for the inability of our study to identify a statistically significant variation in internet access contingent on patient race. With the significant relationship between internet access and income distributions that was demonstrated by our study, which illustrated an increase in internet access as income level increased, race would have compounded rather than eliminated any deviations in internet use because of the lower income levels that are present amongst ethnic minorities in the US. Alternatively, race as predictor of internet use most plausibly proved unattainable because of the demographic underrepresentation of Caucasians and the extraordinary predominance of African Americans in our survey populous at orthopaedic clinics in Philadelphia. Patients participating in our survey consisted of 24% Caucasians (95% CI: 17–34%), 52% African Americans (95% CI: 42–61%), and 24% Hispanics (95% CI: 17–34%), while a 2010 overview of race in the US found a distribution of 72.4% Caucasians, 12.6% African Americans, and 16.3% Hispanics.[21] Increasing the number of Caucasian patients participating in our survey would provide additional data on use of the internet in this ethnic group, allowing for a more representative comparison to African American and Hispanic patients, and thus enhancing the capability of our study to define race as a statistically significant determinant of patient internet use in the urban orthopaedic setting.

Survey participants who reported that a mobile phone was one of the primary ways they accessed the internet were comprised of 74% non-white (Latino or African American) and 26% Caucasian patients, as compared to 38% and 17% respectively, which were found amongst the 25% of all smartphone owners in the US who mostly went online using their cell phone.[16] Our study showed that of patients who used their smartphone as a primary way to access the internet, 48% were high school graduates, 35% attended some college, and 17% had a college degree. In comparison to smartphone users in the US, those found to primarily use their cell phone to go online consisted of 33% high school graduates, 27% individuals who attended some college, and 13% college graduates.[16] Of the patients in our study who primarily used their smartphones to access the internet, 57% had an annual household income less than $35,000, 26% made $35,000 to $50,000, and 17% earned more than $50,000. This was similar to the result from a 2011 US population of smartphone owners, 40% of whom earned less than $30,000 annually, while 29% had income of $30,000 to $50,000, and 17% made greater than $50,000.[16] These similar findings show that the patients on the other side of the "digital divide" identify mobile phones as their primary means of accessing the internet more frequently than other groups, which suggests that the widespread availability of smartphones is most likely another factor underlying the greater amount of African Americans who were found in our study to have access to the internet. Although this suggests some narrowing in the "digital divide" amongst African Americans, a more definite evaluation should be performed to better understand the extent to which increased use of mobile phones, as a primary source for internet access, has influenced this divide in orthopaedic patients of lower income.

In a 2011 survey on health topics, Fox established that of participants who accessed the internet, 63% of Caucasians, 47% of African American, and 45% of Latinos used the internet to look for health information.[3] Similarities were found in our orthopaedic outpatient population, where Latinos (33%) used the internet to research a personal health condition less often than Caucasians (67%) and African

Americans (77%), of which both differences from the Latino race were statistically significant in each comparison independently. Previously, it had been established that there were deficiencies in health topics on the internet that were important for patients, a finding that was particularly striking across Spanish-language sites, where more than half of the condition-related topics were not addressed.[2] Spanish was the primary language spoken by 50% of the Latino patients in our study, while none of the Caucasians and only one African American identified Spanish as their primary language. Although variation in linguistic characteristics did not contribute to a disparity in internet access in our population of orthopaedic outpatients as there was no significant variation between ethnic groups, the language barrier found to be unique to Latino patients in our study was the principle component underlying the propensity for Latinos to have used the internet less than other races to learn about a personal health condition. As opposed to these findings in Latino patients, the percent of African Americans who reported that they had used the internet to look for information about a personal health condition (77%) was unexpectedly high, and controversially surpassed utilization of the internet by Caucasians for this same purpose, a result which had never been found in studies of internet use for healthcare purposes in the US population. In medical patient populations, however, including in the orthopaedic outpatient setting, the influence that ethnicity has on internet use for personal medical education remains undetermined. Our survey of orthopaedic outpatients exemplified that Latino patients search the internet about personal health conditions less than Caucasians and African American patients, while in this urban setting, the use of the internet to learn about a personal health condition amongst African American patients was considerably more than has been reported in the general US population.

Despite providing useful insight into the internet access and characteristic of its use in the orthopaedic outpatient population, our study had a few shortcomings that may have hindered our ability to detect some statistically significant findings in this urban setting. Although the 100 patients who responded to the survey provided enough data to evaluate certain aspects of internet use in our study population, increasing the number of surveys administered, with the primary goals of increasing the size of the Caucasian and Latino groups while providing a greater subpopulation of patients who had access to the internet, would provide sufficient data to evaluate more of the subsequent questions that characterized use of the internet. In evaluating the design of the questionnaire, a shorter survey would have provided for a larger number of patients who chose to answer questions towards the end of the survey, which evaluated patient opinions and experiences based on a scale from strongly agree to strongly disagree. An additional oversight in the structure of the survey was the unexpected ability of patients to select multiple answers when describing their education level and type of health insurance, as this prevented any statistically significant evaluation based on type of health insurance and forced us to analyze results based on education by the highest level of education reported. Finally, in our assessment of smartphone use as a primary source for patient internet access, asking patients about their ownership of a smartphone would have enabled us to determine if owning a phone with online capacities had an influence on internet use based on the race of the patient and on their annual household income. Including this question would similarly have allowed us to more directly compare our patient population to the 2011 US population, which determined the percent of smartphone owners who used this mobile device as their primary means of accessing the internet based on ethnicity, income, and education level. This was opposed to our study, which could only determine the percent of patients reporting their mobile phone as a primary way of obtaining internet access who were of a certain ethnic group or in the various categories of annual household income.

Over the next few decades, the healthcare system in the US will continue to evolve with the more extensive integration of the internet into the field of medicine. While most patients in the US already have access to the internet, their use of online resources for obtaining information about their general health and personal medical conditions continues to progress. Within the orthopaedics community, trends in outpatient access to the internet and in the characteristics of patient internet use had been shown to be predicted by age, income, education level, and furthermore, now additionally by ethnicity. In the future, ethnicity and these other patient demographics should be considered when orthopaedic decisions are made, which may involve or be influenced by a patient's use of the internet, especially in an urban setting that consists of significant minority populations and is characterized by prevalent financial and educational inequalities. Efficiently and effectively improving patient use of the internet to obtain medical information will benefit both the doctor and the patient by enhancing patient education, while also strengthening the doctor-patient relationship and ultimately enabling the doctor to provide a higher quality of patient care.

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Medical Student Research Project

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The Shoe-Surface Interface as a Profile Component Responsible for Knee Injuries in American Football: A Systematic Analysis

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Introduction

Since the late 1940s, there has been concern about the increasing incidence of injuries in competitive sports, particularly the prevalence of lower limb injuries in American football players.[1] Hanley first identified cleat fixation as a potential risk factor of lower limb injuries.[2] Shortly thereafter, in 1969 Rowe et al.[3] and in 1971 Torg et al.[1] described the association between cleat characteristics and knee and ankle injuries in high school football players. Several subsequent studies identified turf characteristics and conditions as additional risk factors contributing to injuries.[4, 5] These early studies and findings were very influential in beginning an important area of research as well as helping to form guidelines for equipment use in organized sports.

Since that time, many studies have looked at the shoe, the surface or both in regards to athletic injuries. While study designs have varied and findings have been inconsistent, the importance of this topic grows with the popularity of football from youth to professional levels. The incidence of ankle, foot, and knee injuries in high school players was recently reported to account for 37.6% of all football injuries. At the NCAA football level, it was found to be 36.9% of all injuries.[6] An additionally interesting and important viewpoint is one of economics. The healthcare cost of treating these injuries has been estimated at many millions annually.[7] Furthermore, as Levy first pointed out, the economics of equipment used (in his case, field turf) is another important consideration.[8] Combining this information with such a large prevalence of injury displays the importance for risk factor identification and injury prevention through guideline formation, i.e. rules and regulations regarding both playing issues, environment, and equipment.

As the times changed, so has the equipment manufactured for athletic use. Updated materials and designs were used for cleats. Newer generations and engineering techniques have been applied to artificial turf.[8] The literature has also changed in order to evaluate the most current equipment being used at the shoe-surface interface. This has made an issue of interpreting seemingly outdated studies and their application for current recommendations. A recent publication has briefly highlighted a few historical points and findings and has called for more epidemiologic studies to investigate this area.[9] Our goal is to more completely inspect the history of the literature from its inception in order to shed light on the current state of the shoe-surface interface in American football injuries.

Methods

Pertinent articles were found using the NCBI's PubMed database and a keyword search including: "cleat," "shoe-surface interface," "football injuries," and "artificial turf." From the results, papers were excluded if they focused on information from European football rather than American football or if the injury topic did not focus on the lower extremity.

After reviewing these articles, levels of evidence were assigned to each publication and included in Table 1. Articles were reviewed for historical contribution to the research subject and relevance to current shoe-surface interface technology. The pool of literature was analyzed for trends and errors that reoccurred throughout the last 40 years.

Results

The database search described above resulted in 32 publications covering the time period from 1969 to May 2012.

As stated previously, in 1969 Hanely and Rowe identified that cleat fixation was a risk factor for injury.[2, 3] Further investigating this risk factor, Torg in 1971 reported on an investigational field study using Philadelphia Public and Catholic high school football league players that shoe designs that decreased foot fixation, the soccer-style shoe, was able to decrease injury rates.[1] Torg's study proposed a recommendation for shoe sole specifications that ultimate changed the equipment guidelines of both the National Federation of High Schools Associations and the National Collegiate Athletic Association. The following year, it was identified that surface characteristic were an additional potential risk factor and that increasingly popular artificial turf had an increased rate of injury compared to natural grass. Further

Table 1. Bibliography Levels of Evidence

Publication	Level of Evidence	Publication	Level of Evidence
Adkison et al.[10]	4	Livesay et al.[11]	2
Andreasson et al.[12]	2	Meyers et al.[7]	2
Bonstingl et al.[13]	2	Meyers et al.[14]	3
Bradley et al.[15]	2	Mueller et al.[5]	3
Bramwell et al.[4]	4	Orchard et al.[16]	3
Cameron et al.[17]	5	Powell et al.[18]	3
Cawley et al.[19]	-	Reider[9]	5
Dick et al.[20]	3	Rowe[3]	3
Dragoo et al.[21]	2	Shankar[6]	3
Ford et al.[22]	4	Skovron[23]	4
Garrick et al.[24]	3	Stanitski[25]	4
Hanley et al.[2]	5	Torg et al.[1]	1
Heidt et al.[26]	2	Torg et al.[27]	1
Keene et al.[28]	4	Torg et al.[29]	3
Lambson et al.[30]	2	Villwock et al.[31]	2
Levy et al.[8]	2	Williams et al.[32]	2

studies confirmed foot fixation as a risk factor and that the soccer shoe decreased injury rates compared to the traditional football cleat.[4, 5, 17]

An early work by Cameron and Davis on the swivel shoe aimed at reducing fixation was more influential in descriptively stating the mechanisms of several different lower limb football injuries.[17]

An interesting and new perspective was introduced by Stanitski in 1974 when he demonstrated that while artificial turf has a higher coefficient of friction, it provided increased player speed. From an injury standpoint, this was potentially a problem in that he proposed that "synthetic playing surfaces changed the complexion of the game by increasing player speed . . . may set the stage for increased collision forces between players with resultant increase in the severity of injuries."[25]

Adkison et al. subsequently demonstrated that not all artificial surfaces could be classified together. It was shown that AstroTurf had a greater injury rate than grass, but a different artificial surface, Tartan Turf, actually had a lower injury rate in a high school football study.[10] Several years later in a different study, Kennen et al. also found that Tartan Turf had a decreased numbers of injuries when compared to other surfaces, although interpretation of the results is possibly limited by study design.[28]

In a novel study in 1974, Torg designed an apparatus that could be used in the laboratory to model the shoe-surface interface with rotational motion. He defined the release coefficient (Force/Weight) and applied this value for guidelines of shoe and surface combinations and their safety factor. On the basis of study, it was concluded that the molded sole soccer type shoe with 15 cleats with a maximum cleat length of ½ inch ½ length and a minimum cleat diameter of ½ inch as being safe on all playing surfaces, both grass and artificial. He further concluded that shoes with this sole configuration were safe on all surfaces, except those constructed with rubber soles, were not safe on all synthetic surfaces.[27] His apparatus was the first of its kind and the model for many subsequent studies.[11, 12, 26, 31] The following year, Bonstingl et al., using a different laboratory apparatus, demonstrated that foot stance developed 70% more torque than did toe stance and that synthetic turf developed more torque than did natural grass.[13]

Supporting these shoe-surface observations, Andreasson et al. further demonstrated in 1986 that not only the design but also the material of the shoe sole was a variable that affected the shoe-surface interface. A laboratory study concluded that lower torque was developed in polypropylene-like soles as compared to polyurethane and rubber soles.[12] Much later, a different publication would speculate about the material used in the shoes upper and how the pliability might affect potential injuries, especially in the ankle.[31]

In 1990, Levy performed a literature review to cover the development and characteristics of artificial playing surfaces on the American football player. It was stated that several benefits of artificial surfaces include lower maintenance, resulting in lower costs, and increased playing time availability. While much of the research showed increased injuries on artificial turf, he conclude that "it seems likely that manipulation of field characteristics are not only possible but essential, if player safety is to be maximized."[8]

In 1992, Powell showed that AstroTurf was implicated in higher rates of ACL sprains and that certain positions (running backs and linemen) under specific circumstance (rushing and passing plays respectively) were more likely to sustain knee injuries.[18] Torg again broadened the knowledge of the subject by reporting the effect of temperature on the shoe-surface interface. He concluded that more pliable cleats had greater release coefficients and that this was directly related to increasing temperatures. He specifically stated that "soft rubber sole shoes on warm AstroTurf create a risk factor."[29] Further studying the design of the football cleat, in a 1996 study, Lambson et al. showed that designs producing higher torsional friction, those with long peripheral cleats, were related with increased ACL injury. It was also reaffirmed that running backs, linemen, and linebackers had higher rates of injury.[30] In another study that year, the concept of spatting, wrapping the cleat and ankle with tape, affects the characteristics of the shoe-surface interface.[26]

In a 2003 study on how weather conditions affect NFL injuries, it was shown that grass produced less ankle sprains than did AstroTurf and less ACL injuries occurred on cold days in open stadiums.[16]

While one study showed that the majority of injuries occurred during non-contact situations in the NFL,[30] two others identified that contact injuries were more frequent in the NCAA.[20, 21]

With the advent of newer models of artificial turf, more recent studies have demonstrated that FieldTurf may actually be safer than grass in that there were more knee injuries and greater time loss injuries on grass in high school football.[14] In addition, there was a decreased incidence of ligament tears on FieldTurf and a higher incidence of ankle

injuries on grass in the NCAA.[7] Although the latest epidemiologic study has stated that there is an increased incidence of ACL injury on artificial turf, especially the newer generations that include granular infill,[21] it should also be noted that the validity of the former study has been called into question because of the source of funding.

Discussion

The purpose of this paper is to present a systematic review of the history of the shoe-surface interface and its role in lower limb injuries in American football. More specifically, it is intended as a response to a recent opinion article by Reider that we believe to have been incomplete on several issues and misguided regarding its conclusions.[9]

Of the 32 articles reviewed, two met the qualification of level 1 level of evidence as a "high-quality prospective study with testing of previously developed diagnostic criteria on consecutive patients." We refer to the 1971 report that on the basis of a three-year prospective clinical study involving 34 high school football teams on the effect of shoe type and cleat length on the incidence and severity of knee injuries investigated within the confines the Philadelphia Public and Catholic Football Leagues, a marked decrease in both the incidence and severity of knee injuries was effected by changing from the conventional shoe with seven ¾-inch cleats to a shoe with a molded polyurethane sole with 14 ⅜-inch cleats.[1] The subsequent laboratory study using an assay device to determine the association of the shoe-surface interface release coefficient to the risk for football knee injuries was published in 1974.[27] Release coefficients, an expression of static friction, were determined for 108 shoe-surface interface combinations. By correlating these values with the results of the Philadelphia High School Study, safety characteristics were described for each shoe-surface combination. The laboratory determinations indicated that the release coefficients varied with the number, length, and diameter of the cleats as well as the nature, natural or artificial, and condition, wet or dry, of the surface. The study concluded that the conventional seven-posted football shoe with seven ¾-inch cleats was not safe on grass. On the basis of this report, both the National Collegiate Athletic Association and the National Federation of High School Associations outlawed these cleats in favor those of ½ inch in length. And this regulation persists today. It was also determined that the molded sole soccer shoe with 15 cleats was safe on all surfaces. Subsequent studies, however, demonstrated that because of the frictional quality of natural soft rubber soles, this material was probably not safe on synthetic surfaces.

An analysis of the above articles reveals that they are limited to what may be described as one leg of a three-legged stool. That is, the possible mechanisms of injury have not been integrated with the shoe-surface interface aspects of the problem and, except for two reports, environmental considerations were not included.[16, 29] With regards to the mechanism of injury issue, foot fixation is clearly associated with rotatory stresses resulting in both knee and ankle joint injuries. As well, injury resulting from exogenous forces such as a valgus strain combined with foot fixation is well recognized. Of note, however, recent understanding of non-contact anterior cruciate ligament injury suggest an axial loading force is the primary mechanism and is probably independent of shoe-surface interface foot fixation.[32] As noted, ambient temperature has been observed to have a direct effect on both the shoe-surface interface release coefficient and the anterior cruciate ligament injury rates.[18, 29]

Most authors agree that the football-induced knee injury problem is complex and multifaceted. We believe that the shoe-surface issue was basically resolved in 1971. That is, maximum cleat length of ½ inch on all surfaces and non-rubber synthetic sole shoes on artificial surfaces. However, knee injuries continue to occur and we believe a prospective attempt to collect data to develop an injury predisposition profile consisting of all possible predisposing factors: shoes, surfaces, injury mechanisms, environmental, and player demographics is indicated.

Conclusion

The shoe-surface variables are important but not the only factors responsible for the occurrence or prevention of athletic-induced knee injuries.

The limitation of football shoe cleat length to ½ inch on all surfaces, as mandated by both the NFHSA and the NCAA in 1974, has withstood the test of time.

The initial reports from the early 1970s recommending multicleated soccer type shoes with polyurethane/polypropylene soles for use on synthetic surfaces has also withstood the test of time.

It is recommend that there be data collection to develop an injury predisposition profile consisting of all possible factors: shoes, surfaces, injury mechanisms, environmental, and player demographics.

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Medical Student Research Project

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Tibial Plateau Fracture Outcomes Following Treatment with Plexur M Bone Graft: A Retrospective Case Review

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Abstract

Objective: To investigate the effectiveness of Plexur M (Medtronic, Minneapolis, MN) bone graft substitute in filling subchondral depression defects of the fractured tibial plateau by comparing its subsidence and complication rates to allograft and calcium phosphate substitutes.

Design: Retrospective chart and radiographic review

Setting: Level 1 regional trauma center

Patients and Methods: One-hundred-twenty-six patients with tibial plateau fractures treated with open reduction internal fixation and bone grafting between January 1st, 2005 and January 31st, 2012 were identified. Eighty-three patients were lost to follow-up and excluded. The remaining 43 patients had at least six months of follow-up with available radiographs, thus were included as study subjects. Thirteen patients received Plexur M bone graft, 14 received calcium phosphate bone grafts, and 17 received allograft. Follow-up digital radiographs were analyzed and subsidence of the articular surface at the fracture site was determined using electronic calipers. The primary outcome measure was subsidence. Secondary outcome measures included infection and reoperation.

Results: Individual comparisons among the three groups revealed that fractures treated with Plexur M had a 14% incidence of subsidence (2/14) compared to a 24% incidence (4/17) in the allograft group and a 15% incidence (2/13) in the calcium phosphate group. The observed differences in rates were not significantly different. Other complications, such as infection and reoperation, did occur but there were no statistically significant differences amongst the groups.

Conclusions: Plexur M is a viable alternative for the treatment of bone voids as a result of tibial plateau fractures compared to cancellous allograft and calcium phosphate.

Introduction

Displaced tibial plateau fractures, particularly those leading to joint instability, are frequently treated surgically, often with open reduction and internal fixation.[1] In the case of depressed fractures, bone graft material (in combination with open reduction and internal fixation (ORIF)) is commonly placed in the depression to fill any defects, provide structural support, and to serve as a scaffold for bone regeneration.[2] It is desirable that bone graft materials here provide immediate structural support, are easy to use by the surgeon intraoperatively, have minimal complications, and allow themselves to be replaced with bone over time.

Multiple bone graft options are available. Options include autografts as well as a variety of synthetic products. Several studies have shown that calcium phosphate graft substitutes are equal or superior to autograft based on clinical outcomes and strength testing, without complications of donor-site pain or infection.[3–6] Calcium phosphate is a commonly accepted bone substitute in the treatment of tibial plateau fractures in addition to allograft.[7,8] Calcium sulfate materials can occasionally be difficult to use intraoperatively due to their material properties. Other bone grafts, such as calcium sulfate materials, have shown extremely rapid resorption and problems with sterile drainage from the surgical incisions.[9,10]

Plexur M graft material (Medtronic, Minneapolis, MN) is a novel graft material consisting of milled cortical allograft bone fibers in a bioresorbable carrier (poly (DL Lactide-co-glycolide) and a calcium porogen). Most importantly, this particular graft material is moldable, and hardens intraoperatively, which we have found to be particularly useful for the surgeon trying to provide subchondral support for a depressed fracture. The objective of this study was to investigate clinical safety and efficacy of Plexur M in the treatment of depressed tibial plateau fractures.

Methods

After appropriate institutional review board approval was obtained, a retrospective review of patient charts was performed. Inclusion criteria consisted of patients admitted to our institution between January 1st, 2005 and January 31st, 2012 with tibial plateau fractures treated with ORIF and bone graft. Patients with less than six months of follow-up were excluded from the present study. Patients were divided

into three groups based upon the treatment used: Plexur M bone graft substitute (Medtronic, Minneapolis, MN), allograft, or calcium phosphate bone graft substitute. Calcium phosphate bone graft substitutes included: Vitoss (Orthovita, Malvern, PA), Hydroset (Stryker, Kalamazoo MI), and chronOS (Synthes, West Chester, PA).

Patient outcomes were determined by reviewing the hospital's electronic medical record system. Data collected via patient chart review included patient age, gender, date of surgery, date of follow-up, surgical fixation method, tobacco use status, diabetes status, compartment syndrome in the surgical leg requiring fasciotomy, as well as open or closed fracture status. X-ray review by two orthopedic surgeons was performed to determine the Schatzker fracture classification (Table 1).

Table 1. Schatzker Fracture Classification System[12]

Schatzker Fracture Classification	Description
Type I	Split or wedge fracture of the lateral tibial plateau, usually occurring in young patients with strong bone
Type II	Lateral split-depression fracture in which the medial aspect of the split is depressed
Type III	A centrally-located depression, usually resulting from lower-energy injuries
Type IV	Fracture of the medial tibial plateau
Type V	Bicondylar fracture of both the medial and lateral tibial plateau
Type VI	Tibial plateau fracture with an associated proximal shaft tibia fracture

Post-surgical subsidence and complications were assessed as primary and secondary outcomes of this study, respectively. Complications were defined as negative outcomes resulting in surgical intervention that could potentially be attributed to the bone graft or bone graft substitute. Included in this outcome measure were infections, which were defined as any peri- or intra-articular infection that, again, required operative intervention (superficial wound infection, septic arthritis, osteomyelitis, etc.). Individual complications are detailed by graft type and Schatzker class in Table 3.

Experimental methods to assess fixation were modeled after previous studies of subsidence in depressed tibial plateau fractures.[7, 13, 14] Subsidence was measured by two separate reviewers (radiologist and orthopedic surgeon) without knowledge of the treatment group, using radiographs viewed at a minimum magnification of 200%. Measurements were taken from the intact articular surface to the surface of the depressed fragment (Figure 2). Repeated measurements were averaged. Measurements were taken immediately postoperatively, and again at a minimum of six months. The difference between the measurement at the time of surgery and at six months was calculated and used to determine subsidence. A distance of 2 mm or greater between the surfaces was classified as positive subsidence.[7]

Radiographs were viewed using a Picture Archive and Communication System (PACS). Radiographs taken at the time of surgery were measured directly using electronic calipers. In some follow-up radiographs where the images were digitized, a known measurement (usually the distance between the most lateral and medial edges of the femoral condyles) was used for calibration in order to measure the subsidence. Figures 1 and 2 are examples of negative and positive evidence of subsidence, respectively. These images were provided to the independent reviewers for reference. Figure 2 also provides landmarks used for radiographic determination of subsidence.

Statistical analyses were performed using univariate logistic regression for each potential factor related to outcomes (complications, infection, subsidence, etc.). Follow-up pairwise comparisons were made among the three treatment groups. A Bonferroni adjustment was made to p-values to account for experiment-wise type 1 error. Individual rates for complication and subsidence were compared using Fisher's exact test.

Results

Retrospective review of tibial plateau fractures at our institution treated with ORIF from January 1st, 2005 to January 31st, 2012 resulted in a total of 273 patients, 43 of whom were included in this study. Of the original 273 patients, 125 underwent ORIF with bone graft or bone graft substitute and had bone graft material information available. After six months, 82 patients did not have radiographs in our PACS system, leaving 43 patients for analysis. They were categorized into three groups as follows: 13 patients received Plexur M bone graft substitute, 13 patients received calcium phosphate bone graft substitute, and 17 received allograft. Average subsidence and standard deviation of the three groups are summarized in Table 2.

Following logistic regression for an overall effect, pairwise group comparisons among the three treatment groups were performed. Fractures treated with Plexur M had a 14% incidence of subsidence (2/14) compared to a 24% incidence (4/17) in the allograft group and a 15% incidence (2/13) in the calcium phosphate group. The observed differences in rates were not significantly different.

Other complications, as previously defined, did occur but were not statistically significant between groups. These comparison were allograft versus calcium phosphate ($p = 0.56$), allograft versus Plexur M ($p = 0.88$), and calcium phosphate versus Plexur M ($p = 0.68$).

Potential confounding factors, including age, gender, smoking, diabetes, compartment syndrome and open fracture status were analyzed to see if there were any between-group differences that could be attributable to these underlying comorbidities. Age was not associated with a greater amount of subsidence ($p = 0.323$) or complications ($p = 0.512$). Gender was not found to impact subsidence ($p =$

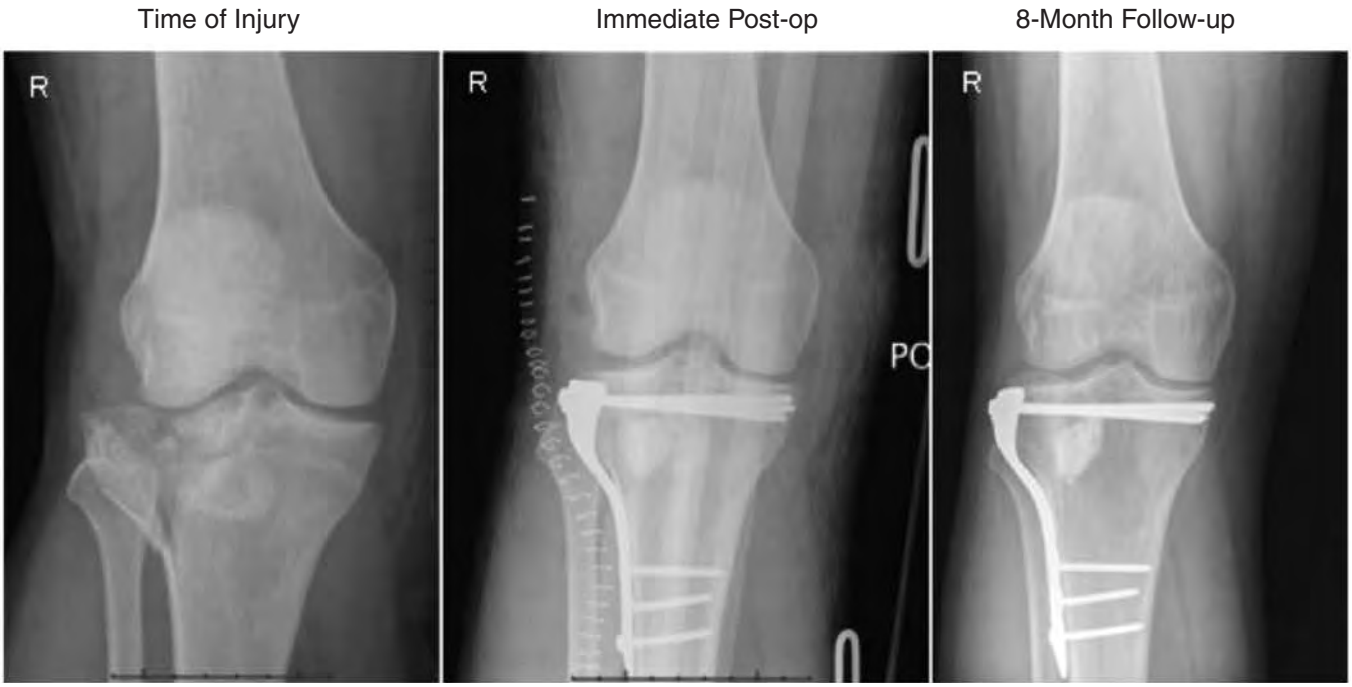

Figure 1. Example of negative read for subsidence.

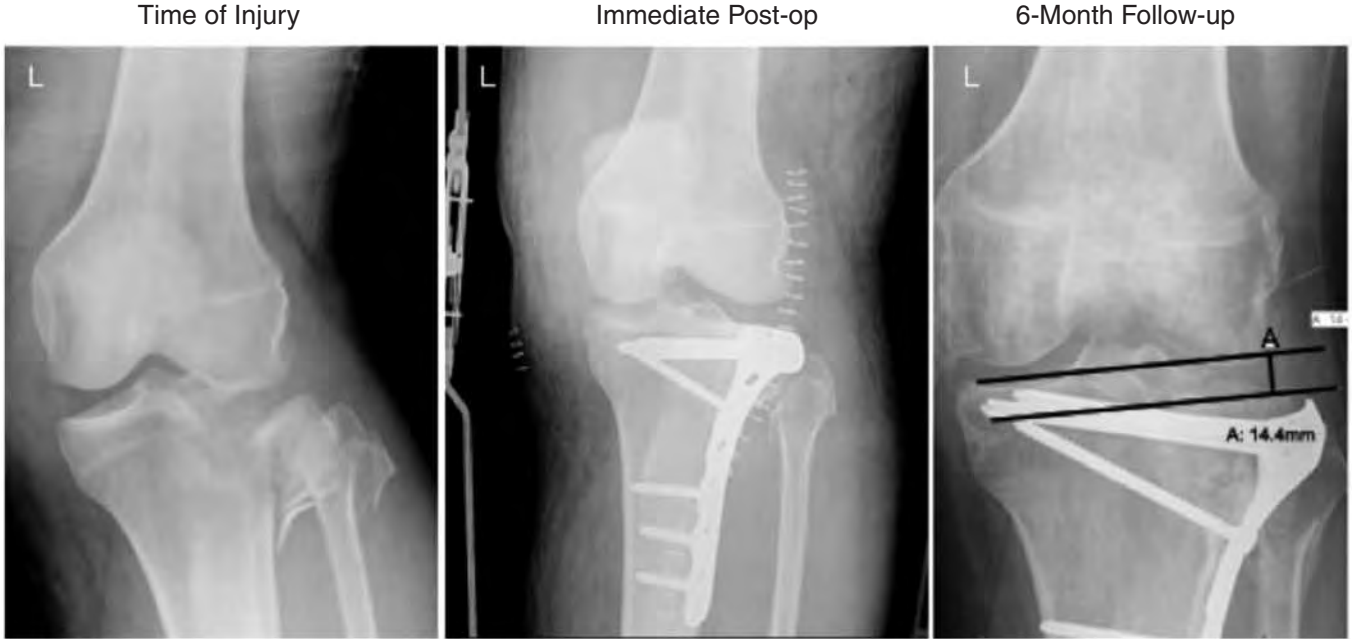

Figure 2. Example of positive read for subsidence. The right pane also illustrates the landmarks used for radiographic determination of subsidence.

0.805) or complications (p = 0.609). Between the allograft, calcium phosphate and Plexur M groups, the number of smokers was 10, four, and seven respectively and the number of diabetics was one, one, and one. Smoking was not found to impact complication (p = 0.907) or subsidence (p = 0.402). Neither compartment syndrome (p = 0.492) nor open fractures (p = 0.973) were found to correlate with complications rates.

Other complications leading to re-operation noted in the Plexur M group included two cases of arthrofibrosis, one case of osteomyelitis, and one case of aseptic knee pain (Table 3).

Discussion

Patients with depressed tibial plateau fractures treated with Plexur M were found to have a lower incidence of subsidence than patients treated with allograft and an equal rate to those treated with calcium phosphate bone graft substi-

tute. Patients treated with Plexur M were found to have a lower incidence of re-operation than patients treated with allograft but a higher rate than those treated with calcium phosphate (Table 2). Plexur M had the lowest infection rate (8%) of all the groups, though this was not statistically significant when pair-wise group comparisons were performed for overall complication rate, as noted in the results above. In general, the relatively high incidence of infection in this study was somewhat higher than expected. That said, the majority of patients who underwent ORIF of a tibial plateau fracture did not have sufficient radiographs available for six-month follow-up in our PACS system and were thus excluded from the study. Many of those patients had radiographs done elsewhere during follow-up due to insurance restrictions, or were not seen in follow-up long enough for meeting the six-month requirement. It could be speculated that whereas patients who did well early in their postoperative course did not continue to follow up, patients with infections required prolonged follow-up and multiple radiographs.

There were two cases of arthrofibrosis associated with the use of Plexur M bone graft substitute. While there is not enough data to support or refute the association of increased peri-articular scarring with the use of Plexur M bone graft, further studies are needed to elucidate the significance this finding.

Table 2. Number of Cases (Percentage) with Either Subsidence Greater than 2 mm or a Complication (as Defined in the Text) Divided by the Total Number of Cases in Each Group

	Subsidence	Complication	Infections
Allograft	4/17 (24%)	7/17 (41%)	6/17 (35%)
Calcium Phosphate	2/13 (15%)	4/13 (31%)	3/13 (23%)
Plexur M	2/13 (15%)	5/13 (38%)	1/13 (8%)

Infections requiring operative intervention are listed here as a subclass of overall complications. For rate of subsidence greater than 2 mm, allograft vs calcium phosphate, p = 0.5860; allograft vs Plexur M, p = 0.5860; calcium phosphate vs Plexur M, p = 1.0000.

Infection and subsidence rates in this study were compared with available literature for the use of allograft and calcium phosphate bone substitutes. Quoted infection rates for calcium phosphate and allograft from a recent meta-analysis are 3% and 9%, respectively.[15] In 2003, Simpsons and Keating reported loss of reduction in 61% of autograft patients and 23% of calcium phosphate patients with lateral tibial plateau fractures,[16] and in 2001 Keating, Hajducka, and Harper reported loss of reduction in 16% of patients treated with calcium phosphate bone graft substitute.[17]

The results of the current study suggest that Plexur M bone graft is a safe and viable alternative to allograft and calcium phosphate bone graft substitutes for treatment of bone voids as a result of tibial plateau fractures. It is not associated with a statistically significant higher incidence of subsidence or rate of other complications including infection or reoperation when compared to allograft and calcium phosphate.

The material properties of Plexur M are arguably preferable to calcium phosphate, synthetic graft substitutes and certain types of allograft because of its moldability, machinability, and set-time. It also avoids the complications of autografts such as donor-site pain or infection, as well as the high incidence sterile drainage that is reported with calcium sulfate grafts, thus making it a desirable material to use in the fixation of tibial plateau fractures.[9–11]

This study is not without its limitations. It is a retrospective chart and radiographic review with a large proportion of patients without sufficient radiographic follow-up in our PACS system for review. The minimum acceptable follow-up period of six months is also relatively short and inconsistent among the analyzed patients. However, follow-up time was not proven to impact subsidence or complication rate.

The patients in this study represent individuals with tibial plateau fractures treated with ORIF and bone grafting over a consecutive eight-year period. It is noted that most patients

Table 3. Summary of Complications and Associated Interventions by Graft and Fracture Type

Graft Type	Case No.	Schatzker Type	Complication; Surgical Procedure
Allograft	1	2	Abscess; debridement
	2	2	Flexion contracture, heterotopic ossification; osteotomy
	3	2	Septic arthritis; debridement, implant removal
	4	2	Wound infection, sinus tract; multiple debridements, implant removal, local flap, skin graft
	5	5	Wound infection; debridement, implant removal
	6	6	Wound infection; debridement, implant removal
	7	6	Wound infection; debridement, implant removal
Calcium phosphate bone graft substitute	1	2	Degenerative joint disease; total knee arthroplasty
	2	5	Persistent wound drainage; debridement
	3	6	Wound dehiscence, septic arthritis; incision and drainage, implant removal, local flap, skin graft
	4	6	Septic arthritis; incision and drainage, implant removal, skin graft
Plexur M bone graft substitute	1	2	Wound infection; implant removal
	2	5	Arthrofibrosis; manipulation, lysis of adhesions
	3	5	Arthrofibrosis; Judet qudricepsplasty
	4	6	Culture negative effusion, persistent knee pain; incision and drainage
	5	6	Osteomyelitis; debridement, implant removal

received allograft near the beginning of the study period, while Plexur M was used more often in the latter part of the study. Calcium phosphate substitutes were used with greatest frequency in the middle of the study period. This is an unintended consequence of the retrospective design of this study and purely reflects the choices made by the surgeon at the time the patients were treated. Plexur M was approved by the FDA in March of 2008 and was not used in any of the patients in this study until after that date.

Subsidence measurements were made using digital x-rays. Some error was noted due to slight variation in the angulation and rotation of the radiographs between immediate postop and follow-up x-rays. Only anterior-posterior images were used to record these measurements. In order to account for this variation and any error in calculating the pixel to millimeter conversion for radiograph reading of subsidence, subsidence measurements less that 2 mm were not considered, similar to previously reported studies.

Larger prospective studies with longer, more consistent follow-up are needed to reliably compare Plexur M with its alternatives and to further delineate the risk factors associated with its use.

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Medical Student Research Project

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Treatment of Articular Fractures with Continuous Passive Motion

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Key Points

• In animal studies, CPM has been shown to improve cartilage healing after injury compared with immobilization. Human studies have also shown the improved rate of hemarthrosis clearance with CPM compared with immobilization.

• Clinical studies of CPM have mostly come from the total knee replacement literature. Its use in joint replacement (which does not rely on cartilage repair), however, only partially translates the potential benefits of CPM.

• CPM has been used extensively in the postoperative care of articular fractures treated with ORIF, a natural extension of the early basic science studies' purported clinical use. It is felt to help improve cartilage repair, range of motion, and clearance of hemarthrosis. However, little attention has been paid specifically to CPM as a treatment modality.

• Better clinical studies of CPM as a treatment modality for articular fracture management are warranted to determine its potential benefits, and to more clearly specify parameters for its use in specific clinical scenarios.

Synopsis

This article presents a review of the basic science and current research that exists on the use of continuous passive motion therapy following surgery for an intraarticular fracture. This information will be useful for surgeons in the postoperative management of intra-articular fractures in determining the best course of treatment to reduce complications and facilitate quicker recovery.

Introduction

Primarily used to reduce joint stiffness following joint surgery or trauma, continuous passive motion (CPM) therapy works to counteract the pathological stages of joint stiffness: bleeding, edema, granulation tissue and fibrosis.[1] This post-operative therapy has been utilized for a variety of orthopedic surgeries including the management of total knee arthroplasty, fracture repair, rotator cuff repair, hand rehabilitation, and ACL reconstruction rehabilitation.[2–4] Salter pioneered the use of CPM in the 1980s after observing the therapy's ability to stimulate articular cartilage healing and prevent complications caused by immobilization post-injury in rabbit models[1, 5] (Figure 1). Further animal studies went on to investigate continuous passive motion therapy's role in reducing joint stiffness following intra-articular injury.[6]

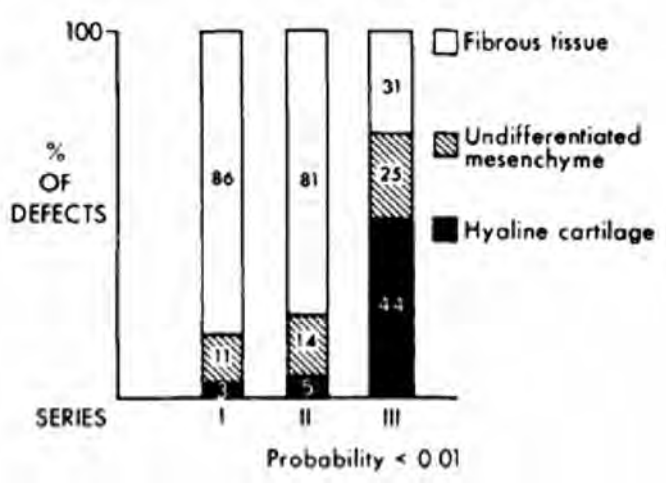

Figure 1. Improved cartilage repair seen with continuous passive motion from animal studies (from Salter et al.,[5] with permission from the publisher).

First index of healing: the nature of the reparative tissue at three weeks in the 36 defects in each of the three series in adult animals. The bars depict the percentages of the 36 defects in each series that exhibited predominantly hyaline cartilage, incompletely differentiated mesenchymal tissue, and fibrous tissue. The nature of the reparative tissue in the defects treated with continuous passive motion (Series III) is far superior to that after either immobilization (Series I) or intermittent active motion (Series II).

However, while studies conducted on animal models demonstrate a significant benefit in CPM use following injury, studies carried out in a clinical setting show more conflicting results.[6–9] The many variations in clinical CPM protocols could be partially to blame for this lack of agreement with no standard method of use dictating the number of degrees per day the machine should advance or the number of hours per day the treatment should last.[10] Despite this lack of conclusive evidence showing definitive benefits when used clinically, CPM therapy has become standard practice in many centers for post-operative treatment of many joint injuries. However, CPM therapy remains a highly-debated treatment with some recent studies highlighting the treatment's disadvantages such as the need for the patient to stay in bed, the increased costs of maintaining and operating the units, and the extra technical support patients require from their nurses.[11] With CPM's potential to facilitate faster recovery, shorten patients' lengths of stay, and as a result, reduce costs,

many hospitals could benefit from a definitive verdict on the effectiveness of CPM therapy.

Much of the clinical research focuses on the efficacy of this treatment in increasing range of motion and decreasing hospitalization time and post-operative complications following total knee arthroplasty when compared to a regimen focused on physical therapy alone.[12] However, very little research exists on the use of CPM for the management of articular fractures. Many articular fractures, such as tibial plateau fractures, can develop stiffness as a sequelae.[13] Recovery from this fracture, as an example, is also often further complicated by significant soft tissue injury and can involve collateral ligaments and the anterior and posterior cruciate ligaments.[14] In addition, the significant amount of bleeding associated with the soft tissue injury and fracture of the proximal tibial metaphysis can lead to compartment syndrome, and post-operative complications such as deep vein thrombosis can develop.[14] Given the nature of these possible complications and the proposed benefits of CPM which include the potential to decrease hemathrosis, and decreasing the incidence of deep vein thrombosis in trauma patients, CPM therapy has the potential to offer many advantages post-operatively.[15] However, our understanding of the efficacy of CPM in the management of articular fractures is not well understood, as few studies have specifically examined CPM in this setting, although its use in total knee arthroplasty patients has been examined.

In this paper, we review the rationale and basic science evidence for CPM in articular injuries and review the clinical evidence in the postoperative treatment of intra-articular fractures.

Continuous Passive Motion Therapy: Investigating Potential Benefits

The historical progression to the development of CPM started off with early research done through the 1950s, 60s and 70s which demonstrated the effects of immobilization compared to joint motion on articular cartilage. These early studies provided evidence of the harmful effects of immobilization which caused deterioration and articular cartilage loss in animal models. Fibrocartilage replaced the articular cartilage, and adhesions developed after immobilization, and after 30 days of immobilization, the cartilage damage could not be reversed like it could be with changes seen in soft tissue. However, this damage could be prevented if immobilization was limited and early exercise was emphasized.[16]

Salter pioneered the use of continuous passive motion through his early work starting in the 1970s. He and his colleagues conducted numerous studies on rabbit models and specifically looked at CPM therapy in improving the outcomes in synovial joint injuries. Salter compared CPM therapy to immobilization in his rabbit models, starting CPM immediately after surgery and continuing non-stop for one to four weeks. He found that the new therapy stimulated healing of the articular cartilage and led to faster and better healing when compared to both immobilization and limited active motion. In looking specifically at intra-articular fractures, CPM therapy stimulated articular cartilage growth and, therefore, was protective against degenerative arthritis development and resulted in better surgical wound healing.[1] In his 1984 publication, he summarized his findings as well as presented an early report on the clinical applications of continuous passive motion. In this retrospective study, he observed the effects of continuous passive motion for various joint injuries of the hip, knee, ankle, elbow and finger. Salter's early research summarized in this case study support the use of CPM therapy in preventing joint stiffness and facilitating healing, specifically for articular cartilage. Early success with rabbit models in the treatment of full-thickness articular cartilage defects, intra-articular fractures, acute septic arthritis, MCL reconstruction, and lacerations of tendons encouraged the use of CPM therapy in clinical applications. The nine cases Salter reviewed utilized CPM therapy for a variety of injuries (two intraarticular femur fractures, a patellar dislocation, two elbow fractures, one acetabular fracture, one intraarticular finger fracture, one hip infection, and one case of arthrofibrosis). This is clearly a very heterogenous group of cases to comprise this study. The protocol for CPM therapy followed by and recommended by these case studies indicates immediate post-operative use, starting in recovery and continuing without prolonged interruption for one week at one cycle per 45 seconds. Success in the clinical setting mimicked the early experimental success with patients treated with the CPM therapy reporting that they tolerated the treatment well and maintained the increased range of motion achieved through their respective surgical procedures. In addition, the case studies showed no CPM-related complications, periods of prolonged hospitalization or increase in patient pain or discomfort.[17]

Basic Science Evidence

Tendon Strength: Early reports of success with CPM therapy motivated further studies and its benefits on animal models. Loitz et al. used rabbit models to investigate the effect of continuous passive motion versus immobilization on the mechanical properties of tendons deprived of normal weight-bearing stimulation.[18] This design attempted to mimic the state of tendons following an injury, such as a fracture, which prohibited normal weight bearing. In this experiment, the 26 rabbit models were divided into two groups: an eight-member control group received no treatment and an experimental group of 18 rabbits received CPM to one ankle and immobilization for three weeks to the other after receiving an articular injury to both ankles without injury to surrounding tendons. The researchers then tested the collagen composition of the tendons and the mechanical properties. The thickness of the dissected tendons was measured with a digital micrometer and the mechanical strength by a servocontrolled electromechanical materials testing system. In addition, samples of the tendons were analyzed

for hydroxyproline content. While the cross-sectional area of control and experimental tendons were similar, averaging 0.9 $mm^2 \pm 0.2$ mm^2, the linear load for the immobilized tendons was found to be 16% less than the control tendons. The value for the CPM treated tendons was similar to that of the control tendons. In addition, the study found a significant difference in the strength of the control and immobilized tendons, with control tendons 20% stronger than immobilized and 16% stronger than CPM treated tendons. Looking at tensile strength, they found the control and CPM tendons to be similar with immobilized tendons showing 25% less strength than both. The composition of the tendons between the groups also differed, though not significantly; the hydroxyproline concentrations of the CPM tendons was 6% greater than both the control and immobilized tendons demonstrating the increased healing taking place. Overall, the study found the control tendons, as expected, were the strongest of the three while the tendons coming from the immobilized limbs the weakest. The tendons taken from injured joints and treated with CPM therapy fell in the middle and therefore demonstrated the role of CPM therapy in countering the harmful effects of short-term immobilization.

Joint Motion: Also comparing CPM therapy to immobilization in an animal model, Namba et al. focused on treating post-traumatic joint stiffness.[6] This experiment again utilized rabbit models. After sustaining intra-articular ankle injuries in two of their ankles, the 10 rabbits received the two different treatments: one ankle was treated with immobilization in a cast at 90 degrees flexion and the other with a CPM machine for three weeks at 24 hours a day. Evaluating joint stiffness specifically, Namba found that at three weeks, the immobilized joint was 2.6 times stiffer than pre-injury levels while the CPM treated joint showed no significant difference when compared to pre-injury levels. While CPM helped maintain joint function post-injury, no significant difference was found between the groups in terms of joint swelling.

Wound Healing: The effect of CPM therapy on wound healing is another important consideration in evaluating the treatment. Van Royen et al. compared the effects of CPM to cast immobilization in post-operative wound healing.[19] His histological and functional tests found that CPM treated wounds were significantly stronger and the histological structure of the collagen fibers showed better organization in the CPM treated wounds. In this experiment, van Royen used rabbits as his animal models and made skin incisions around the patella and into the knee joint. He then divided the rabbits into two groups: the immobilization group's knees were held at 80 degrees flexion for three weeks, while the CPM group received the therapy for the same duration of time. After three weeks of treatment, van Royen collected samples from the healing wound to observe the collagen organization and test the strength. Finding improvements in the strength and healing of the CPM treated wound, the study concluded that the added tension from the therapy improved the healing of the wounds.

Tissue Repair and Regeneration: Beyond being used to reduce joint stiffness and increasing tendon strength after injury, two studies done by O'Driscoll et al. and Kim et al. looked at CPM therapy's potential to stimulate neochondrogenesis and peripheral nerve repair.[20, 21] Using animal models, O'Driscoll found that a periosteum graft put into the knee joints of 30 rabbits showed evidence of articular cartilage growth after two weeks in the CPM-treated group when compared to the immobilized group. The CPM group had significantly more cartilage than the immobilization group: 59% of the graft consisting of cartilage in comparison to 8% respectively. Using animal models, Kim et al. found no statistically significant difference between the CPM group and the immobilization group in average nerve conduction and average fiber density following nerve transection. Therefore, as previous research demonstrating the benefits of CPM therapy suggest, CPM has the potential to stimulate cartilage growth, but does not appear to have any effect on nerve repair.

Frequency and Treatment Parameters: Basic Science and Clinical Evidence

As mentioned previously, CPM is used frequently by clinicians, but there are few guidelines for the timing of treatment, frequency, duration, and other treatment parameters. Studies done by Gebhard et al. and Shimizu further demonstrated the benefits of CPM therapy, and also set forth more specific parameters of use.[22, 23] Both studies used animal models to find the ideal number of hours per day needed to get the benefits of CPM therapy. Another study done by Takai et al. looked at the effect of the frequency of the CPM machine cycles on the healing of tendons.[24] His study indicated that the frequency might allow for a shorter duration of use with the same benefits.

Investigating duration of treatment, Gebhard et al. looked specifically at joint stiffness, muscle mass, bone density and regional swelling following intra-articular injury.[22] Again using rabbit models, 30 rabbits received an intra-articular injury by drilling a tibial pin into their ankle joints. The rabbits were then divided into five groups to receive four, eight, 12, 16 or 24 hours of CPM each day on one injured ankle and immobilization on the other ankle. When not undergoing CPM therapy, the rabbits were immobilized. After three weeks, the rabbits were evaluated. In looking at each of the parameters measured, Gebhard and colleagues found that only the rabbits treated with either 16 or 24 hours of CPM therapy saw any benefits in reducing joint stiffness. In fact, rabbits that received the shorter duration CPM therapy actually showed a worsening in mobility, with the CPM-treated limbs as much as four times stiffer than immobilized limbs. In terms of swelling, the 24-hour group was the only to show any benefit, though the decrease was not significant. All of the CPM groups increased in muscle mass being 13% greater than the immobilized limb. However, bone density went against the previous trend with longer CPM duration having more benefits, and an increase in bone density was observed

only in those treated with 12 hours or less of CPM therapy. Bone density data showed a statistically significant inverse relationship between duration and bone density: those treated with 12, eight and four hours of CPM per day had progressively more bone density than those with immobilization or 16 and 24 hours CPM per day. Through his experiments with animal models, Gebhard demonstrated the differing effects of CPM therapy on different tissue types and recommended that the therapy be employed for at least 16 hours per day to prevent stiffness, reduce swelling and increase muscle mass without having detrimental effects on bone density.

Shimizu et al. also focused on the dose-response relationship of continuous passive motion therapy.[23] The study again utilized rabbit models, and in both knees of all 34 rabbits, they exposed the knee joint and dislocated the patella as well as put holes in the articular bone of the femur. Post-operatively, the rabbit subjects were divided into groups based on the number of hours per day they would receive CPM treatment. All CPM machines were set at the same arc and cycle duration and the same immobilization cast, set at 90 degrees flexion, was utilized. Ten rabbits received CPM therapy 24 hours a day, six rabbits received CPM for eight hours a day and immobilization for the remaining time on one joint and CPM for two hours a day with immobilization on the other, seven rabbits remained immobilized for the full two weeks, nine rabbits were allowed normal cage activity for the full duration, and five rabbit knees received immobilization for one week followed by one week of 24-hour-a-day CPM therapy. After treatment, the rabbits were allowed normal cage activity for an additional five weeks before being evaluated. Shimizu and his colleagues examined mobility, histological features, and the extent of cartilage repair. While no significant difference was found in passive mobility, visual and histological analysis of the joints treated with CPM for 24 hours per day and for eight hours a day showed better repair and healing in comparison to the immobilized and cage activity groups. In addition, the CPM conducted after one week of immobilization did not overcome the initial harm caused by immobilization. The findings led the group to recommend that continuous passive motion therapy should be started as soon as possible and that the most favorable results would be achieved when CPM is carried out for 8–24 hours a day, though brief periods of immobilization left no ill effects.

Takai and co-authors suggested that the cycles per minute of the CPM machine might allow for shorter durations of use.[24] In his study, he used dogs as the animal model and following flexor tendon injury and repair, the dogs were divided into two treatment groups. One group received CPM therapy for five minutes per day at 12 cycles per minute while the second group got the same therapy for 60 minutes a day at one cycle per minute. These parameters worked out to the same number of cycles per day, but at different frequencies. After harvesting the tendons, the gliding function and strength of the tendons were evaluated at three weeks and six weeks. While the function of the tendons was the same for both groups, the tendon strength of the higher frequency group was significantly greater. Therefore, while duration of CPM therapy is an important variable in the effectiveness of the therapy, the frequency of cycles might have an even greater effect on outcome.

Another important parameter that, like the others discussed above, remains unstandardized is the number of days the patient must use the CPM machine in order to see any benefits. Several clinical studies have looked at this variable. One study determined that three days of CPM therapy was sufficient after looking at effects of the therapy on two groups of patients.[25] The first group experienced post-operative knee or elbow stiffness that existed for some time before therapy while the second group used CPM therapy immediately following the injury. After only three days of therapy, the first group saw significant improvements in range of motion which was maintained on follow-up while the second group regained their pre-injury range of motion with the reduced CPM therapy duration as well. Other studies looked at patients following total knee arthroplasty. In a study by Bennett et al., an early flexion CPM group started a group at a greater degree of flexion in recovery and continued the treatment for seven days comparing the outcome with a standard CPM group and a control with no CPM therapy.[26] Overall, the early flexion group showed significantly greater range of motion in early on, but the groups showed similar results after one year of follow-up. Similarly, other studies comparing the number of hours per day dedicated to CPM therapy found no significant difference in the range of motion of the total knee arthroplasty patients.[10, 27] Overall, the literature suggests that no consensus has been reached on the optimum number of hours per days and the number of days the continuous passive motion therapy should be administered.

Mechanisms of Action

While these previous studies illustrated the potential benefits of CPM therapy, O'Driscoll et al. investigated the mechanism behind the beneficial effects of continuous passive motion therapy.[7] O'Driscoll hypothesized that clearance of this blood from the joint with CPM can facilitate recovery and reduce stiffness. In his experiment using rabbit models, seven of the 16 received labeled erythrocyte injections into their knees and were scanned that day and subsequently on days one, two, three, four, and seven. Nine rabbits were injected with unlabeled blood as controls. After the injections, one knee was immobilized while the other underwent continuous passive motion therapy continuously for seven days. After seven days of treatment, the knee joints of the rabbits were dissected and examined. The results from the scans taken during the treatment showed that after 48 hours of CPM therapy, the knee synovial fluid was clear in comparison to the fluid taken from the immobilized joint which was bloody. Overall, the rate of clearance was twice as fast

in the CPM-treated joint than in the cast-immobilized joint with the clearance of 50% of the blood occurring in 2.2 days compared to 5.5 days respectively (Figure 2). In looking at the joint after the seven days, 7.1% of the original injected number of erythrocytes were found in the CPM-treated knee in comparison to the 13.2% found in the immobilized knee. The authors explained this difference by postulating that during the continuous passive motion treatment, the intra-articular pressure in the joint is raised and lowered creating a pumping effect that aids in clearance.

By measuring the intra-articular pressure of a human knee during CPM therapy in a separate study, Pedowitz and colleauges supported the hypothesis put forth by O'Driscoll.[28] In a study with 16 patients, the CPM machine was set at zero to 90 degrees of flexion with one cycle per 150 seconds. After taking pressure measurements at full extension and flexion for three complete cycles for 90 minutes, the researchers found that the pressure was the most at the extremes of joint flexion and extension. The minimum pressure occurred at 30 to 60 degrees of flexion (Figure 3). These cyclic pressure gradients both aid in fluid clearance and helped stimulate tissue healing, explaining the benefits seen with CPM therapy.

Clinical Evidence for CPM in Articular Fracture Management

Clinical use of CPM has been investigated, with relatively mixed results, in the total knee arthroplasty literature. But the full benefit of CPM would theoretically be seen with articular cartilage injury treatment, in accordance with the animal data discussed above. For many periarticular fractures, early motion is indeed emphasized to prevent "fracture disease," as popularized by the AO movement. But specific details about the clinical efficacy of CPM machines for management of particular articular fractures with regard to the optimal timing, duration, frequency, and motion parameters is not well studied. In this section, we will review the available clinical literature in an attempt to address this. However, although CPM is used frequently for these injuries, few studies actually specifically investigate CPM.

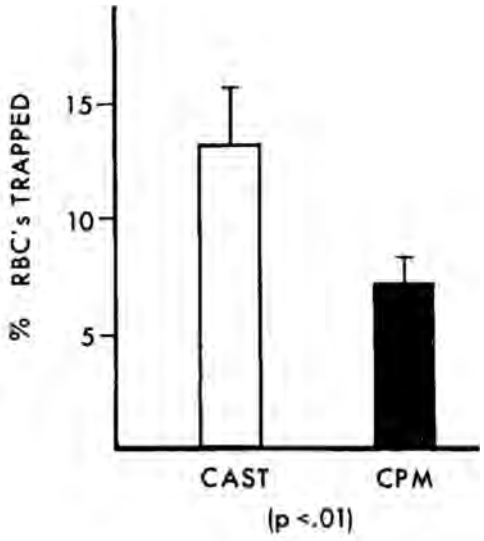

Figure 2. Clearance of hemarthrosis with CPM compared with cast immobilization of the knee (from O'Driscoll et al.,[7] with permission from the publisher).

The bars represent the percentages of injected ^{111}In-labelled erythrocytes that remained trapped in the synovium after seven days. Treatment by continuous passive motion decreased this trapping by approximately 50%. Values are expressed as mean ±1 standard error of the mean.

An Example of the Benefits of Early Motion: Tibial Plateau Fractures

Tibial plateau fractures represent a periarticular fracture group for which surgery is frequently done, and early motion is typically recommended. Gausewitz et al. reviewed the treatment of 122 acute tibial plateau fractures to determine the effects of early mobilization in rehabilitation. While the earlier studies demonstrated benefits for early motion, certain risks such as loss of fracture reduction, failure of internal fixation and compromised healing remained. Dividing patients into groups based on the amount of time they spent immobilized, Gausewitz and colleagues measured overall outcome by analyzing knee flexion, loss of fracture reduction, hospital length of stay and ligamentous laxity.[29] The review of patients and results revealed that patients treated without surgical intervention and immobilized for up to six weeks regained full range of motion. However, patients treated surgically with open reduction and internal fixation developed stiffness after only two weeks of immobilization. While the range of motion measurements were not statistically significant, after two to six weeks of immobilization, four of the 13 patients had flexion of less than 105 degrees and three had flexion contractures in comparison to the immediate motion group, which had only one flexion contracture and one patient with less than 105 degrees flexion. Despite the improvements in range of motion in the patients with shorter immobilization times, the patients' lengths of stay was found to be longer. The 23 patients with less than two weeks of immobilization stayed an average of 18.1 days in comparison to those with greater than two weeks of immobilization who stayed an average of 5.7 days. These values could be a slight misrepresentation of drawbacks to the treatment, however, since the longer stay of the patients with earlier mobilization was often due to the use of traction and a cast brace in comparison with patients who were simply discharged in a cast. The primary impact of the study was to highlight the benefits of early mobilization for surgically-treated fractures in recovering and maintaining range of motion.

Blokker et al. also related patient outcome to immobilization time in patients recovering from tibial plateau fractures.[30] In his review, he considered adequacy of reduction, immobilization time, fracture type, treatment method and overall result when evaluating patient outcome. However,

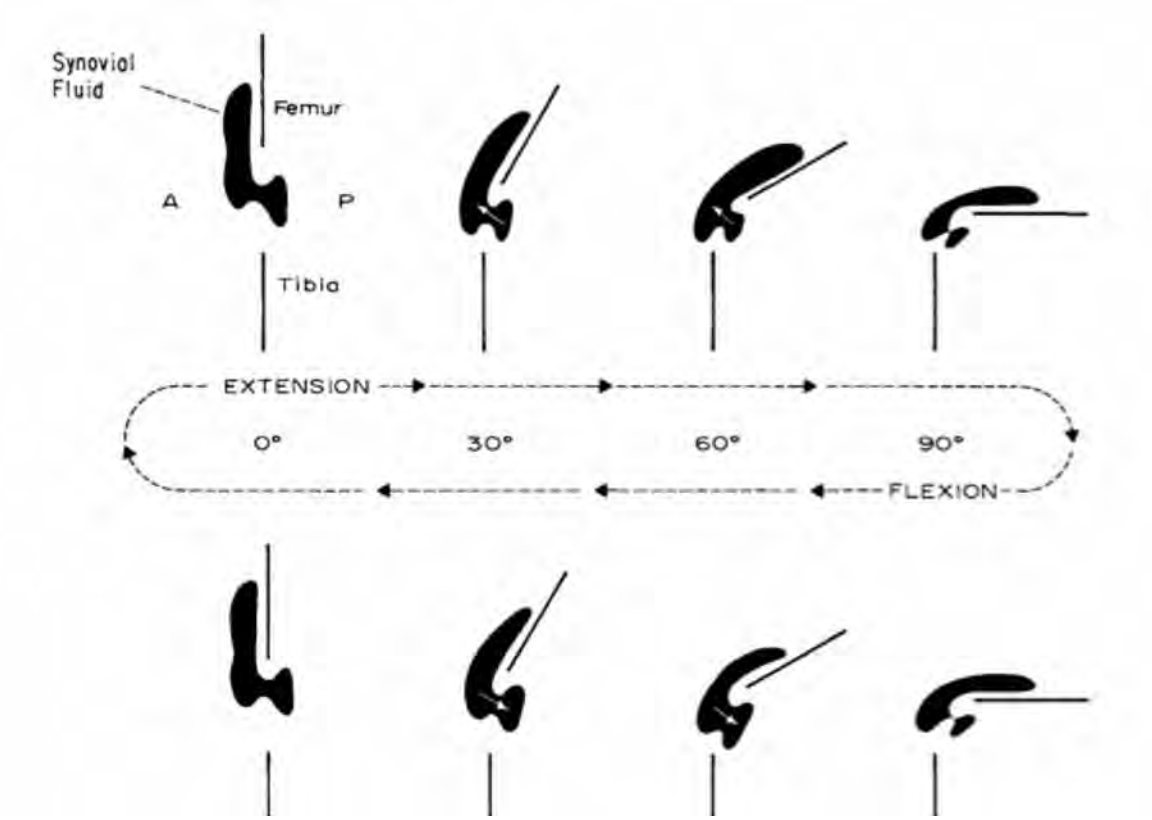

Figure 3. Intraarticular fluid flow during CPM, consistent with "physiologic compartmentation" of the human knee (from Pedowitz et al.,[28] with permission from publisher).

the results of his review could not support the earlier findings that demonstrated the strong relationship between early motion and better outcome in the patient. Patients were reported to have a satisfactory outcome if they achieved a range of motion of at least 90 degrees flexion, a lack of extension of less than 10 degrees and had returned to full activity with occasional mild pain or ache. 75% of the 60 patients evaluated reported a satisfactory result. He found no difference in outcome in patients who started knee movement in the first two weeks compared to those who started after. However, the authors also recognized that if motion was started by the first or second day of recovery rather than simply within the first two weeks, the results might have been more favorable for early motion. In addition, the assessment of satisfactory versus unsatisfactory result was done three years after the injury and no intermediate assessments were included. Therefore, the possibility existed that some more significant differences between the early mobilization group and the group immobilized for more than two weeks could have been observed.

In a similar study involving tibial plateau fractures, Lachiewicz et al. focused on cases treated surgically with open reduction and internal fixation.[31] This study reviewed the cases in order to determine the factors that influenced clinical and radiographic outcomes. The 43 fractures evaluated received several different post-operative treatments with 17 getting CPM therapy, 12 having a long-leg cast for six weeks because of ligament injuries, six in a knee immobilizer for six weeks, and the final 16 receiving instruction in range of motion exercises. However, with the focus of the study not being on post-operative treatments, the patients were not further divided up or analyzed based on these various treatments specifically. Overall, the authors found that 35 of the 43 had excellent results, with bicondylar fractures presenting the most difficulties. Patients who experienced this type of fracture were found to have an 18 degree decrease in range of motion in comparison to other patients. Interestingly, 10 of the 15 patients were in an immobilizer for more than three weeks. In addition, other patients treated postoperatively with an immobilizer for more than three weeks also experienced a decreased mean range of motion, measuring 14 degrees lower than those in an immobilizer for a shorter time period. This difference was found to be statistically significant. In summary, Lachiewicz demonstrates preliminary support for the use of early continuous passive motion in the treatment of tibial plateau fractures.

In a prospective study, Gaston et al. evaluated knee function during recovery of a fracture of the tibial plateau.[32] Fifty-one of the 63 patients were treated surgically with internal fixation while another five were treated with a combination of internal and external fixation and a final seven were treated non-operatively. Gaston and his colleagues tested joint movement and muscle function at three, six, and 12 months. At 12 months, only 14% had regained normal

quadriceps muscle strength while 30% had normal hamstring muscle strength. In addition, 21% suffered from residual flexion contractures. All the patients underwent standard physical therapy treatment for 12 weeks after surgery. The study outlined the slow recovery process after fractures of the tibial plateau. Whereas 82% of the patients had achieved 100 degrees of knee flexion, 21% still had extension deficits greater than five degrees, and this deficit was especially pronounced in patients over 40 years of age.[35] These deficits have an impact on daily life for patients with a minimum of 65 degrees of flexion needed for the swing phase of normal gait, a 90 degrees minimum needed to descend stairs and 105 degrees necessary for getting up from a chair.[12]

In a separate retrospective study, outcomes were measured differently by using HSS and Lysholm scores after tibial plateau fracture treatment along with CPM.[33] In this study, the knee was immediately placed in continuous passive motion, though the specifics of the CPM protocol were not included in the study. The study showed a significant decrease in activity due to knee complaints in the first two to three years following the injury; however, at three to six years following injury, these scores had increased to show good function and a return to pre-injury activity levels. This trend reversed itself after six years of follow-up though with patients again deteriorating due to an increase in arthritis. In contrast to the previous study which found age predicted a worse outcome, this study, which relied more on patient self-evaluation, found that the younger patients felt more impaired by the injury.

The results from the previous two studies above highlight the long recovery process that patients with tibial plateau fractures face, and the lack of consensus on post-operative treatment between the two further supports the need for more directed research.

Clinical Data for Articular Fractures and CPM

Several studies have recommended, without clear evidence, that CPM and early passive motion is important for recovery.[34, 35] We will hereby review clinical studies in which CPM was used for articular fractures as part of the treatment.

Proximal Tibia Fractures: Tibial eminence fractures are frequently treated with arthroscopic methods and fixation, with CPM used frequently postoperatively. Osti and co-authors reported 10 patients in which this was performed, and CPM was used postoperatively.[36] No specific data regarding timing, frequency, or other parameters for the CPM were provided. Patients achieved full extension, and flexion between 125 and 135 degrees. In a separate study, CPM was also used in 32 patients with tibial eminence fractures treated with arthroscopic fixation starting on postoperative day one.[37] Near full range of motion was achieved in all patients.

CPM therapy was used in some patients postoperatively treated with arthroscopic assisted tibial plateau fracture fixation as reported by Caspari et al.[38] Twenty-nine patients were treated in this case series, but there was no indication of how many patients were treated with CPM, or why it was chosen for those patients. In a similar study, arthroscopic-assisted tibial plateau fracture fixation was performed in nine cases with CPM used postoperatively.[39] Although no particular parameters were described, CPM was used for five days postoperatively and "clinical function was quite satisfactory" without more detail provided. Ohdera and co-authors did a comparison of arthroscopic with open reduction methods for treatment of tibial plateau fractures with CPM used for all patients postoperatively.[40] Parameters for the CPM were not described, nor was the exact timing ("several days after surgery"). In a similar study, 25 patients with tibial plateau fractures from skiing injuries treated with arthroscopic reduction techniques were managed with CPM postoperatively.[41] Again, no specific information was provided regarding the timing, frequency, or CPM parameters used other than it was used in the hospital postoperatively.

CPM therapy was also used postoperatively from 0–30 degrees in four patients with severe bicondylar tibial plateau fractures treated with combined anterior and posterior ORIF methods.[42] In another technique article, CPM was used immediately postoperatively and continued for two weeks at home, if indicated, for bicondylar tibial plateau fractures treated with ORIF.[43] Carlson described his experience with dual incision posterior ORIF of bicondylar tibial plateau fractures in eight patients who also had CPM postoperatively.[44] These patients were treated with CPM postoperatively in the hospital and continued until knee flexion was near 90 degrees. No other specific information regarding the CPM was provided in his report. There does not appear to be any particular problems with CPM use in the elderly as reported by Biyani and colleagues after ORIF of tibial plateau fractures.[45] In this particular study, CPM with cast bracing was compared with cast bracing alone postoperatively, with no significant difference in clinical outcomes. But there was no indication as to when CPM was chosen, and there were multiple fracture patterns with different surgeons and surgical approaches, making it difficult to draw conclusions regarding the efficacy of CPM for these patients.

Distal Femur Fractures: Similarly to treatment of tibial plateau fractures, surgeons have used CPM postoperatively after treatment of distal femur fractures. Shewring and Meggitt reported on 21 cases of distal femur fractures treated with the AO dynamic condylar screw and CPM started on the second postoperative day.[46] CPM was used twice a day for two weeks. Unicondylar femur fractures treated by ORIF in 16 cases in a separate study were also treated with CPM on the first postoperative day.[47] No other data regarding the specifics of the CPM treatment were provided. Other intraarticular femur fractures such as the "Hoffa fracture" have been treated with ORIF followed by CPM.[48]

Elbow Fractures: The elbow, unfortunately, is particularly prone to developing stiffness after trauma and surgery. CPM has, therefore, been looked to as a possible treatment

both after fracture fixation as well as surgery for release of arthrofibrosis. Frankle and co-authors reported 21 patients with elbow dislocations and radial head fractures treated by ORIF and benign neglect depending on the severity of the injury.[49] Early motion was performed in all patients, with CPM in only two patients. No additional data regarding the CPM was given in this study. Athwal et al. reported on 37 patients who underwent ORIF for AO/OTA type C distal humerus fractures, with some patients also having postoperative CPM treatment.[50] No specific data regarding timing, frequency, or CPM parameters were provided.

Other Articular Fractures: CPM has also been used extensively for postoperative management of articular fractures of the ankle, hip, shoulder, and fingers, in addition to the knee and elbow which have already been discussed. Acetabulum fractures frequently lead to hip stiffness post-traumatic arthritis, for instance, and can be potentially helped with postoperative CPM.[51, 52] For instance, Brumback et al. reported on 58 patients with posterior acetabulum fracture dislocations treated with ORIF and CPM, and many cases also had postoperative skeletal traction, although specific data regarding the CPM was not provided.[51] CPM was the focus of one particular study of ankle fractures treated with ORIF.[3] Farsetti and colleagues described a retrospective series of 22 patients each who underwent ORIF of a malleolar ankle fracture and had 10 years of follow-up. In the first group, CPM was applied immediately postoperatively and for three weeks. In the second group, a plaster splint or cast was applied for three weeks. Patients with CPM had higher AOFAS scores and fewer cases of osteoarthritis at 10-year follow-up than patients treated in a cast. Although this was not a controlled study and had relatively few patients, it does demonstrate the potential functional benefits of CPM compared with immobilization.

Sequelae of Articular Fractures: Arthrofibrosis and Heterotopic Ossification: Though this is not the focus of this particular paper, it is important to note that CPM is also used frequently postoperatively after open or arthroscopic treatment of arthrofibrosis of the elbow. Duration of treatment are reported from one to six weeks postoperatively, although we are not aware of any studies which have investigated CPM specifically.[53–57] Bae and co-authors were particularly aggressive and liberal with CPM treatment, applying this in the recovery room after a medial elbow release and used for 23 hours a day for three weeks, followed by nighttime use for an additional three weeks in addition to physical therapy throughout this time.[54] Alternatively, Kraushaar and colleagues did not feel that CPM was needed in a series of 12 patients with post-traumatic elbow flexion contracture treated with an open lateral release technique.[58]

Contractures of the knee are occasionally treated with the Judet quadricepsplasty, and CPM is frequently used postoperatively, as described by Ali in 10 patients in which CPM was used.[59] In this particular study, immediate CPM at a "slow" rate was applied from 0–60 degrees under epidural control and ice packs. The range of motion and rate (speed) of the CPM was gradually increased up to maximal possible flexion.

Summary

Continuous passive motion therapy clearly has some basic science and animal data to support its use in the management of articular cartilage lesions, which can be extrapolated clinically to the treatment of articular fractures. Interestingly, most clinical studies looking specifically at CPM treatment come from the total joint replacement literature in which patients without articular cartilage lesions are treated. The goals in these cases are not to improve articular cartilage repair, of course, but to essentially improve range of motion, and results have been mixed. Although CPM is used in other cases such as articular fractures, ligament reconstruction, articular cartilage repair surgery, and arthrofibrosis release surgery, it has not been well studied as a treatment for these indications. Very few studies have actually looked at CPM as a treatment modality with articular fractures, so we have very little guidance regarding the recommended time of onset, rate, duration, and other parameters that should be used. The heterogeneity of articular fractures, along with the multitude of surgical treatment factors that can affect range of motion, cartilage repair, and functional outcomes, make it difficult to study CPM from the available data in the literature. Meaningful information could potentially be obtained from a narrow injury subtype, in a randomized controlled study, just to even determine if CPM is beneficial at all, and if so, how it should be used. It appears that there is enough basic science evidence and reported use of CPM for articular fractures to warrant such a study, as we still have room for improvement with our management of articular fractures.

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Medical Student Research Project

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Efficacy and Cost Effectiveness of Prophylactic Knee Bracing in Tackle Football

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Abstract

The practice of prophylactic knee bracing has been controversial since its inception 40 years ago. Designed to prevent medial collateral ligament (MCL) injury, studies have not consistently shown that the use of prophylactic knee braces have reduced these injuries in American football. Regardless of their lack of effectiveness, preventing isolated MCL injuries need be considered in view of their relative mildness, favorable response to conservative management, lack of long-term sequellae, and early return to contact activities compared to other traumatic knee injuries. Also to be considered is the expense in that providing one non-skilled player with a pair of braces costs $500 and fitting all non-skilled players in NCAA intercollegiate football would cost approximately 30 million dollars. Considering the expense involved of fitting players with prophylactic knee braces in view of lack of evidence that they project against MCL and ACL injuries, we recommend that athletic directors, equipment managers, and coaches should take these factors into consideration in the decisions whether or not to require players to wear these braces.

Introduction

In 1988, the American Association of Orthopedic Surgery (AAOS) recommended against the use of prophylactic knee braces citing a lack of evidence of the efficacy of their use.[1] They did acknowledge that no definite conclusion about the devices could be made and that further research and testing was needed. Since this initial recommendation, the AAOS published a new statement in 2003 regarding prophylactic knee braces stating, "Prophylactic knee braces may provide limited protection against injuries to the MCL in football players."[2] However, in 2007, the AAOS has since retired this position statement and has not offered a new position on the use of prophylactic knee braces.

Despite no clear endorsement statement from the AAOS and a lack of convincing studies that demonstrate efficacy, many high school and college football programs either encourage or have mandatory usage of the braces in their programs. Many team physicians, trainers, coaches, and players believe the cost of the knee braces is justified because of claims that these braces may prevent knee injuries. However, the braces are designed only to prevent medical collateral ligament (MCL) injuries, which are relatively mild, experience a favorable response to conservative management, and lack long-term sequellae. Also, there is early return to contact activities compared to other traumatic knee injuries.[3, 4] The major knee injury that devastates most high school and college football athletes is disruption of the anterior cruciate ligament (ACL) and, on occasion, the posterior cruciate ligament (PCL), both of which have a limited potential for spontaneous healing and usually require surgery.[4] Prophylactic knee braces were neither designed to prevent ACL or PCL injuries, and no study has demonstrated evidence of their preventing these injuries.

Background

Football is a popular sport in America and it is estimated that over 1.3 million high school athletes and over 75,000 collegiate athletes play contact football in organized programs.[5] Contact football at these levels involves numerous injuries, particular to the knee in which the literature has varying estimates of occurrence from eight percent to 36 percent of all football injuries.[6]

Previously, a grade III MCL injury was considered severe requiring surgery with a resulting great loss of playing time. The lost playing time and sequellae resulted in the use of prophylactic knee braces. More recent data has afforded a better understanding of the three degrees of MCL injuries with regard to management guidelines. Greater understanding of non-operative management have substantiated that these injuries do not lead to significant time loss or long-term sequellae. Specifically, players sustaining a grade I tear return to the playing field in about 11 days, grade II in 20 days and the most severe grade III in just 34 days.[4] MCL injuries associated with tears to the ACL or PCL require operative management; however, current data indicates that prophylactic braces are not protective for these injuries.[4] ACL injuries usually occur due to a non-contact mechanism and PCL injury from anterior contact to the knee, whereas a

prophylactic brace only claims to protect injuries occurring due to lateral contact.

Prophylactic knee braces have been used in high school and college football programs since the early 1980s. The first prophylactic knee brace was developed in 1978 by George Anderson, head athletic trainer for the Oakland Raiders. The brace was developed in response to an injury to quarterback Ken Stabler, which was a double-hinged brace coined the Anderson Knee Stabler.[7] Importantly, Anderson reported that these knee braces were only effective at preventing re-injury to several players.

The Anderson Knee Stabler, along with other commercially available versions of this device, were use by other players who had never been injured in attempt to prevent MCL injuries. However, based on Anderson's report and the perceived success, the use prophylactic knee braces spread throughout college and high school football programs, and became mandatory in many programs by the early and mid 1980s.

Since the braces have come into use in the 1980s, many researchers have attempted to answer the question of whether or not these knee braces are effective at preventing MCL injuries. Table 1 summarizes the results of these studies which have no consensus and vary greatly in their conclusions. The first significant research reports came out in the mid 1980s and showed various results. A study by Teitz suggested that prophylactic knee braces did not prevent knee injuries and actually increased the risk of knee injury to the players that wore them.[8] Studies by Hewson and Rovere found no statistically significant evidence to suggests prophylactic knee bracing had any positive effect for preventing knee injuries.[9, 10] However, two studies — one by Hansen and another by Schriner — displayed statistically significant evidence that prophylactic knee braces effectively reduced the number of MCL injuries.[11, 12] Quillan, in a non-statistical study of 250 players, suggested a positive effect of prophylactic bracing.[13]

Table 1. Summary of Previous Studies and Conclusions

Author	Year	Conclusion
Hansen[11]	1985	Effectively reduces knee injuries
Hewson[9]	1986	No statistically significant evidence of a positive effect
Teitz[8]	1987	No positive effect and increases risk of injury
Rovere[10]	1987	No statically significant evidence of a positive effect
Schriner[12]	1987	Effectively reduces knee injuries
Quillian[13]	1987	Small evidence of reducing injuries
Grace[14]	1988	No positive effect and may increases risk of injury
Zemper[15]	1990	No positive effect and may increases risk of injury
Sitler[17]	1990	Effectively reduces knee injuries based on player position
Deppen[16]	1994	No statistically significant evidence of a positive effect
Albirght[19]	1994	No statistically significant evidence of a positive effect
Pietrosimone[6]	2008	No statistically significant evidence of a positive effect
Salata[18]	2010	No statistically significant evidence of a positive effect

Since 1988, many additional studies have been undertaken with varying results. Studies by Grace and Zemper indicated that the prophylactic knee braces demonstrated no positive effect in preventing injuries and data suggested an increase in the risk of knee injury.[14, 15] A study by Deppen found no statistically significant difference between the braced and non-braced players.[16] Lastly, a study by Sitler following cadets in a US military academy who participated in eight-man intramural football found a significant reduction in MCL injuries that was dependent on player position.[17] A more recent study in 2008 by Pietrosimone attempted to determine a relative risk difference between braced and non-braced players by retrospectively analyzing previous studies.[6] The authors concluded that no definitive conclusion could be drawn. The most recent study in 2010 by Salata[18] concluded that no consistent effect of prophylactic knee bracing could be determined.

Many of these studies attempted to comment on the incidence of ACL injuries between braced and non-braced players but were unable to demonstrate a statistically positive effect. Many collegiate and high school programs encourage the use of prophylactic knee braces and some programs require certain players to wear them despite no recommendation from the AAOS and a lack of evidence of the effectiveness of the braces. A typical brace such as the Bledsoe Axoim retails $500 per brace (Figure 1).

Clearly, analysis of the above cited data pools raise the question of the efficacy of prophylactic knee braces protecting against any knee injury with particular regard to the MCL. Corollary to this question are two issues: one, are MCL injuries of significance with regard to time lost and sequela; two, are the expenses of braces justified?

Methods

Relevant literature articles were found by searching the PubMed database. The database was searched by using the MeSH for knee injury, sub title prevention and control and MeSH for football. The search returned 31 results. Many of these 31 results are relevant to the study of prophylactic knee bracing. The remaining relevant articles were found by reading two relevant review articles by Requa and Pietrosimone.[6, 20] The review article by Requa provides a review of the study methods used, offering superb commentary on the limitations and difficulty of the prophylactic knee bracing studies conducted prior to 1990. After finding the studies, a level of evidence score was assigned to each study. Table 2 provides a summary each of the authors' method of study.

A search for medial collateral ligament injuries was searched using the PubMed database by searching MeSH medical collateral ligament knee subtitle injury and MeSH football injuries. Nine results were retrieved, one of which was relevant. The relevant paper talked about lateral collateral ligament (LCL) of the knee but several references in the paper were made to studies about the MCL which were very useful.

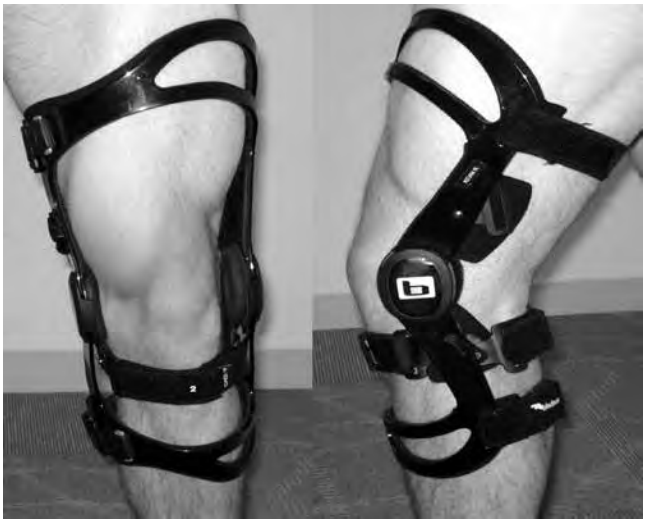

Figure 1. The Bledsoe Axiom brace, a typical prophylactic knee brace

Consultation with the head athletic trainer for the Temple University football program provided information of the cost and use of the prophylactic knee braces. A calculation of Temple University's football program of prophylactic knee bracing policy was applied to all NCAA football teams to provide aggregate cost of prophylactic knee bracing for offensive lineman in all of college football.

Results

Most of the studies were done by looking at players either non-braced or braced; however, two studies, Deppen and Albright, calculated players knee exposures and further divided the knee exposures into a non-braced and braced group with knee exposure being defined by the author and greater weight given to hours played in a game versus practice. All of the studies provided data for total MCL injuries, displayed in Table 3. Studies for which MCL injuries were defined by severity or by days missed are included in Table 4 to display the number of grade III MCL injuries. Several authors have attempted to study the effect of bracing and ACL injury prevention, summarized in Table 5.

Discussion

Most all of the studies agree that knee bracing provides no significant clinical evidence that either an MCL or ACL injury can be prevented (Table 5). Analysis of playing time lost by the worst case scenario, a grade III MCL injury, which was 34 days and has been the consistent since the 1990s.[4] The standard of care for a grade III MCL injury is an examination to ensure integrity of the ACL and PCL as well as the menisci, followed by the use of a stabilizing brace and crutches for 14 days with limited knee exercise to promote mobility. After these 14 days, the knee is usually stabilized, crutches and bracing is discontinued and rehabilitation initiated. Typically, a stationary bike is used at first, then jogging and running are introduced and, after 30 to 40 days of injury, a player can return to contact sports. A functional brace may be recommended for the remainder of the season but is usually discontinued the following season.[3, 4] This particular standard of care is the most effective management for a grade III MCL and it is significantly less expensive than the operative treatment an ACL injury requires.

The fact that prophylactic knee braces do not prevent ACL injuries and that a grade III MCL injury neither leads to extensive playing time lost nor is expensive to treat led us to question the cost to outfit a player and/or team with prophylactic knee braces. We discovered that the bracing policy is mandatory for offensive linemen at some institutions and that many programs require all non-skilled players, defined as offensive linemen, defensive linemen and linebackers, to wear prophylactic braces. Historically, offensive linemen suffer the greatest number of knee injuries via lateral contact followed by linebackers and defensive linemen.[8, 11] A typical prophylactic brace, such as the Bledsoe Axiom, costs approximately $500 for a pair. College football teams usually have 15 to 20 offensive linemen, 12 to 17 linebackers, and 12 to 17 defensive linemen on a team per year. There are currently a total of 624 D-I, D-II and D-III football teams in the NCAA. We calculated that if every college football program outfitted their non-skilled players with knee braces, it would cost between $14 and $17 million per year. Considering that braces may last less than a year and that many collegiate teams carry reserve players not on the roster and may have additional practice squads, the true cost of outfitting all non-skilled players with prophylactic braces is conservatively estimated in excess of $30 million per year.

Conclusion

Several decades of research provides no definitive conclusion regarding the effectiveness of prophylactic knee braces and injuries to the MCL. Furthermore, no study has been able to demonstrate a positive effect on ACL injury rates (Table 6). A calculation of the cost of providing all non-skilled position players in NCAA football programs with braces would cost an estimated 30 million dollars. Also to be considered is the potential exponential cost of prophylactic bracing of high school players. To be noted is that prophylactic knee bracing is not employed at the professional level.

Considering the high expense involved of fitting players with prophylactic knee braces and the lack of evidence of either MCL and ACL injury prevention, we believe that athletic directors, equipment managers, and coachers should take this into consideration in the decision-making process of whether or not to require players to wear these braces.

Acknowledgement

We would like to thank Dwight Stansbury, the head trainer for the Temple football team, for his time and expertise.

Table 2. Summary of Previous Studies Methods and Level of Evidence

Author	Year	Player Level	Non-braced	Braced	Length of Study	Method	Level of Evidence
Hansen*	1985	Collegiate	329	148	4 years	Reviewed medical records and recorded number and types of knee injuries and compared braced to unbraced	II
Hewson*^	1986	Collegiate	226	224	4 years	Analyze and compare type of injury, severity, player position and days lost from injury	II
Teitz*	1987	Collegiate	7,011	4,686	2 years	Analyzed and compared type of injuries according to player position, severity, non-braced teams and braced teams	II
Rovere*^	1987	Collegiate	70	62	4 years	Comparison of knee injuries between unbraced and braced players	II
Schriner	1987	High school	1,357	433	2 years	Comparison of knee injuries between unbraced and braced players broken down by lateral force contact and medial, posterior and hyperflexion contact	II
Quillian	1987	High school	194	50	2 years	Comparison of knee injuries between braced and non-braced groups based on contact hours	II
Grace^	1988	High school	253	330	2 years	Comparison of knee injuries between unbraced and single-hinge brace, unbraced and double-hinge brace	II
Zemper*	1990	Collegiate	4,485	1,744	2 years	Comparison of total knee injuries between unbraced and braced, comparison of MCL injuries, severity of knee injuries	II
Sitler*^	1990	Collegiate intramural eight-man tackle football	705	691	2 years	Comparison of total knee injuries between unbraced and braced, type of injury and severity	I
Deppen^	1994	High school	19,458 knee exposures	21,641 knee exposures	4 years	Comparison of total knee injuries between unbraced and braced, type of injury and severity, mechanism of injury	II
Albirght*^	1994	Collegiate	76,811 knee exposures	78,911 knee exposures	3 years	Comparison of total knee injuries between unbraced and braced, type of injury and severity, mechanism of injury	II
Pietrosimone	2008	Various	Systematic review of seven studies indicated by * next to author name		Various	Compute the relative risk increase or relative risk reduction of knee injury in each of the seven previous studies	IV
Salata	2010	Various	Review of six studies indicated by ^ next to author name		Various	Review of previous results and discussion of findings	

Table 3. Summary of Previous Studies and Total MCL Injuries (Note NB Is Non-braced and B Is Braced)

Author	NB Players	MCL Injuries NB	B Players	MCL Injuries B
Hansen	329	17	148	2
Hewson	226	41	224	33
Teitz	7,011	143	4,686	175
Rovere	70	16	62	18
Schriner	1,357	85	433	10
Quillian	194	6	50	0
Grace	250	6	330	17
Zemper	4,485	69	1,744	32
Sitler	705	25	691	12
Total	**14,627**	**408**	**8,368**	**299**
Author	**NB Exposures**	**MCL Injuries NB**	**B Exposures**	**MCL Injuries B**
Deppen	19,458	12	21,617	16
Albright	76,811	47	78,911	53
Total	**96,269**	**59**	**100,528**	**69**

Table 4. Summary of Previous Studies and MCL Grade III Injuries (Note NB Is Non-braced and B Is Braced)

Author	NB Players	MCL III NB	B Players	MCL III B
Hewson	226	15	224	8
Teitz	7,011	21	4,686	19
Rovere	70	1	62	1
Grace	250	4	330	7
Zemper	4,485	6	1,744	3
Sitler	705	3	691	1
Totals	**12,747**	**50**	**7,737**	**39**
Author	**NB Players**	**MCL III NB**	**B Players**	**MCL III B**
Deppen	19,458	3	21,617	3

Table 5. Summary of Previous Studies and ACL Injuries (Note NB Is Non-braced and B Is Braced)

Author	NB	ACL Tears NB	B	ACL Tears B
Hansen	329	6	148	1
Hewson	226	6	224	5
Teitz	7,011	33	4,686	18
Rovere	70	1	62	3
Quillian	194	1	50	0
Grace	250	1	330	1
Sitler	705	7	691	2
Total	**8,785**	**55**	**6,191**	**30**
Author	**NB**	**ACL Tears NB**	**B**	**ACL Tears B**
Deppen	19,458	7	21,617	2

Table 6. Summary of Previous Studies and Detailed Conclusions

Author	Year	Total Knee Injury	Injury Severity	Recommended
Hansen	1985	Reduction in MCL and meniscus injuries	Less players needed surgery	Yes, OL and LB
Hewson	1986	No significant reduction	No change due to bracing	No, no significant effect noted
Teitz	1987	More injuries with brace on lateral contact, especially RB and DB	No change due to bracing	No will cause more injuries
Rovere	1987	More injuries with brace	No change due to bracing	No will cause more injuries
Schriner	1987	Significant reduction 5.4%	Not determined	Yes, high school only
Quillian	1987	Reduction in MCL injuries	Less severe injuries	Longer prospective study needed
Grace	1988	Increased knee injuries for single-hinge braces, no increase in knee injuries for double hinge. However, both types of braces show an increase in injury of distal leg and foot	No change due to bracing	No will cause more leg and ankle injuries
Zemper	1990	Knee injuries are higher in braced group	No change due to bracing	No more injuries are noted

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Case Report

Progressive Fusionless Correction of Adolescent Idiopathic Scoliosis with the Anterior Vertebral Body Tether: A Case Study

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Introduction

Traditionally, scoliosis has been treated with bracing or spinal fusion. Yet, bracing is not always an efficacious treatment option: psychosocial elements surrounding brace-wear exist,[1] and bracing often fails to halt curve progression.[2] Spinal fusion presents its own problems: fusion often inhibits growth over the length of the construct, can lead to the development of adjacent level disc degeneration, and cause other difficulties including decreased range of motion and decreased spinal mobility postoperatively.[3] Because of these concerns, alternative surgical approaches that permit growth of the spine and halt curve progression have been proposed. Anterior vertebral body tethering (AVBT) has been implemented as an alternative to both spinal fusion and other fusion-less techniques. The biomechanical basis for growth modulation via a flexible tether has been shown in animal models;[4] however, there is a paucity of data investigating the efficacy of tethering in human subjects. To date, one case has been reported in literature.[5] In this report, we present the first such AVBT at our institution.

Case Study

A twelve-year-and-five-month-old female presented with a right sided, 34° main thoracic curve from T6–T11 and a lumbar curve of 34° from T10–L4 (Figure 2). The patient's thoracic kyphosis was 22° (T5–T12). The patient had a Risser score of 0, and a Sanders score of 3. Flexibility of the curve was measured at 12° upon lateral bending. On forward bending, the patient's thoracic hump and lumbar prominence both measured 10°. The patient's curve magnitude and her skeletal immaturity made her a prime candidate for the AVBT procedure.

Description of the Procedure

The patient was positioned in the lateral decubitus position with the right side up. Two small working thoracoscopic portals were made in the anterior axillary line. A 15 mm working portal and a small thoracotomy incision were also made on the mid axillary line. Screw holes were drilled and tapped and a 3-prong staple was impacted into place on the anterior aspect of the vertebral bodies under C-arm guidance. 6.0-millimeter screws were placed along the length of the construct. A flexible tether (Zimmer Dynesys, Raytham, MA) was then placed through the thoracotomy site into the tulip of the superior most screw, which was then locked down with the set screw. The tether was then laid into the tulips of all the screws. Tension was then placed onto the next caudal screw (the 2nd vertebral body of the construct). Careful reduction translation force was placed onto the spine at both of these levels as the tether was tightened, and the set screw tightened at the second body. The surgery progressed in a similar fashion distally with the tether being attached at caudal levels. All levels were sequentially tensioned (Figure 1). As previously described,[5] anterior stapling of the lumbar curve was performed at this time as well. After all instrumentation was completed, global imaging of the spine in both AP and lateral views was performed; visualization of significant reduction of the curvature in the coronal plane and appropriate alignment in the sagittal plane was obtained.

Results

The main curve measured 11° intraoperatively while the patient was in the supine position. Immediately postoperatively, the patients curve measured 15° in the upright position (Figure 5). Follow-up x-rays were obtained at six weeks, three months, six months, and one year. The patient's films showed gradual and progressive correction of the coronal deformity over the course of the follow-up period (Table 1). The thoracic curve measured 13° at six-week follow-up, 10° at three months, and 6° at six months. At 12-month follow-up, thoracic kyphosis and lumbar lordosis were well maintained and the patient's main thoracic curve measured 0°, representing a 100% correction (Figure 9).

Discussion

Several fusionless surgical options for the treatment of idiopathic scoliosis have been described. Posterior approaches with growing systems are often utilized. Problems with these include the need for serial lengthening with growing rods and expansion of the Vertical Expandable Prosthetic Titanium Rib (VEPTR) system every six months (both of these necessitate additional surgery). Anteriorly, vertebral body stapling has been applied in patients with idiopathic scoliosis (and high risk for progression) as an

alternative to bracing and/or fusion. The results of stapling suggest that the technique provided good correction of the scoliotic spine, while allowing for continued axial growth; Betz et al. report a 79% success rate in thoracic curves measuring less than 35°. However, for thoracic curves greater than 35°, stapling was unsuccessful and patients ultimately required alternative treatment.[6] This led the authors to pursue anterior vertebral body tethering (AVBT) as an alternative surgical option for skeletally immature patients who are at a high risk for progression.

It is clear from our case study that the use of the AVBT was not only able to achieve an initial coronal correction, but it has been able to control and shape the growth of the scoliotic curve over time. The patient's axial growth, along with the tension created by the tether on the convex side of the curve, has resulted in a positive and progressive correction of the curve at 12-month follow-up. By partially restraining growth on one side of the spine (the coronal convexity), the technique has allowed growth on the contralateral side to reverse the abnormal scoliotic growth pattern. This treatment option addresses many of the concerns associated with other traditional surgical techniques. The treatment is fusionless and thus avoids the difficulties associated with spinal fusion, including stiffness and trunk shortening. This technique has the additional advantage of not necessitating multiple trips to the OR (as is the case with serial lengthening of the VEPTR system or growing rods).

The authors are cautiously optimistic that the use of the AVBT will ultimately prove to be a viable treatment option for skeletally immature patients. However, several unknowns still exist surrounding the AVBT technique. Some surgeons have begun using this technique in the lumbar spine; however, no data has been published reporting its efficacy in that region. Additionally, in patients who have achieved close to 100% correction (including this case), there is concern that further skeletal growth, along with the mechanical forces of the tether, will begin to create a scoliotic curve in the opposite direction. In light of this potential hurdle, a key consideration is how much "residual curve" to leave behind in an initial tethering procedure. This decision will likely hinge on how much growth the child has remaining. If over-correction does occur, it may potentially be alleviated thoracoscopically by loosening or removing segments of the tether, thus relieving the tension in one or more segments of the construct. Clearly, though, the biomechanical principles underlying the anterior vertebral body tethering technique dictate the need for increased follow-up time to characterize the temporal relationships between dynamic tethering, skeletal growth and final correction.

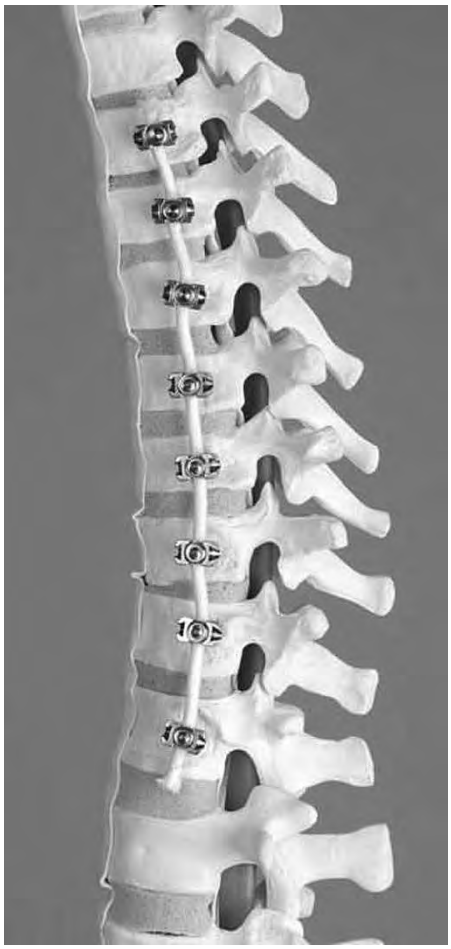

Figure 1. Saw-bone model of tethering construct

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Table 1. Coronal Cobb Angle

	Preoperative	Intraoperative	First Erect	6 Weeks	3 Months	6 Months	12 Months
Thoracic Cobb	34°	11°	15°	13°	10°	6°	0.6°

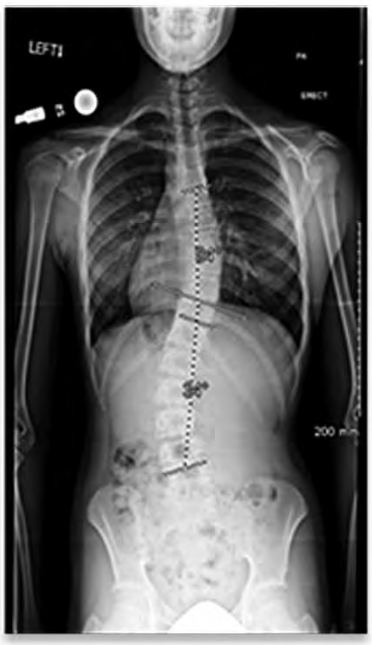

Figure 2. Pre-Operative Coronal 34°

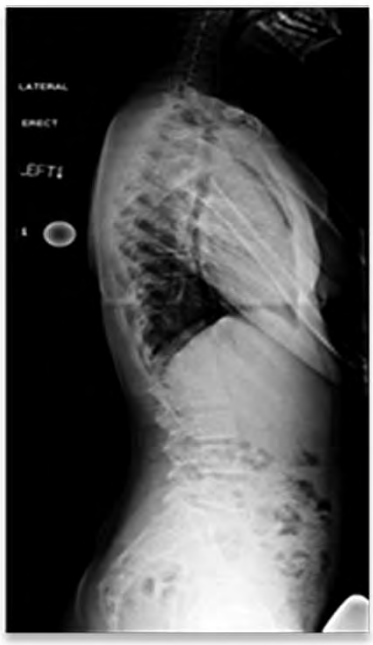

Figure 3. Pre-Operative Thoracic Kyphosis 21°

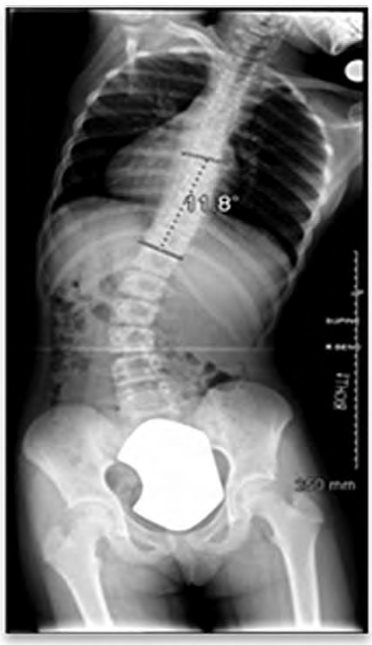

Figure 4. Pre-Operative Cobb on Right Bend 12°

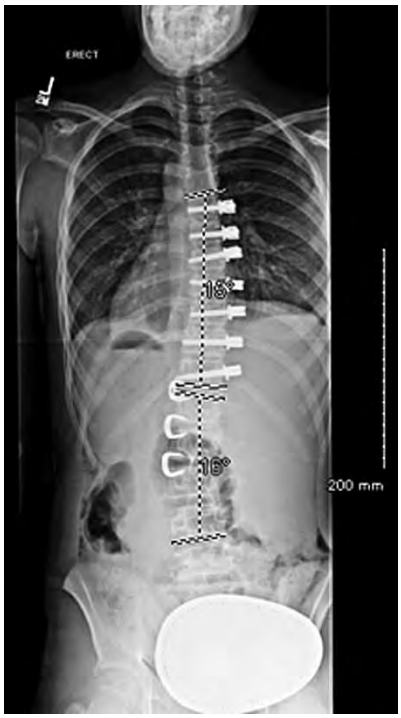

Figure 5. First-Erect Coronal 15°

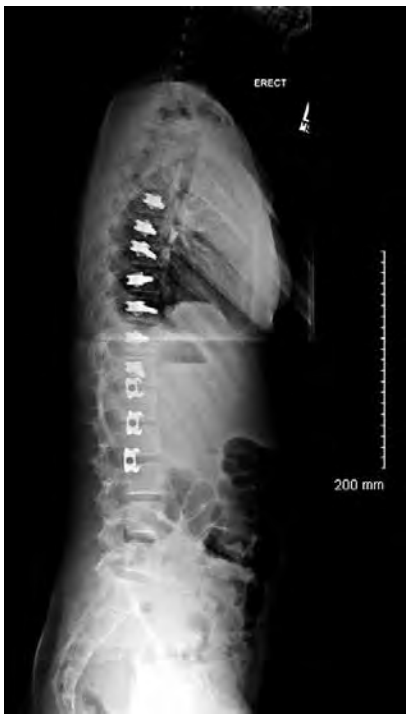

Figure 6. First-Erect Thoracic Kyphosis 16°

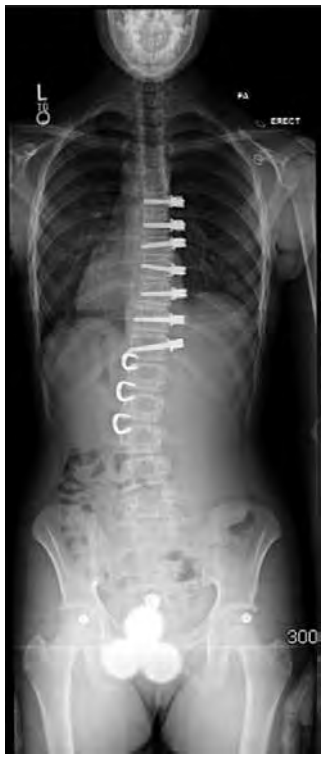

Figure 7. Six-Month Post-Operative Coronal 6°

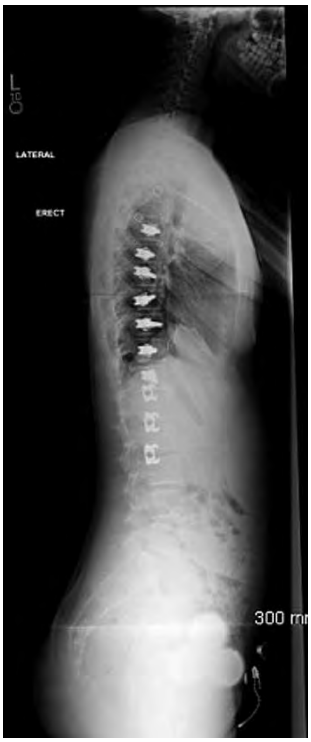

Figure 8. Six-Month Post-Operative Thoracic Kyphosis 22°

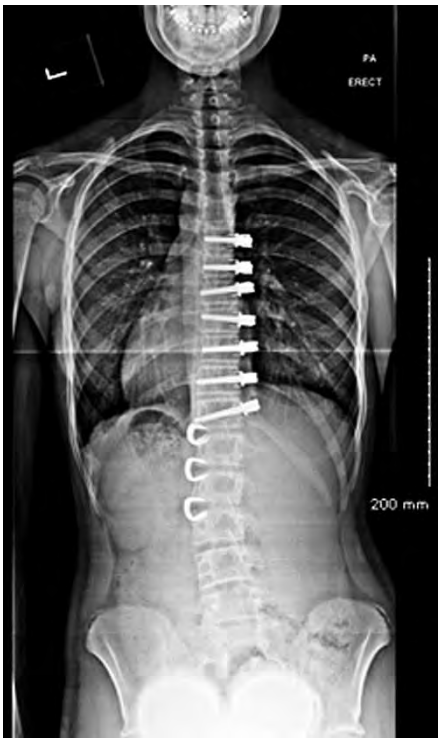

Figure 9. Most Recent Coronal Cobb 0°

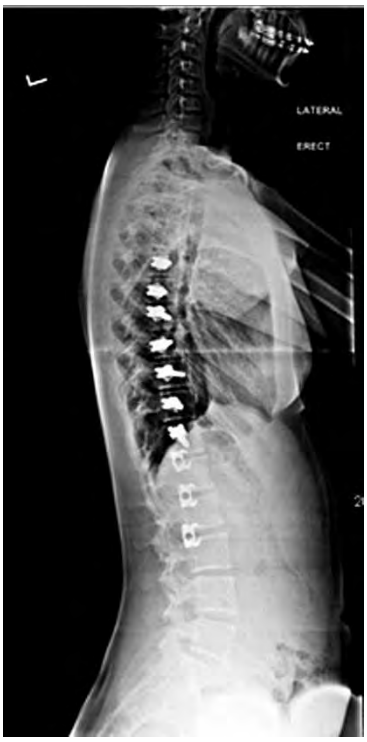

Figure 10. Most Recent Thoracic Kyphosis 23°

Review Article

Connective Tissue Growth Factor (CTGF): Current Understanding and Clinical Implications in Bone Disorders

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Abstract

It is well established that the matricellular protein connective tissue growth factor (CTGF) is produced in bone during skeletal development and fracture healing. While its importance in skeletal formation has been clearly shown through the use of genetically engineered mouse models in which CTGF production is ablated, the ramifications for targeting CTGF clinically remain controversial. Since a monoclonal antibody targeting CTGF is currently in clinical trials for the treatment several non-skeletal disorders, it is critical that we identify any potential application for targeting CTGF clinically in bone. In this article, we provide our current understanding of the role of CTGF in bone formation and potential clinical implications involving it as a target in treatment modalities.

CTGF: A CCN Family Member

The 38kDa connective tissue growth factor (CTGF) protein was first identified in the conditioned media of human umbilical vein endothelial cells and given its name due to the mitogenic role it played in these cells.[1] A role for it in bone development was demonstrated in our lab using a rat osteopetrosis model in which CTGF was shown to be highly expressed in bones.[2] The stimulation of CTGF expression by transforming growth factor beta (TGF-β), the most potent inducer of CTGF expression, was first demonstrated in human skin fibroblasts during wound repair *in vivo* where there is a coordinated up-regulation of TGF-β followed by CTGF expression.[3] In addition to TGF-β, CTGF is also upregulated by other growth factors and is ultimately secreted into the extracellular matrix (ECM), where it exerts its effects in an autocrine and paracrine fashion on cells through interaction with other ECM components; this is made possible by its modular structure.

The CTGF transcript contains five exons, which code for a signal peptide and four modules. Module I is an insulin-like growth factor (IGF)-binding domain, Module II a von Willebrand type C (vWC) domain, Module III a thrombospondin-1 (TSP-1) domain, and Module IV a C-terminal domain containing a putative cysteine knot.[4–6] CTGF has been grouped with other proteins with similarly conserved modules into the CCN family of proteins. Named for the three original members — Cysteine-rich 61 (Cyr61), CTGF, and Nephroblastoma overexpressed (Nov) — the CCN family also includes the Wnt-induced secreted proteins (WISP) 1–3.[7] CCN proteins regulate an array of cellular and physiologic functions, including those crucial for bone formation, growth, and maintenance.[8]

It is important to note that to date no single, unique CTGF receptor has been identified. Instead CTGF exerts cellular effects by binding to cell-surface integrin receptors and interacting with co-receptors in the ECM. At least eight types of integrins have been shown to bind to CCN proteins.[9] Additionally, CTGF has been shown to bind different integrin complexes on the same type of cell.[8] CTGF also interacts with multiple types of co-receptors. These include heparan sulfate proteoglycans (HSPGs), low-density lipoprotein (LDL) receptor-related proteins, and the neurotrophic tyrosine kinase receptor type 1 (NTRK1 or TrkA).[10] Through its interaction with these integrin receptors and co-receptors, CTGF functions in three general ways: 1) CTGF signals in an effector cell through direct integrin binding on the cell surface; 2) can alter the biologic activities of growth factors and cytokines through signaling crosstalk; and 3) CTGF can both regulate the expression of biologically active molecules (e.g. growth factors, cytokines), and functionally interact with them in the ECM to affect their bioavailability and activity. As a result, the complex interaction of CTGF with other molecules results in a functional versatility in a cell- and context-specific manner.

CTGF Is Required for Skeletal Development

A role for CTGF in skeletal tissues first emerged from studies demonstrating its expression in developing cartilage, bone, and teeth.[11–14] However, the key finding for the indispensability of CTGF in skeletogenesis came with the generation of genetically engineered mice lacking CTGF production. These CTGF knockout (KO) mice have provided great insight into the importance of CTGF in normal skeletal development. The initial study demonstrated that CTGF KO mice are born with multiple skeletal dysmorphisms, including bends (or kinks) in specific long bones, craniofacial

abnormalities, and misshapen ribs.[12] CTGF KO mice die shortly after birth due to respiratory distress; this is believed to be the result of the misshapen ribs as well as pulmonary hypoplasia.[12, 15] Since these mice cannot be studied postnatally, the lack of CTGF in adult bone maintenance and fracture repair has not been pursued extensively. This gap in our understanding of CTGF in adult bone constitutes a difficulty when considering the application of a monoclonal antibody targeting CTGF for clinical treatments (see below). Our laboratory recently demonstrated that the role of CTGF in prenatal bone development is skeletal site-specific, such that the loss of CTGF, while critical for the proper formation of some bones (e.g. tibiae), is not necessary for normal formation of others.[16] This adds yet another level of complexity in understanding the role of CTGF in skeletal tissues, one that is necessary should it be used in clinical applications.

The Osteoinductive Promise of CTGF

CTGF is produced and secreted by osteoblasts and has been shown to stimulate osteoblast differentiation, matrix production, and differentiation *in vitro*.[2, 13, 17–20] Considering the apparent beneficial effects of CTGF on osteoblast differentiation and bone formation, studies have tested applications of its use in bone regeneration to promote bone healing or new bone formation. Injection of recombinant CTGF (rCTGF) into the marrow cavity of rat femurs elicited an osteoinductive response in the form of osteoblast differentiation and active bone formation.[2, 13] When a hydroxyapatite carrier loaded with CTGF was implanted into bone defects within a rabbit mandible, CTGF significantly enhanced the proliferation and migration of human bone marrow stromal cells, induced cell invasion and enhanced bone formation compared with the carrier alone.[21] Using the intractable bone defect model, treatment with rCTGF induced the osteoblast mineralization markers and enhanced the bone regeneration.[22] The majority of *in vivo* and *in vitro* studies support an anabolic role for CTGF on bone formation, and therefore, this factor is a candidate for the development of novel clinical therapeutic approaches to stimulate bone formation.

CTGF Promoter Polymorphism and Systemic Sclerosis

Recent evidence demonstrates that a polymorphism in the CTGF gene promoter is associated with a susceptibility to systemic sclerosis (SSc). The polymorphism constitutes a G-945C substitution, the effect of which is to decrease the binding of the key transcriptional repressor, SP3, resulting in increased expression of CTGF.[23] However, this (G-945C) polymorphism does not correlate with increased plasma CTGF levels.[24] SSc, also known as scleroderma, is a heterogeneous disorder of the connective tissue and includes in its disease progression immune activation, vascular damage, and eventual tissue fibrosis.[25] Due to the lack of efficient antifibrotic therapeutics, severe SSc results in mortality due to the fibrosis and subsequent loss of function of skin, vasculature, musculoskeletal system, and internal organs.[23, 25] In addition to the well-known fibrotic changes seen in SSc, bone mineral density (BMD), bone mass, and bone quality in these patients are deleteriously affected,[26, 27] including both cancellous and cortical bone.[28] Further studies are necessary to determine if there is a direct link between the G-945C CTGF promoter polymorphism and effects on bone in SSc patients.

WISP-3/CCN6 and Progressive Pseudorheumatoid Dysplasia

Members of the CCN family, such as CTGF, may play a role in regulating the expression of other family members. Importantly, this has been suggested from studies using the CTGF KO mouse in which Nov/CCN3 expression increased in chondrocytes in the absence of CTGF.[29] Therefore, it is becoming increasingly evident that examining the expression levels of all six CCN family members is prudent in cases of individual mutations causing skeletal abnormalities. In 1999, mutations in Wisp-3/CCN6 were identified as the molecular cause for the syndrome Progressive Pseudorheumatoid Dysplasia (PPD). PPD is an autosomal recessive disorder characterized by juvenile onset arthropathies and progressive erosive bone and joint changes.[30] However, when mice were generated with the mutations seen in human PPD, the skeletal function was normal.[31] This highlights the fact that murine models involving genetic mutations in CCN proteins do not always recapitulate human pathologies. Further investigation is necessary to determine if any CTGF expression is affected as a result of Wisp-3 mutations in PPD.

Current Anti-CTGF Treatment: FG-3019

Levels of CTGF have been shown to be markedly elevated in various injured tissues that develop fibrosis including the skin, kidney, liver and lung.[32–36] More recently, CTGF overexpression has been implicated in the pathophysiology of dystrophic skeletal muscles where it is believed to contribute to the deterioration of skeletal muscles and their function in addition to mediating the ensuing fibrosis of the damaged muscle tissue.[37] Clinical trials are currently underway using a monoclonal antibody (FG-3019) that recognizes module II of human and rodent CTGF as a novel therapy to treat patients with diabetes, advanced kidney disease, pancreatic cancer, idiopathic pulmonary fibrosis.[38] This antibody has also been used to treat CTGF-expressing tumors in mice, where it abrogated CTGF-dependent pancreatic tumor growth and lymph node metastasis without any toxic side effects in mice.[39] FG-3019 has also been used as a therapy in a mouse model of Duchenne muscular dystrophy, where it reversed the fibrosis of muscular tissue and even allowed return of skeletal muscle function (personal communication, Dr. Enrique Brandan). These studies support the concept of using drugs that specifically target CTGF as a treatment for

various human diseases. However, the effects of FG-3019 on underlying bone have not yet been investigated.

Targeting CTGF Clinically: A Paradox

The utility of targeting CTGF clinically to treat conditions of insufficient bone formation, while ripe with potential, is still far from being a complete picture. As mentioned previously, studies in which rCTGF was injected into the marrow cavity of rat femurs demonstrated an anabolic response to CTGF in bone formation.[2, 13] However, since the functional diversity of CTGF largely depends on the matricellular molecules with which it interacts, the anabolic nature of CTGF in bone is likely contingent upon the presence and/or absence of specific molecules at a target skeletal site; therefore, the effect of CTGF on bone would be variable from site to site. To illustrate this, consider the interaction of CTGF with the TGF-β and bone morphogenetic proteins (BMP) pathways. While both of these pathways are critical in bone formation, it has been shown that CTGF can positively enhance TGF-β signaling through interactions with TGF-β1 while inhibiting BMP receptor signaling through interaction with BMP-4.[40] Furthermore, we have demonstrated that treatment of primary osteoblasts that lack CTGF (isolated from CTGF KO mice) with rBMP-2 causes enhanced differentiation and signaling when compared to control osteoblasts (isolated from wild-type mice; see paper by Mundy et al. in this edition). These data suggest that CTGF acts to inhibit the anabolic effect of BMP-2 on bone formation.

Herein lies the current paradox of targeting CTGF clinically in bone: how is it that CTGF alone produces osteoinductive effects, while also potentially countering the well-established osteoinductive effects of BMP-2? While there is insufficient research evidence to currently tease apart this discrepancy, both have current clinical implications. As CTGF expression normally increases during fracture repair, one could postulate that abrogation of CTGF signaling through FG-3019-mediated blockade would negatively affect fracture healing and potentially hasten development of age-related osteoporosis.[13, 14, 41, 42] This would present a serious detractor to FG-3019 therapy, particularly in postmenopausal women. Additionally, knowledge of the CTGF-BMP interaction could identify a potential use for the addition (or blockade) of CTGF in concert with recombinant BMP2 (rBMP-2) administration in treatment of bony defects and malunions. To fully understand the therapeutic potential of CTGF in bone formation, studies must utilize the following: 1) animal models simulating various clinical scenarios, such as fracture repair and osteoporosis; and 2) local or global treatment using rCTGF or FG-3019 with or without rBMP-2. Once results from these *in vivo* models are obtained, only then can one understand the clinical applications of CTGF for treatment of patients with bone disorders.

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Senior Abstract

Are Young Adults With Low Energy Distal Radius Fractures Vitamin D Deficient? A Prospective Pilot Study

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Senior Bio Questions

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- Where do you want to practice? MD/DC/VA area or Atlanta

Introduction

Low serum levels of Vitamin D (25-hydroxyvitamin D_3) have been associated with low energy fractures in the elderly; however, no investigations to date have correlated Vitamin D with low energy fractures in young adults. The purposes of this study were to 1) measure the serum levels of 25-hydroxyvitamin D_3 in young adults who sustained a low energy distal radius fracture and compare those values to that of healthy individuals without a history of fracture; and 2) determine the prevalence of vitamin D deficiency in this population.

Methods

A single-center, prospective study of low energy distal radius fractures was performed at an urban, level I trauma center in the northeastern region of the United States from 2011–2012. All subjects were aged 18–45 years. Demographic information such as age, body-mass index, race, gender, medical history, and history of previous fracture were recorded. For the study group, all patients were tested for serum 25-hydroxyvitamin D_3 within 30 days of the injury; patients who sustained an injury via a high-energy mechanism were excluded. For the control group, individuals were selected from a database of healthy patients followed by the internal medicine service; they were excluded if a history of comorbidity or previous fracture was identified.

Results

A total of 15 distal radius fractures and 67 healthy controls met inclusion criteria. The overall range of 25-hydroxyvitamin D_3 level was 7.0–50.2 ng/mL, and the average measurement was 22.4 ng/mL in the control group and 21.4 ng/mL in the study group; these differences were not statistically different ($p = 0.9761$). In patients who sustained a distal radius fracture, vitamin D levels were categorized as the following: deficiency in 13.3%, insufficiency in 46.6%, and adequacy in 40.0%. Of the fracture cases, seven of 15 were managed by operative fixation, and patients who underwent surgery did not have significantly different values than those treated by nonoperative management ($p = 0.7788$).

Conclusions

The overall serum 25-hydroxyvitamin D_3 levels for young adults in this region were in the low-normal range. Patients who sustained a low energy distal radius fracture did not have significantly lower vitamin D levels than relatively similar, healthy patients without a history of fracture.

Senior Abstract

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Nerve and Tendon Injury with Percutaneous Fibular Pinning: A Cadaveric Study

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Objective

The purposes of this study were to measure the average distance from a percutaneous pin in each quadrant of the distal fibula to the sural nerve and nearest peroneal tendon, and define the safe zone for pin placement as would be used during surgery.

Method

Ten fresh-frozen cadavers underwent percutaneous pin fixation into four quadrants of the distal fibula. The sural nerve and peroneal tendon were identified as they coursed around the lateral ankle. Distances from the K-wire in each quadrant to the anatomic structure of interest were measured.

Results

Average distances (mm) from the K-wire to the sural nerve in the anterolateral, anteromedial, posterolateral, and posteromedial quadrants were 19.1 ± 8.9 (range, 5.1–35.5), 12.8 ± 8.2 (range, 0.3–27.8), 12.6 ± 6.8 (range, 3.0–27.8), and 5.9 ± 5.5 (range, 0.1–19.9), respectively. Average distances from the K-wire to the nearest peroneal tendon in the anterolateral, anteromedial, posterolateral, and posteromedial quadrants were 15.7 ± 4.4 (range, 9.5–23.1), 11.9 ± 5.2 (range, 3.2–21.7), 6.3 ± 3.9 (range, 0.1–14.4), and 1.0 ± 1.6 (range, 0–5.6), respectively.

Conclusions

Percutaneous pinning of distal fibula fractures is a successful treatment option with minimal complications. Our anatomical study found the safe zone of percutaneous pin placement to be in the anterolateral quadrant. The sural nerve can be as close as 5.1 mm and the peroneal tendons as near as 15.7 mm. In contrast, the posteromedial quadrant was associated with the greatest risk of injury to both the sural nerve and peroneal tendons.

Senior Abstract

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Is Antibiotic Prophylaxis Necessary in Elective Soft Tissue Hand Surgery?

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Background

Antibiotic prophylaxis for clean soft tissue hand surgery is not yet defined. Current literature focuses on overall orthopedic procedures, traumatic hand surgery, and carpal tunnel release. However, a paucity of data exists regarding the role of antibiotic prophylaxis in a broader variety of soft tissue hand procedures. The goal of the current study was to evaluate the rates of surgical site infection following elective soft tissue hand surgery with respect to administration of prophylactic antibiotics.

Methods

A multicenter, retrospective review was performed on 600 consecutive elective soft tissue hand procedures. Procedures with concomitant implant or incomplete records were excluded. Antibiotic delivery was given at the discretion of the attending surgeon. Patient comorbidities were recorded. Outcomes were measured by the presence of deep or superficial infections within 30 days postoperatively.

Results

The four most common procedures were carpal tunnel release, trigger finger release, mass excision, and first dorsal compartment release. The overall infection rate was 0.66%. All infections were considered superficial, and none required surgical management. In patients who received antibiotic prophylaxis (n = 212), the infection rate was 0.47%. In those who did not receive prophylaxis (n = 388), the infection rate was 0.77%. These differences were not statistically significant (P = 1.00).

Discussion

Most of literature has evaluated the efficacy of antibiotics in hand surgery as a function of trauma or carpal tunnel syndrome. We aimed to expand the body of knowledge by studying a variety of procedures. With increasing threats of withholding reimbursement for non-compliance with SCIP measures, additional data will be useful for guiding future health policy. Furthermore, with increasing antimicrobial resistance, judicious use of antibiotics is warranted. Limitations include retrospective design, lack of randomization, and potential type II error.

Conclusion

The overall rates of infection following elective soft tissue hand surgery are very low. Antibiotics did not appear to confer additional protection from surgical site infection.

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The Etiology of Childhood Limp Presenting to a Tertiary Care Pediatric Emergency Department . . . Risk Factors Predictive of Hospital Admission

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Background

The differential diagnosis for a child with a limp/inability to bear weight is extensive. While many etiologies of limp do not require urgent attention, several orthopedic conditions require hospital admission for management. The purpose of this study is to identify the etiologies of childhood limp presenting to a tertiary care pediatric emergency department, and to isolate patient factors that predict need for urgent orthopedic care.

Methods

Electronic medical records of all patients presenting to a tertiary care pediatric emergency department from 1/1/2010 to 4/1/2010. The search identified 16,056 patients of which 1,776 had a musculoskeletal complaint; of those patients, the medical record was evaluated to delineate individuals with a lower extremity injury, limp, and/or inability to bear weight. These patients underwent a full chart review to determine the exact etiology. Univariate analysis and multiple logistic regression were used to compare groups.

Results

Respiratory complaints were the most common reason for presentation to the emergency department (4,173 patients, 26%), followed by gastrointestinal/abdominal (2,538 patients, 16%), ear, nose, and throat (2,345 patients, 15%), musculoskeletal (1,776 patients, 11%) and dermatological (1,104 patients, 7%). Of the musculoskeletal complaints, 779 patients had a lower extremity injury, limp and/or inability to bear weight. The most common diagnosis was sprain/strain (205 patients, 26%). This was followed by contusion (148 patients, 19%), fracture (110 patients, 14%), cellulitis/abscess (73 patients, 9%), and abrasion/laceration/puncture (61 patients, 8%). Transient synovitis was discovered in 15 patients (1.9%), and septic arthritis in 2 patients (0.3%). An ingrown toenail or avulsion of toenail was found in 14 patients (1.8%). Other causes of limp included animal bite (6 patients, 0.8%) back spasm (5 patients, 0.6%), sickle cell crisis (5 patients, 0.6%), apophysitis (4 patients, 0.5%), burn injury (3 patients, 0.4%), frostbite (2 patients, 0.3%), slipped capital femoral epiphysis (SCFE) (1 patient, 0.2%), psoas abscess (1 patient, 0.1%), deep venous thrombosis (1 patient, 0.1%), rhabdomyolysis (1 patient, 0.1%), testicular torsion (1 patient, 0.1%). Overall, 59 patients (7.6%) who presented with a complaint of a limp were subsequently admitted. These patients represented only 0.4% of the patients who presented to the emergency department. The average age was 8.5 ± 5.4 years old. Duration of symptoms had a mean of 3.1 ± 7.9 days. In regards to mechanism of injury, 30 patients (51%) had a traumatic event and 29 patients (49%) had an atraumatic onset. The most common admitting diagnosis was fracture (21 patients, 36%), followed by infection which included cellulitis/myositis/abscess/psoas abscess/bacteremia with foot contusion/septic joint (16 patients, 27%), transient synovitis (5 patients, 8.5%), sickle cell crisis (4 patients, 6.8%), SCFE (1 patient, 1.7%).

The patients admitted differed significantly from those not admitted in regards to average age, mechanism of injury, presence of a fever, inability to bear, whether or not there was a past medical history, if advanced imaging was obtained, serum white blood-cell count, and if laboratory workup was performed. Multivariate analysis revealed positive clinical predictors of admission to be inability to bear weight, presence of a fever, younger age, and atraumatic mechanism of injury.

Conclusions

The wide differential diagnosis for a child presenting with a complaint of limp/inability to bear weight on the lower extremity makes efficient and accurate diagnosis challenging. In the emergency department, traumatic etiologies predominate with sprain/strain, contusion, and fracture accounting for nearly half of all visits. A thorough history and physical exam, coupled with radiographs, is sufficient to diagnose limp in most cases. Resources such as laboratory studies and advanced imaging are best utilized for younger children without a history of trauma, with inability to bear weight, and a fever upon presentation to assist in establishment of diagnoses that require urgent treatment and/or hospital admission.

Special Event

John Lachman Lecture at the Pennsylvania Orthopaedic Society Fall Meeting

This year's fall 2012 meeting of the Pennsylvania Orthopedic Society meeting titled "Sports Medicine: From Sidelines to Surgery and Back Again!" held an impressive line-up of speakers, including our own Dr. J. Milo Sewards who delivered the above-mentioned annual Lachman Lecture. Beginning with an intriguing history from ancient wars to the introduction of general anesthetic in the Crimean War, the advancements in hand surgery, intramedullary nailing and antibiotics in WWII to the evolving MASH units and urgent evacuations in Vietnam, he described the now-modern TCCC, or Tactical Combat Casualty Care, and the role of damage control orthopedics in the Middle East. While no definitive implantable fixation is used in the combat zone for our troops, the methods and tools of stabilization were outlined along with a myriad of pictures illustrating the trauma that is seen and how it is compared to certain extreme cases at our own home institution. A stimulating discussion was held afterwards describing the ways in which both local fighting militia and insurgents are treated at our surgical units in the combat zones, and the unique ways these cases must be treated. We are all proud of Dr. Sewards' service to our country and honored to have him working and teaching at Temple.

Colin Mansfield, MD

Gregory Gallant, MD, MBA, past president of the Pennsylvania Orthopaedic Society, and J. Milo Sewards, MD

Special Event

The Fourth Annual Delaware Valley Orthopaedic Trauma Symposium

The Fourth Annual Delaware Valley Orthopaedic Trauma Symposium was held on June 15th–16th, 2012. The event took place at the Temple University School of Medicine and was organized by the course chairmen, Dr. Saqib Rehman, Orthopaedic Traumatologist and Assistant Professor of Orthopaedic Surgery and Sports Medicine at Temple University Hospital, and Dr. Asif Ilyas, Associate Professor and Director of the Hand and Upper Extremity Surgery Fellowship at the Rothman Institute. The event was highly attended by athletic trainers, physicians' assistants, local residents and attendings. The day was greatly successful due the efforts of the 20 plus moderators who were able to provide their expertise on various topics. Guest faculty included Peter Cole, MD, Chief of Orthopaedic Surgery at the University of Minnesota as well as David Ring, MD, PhD, Director of Hand Research at Massachusetts General Hospital. This year, the focus of the symposium was on Upper Extremity Fracture Management: Tips and Techniques. Numerous topics were covered including, complex upper extremity injuries, fractures about the elbow, scaphoid and carpal injuries and a plethora of other subjects involving adult and pediatric upper extremity injuries. To supplement the lectures were workshops equipped with sawbones and various implants as well as lectures and clinical case presentations and discussions. The hands-on experience provided excellent opportunity to practice surgical technique as well as avoidance of certain pitfalls that are often encountered in surgery. Onsite were numerous orthopaedic vendors debuting some of their products such as implants and educational textbooks. Furthermore, research posters from Temple and many other local institutions were on display. Overall, the event was well orchestrated and provided an excellent opportunity to enlighten the orthopaedic and medical community on conditions affecting the upper extremity. With such a successful and well-attended event, we look forward to what this year's 2013 symposium will bring.

Emmanuel Atiemo

Special Event

The Howard H. Steel Lecture at the Philadelphia Orthopaedic Society

Presented by:

J. Richard Bowen, MD
Professor, Alfred I. DuPont Institute

"Adult Manifestations of Childhood Hip Diseases"

Another fantastic installment of the annual Howard H. Steel Pediatric Lecture occurred this year. As Dr. and Mrs. Betty-Joe Steel invited the Temple juniors to eat dinner at their table, Mrs. Steel recounted the many stories they have from their vacation ranch in Montana and volunteered some of their early stories from Seattle over the table's very generous glasses of wine and steak buffet.

The lecture following dinner was just as lively and jovial, being presented by the esteemed Dr. J. Richard Bowen. After recounting a case in which he was able to scrub in with the famed Dr. Steel early in his career (and managed to hold an extremely large leg for the entirety of the case without an overly amount of mishap), he delved into a stimulating set of cases. The crowd all enjoyed hearing about Sloshy Steele, the Dega Squirt, Bony Acetabulum and the Fallen Pelvis. In all, we were able to cover his three main talking points of DDH, FAI, and Necrosis. In the end, a stimulating discussion rounded out another successful and humbling Howard Steel Pediatric Lecture, with a round of applause and ovation for both the Guest Speaker and the great man for which the lecture was named.

Colin Mansfield, MD

Front Row: Mrs. Betty-Jo and Dr. Steel; Back row: Matthew L. Ramsey, MD, President of Society; J. Richard Bowen, MD, Guest Speaker; and Donald W. Mazur, MD, Board of Directors, Program Chair

Special Event

Pennsylvania Orthopaedic Society Spring Meeting

The Pennsylvania Orthopaedic Society (POS) hosted its annual Spring Meeting in Miami, Florida at the legendary Fontainbleu Hotel Miami Beach. It seemed only fitting that PA's best and brightest musculoskeletal specialists would gather around the luxurious poolside area of the Fontainbleu, which was featured in bad-boy blockbuster films such as *James Bond-Gold Finger, The Bodyguard, The Specialist,* and *Scarface.* Joining the ranks of those great men of style at the Fontainbleu poolside bar were Temple Orthopaedics Chairman Joe Thoder and resident Rick Tosti, who were obligated to imbibe "something more fruity" than the usual choice lagers (see photo). Shortly thereafter, they were joined by faculty members, Alyssa Schaffer and Wade Andrews, at the Gotham Steakhouse.

The meeting, entitled "Controversies in Upper Extremity Surgery: East vs. West," featured debate-style didactics on various controversial topics. Dr. Thoder was invited to debate Dr. Anthony Romeo, team physician of the Chicago White Sox, on the merits of open reduction internal fixation versus total elbow arthroplasty for acute fractures of the distal humerus. Although Dr. Thoder was our only faculty speaker, Temple Orthopaedics had a significant presence at the meeting. Ortho Resident Rick Tosti presented research entitled "Is Antibiotic Prophylaxis Necessary in Elective Soft Tissue Hand Surgery?" The project was recently given the honor of "Best Presentation" at the Hand and Wrist section at the American Academy of Orthopaedic Surgeons (AAOS) 2012 meeting, and it was similarly well received at the POS. Furthermore, alumnus Asif Ilyas was the co-chairman of the program, moderator of the section on elbow controversies, and speaker of exploring radial nerve palsies after fractures of the humerus. Additionally, alumnus David Yucha was invited to speak about biceps tenodesis in the 40 year old and debated against the option of SLAP repair. Last, former faculty member and sports fellow, John Kelly, lectured on the value of MRI versus ultrasound the diagnosis of rotator cuff tears. Truly showing that old habits die hard, he began his discussion with a video of a chimpanzee urinating a clear yellow stream into its mouth and said "this is the value of ultrasound in diagnosing cuff tears . . . be a man and order an MRI!"

Rick Tosti

Special Event

2012 American Academy of Orthopaedic Surgeons Meeting

Temple Orthopaedics was well represented at the 2012 American Academy of Orthopaedic Surgeons meeting in San Francisco. The chief resident class of 2012, including John Fowler, John Richmond, Jung Park and Nate Bodin, made the trip to the Bay Area and certainly made the most of the experience. In addition to going to several lectures at the San Francisco Convention Center, they took full advantage of beautiful Northern California by taking a day trip with several of the faculty to Napa Valley for a wine tasting tour. They also "shacked up" in quite a posh suite in the downtown San Francisco with a lovely, picturesque view of the City by the Bay.

The meeting itself featured three poster presentations, two podium presentations and a scientific exhibit from the Temple University Department of Orthopaedics and Sports Medicine. Rick Tosti, second-year resident, gave an extremely well-received presentation on his paper, *Is Antibiotic Prophylaxis Necessary in Clean Soft Tissue Hand Surgery?,* a paper which later went on to get published in the journal, *Orthopedics.* John Fowler presented a paper from the Kinesiology Department at Temple University on *Athletic Induced mTBI and Catastrophic Intracranial Injuries: Helmet Efficacy and Predisposing Profiles.* Third-year resident Rich Han presented his poster titled, *Evaluation of Glenohumeral Bone Defects in Shoulder Instability: Interobserver Reliability Across Modalities.* Fourth-year resident Emmanuel Atiemo displayed his poster, *Why Do Corticosteroids Work in Stenosing Tenosynovitis: Histologic Evaluation of the Tenosynovium.* Fellow fourth-year resident Matt Kleiner presented his poster, *Enoxaparin and Warfarin for VTE prophylaxis in THA: To Bridge or Not to Bridge.* Finally, faculty member Saqib Rehman and Rich Han enlightened passers-by with a captivating exhibit conceived of Temple's intimate experience with orthopaedic injuries from penetrating gun shot wounds titled, *Ballistics: Current Trends in Firearms (Experiences from Temple University Hospital).*

Congratulations to all of the participants for their hard work and dedication to their research and for representing Temple well. Keep up the good work!

Matthew T. Kleiner

Special Event

Resident Research Day 2012

Held on April 21, 2012 in the Clancy Conference Room, Resident Research Day for Temple University Department of Orthopaedic Surgery and Sports Medicine displayed the most recent contributions to the department's excellent research tradition. The program cover featured a drawing of Dr. John Lachman making a teaching point, and each presentation demonstrated the pursuit of better understanding of orthopaedic medicine and more effective clinical solutions that his legacy engenders.

The program started with an excellent grand rounds presentation by Nancy Pleshko, PhD, a professor in the Department of Bioengineering at Temple University and director of Temple's Tissue Imaging and Spectroscopy Laboratory (TISL). Dr. Pleshko is a leading researcher in cartilage imaging and analysis, and her talk, "Cartilage Degradation and Repair: Progress Towards Clinical Spectroscopic Assessment," reviewed the development of infrared spectroscopic analysis to evaluate cartilage lesions and cartilage repair techniques. In addition to educating the crowd regarding the molecular properties of cartilage that make it such an important factor in orthopaedic pathology, Dr. Pleshko discussed potential clinical benefits of translating her bench laboratory techniques directly to *in vivo* analysis through the development of spectroscopic probes. Throughout her talk, Dr. Pleshko was able to keep focus on the link between basic science principles and their clinical applications. And being from our home institution, Dr. Pleshko and the TISL offer endless possibilities as a resource for collaboration in the future.

The day continued with strong resident presentations that addressed many hot topics in modern orthopaedic practice. Matthew Kleiner, MD addressed postoperative anticoagulation protocols with his presentation "The Lovenox Leak: To Bridge or Not to Bridge." Richard Han, MD presented "Evaluation of Glenohumeral Bone Defects in Shoulder Instability: Inter-observer Reliability Across Modalities," which had previously garnered a lot of interest as a poster presentation at the AAOS national meeting. And Samuel Popinchalk, MD gave a talk titled "Development of a Pollicization Clinical Outcomes Measure" that tied in unique practices at Temple's affiliate, Shriners Hospital for Children. A highlight of this section of the program was the presentation, "A Review of Atypical Femoral Fractures from a Tertiary Care Teaching Hospital. Is there an Alarming Trend?" by John-David Black, MD. Dr. Black is a resident at St. Luke's University's Department of Orthopaedic Surgery. His inclusion in the program brought in new research and fresh perspective from an area institution outside of the Temple network, and the department was happy to welcome him. It also opened up discussion regarding the "Own the Bone" program, which the department has been trying to initiate at Temple.

Resident Research Day also serves as a culmination for the hard work of each class of senior residents. The PGY-5 class once again showed that their leadership skills extend from the hospital wards and the operating rooms to the laboratory. Presentations were as follows: "Flouroscopic Evaluation of DRUJ Violation with Plating of Distal Radius Fractures" by Nathan Bodin, MD; "Bacterial Adherence to Barbed Monofilament Suture in a Contaminated Wound Model" by John Fowler, MD; "Biomechanical Comparison of Locked Plating and Spiral Blade Retrograde Nailing of Supracondylar Femur Fractures" by Jung Park, MD; and "Access to Care Following Acute Anterior Cruciate Ligament Injury: 2 Year Evaluation of a Single Urban Academic Center" by John Richmond.

In the end, presentations were judged by a panel that included Dr. Pleshko and numerous department attendings. Drs. Fowler, Richmond, and Park were awarded first, second, and third place prizes respectively. On a day that showcased the department's strong research initiative, there were also intriguing collaboration opportunities, interesting outside perspectives that promoted meaningful academic discussion, and an award-winning send off for the PGY-5 class. And the prevailing sentiment of the day was excitement for what the department can produce over the next academic year.

Scott Barbash, MD

Special Event

Alumni Day 2012

This year's Alumni Day began on a rainy Friday morning on May 4th, 2012 with a record number of attendees gathering at Lulu Country Club in Glenside, PA. A long line of Temple physicians were in attendance, from the class of 1968 to the newly established interns who were just finishing their first year. A series of lectures from program alumni, including Glenn Lieberman, MD, Joseph Milo Sewards, MD and Neil MacIntyre, MD, were given on trauma, joint replacement, and other current topics.

The day was dedicated to Dr. Edward Resnick. Dr. Resnick was a compassionate man who devoted his life to orthopedics and his patients. He was humorous and witty, and truly enjoyed educating his residents. He worked as an x-ray technician in the Army Medical Corps in Europe from 1944 to 1946 and travelled to Kenya, Tunisia, Peru, and the Dominican Republic to share his orthopaedic knowledge with other physicians and healthcare workers.

By the time lectures and lunch finished, the sun had swept the rains with temperatures climbing to a beautiful 85 degrees. The golf course, designed by legendary architect Donald Ross, was host to several foursomes of golfers competing for awards such as Closest to the Pin and Longest Drive. Residents uncertain of their golfing skills took to the fairways on carts, but none could be persuaded to caddy.

Because I know stories change face over time, I feel as though I should put this in writing: Dr. Milo Sewards, our program director, won the Closest to the Pin award but Sam Popinchalk, PGY3 and resident golf favorite, was sidelined due to an injury which allegedly occurred just hours prior to the event as they crossed paths in the parking lot.

At cocktail hour and dinner, the current residents listened to Dr. Thoder, Chairman of Temple Orthopaedics, reminisce with his past resident classmates about "the good old days." As they laughed and exchanged stories, it was clear that our orthopaedic program is a second family few are lucky to experience, regardless of where graduates start their new careers or the fields they pursue.

Justin Iorio

Special Event

Second Annual Ponderosa Bowl

December 8, 2012

On an early December morning on the muddy plains of the Ponderosa, 12 gathered for the Second Annual Ponderosa Bowl (formerly the Shrine Bowl). The Cherry Team was led by veteran and captain John Fowler while the White Team was spearheaded by the triple threat of chief residents Katherine Criner, Joseph Dwyer and Matthew Kleiner. It was a back and forth battle during the first half with a tie game of 20–20 at the midway point. The White Team started the second half with a long scoring drive, highlighted by Stephen Refsland's personal foul for an inappropriate "high tackle" on Criner which kept the drive alive. International threat, Liam Woozley, scored a play later on a short wide receiver screen to take the lead 28–20. However, at the end of the day, the depth of the Cherry squad proved too much as they scored four consecutive touchdowns to close the game. Craig Steiner was able to bounce back one play after an embarrassing and costly (ruined jeans) slip in the mud to throw an essential block on the go ahead score by Rookie of the Year Jim Lachman. It was all over for the White Team when a questionable pass interference call on Christopher Haydel in the defensive red zone led to Fowler's second score on the day, putting the game well out of reach. Fowler and Lachman each had multiple scores on the day, allowing the Cherry Team to take home the coveted Joseph Torg Cup. Final score: Cherry 48–White 28.

Mark Solarz

Departmental News

Faculty

Temple University Department of Orthopaedic Surgery and Sports Medicine

Chairman

Joseph Thoder, MD, *The John W. Lachman Professor*

Professors

William DeLong, MD
Pekka Mooar, MD
Ray Moyer, MD, *The Howard H. Steel Professor*
Joseph Torg, MD
F. Todd Wetzel, MD, *Vice Chairman*

Associate Professors

Easwaran Balasubramanian, MD
Saqib Rehman, MD
Bruce Vanett, MD
Albert Weiss, MD

Assistant Professors

Joseph Eremus, MD
Christopher Haydel, MD
Eric Kropf, MD
Matthew Lorei, MD
Stanley Michael, MD
Alyssa Schaffer, MD
J. Milo Sewards, MD

Adjunct Faculty — Philadelphia Shriners Hospital

Randal Betz, MD, *Chief of Staff*
Philip Alburger, MD
Patrick Cahill, MD
Richard Davidson, MD
Corinna Franklin, MD
Scott Kozin, MD
G. Dean MacEwen, MD
Howard Steel, MD, *Emeritus Chief of Staff*
Joshua Pahys, MD
Amer Samdani, MD
William Schrantz, MD
Harold van Bosse, MD
Daniel Zlotolow, MD

Adjunct Faculty — Abington Memorial Hospital

Andrew Star, MD, *Chief of Orthopaedics*
Shyam Brahmabhatt, MD
David Craft, MD
Matthew Craig, MD
Greg Galant, MD
Michael Gratch, MD
Victor Hsu, MD
David Junkin, Sr., MD
Moody Kwok, MD
Guy Lee, MD
Thomas Peff, MD
T. Robert Takei, MD
Jeffrey Vakil, MD

Adjunct Faculty — St. Christopher's Hospital for Children

Peter Pizzutillo, MD, *Chief of Orthopaedics*
Kiersten Arthur, MD
Alison Gattuso, DO
Martin Herman, MD
Michael Kwon, MD
Juan Realyvasquez, MD
Joseph Rosenblatt, DO
Shannon Safier, MD
Michael Wolf, MD

Temple University Hospital
Department of Orthopaedic Surgery and Sports Medicine
Faculty 2012–2013

Joseph Thoder, MD
John W. Lachman Professor
Chairman
Hand & Upper Extremity
General Orthopaedics

Eswarian Balasubramanian, MD
Joint Reconstruction
General Orthopaedics

William DeLong, MD
Orthopaedic Trauma
Sports Medicine
General Orthopaedics

Joseph Eremus, MD
Foot and Ankle
General Orthopaedics

Christopher Haydel, MD
Orthopaedic Trauma
General Orthopaedics

Eric Kropf, MD
Sports Medicine
General Orthopaedics

Paul Lento, MD
Physical Medicine
and Rehabilitation

Matthew Lorei, MD
Joint Reconstruction
General Orthopaedics

Stanley Michael, MD
Sports Medicine
Joint Reconstruction
General Orthopaedics

Pekka Mooar, MD
Sports Medicine
Joint Reconstruction
General Orthopaedics

Ray Moyer, MD
Howard Steel Professor
Sports Medicine

Saqib Rehman, MD
Orthopaedic Trauma
General Orthopaedics

Alyssa Schaffer, MD
Hand Surgery

J. Milo Sewards, MD
Sports Medicine

Joseph Torg, MD
Sports Medicine

Bruce Vanett, MD
General Orthopaedics

Albert Weiss, MD
Hand & Upper Extremity
General Orthopaedics

F. Todd Wetzel, MD
Vice-Chairman
Spine Surgery

Orthopaedic Surgery
& Sports Medicine
Temple University Health System

Temple University Hospital
Department of Orthopaedic Surgery and Sports Medicine
House Staff 2012–2013

Emmanuel Atiemo, MD
PGY-5

Katharine Criner, MD
PGY-5

Joseph Dwyer, MD
PGY-5

Matthew Kleiner, MD
PGY-5

Scott Barbash, MD
PGY-4

Richard Han, MD
PGY-4

Emeka Nwodim, MD
PGY-4

Samuel Popinchalk, MD
PGY-4

Stephen Refsland, MD
PGY-3

Craig Steiner, MD
PGY-3

Rick Tosti, MD
PGY-3

Justin Iorio, MD
PGY-3

Rupam Das, MD
PGY-2

Colin Mansfield, MD
PGY-2

Kaziemierz Komparda, MD
PGY-2

Mark Solarz, MD
PGY-2

Dustin Greenhill, MD
PGY-1

James Lachman, MD
PGY-1

Anastassia Persidsky, MD
PGY-1

Ariana Trionfo, MD
PGY-1

Temple University Department of Orthopaedics — Publications by Faculty 2012–2013

Podium/Poster Presentations

2013

Padlakar M, McGoverin C, Barbash S, Kropf EJ, Pleshko N. Differentiation of ligament and tendon with near infrared spectroscopy. *ORS Annual Meeting,* San Antonio, Texas, January 26–29, 2013 (poster).

Whitaker JJ, Williamson C, Fowler JR, Kleiner MT, Haines C, Herman MJ. The Etiology of Childhood Limp Presenting to a Tertiary Care Pediatric Emergency Department: Risk Factors of Hospital Admission. *AAOS Annual Meeting,* Chicago, IL, March 18–23, 2013 (poster).

Steiner C, Richmond J, Solarz M, Kropf EJ. Access to care following acute anterior cruciate ligament injury: 2 year evaluation at a single urban center. *AANA Annual Meeting,* San Antonio, Texas, April 25–27, 2013 (e-poster).

2012

Mascarenhas R, Kropf EJ, Irrgang JJ, Chu CR, MacDonald PB, Harner CD. Inter-rater reliability in the grading of articular cartilage lesions of the knee: A comparison between arthroscopic and magnetic resonance grading. *AANA Annual Meeting,* Orlando, FL, May 17, 2012 (podium).

Mascarenhas R, Kropf EJ, Irrgang JJ, Chu CR, MacDonald PB, Harner CD. Arthroscopic and magnetic resonance imaging of articular cartilage lesions of the knee: Inter-rater and reliability between modalities. *Canadian Orthopaedic Association 67th Annual Meeting,* Ottawa, ON, Canada, June 8, 2012 (podium).

Han R, Mullen J, Ali S, Kropf EJ. Radiographic, CT and MRI Evaluation of Glenohumeral Bone Defects in Recurrent Shoulder Instability: Interobserver Reliability and Agreement Between Modalities. *AANA Annual Meeting,* Orlando, FL May 17–19, 2012 (e-poster).

Duncan I, Kleiner M, Atiemo E, Michael S. Kropf EJ, Sewards JM. Is lateral decubitus position better than beach chair at providing visualization of the posterior labrum of the shoulder? *AANA Annual Meeting,* Orlando, FL, May 17–19 2012 (e-poster).

Tosti R, Fowler JR, Dwyer J, Gaughan JP, Ilyas AM, Thoder JJ. Is antibiotic prophylaxis necessary in clean soft tissue hand surgery? *American Academy of Orthopaedic Surgeons Annual Meeting,* February 7–11, 2012: San Francisco, CA.

Fowler JR, Perkins TA, Buttarro BA, Truant AL, Torg JS. Bacteria adhere less to barbed monofilament than other suture types in a contaminated wound model. *Orthopaedic Research and Education Foundations (OREF) Orthopaedic Research Society (ORS) Resident Research Competition – Philadelphia Region,* June 8–9, 2012: Philadelphia, PA.

First Place Winner, Podium Presentation

Torg JS, Boden BB, Hirsch H, Fowler JR, Gaughan JP, Comstock D, Tiernery R. Athletic Induced mTBI and Catastrophic Intracranial Injuries: Helmet Efficacy and Predisposing Profiles. *American Academy of Orthopaedic Surgeons Annual Meeting,* February 7–11, 2012: San Francisco, CA.

Publications in Peer Reviewed Journals

2013

Kropf EJ, Shen W, van Eck C, Musahl V, Irrgang JJ, Fu FH. ACL-PCL and intercondylar notch impingement: Magnetic Resonance Imaging of native and double-bundle ACL-reconstructed knees. *Knee Surg Sports Trauma* 2013 Mar;21(3):720–5.

2012

Mascarenhas R, Tranovich MJ, Kropf EJ, Fu FH, Harner CD. Bone-patellar tendon-bone autograft vs hamstring autograft anterior cruciate ligament reconstruction in the young athlete: A retrospective matched analysis with 2–10 year follow-up. *Knee Surg Sports Trauma* 2012 Aug;20(8): 1520–7.

Auerbach AD, Rehman S, Kleiner MT. Selective transcatheter arterial embolization of the internal iliac artery does not cause gluteal necrosis in pelvic trauma patients. *J Ortho Trauma.* 2012 May;26(5):290–5.

Fowler JR, Criner K, Craig MR. Prophylactic Intramedullary Fixation for Bisphosphonate-related Subtrochanteric Stress Fracture. *Orthopaedics* 2012 Jun 1;35(6):e954–7.

Fowler JR, Kleiner MT, Das R, Gaughan JP, Rehman S. Assisted closure of fasciotomy wounds: A descriptive series and caution in patients with vascular injury. *Bone and Joint Research* 2012 March;1(3):31–35.

Fowler JR, Craig MR. Association of low energy femoral shaft fractures with bisphosphonate use. *Orthopaedics* 2012 Jan;35(1):20.

Fowler JR, Ogrich L, Evangelista P, Schaffer AA. Assessing injection techniques in the treatment of trigger finger. *Modern Plastic Surgery* 2012 Oct;2(4):83–86. doi: 10.4236/mps.2012.24020.

Piposar J, Fowler JR, Gaughan JP, Rehman S. Race may not effect outcomes in operatively treated tibia fractures. *Clin Orthop Relat Res* 2012 May;470(5):1513–7.

Rehman S, Salari N, Codjoe P, Rehman M, Gaughan J. Gunshot femoral fractures with vascular injury: A retrospective analysis. *Orthop Surg* 2012 Aug;4(3):166–71.

Tosti R, Fowler JR, Dwyer J, Gaughan JP, Ilyas AM, Thoder JJ. Is antibiotic prophylaxis necessary in clean soft tissue hand surgery? *Orthopaedics* 2012 Jun;35(6):e829–833.

Textbook

Ilyas AM, Rehman S, eds. *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Book Chapters/ePublications

Rehman S, Criner K. "Tibial shaft fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Ilyas AM, Rehman S. "Distal humerus fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Manthe M, Suk M, Rehman S. "Distal tibia fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Haydel C, Rehman S. "Femoral neck fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Mellon M, DeLong W, Rehman S. "Distal femur fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications, Volumes 1 and 2.* Jaypee Med. Publishing, 2013.

Kleiner M, Kropf EJ. *Osteotomy for Knee Osteoarthritis — A Lost Art?* Orthopedics Hyperguide http://www.ortho.hyperguides.com/index.php?option=com_content&view=article&id=2336. January 2013, Vindico Medical Education Publisher.

Fowler JR, Thoder JJ. "Radial Head Fractures and Elbow Dislocations." In *Contemporary Surgical Management of Fractures and Complications.* Ilyas AM, Rehman S. Jaypee Medical Publishers New Delhi, India. April 2013.

Tosti R, Sheikh E, Ilyas AM. "Humeral Shaft Fractures." In Ilyas AM, Rehman S, eds., *Contemporary Surgical Management of Fractures and Complications.* Jaypee Medical Publishers, New Delhi, India, April 2013.

Kleiner M, Kropf EJ. "Patellofemoral Instability: Evaluation and Management," Chapter 10, pp 195–209. *The Knee: Current Concepts in Kinematics, Injury Types, and Treatment Options.* Nova Science Publishers Inc. August 2012, Hauppauge, New York.

Fowler JR, Guille J. "The Limping Child." In Elzouki AY, ed., *Textbook of Clinical Pediatrics.* Second Edition, Volume IV. Section 25 Pediatric Orthopaedics 2013;(405):3909–3916.

Wang JH, Kropf EJ, Fu FH. Double Bundle ACL Reconstruction. In Ryu RK, ed., *AANA Advanced Knee Arthroscopy.* 2010.

Kropf EJ, Haner CD. "Meniscus Root Repair." In Fu FH, ed., *Masters Techniques in Sports Medicine.* 2010.

Wong AK, Kropf EJ, Fu FH. "Anterior Cruciate Ligament." In Ranawat A, Kelly B, eds., *Musculoskeletal Examination of the Hip and Knee: Making the Complex Simple.* 2010.

Mascarenhas R, Kropf EJ, Harner CD. "Ligamentous Injuries of the Knee." In Sivananthan S, Miller MD, Sherry E, Warnke PH, eds., *Mercer's Textbook of Orthopaedic and Trauma Surgery.* 10th Edition. London, UK, 2009.

Torg, JS. "Cervical Spine Problems in the Adult Athlete in Orthopedic Sports Medicine, 3rd Edition." DeLee and Drez, eds. WB Saunders Co., Philadelphia, pp 665–700, 2009.

Wetzel FT. "Image Guided Therapy and Its Relationship to Conventional Surgical Management." In Mathis JM, Golovac S, eds., *Image-Guided Spine Interventions, 2nd Edition.* New York: Springer Science+ Business Media, 2009, pp 267–289.

Wetzel FT, Saulino ML. "Surgery for Chronic Pain." In Herkowitz H, Garfin S, Eismont FJ, Bell GR, eds., *Rothman-Simeone The Spine, Sixth Edition.* Philadelphia: Saunder Elesevier, 2009 (in press).

Grand Rounds 2012–2013

August 15	Kurosh Darvish, PhD: "Introduction to Spinal Biomechanics" Katharine Criner, MD: "The Relevance of Biomechanical Studies to Clinical Orthopaedic Practice"
September 15	Hassan Mir, MD: "Culturally Competent Care" Samuel Popinchalk, MD: "Complications with Subtrochanteric Femur Fracture Management"
October 17	Peter Pizzutillo, MD: "Developmental Dysplasia of the Hip" Scott Barbash, MD: "Complications with Management of Periarticular Fractures in the Pediatric Knee"
November 3	Andrew Star, MD: "Is There Really Such a Thing as Minimally Invasive Joint Replacement?" Richard Han, MD: "Vascular Complications in Total Knee Replacements"
November 21	Harold van Bosse, MD: "An Orthopaedist's Algorithm for Treatment of the Arthrogrypotic Child" Matthew Kleiner, MD: "Proximal Femoral Focal Deficiency"
December 12	Christopher Haydel, MD: "Staying Out of Trouble with Tibial Plateau Fractures" Emeka Nwodim, MD: "Proximal Tibia Fractures in Children and Adolescents"
January 16	F. Todd Wetzel, MD: "Conflicts of Interest" Stephen Refsland, MD: "Current Legal and Ethical Issues in Sports Orthopaedics"
January 19	Paul Lin, MD: "Pars Defect Repair" Joe Dwyer, MD: "Spondylolysis and Spondylolisthesis"
February 13	Mohit Bhandari, MD, PhD: "Think Big" Emmanuel Atiemo, MD: "Current Evidence with Intertrochanteric Hip Fracture Management"
March 9	Keith Wapner, MD: "Management of Hallux Valgus Deformity in the Adult" Richard Tosti, MD: "Stress Fractures of the Tarsal Navicular and Fifth Metatarsal"
March 27	Peter Lelkes, PhD: "Smart Biomaterials for Orthopaedic Applications" Justin Iorio, MD: "Methods to Minimize Blood Loss in Pediatric Spine Deformity Surgery"

Dr. Rehman and Dr. Bhandari


Dr. Torg and Dr. Wapner

Instructions to Authors

Editorial Philosophy

The purpose of the *Temple University Journal of Orthopaedic Surgery & Sports Medicine (TUJOSM)* is to publish clinical and basic science research performed by all departments of Temple University that relate to orthopaedic surgery and sports medicine. As such, *TUJOSM* will consider for publication any original clinical or basic science research, review article, case report, and technical or clinical tips. All clinical studies, including retrospective reviews, require IRB approval.

Editorial Review Process

All submissions will be sent to select members of our peer review board for formal review.

Manuscript Requirements

Manuscripts are not to exceed 15 double spaced type-written pages and/or 5,000 words (minus figures/tables/pictures). The manuscript should contain the following elements: Title page, Abstract, Body, References, and Tables/Legends. Pages should be numbered consecutively starting from the title page.

(1) Title Page — The first page, should contain the article's title, authors and degrees, institutional affiliations, conflict of interest statement, and contact information of the corresponding author (name, address, fax, and email address).

(2) Abstract — The second page, should be a one-paragraph abstract less than 200 words concisely stating the objective, methods, results, and conclusion of the article.

(3) Body — Should be divided into, if applicable, Introduction, Materials & Methods, Results, Discussion, and Acknowledgements. Tables and figures (in JPEG format) with their headings/captions should be listed consecutively on separate pages at the end of the body, not continuous within the text.

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1. Based on company reports for global sales Jan-Dec 2011. **2.** PMA approved P900009/Supplement 6, Summary and Effectiveness Data. **3.** Heckman JD, Ryaby JP, McCabe J, Frey JJ, Kilcoyne RF. Acceleration of tibial fracture healing by non-invasive, low-intensity pulsed ultrasound. *J Bone Joint Surg Am.* 1994;76:26-34. **4.** Kristiansen TK, Ryaby JP, McCabe J, Frey, Roe LR. Accelerated healing of distal radial fractures with the use of specific, low-intensity ultrasound. *J Bone Joint Surg AM.* 1997;79(7):961-973.

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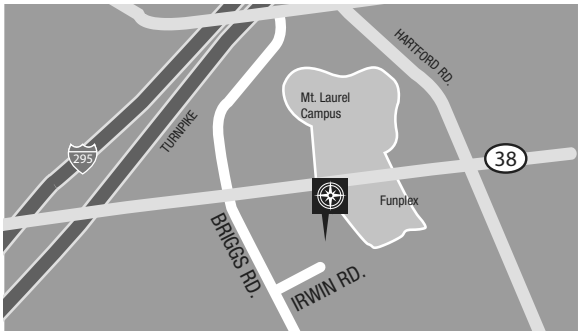

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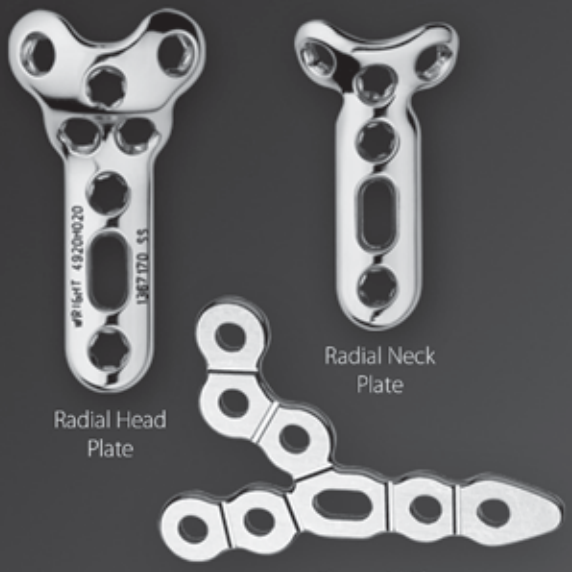

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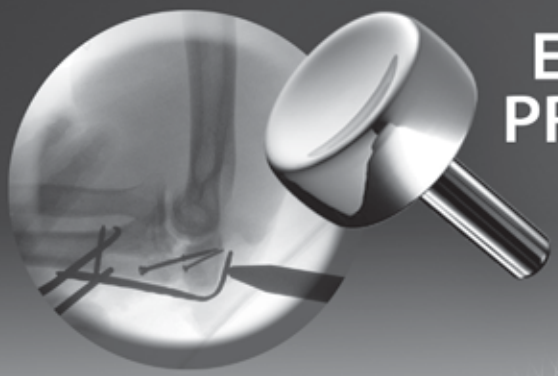

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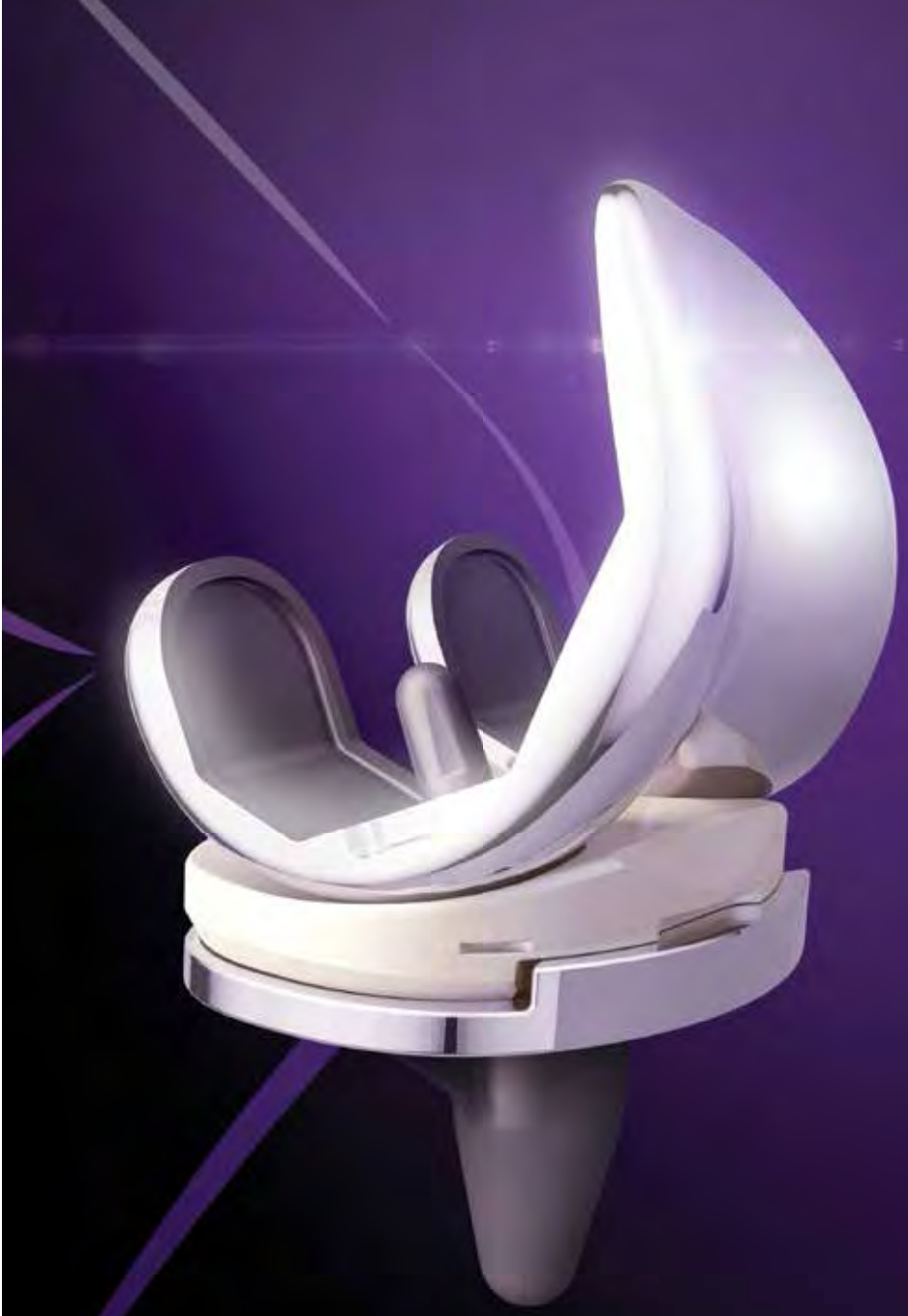

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