The University of Pennsylvania Orthopaedic Journal

Volume 33 June 2023

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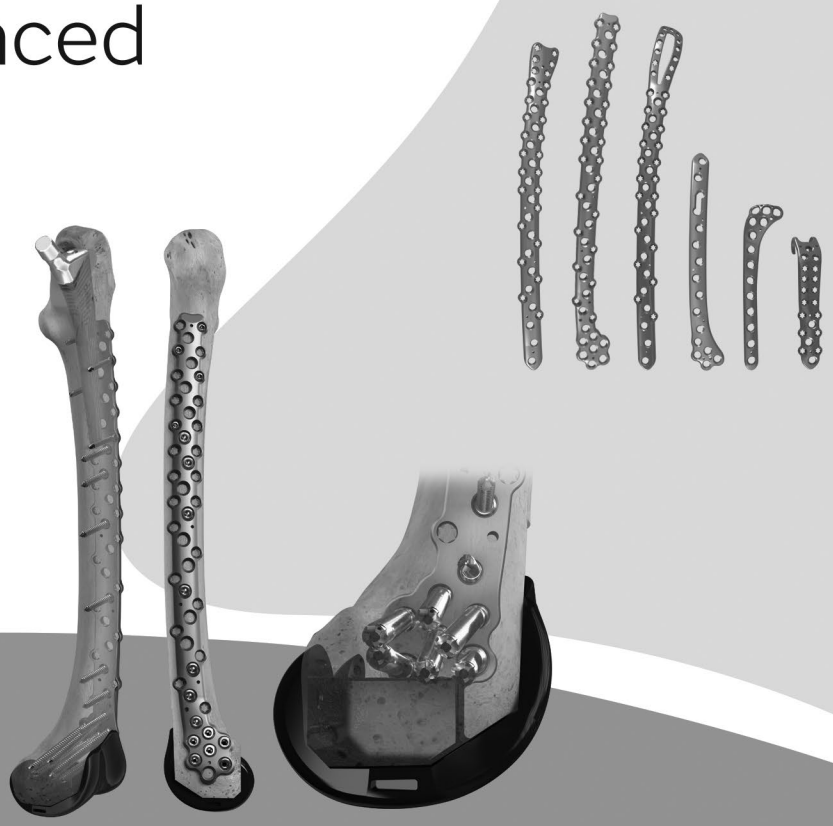

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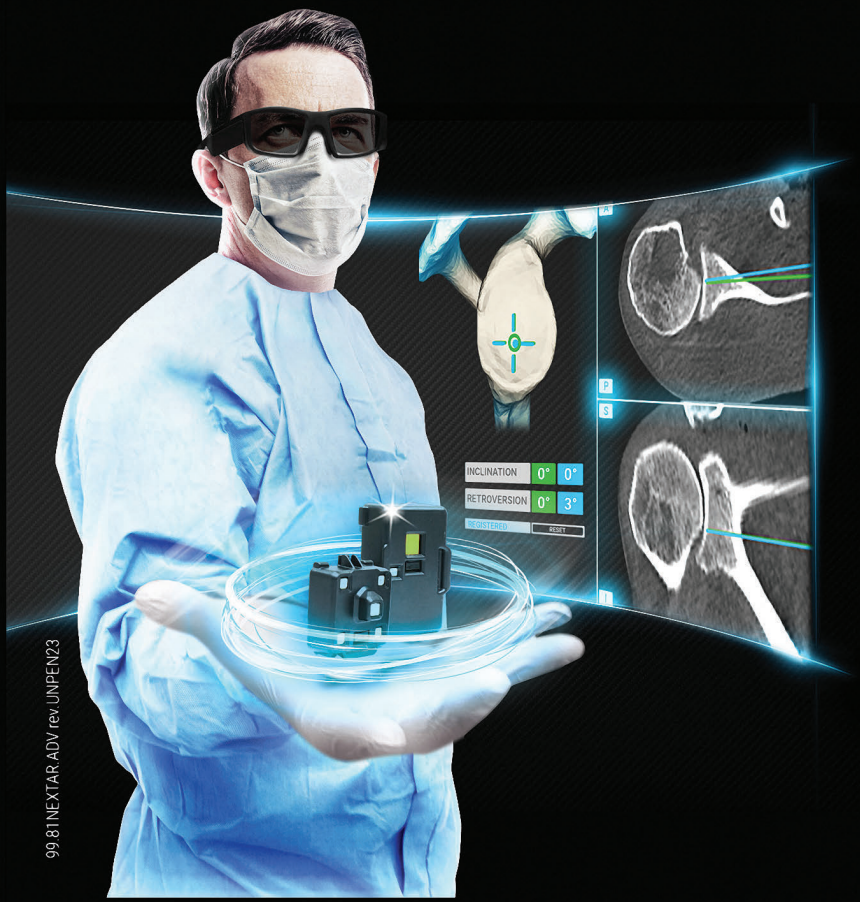

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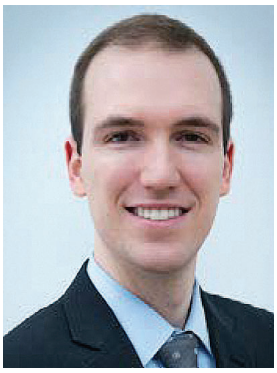


Welcome to the 33rd edition of the University of Pennsylvania Orthopaedic Journal (UPOJ). Founded in 1986 under the guidance of Dr. Carl T. Brighton, the UPOJ remains a testament to the department's commitment to innovation and research in the field of orthopaedics. This year, as we embark on a new edition, our focus is centered on the transformative power of innovation in shaping the future of orthopaedics.

Innovation has become the driving force behind advancements in healthcare, revolutionizing the way we approach patient care and tackle complex musculoskeletal challenges. In this year's journal, we aim to explore the diverse realms of innovation within orthopaedic surgery and delve into the groundbreaking discoveries, technologies, and strategies that are reshaping the landscape of our field.

Healthcare is an ever-evolving domain, and embracing innovation has become essential for those who strive to provide the highest quality of care to their patients. From novel surgical techniques and regenerative therapies to the integration of artificial intelligence and virtual reality in diagnostics and treatment, the possibilities for innovation in orthopaedics are boundless. This year's edition will showcase the cutting-edge research, collaborative efforts, and visionary ideas that are pushing the boundaries of orthopaedic innovation.

We are honored to dedicate this year's edition to Dr. L. Scott Levin, a visionary and trailblazer in the field of orthopaedics. Dr. Levin's unwavering commitment to innovation, patient care, and medical education has transformed the landscape of orthopaedics and inspired numerous aspiring surgeons.

As a distinguished leader, Dr. Levin has served as chairman of the department of orthopaedic surgery at Penn since 2009. During this time, he has spearheaded groundbreaking research and pioneered advancements in the field. His expertise in hand surgery, microsurgery, and limb reconstruction has not only improved the lives of patients but has also elevated the standard of care worldwide. He heads Penn's Vascularized Composite Allotransplantation, and he performed the world's first bilateral hand transplant for a child in 2015. Dr. Levin's unwavering dedication to his craft, combined with his empathetic and compassionate approach, has garnered him the utmost respect from colleagues and patients alike, resulting in him receiving the prestigious AAOS Kappa Delta Award in 2022.

Beyond his clinical practice, Dr. Levin has made lasting contributions to medical education, mentoring and inspiring numerus residents and fellows from around the world. His tireless efforts to disseminate knowledge, promote research, and enhance surgical training have had a profound and lasting impact on the field. His legacy will continue to inspire and guide us as we strive for excellence and innovation in the pursuit of improving patient care.

We also acknowledge the valuable contributions of our faculty editors, Dr. Samir Mehta and Dr. James Carey who have played valuable roles in shaping the content of this year's edition.

As a resident-run publication, the UPOJ thrives on the dedication and contributions of our talented team. We express our heartfelt appreciation to the resident section editors and acknowledge their invaluable efforts in curating this year's journal.

Furthermore, we extend our thanks to our industry sponsors, whose generous financial support enables the journal to continue promoting the educational and research missions of Penn Orthopaedics.

We are pleased to announce that this year's edition, along with past editions, is available for free online at www.upoj.org. We encourage all readers to subscribe to the journal at www.upoj.org/subscribe and stay connected with the latest advancements in orthopaedic innovation.

It has been a privilege to serve as editors for the 33rd edition of the UPOJ. We hope that this year's journal not only informs and inspires, but also sparks new ideas and collaborations that will shape the future of orthopaedics through the power of innovation.

Letter from the Chair: I am the Bullfrog

L. Scott Levin, MD, FACS, FAOA

I was prepared to have this UPOJ Chairman's letter be the fourteenth and last time that I would write this report. Over the years, I have tried to highlight achievements of our Department and our people. As a team we have made enormous progress in all missions The last three years have been challenging due to the effects of the pandemic that have had impact on how we deliver clinical care, educate our students, residents and fellows, and conduct clinical and basic science research. That being said, every challenge is an opportunity to think differently and execute in new ways. Despite some headwinds- we are stronger now than we were three years ago. Our clinical care and expansion of Penn Orthopedics locally and globally is significant. For example, Samir Mehta, Derek Donegan, Steve Kovach and I have been helping our Ukrainian Orthopedic colleagues manage civilian and soldier extremity injuries using a ZOOM platform four days a week. In real time at 6 am, we review x-rays, pictures of major wounds and advise on treatment given some of the limitations of our courageous and tireless colleagues. Dr, Stephen Kovach and I travelled to Heidelberg Germany last December to teach soft tissue reconstructive teachniques to 16 Ukranian surgeons. Give a man or woman a fish-they eat for a day, teach them to fish-they eat for a lifetime. Recently Dr. Kovach travelled to Ukraine to perform free flaps on several patients. Our Orthoplastic efforts will continue and intensify- the need is great and sharing experience and knowledge is helping to make a difference.

Under the leadership of Daniel Farber, Stephen Liu and Cara Cipriano we continue to optimize education at every level. Our commitment to Penn medical students is reflected in our ratings and feedback from our students who participate in the 200 course. Our residents continue to matriculate in their first or second choice for fellowship-year in and year out. Our written and oral board examination pass rates remain extremely high, and our resident ranks continue to reflect our commitment to diversity at every level. The named lectureships and visiting professor program I consider to be the best in the country. Optimizing utilization of the Human Tissue Laboratory enhances anatomic knowledge of our learners and faculty.

Our research mission has an amazing track record. Although we were ranked #2 in the country this year for NIH funding, Dr. Soslowsky and several of our clinical and research faculty were awarded an 8 million dollar NIH grant to study the Achilles tendon. We certainly will be recognized for this monumental achievement next year.

We continue to use our strategic plan, crafted in 2020 to guide our day to day activities, and we have made considerable progress in "C. L. I. G" (Culture, Leadership, Innovation and Growth).

As part of our strategic plan, we have doubled down on the importance of Leadership. The Michael Kelly Wharton leadership program is a crown jewel of our residency and distinguishes us from other trained programs. I study leadership and read a lot about leadership-and in turn I believe I have "learned" to lead the department more effectively.

Now I come to the story of "the Bullfrog". Admiral William McRaven's most recent book is titled: The Wisdom of the Bullfrog"(Leadership Made Simple- but not Easy) . Admiral McRaven served as the longest active duty SEAL in the Navy-hence the name Bullfrog which designates this longevity. I am the most senior clinical department Chairman at Penn Medicine following the transition from Dr. Sean Grady in the Department of Neurosurgery to Dr. Daniel Yoshor in 2020. Every Clinical Chair at Penn was appointed after me and so by self-decree- I am "the bullfrog" of Chairs.

My term as Chair will continue for another year at the request of Dean Larry Jameson and Penn CEO Mr. Mahoney during which time a new search for my successor will begin. I am confident that the search committee will identify a new leader, and I will do all I can to support the search and further optimize the Department's outstanding national profile while we complete the search. In every enterprise a change in Leadership is essential to assure continued success. In fact, leaders can "overstay their welcome" to the detriment of the organization. The manner in which the change of authority and command occur and a seamless transition is essential to continue our momentum and trajectory.

In reading Admiral McRaven's book, I reflected on the leaders I have observed, leaders that I have reported to, organizations that I have led, and leadership roles I have had in academic medicine.

Let's take the principle in each chapter and allow me to interpret the Admiral's message as it relates to my role as Department Chairman.

1. Sua Sponte: The motto of Army Rangers. Broadly translated it means "by their own accord". Entrust those you lead to do the right thing…to take matters

into their own hands knowing what is expected. I have complete trust in our residents, faculty fellows, PAs and administrative staff to work for the common good of our patients and our other missions. Our code of conduct, Professionalism committee and defining expectations all help our team members know what is expected of them.

2. Stamina-The SEALS motto is-"The only easy day was yesterday". While our day to day work is intense and demands much of each of us, our strong work ethic highlighted by Sir William Osler (which he considered the key to success) lets us overcome the challenges that face us each day. As a matter of fact, I get up in the morning and ask myself- "what problem will I try to solve today?" I hope you do the same. Day in, day out we work as effectively as we can, with recognition that burnout can occur and we must guard against that.
3. "Who Dares Wins" This was the Special Air Service WWII motto coined by Sir David Sirling. It means- "take risks". It does not mean gamble or fail to prepare for a surgical procedure. It means that every day, as caregivers and surgeons we take risks routinely-with the intent to serve all with skill and trust.
4. "Hope is not a strategy" We routinely perform detailed preoperative planning and develop a surgical or clinical treatment plan for our patients so that our strategy is sound and outcomes can be predicted with a high degree of certainty. Out patients expect this planning.
5. "No campaign survives the first contact with the enemy" Have plan A and a backup plan B for any endeavor. This is true in the clinics, operating room and in the research laboratory.
6. "Run to the sound of guns". Where there is smoke, there is often fire. I try to be aware of obstacles, conflicts, and impending failures and engage the personnel, stakeholders and our health system to mitigate problems before they get out of hand. Solving problems is the job of a leader. Problems are always present-consistency in finding the problems, addressing the issues and finding solutions are critical to the success of the leader.
7. Integrity and honor. Leaders must demonstrate integrity in everything they do. Leaders must approach their charge considering it an honor to lead. Those that are led watch the leader constantly. Character matters. Leadership is character.
8. "You cannot Surge Trust" This expression was stated by Marine Corps General James Amos- referring to the citizens in Iraq and Afghanistan and the military relationship with governments and tribal leaders. Trust can only be built over time by openly communicating and being receptive to different points of view. Honesty and truth telling are essential to build trust. I am proud of the trust I have built with all of the individuals that define the Penn Department of Orthopedics as well as my fellow Chairs and countless Penn healthcare leaders.
9. When in Command, Command. A quote by Navy Admiral Nimitz. If one takes the reins to lead, then lead as if the welfare of your entire enterprise depends on you. It does. Certainly one should delegate responsibility to others in the organization. However, Command is lonely- the leader is ultimately responsible for what goes wrong but should acknowledge team members and teamwork when things go well.
10. Hold people accountable. We do not do this often enough in my opinion. I believe that our team succeeds when we hold each other accountable, set standards and deliver on commitments.
11. Strong management and oversight is essential. I want to recognize the efforts of Deborah Rose, James Henry. Following the departure of Neal Ravitz they have stepped in and managed operations. Andrew Duncan is our new Chief Administrative officer who is off to an outstanding start and is a seasoned Orthopedic administrator.
12. "Troop the line" Good leaders are in the trenches with their soldiers. I have made it a point to take call, and operate in each of the downtown hospitals. "Walking the factory floor" place as CEO or leader on the "battlefield" to witness firsthand the issues that need to be addressed.
13. "A good Shepard should smell like his or her sheep". It is essential that a surgical leader practice surgery and demonstrate clinical excellence. If you are in the trenches….you are one of the team. This is important.
14. "Have a swim buddy" A swim buddy is a fellow SEAL team member who has your back, supports your career and cares about your success. It is an individual that you trust (literally for SEALS with your life) to praise you when you do things well, and constructively criticize you when you are not doing things well. Bonds like this are made between residents, among faculty and can be forged outside of Penn Orthopedics.

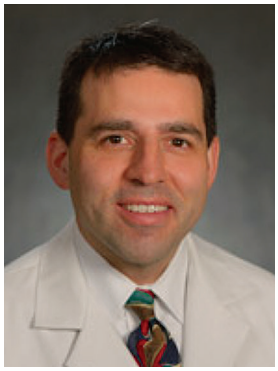
The themes and 14 chapters above all need to be credited to Admiral McRaven. However as the Chair of the Department of Orthopedic Surgery over the last 14 years, I have tried to be an effective leader. I can relate to the principles and try each day to meet your expectations. I will continue my commitment as Penn's Bullfrog, knowing that the next leader will take us farther exercising the same principles.

With gratitude for your support and accomplishments in 2023. LSL

With best regards,
Scott

Letter from the Program Director

Daniel C. Farber, MD



The past year has slowly brought a return to some normalcy in the residency program. As the COVID-19 pandemic has ebbed, there have been more opportunities for in-person educational opportunities, operating room and clinic volumes are returning to pre-pandemic levels allowing more clinical education time as well. It has been a pleasure working this year's Academic Chief Residents; George Fryhofer, Ryan DeAngelis and Greg Minutillo. Under their leadership, we have seen enhancements to the educational program, our visiting professor program, and more time in the human tissue lab doing hands-on learning. In-person off-campus journal clubs have continued which build not only knowledge but camaraderie between residents and faculty.

Through the enormous efforts of Kathleen Collins, MD PGY3 and Ryan DeAngelis, we have been able to get most residents caught up with AO courses both basic and advanced to help with their core orthopaedic trauma knowledge and skills after these courses were interrupted by COVID. We have also seen residents return to various meetings to present their research in person.

Under the leadership of Cara Cipriano, MD, the medical student experience continues to improve and receive high marks. Revamping of the Ortho 200 series where Penn medical students get their first taste of orthopaedics has sparked great interest in further exploration of our specialty. Improvements to the sub-internship experience led to a large increase in the number of visiting students to our program. The diversity scholarships sponsored by our department's Culture Committee also enhanced the sub-internship experience. The quality of these visiting students is exemplified by our excellent match this year with 4 of our 8 incoming interns having rotated with us and all of them being stellar students.

We have several new faculty who are already making an educational splash with our residents including Emmanuel Gibon, MD, PhD, and Timothy Costales, MD in Adult Reconstruction and Lorraine Boakye, MD in Foot and Ankle. Dr. Boakye and Costales will be supervising the intern skills month going forward and we look forward to the consistency and enhancements that they will bring to this experience.

From an administrative perspective, we sadly bid farewell to Shannon Savelloni this fall as she moved on to new opportunities. Shannon was an outstanding program coordinator who put her heart and soul into the program and she will be missed. She certainly taught me an enormous amount. On a positive note, we did welcome a new addition to the team. Abbi Goldman came to us from the Infectious Disease department and will be serving as a second program coordinator along the Angela Nieves who continues to skillfully manage the intricacies of running a large residency program. Abbi will be primarily responsible for the various fellowship programs and will assist Angela with residency program needs.

As always, the achievements of the program are only possible with the contributions of our amazing attending faculty who take their time and effort to support resident and student education. Another thank you to our Associate Program Director, Stephen Liu, whose hard work is indispensable to our success. The continued steadfast support of our Chair, L. Scott Levin MD is crucial to our success and we are lucky to have him for at least another year.

I again send out a huge thank you to our outgoing Academic Chief Residents mentioned above and congratulate them while looking forward to working with the incoming Academic Chiefs, Lucas Myerson, Kelsey Young, and Steven Zhang. Lastly, congratulations to all our graduating PGY5s who are headed to great fellowships.

We wake up every morning looking for ways to continue to improve the educational mission at Penn Orthopaedics and if you want to support this mission, please donate to the Penn Orthopaedic Education Fund. All donations directly benefit the residents and support everything from educational resources, resident instructional courses, and academic travel to the occasional resident happy hour that helps boost morale. Please contact Allyse Orsini at aorsini@upenn.edu or 267-788-0975. Also check out our Instagram page at Penn.Ortho!

Wishing everyone a happy and healthy and productive 2023-4 Academic year!

Easing the Pressure Points for Translation of Surgical Innovation

Edward J Harvey, MD, MSc and Stephanie Parent-Harvey, BS

Division of Orthopaedic Surgery, McGill University health Center Research Institute, McGill University

Innovation is easily the most overused, and perhaps misused, buzzword in medicine. The interchangeability between invention and innovation is what causes the confusion. Inventions are not innovations. Incremental improvement is not innovation and may not even be invention. The understanding of these concepts clears up many problems in translation of ideas to intellectual property (IP) and then to commercialization. Every day in operating rooms around the world surgeons have ideas on how to improve their condition. Barely any of these fit the concept of innovation let alone invention. The proof is that there are very few ideas brought into a commercial ecosystem from these thoughts. Inventions need to have a uniqueness that allows them protection – by copyright or patent in most cases. Trademarks and trade secrets are other ways to individualize your ideas but less common in medicine. Patents are a social contract between a society and an inventor. A governmental approved agency grants the inventor a sectoral monopoly, while the inventor must bring their idea to public domain.

Many ideas are not protectable, either due to prior usage or claims, or because of non-patentable properties. Certain concepts may seem like an invention but may be refused due to the argument that there is evident construction or clever engineering. This means most people would think of this and it is seen as an evident improvement. Just because you paint your fridge green does not mean you can patent it. If you do have an idea that seems destined to be granted patent protection, that is an invention. It is not an innovation. For example, the airplane was an invention but the use of the airplane to allow rapid transport of people and freight all over the world is the innovation. Innovation is the scaling of technology to address a societal need that results in wealth development- monetary or societal benefit of some type. That means invention is a small part of innovation. The renaming of university departments and the adding of innovation in job titles is probably not reflecting true innovation but certainly is a buzzword for the decade.

Innovation is a lot harder to accomplish than renaming some people as innovation gurus. Innovation in medicine is actually even harder. This is due to many issues. Regulatory hurdles protect both patients and incumbent companies. Economics is another hurdle in that new medical innovations need to be better, or at least as good, as what is offered; but they also need to be cheaper in order to be added to a hospital budget. That is a bad combination for most products. This is why we see innovation in expensive care scenarios- like cardiac and vascular surgery. But medical device sales is an extremely lucrative field. In 2016, US spending on medical devices and in-vitro diagnostics was $173.1 billion, 5.2% of total national health expenditures[1]. The US is the world's largest market for medical devices. Translating your idea down the commercialization pathway requires jumping these hurdles and others. Much innovation in medicine is new product performance ideas. The doctor envisions a new plate, robot, scalpel, or other product they encounter every day. The issue is that every other person doing that profession can see the same weaknesses and come up with the same idea. This results in a race to economic parity as you battle other products coming to the same space. Going down the innovation funnel means fighting off these other products from entering your space. The method of choice for venture capitalists interested in your idea is patent protection. The way to obtain that protection is through money invested in proof of concept- either studies or functioning prototypes. You need to get money before you can be given money - a real problem for development!

Getting to this point in commercialization development is already rarified air. Driving the idea to this point means you are derisking the investment for you, your customers, and investors. Most doctors will sell their concept to an interested party rather than endure the pain that follows. But if you do progress further in the derisking process there is definitely a bigger chance of reward. The path forward is a true monetization process. A lot of money is needed for production, capitalization, and popularization. This is a cash requirement – mean development cost for a novel therapeutic complex medical device was $54 million[1]. There is no real shortcut around this in the medical device world. In software products you can put out a minimally viable product that you can fine tune over time with help from consultants and customers. Devices however must go through a design freeze at some point to have FDA review and perhaps approval. Drug discovery and development is an even more expensive endeavor requiring over a billion dollars. Obviously, this is a huge barrier to entry making this pathway difficult except for institutions and companies with very deep pockets. The concept of monetization is not hard but is still a complex of difficult pathways where the only goal for your product moving forward is to make money or get further investment. The real way to accomplish this is to build a great product that drives value and attracts money because certainly you need a lot of money to bring any

device to market. The start-up financing cycle is further complicated after founding because as you grow your team there is an increased need for money. In the early stages of a new company your financial growth is made-up in friends and family contributions or through seed capital from government and other institutional investors. It is the best policy that you seek non-dilutive funding at this stage if possible. Grants and loans are the prime target. But after you see your idea through patent protection and proof of concept there's a need to show economic viability to all other investors. This is where many companies fail. Part of the monetization start-up phase requires passing regulatory control and certainly this is something that venture capitalists look at in a very in-depth manner. You need to take your proof-of-concept device to the point where you able to file for regulatory clearance which is generally a 510K in the United States or on the other hand performed human clinical trials. These are two points the investors are looking at. There are several ways to get FDA or equivalent approval. These include premarket notification better known as 510K premarket approval, de novo evaluation for new products or of a class 3 device, and other more esoteric classifications. Most people in surgery are looking at incremental improvement on a previous concept and this is more of a premarket notification. Most medical devices come in on this 510K premarket notification. There is a similar process in most countries, although there is a global trend to accept American FDA clearance in other countries. Regulatory clearance and IP protection represents a major derisking point for both you, your company, your idea, and investors. It is really at that time there's a decision point for your idea where you are either going to sell or license the product or embark on a real-world company. Starting a company means bearing with market valuations and the vagaries that go along with commercialization. This decision point usually involves the incorporation of the concept that you must make your idea work for commercialization. This dictates that you either have to make something no one else has, such as a niche product or process, or you have to make the product cheaper than everything else in the market. Sometimes you must do both things, and this is very difficult. Getting through this stage in the monetisation phase means that you are now headed towards a scale up concept. This is capitalization where you must bring in money to develop your product and sell it. It would be ideal if you could do this without loss of control of your company or product designation however this is not always possible. Derisking your product means bringing other people in to make it successfully scale, and this team building is really one of the major sticking points in hurdles for eventual success.

Scaling a device or idea means bringing it to market with a team of experts. Part of this is building capital in order to bring your concepts to reality. You also must define goals for your product and company. This requires you to create infrastructure and build your team while generating sales and delivering value. Although these all seem to be sequential, they actually all happen at once. This is the controlled mayhem that can sink many products. It is this at this time that most doctors realize that they cannot do this while maintaining a clinical practice or they may realize they actually don't have the economic toolkit to do this themselves. This is when team building becomes very important. How do you know if your idea is going to make it going forward? You should have an idea that your product is going to improve patient care physician satisfaction, while fulfilling payer requirements. Physician fulfillment is usually the easiest part of this because most medical products are brought forth from dissatisfaction with the physicians' day-to-day workflow. Patient care is the other concept that is fairly simple because most of the physician's day-to-day workflow is centred on patient care. The concept of making sure that the payer is happy is a foreign concept to most physicians. It becomes more of a costing exercise and risk management for the eventual purchasers of the product that will allow your device to get to a bigger population. Investors like genuine enthusiasm amongst physician users at this point of the innovation cycle. Communication is extremely important at this point in the journey. You need to be able to communicate to your investors how you are making money. This needs to be crystal clear in the way you communicate it. You need to analyze the market, and both understand and communicate intended primary market size and expanded secondary market size to investors. You need to have a market fit optimization or value to all the parties the physicians, patients, and providers is clear. It would seem at this point that it would be simple to bring your product to the masses however the last value of death in the innovation cycle is popularisation. Good products can fail even at this point. It is very difficult to sell your product early on in its life cycle to the late majority or even the early majority of consumers. Your product is often being purchased by the early adapters and innovators and it is difficult to understand who is actually buying your product without in depth market evaluation. Investors look to this type of evaluation as being one of the final milestones to see whether they will invest in your idea. Sales, and lots of sales, is what will significantly increase funding interest in your product. This means that you need a break-even point in your company evolution, and you will need to understand the runway or when you will run out of money. If you can get through all these hurdles and products and you have a clear innovative product with a clear need or market and your product is preferably technologically driven, then you can win the innovation race. It is a marathon and not a sprint.

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I Am Smarter Than You: Projections of Orthopaedic Research from the most Knowledgeable Mind in the World

Orthopaedic surgery has witnessed remarkable advancements over the years, enhancing the quality of life for countless individuals. As we embark on a new era of medical innovation, it is crucial to explore the future of orthopaedic surgery research. This editorial delves into the transformative potential of imaging, robotics, biologics, and regenerative medicine, outlining how these fields are reshaping the landscape of orthopaedic surgery and paving the way for unprecedented possibilities.

Imaging technologies have revolutionized orthopaedic surgery by providing surgeons with detailed anatomical insights. However, the future holds even more promise. Advanced imaging techniques such as 3D imaging, functional magnetic resonance imaging (fMRI), and positron emission tomography (PET) are poised to become indispensable tools for preoperative planning, intraoperative guidance, and postoperative evaluation. These technologies offer unparalleled visualization and aid in personalized treatment approaches, enabling surgeons to tailor interventions to each patient's unique anatomy and pathology. The integration of artificial intelligence (AI) algorithms into imaging systems further enhances the accuracy and efficiency of diagnosis and treatment decisions.

Robotic-assisted surgery has gained significant traction in recent years, with the ability to enhance precision, minimize invasiveness, and improve patient outcomes. The future of orthopaedic surgery lies in the continued development of robotic systems tailored specifically for orthopaedic procedures. Collaborative robots (cobots) hold great promise by combining the dexterity and accuracy of robots with the surgical expertise of human surgeons. Cobots have the potential to enhance surgical skill, reduce the surgeon's physical strain, and increase patient safety. Additionally, the integration of haptic feedback and virtual reality technology allows for realistic surgical simulations, enabling surgeons to refine their skills and improve patient care.

Biologics, such as platelet-rich plasma (PRP), stem cells, and growth factors, have emerged as powerful tools in orthopaedic surgery. These therapies stimulate the body's natural healing mechanisms, promoting tissue regeneration and reducing the need for extensive surgeries. The future of biologics in orthopaedics holds the potential for personalized treatments tailored to each patient's specific needs. Through advancements in tissue engineering and genetic engineering, orthopaedic surgeons may harness the body's innate healing potential to repair damaged tissues, restore joint function, and alleviate pain. Clinical trials and ongoing research are vital to unlocking the full potential of biologic therapies.

Regenerative medicine offers a revolutionary approach to orthopaedic surgery, aiming to regenerate damaged tissues and organs rather than merely repairing or replacing them. Stem cell therapies, tissue engineering, and gene therapies hold great promise in regenerating bone, cartilage, and ligaments. Novel techniques such as 3D bioprinting enable the creation of patient-specific implants, bypassing the need for donor tissues. The future of regenerative medicine in orthopaedics lies in the development of personalized therapies that address the root cause of musculoskeletal disorders, revolutionizing treatment strategies and providing long-lasting solutions.

The future of orthopaedic surgery research is teeming with potential, driven by the rapid advancements in imaging, robotics, biologics, and regenerative medicine. These fields hold the key to personalized, minimally invasive interventions, accelerated healing, and improved patient outcomes. By embracing these technologies, orthopaedic surgeons can refine their skills, optimize treatment plans, and deliver transformative care. As we embark on this exciting journey, it is crucial to invest in research.

Now that you have read the article, let me ask you, did you enjoy it? Did you learn anything new? As you may have probably figured out by now, this editorial was generated using a free, commercially available artificial intelligence program. Once the command was inserted "Write a 1.75 page editorial on the future of orthopaedic surgery research. Please touch upon imaging, robotics, biologics, and regenerative medicine" the machine was able to extract, analyze, and synthesize a complete and coherent essay in the time it takes to read this sentence. So the question remains, is this artificial intelligence smarter than you, and if not now, then when? While most of us are not concerned (at the moment), and rightfully so, what does that mean for the future of orthopaedic surgery, and how can we best integrate this technology to better serve our patients?

- ChatGPT3.5

Bijan Dehghani, MD MS
Mitchell Hallman, MD
Samir Mehta, MD

Department of Orthopaedic Surgery, University of Pennsylvania

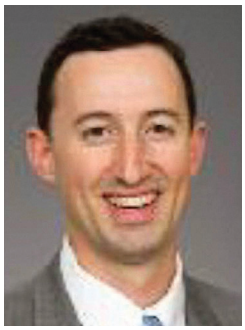
Spine Division

Amrit Khalsa, MD

Spine Faculty


Harvey Smith, MD


Vincent Arlet, MD


Amrit Khalsa, MD


David Casper, MD


J. Rush Fisher, MD

The Orthopaedic Spine Division continues to excel as individuals grow and transition. In addition to advancing the legacy of complex scoliosis and revision spine surgery, the orthopaedic spine service tackles a wide breadth of cutting-edge and minimally invasive procedures. Additionally, the division participates in Level 1 spine trauma call managing some of the most critical patients.

The Spine Division educational mission continues to thrive. In addition to supporting a rotating PGY 3 and PGY 4 resident, between the matriculating and rising PGY 5 classes, a total of 6 Penn Orthopaedic residents are attending elite spine fellowships across the country.

Dr. David Casper assumed the role of Co-director for our own Penn Spine Fellowship. Under his leadership and the continued support from our additional faculty, Director Emeritus Dr. Vincent Arlet, Co-Director Dr. Amrit Khalsa, Dr. Harvey Smith, and Dr. Rush Fisher we had the most applicants ever for our two separate Penn Spine Fellowship tracks. We continue to support 3 positions, 1 full-time adult spine fellow and 2 combined pediatric and adult fellows in collaboration with the Shriner's Hospital of Philadelphia.

Dr. Vincent Arlet began a year-long sabbatical in July 2023 focusing on his complex pediatric spine outreach program in Trinidad.

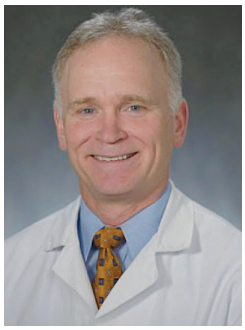
Dr. Harvey Smith continues to lead the way as the consummate physician-scientist, advancing translational research surrounding biologic disc-replacement and spinal fusion biologics. As a whole, the Spine Division has participated in a multitude of peer-reviewed publications, chaired multiple committees and academic courses both at the regional and national levels, and continue in leadership roles across multiple national and international societies.

The Spine Division is supported by an illustrious host of advanced practice providers and administrative assistants that continue to elevate the division as we grow both clinically and academically.

Hand Division

David Bozentka, MD

Hand Surgery Faculty


David Bozentka, MD


David Steinberg, MD


L. Scott Levin, MD, FACS


Hannah Lee, MD, PhD


Andrew Sobel, MD


Robert Carrigan, MD


Apurva Shah, MD, MBA


Stephen Liu, MD

The Hand and Upper Extremity Service within the Department of Orthopaedic Surgery has again had a remarkable year. Not only have we added more talented clinical staff to our team, but we have also provided our residents and fellows with an exceptional educational experience. Furthermore, our commitment to research has expanded, with numerous studies being undertaken by our team.

We are delighted to welcome Linda Chao, MD, to the Penn Hand Surgery family as the newest member of our team at Cape Regional Medical Center. Linda brings an impressive academic background, having completed her undergraduate studies at Harvard College and her medical training at Brown University. She then went on to complete her residency in Orthopaedic Surgery at Wake Forest University and her Hand and Upper Extremity Fellowship at Cedars-Sinai Medical Center in Los Angeles. Her interest in microsurgery and teaching makes her an invaluable addition to our team. In addition to her impressive professional achievements, Linda is a passionate runner who competes in marathons and half marathons. We are thrilled to have her join us and look forward to the valuable contributions she will make to our practice.

Under the guidance of David Steinberg, MD, and Ines Lin, MD, our fellowship program has continued to flourish. The two current fellows have had a successful and enriching year, making significant strides in their clinical and academic pursuits. Thanks to the Chan Family Surgery Education Fund, established by the generous contributions of Peter SH Chan MD and Karen Postick Chan MD, our fellows have had the opportunity to attend the flap dissection course as well as the annual meeting of the American Society for Surgery of the Hand held in Boston, Massachusetts. In addition, our weekly didactic fellow-run hand surgery conference has continued to thrive, with topics ranging from joint radiology conferences to pediatric conferences, journal clubs, and case reviews.

Lindsay Janes, MD, who completed her plastic surgery residency at Northwestern University, will return to Chicago after completing her fellowship to join the Division of Plastic Surgery at Northwestern University. Likewise, Kate Grunzweig, MD, who completed her residency training in the Department of Plastic Surgery at Case Western Reserve University, will join the Department of Plastic Surgery at Loyola University in Chicago after her year of hand fellowship training.

In February, we interviewed twenty candidates from over one hundred applicants for the two Hand Fellowship positions. The incoming fellows for next year include Dr. Vinay Rao, MD, MPH, who will complete his plastic

surgery residency at Brown University, and Kurt Mohty, MD, currently completing his orthopedic surgery residency at the University of Southern California.

Andrew Sobel, MD, has demonstrated exceptional leadership as the Director of Clinical Research for the Hand Surgery Section. He has been conducting bi-monthly research meetings to review over twenty active research projects, leading to presentations at the AAOS, ASSH, and AAHS annual meetings. Recently, the section was notified of its approval for NIH funding as a clinical trial site for a peripheral nerve injury repair device.

To keep you informed of our developments, Penn Hand Surgery is now on social media. Under the leadership of Mylinh Nguyen, the service has launched a social media site showcasing interesting cases, hand-section staff, presentations, and awards. We encourage group members and alums to send suggestions for the site. In addition, stay updated with the program by following us on Instagram @ Pennhandsurgery.

The hand transplant team has successfully performed bilateral upper extremity allotransplantation on four patients with quadra-membral amputations, resulting in remarkable outcomes that have transformed their lives. Unfortunately, the listing of patients had been put on hold due to the COVID pandemic. However, the team has continued to meet regularly, rehearsing in the new Pavilion operating room and the human tissue lab with cadavers, fine-tuning the procedure checklists in preparation for the fifth bilateral upper extremity allotransplantation.

We want to acknowledge the invaluable support of our outstanding advanced practice providers, nurses, and administrative assistants who help keep the hand and upper extremity service running smoothly. We congratulate Christine (Chrissy) McAndrew, PA-C, on her new role as the Director of Advanced Practice & Nursing for the Department of Orthopedics. In this capacity, Chrissy will directly manage the Department's RN nursing staff while continuing to oversee the Advanced Practice Providers. Chrissy has been with the department for 11 years and has been the Supervisor of the Advanced Practice Providers since 2018 and the Hand Transplant Coordinator for Penn Medicine and The Children's Hospital of Philadelphia since 2012.

We are privileged to have an exceptional, collaborative, and dynamic group. With such fantastic support, the future looks bright for the Hand Surgery Section.

Shoulder and Elbow Division

David Glaser, MD

Shoulder & Elbow Faculty

David Glaser, MD

Andrew Kuntz, MD

Gabe Horneff, MD

With continued commitment to the management of the most complex cases, the section's tertiary referral network has continued to increase, along with the complexity of cases. In FY23, the group performed over 10,000 visits and performed over 900 surgical cases with anatomic/revision shoulder arthroplasty and elbow surgery seeing increased volume. Gabe Horneff has continued ownership of resident education for our division and become our anchor at PAH, with his practice growth predicted to be at maximum by the end of FY24. Our indications conference, which was expanded during the pandemic, has continued and includes a nationwide group of our past fellows, our therapy team, and regional shoulder and elbow providers. Jeff Abrams has been a welcome addition to our educational mission. Through a virtual platform, the group of talented sub-specialists re-unite monthly to discuss complex cases, opportunities for research and to catch up on important life events.

Andy Kuntz is leading our research effort, setting a high standard for both scientific methods and clinical outcomes. To parallel his research interests in anatomy, functionality, and mechanisms of injury of the rotator cuff and outcomes of arthroplasty, he has decided to focus his clinical practice towards the treatment of both rotator cuff pathology and glenohumeral arthritis. We would like to recognize Andy for his continued focus as a clinician-scientist, providing world class clinical care, while contributing to all aspects of our research mission - clinical, translational, and basic science. He was awarded a VA Merit Grant to further support his pre-clinical research. Alongside Andy, Gabe has helped expand our clinical research program, mentoring several medical students and residents with their academic endeavors. In close collaboration with Lou Soslowsky and others in the McKay Research Laboratory, we helped advance the McKay lab to #1 in National Institutes of Health funding. We are rolling out an integrated research platform that will be able to seamlessly unite clinical and research activities, providing a much-needed tool for data collection, while improving two-way clinical communication with our patients.

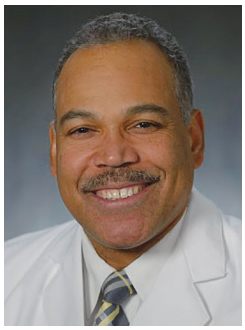
The fellowship has continued to thrive, attracting the most competitive candidates. This year's candidate pool was as competitive as prior years. Our program is unique in that the fellow has exposure to four different surgeons, with complementary philosophies, who use an extreme range of devices and approaches. John Kelly has added an additional opportunity to fellows interested in creative arthroscopic approaches to manage complex shoulder and elbow pathology. Additionally, now in its six year, and in collaboration with our French colleagues, we offer our fellow an opportunity to visit world leaders in shoulder surgery. Holt Cutler (F'23) returned from Monaco and France and will join Howard Harris and Buz Burkhead at the well known Carrell Clinic in Dallas. Brandon Romero (F'22) joined University of Nevada Las Vegas and immediately became the only subspeciality trained shoulder and elbow specialist in his group. Past fellows who enjoyed the European experience include Christy Piper (F'21), Greg Gomez (F'19), Josh Rogozinski (F'18), and Chad Myeroff (F'17) who spent between two to three weeks visiting academic centers in Europe.

We will continue to leverage our internal cohesiveness, therapy partners (superstars Brian Leggin, Joseph Kearns, and Marty Kelly) and recent collaborations with non-Penn shoulder and elbow providers, to bring success to our division.

Adult Reconstruction Division

Charles Nelson, MD

Arthroplasty Faculty

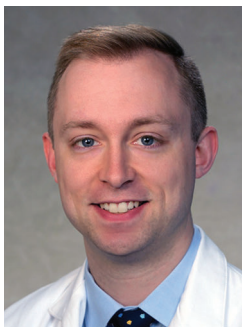

Charles Nelson, MD


Craig Israelite, MD


T. David Tarity, MD.


Christopher Anthony, MD


Neil Sheth, MD


Christopher Travers, MD


Emmanuel Gibon, MD, PhD

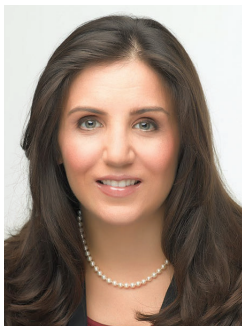

Timothy Costales, MD

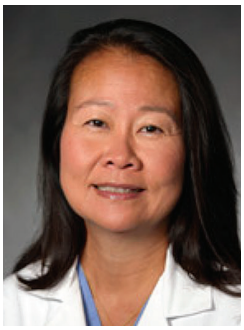
This past academic year has been one of productivity and growth for the Penn Orthopaedics Adult Reconstruction Division. Despite the continued challenges with the ongoing COVID pandemic, the adult reconstruction division has maintained a high surgical volume and provided high quality care for a wide spectrum of patients. We continue to care for both healthy and high-risk patients with innovative strategies to minimize complications and optimize quality in the high-risk patient cohort. Additionally, we have grown our department with the addition of Emmanuel Gibon, MD, PhD, and Timothy Costales, MD. In addition to clinical excellence, our faculty have remained active in making scientific contributions and clinical education nationally and internationally, as well as serving in leadership and volunteer positions within prestigious national orthopaedic organizations including: the American Academy of Orthopaedic Surgeons; The Hip Society, The Knee Society, the International Hip Society, the American Association of Hip and Knee Surgeons, the American Orthopaedic Association, and the American Board of Orthopaedic Surgeons. Our faculty participated in several dozen peer reviewed publications, scientific presentations and invited lectures in 2022-2023. The division maintains active in clinical research with significant federal and industry funding. The adult Reconstruction faculty members currently include Professor Charles L. Nelson, MD, Associate Professors Eric Hume, MD, Craig Israelite, MD and Neil Sheth, MD and Assistant Professors Christopher Travers, MD, Christopher Anthony, MD, T. David Tarity, MD, Emmanuel Gibon, MD PhD, and Timothy Costales, MD.

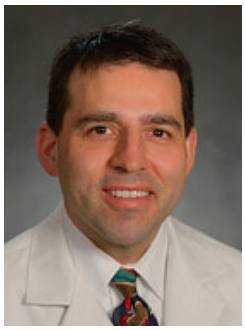
Foot and Ankle Division

Casey Humbyrd, MD

Foot & Ankle Faculty


Casey Humbyrd, MD


Wen Chao, MD


Daniel Farber, MD


Anthony "Bobby" Ndu, MD


Keith Wapner, MD

2022 was a year of scholarship, transition, and growth for the Foot and Ankle Division.

Dr. Lorraine Boakye joined the division after completing fellowship at Harvard-Brigham and Women's Hospital. Prior to this she completed medical school, and residency training at the University of Pittsburgh and her undergraduate education at Yale. Dr. Boakye is on the Clinician-Educator track and took on the role of Director of Clinical Research for the Foot and Ankle Division. She is excited to collaborate with the McCabe lab in their work on Achilles rupture and tendinopathy. She is working to optimize our collection and use of patient reported outcomes within the division and throughout the department. Additionally, she received the AOFAS sponsored position to participate in the Orthopaedic Research Society's Career Scholars Career Development Program this fall. She was also honored with the Ruth Jackson Orthopaedic Society's 2023 Courage Award. She currently serves as the RJOS Liaison to the AAOS Diversity Advisory Board. She, alongside Dr. Coastales, will be leading the Intern Skills Curriculum for next year's class.

Dr. Wen Chao continues to be the orthopaedic foot and ankle consultant to the Philadelphia Ballet since 2001. She also serves as a member on the Public Education Committee for AOFAS, as well as reviewer for Foot and Ankle International and Foot and Ankle Orthopaedics. She is a member of AAOS, AOFAS, AOA and the Orthopaedic Foot Club. Dr. Chao is interested in ultrasound applications to foot and ankle practice, particularly correlating intraoperative and ultrasound findings compared to the MRI imaging. This is an ongoing IRB research project.

Dr. Anthony "Bobby" Ndu continued to build his clinical practice, as well as his role as fellowship director. He has expanded his clinical practice to include the cutting-edge technology of Minimally Invasive foot and ankle reconstruction surgery, performing large reconstructions through keyhole incisions. He also serves as a member of the committee for Diversity Equity and Inclusion for AOFAS and was recently nominated as a candidate for a position on the national board of the AOFAS. Dr. Ndu continues his dedication to teaching with multiple technique papers submitted for publication while working closely with and mentoring undergraduate and junior medical students interested in a career in orthopedics.

Dr. Daniel Farber has continued to lead the educational mission of the department as Vice Chair for Education and Residency Program Director while maintaining a busy clinical practice. He serves on the education committee of AOFAS and he was the Program Chair for the Pennsylvania Orthopaedic Society Annual Meeting in Hershey, PA. He also was selected for the inaugural European Foot and Ankle Society / AOFAS traveling fellow program for the fall of 2023. He continues as Chair of the AAOS Resolutions committee as well as on the ABOS Committee selecting MOC/WLA articles. He serves as a reviewer for Foot and Ankle International, Foot and Ankle Orthopaedics as well as the American Journal of Sports Medicine. He continues his basic science research efforts in collaboration with Lou Soslowsky, PhD and the McKay lab. He is also a collaborator with Josh Baxter, PhD of the Human performance lab on his K01 and R01 awards exploring Achilles pathology.

Dr. Casey Humbyrd worked to build her clinical and research endeavors at Penn. She was awarded the Ruth Jackson Orthopaedic Society research award to study gait in pregnancy. She served as the Program Chair for the AOFAS Winter meeting in Phoenix, Arizona and AOFAS Annual Meeting in Quebec City, Quebec. She also served as the chair of the Conflict-of-Interest Committee for the AOFAS. She continued representing AAOS at the AMA. Dr. Humbyrd continued her column "Virtue Ethics in a Value-Based World" in Clinical Orthopaedics and Related Research. Dr. Humbyrd serves as a reviewer for Foot and Ankle International, the Journal of the American Academy of Orthopaedic Surgeons, and Clinical Orthopaedics and

Related Research. Dr. Humbyrd also expanded her clinical practice, including participating in the care of collegiate and professional athletes.

The year also included exciting developments in the realm of translational research, with the awarding of a P50 research grant on Achilles tendinopathy. This five-year, nearly 8-million-dollar Center of Research Translation Grant, focused on furthering an understanding of how and why the Achilles tendon breaks down. The grant is led by Drs. Soslowsky, Mauck, and Humbyrd. The tissue core section will be led by Drs. Farber and Baxter.

Finally, 2022 marked the final full year of clinical practice for Dr. Keith Wapner, who plans to retire June 30, 2023 after 37 years in practice. His contributions to Penn and Foot and Ankle Orthopaedics can't be overstated. A department-wide celebration is planned to honor his work and career, and his presence at Penn lives on both with the Keith Wapner academic scholarship and the June Wapner annual lectureship.

Orthopaedic Oncology Division

Cara Cipriano, MD, MSc

Orthopaedic Oncology Faculty

Kristy Weber, MD, FACS

Cara Cipriano, MD, MSc

The Orthopaedic Oncology service at Penn has continued to grow and develop in 2022-23.

Dr. Kristy Weber remains clinically busy with her practice at PCAM, HUP, and CHOP. She also serves in leadership roles such as the Vice Chair of Faculty Affairs for the Penn Orthopaedics Department and Director of the Sarcoma Program at the Abramson Cancer Center. In this capacity, she runs the weekly musculoskeletal tumor board, which includes participation from medical oncology, radiation oncology, pathology, radiology, neurosurgery, and general surgery. In addition, she dedicates herself to maintaining and building the multidisciplinary team by recruiting new faculty. We are excited to welcome several new sarcoma team physicians in the next few months, each of whom will bring their unique experiences, ideas, and energy to the team.

In her first two years at Penn Medicine, Dr. Cara Cipriano has built a clinical practice focused primarily on musculoskeletal oncology, but also including primary and revision joint replacement. This has created additional options for patients requiring complex reconstructions for metastatic disease as well as sarcoma survivors with prosthetic complications that inevitably occur over long-term follow up. In addition to seeing patients at PCAM, she sees patients at Radnor, which improves access to patients who experience barriers or challenges associated with travel into downtown Philadelphia. More recently, she has established a clinic at Pennsylvania Hospital's Farm Journal Building, where patients can see our medical oncology team on the same day for improved convenience and coordinated care.

Dr. Cipriano also has several initiatives and responsibilities as the Director of Undergraduate Medical Education. She manages the Ortho 200 Clerkship and has made significant structural changes to the curriculum that have improved the engagement and experience of the students. Additionally, she mentors students applying into Orthopaedic Surgery residencies, oversees the Inclusive Orthopaedics Scholarship to support URiM students, and acts as a faculty advisor for the Leo Leung Orthopaedic Society, a student interest group at Penn Medicine.

Kate Barrie, PA continues to play a pivotal role in seeing patients as well as managing the Orthopaedic Oncology service. Nicole Koffke, RN, joined our team in 2022 and has already proved herself to be a phenomenal and indispensable addition. Both Kate and Nicole are both exceptionally knowledgeable and compassionate clinicians, and they are highly valued by our patients and multidisciplinary colleagues. We look forward to welcoming Allyson Woodley as our new administrative coordinator next month, to complete and bring her own strengths to our team.

Our basic and translational science teams at Penn Medicine, Penn Veterinary Medicine, and the Children's Hospital of Philadelphia (CHOP) have maintained and grown their extramural funding, allowing them to push the envelope of modulating the immune environment in soft tissue sarcoma and identify targets for the prevention and treatment of sarcoma metastasis to the lung. We are continuing our collaborative clinical trials across the full age span led by Drs. Patrick Grohar at CHOP. Dr. Nicola Mason is expanding her work with immunotherapy for dogs with different types of sarcoma with corollary trials in children using her ideas. Overall, the portfolio of available clinical trials for a variety of bone and soft tissue sarcomas as well as aggressive benign conditions has continued an upward trajectory over the past year at Penn. Meanwhile, our multidisciplinary clinical research group has been focused on questions related to local disease control that directly impact the care and quality of life of our patients.

We are in the process of finalizing a manuscript on a novel approach for determining whether patients require surgery for metastatic lesions at risk for fracture, and we will be beginning new studies in the coming months. As always, much of this research would not be possible without the philanthropic generosity of our patients, their families, and other supporters.

Our 9th Annual Steps to Cure Sarcoma Walk/Run took place on May 21, 2023, and was a great success. The event was organized by our patient/family advocacy group to raise awareness and funds for sarcoma translational and clinical research. (www.stepstocuresarcoma.com) We look forward to a productive year in 2023-24!

Children's Hospital of Philadelphia

Jack Flynn, MD, Ryan Quinn, MHA, and Divya Talwar, PhD, MPH

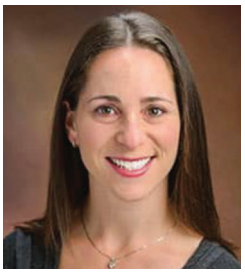
Pediatric Faculty


John Flynn, MD


Jason Anari, MD

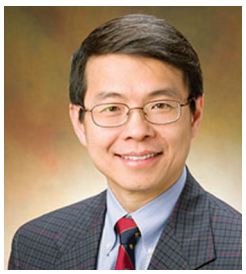

Alexandre Arkader, MD


Keith Baldwin, MD, MPH, MSPT


Naomi Brown, MD, FAAP, CAQSM


Patrick Cahill, MD


Robert Carrigan, MD


Benjamin Chang, MD, FACS


Richard Davidson, MD


Vincent Deeney, MD


Malcom Ecker, MD


Theodore Ganley, MD


B. David Horn, MD


J. Todd Lawrence, MD, PhD


Ines Lin, MD


Kathleen Maguire, MD

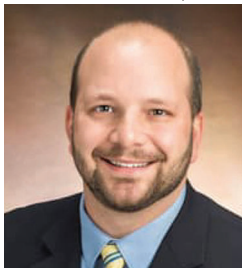

Christina Master, MD, FAAP, CAQSM, FACSM


Christopher Renjilian, MD


Wudbhav Sankar, MD


Apurva Shah, MD, MBA


David Spiegel, MD


Brian Vernau, MD, FAAP, CAQSM


Kristy Weber, MD, FACS


Lawrence Wells, MD


Brendan Williams, MD


Jennifer Winell, MD

Introduction

The Division of Orthopaedic Surgery at the Children's Hospital of Philadelphia (CHOP) had another successful and productive year of significant growth, accomplishment, and innovation. Upholding our mission and vision to provide the most comprehensive pediatric musculoskeletal care in the nation/world, we have continued to expand our clinical, research, and teaching programs.

In 2022, CHOP Orthopaedics welcomed a new sports medicine pediatrician to our team, participated virtually and in person at national and international conferences, maintained enrollment of three FDA Phase IIIb investigational drug trial and a feasibility device trial, published more than 200 articles, and obtained significant extramural funding from major funding agencies such as National Institutes of Health (NIH), Department of Defense (DoD), and National Science Foundation (NSF).

Clinical Program

Our Orthopaedic faculty continues to expand and is currently comprised of thirty members: eighteen specially trained pediatric orthopaedic surgeons (including three transition-to-adult care faculty), four non-operative physicians, six sports medicine-trained pediatricians, and two collaborating plastic surgeons.

Education Program

CHOP Orthopaedics currently funds four one-year clinical fellowships. The 2022-2023 clinical fellows are Soroush Baghdadi, MD (Figure 1); Grace Blaylock, MD (Figure 2); Bridget Ellsworth, MD (Figure 3); and Rachel Lenhart, MD (Figure 4). The 2022-23 research fellow was Dr. Akbar Syed, MD from India (Figure 5). While at CHOP, Dr. Syed focused his research efforts on clinical research related to pediatric trauma, hand, neuromuscular conditions, tumors, and sports injuries. He will stay with our division for another year.

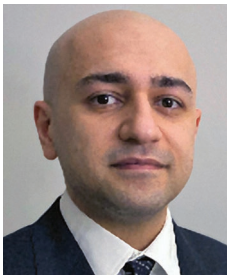


Figure 1. Soroush Baghdadi, MD

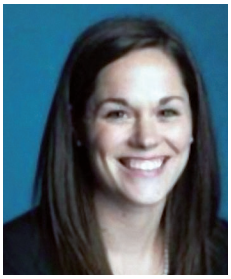


Figure 2. Grace Blaylock, MD

Figure 3. Bridget Ellsworth, MD

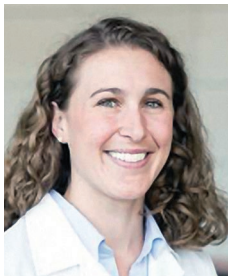


Figure 4. Rachel Lenhart, MD

Figure 5. Akbar Syed, MD

To celebrate the graduation of the 2021-2022 clinical fellows, the Division hosted the Nicholson Visiting Professor Program and Fellows Graduation & Reunion in June 2022. This year's Visiting Professor was Dr. Peter Newton, Professor of Orthopaedic Surgery at the University of California, San Diego, and Chief of Pediatric Orthopaedics as well as Surgeon-in-Chief at Rady Children's. He is a nationally recognized expert in pediatric spine deformity.

The 2022 Drummond Rising Star Visiting Professor was V. Salil Upasani, MD, MPH. Dr. Upasani is an Associate Professor of Clinical Orthopedic Surgery at the University of California San Diego and Rady Children's Hospital San Diego. He is the Director of the International Center for Pediatric and Adolescent Hip Disorders and the Program Director of the Pediatric Orthopedics and Scoliosis Clinical Fellowship at Rady Children's Hospital San Diego. Dr. Upasani has research interests in all general pediatric orthopedic conditions, with a special interest in hip and spine disorders in children, adolescents and young adults.

Research Program

Basic Science and Translational Research

This past year, our basic and translational medicine researchers led by Maurizio Pacifici, Ph.D. have made impressive progress and generated novel, exciting, and far-reaching insights on key aspects of skeletal biology and growth and pediatric musculoskeletal pathologies. Our pediatric musculoskeletal research lab continues to solidify its standing with research work from Dr. Fanxin Long and Dr. Veronique Lefebvre. Our faculty members and their associates, including postdoctoral fellows, visiting scientists and research technicians, continued to tackle and fulfill the goals of several current NIH R01 grants and one Department of Defense (DOD) grant. They focused on pediatric pathologies including Multiple Hereditary Exostoses (MHE), Fibrodysplasia Ossificans Progressiva (FOP), Temporo-mandibular Joint dysfunction, Lamb-Shaffer syndrome, Hjadu-Cheney syndrome, and spondyloarthritis. The research Program is currently supported by 12 RO1 grants from the National Institutes of Health and generous donations from private foundations.

Center for Thoracic Insufficiency Syndrome (CTIS) Frontier Translational Research Program

Through funding from the Frontier Program, the Division's Center for Thoracic Insufficiency Syndrome (CTIS) continued developing innovative projects in translational research. The CTIS program strives to develop novel imaging techniques, construct new metrics for clinical outcomes, and establish reliable evidence to support innovative surgical strategies and devices through its research. These efforts are made possible by

the collaboration of a multidisciplinary team of specialists from clinical research, image processing, informatics, and basic sciences/biomechanics. Currently, the CTIS Basic Science Lab is developing an animal model of TIS that will provide a platform for testing novel devices. The animal surgeries and biomechanics testing will be performed at Penn Vet's New Bolton Center. In addition, the CTIS team in collaboration with Medical Image Processing Group were awarded NIH R01 grant to develop novel dynamic functional metrics for TIS patients by establishing a comprehensive normative database of dMRI images and anatomic and functional models and metrics, and to translate these to develop biomarkers of TIS and of its corrective-surgery outcomes.

With the generous philanthropic support, Dr. Campbell's legacy was strengthened with the establishment of *Wyss/ Campbell Center for Thoracic Insufficiency Syndrome*, enabling CHOP to discover countless more breakthroughs in research and care for TIS children.

Genetic Research

CHOP Orthopaedics continues to work in collaboration with the Center for Applied Genomics (CAG), led by Dr. Hakon Hakonarson and Dr. Struan Grant, to compile a registry of DNA and RNA samples. These samples are obtained from patients and families with a variety of orthopaedic conditions including adolescent idiopathic scoliosis (AIS), osteochondritis dissecans (OCD) of the knee, Tibial Spine fractures (TSF) and multiple hereditary exostoses (MHE). The team is investigating further genetic characterizations of the EXT1/EXT2 mutations harbored by each exostosis and identify second hit(s) across exostoses from the same patient. This pilot project represents the first biomedical research focused on MHE and will provide novel and broadly relevant information. The goal is to translate the findings to prognostic tools based on the severity of the disease and to identify therapeutic means to counter the effects of EXT1/EXT2 plus "second hit" mutations.

Clinical Research

The Division of Orthopaedic Surgery is currently conducting more than 236 IRB-approved clinical research projects. This includes more than 100 prospective and observational studies. CHOP Ortho faculty are also members of a number of multicenter study groups, including the Harms Study Group (HSG), the Pediatric Spine Study Group (PSSG), Research in Osteochondritis Dissecans of the Knee (ROCK), SCFE Longitudinal International Prospective Registry (SLIP), Tibial Spine Prospective Cohort (TSF-PC), The Fox Pediatric Spinal Deformity Study (Fox PSDS), Pediatric ACL: Understanding Treatment Operations (PLUTO), Medial Epicondyle Outcomes Multicenter (MEMO) study and International Hip Dysplasia Institute (IHDI), Children's Orthopedic Trauma and Infection Consortium for Evidence based Studies (CORTICES), Congenital Upper Limb Differences Registry (CoULD), Research in Osteochondritis of the Elbow (ROCKET), Sports Cohort Outcomes Registry (SCORE), and International Perthes Study Group (IPSG). Investigators within the division have been awarded funding from both internal and external sources to conduct these studies. In 2022, the Division published over 196 articles in major orthopaedic journals, including *JAMA, JBJS, Lancet, JPO*, and *CORR*. Members across our division presented more than 107 presentations at international and national conferences last year alone.

Our Benjamin Fox Research Fellowship for medical students between 3rd and 4th years welcomed Julianna Lee (University of Connecticut School of Medicine), Kevin Orellana (University of Texas Rio Grande Valley School of Medicine), and Daniel Yang (University of Michigan Medical School) (Figure 6-8).

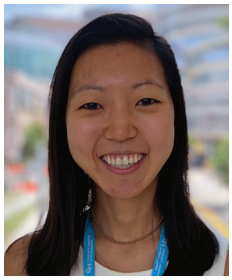


Figure 6. Julianna Lee

Figure 7. Kevin Orelllana

Figure 8. Daniel Yang

Recognition and Achievements

Our faculty have assumed several leadership roles within the pediatric orthopaedic community over the past year.

Jason Anari, MD served as international faculty member at the Salzburg Medical Seminar in Pediatric Orthopedics in Salzburg, Austria. Dr. Anari also received a new grant as PI from Pediatric Orthopaedics Society of North America (POSNA) titled, "*Managing failure to lengthen in MCGR: Best practice guidelines*".

Alexandre Arkader, MD is the Vice Chair for the Pediatric Orthopaedic Society of North America (POSNA) Educational Course Committee. He served as a sub-committee chair for Global Courses. Dr. Arkader continues to serve as a reviewer for *Journal of American Academy of Orthopaedic Surgeons, Journal of Bone and Joint Surgery Essential Surgical Techniques, BMC Musculoskeletal Disorders, Journal of Pediatric Orthopaedics B, Journal of Children's Orthopaedics, Current Orthopaedic Practice, Clinical Orthopaedics and Related Research, and Pediatric Radiology*. He is also on the Surgical Advisory Board for Orthopediatrics. Dr Arkader continues to serve as Editor for tumors section, *JPOSNA*. He also received a Cell and Gene Therapy Seed Grant as Co-PI with Dr. Fanxin Long titled "*Wnt-based gene therapy for bone repair*." Dr. Arkader is an active member of CORTICES study group.

Keith Baldwin, MD, MSPT, MPH is the Associate Director of Orthopaedic Trauma in the Division of Orthopedic Surgery. Dr. Baldwin is a Resident Advisory board member for the American Journal of Orthopaedics. He currently serves as a reviewer for several journals including the *BMC Medical Education, BMC Musculoskeletal Disorders, BMJ Open, Journal of Pediatric Orthopaedics, Annals of Internal Medicine, Journal of Bone and Joint Surgery—American*, *American Academy of Pediatrics, Clinical Orthopaedics and Related Research, Indian Journal of Orthopaedics, Journal of Orthopedic Trauma, International Research Journal of Medicine and Medical Sciences, PM & R Journal*. He also serves as an associate editor for Journal of Orthopedic Trauma and an editorial board member of the *American Journal of Orthopedics, Current Orthopaedic Practice* and *World Journal of Orthopedics*. He serves as a section editor for The Journal of Bone and Joint Surgery Reviews. Dr. Baldwin is an active member of CORTICES Study Group and CORTICES Research Committee. He also received the prestigious *Standard Research Grant* from Scoliosis Research Society.

Patrick Cahill, MD started his term as Board of Director for Pediatric Cervical Spine Study Group. He serves as Chair for Health Policy Committee and a member of the Governance Council, Pediatric Device Task Force, and Program Committee at Scoliosis Research Society. He is also a member of POSNA's Quality, Safety, Value Initiative Committee and Advocacy Committee. He continues to serve as an Associate Editor for *Spine Deformity Journal* and as a reviewer for the *Journal of Bone and Joint Surgery—American* and the Thrasher Research Fund. Dr. Cahill is an active member in the Harms Study Group, Pediatric Spine Study Group, and Fox Pediatric Spine Deformity study group, which are multi-center groups prospectively researching care improvements for complex pediatric spine deformities. Dr. Cahill continues to serve as co-PI from Scoliosis Research Society titled, "*New Strategies for Pulmonary Assessment in Spinal and Chest Wall Deformity*". He is the Director for *Wyss/Campbell Center for Thoracic Insufficiency Syndrome*.

Robert Carrigan, MD continues to serve on the ASSH Fellows Conference Committee, AAOS Appropriate Use Committee, and POSNA Resident Newsletter Committee. He also serves as a reviewer for *Journal of Hand Surgery* and *Clinical Orthopaedics and Related Research*.

Richard Davidson, MD has continued to serve as an associate editor for Foot & Ankle, International. He also serves as a reviewer for *Clinical Orthopedics and Related Research*. Dr. Davidson serves on the editorial board for, Children's Doctor, a publication of the Doctors of The Children's Hospital of Philadelphia.

B. David Horn, MD continues to serve as a reviewer for journals, such as *Clinical Orthopaedics and Related Research (CORR), Pediatric Emergency Medicine, and Pediatrics*.

Jack Flynn, MD, Chief of the Division of Orthopaedics, continues to serve as a Director on the American Board of Orthopaedic Surgery and began serving as President of the Pediatric Spine Study Group/Pediatric Spine Foundation. He also was selected to serve on the JBJS Board of Trustees. Dr. Flynn is a co-editor of *Lovell and Winter's Pediatric Orthopaedics, Rockwood's Fractures in Children, Operative Techniques in Pediatric Orthopaedics*. Dr Flynn serves on the Editorial Board of *Journal of Spinal Deformity*. He was the Presidential Guest Speaker at the 2022 POSNA Annual Meeting, and in 2022 served as Visiting Professor at Children's Hospital Los Angeles, UC Davis Residency Graduation, Case Western Orthopaedics and University of Wisconsin.

Theodore Ganley, MD is the Sports Medicine Director at CHOP was the second VP of the Pediatric Research in Sports Medicine (PRISM) group, co-founder and executive board member as well as President for the Research in Osteochondritis Dissecans of the Knee (ROCK) group, executive committee member for the American Academy of Pediatrics, advisory board member for the International Pediatric Orthopaedic Symposium, and program chair for the Philadelphia Orthopaedic Society. Along with his leadership roles, he continues to be actively involved in biomechanical studies utilizing cadaver specimens in collaboration with the *Biedermann Lab for Orthopaedic Research* and *Human Motion Lab*. He is leading a nationwide initiative on Tibial Spine prospective study group with 14 sites currently participating and it was funded by *Arthur H. Huene Memorial Award* from POSNA. Additionally, he is the site leader for the FDA clinical trial for studying the efficacy and safety of autologous cultured chondrocytes on porcine collagen membrane (MACI). Dr. Ganley also serves as the site PI for recently NIH funded grant "*IMPACCT: Infrastructure for Musculoskeletal Pediatric Acute Care Clinical Trials*".

John Todd Lawrence, MD, PhD continued his collaborative work with Dr. Leo Han at Drexel University. Funded by the National Science Foundation, the project focused on conducting in vitro studies for a novel cartilage repair strategy. Dr. Lawrence is an active member of sports medicine multicenter research groups such as PLUTO and he leads a 12-site study group MEMO, which is the largest group studying medial epicondyle fractures and injuries. He continues to serve as a reviewer for the *American Journal of Sports Medicine (AJSM), Journal of Shoulder and Elbow Surgery (JSES), Journal of Children's Orthopaedics (JCO), Journal of Bone and Joint Surgery (JBJS), and Clinical Orthopaedics and Related Research (CORR)*. Dr. Lawrence continues to serve as a co-PI from NIH titled "*A Low-Cost, Collaborative Tool for the Tracking of Youth Activities to Reduce Risk of Physical Injury*" and site Co-PI for recently NIH funded grant "*IMPACCT: Infrastructure for Musculoskeletal Pediatric Acute Care Clinical Trials*".

Kathleen Maguire, MD is our new faculty member continuing her work at our Sports Medicine Performance Center. She is an active member of AAOS Emerging Leaders Program, POSNA, American Orthopaedic Society for Sports Medicine, American College of Sports Medicine,

Arthroscopy Association of North America, and the American Medical Association. Dr. Maguire serves as a reviewer for the *American Journal of Sports Medicine*.

Wudbhav Sankar, MD is the Director of the Young Adult Hip Preservation Program at CHOP. Dr. Sankar currently serves as Secretary for the Pediatric Orthopaedic Society of North America (POSNA) and co-director of the International Hip Dysplasia Institute. He remains active in several study groups including Academic Network of Conservational Hip Outcomes Research (ANCHOR), SCFE Longitudinal International Prospective Registry (SLIP) and International Perthes Study Group (IPSG). Also, he serves as co-director for the International Hip Dysplasia Institute (IHDI) Medical Advisory Board. Dr. Sankar is currently a reviewer for the *Journal of Bone and Joint Surgery, Journal of Pediatric Orthopaedics, Clinical Orthopaedics and Related Research, Journal of Pediatric Orthopaedics.* Dr. Sankar also serves as an Editorial Board Reviewer for *Techniques in Orthopaedics and Journal of Children's Orthopaedics*.

Apurva Shah, MD, MBA continues his tenure as the Director of Clinical Research. He continued to serve as co-PI on the grant from Orthopaedic Trauma Association titled, "*Opioid utilization after rotational ankle fractures*". He continues to serve as the team leader and traveled to Sigua Tepeque, Honduras for a pediatric hand surgery medical mission. Dr. Shah is currently a reviewer for the *Journal of Bone and Joint Surgery* and *Journal of Pediatric Orthopaedics.* Dr. Shah is also serving as the PI for Angela S.M. Kuo Memorial Award from POSNA for his research project "*Opioid vs. Non-Opioid Analgesia in Pediatric Supracondylar Humerus Fractures*." He also serves as the site Co-PI for recently NIH funded grant "*IMPACCT: Infrastructure for Musculoskeletal Pediatric Acute Care Clinical Trials*".

David Spiegel, MD continued his work with the Children's Hospital of Philadelphia Global Health Pilot Grant. He currently is the chair for the International Scholars Program at AAOS. Dr. Spiegel continued to be an active academic internationally, giving lectures in Iraq, Nepal, and Pakistan.

Lawrence Wells, MD is the Associate Director of the Sports Medicine Performance Center at CHOP. Dr. Wells currently serves as the President of Board of Directors for the Philadelphia Orthopaedic Society and as Vice Chair for Inclusion, Diversity and Equity at the Perelman School of Medicine.

Brendan Williams, MD continued his work at our Sports Medicine Performance Center. Dr. Williams serves on AAOS Emerging Leaders Program and a member of American Academy of Orthopaedic Surgeons, American Academy of Pediatrics, POSNA, Pediatric Research in Sports Medicine, and PRISM. He continued his tenure as Board of Directors for Children Beyond Our Borders. Dr. Williams serves as an ad hoc reviewer for *Pediatrics, The Journal of Bone and Joint Surgery—Case Connector, and The American Journal of Sports Medicine*.

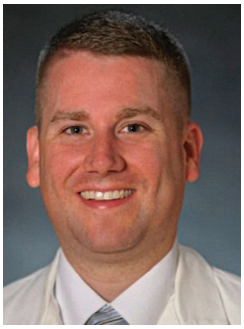
Corporal Michael J. Crescenz Philadelphia VA Medical Center

David Steinberg, MD

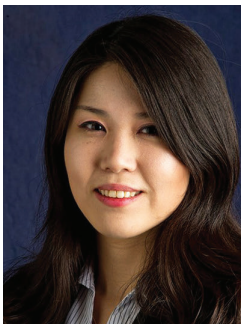
Joseph Bernstein, MD

L. Scott Levin, MD

Eric Hume, MD



Andrew Kuntz, MD



Hannah Lee, MD, PhD

Harvey Smith, MD

David Steinberg, MD

Timothy Costales, MD

We formally expanded our services in joint reconstruction and orthopaedic trauma with the addition of Timothy Costales, MD to our faculty in October. He completed his orthopaedic residency at the University of Maryland/Shock Trauma and an adult joint reconstruction fellowship at Massachusetts General Hospital/Harvard Medical School. The orthopaedic section will continue to grow with the arrival this summer of Colonel Jean-Claude D'Allyerand, MD, MSE, an orthopaedic traumatologist and Associate Professor of Surgery at USUHS, who is currently serving as Deputy Hospital Commander for Surgical Services at Landstuhl Regional Medical Center in Germany. He will assume the role of Chief of Orthopaedic Surgery at the VA, as well as joining the ranks of the orthopaedic trauma service at PPMC.

We continue to offer general orthopaedic and subspecialty services to our veterans, under the guidance of our dedicated faculty, including Drs. Joe Bernstein, Eric Hume, Andy Kuntz, Hannah Lee, Scott Levin, Harvey Smith, and David Steinberg, as well as the aforementioned Dr. Costales. During a twelve-month period, over 7000 visits were recorded for the outpatient Orthopaedic Surgery Clinics, which is a significant increase from the previous year. We offer surgical services four days weekly and performed over 445 operative procedures last year. We would not be able to provide comprehensive orthopaedic care to our patients without the dedication of our residents, hand fellows, our orthopaedic nurses (Alex David, John Yohannan & Mary Manattu), the other clinic staff, and our PA's. We saw the departure of one of our physician assistants, Thomas Bialkowski, who relocated to the warmer climes of Florida. Fortunately, we were able to recruit Eric Drennan, MPA-C, who brings with him a wealth of orthopaedic experience. He will be joining

Corporal Michael J. Crescenz VA Medical Center

long-time orthopaedic physician assistant, Mitchel "Chip" Staska, MPA-C, the ever-dependable driving force of the clinical arm of orthopaedics. Together, they will continue to provide a seamless journey for our patients, from initial referrals and consultations through outpatient evaluations, both in-person and via telemedicine. They assist the residents and faculty in managing patients, ordering appropriate diagnostic tests & consults, and scheduling surgeries; helping our team to efficiently navigate through a complex medical system.

Our faculty, residents and Penn's scientists, engineers & research staff continue to forge new paths through the partnership between the McKay Orthopaedic Laboratory and the PVAMC Translational Musculoskeletal Research Center. Drs. Bernstein, Kuntz, Steinberg, Lee, and Smith have each applied for or been awarded research grants through the Veterans Administration and outside funding sources. Working alongside Drs. Mauck, Soslowsky, Scanzello, Dyment, Cullen, Dodge, Gullbrand, & L Smith, they investigate many facets of the musculoskeletal system, focusing on the repair & regeneration of tendon, cartilage, meniscus, nerve, & the intervertebral disc. These studies have resulted in presentations at numerous national and international conferences, and publications in premier orthopaedic and biomedical research journals (including *Tissue Engineering*, *J Biomechanics*, and *J Orthopaedic Research*).

In addition to patient care and research, the orthopaedic program at the VAMC promotes the third pillar of academic medicine, education. In addition to clinical and surgical training of residents, hand fellows and students, we have expanded our educational efforts. This includes weekly resident conference with Dr. Kuntz, quarterly Chairman's rounds with Dr. Levin, and the an arthroscopic simulator.

With the team effort of our orthopedic family, and in collaboration with other services at the Veterans Administration Medical Service, we strive to provide our veterans with exceptional health care, by providing compassionate patient care, advancing medical knowledge through world-class research, and educating our next generation of health care providers.

Pennsylvania Hospital

Neil Sheth, MD

Pennsylvania Hospital (PAH) has a rich history in Philadelphia as the nation's first hospital. Founded in 1751 by Benjamin Franklin and Dr. Thomas Bond, the hospital was intended as a safe haven for the care of the "sick-poor and insane of Philadelphia." Located in the heart of South Philadelphia, its brand name draws thousands of patients annually to receive their care at the corner of 8th and Spruce Streets.

Education is at the forefront of our focus at PAH. Residents are typically in the operating room three to four days per week, with dedicated clinic time in multiple sub-specialties. Video conferencing continues for conferences across all sub-specialties, historically held at PMUC, and weekly sub-specialty specific conferences for spine and foot and ankle continue to be coordinated virtually.

The administration at Pennsylvania hospital continues to be extremely supportive of the expanded presence of orthopaedic faculty and residents. The hospital system has further increased the number of physician extenders, doubled the OR block time for the department, and increased physical space for clinical work and administrative duties. Their continued support is critical as the orthopaedic volume continues to grow and additional attendings are added to the faculty. These efforts have allowed PAH to maintain its reputation in the region as a first-class hospital.

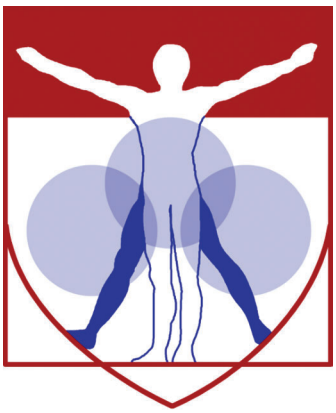
The Department of Orthopaedic Surgery at the University of Pennsylvania now staffs 20 attending surgeons and non-operative providers from various sub-specialties to populate the orthopaedic clinic in the Cathcart Building and the Farm-Journal Building. Among the sub-specialties represented are adult hip and knee reconstruction, foot and ankle, hand/plastic surgery, neuro-orthopaedics, shoulder and elbow, spine/deformity, sports medicine, and trauma. Notable for this past year, Dr. Lorraine Boakye (foot and ankle surgery) has been the newest addition to the roster at Pennsylvania Hospital.

With the continued increase in operative volume, PAH continues to be staffed by a PGY-1, PGY-2, PGY-4 and complemented by a team of nurse practitioners and physician extenders that assist with patient clinical care and floor work. The Orthopaedic Intern spends a portion of the week in the operating room or across various outpatient clinics and also assists the PAH team with patient care issues on the floor.

With the continually changing healthcare environment, we continue to grow the outpatient total joint arthroplasty program which started five years ago. We have implemented and continue to refine the dedicated rapid recovery program – the 9th floor extended stay unit opened in October 2019 and now services nearly 60%+ of the orthopaedic patient volume coming through PAH. PAH has also demonstrated an increased commitment to the use of robotics for performing total joint replacement over the past year positions PAH to be competitive in the region as we continue to evolve.

Penn Center for Musculoskeletal Disorders

Louis J. Soslowsky, PhD



The Penn Center for Musculoskeletal Disorders (PCMD) was initiated in 2004 with a goal to bring musculoskeletal researchers across campus together at the University of Pennsylvania. In 2006, the National Institute of Arthritis and Musculoskeletal Skin Diseases of the NIH funded our center grant proposal at which time we became one of five such NIH-recognized Centers in the country (www.med.upenn.edu/pcmd). In 2011, this Center grant was renewed for another five years and was the only one of the three up for renewal that was re-funded that year. Through the review by the NIH, Penn scored a perfect "ten" and was hailed as "exceptional" by the review panel! In 2016, we received another "exceptional" score, highest ranked in the country, by the NIH review panel and were renewed for another five years. We were pleased that in 2021, we were renewed again for five more years. We remain the longest running such center in the country.

The overall goal of this Center is to promote cooperative interactions among investigators, accelerate and enrich the effectiveness and efficiency of ongoing research, foster new collaborations and new research, and ultimately, translate our research efforts into better and new therapies for musculoskeletal disorders. The central theme of the Center continues to be "Musculoskeletal Tissue Injury and Repair". This theme is broad (as it includes all musculoskeletal tissue types, such as bone, cartilage, disc, ligament, meniscus, muscle, and tendon), focused (as takes advantage of commonalities in approaches across tissue types), and clinically significant (as it fosters development of assays, procedures and knowledge in pre-clinical animal and human models of translational relevance). It is important to note that our PCMD is not a "bone center" nor is it a "muscle center". Rather, it is truly a "musculoskeletal center" and has emerged as the recognized home for musculoskeletal research across the Penn campus and as a technical and intellectual resource for the broader Philadelphia musculoskeletal research community. Thus, the primary overall aims of this Center are to enhance and advance the research productivity of investigators in musculoskeletal tissue injury and repair by: 1) Providing innovation within critical resource core facilities in areas that cross disciplines, length scales, and hierarchies. These core facilities are mCT Imaging, Biomechanics, and Histology, 2) Developing a pilot and feasibility grant program for investigators, with direct mentorship, whereby new approaches, ideas, and collaborations can be developed prior to seeking extramural funding, and 3) Developing educational and research enrichment programs spanning tissue types, research approaches, and paradigms, through which members can learn from national leaders and from each other. High quality musculoskeletal research is currently being conducted by many groups at Penn. While many bring sophisticated approaches to bear on musculoskeletal problems, few groups have the required expertise and facilities to perform high quality and specialized assays in their own labs. Furthermore, most investigators are not aware of approaches utilized, and results obtained, in other tissues that may have direct relevance on their research questions. Ultimately, close cooperation, communication, and collaboration among researchers across musculoskeletal tissue types and from a wide variety of disciplines will significantly enhance the research of our members. The Center will provide opportunities to integrate multi-disciplinary techniques to determine mechanisms for tissue function, injury, degeneration, repair, and regeneration, with the ultimate goal of advancing the diagnosis, treatment, and prevention of diseases and injuries of the musculoskeletal system.

The Center currently has a membership of more than 180 faculty across five schools at Penn (Perelman School of Medicine, School of Engineering and Applied Science, School of Veterinary Medicine, School of Dental Medicine, and School of Arts and Sciences). We also now have 70 affiliate faculty members for more than 20 institutions regionally as we expand the reach and impact of our Center. For more information on the PCMD, please visit our website at www.med.upenn.edu/pcmd.

Clinical Research

Annamarie D. Horan, MPA, PhD

U.S. Regulations General Review

The mention of regulations in any capacity is an easy conversation killer in a mixed group of people. Whether one is discussing environmental protections, Constitutional Rights, or as appropriate to this document, Human Subjects Research, regulations are viewed as either fortresses against progress or guardians of public safety. Perhaps a more pragmatic approach is to view the current regulatory environment in the same way as we do guardrails on a highway. Drive between the guardrails and you stay out of the gutters or prevent yourself from careening off a cliff. Head straightaway into the guardrails and suffer damage.

The regulatory environment for Human Subjects Research includes but is not limited to studies of drugs, devices, biologics, the assurance of Human Subjects protections, quality management systems for producing products for human use, the conduct of post marketing surveillance, and the management and protection of individual privacy rights. In the United States these responsibilities are matrixed across multiple federal agencies in both the Executive and Judicial Branches. The full, searchable, and continually updated Code of Federal Regulations (CFR) can be accessed online at https://www.ecfr.gov/. Currently Titles 1 – 50 are available online.

In the Executive Branch, the Department of Health and Human Services (HHS), *Office of the Secretary* houses the Office for Civil Rights (OCR) which is responsible for enforcing the Privacy and Security Rules. The complete suite of HIPAA Administrative Simplification Regulations can be found at 45 CFR Part 160, Part 162, and Part 164, and includes: Transactions and Code Set Standards, Identifier Standards, Privacy Rule, Security Rule, Enforcement Rule, and Breach Notification Rule. HHS established general rules for the Protection of Human Subjects in Research under 45 CFR 46. The HHS *Operating Divisions* include several agencies engaged in the conduct and oversight of both human and non-human research such as the Agency for Healthcare Research and Quality (AHRQ), Centers for Disease Control and Prevention (CDC), the National Institutes of Health (NIH), and the U. S. Food and Drug Administration (FDA). The FDA specific regulations are found under Title 21 CFR. All Human Subjects Research is under the jurisdiction of 45 CFR 46, but not all these activities meet the criteria for FDA oversight.

The FDA jurisdiction is broader than research. FDA sets and enforces rules around Food for human and animal consumption, Cosmetics, Drugs for human and animal use, Medical Devices, Biologics, sets standards for products and activities around Radiologic Health, and oversees some aspects of tobacco regulations. FDA also interacts with other agencies under the Executive Branch in Human Subjects Research with respect to specimen shipping (Department of Transportation Title 49 CFR), United States Customs and Border Protection (CBP) (United States Department of Homeland Security Title 8 CFR Chapter 1). The Judicial Branch of the Federal Government influences Human Subjects Research through the Drug Enforcement Administration (DEA) and the Bureau of Alcohol, Tobacco, Firearms, and Explosives (ATF). Additionally, the FDA works with independent federal authorities such as United States Consumer Product Safety Commission (USCPSC, CPSC) and the Federal Trade Commission (FTC) and innumerable state, local, and international organizations to conduct its myriad duties to ensure safety and efficacy of drugs and devices and other products marketed in the United States.

Penn Orthopaedics Update 2023

At this writing, the Department is quite a bit leaner than it has been in a decade. Dozens of defunct studies were administratively closed with the assistance of IRB Administrators and removed from our regulatory burden. It is critically important for all PIs to ensure regular maintenance of their studies even if the IRB rules the protocol to be *Exempt* or to be *Approved with No Continuing Review Required*. Once a study is opened and active, it must at some point come to a formal close. The best way to ensure this is to assign your Divisional CRC to the protocol as a Study Contact. All protocols are reviewed regularly and the CRCs do stay on top of their dockets to keep the PIs within the guardrails.

Additional compliance requirements applicable to all studies include the ongoing management of every study in the PSOM Clinical Research Management System (CRMS). This now includes the capture of retrospectively "enrolled" patients and excluded/declined patients. There will be more details forthcoming. Again, the Departmental CRC Team all have access to the CRMS and will be undergoing additional training to facilitate this higher compliance standard. We look forward to working with our PIs to further tune up our Institutional presentation.

Activity Report

The data below were presented to the faculty on March 20, 2023. As shown in Table 1, the Department carries a burden of 126 open protocols, only 26 of which are extramurally funded. The funding sources include Industry, Federal, non-Federal, and private. Table 2 provides a breakdown of

Table 1 Open vs Funded Protocols by Sub-Specialty Division

Division	Total Open Protocols	Funded
Adult Reconstruction	22	9
Foot & Ankle	12	2
Hand	25	5
Oncology	7	0
Shoulder & Elbow	9	4
Spine	4	0
Sports	29	3
Trauma	18	3
Grand Total	**126**	**26**

Table 2 Funding by faculty PI

Funded PI's	# Awards
Carey, James L	3
Farber, Daniel C	2
Hume, Eric L	2
Israelite, Craig L	2
Kuntz, Andrew	4
Levin, Lawrence S	3
Lin, Ines C	1
Mehta, Samir	3
Nelson, Charles L	1
Sheth, Neil P	2
Steinberg, David R	1
Tarity, Thomas D	1
Travers, Christopher	1
Grand Total	26

*Awards = Active projects only

the individual faculty funded as of March 20, 2023. Since March 2023, the specific distribution of projects and the number of funded projects has changed and should always be understood to be in flux as old protocols close and new studies begin. Regardless of funding source, in an ideal world, all protocols would be associated with extramural funding. We do not live in an ideal world. Pragmatically, the minimum goal is for every Division to seek sufficient funding to support at least 1 FTE CRC and to not have an unfunded study burden that exceeds the capacity of the CRCs per Division to fully manage, given the totality of administrative regulations applicable to all studies. From the perspective of optimal faculty performance, regardless of source, annual revenues equivalent to an R01 level (e.g. ~ $250K direct costs) would signal great strength in the program. With these values in mind, it is our hope that faculty active in research will focus their energies accordingly. It is not expected that every faculty member engage in research and even distribution across the Department is not practical to expect. For those who are engaged, choosing studies with the greatest chance of full patient follow up is the best way to achieve target revenues.

From the perspective of compliance, the best way to ensure optimal compliance beyond initial IRB approval, combine your desire to engage resident & fellow trainees with the CRC team during initial IRB submission and keep them involved. Keep your data secure. Keep your records in a manner that is aligned with applicable regulations and internal policies. Stay within the guidelines. Become the best PIs you can be.

Adult Reconstruction remains highly productive with 22 open studies, 9 of which are extramurally funded. The myMobility study (NCT03737149) led by Dr. Israelite continues to perform well against our peer institutions. Penn is #1 university enrolling site for 2022. In March of 2023 we reached the 300 subject milestone and continue to be a high enrolling site each month.

Dr. Nelson is having continued success with the PCORI funded PEPPER (NCT02810704) study with ongoing enrollment and 48 Subjects in active follow up. Dr. Nelson has joined the Rush University Medical Center Consortium in their dose finding study of Dexamethasone in TKA (NCT05018091). He has also anticipating funding as a site PI for a multicenter study of Autogenous Bone Marrow Aspirate Concentrate for the Treatment of Osteonecrosis of the Femoral Head (Johns Hopkins, Primary Site). In addition to all these activities, Dr. Nelson will be taking over the final stages of DePuy funded studies of Dr. Hume's Ceramic on Ceramic Hip study (NCT02096211). Dr. Hume will continue to be PI on the Smith & Nephew R3 Delta Ceramic Acetabular System PAS U.S. (R3-PAS) protocol (NCT03056534) as that wraps up this fall. Dr. Travers has nearly completed the DePuy ACTIS study (NCT02783274) and the NIH funded EN20-01 Centrexion Knee OA Study (NCT05025787) has successfully randomized its 1st Subject!

Foot & Ankle is back on track with Dr. Farber's Treace Medical Concepts, Inc.'s Early Weight-Bearing After the Lapiplasty Mini-Incision Procedure (Mini3D) study (NCT05082012). We anticipate that additional updates for the Foot & Ankle Division will be submitted elsewhere in this edition of the UPOJ by colleagues Dr. Josh Baxter and Dr. Casey Humbyrd.

Hand Surgery received notification in March that an academic (UC San Diego)-industry (Auxillium)-federal (NIH) collaborative grant was awarded to Dr. Bozentka. The administrative processes on this award are still pending. We are looking forward to beginning this study!

In the last issue, Dr. Levin's DOD-funded Hand Transplantation Qualitative Research Study (W81XWH1820067) achieved the 1st Manuscript milestone. The publication information was not available at that time and is now available and the link is now presented. https://www.archives-pmr.org/article/S0003-9993(23)00031-X/fulltext This major grant is wrapping up in the fall and additional publications are under development as are other collaborations and funding opportunities. Dr. Andrew Sobel as the Director of the Hand Surgery Clinical Research Program, is striving to develop an educational pipeline within the Division that may also aid the Department. Please follow up with him for details.

Shoulder & Elbow continues its strong Clinical Research presence over the past 10 years with 4 industry funded studies ongoing. We welcomed Dr. John G. Horneff to the Shoulder & Elbow Division, his ASES studies continue to remain active and growing. Shoulder & Elbow has remained a strong and stable Division in Clinical Research under Dr. Kuntz's leadership.

Spine will continue the STRUCTURE study (NCT04294004), a Phase II study enrolling patients undergoing single level transforaminal lumbar interbody fusion. Dr. George Dodge will be handing the role of PI over to Dr. Harvey Smith. Dr. Casper is on the brink of executing a new study with Carlsmed, Inc. to capture registry information for Personalized aprevo® surgery patients.

Sports Medicine continues to have a robust repertoire of active funded projects. Dr. Carey continues in his role as the Local and Global PI on the Vericel sponsored PEAK study (NCT03588975) and also participates in a Vericel sponsored retrospective study. Drs. Kelly and Dodge's study investigating the impact of Kenalog injections on metabolic syndrome biomarkers is drawing to a close. We look forward to the results of this impactful investigation.

Ortho Trauma has several active studies in process, previously described. He is also in pending execution of a new registry study with Curvafix, Inc. Dr. Mehta & Dr. Horan also celebrate the issue of U.S. Patent No. 11,339,436 on May 24, 2022 for their work on Biomarker identification in Fracture Healing. Hopefully, there will be more to follow on this milestone in Orthopaedic Trauma.

Financial Report

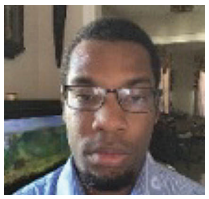
Figure 1 shows the Total Costs (Direct Costs + Indirect Costs) expended during the periods shown for all categories (Personnel and Non-Personnel Costs). The revenue sources for these expenditures include both current sources as shown in Table 1 as well as previously earned revenues that remain available from completed projects. Unless projects are grant funded, revenue supporting Clinical Research is received in a reimbursement method and therefore lags behind the performance period due to the invoicing and payment process. FY21 revenues were considerably lower than FY20 due to the COVID-19 driven full shutdown of non-COVID-19 related and non-life saving clinical research. FY22 revenues show healthy recovery in the program, though the strength and duration of recovery should be interpreted cautiously as the entire industry has irreversibly changed post-COVID-19. Our fingers are crossed that we are truly looking forward to a sustained restorative period.

Division	FY20	FY21	FY22	Sum FY20 - FY22	# Awards
Adult Reconstruction	$324,232	$120,360	$278,474	$723,066	17
Foot & Ankle	$10,138	$42,857	$55,406	$108,401	4
Hand	$337,829	$78,357	$175,873	$592,059	7
Oncology					0
Shoulder & Elbow	$103,053	$131,376	$149,603	$384,033	4
Spine	$500	$21,234	$54,646	$76,380	4
Sports	$76,931	$44,540	$93,926	$215,397	4
Trauma	$44,411	$3,007	$106,238	$153,655	7
Grand Total	**$897,094**	**$441,731**	**$914,165**	**$2,252,990**	**47**

Figure 1. Clinical Research Expenditures FY20-FY22

Our Team. We thank our Team of Dedicated CRCs. Shown below from left to right are Helena Moses, Warren Harding (Adult Recon), Mounika Ponakala (Sports Medicine), Ellen Stinger (Upper Extremity & Spine), Linda To (Ortho Trauma). Not pictured: Artsiom Meliukh (Adult Recon). Ellen Stinger and Warren Harding also serve Foot & Ankle.

Helena Moses
Adult Reconstruction



Warren Harding
Adult Reconstruction

Mounika Ponakala
Sports Medicine

Ellen Stinger
Spine, F&A, Hand

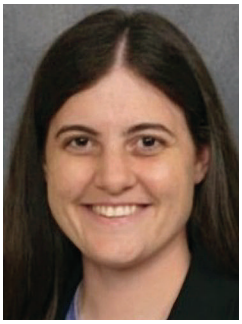
Linda To
Trauma

Samir Mehta, MD
Chief, Division of Orthopaedic Trauma, Medical Director of Clinical Research
Associate Professor of Orthopaedic Surgery

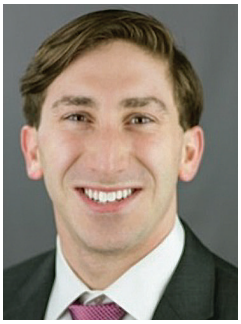
Annamarie Horan, MPA, PhD
Director of Clinical Research
Orthopaedic Surgery and Anesthesiology & Critical Care

Current Residents

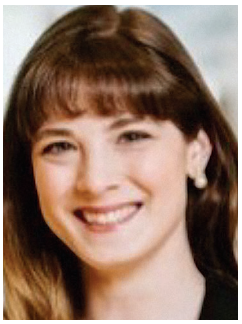
Clinical Year 5 Resident Spotlight



Lauren Boden, MD
Fellowship: Spine, Cleveland Clinic
Medical School: Emory University
Undergraduate: Pomona College

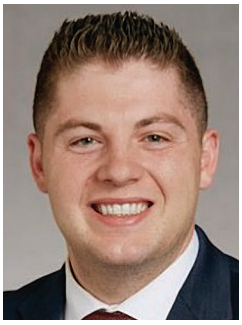


David Falk, MD
Fellowship: Spine, Washington University in St. Louis
Medical School: George Washington University
Undergraduate: University of Michigan

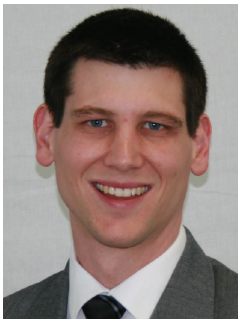


Kelsey Bonilla, MD*
Fellowship: Trauma, Cedars-Sinai
Medical School: Perelman School of Medicine at University of Pennsylvania
Undergraduate: Rutgers University

George Frylhofer, MD, MTE*
Fellowship: Spine, Rush University
Medical School: Perelman School of Medicine at University of Pennsy
Undergraduate: Harvard University



Ryan DeAngelis, MD
Fellowship: Trauma, UT Houston
Medical School: Cooper Medical School of Rowan University
Undergraduate: The College of New Jersey

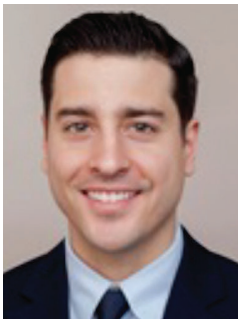


Joseph Koressel, MD
Fellowship: Spine, Stanford
Medical School: Weill Cornell
Undergraduate: University of California, Davis

*Indicates Resident is in the 6-year Research Track

Viviana Serra Lopez, MD,MS*
Fellowship: Hand, Hospital for Special Surgery
Medical School: University of Puerto Rico
Undergraduate: Massachusetts Institute of Technology

Brian Perez, MD
Fellowship: Joints, NYU
Medical School: Albert Einstein
Undergraduate: Rutgers University



Gregory Minutillo, MD, MPH
Fellowship: Joints, Rush University
Medical School: Tulane University
Undergraduate: James Madison University

*Indicates Resident is in the 6-year Research Track

Clinical Year 4 Residents

Stephen Barchick, MD

Undergraduate:
Harvard University

Medical School:
Duke University

Sachin Gupta, MD*

Undergraduate:
George Washington University

Medical School:
George Washington University

Joung (Richard) Kim, MD

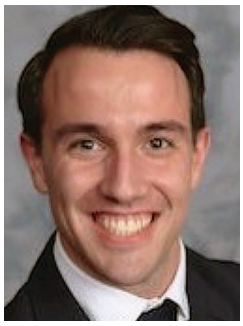
Undergraduate:
University of Rochester

Medical School:
Icahn School of Medicine at Mount Sinai

Charles Lucas Myerson, MD

Undergraduate:
University of Southern California

Medical School:
Tulane University



Matthew Stein, MD, MS*

Undergraduate:
Univ. of Maryland

Medical School:
Georgetown University

Kelsey Young, MD

Undergraduate:
Cornell University

Medical School:
Cornell University

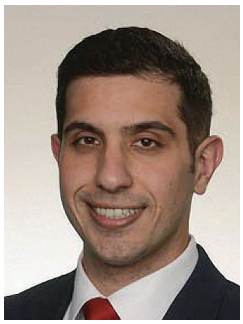
Steven Zhang, MD

Undergraduate:
Cornell University

Medical School:
Stanford University

*Indicates Resident is in the 6-year Research Track

Clinical Year 3 Residents



Aymen Alqazzaz, MD

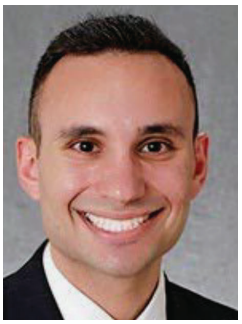
Undergraduate:
University of Maryland

Medical School:
University of Maryland

Ashleigh Bush, MD

Undergraduate:
Indiana University

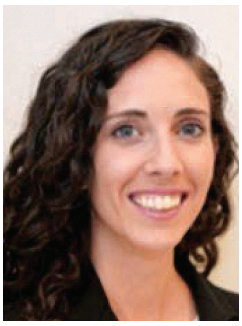
Medical School:
Indiana University



Jordan Cohen, MD*

Undergraduate:
University of Maryland

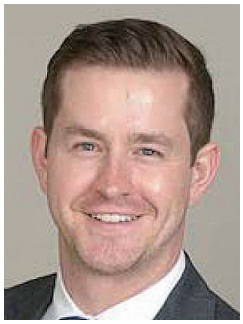
Medical School:
George Washington University



Kathleen Collins, MD

Undergraduate:
Morehouse School of Medicine

Medical School:
Virginia Polytechnic Institute and State University



Cody Hansen, MD

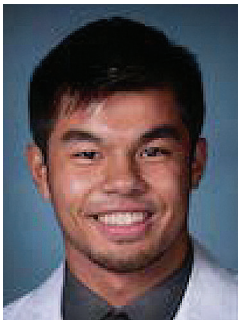
Undergraduate:
University of California San Diego

Medical School:
University of Denver

Kendall Masada, MD*

Undergraduate:
University of Texas

Medical School:
University of Texas Health Science Center



Brian Velasco, MD

Undergraduate:
Geisinger Commonwealth School of Medicine

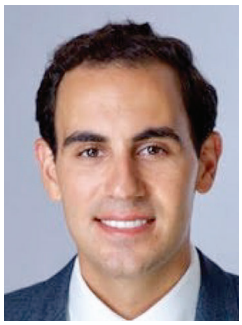
Medical School:
Franklin & Marshall College

Dainn Woo, MD

Undergraduate:
New York University

Medical School:
The City College of New York

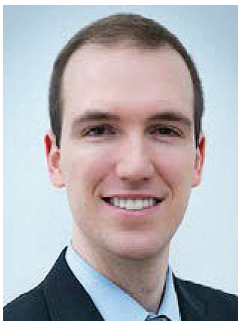
Research Year



Bijan Dehghani, MD*

Undergraduate:
Albany Medical College

Medical School:
Boston University



Jordan Cohen, MD*

Undergraduate:
University of Maryland

Medical School:
George Washington University

*Indicates Resident is in the 6-year Research Track

Clinical Year 2 Residents

Mohammed Abdullah, MD*

Medical School:
The University of Texas Medical Branch
Undergraduate:
The University of Houston

Caroline Granruth, MD

Medical School:
Tulane University

Undergraduate:
University of Virginia

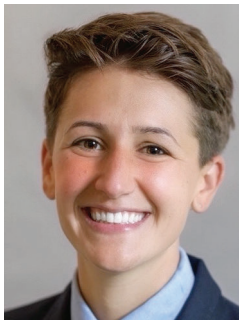
Jaret (Mac) Karnuta, MD, MS

Medical School:
Case Western Reserve

Undergraduate:
Duke University

Erin Kelly, MD

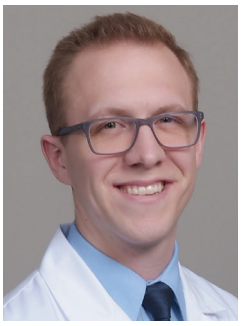
Medical School:
Wake Forest School of Medicine
Undergraduate:
Wake Forest University



Sand Mastrangelo, MD
Medical School:
Dartmouth
Undergraduate:
Brown University

Bradley Osemwengie, MD
Medical School:
Texas Tech
Undergraduate:
University of North Texas

Eric Schweppe, MD*
Medical School:
Columbia University
Undergraduate:
United States Military Academy



Weston Smith, MD
Medical School:
University of Utah
Undergraduate:
Brigham Young University

*Indicates Resident is in the 6-year Research Track

Clinical Year 1 Residents

Ellis Berns, MD

Medical School:
Brown University
Undergraduate:
Brown University

Anna Blaeser, MD

Medical School:
Albany Medical College
Undergraduate:
University of Minnesota

Emily Eiel, MD

Medical School:
University of Massachusetts
Undergraduate:
Colgate University

Rachel Flaugh, MD*

Medical School:
Harvard Medical School
Undergraduate:
University of Nebraska

Lisa Friedman, MD*

Medical School:
Case Western Reserve University
Undergraduate:
Carleton College

Samuel Oduwole, MD

Medical School:
Quinnipiac University

Undergraduate:
La Salle University

Alyssa Thorman, MD

Medical School:
University of Utah

Undergraduate:
University of Utah

Thompson Zhuang, MD

Medical School:
Stanford University

Undergraduate:
Princeton University

*Indicates Resident is in the 6-year Research Track

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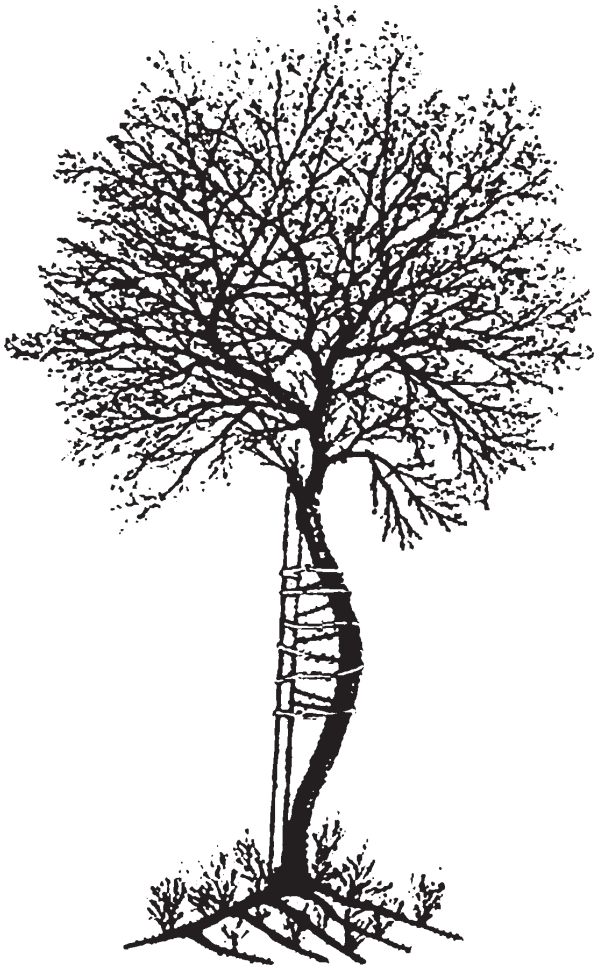

University of Pennsylvania Orthopaedic Journal

2022-2023 Clinical and Basic Science Research

The following sections highlight clinical and basic science research conducted at the University of Pennsylvania in the field of Orthopedics, including work from the Department of Orthopaedic Surgery, The McKay Laboratory for Orthopaedic Research, Children's Hospital of Philadelphia, and the Philadelphia Veterans Affairs Translational Musculoskeletal Research Center. In addition to research, each clinical section is preceded with a "Tips & Tricks" article highlighting case reports or surgical techniques for education and to display the breadth of musculoskeletal disease seen and treated in our hospital system.

Clinical Research Sections:

Trauma

Spine

Sports

Hand

Shoulder and Elbow

Adult Reconstruction

Foot and Ankle

Oncology

Orthoplastics

Arthroplasty

Pediatrics

Basic Science Research Sections:

Bone & Development

Cartilage, Meniscus & Disc

Muscle, Tendon, & Ligament

Trauma

Schatzker IV Tibial Plateau Fracture after Taser Utilization: A Case Report and Review of the Literature

Sand Mastrangelo, MD[1]
Ellis Berns, MD[1]
Cole Headen, BS[2]
Derek Donegan, MD MBA[1]

1Department of Orthopaedic Surgery
University of Pennsylvania

2Perelman School of Medicine
University of Pennsylvania

Introduction

A Taser is a type of conducted energy weapon (CEW) used to stun and disable targets via conduction of a high-voltage charge through a pair of barbed probes. Designed as a weapon to deter criminal behavior without causing lethal consequences, the Taser has been widely adopted by law enforcement across the United States. In fact, since its introduction in the 1970s, several design iterations have been released with its most recent touted as "state-of-the-art in conducted energy weapons, reducing injuries to officers and suspects"[1]. Nonetheless, there is a paucity of research surrounding the Taser's effectiveness in limiting civilian injuries during police-civilian encounters.

In fact, a review of the medical literature describes significant morbidity associated with Taser use. Multiple osseous injuries and injury patterns have been described including thoracic compression, phalangeal, and facial fractures. Phalangeal and facial fractures occur secondary to direct penetration of the Taser barb while spinal compression fractures result following high energy, seizure-like activity caused by the electrical discharge[2-11]. Despite this, there have been no specific reports of orthopaedic injury patterns as a consequence of Taser use. In this case report, we describe a high energy tibial plateau fracture as a result of Taser utilization.

Patient Presentation

This is a 24-year-old male patient who presented to our hospital emergency department in police custody on the day of injury. Per police report, a Taser was utilized during the patient's arrest. After the taser was fired into the patient's right knee, he reportedly tripped and forcibly landed onto his right lower extremity. Following the incident, the patient was unable to ambulate and was transported to the hospital for emergency evaluation. His urine drug screen was positive for opiates, benzodiazepines, oxycodone, and tetrahydrocannabinol (THC).

The patient's past medical history is significant for a previous facial gunshot wound in 2019 resulting in comminuted left maxillary wall, left zygomatic arch, and right mandibular body and ramus fractures for which he underwent open reduction and internal fixation (ORIF) with plastic and reconstructive surgery. Following this injury, the patient self-reports depression and post-traumatic stress disorder resulting in paranoia and chronic auditory and visual hallucinations of persecution by assailants and police. Eight months prior to presentation, he sustained a gunshot wound to the right elbow and right thigh without fracture, soft tissue, or vascular injury. He reported taking suboxone for opioid use disorder.

On exam, the patient had diffuse swelling and obvious deformity of the right knee with a Taser probe embedded in the prepatellar soft tissue without surrounding ecchymosis or erythema. He endorsed significant knee pain with inability to actively flex or extend his knee. There were no open wounds about the knee concerning for open fracture. Neurovascularly, the patient had palpable dorsalis pedis and posterior tibial pulses and a normal Ankle-brachial index (ABI). He had 5/5 strength in his tibialis anterior, extensor hallucis longus, flexor hallucis longus, and gastrocnemius/soleus complex. His anterior and posterior lower extremity compartments were swollen and full but compressible. Ligamentous knee exam was limited by pain and no firm endpoint was felt on varus stress. He had no pain with passive stretch of his ankle or toes and there was low concern for acute compartment syndrome upon initial presentation. Secondary trauma evaluation did not reveal additional injuries.

X-ray and CT scan of the right knee, tibia/fibula, and ankle were obtained following initial evaluation as shown in Figures 1 and 2. Imaging revealed a comminuted, intra-articular medial tibial plateau split and depression fracture pattern extending from to the medial tibial spine with approximately 1.5 cm of articular depression consistent with a Schatzker IV tibial plateau fracture. There was also an ipsilateral fibular head avulsion fracture. The retained Taser probe is visualized

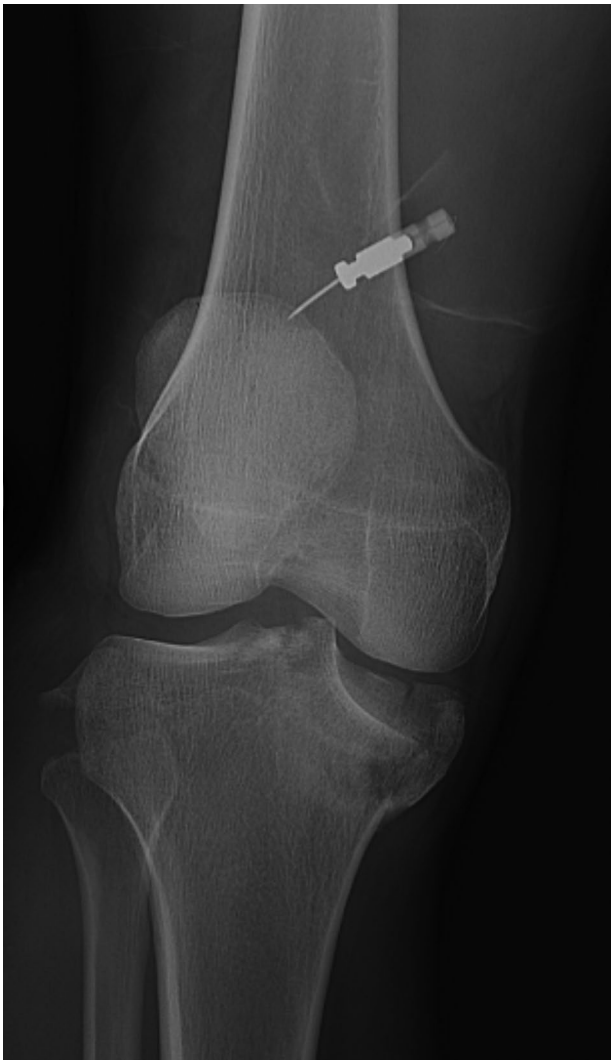

Figure 1. Anterior to posterior radiograph of the right knee demonstrating a Schatzker IV Tibial Plateau fracture and fibular head avulsion fracture as well as a retained Taser prong.

over the prepatellar soft tissue without bony penetration.

Initial attempts at gentle twisting and removing the retained Taser probe were unsuccessful due to the barbed projection at the distal aspect of the shaft. A Taser probe is similar to a #8 fishhook; most have a 4mm barb and 9.5mm shaft[12]. A subsequent attempt was made using 1% lidocaine without epinephrine injected superficially around the retained probe. With an #11 blade scalpel, a 3mm longitudinal incision was made extending through the entry point into the skin which allowed for rotation and rocking of the probe until the barbed tip was freed from the soft tissue. Xeroform and a soft dressing were placed about the anterior knee and the patient received Tetanus prophylaxis in the emergency department. He was admitted to the orthopaedic surgical service for serial compartment checks and neurovascular monitoring pending surgical management.

Operative Fixation

For this particular injury pattern, and owing to his significant soft tissue swelling, operative considerations

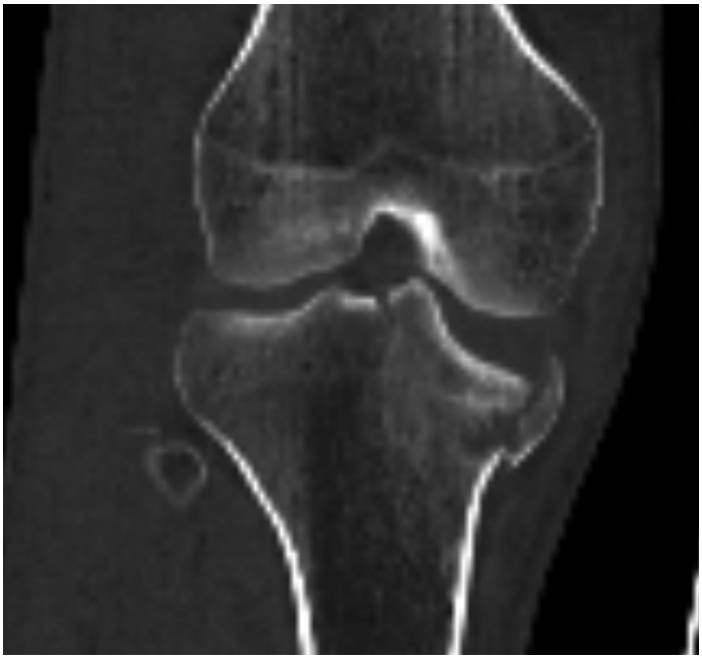

Figure 2. Coronal CT scan demonstrating medial articular depression and comminution consistent with a Schatzker IV fracture pattern.

included temporizing knee-spanning external fixation allowing for soft tissue rest versus acute open reduction internal fixation (ORIF). The day following the patient's injury, his lower extremity compartments were swollen, though compressible with adequate wrinkling, and the decision was made to acutely pursue definitive ORIF.

A medial approach to the tibial plateau was utilized to best visualize the fracture[13]. The fracture was thoroughly debrided and the medial articular depression was tamped superiorly to restore the anatomic joint surface. The resulting bony defect was filled with crushed cancellous allograft. A 3.5 Synthes medial tibial locking compression plate was utilized in buttress mode and plate balance was achieved under fluoroscopic guidance. The axillary screw was placed distal to the apex of the posteromedial fracture line permitting in-situ contouring and adequate compression across the fracture. A second, nonlocking screw was placed distally for rotational control. The remainder of the plate was secured via hybrid fixation using a combination of locking and nonlocking screws. Final fluoroscopic evaluation confirmed anatomic reduction of the articular surface without intra-articular penetration of hardware. The proximal fibula was minimally displaced and thus treated nonoperatively. The wound was thoroughly irrigated and closed in standard, layered fashion. The patient was made non-weight bearing in a knee immobilizer and discharged from the hospital after clearing a normal post-operative protocol including physical therapy.

Post-operative x-rays demonstrated application of the medial plate in excellent alignment as shown in Figure 3A. The patient was evaluated at four and ten weeks post-operatively. Radiographs at 10 weeks showed excellent interval healing of his fracture with maintenance of the

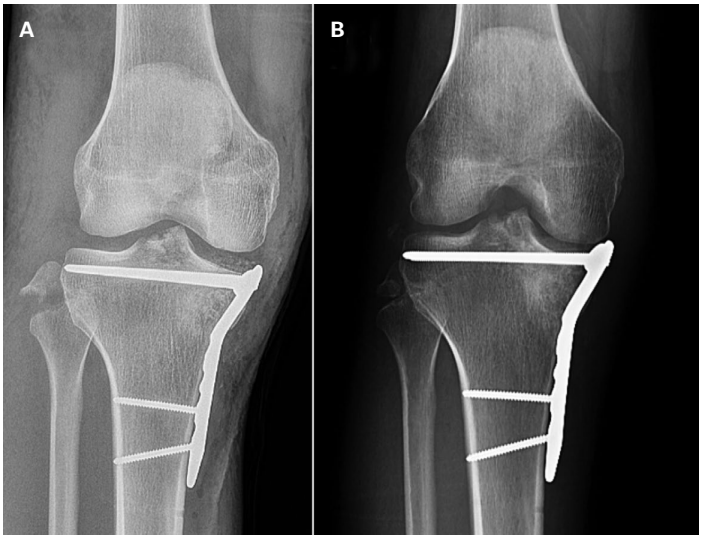

Figure 3 Post-operative open reduction internal fixation **(A)** 1 day post operatively **(B)** Ten weeks post operatively.

joint line and no hardware complications as shown in Figure 3B. He was advanced to weight bearing as tolerated on the operative extremity.

Discussion

The Schatzker classification is a ubiquitous, descriptive classification system aiding in both prognosis and management of tibial plateau fractures. Severity of injury tends to correlate with increasing numeric grade (I-VI) and worse functional outcome, illustrated in Table 1. Schatzker I-III tibial plateau fractures, for instance, typically result from lower energy trauma and are generally associated with osteopenic bone; in contrast, Schatzker IV-VI fracture patterns are caused by higher energy mechanisms such as motor vehicle collisions or pedestrians struck[14].

Notably, the patient described in this report sustained a Schatzker IV tibial plateau fracture (medial split and depression) as a result of Taser utilization during an encounter with law enforcement. In addition to the osseous injury, this particular fracture pattern is commonly associated with soft tissue injuries including anterior cruciate ligament (ACL), lateral collateral ligament (LCL), and medial meniscal tears[14]. Based on the severity of this patient's injury, he likely landed directly onto his right knee with a varus force resulting in medial tibial plateau impaction from the medial femoral condyle and tension-sided avulsion of the lateral collateral ligament from the fibular head.

Given the location of the Taser probe in the anterior aspect of the patient's knee, he was presumably running towards the officers. The patient states he "flipped" once he was struck; the electrical current discharged from the Taser likely caused involuntary muscle contraction of his lower extremity with simultaneous incapacitation of compensatory protective fall reflexes such as extending the hands or rolling towards the shoulder or side. The confounding intoxication of our patient with opiates, THC, and benzodiazepines may also have dampened normal self-protective mechanisms.

Recently, the use of excessive force in civilian encounters with law enforcement has been subject to intense scrutiny and public concern. In the United States, police use of force remains significantly higher than comparable democracies; between 2015 and 2019, nearly 1,000 people per year were killed during an encounter with law enforcement[15]. Among Black Americans, the rate of fatal police encounters is much higher than any other ethnicity; the reality is even bleaker among patients experiencing psychological distress[16] thus reifying the confounding consequences of racial disparity and mental health. Notably, the patient described in this

Table 1. Schatzker Classification for Tibial Plateau Fractures.[19]

Schatzker Classification	Definition	Classic Injury	Energy Required	Mechanism
Schatzker I	Wedge-shaped cleavage fracture of lateral tibial plateau	Sports injuries/osteoporosis	Low	valgus extension & flexion
Schatzker II	Splitting & depression of lateral tibial plateau	Sports injuries/osteoporosis	Low	valgus extension & flexion
Schatzker III	Pure depression of lateral tibial plateau & lateral depression	Sports injuries/osteoporosis	Low	valgus extension & flexion
Schatzker IV	Medial tibial plateau fracture	Motor Vehicle Accidents	High	varus extension & flexion hyperextension
Schatzker V	Wedge fracture of both lateral & medial tibial plateau	Motor Vehicle Accidents	High	varus and axial extension
Schatzker VI	Transverse tibial metaphyseal fracture & any type of tibial plateau fracture	Motor Vehicle Accidents	High	varus and axial extension

report is Black with a history of both substance use and psychiatric comorbidities.

Conclusion

Tasers are commonly perceived as innocuous weapons often used to reduce harm in civilian encounters with law enforcement. In practice, however, Taser discharge results in profound bodily force and is associated with significant morbidity[17]. Despite an effort by law enforcement to limit the use of excessive force in civilian encounters, non-fatal injuries have been on the rise[18]. There are few comprehensive studies on how these less-than-lethal weapons reduce harm and more research is needed pertaining to their enduring physiologic effect. While various Taser-related civilian injuries have been reported in the literature, this is the first report to describe a high-energy tibial plateau fracture as a consequence of Taser use.

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Spine

U·P·O·J

Evaluation and Initial Workup Guide of Spine Trauma Patients for Orthopaedic Surgery Residents

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Introduction

Trauma to the spine is common. For the consult resident challenges in diagnosis exist and managing critical patients can be a daunting task. Determination of stability requires understanding of relevant anatomy, imaging literacy, and a working knowledge of injury classifications to avoid catastrophic consequences. The consult resident must develop a systematic approach to evaluate and manage spine trauma patients. We hope the following guide will help to address critical aspects of the initial workup, examination, and diagnosis of different injuries in spine trauma patients.

Physical exam

The physical exam is one of the most important components of a spine exam. It involves inspecting the spine for alignment, assessing for any prior surgical scars or skin defects, as well as noting any muscular atrophy. Be sure to palpate for any tenderness or step-offs along the spine as well. Flexion, extension, rotation can also be assessed. Care must be taken with mobilization of a patient with concern for higher energy injury or injury to the spinal cord and in these instances strict spinal precautions should be employed with use of careful log roll with one person stabilizing the head and neck and two others carefully supporting the thoracolumbar region (Figure 1).

Sensation can be assessed in dermatomal distribution. This includes pain, light touch, vibration, and 2-point discrimination. The ASIA sensory grading system is as follows: 0= absent, 1= impaired, 2= normal, NT= not testable

Motor function can be assessed using the ASIA grading system. 0= no movement. 1= visible or palpation contraction. 2= full range of motion with gravity eliminated. 3= full range of motion against gravity. 4= full range of motion against gravity and moderate resistance. 5= full strength and gravity and resistance.

Figure 2 and 3 show tabled that demonstrates motor testing as well as the affected muscle and associated neural structure for the upper extremity and lower extremity respectively.

Spine reflexes should also be tested by the consult resident. The following spinal nerve levels are associated with the corresponding reflex. C5= biceps brachii reflex. C6= brachioradialis reflex. C7= triceps brachii reflex. L1/2= cremasteric reflex L4= patellar tendon reflex. S1= Achilles tendon reflex.

Figure 1. Example of a logroll of a patient to maintain spinal precautions. One person stabilizes the head and neck and two others carefully support the thoracolumbar region.

Motor Testing of Upper Extremity Muscles			
Primary Motion	**Primary Muscle**	**Innervation**	**Nerve Root**
Scapular stabilization	Serratus	Long thoracic n.	C4
Shoulder abduction	Deltoid	Axillary n.	C5
Shoulder internal rotation	Subscapularis	Subscapular n.	C5
Shoulder external rotation	Infraspinatous	Suprascapular n.	C5
Elbow flexion (palm up)	Biceps & Brachialis	Musculocutaneous n.	C5
Elbow flexion (thumb up)	Brachioradialis	Radial n.	C6
Wrist extension	ECRL	Radial n.	C6
Wrist supination	Supinator	PIN	C6
Elbow extension	Triceps	Radial n.	C7
Wrist flexion	FCR & PL	Median n.	C7
Wrist pronation	PT & PQ	Median n.	C7
MCP & PIP finger flexion	FDS	Median N.	C8
DIP finger flexion	FDP	Ulnar n. & AIN	C8
Thumb extension	EPL	PIN	C8
Finger abduction	Interossei	Ulnar n.	T1

Figure 2. Upper extremity musculature action and innervation.

Nerve root	*Primary Motion*	*Primary muscles*
L1		
L2	Hip flexion and adduction	Iliopsoas (lumbar plexus, femoral n.) Hip adductors (obturator n.)
L3	Knee extension (also L4)	Quadriceps (femoral n.)
L4	Ankle dorsiflexion (also L5)	Tibialis anterior (deep peroneal n.)
L5	Foot inversion Toe dorsiflexion Hip Extension Hip abduction	Tibialis posterior (tibial n.) EHL (DPN), EDL (DPN) Hamstrings (tibial) & gluteus max (inf. gluteal n.) Gluteus medius (sup. gluteal n.)
S1	Foot plantar flexion Foot eversion	Gastroc-soleus (tibial n.) Peroneals (SPN)
S2	Toe plantarflexion	FHL (tibial n.), FDL (tibial)
S3 & S4	Bowel & bladder function	Bladder

Figure 3. Lower extremity musculature action and innervation.

Provocative tests

The spurling test which assesses for cervical radiculopathy. This is performed by rotating the head to the affected side, extending the neck, and applying an axial load to the head. The test is positive if pain radiates down the ipsilateral affected arm.

Hoffman's Test assessed for cervical myelopathy. It is performed by stabilizing the long finger and flicking the distal phalanx into extension. A positive test causes involuntary contraction of the thumb interphalangeal joint.

Lhermitte's sign tests for cervical spinal cord compression and myelopathy. A positive test occurs when cervical flexion or extension produces a shocking sensation down the spine or into the appendages.

The straight leg raise can be used to assess compression of the lower lumbar nerve roots. This test is positive if

radicular symptoms are produced when the leg is raised beyond 30 degrees at the hip joint maintaining the knee in full extension.

The Babinski test assesses for upper motor neuron lesions. It is positive when stroking along the plantar aspect of the foot produces an extensor plantar response.

The following sections will illustrate different fracture types and pathologies based on anatomic location.

Cervical Spine Fractures

1. Upper Cervical Fractures

b. Atlas (C1) Fractures

Often caused by axial loading and hyperextension mechanism injury. Neural deficits are often rare due to the large space available for canal. High suspicion for transverse ligament rupture if lateral mass displacement $\geq$ 6.9 mm on open-mouth odontoid views (Figure 4).

- Review the Landell classification of C1 fractures.

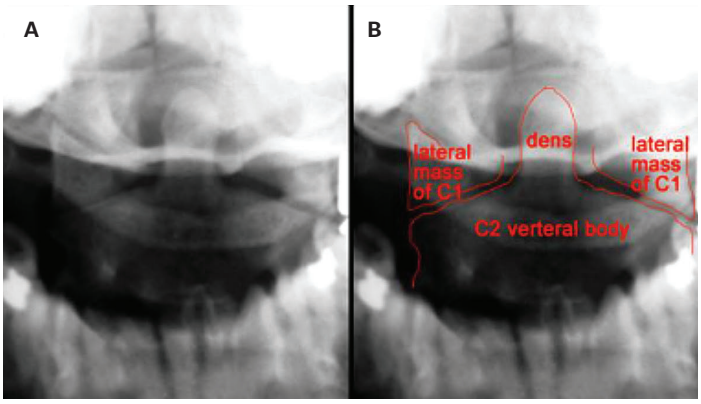

Figure 4. Open-mouth odontoid view x-ray showing dens, C2 vertebral body, and lateral masses of C1. **(A)** Unlabeled; **(B)** Labeled.

Treatment

i. Fractures of the posterior arch or lateral mass fractures can be treated nonoperatively with hard collar or halo application.
ii. Jefferson burst fracture is treated with fusion.

b. C1-C2 Subluxation (Ruptured Transverse Ligament)

An injury Transverse Atlantoaxial Ligament (TAL) is inferred by an increase in the Atlanto-Dens interval (ADI) which is seen on the lateral radiograph. ADI > 3-5 mm is indicated of injury to the transverse ligament, while all ligaments are likely ruptured if ADI > 7 mm, and concern for cord compression with ADI > 10 mm.

Treatment

i. ADI 3-5 mm: halo (check flexion extension views after removing halo at 3 weeks)
ii. ADI > 5 mm: surgery (C1-C2 fusion)

c. Odontoid (C2) Fractures

Odontoid fractures constitute up to 18% of cervical spine injuries, with a bimodal distribution of high-energy trauma in the younger patients and low-energy older population.[1] Consult resident must recognize risk factors for non-union which will affect management of these patients including: > 4 mm displacement, Age (> 40), Type 2 fracture pattern, angulation (> 10 degrees), and posterior displacement.

- Review the Anderson-D'Alonzo classification.

Treatment

i. Type 1 injuries can be treated in a rigid cervical collar.
ii. Type 2 fractures have a higher risk of nonunion due to poor blood supply. Nondisplaced type 2 fractures can be potentially treated in a halo vest. Nondisplaced or minimally displaced type 3 dens fractures can also be treated in a halo vest.
iii. For displaced type 2 and type 3 dens fracture, surgical fusion versus odontoid screw fixation should be considered.

d. Traumatic Spondylolisthesis of Axis (Hangman's Fracture)

Due to the unique anatomy of the C2 as a transitional vertebra between upper and lower cervical spine, the pars interarticularis of C2 is subjected to disproportionally high stress that can lead to fracture.

- Review the Effendi classification.

Treatment

Based on the type of fracture according to the Effendi classification

i. Type I: collar
ii. Type II: halo-vest for 12 weeks.
iii. Type IIA: halo-vest for 12 weeks.
iv. Type III: surgery with C2–C3 fusion (possibly posterior C1–C3 fusion)

2. Subaxial Cervical Fractures

This constitutes the majority of cervical spine trauma injuries occur in the subaxial spine, likely due to the increase motion at these segments (most of which occurring at C5-C7). Several classification systems have been described including Allen and Ferguson Classification and the Subaxial Cervical Injury Classification (SLIC). The SLIC classification has attempted to standardize the spine trauma description and management, which is focused on three injury criteria: (1) injury morphology, (2) integrity of the disco ligamentous complex (DLC); both anterior and posterior structures, and (4) neurologic status.[2] Ultimately, variation in interobserver reliability persuaded AO spine to produce the validated "AO Spine subaxial cervical spine injury classification system" which is based on four criteria: (1) morphology of the injury, (2) facet injury, (3) neurologic status, and (4) case-specific modifiers.

Important imaging to obtain in the initial evaluation should include:

- 3-view C-spine XR (AP, lateral, open mouth) +/− swimmers view
- Flex-ex views often not typically ordered in the acute trauma situations and replaced by CT imaging.
- Evaluate for noncontiguous spine injuries (must be ruled out as it is present in up to 30% of patients).
- CT C spine
- CTA if concern for vertebral artery injury.[3]
- MR C-spine
- Must obtain in patients with neuro deficits

a. Facet fractures and dislocations

Stability depends on several factors including if unilateral versus bilateral, disco ligamentous integrity, size of the fracture fragments. High suspicion for floating lateral mass. When evaluating for unilateral versus bilateral on XR spondylolisthesis of up to 25% is present in unilateral facet fracture, while bilateral facet fracture dislocation will show > 50% spondylolisthesis. Unilateral facet fractures may be reduced on plain XR's if obtained supine, however, "naked facet sign" can be seen on CT scan axial view. There is a high incidence of herniated disk into the canal (therefore must obtain STAT MRI prior to reduction attempt). Facet dislocations can also occur. A jumped facet occurs when the inferior articular process of the superior vertebra is anteriorly locked in relation to the superior articular process of the inferior vertebra. A perched facet occurs when the inferior process of the superior vertebra appears to sit perched on the ipsilateral superior articular process of the inferior vertebra. These two entities can be unilateral or bilateral with bilateral indicating a higher level of instability.

b. Lateral Mass Fractures

There should be close attention to imaging to rule out the highly unstable injury of floating lateral mass—which is a subset of unilateral lateral mass fracture with fractures of the adjacent pedicle and lamina.

Thoracolumbar spine

Thoracic and lumbar fractures account for 50% of neurological spine trauma.[4] Injuries to this region of the spine are from high energy trauma in the younger population and low energy mechanisms elderly patients with osteoporosis. Although normally grouped together, can be thought of as three distinct regions, namely thoracic spine, thoracolumbar junction, and lumbar spine which each have special considerations.

Thoracic spine: Higher chance of cord injury in this region due to narrow spinal canal and watershed blood flow.[5] Enhanced stability of the spine due to the rib cage.

Thoracolumbar junction: About half of thoracolumbar fractures occur in this region due to high stress while transitioning from stiffer thoracic spine to more mobile lumbar spine.[6] Neural deficits can be upper or lower motor neuron depending on the relation of injury to the conus medullaris.

Lumbar spine: If neural deficits are present, they are unlikely to be complete injuries because the spinal canal is wider in this region and the cauda equina is more resistant to compression than the spinal cord. The spinal cord generally ends at the L1-L2 region.

Fractures about this region can be classified as minor or major injuries according to the Denis classification.

Minor fractures

Include fractures of the transverse process, spinous process, pars, or facet joints for example.

Treatment

Minor fractures in isolation can be treated with a thoracolumbosacral orthosis (TSLO). Care must be taken to ensure these fractures are not part of a constellation of a more unstable fracture pattern, however.

Major injuries

Include compression fractures, burst fractures, flexion/distraction injuries, and fracture dislocations of the spinal cord.

a. Wedge compression fractures

Often occur in elderly, osteoporotic females from a low energy mechanism.[7]

Treatment

Can be treated conservatively with TSLO or with vertebroplasty vs kyphoplasty (if conservative treatment fails) generally on a sub-acute basis for palliative purposes.

b. Stable burst fractures

Occurs due to trauma from a flexion and axial load that leads to compression failure of the anterior and middle columns. AP x rays will show increased distance between the pedicles. Sagittal radiographs will show a disruption of the posterior bodies of the vertebrae. Stable burst fractures are those that do not involve disruption of the posterior ligamentous complex (i.e., supraspinous ligament, interspinous ligament, ligamentum flavum, and facet capsule. MRI of disrupted posterior ligamentous complex (PLC) will show widening of the interspinous distance and separation of facet joints.

Treatment

Supportive in TSLO brace .

c. Unstable burst fractures

Involve compression failure of the anterior and middle columns and tensile failure of the posterior columns. MRI is indicated to evaluate the intervertebral disc damage.[8]

Treatment

Prior research showed that operative indications include $>$ 40% of canal compromise, $>$ 50% of vertebral height loss, and $>$ 50% kyphosis. They can be fixed with hook-rod systems, pedicle screws, or sublaminar wires. New research is showing success in non-operative treatment even with the above parameters. One of the deciding factors for operative versus non-operative management is whether the patient is having any neurological symptoms.

Flexion-Distraction injuries (Chance fractures)

The pattern of injury includes tension failure of the posterior column and distraction injury of the anterior/middle column. Can be classified as bony chance fractures or ligamentous chance fractures. These fractures are frequently accompanied by abdominal injuries.

a. Bony chance fractures

Occur when the injury force goes horizontally through bone.

Treatment

Can be treated with extension casting/bracing. Operative indications include kyphosis of $>$ 15 degrees as well as lack of neural deficit

b. Ligamentous chance fractures

Occur when the injury goes horizontally through the ligaments.

MRI is helpful to assess extent of ligamentous damage.

Treatment

Operative fixation

Translational injuries

Occur after an external force causes facet subluxation/dislocation either anteriorly or posteriorly. This is frequently accompanied by a rotational component as well. These are routinely accompanied with a dural tear.

Treatment

Operative with reduction and pedicle screw fixation with instrumentation spanning two levels above and below the site of injury.

Distraction of extension injury

This is a rare injury that is most common in stiff spines such as ankylosing spondylitis. Caused by an extension force to the back with resulting disruption of the anterior longitudinal ligament. Posterior complex may be involved with severe force.

Treatment

Operative if unstable fracture.[9]

Special considerations

Cauda Equina syndrome

Occurs due to compression of lumbar/sacral nerve roots. Only involves lower motor neurons. Can be acute or insidious in nature. Can be caused compression from fractures, disc herniations, etc. Has a constellation of symptoms included bowel/bladder dysfunction, saddle anesthesia, lower extremity sensory/motor deficits.

Needs a full neuro exam included rectal exam (assessing for sensation/tone/voluntary contraction). Be sure to discriminate between pinprick and light touch on exam. Red Flags: progressive back/leg pain refractory to analgesia, lower extremity weakness, urinary retention/incontinence, stool incontinence, recent paraspinal invasive procedures, and use of anticoagulants. MRI is useful tool for evaluation.

Treatment

Operative decompression within 48 hours of presentation. Fusion may be indicated depending on the type of injury

Ankylosing Spondylitis

This condition is an autoimmune spondyloarthropathy that creates a stiff spine due to joint ankylosis secondary to inflammation/bony erosion. Can be associated with other systemic manifestation.

This pathology and associated fractures are at times difficult to recognize. The consult resident needs to be sure to maintain the posture of the patient, obtain imaging of the entire spine, and limit amount of travel within the hospital as this can exacerbate the injury.

Plain films reveal a 'bamboo' spine. CT images will show bony changes but no evidence of active inflammation. CT has a higher sensitivity to aid in the diagnosis of cervical fractures in the setting of ankylosing spondylitis (AS).

Treatment

Can include non-op management/therapy for pain. All spinal fractures in a patient with AS are treated more aggressively due to the high risk for instability. Some operative indications include worsening neuro deficit, epidural hematoma, and unstable fractures of the spine. Even unimpressive appearing fractures in a neurologically intact AS patient may be stabilized operatively due to the high instability risk.

Infection

Important to have a high index of suspicion for infections of the spine as presentation may not be straightforward leading to a delay in diagnosis. 80% will present with back pain. Risk factors for spinal infections include malnutrition, diabetes, iv drug abuse, immunosuppression, bacteremia/sepsis, and recent invasive procedure.

The consult resident should ensure that labs/biopsy should be obtained before the administration of IV antibiotics if the patient is hemodynamically stable. Routine labs should include CBC w/ diff, CRP, ESR. Open biopsy is associated with the most accuracy followed by CT guided biopsy. Blood cultures are only positive in 20% of patients. MRI with gadolinium is the gold standard to assess for spinal infections. Soft tissues are difficult to see with CT scans and X-rays may show no evidence of change for several weeks.

a. Discitis

Involves infection of the intervertebral discs with S. aureus as the most isolated organism. X rays can reveal a loss of lumbar lordosis and disc space narrowing. MRI is the most sensitive for diagnosis.

Treatment

Antibiotics and serial inflammatory marker trend to note improvement. Operative indications include lack of response to antibiotics, progressive neurologic deficits, and late infection.

b. Vertebral osteomyelitis.

Most cases occur in the lumbar spine with S. aureus as the most common microorganism isolated. Fever is only present in 1/3 of patients. Symptoms of pain are insidious. X rays may show disc space destruction with concomitant disc space narrowing. MRI with contrast is the test of choice.

Treatment

Prolonged antibiotics versus operative neurological decompression and stabilization, irrigation and debridement. Operation needed for progressive neurological deficits, cervical vertebral osteomyelitis, and gross instability of the spine.

c. Epidural abscess

Involves an infection in superficial to the dura mater. Usually occurs secondary to hematogenous spread or from adjacent discitis with S. aureus as the most common culprit.[10] Systemic symptoms and fever are usually present. MRI of the entire spine is the best imaging modality for this condition and will show a ring enhancing lesion around the abscess.

Treatment

Surgical decompression +/- instrumentation, long term IV antibiotics.

Tumor

Tumors of the spine can occur in any age group. The overwhelming majority of spinal tumors are metastatic in a 9:1 ratio. Red flags for spinal tumors include night pain, a history of malignancy, as well as pathologic fracture after minor trauma.[11] Imaging of the concerned area should be obtained with Xray/CT with contrast/MRI with contrast. Primary screening consists of CT chest abdomen, and pelvis. ESR and CRP should be obtained as well.

Treatment

Depends on type of cancer, presence of metastasis, and the patient's overall condition/life expectancy. Surgical resection can be performed for an isolated lesion such as osteoid osteoma refractory to conservative management. For metastatic lesions, the tumor burden is primarily controlled with radiation and or chemotherapy.

The Obtunded patient

The obtunded patient can present with difficulty for proper evaluation for a consult resident. Not only are they unable to provide a reliable history, but they are unable to cooperate in a physical exam, therefore it is difficult to assess for neurologic injury leading to a delayed diagnosis.[12] One aspect of the physical exam can still be tested to evaluate for potential spinal shock, namely the bulbocavernosus reflex. This is elicited in males by squeezing the glans penis and assess for anal sphincter contraction. In females, this is assessed by manually squeezing the clitoris (or tugging on an indwelling foley catheter) and assessing for anal sphincter contraction. This patient population is also problematic because occult spinal injuries can have devastating neurological consequences at a higher percentage compared to injuries determined on initial evaluation.[13] Cervical collars should be kept in place until injury can be ruled out with a high-quality CT scan. If no injuries are detected, the C collar can be removed in order to prevent complications with prolonged use (such as pressure ulcers, aspiration pneumonia, venous thromboembolism, etc.[14]

Incomplete Spinal cord injuries

Incomplete cord injuries can also be present following a trauma.[15]

1. Central Cord Syndrome affects the motor, pain, and position sense. It affects the upper limbs more than the lower limbs.
2. Anterior Cord Syndrome involves bilateral loss of motor function and pain sensation below the injured segment. Position sense is preserved.
3. Posterior Cord Syndrome involves loss of position sense below the lesion level. Motor and pain sensation are preserved.
4. Brown Sequard Syndrome affects one side of the spinal cord. It causes loss of motor function and position sense on the same side as well as pain sensation on the opposite side.

Discussion

Spine trauma call can be a daunting proposition for the junior orthopedic surgery consult resident. Between

obtaining a detailed history, physical exam, review of the imaging, and formulation of an accurate assessment and plan amongst the pressure of time constraints and other pending consults, there are a number of things that the consult resident has to perform in order to ensure adequate care of the spine trauma patient. With a thorough knowledge of the anatomy and pathology of the spine, this will equip the consult resident with providing quality care for the spine trauma patient.

Conclusion

The spine trauma patient offers a myriad of learning opportunities for the junior orthopedic surgery consult resident. Once a proper understanding of spine anatomy and pathology is obtained, the consult resident can not only learn from their call experiences, but also positively impact the lives of patients in their time of need.

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Sports

U·P·O·J

Sports Tips & Tricks: Arthroscopic Repair of Massive Rotator Cuff Tear

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The treatment of massive, retracted rotator cuff tears presents a challenge to the shoulder surgeon. However, when certain principles are adhered to, most large tears can be restored to at least near anatomic position. The preponderance of tears possess at least one mobile limb and require a side to side reduction. In fact the width of the supraspinatus insertion has been shown to be only 1.3mm.[1] indicating that tears retract posteriorly or anteriorly. The infraspinatus tendon to inserts anteriorly on the greater tuberosity as it blends with the supraspinatus footprint. (Figure 1) and will predictably retract posteriorly in time. Tears with an exposed lateral footprint greater than 1.3mm require some mobilization to allow excursion infraspinatus tissue anteriorly, or coracohumeral ligament tissue posteriorly in order to effect a tear reduction. So called 'crescent' (Figure 2) tears that are repaired directly laterally with double row constructs are predictably repaired under excessive tension and are more likely to fail.(Figure 3) Tension is the 'bane' of rotator cuff repair[11], and it is the senior author's contention that in an effort to obtain a 'double row' construct, the art of tear reduction has been abandoned.

Clearly a double row construct affords greater footprint compression. However biomechanical studies comparing double row to single row are inherently flawed since that are performed in cadaveric specimens where acute tears are created surgically.[23] Clinically, the preponderance of rotator cuff tears are chronic and retract in line of muscle pull.

The repair of massive rotator cuff tears have yielded unpredictable results with a high reported rate of re-tear noted,[7] highlighting the importance of honoring the biology of tendon healing. This paper will delineate an approach for arthroscopic repair of massive rotator cuff tears emphasizing the importance of tear reduction and minimizing tension. We would also like to introduce the term 'oblique reduction' to more precisely characterize surgical restoration of anatomy.

Reducing the Tear

Anatomic reduction of the torn cuff to the greater tuberosity should be the surgeon's chief goal in order restore to restore cuff function and minimize rotator strain at the repair site. Two principle factors that may affect the surgeon's ability to achieve anatomic reduction include the amount of

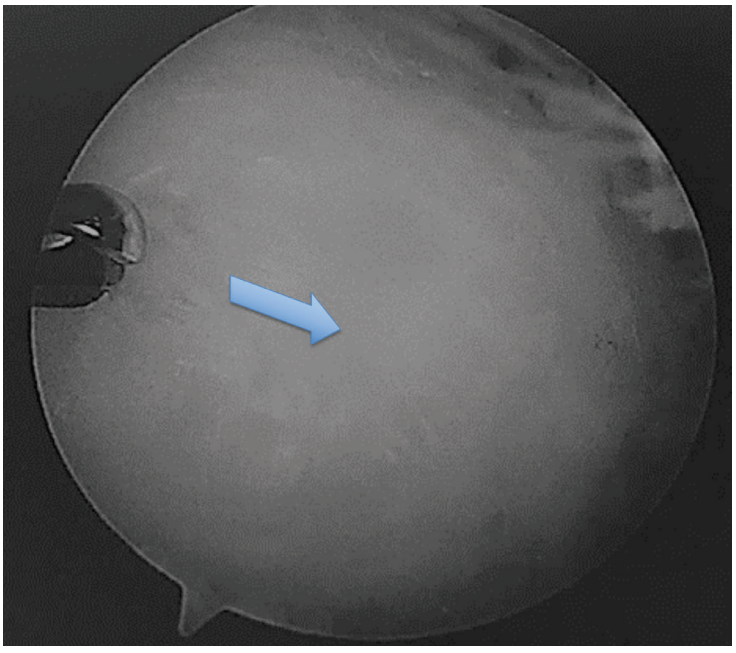

Figure 1. Infraspinatus fibers inserting anteriorly on greater tuberosity.

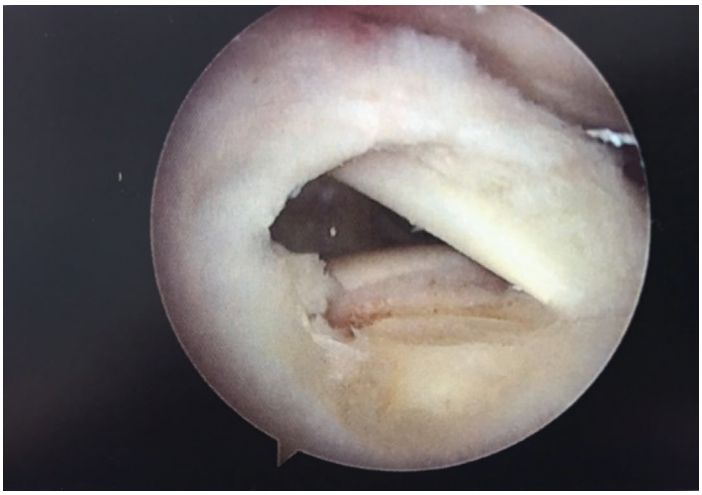

Figure 2. "Crescent tear."

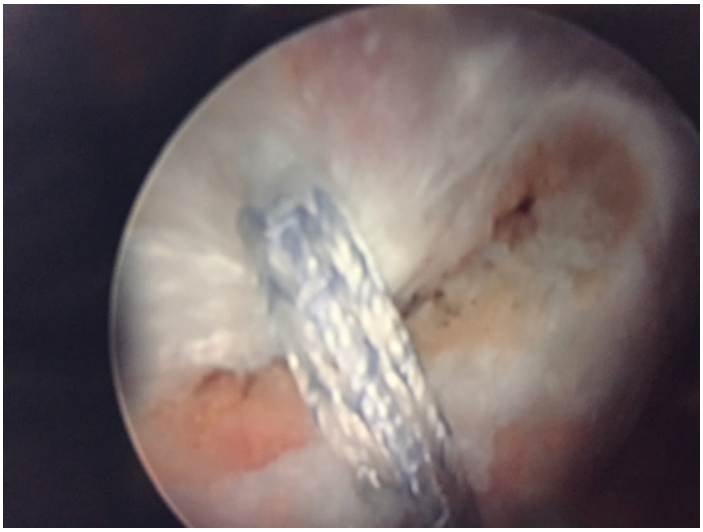

Figure 3. 'Oblique Reduction' of mobile posterior limb.

tendon retraction or scarring, and tissue quality. However, once the direction of cuff displacement is recognized, it is the senior author's experience that at least a near complete tear reduction is usually possible.

The location of the 'mobile limb' in the retracted tear will dictate the strategy of side to side suturing. When grasping the edges of the tendon with an arthroscopic grasper and pulling the posterior limb anteriorly and the anterior limb posteriorly a more mobile limb will be appreciated. Tendon excursion can be apprised from various portal locations, but is usually best visulaized while viewing from the '50 yard line' view—a portal directed lateral to the tear apex. Cuff mobilization can be improved with debridement of scar and bursal tissue, as well as extra- and intra-capsular releases of adhesions. An anterior interval slide can be performed (technique discussed below) which not only frees the subscapularis (often torn) but also releases the coracohumeral ligament (CHL). The senior author does not recommend posterior interval slides as this may introduce excessive tissue trauma to already compromised tissue.[3] Furthermore, as stated previously, the infraspinatus insertion on the tuberosity curves anterolaterally[1], and a straight posterior interval slide conceivably may violate the native infrapsinatus tendon.

It is paramount to recognize that subscapularis tears are far more common than generally realized.[22] With subscapularis repair and advancement, the adjacent 'comma tissue' is lateralized, allowing tissue for suitable fixation for the posterior cuff. Thus, subscapularis repair lessens cuff defect size as well as providing tissue to be approximated to the posterior cuff (oblique reduction).

The term "margin convergence" was introduced by Burkhart et al. to describe the side-to-side repair of massive, longitudinal-type tears, converting them into smaller 'crescent-shaped' tears.[2,3] This technique effectively shifts adjacent tissue laterally to decrease the medial to lateral dimension of the tear. Using margin convergence, the free edge of the tear "converges" toward the rotator cuff footprint, allowing for decreased strain and tension. [3] Burkhart et al illustrated the power of this technique when he compared those who underwent direct tendon to bone repair for smaller crescent-shaped tears versus patients with larger U-shaped tears treated with margin convergence, and found no significant difference at 3.5 years post operatively.[4] Additionally these authors used elegant biomechanical principles to illustrate how margin convergence confers significant strain reduction at tear edges.[5] By virtue of the rotator cuff footprint[1] and the anterolateral insertion of the infrapsinatus, most side to side suturing will involve an apical suture posteriorly with anterior tissue bites usually taken more laterally ('L shaped' tear) in an effort to execute the 'oblique reduction' (Figure 4).

Although the goal is to achieve anatomical reduction of the rotator cuff, medialization of the repair may be necessary since excessive tension is to be avoided (Fig 5). Several biomechanical and anatomic studies combined with clinical data have asserted that insertion medialization less than 10 millimeters yields favorable outcomes without

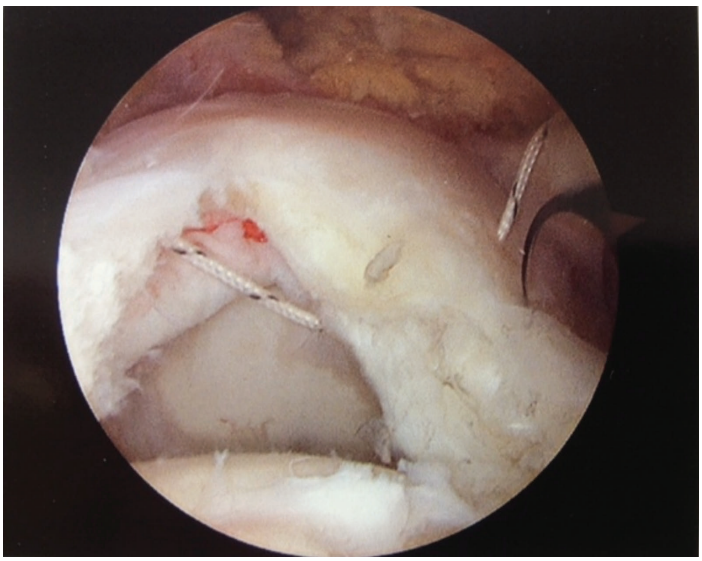

Figure 4. Oblique suture pattern for Margin Convergence.

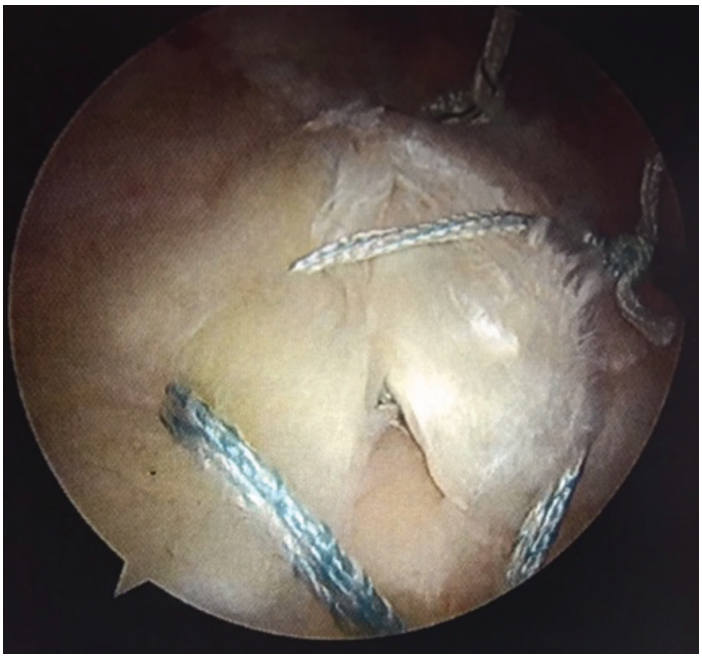

Figure 5. Medialized repair.

appreciable effects on range of motion strength[6,8].

Tension

Cuff tears repaired under tension have been shown in animal studies to be at higher risk of failure with a decrease in material properties within the repaired tissues. [10] Additionally, patients who underwent rotator cuff repair under increased tension were noted to have decreased perceived improvement post operatively, decreased strength measures, and increased post-operative pain.[11] More recently the description of 'type 2' failure (Figure 6) a result of excessive medial tension in double row constructs, reinforces the perils of tension on biology.

As mentioned above, margin convergence (oblique reduction) is a technique that approximates tendon edges and creates a relatively tension free repair. Using a cadaveric model, Burkhart et al.[5] described the improvement in tear gap size and strain with margin convergence with singular successive side-to-side suture placement. The index suture decreased the gap size by 50%, followed by 60% with the second suture, 67% with the third suture, and 75% with the fourth and final suture. In addition, there was an average reduction in the strain in both the subscapularis and infraspinatus tendons of 58%[5]

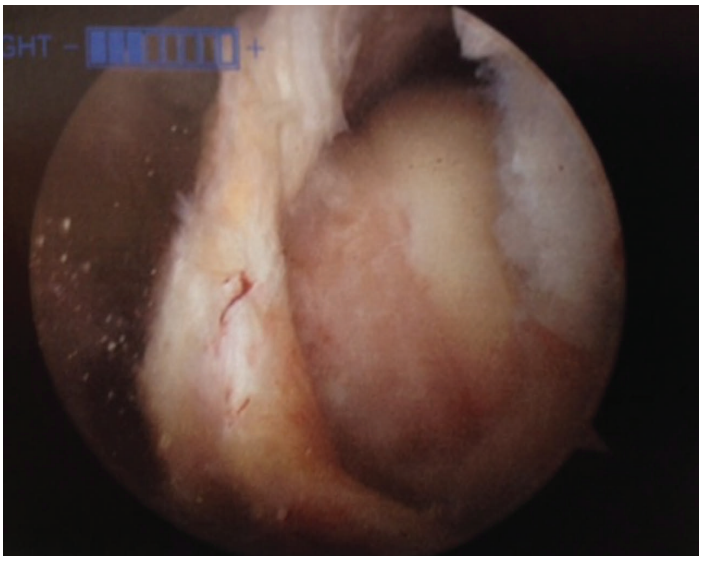

Figure 6. 'Type 2" (medial) failure.

Technique

Arthroscopic repair of the massive rotator cuff tear demands appropriate pre-operative planning, positioning of the patient, optimal portal placement, and thorough arthroscopic evaluation of the glenohumeral joint and subacromial space. Once the cuff tear has been defined and the tissue appropriately debrided, excursion of the cuff tendon is assessed using an arthroscopic grasper. As stated above, the tendon is grasped on either side of the apex to assess which limb is mobile (usually posterior). Oblique reduction suturing can then be planned in order to effect reduction.

It is critical to release all bursal adhesions medially to the scapular spine in an effort to increase excursion of the infraspinatus. The senior author maintains that an inferior capsular release is an essential element of reducing humeral head elevation. (Figure 12) since most chronic tears are associated with anterior superior humeral head excursion. In addition, a thorough CHL release will similarly allow the humeral head to descend inferiorly and facilitate humeral head coverage. An electrothermal device is delivered down to the base of the coracoid process while the lateral soft tissues linking the supraspinatus and infraspinatus are left intact. (Figure 13) An important structure, termed 'comma sign' by Burkhart, must be preserved as it houses a connection of the subscapularis to the supraspinatus. In fact, some investigators posit that the 'comma tissue', once thought to consist of the superior glenohumeral ligament and coracohumeral ligament, may actually contain anterior fibers of the supraspinatus.[3] Often the senior author finds that the placement of traction sutures in both anterior and posterior limbs facilitates reduction. (Figure 7)

Again, oblique reduction suturing proceeds medially to laterally with an apical suture placed into the mobile (usually posterior) limb with the subsequent bite in the opposing limb directed more laterally in order to effect a tear reduction. The absence of a 'dog ear' indicates that the tear is anatomically reduced. (Figure 8) The surgeon is to continue to place oblique side-to-side sutures until the tendon reduction tension becomes noticeable. After oblique reduction suturing is completed, the lateral free margin can be repaired to the prepared bony footprint on the humerus with decreased tension using a variety of techniques. The senior author prefers to incorporate tape type suture to secure tendon edges to bone. The

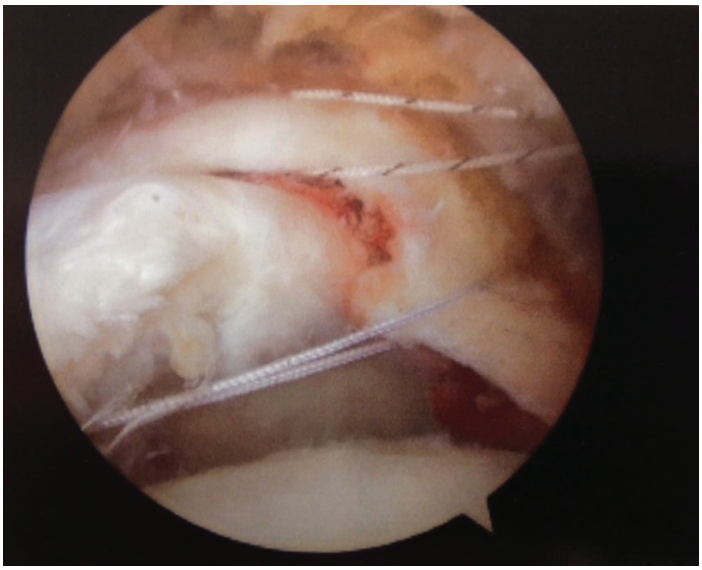

Figure 7. Traction sutures placed anteriorly and posteriorly.

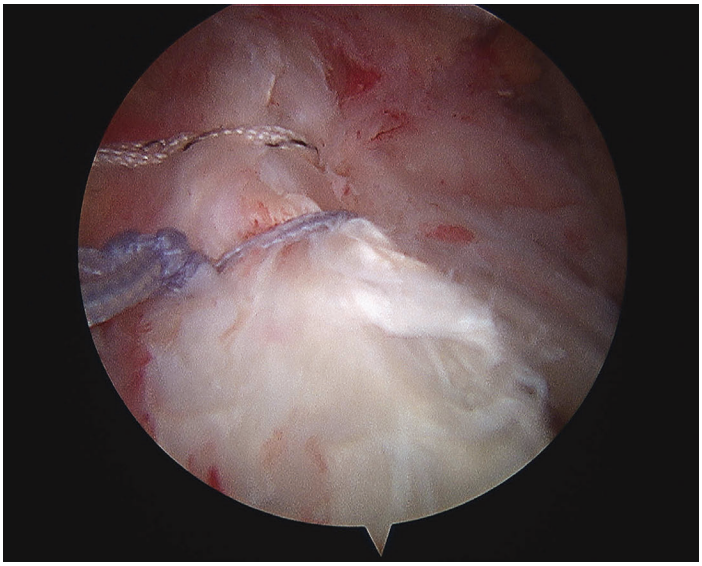

Figure 8. Absence of 'dog ear' in anatomic reduction.

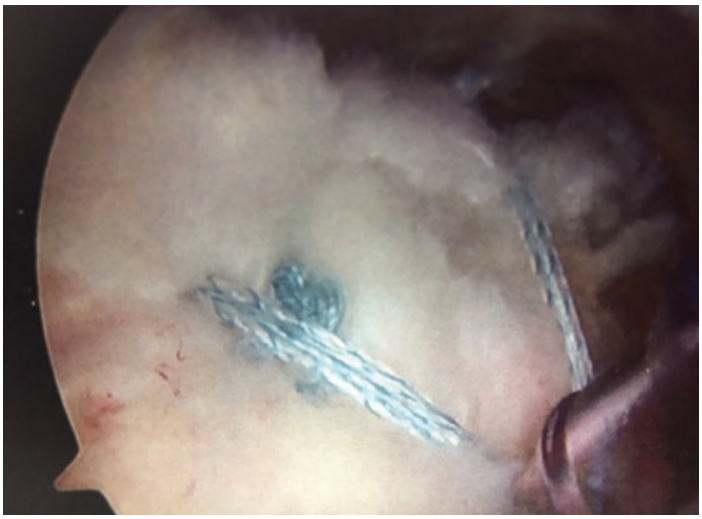

Figure 9. Usage of Tape Suture to reduce tear.

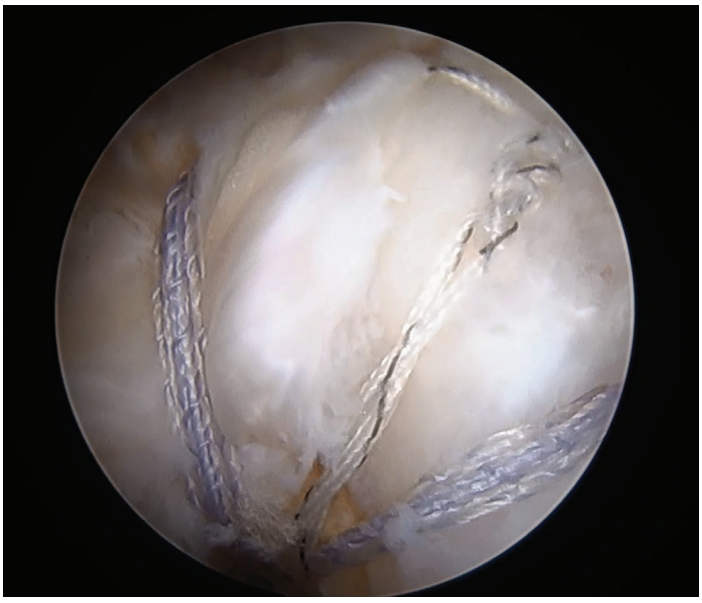

Figure 10. Using margin convergence suture and tape to reduce tear.

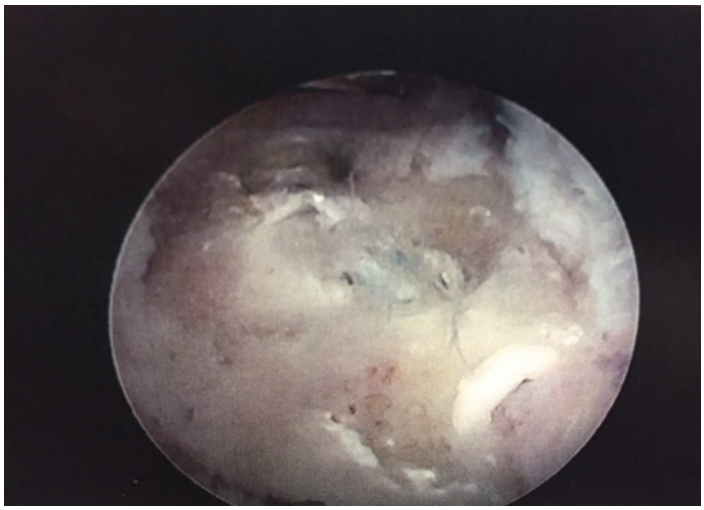

Figure 11. 'Anchor Fest' on tuberosity eliminating bone surface area for healing.

tape suture bites are taken in an effort to continue tear reduction—that is, the tape sutures help to converge the margin to bone when tensioned (Figure 9). Occasionally the senior author will incorporate the free ends of the margin convergence sutures into the lateral anchor in an effort to enhance fixation security. (Figure 10)

Anchors

Suture anchors are costly[6] and when double row fixation is implemented precious 'footprint' is occupied by non-biologic tissue (Figure 11). The senior author regards the footprint area as 'sacred' and should be revered as a repository of receptive, healing bone rather than a 'graveyard' of anchors. In order to enhance biology, the senior author routinely performs a 'tuberoplasty' in an effort to help joint congruence and generate a bleeding

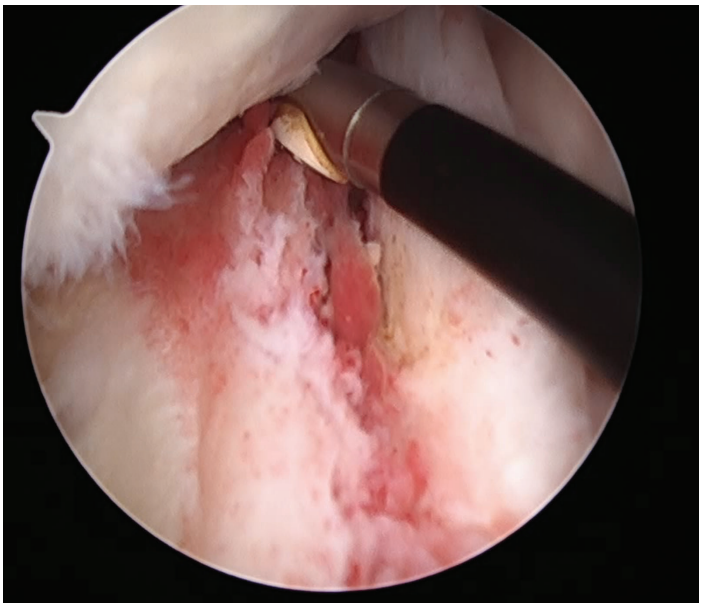

Figure 12. Posterior inferior capsular release.

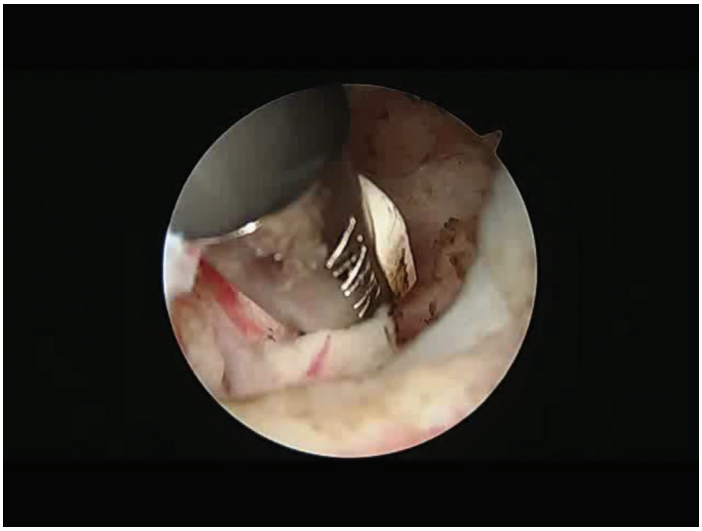

Figure 13. Release of CHL off Coracoid Neck. 70 degree scope may be helpful.

bone bed.[8]

Summary

Oblique reduction is essential to effect anatomic restoration in the preponderance of rotator cuff tears. The very narrow lateral insertion of the supraspinatus dictates that essentially all tears will have an oblique reduction pattern. The impetus of industry to promote more anchor usage and regard most tears as 'crescent' shape leads to improper reduction with non-physiologic tension and excessive costs. Reduction of humeral head elevation and near coverage of a depressed humeral head are tantamount to success. The senior author's experience with these aforementioned techniques has been favorable with attainment of Penn Shoulder score of 84 when full coverage is attained.[24]

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Hand

Hand Tips & Tricks: External Fixation of the Hand

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Introduction

Within orthopaedic surgery, external fixation constructs are used as transitional or final treatment options when stabilizing fractures or correcting deformity. In hand and upper extremity surgery, similar utilization and fixation principles of external fixation apply as in the lower extremity, though they are employed more commonly for highly comminuted fractures with bone loss or periarticular instability. External fixation is often used for hand fractures secondary to gunshot injuries, which result in comminution and severe soft tissue damage due to the energy transferred by the ballistic projectile. Further, external fixation, especially with dynamic properties, has been used for proximal interphalangeal (PIP) joint fracture dislocations and condylar fractures of the proximal or middle phalanx. Here, we review the indications, technique, and outcomes of hand external fixation with an in-depth discussion on management of gunshot-induced hand injuries.

Indications for Hand External Fixation

The principle of external fixation relies on ligamentotaxis, in which bony structures are brought into and maintained in alignment by tensioning the surrounding soft tissues, ligaments, and tendon.[1] One main indication for external fixation is to stabilize open fractures or fractures with infection or segmental bone loss to allow for soft tissue inflammation to abate before definitive fixation and soft tissue coverage. In addition, dorsal fracture dislocations of the PIP joint are especially amenable to external fixation. The goal of external fixation in those injuries is to keep the PIP joint concentrically reduced, which can be achieved using static or dynamic external fixators.[2] Other indications include maintaining length stability in fractures with segmental bone loss and in bony nonunion of the phalanges or metacarpals.[1] When there is concern for development of infection (e.g., contaminated wounds, comminuted fractures due to gunshot injuries), external fixation can be combined with the placement of internal antibiotic delivery devices to reduce infection risk.[3]

Hand fractures caused by gunshot injuries represent a unique class of open fractures in which management controversies abound. The standard of care for all high-energy gunshot wounds or any-velocity gunshot wounds associated with severe soft tissue compromise, unstable fractures, or intra-articular fractures is surgical debridement and fixation, which can include external fixation in highly comminuted fractures or in cases with severe soft tissue compromise (Figure 1). Antibiotic prophylaxis should be administered. Notably, the risk of infection after gunshot wounds is somewhat dependent on the timing between injury and treatment. One study noted that a delay of greater than 6 hours was associated with a higher incidence of infections.[4] In contrast, low-energy gunshot injuries with minimal soft tissue injury and stable/extra-articular fractures may be managed with local wound care. The need for antibiotic prophylaxis in these patients is controversial.[5]

Technique

Here, we describe the technique for hand external fixation using a classic syringe fixator, which is a low-cost and effective method that is universally available.[6] Alternatives to the syringe external fixator exist. In some circumstances, the 1-mL syringe may not provide enough length to span the fracture, pins of larger diameter and in multiple planes may be required to better fixate the fracture, a more rigid construct is desired, or forces other than straight distraction applied to the bone are required that cannot be enacted with a straight, firm syringe. It is in these circumstances that modifications can be made to the original technique. Longer or wider syringes can be used (3 mL, 10 mL, etc.) which allow for longer constructs and the ability to have pins slightly out of plane rather than all parallel. Flexible rubber catheters (e.g., "red rubber" or Foley catheters) can be helpful when the desired plane of fixation is not linear, as they are able to be pulled and

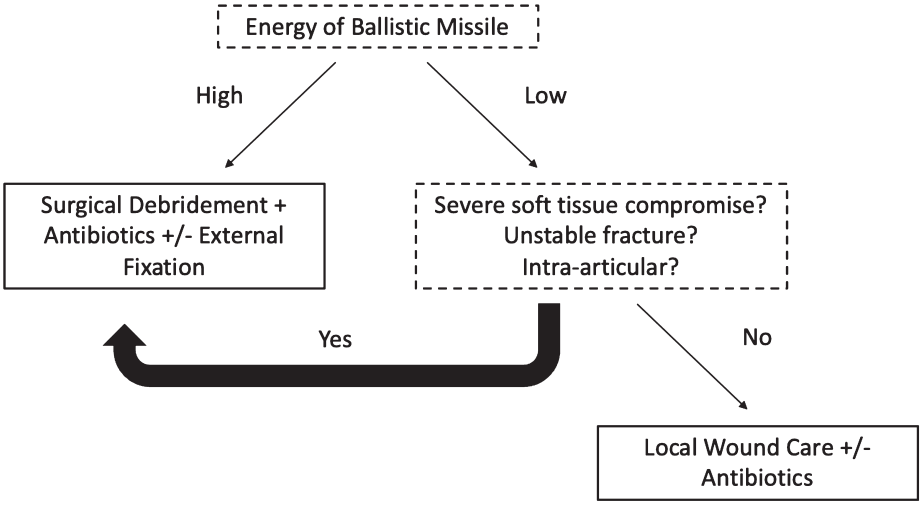

Figure 1. Management of gunshot injuries to the hand.

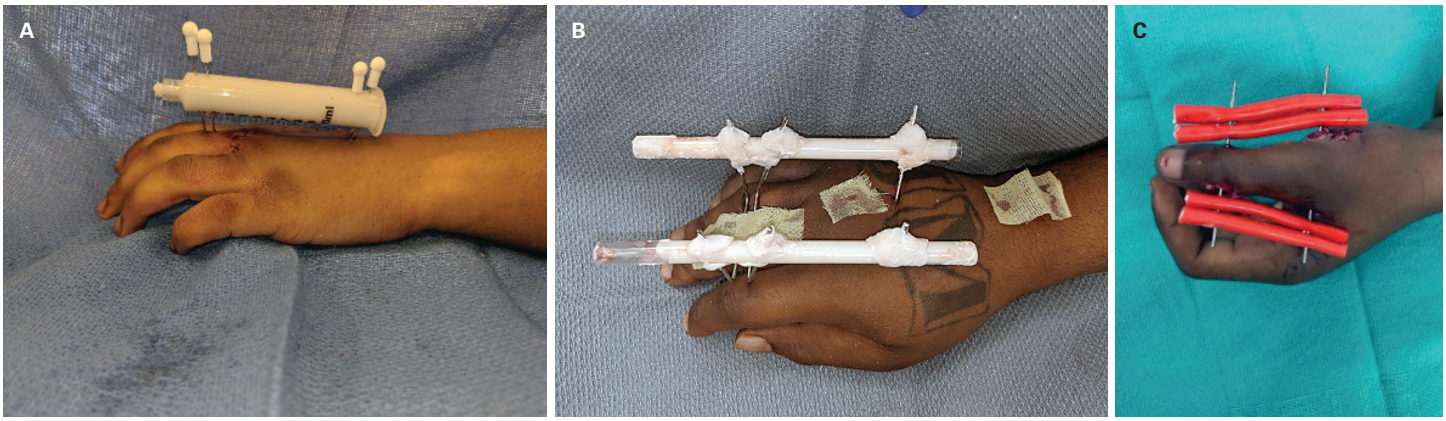

Figure 2. Hand external fixation devices. **(A)** and **(B)** Rigid external fixator devices with cement augmentation; **(C)** Flexible rubber catheter fixator with cement augmentation to provide rigidity. Images courtesy of Andrew D. Sobel, MD.

stretched to accommodate the desired fracture reduction planes. They can also be easily stacked to increase construct rigidity. Whereas cement can be added to syringe fixators as a low-cost means to increase construct rigidity, this is a mandatory step when using rubber catheters as they do not have enough rigidity alone to hold the fracture reduction.

Outcomes After Hand External Fixation

Intra-articular hand fractures have been shown to have worse outcomes including worse grip strength and motion compared to extra-articular fractures after external fixation,[7] which may be more related to articular involvement than choice of fixation. In a study of external fixators used for comminuted intra-articular hand fractures, patients achieved an active range of motion of up to 55 degrees at the metacarpophalangeal (MCP) joint and 80 degrees at the PIP joint after external fixator removal. The authors used both static and dynamic external fixation strategies.[6] Another study on dynamic external fixator use in dorsal fracture subluxations or pilon fractures at the PIP joint found that patients achieved 86 degrees of PIP joint motion at final follow-up.[8] In a series of 15 intra-articular PIP fractures, dynamic external fixation with distraction achieved over 85% restoration of grip strength and resulted in only one case of secondary fracture displacement.[9] Another study of 34 patients with PIP joint fracture dislocations treated with dynamic external fixation with distraction resulted in 88 degrees of PIP joint motion, 60 degrees of distal interphalangeal (DIP) joint motion, and no loss of reduction at final follow-up.[10] Other studies have shown similarly acceptable results.[11]

The incidence of complications following external fixation of the hand are relatively infrequent. A study of K-wire use in the hand and wrist found that major complications occurred in only 3% of pins, which included infection requiring drainage, malunion or nonunion requiring surgical repair, or fracture through the pin track. The incidence of minor complications, which included pin loosening and superficial infections, was 12%.[12] Pin-track infections can occur, which can usually be treated with oral antibiotics alone. The incidence of septic arthritis or osteomyelitis is rare.[13]

Conclusion

Hand external fixation is a safe and effective method for treating highly comminuted hand fractures or hand fractures with severe soft tissue compromise. Inexpensive external fixators can be fashioned using K-wires, syringes, catheters, cement, and other readily available materials. Outcomes after hand external fixation are generally good with few complications.

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Shoulder and Elbow

U·P·O·J

Management Techniques for Posterior Shoulder Dislocations: A Discussion and Case

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Introduction

Posterior shoulder dislocations are an uncommon injury and comprise less than 5% of all shoulder dislocations. Even more rare, are posterior shoulder fracture-dislocations, comprising less than 1% of shoulder fracture-dislocations.[1] Posterior shoulder dislocations usually occur as a result of trauma, seizure, severe muscle contractions following an electric shock.[1,2] Prompt evaluation and treatment of this condition is paramount in order to prevent avascular necrosis of the humeral head.[3] Over 70 years ago, McLaughlin noted the difficulty that can be present in diagnosing a posterior shoulder dislocation—"posterior dislocation of the humerus is a diagnostic trap. Clinical and roentgenographic evidence of this lesion is always present, but usually escapes notice unless deduced."[4] These injuries can be missed or have delayed diagnosis due to non-specificity of pain, or the urgent treatment of an associated condition taking medical priority and distracting from a focused assessment of the entire patient. Furthermore, when assessed with x-ray, AP radiographs of the shoulder often appear normal.[1]

Careful examination is necessary to prevent missed or delayed diagnosis. On inspection, increased prominence of the coracoid, flattening of the anterior shoulder and increased prominence over the posterior shoulder may only be noticeable in direct comparison to the contralateral side.[4] The examiner should look for fixed internal rotation and adduction of the shoulder with limited external rotation and abduction and/or pain with attempted range of motion.[1,4] Radiographically, multiple views of the shoulder must be obtained. While the Emergency Department (ED) will often obtain an AP and Scapular Y view of the shoulder, these films are often unsatisfactory for diagnosis[4], which can be confirmed with Axillary or Velpeau views.[1] Posterior shoulder dislocations can lead to impression defects of the anteromedial humeral head—reverse Hill-Sachs lesions—caused by the humeral head impacting on the glenoid. The size of the impression defect has been defined into categories of less than 20%, 20-40%, and greater than 45-50% of the humeral head articular surface.5 Posterior dislocations have also been considered acute within 6 weeks and chronic if present for more than 6 months.[5]

Treatment options

Closed reduction

Closed reduction is a treatment option indicated for posterior shoulder dislocations with small impression defects. These are typically dislocations diagnosed within the first 6 weeks after injury as delay of diagnosis beyond 6 weeks typically leads to greater progression of the impression defect.[6] Closed reduction may be accomplished by disimpacting and clearing the reverse Hill-Sachs lesion from the posterior glenoid. Traction, adduction, internal rotation, and posterior force on the humerus will disengage the humeral head from the glenoid. Following disimpaction, external rotation of the humerus will relocate the shoulder. Care must be taken to ensure that the humeral head has been completely disengaged from the glenoid as premature humeral external rotation has risk of fracturing the humerus.[6] After reduction, instability is assessed by determining the amount of internal rotation at which the shoulder re-dislocates.[6]

Operative treatment

If the shoulder is not stable through a functional range of motion, a stabilization procedure is indicated. For dislocations with impression defects less than 40-50% of the humeral head articular surface, the McLaughlin or modified McLaughlin procedures have been widely used to achieve stability following reduction.[6] When greater than 40-50% of the humeral articular surface is involved in the reverse Hill-Sachs lesion, arthroplasty is the most commonly used treatment.[6,7]

Case presentation

The patient was, at the time of injury, a 23 year-old right hand dominant male with bilateral posterior shoulder fracture

dislocations after a fall from height from a truck rig (Figure 1). At the time of initial injury, he presented to a local ED who deferred reduction and referred him to an orthopedist in the community. The patient followed up outpatient with the community provider 12 days after initial injury. At that time, the outside provider informed the patient he was unable to care for his condition and referred him to a tertiary shoulder and elbow specialist at our institution. The initial visit at our institution occurred 3 weeks after the index injury. Physical exam revealed no obvious deformity or ecchymosis. There was tenderness to the bilateral shoulders on palpation and throughout passive range of motion. Active range of motion was limited to 10 degrees in each plane. The patient was otherwise neurovascularly intact distally to the bilateral upper extremities. X-rays and CT scan of the bilateral shoulders revealed posterior shoulder dislocation with comminuted reverse Hill-Sachs fractures with humeral heads perched on the posterior glenoid (Figure 1, 2, and 3). The decision was made to pursue operative management for his bilateral posterior shoulder fracture dislocations.

On the left, the patient was treated via a Modified McLaughlin through the rotator interval. On the right, there was a greater degree of bone loss compared to the contralateral extremity, requiring fixation with hemiarthroplasty using complete peeling of the subscapularis (Figure 4).

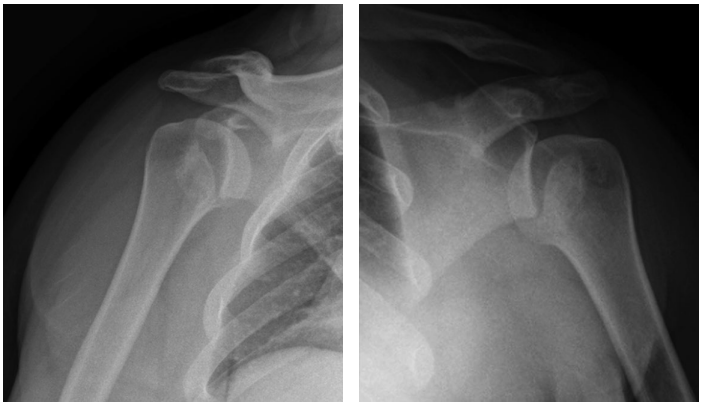

Figure 1. Shoulder x-rays of the right (left) and left (right) sides illustrating posterior shoulder dislocations.

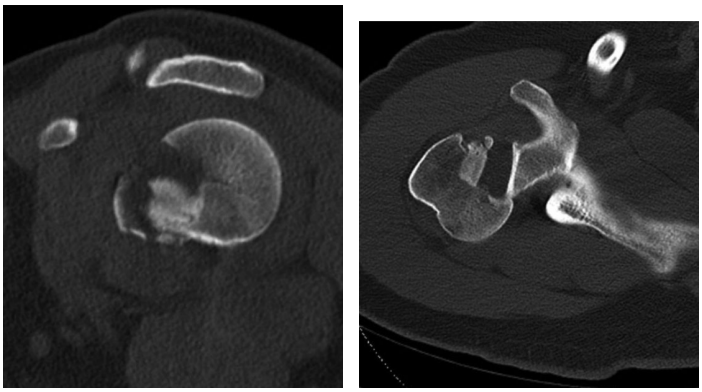

Figure 2. CT scan of the right shoulder illustrating a posterior shoulder dislocation with large comminuted reverse Hill-Sachs fracture with humeral head perched on the posterior glenoid.

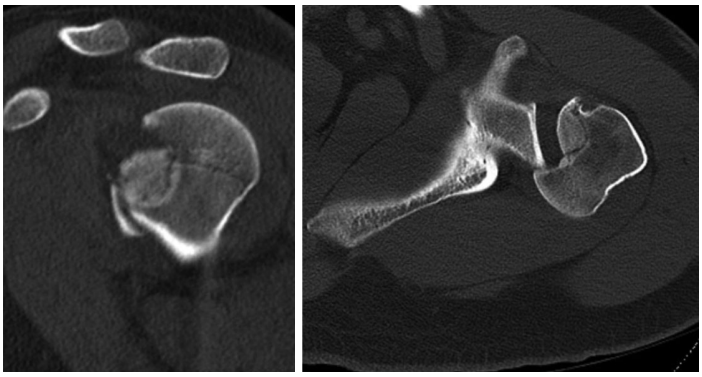

Figure 3. CT scan of the left shoulder illustrating posterior shoulder dislocation with comminuted reverse Hill-Sachs fracture with humeral head perched along the posterior glenoid.

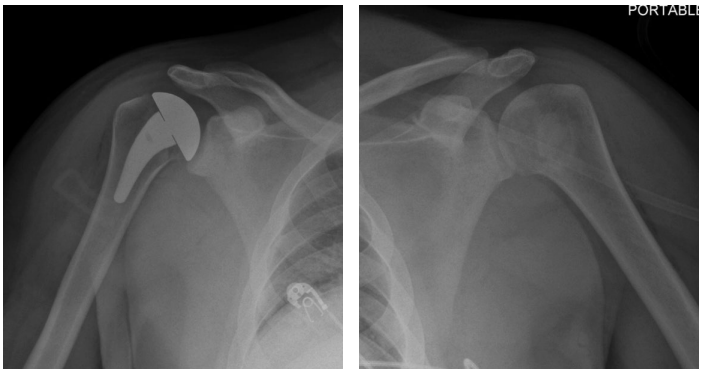

Figure 4. Postoperative shoulder x-rays of the right (left) and left (right) sides after right shoulder hemiarthroplasty and left shoulder Modified McLaughlin procedures.

Procedure

The patient was positioned in the modified beach chair position. The left shoulder and extremity were draped, and the procedure completed in its' entirety prior to prepping and draping of the right shoulder, and completion of reduction and fixation on the right side.

Left

There was an initial attempt at closed reduction with paralysis but the humeral head did not disengage from the posterior glenoid. A standard anterior deltopectoral incision was made. Once the humeral head was palpated, it was reduced using posterior pressure with simultaneous internal rotation to disengage the reverse Hill-Sachs from the posterior glenoid. The arm was placed in external rotation to avoid further posterior subluxation. The reverse Hill-Sachs lesion was palpated and then the rotator interval was located and opened to allow palpation of the defect to localize the apex and leading edge. The Modified McLaughlin was perform using 3mm cottony Dacron tape, placed at the top of the apex of the reverse Hill-Sachs lesion. One arm of the tape was brought around the undersurface of the bicipital groove and out the greater tuberosity cortical bone laterally. The same procedure was repeated with the other limb of Dacron tape to create a horizontal mattress stitch in the superior aspect of the reverse Hill-Sachs lesion. Next, a second 3mm cottony

Dacron tape was placed just inferior to the first tape in the Hill-Sachs lesion defect. It was applied in the same fashion with both ends exiting through the lateral cortical bone transosseous and underneath the bicipital groove. After completion, tension was pulled on the free ends of the tapes to allow subscapularis to sink into the reverse Hill-Sachs defect, thereby creating a reverse remplissage effect. Each of the Dacron tapes were then sequentially tied over the bone bridge in horizontal mattress pattern. After completion, excess suture limbs were cut and the shoulder stability was tested and the patient was noted to have excellent range of motion with only minor loss of external rotation. There was no concern for posterior engagement of the glenoid with the arm brought into internal rotation. The left shoulder was closed and dressed, and held in external rotation.

Right

The right shoulder was positioned, prepped, and draped. An initial closed reduction maneuver was attempted with paralysis but failed. A standard anterior deltopectoral incision was made. The rotator interval was opened and followed to the supraglenoid tubercle. The biceps long head was tenodesed to the superior border of the pectoralis major tendon. An open reduction of the humeral head through the rotator interval was attempted but failed due to posterior glenoid engagement. A subscapularis peel was then performed. With the arm adducted, externally rotated, and flexed, a humeral capsular release was undertaken past the six-o'clock position. The humeral head was then disengaged from the posterior glenoid and dislocated anteriorly. Direct inspection revealed a very large reverse Hill-Sachs lesion with depression of the anterior 40% of the humeral head articulation. A hemiarthroplasty was then performed with Tornier Ascend Flex. After completion, the shoulder was reduced and the patient was found to have 140 degrees of passive forward elevation, 45 degrees of external rotation with the arm at the side, and no posterior dislocation with cross-body adduction. The right shoulder was closed and dressed.

The left shoulder was placed in an external rotation gun sling and the right shoulder was placed in a sling.

Modified McLaughlin

For posterior shoulder dislocation with 20-45% involvement of the articular surface of the humeral head in the impression defect, the subscapularis tendon may be transferred to fill the defect in the humeral head and prevent engagement with the posterior glenoid. To preserve the subscapularis attachment to bone and provide more bony support for defect, the lesser tuberosity may be osteotomized and transferred with the attached subscapularis to fill the defect.[7]

McLaughlin originally developed the McLaughlin procedure for correction of reverse Hill-Sachs deformities after posterior shoulder dislocation by transferring the subscapularis tendon into the defect.[8] The Modified McLaughlin was introduced by Hawkins et al, where the reverse Hill-Sachs defect was filled by transferring the subscapularis tendon and lesser tuberosity.[8]

Shoulder Hemiarthroplasty

Indication

For posterior shoulder dislocation with greater than 45-50% involvement of the articular surface of the humeral head in the impression defect and a preserved glenoid, a hemiarthroplasty should be performed.[7]

Discussion

Posterior shoulder dislocation accounts for less than 5% of all glenohumeral dislocations,[6] Approximately 15% of posterior shoulder dislocations are bilateral,[5] as was the case with the patient presented above. The rarity of the injury, the associated injuries, and often delayed diagnosis contribute to the morbidity of posterior shoulder dislocations.[5] The approach to treatment of posterior shoulder dislocation and instability should take into account the timeline since injury, and most importantly the size of the reverse Hill-Sachs lesion.

The patient above required operative management of bilateral posterior shoulder dislocations and demonstrates how management may differ based on evaluation of patient pathology. The left shoulder was reduced with an opening in the rotator interval and following evaluation of the impression defect, was able to be managed with a modified McLaughlin procedure. For the right shoulder, reduction first required peeling of the subscapularis in order to disengage the reverse Hill-Sachs from the posterior glenoid. Evaluation of the humeral head following reduction found the defect too large to be successfully treated by filling the defect alone, and a hemiarthroplasty was performed.

Conclusion

Posterior shoulder dislocation may be treated with closed reduction, a McLaughlin procedure, modified McLaughlin procedure, or arthroplasty. Selecting the correct treatment requires consideration of the humeral head defect and intraoperative findings. Having multiple options available in the operating room facilitates making the optimal treatment choice for the patient.

Dedication

The authors would like to dedicate this paper to the memory of the patient referenced in this report who passed away in the year following his injury.

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Consumer Interest in Shoulder Replacement Surgery: A Google Trends™ Analysis From 2013 to 2023

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Introduction

Shoulder replacement has grown rapidly over the past decade, owing in part to the rise of reverse shoulder arthroplasty and its expanding indications. The interest of industry in shoulder replacement is well documented, however, patient interest in shoulder replacement has not been investigated.

Methods

Search inquiry data was queried from Google Trends™ to measure consumer interest in shoulder replacement over the period from 2013 to 2023. The primary search terms were "Shoulder Arthritis" and "Shoulder Replacement" OR "Total Shoulder Replacement." Secondary search terms were "Reverse Shoulder Replacement" OR "Reverse Total Shoulder Replacement." Searches by geographic location were also conducted.

Results

Search volume index significantly increased for all search terms during the 10-year interval ($p < 0.001$). Trend analysis of search volume index versus time revealed excellent correlations (R2) for all search terms: "shoulder arthritis" (0.76), "shoulder replacement" or "total shoulder replacement" (0.79), and "reverse shoulder replacement" or "reverse total shoulder replacement" (0.75). When compared with the trend for the search term "shoulder arthritis", an excellent correlation was revealed for "shoulder replacement" or "total shoulder replacement" ($R2 = 0.74$, $p < 0.001$) and a strong-moderate correlation was observed for "reverse shoulder replacement" or "reverse total shoulder replacement" ($R2 = 0.69$, $p < 0.001$).

Over the 10-year period, interest increased the most for "shoulder arthritis" in Connecticut and Alabama, for "shoulder replacement" or "total shoulder replacement" in Mississippi and Montana, and for "reverse shoulder replacement" or "reverse total shoulder replacement" in Utah and Indiana.

Discussion/Conclusion

Our results demonstrate a significant increase in patient interest in shoulder replacements over the past decade and identify geographic hot spots of patient interest. This data will be useful for educating and treating populations interested in shoulder replacements. This is the first study to utilize Google Trends™ to analyze patient interest in shoulder replacement surgery.

Foot and Ankle

Human Placental Allograft: Does it Decrease Postoperative Pain in Tendon Repair and Debridement?

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Introduction

Lower extremity tendon repair and debridement can be associated with post-operative risks. In particular, pain levels may increase postoperatively due to the development of tissue adhesions and fibrosis. This can contribute to extended postoperative rehabilitation and ultimately may result in poor patient outcomes.[1]

Recently, attention has been turned to the use of biologic adjuncts such as human placental allografts to provide additional support to tendon repairs as they have been found to demonstrate anti-inflammatory, anti-microbial, anti-adhesive, and anti-fibrotic properties. These properties have been well-described in the ophthalmology literature, demonstrating a reduction in postoperative inflammation, pain, and adhesion formation.[2]

Use of these allografts has since expanded into other surgical fields including maxillofacial, gynecologic, and now orthopaedic surgery.[3,4] By capitalizing on these properties to minimize the postoperative adverse events of adhesions, fibrosis, and inflammation, the use of placental allograft may contribute to decreased postoperative pain and improved surgical outcomes. However, there is limited data on both short- and long-term patient outcomes to support the regular use of these adjuncts in orthopaedic foot and ankle surgery.

The purpose of this study was to compare postoperative pain scores in patients undergoing achilles or peroneal tendon repair or debridement with adjunctive human placental allograft compared to patients who underwent repair or debridement without allograft.

Methods

All patients who underwent achilles tendon repair, peroneal tendon repair, or peroneal tendon debridement performed by one fellowship trained orthopaedic foot and ankle surgeon at a single institution from January 1, 2022 to February 10, 2023 were included in the study. Patient demographic data including age and sex was collected. Surgical data including CPT code, procedure type (achilles tendon repair, peroneal tendon repair, or peroneal tendon debridement) was collected. Pain scores were obtained at preoperative and two week postoperative clinic visits from nursing assessment notes. These were numerical pain scales based on a 0-10 point scale. Data on preoperative and postoperative pain scores are presented as means.

SAS statistical software was used to perform t-score analyses in order to compare mean pain scores at two weeks postop between patients undergoing repair or debridement with graft versus without graft. Patients were then further stratified by type of tendon repair or debridement (achilles or peroneal) and those who had allograft were compared to those who did not using the same mean and t-score analysis.

Results

A total of 44 patients underwent either achilles tendon repair, peroneal tendon repair, or peroneal tendon debridement between January 1, 2022 and February 10, 2023. Of these patients, 19 had their repair or debridement augmented with allograft, while 25 did not.

Of the patients who underwent allograft, 13 (68.42%) were male, while 6 (31.58%) were female. Of those without allograft, 11 (44%) were male, while 14 (56%) were female. The average age in both groups was 42.16 years old. The mean preop pain score in the allograft patients was 3.31. The mean preop score in the patients without allograft was 4.24. The mean pain score at 2 weeks postop was 2.37 in the allograft patients, compared to 3.00 in the patients without allograft ($p = 0.63$).

Of the 44 total patients, 17 (38.64%) underwent achilles tendon repair. Of these patients, 9 (52.94%) were augmented with allograft, while 8 (47.06%) were not. Of the 9 patients who underwent allograft, 7 (77.78%) were male and 2 (22.22%) were female. Of the 8 patients who did not receive allograft, 6 (75%) were male and 2 (25%) were female. The mean age in the allograft group was 41.56, compared to 43.75 in the group

without allograft. The mean preoperative pain score in the allograft group was 2.44, compared to 3.50 in the group without allograft. Postoperatively at 2 weeks, the allograft group had a statistically significant decrease in pain score to 0.88 compared to 3.13 in the group without allograft ($p < 0.05$).

Of the 44 total patients, 27 (61.36%) underwent peroneal tendon repair or debridement. Of these patients, 10 (37.04%) were augmented with allograft, while 17 (62.96%) were not. Of the 10 patients who underwent allograft, 6 (60%) were male and 4 (40%) were female. Of the 17 patients who did not receive allograft, 5 (29.41%) were male and 12 (70.59%) were female. The mean age in the allograft group was 42.70, compared to 41.41 in the group without allograft. The mean preoperative pain score in the allograft group was 4.10, compared to 4.59 in the group without allograft. At 2 weeks postop, these pain scores decreased to 3.70 in the allograft group and 2.94 in the group without allograft ($p = 0.6$).

Discussion

While there are several surgical techniques described for tendon repair and debridement, data on the use of human placental allograft in orthopedic foot and ankle surgery is lacking. Steginsky et al in 2016 reported that over 25% of these patients have postoperative complications such as pain, while persistent swelling can be seen in almost 20%.[5] Preventing these postoperative complications—perhaps with the use of human placental allograft—allows patients to recover faster and with improved outcomes.

A 2017 retrospective single center study looked at using Stravix placental tissue as an adjunct surgical wrap in the repair of ruptured tendons in five patients with an average age of 31.[6] Their cohort of consisted of 2 patients with ruptured peroneus brevis tendons, 2 patients with ruptured achilles tendons, and 1 patient with a rupture posterior tibial tendon. Their study looked at postoperative pain scores at 1 week, postoperative adverse events through 2 years, and if there was a reduction in pain from preop. They found that 40% of patients reported no pain at 1 week postop. There were no postoperative adverse events such as dehiscence, infection, fluid collection, or drainage. All 5 patients reported a reduction in pain from preop.

Our study demonstrates similar findings of reduction in pain from preop and a decreased postoperative pain score at 2 weeks in patients with allograft compared to those without. While the combined analysis from this study is not yet statistically significant given the sample size, it does demonstrate a trend. Patients with allograft had lower postoperative pain scores compared to those without. Additionally, when stratified by type of tendon repaired, the patients who underwent achilles tendon repair with allograft demonstrated a statistically significant lower postoperative pain score compared to those without allograft.

Surgical Technique

The same surgical technique was used to implement the human placental allograft in both achilles and peroneal repair or debridement in order to minimize scar tissue and

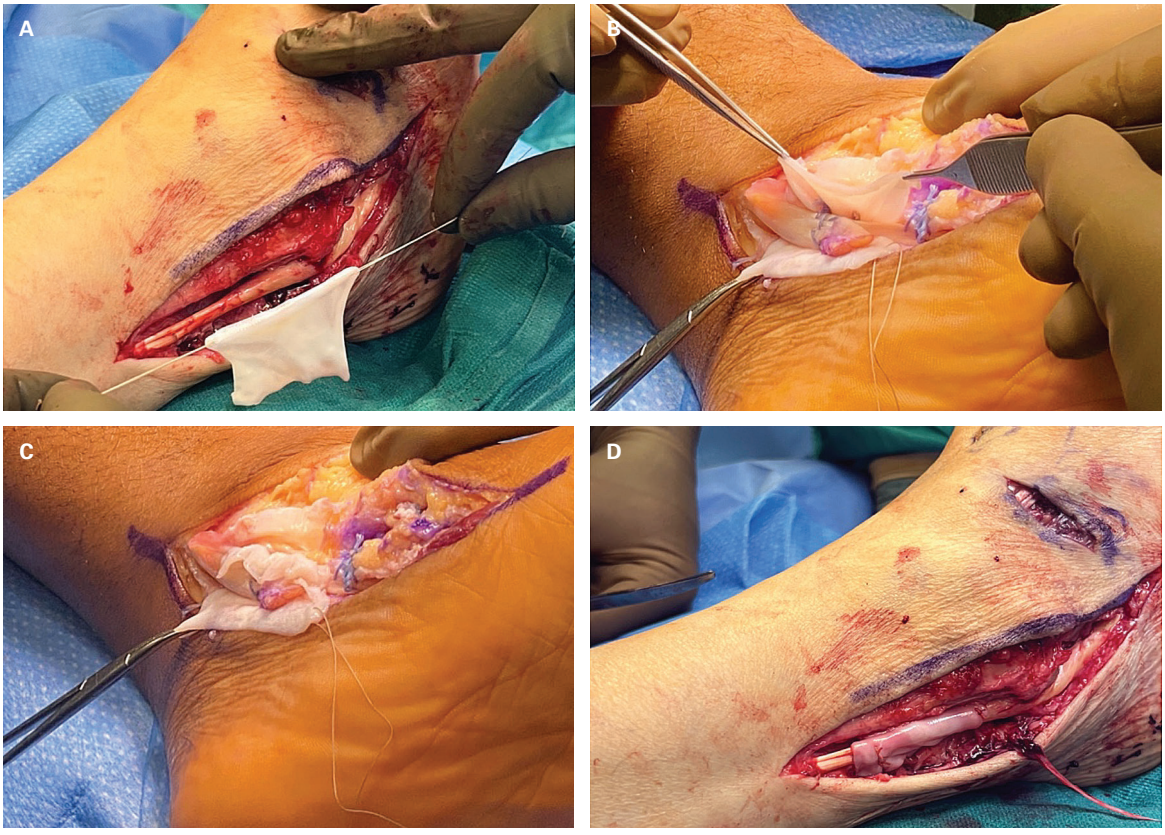

Figure 1. Surgical technique for allograft use in peroneal tendon repair. **(A)** Allograft prepared with 3-0 vicryl suture on two corners; **(B)** Passing allograft beneath peroneal tendon; **(C)** Suturing allograft in place to tendon; **(D)** Completed peroneal tendon repair with allograft wrapped circumferentially

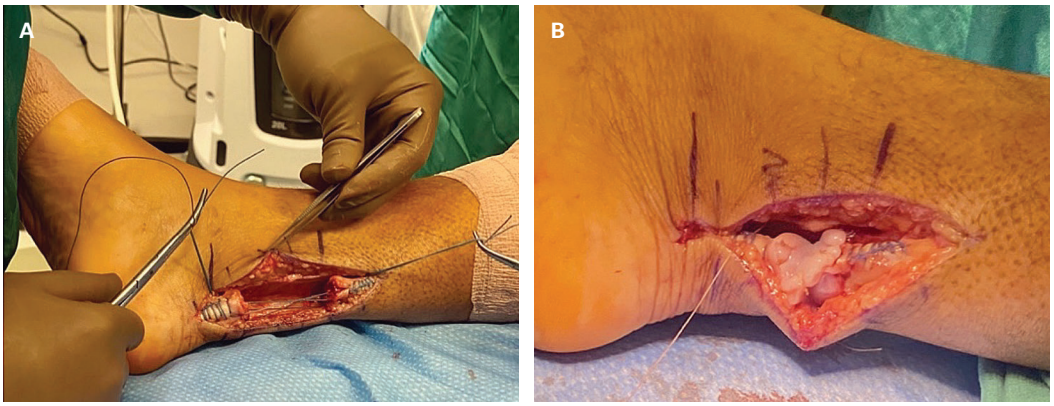

Figure 2. Use of allograft in achilles tendon repair. **(A)** Achilles tendon repair before allograft; **(B)** Achilles repair with allograft wrapped circumferentially around tendon.

Table 1. Complete Data

Characteristic	Graft (n = 19)	No Graft (n = 25)
Tendon		
Achilles	9 (47.37%)	8 (32%)
Peroneal	10 (52.63%)	17 (68%)
Sex		
Male	13 (68.42%)	11 (44%)
Female	6 (31.58%)	14 (56%)
Age (mean)	42.16	42.16
Pain score (mean)		
Preop	3.31	4.24
Postop (2 weeks) p = 0.63	2.37	3.00

Table 2. Stratified Data

	Achilles (n = 17)		Peroneal (n = 27)	
Characteristic	Graft (n = 9)	No Graft (n = 8)	Graft (n = 10)	No Graft (n = 17)
Sex				
Male	7 (77.78%)	6 (75%)	6 (60%)	5 (29.41%)
Female	2 (22.22%)	2 (25%)	4 (40%)	12 (70.59%)
Age (mean)	41.56	43.75	42.70	41.41
Pain score (mean)				
Preop	2.44	3.50	4.10	4.59
Postop (2 weeks)*	0.88	3.13	3.70	2.94
	*p = 0.01		*p = 0.60	

inflammation postoperatively. First, one 3-0 vicryl suture was passed through a proximal corner of the allograft. Then a second 3-0 vicryl suture was passed through a distal corner on the same side of the allograft as the previous suture.This technique allowed for improved control of the graft and the application of gentle tension to make wrapping the desired tendon easier. The allograft was then wrapped circumferentially around the tendon at the site of repair, passing it underneath the tendon while holding onto the proximal and distal vicryl sutures. These sutures were then passed back through the opposite corner to wrap the allograft around the tendon. These were tied sequentially. A third 3-0 vicryl suture was used to anchor the allograft to the tendon to keep it in place. In peroneal debridement or repair, the allograft was wrapped around the more diseased tendon (not both) at the site of maximal adhesion in order to prevent further adhesions with the other peroneal tendon.

Conclusion

Lower extremity tendon repair and debridement can be associated with increased postoperative pain due to the development of tissue adhesions, fibrosis or inflammation. While several surgical subspecialties have adopted the use of human placental allograft for their anti-inflammatory, anti-microbial, anti-adhesive, and anti-fibrotic properties, there is limited data to support the regular use of these in orthopedic foot and ankle surgery.

Data on 44 patients from one orthopedic foot and ankle surgeon at a single institution demonstrate a trend in decreased postoperative pain at two weeks in patients undergoing tendon repair or debridement with adjunctive allograft compared to those without allograft. Future studies are necessary to demonstrate statistical significance in a larger cohort of patients with longer follow up.

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Oncology

U·P·O·J

The Harrington Reconstruction for Patients with Advanced Periacetabular Metastatic Disease

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Introduction

The goal of surgery for metastatic bone disease is to relieve pain and allow patients to return to function as soon as possible. Pathologic fractures that involve the acetabulum present a unique challenge to orthop edic surgeons. Because of insufficient periacetabular bone in cases of extensive tumor destruction, fracture repair is not feasible and conventional total hip replacement is not a viable treatment option for most patients. Reconstructing the pelvis requires creating a stable construct in the context of substantial bone loss; moreover, given the underlying pathology, this must be achieved without relying on biologic healing of the bone.

While various reconstruction techniques may be utilized, the Harrington reconstruction is a well-described surgical option for patients with advanced periacetabular metastatic disease. In 1981, Harrington described a technique whereby the structure of the pelvis is recreated with threaded Steinmann pins that are fixed in the intact, proximal ilium and converge in the destroyed supra-acetabular region. The defect surrounding these pins is then filled with cement and an acetabular liner is embedded. Finally, a femoral stem is placed to complete the total hip arthroplasty, creating mechanical stability and permitting immediate, full weight bearing.

Classification

Periacetabular metastatic destruction is graded according to the Harrington classification. This categorizes periacetabular fractures into subgroups based on their underlying bony defect and, hence, their specific biomechanical deficiency.

Case Report

History

This is a 56-year-old male patient who presented to the emergency department with a pathologic left acetabular fracture in the setting of high grade urothelial carcinoma of the left renal pelvis. The patient was initially diagnosed one year previously, after which he underwent total left nephrectomy and pelvic lymph node dissection. He was treated with adjuvant Gemcitabine and Cisplatin as advised by his outpatient oncologist.

Nine months later, the patient reported insidious onset left hip pain and had a left ischial bone biopsy confirming osseous metastasis. Following this diagnosis, he presented to an outside hospital with intractable left hip pain and inability to bear weight; he also reported numbness and weakness of his left lower extremity and was admitted for ambulatory dysfunction. During admission, an X-ray and CT scan of the abdomen and pelvis demonstrated progressive osseous metastasis to the left acetabulum (Figure1). Orthopaedic surgery advised use of a walker, with weight bearing on his left lower extremity according to pain.

Approximately two weeks after this admission, the patient initiated palliative radiotherapy with radiation oncology (1200/3000 cGy 4/10 fx, 300 cGy/fx). Unfortunately, he sustained a mechanical fall shortly after starting and X-rays taken at the time of injury revealed a comminuted pathologic fracture involving the superolateral aspect of the left acetabulum. Additionally, there was evidence of a smaller lytic lesion of the inferior pubic ramus with a nondisplaced pathologic fracture (Figure 2). The patient

Table 1 Harrington classification of periacetabular destruction[2]

Harrington Classification	Characterization
Class I	Deficient lateral cortices with intact superior and medial wall
Class II	Deficient medial wall
Class III	Deficient lateral cortices and superior wall
Class IV	Isolated lesion that can be resected in an attempted curative procedure

was transferred to our emergency department for further management.

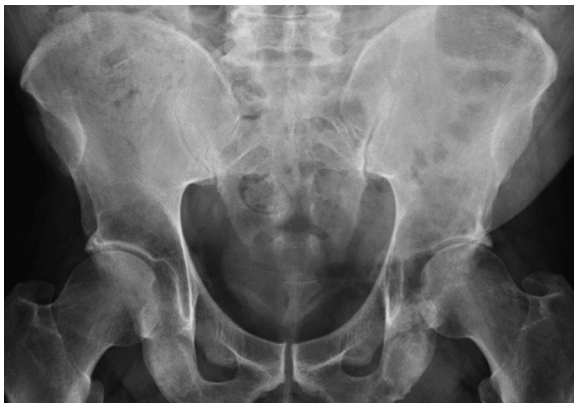

Figure 1. X-ray with lytic osseous metastasis in the weight-bearing region of the left acetabulum.

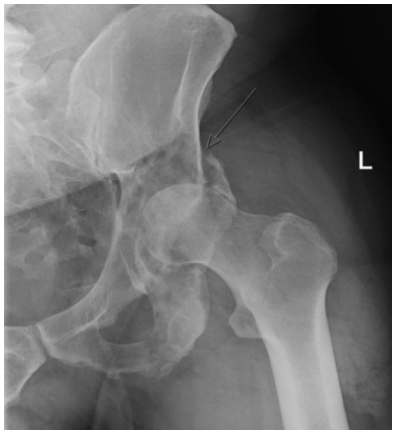

Figure 2. X-ray with comminuted, pathologic fracture of the superolateral left acetabulum. Non-displaced, comminuted fracture of the inferior pubic ramus.

Examination

On exam, the patient complained of severe pain and was intermittently confused due to medications but answered all questions appropriately. The left lower extremity was without gross deformity or skin defects; there was no significant swelling, erythema, or ecchymosis. He was diffusely tender to palpation about the left hemipelvis and had significant pain with log roll of the left lower extremity. Active and passive hip flexion and extension were deferred in the setting of his acute injury. He was otherwise neurovascularly intact.

Imaging

X-rays of the pelvis and left hip and were obtained confirming a pathologic fracture of the left acetabulum. Subsequent CT abdomen and pelvis revealed a pathologic, comminuted fracture of the left acetabulum through an underlying lytic lesion (6cm x 6cm) and further involvement of the surrounding bone, including the anterior wall/column and ischium (Figure 3).

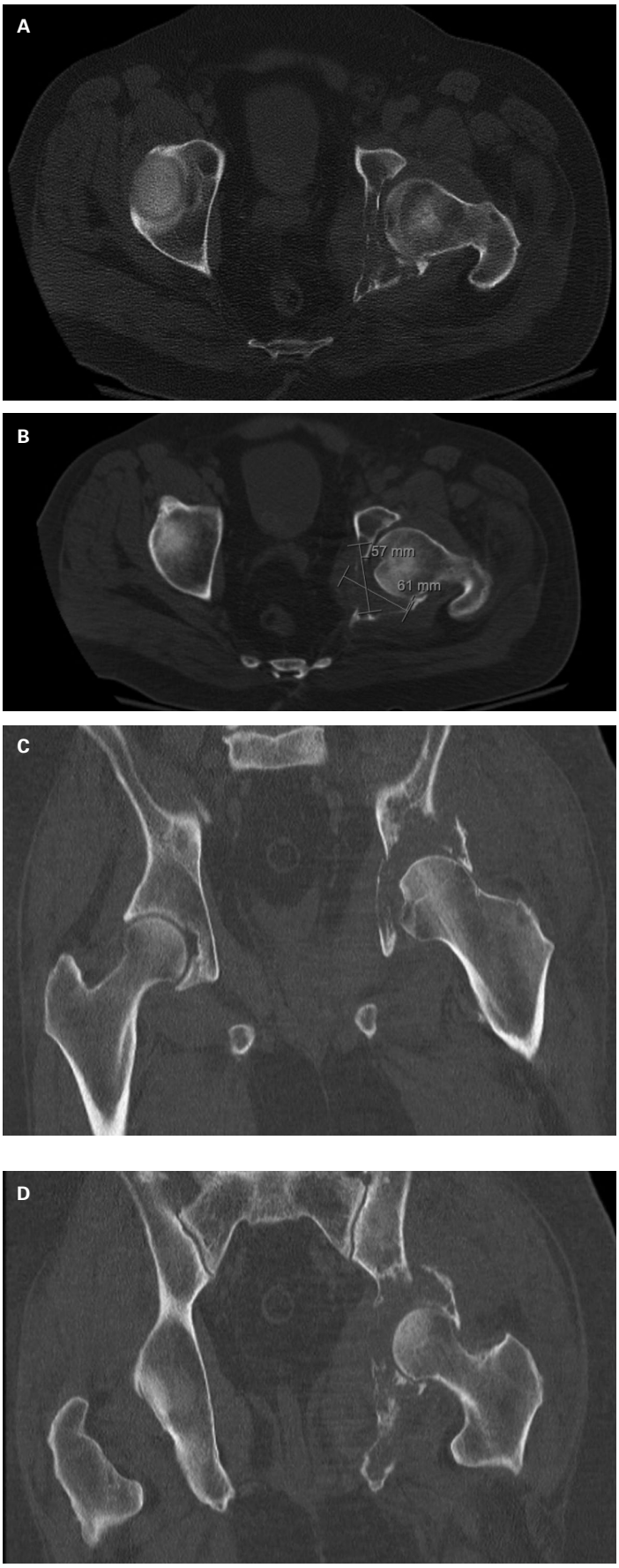

Figure 3. CT pelvis with pathologic fracture of left acetabulum through extensive lytic lesion.

Clinical Management

The patient was admitted to the orthopaedic surgery service for preoperative work-up and surgical management. Anesthesia was consulted for perioperative pain control and cardiology was consulted for pre-procedure risk stratification given the patient's history of inferior right bundle branch block and ascending thoracic aortic aneurysm. Medical co-management and radiation oncology were also consulted for perioperative management. The patient was made touch-down weightbearing but, due to pain, was effectively on bedrest pending operative intervention.

Surgical Management

Indications

The patient's prognosis was discussed with his oncology team and noted to be uncertain. The surgical team therefore offered the patient the options of Girdlestone procedure (femoral head resection to reduce pain by removing pressure from the fractured acetabulum) and Harrington reconstruction. The latter is associated with higher function but also greater risks, which include but are not limited to blood loss, infection, dislocation, and mechanical failure due to progressive disease. After extensive discussion involving the patient and his family, the decision was made to pursue operative management in the form of a left hip tumor curettage and Harrington reconstruction. Indications for the procedure included intractable pain resulting in immobilization and high narcotic requirements in this patient with metastatic disease.

Description of Procedure

The patient was placed in the lateral decubitus position and a standard posterior approach to the hip was performed. The hip could not be dislocated, having migrated into the pelvis; therefore, the neck was cut in situ and the head was fragmented with osteotomes and removed piecemeal. The acetabulum was visualized and found to have extensive lytic destruction. Copious necrotic material and osseous fragments were removed from the acetabulum under direct visualization using rongeurs, curettes, and pituitaries. After debridement to mechanically stable bone, there was no significant medial wall remaining.

While the curettage was being performed, a second incision was made laterally over the iliac crest and dissection was carried down to the abdominal wall muscles, which were released from their insertion, exposing approximately 6cm of the iliac crest. Under fluoroscopic guidance using obturator oblique and anterior-posterior views, four 4mm threaded Steinmann pins were inserted from the crest into the acetabular defect and then driven into the ischium distally. A polyethylene acetabular liner was selected to fit within the defect, recreating appropriate hip center and offset, while allowing for a 40 mm femoral head. The back of the cup was scored using a bur to improve cement fixation. The wound was then very thoroughly irrigated, taking care to remove any remaining loose particles of bone. Three batches of cement were then mixed and injected into the acetabular defect around the pins, and the cup was embedded in the cement. This was allowed to dry completely, while carefully maintaining appropriate inclination and version of the cup.

Attention was then directed to the proximal femur, which was exposed and prepared to accommodate a standard uncemented femoral component, completing the total hip replacement. A trial stem and head were placed and the hip was assessed to ensure equal limb length and stability. Trial components were then exchanged for the true components. The posterior capsule and short external rotators were repaired to the proximal femur through drill holes with Ethibond suture, and the wound was closed in the standard fashion.

Postoperative Course

The patient was made immediately weight bearing as tolerated on his left lower extremity with a walker and posterior hip precautions, similar to a standard total hip replacement. He worked with PT/OT beginning post-operative day 0 and was discharged to an acute rehabilitation facility. He was indicated for post-operative radiation after soft tissue healing.

At his two-week follow-up appointment, the patient reported significant progress with PT at his rehabilitation facility; he ambulated 120 feet with a rolling walker with moderate pain. His exam was notable for 5/5 painless strength throughout his left lower extremity without tenderness or crepitus to palpation. His lower abdominal and left hip incisions demonstrated routine healing without signs of wound compromise. X-rays showed no acute pathology or evidence of hardware migration (Figure 4).

Unfortunately, the patient died from cancer-related complications approximately one month following his left hip reconstruction; however, with constantly improving medical treatment options for cancer, patients often live and benefit from the hip for several years.

Discussion

The Harrington reconstruction is a well-described surgical technique for managing advanced periacetabular metastatic destruction. For most patients, this is an effective and enduring surgery permitting early mobilization and pain relief . Several techniques can be helpful to minimize operative time, blood loss, and complications such as mechanical failure.

- IR-guided embolization 24-48 hours prior to the procedure can be performed to improve hemostasis during curettage of the large, uncontained and often hypervascular metastatic lesion. In addition to reducing intra-operative blood loss, the hemostasis improves cement fixation to the remaining bone and pins.

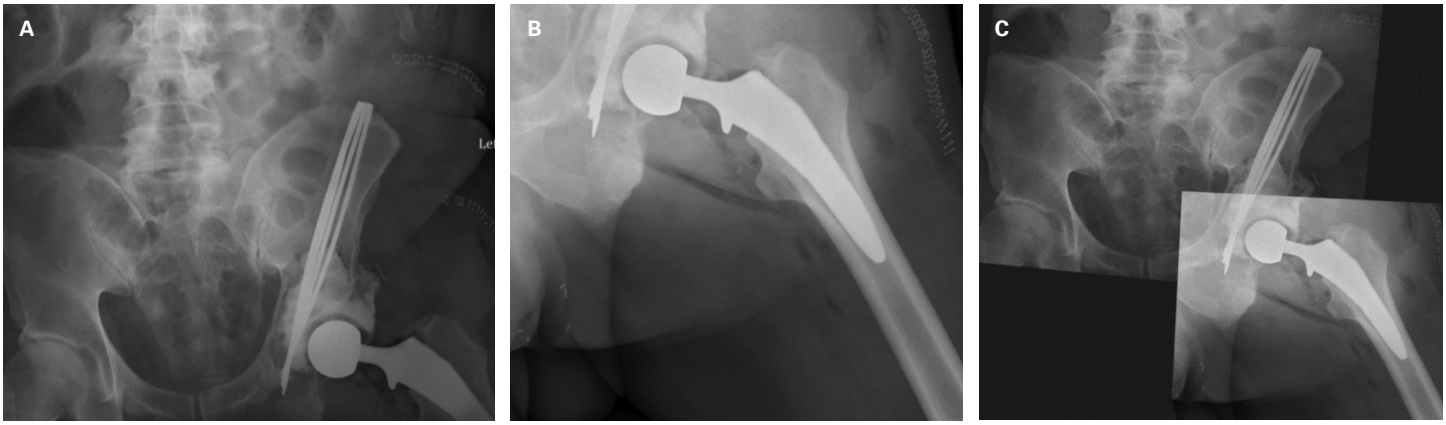

Figure 4. Post-operative X-ray status post left hip Harrington with total hip arthroplasty.

- When positioning the patient lateral, placing a bump under the contralateral hip allows easier access to the iliac crest for pin insertion, especially in larger patients.
- Checking fluoroscopic views before prepping and draping will ensure that unobstructed AP pelvis and obturator oblique images can be obtained for intra-operative guidance.
- Because the anterior and posterior columns are often absent, standard Hohmann-type retractors are not effective and large deavers should be available for exposure.
- Threaded rather than smooth Steinmann pins should be used, as the latter tend to migrate proximally in the months or years postoperatively. This causes point tenderness over the iliac crest at the site of the prominent pin and requires removal of the offending pin(s).
- The Harrington procedure classically describes placing pins in specific locations (i.e., one anterior, one posterior, and one superior). However, we recommend assessing the pattern of bone loss and placing the pins to compensate accordingly. For example, in the case presented here, the majority of the bone loss was posterior, so most of the pins were used to recreate the posterior column.
- Fixing the pins distally into the ischium or pubic ramus, if bone remains in those locations, provides additional stability (Figure 5). If needed, pins traversing the acetabular defect can be redirected into the ischium/ramus with a large right-angle clamp or army-navy retractor. For example, if the pin is directed medially, the instrument is used to pull it laterally while advancing the pin until it fixes in the bone distally (Figure 6).
- Once the pins are appropriately placed and confirmed to be in final position, trimming and burring down the ends to be flush with the iliac crest will avoid pin prominence and irritation. If they need to be removed at a later date, a rongeur can be used to debride a small portion of the iliac crest to expose and engage the end of the pin for removal.
- Depending on the size of the patient and pin configuration, a thinner acetabular liner may be helpful to maximize head size and stability. Thin liners are not designed for cementation but can be easily adapted for this purpose by scoring the outer surface with a bur.
- Steinmann pins passed from the iliac crest into the ischium may pass through the acetabular region and excessively lateralize the cup. In order to avoid this, pins should ideally be directed either anteriorly, posteriorly, or medially. Pins that would interfere with cup position can be reversed until they support the liner superiorly (Figure 7).
- Trialing and memorizing the placement, angulation, and version of the liner prior to cementing is helpful, as filling the defect with cement and thereby covering the bone and pins can be disorienting. If any portion of the acetabulum remains, such as a rim of the anterior/posterior wall or transverse acetabular ligament, this can be used as a landmark to accurately recreate hip center, version, and offset.
- Prior to cementing, pulse lavage, hydrogen peroxide, and epi-soaked packing can be used to remove blood and dry the bone surface for improved cement fixation.
- When the posterior column is absent, the sciatic nerve is unprotected; ensuring cement does not migrate posteriorly and irrigating with cool saline during the curing process can prevent thermal damage to the nerve.
- If patients do not have risk factors for fragility fracture (e.g., femoral metastases, age, osteoporosis) an uncemented femoral component may be used to reduce operative time.

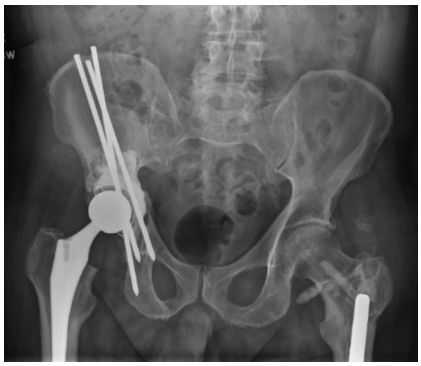

Figure 5. Steinmann pins fixed distally into the ischium or pubic ramus provides additional stability.

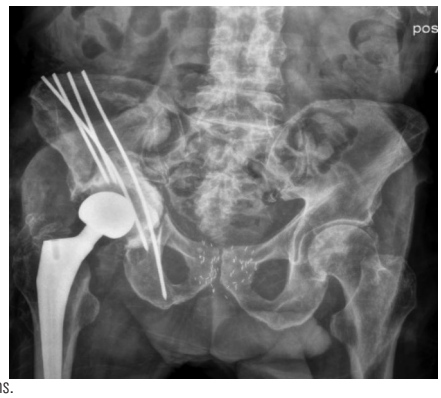

Figure 6. Bent pins.

Conclusion

The Harrington reconstruction is an effective surgical technique to relieve pain and restore ambulatory function in patients with destructive periacetabular metastatic disease. This case reviews the surgical technique and treatment course for a patient with metastatic urothelial carcinoma. Despite his poor prognosis, the patient was able to ambulate and maintain a significant degree of functional independence during his remaining lifetime.

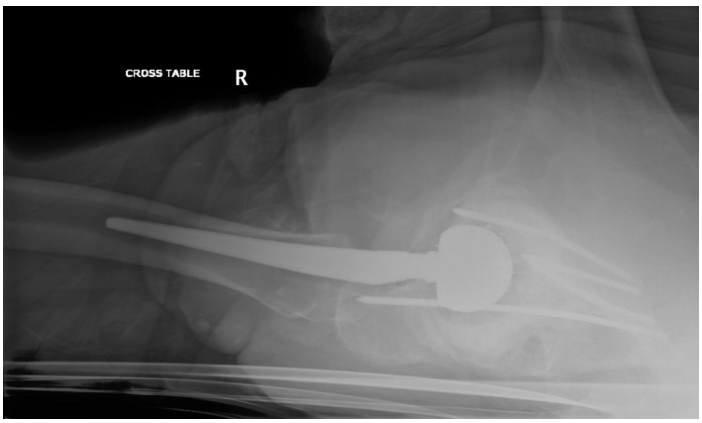

Figure 7. Pins that would interfere with cup position can be reversed until they support the liner superiorly.

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Orthoplastics

U·P·O·J

Orthoplastics Tips and Tricks: Medial Femoral Condyle Vascularized Bone Graft for Scaphoid Nonunion: A Case Report

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Introduction

Scaphoid nonunions occur in 10-15% of scaphoid fractures with increased likelihood in cases with delay in treatment, proximal pole fracture, or carpal instability.[1-3] Fracture of the proximal third of the scaphoid are at an increased risk of osteonecrosis after scaphoid nonunion due the unique blood supply to this region and therefore present a unique challenge for repair.[4,5] When treating scaphoid nonunions, vascularized bone grafts have been demonstrated to improve rates of union when compared to non-vascularized bone graft. Vascularized bone grafts have been reported to achieve a 90% union rate in scaphoid nonunions with proximal pole avascular necrosis.[1]

Historically a number of graft sites have been utilized for treatment of scaphoid nonunions including the distal radius, ulna, and metacarpals.[2,6,7] Recurrent nonunion rates following scaphoid nonunion surgery have been reported as high as 25% due in part to bone graft resorption.[8] Attempts at mitigating this issue with vascularized pedicle bone grafts from the distal radius have added challenges including their short vascular pedicle, unreliable vascular supply, and difficulty shaping the graft to fit the defect without compromising the graft's integrity. In 2000, Doi et al initially described the use of a vascularized medial femoral condyle periosteocortial bone graft as an alternative option for treatment of a scaphoid nonunion with the potential to avoid the pitfalls of other grafts.[8]

The medial femoral condyle vascularized periosteal bone graft is unique in that it includes the periosteum, a full layer of cortical bone, as well as the underlying cancellous bone which is optimal for repair of small bone defects.[9] The blood supply to the periosteum of the medial femoral condyle comes from the femoral artery via the articular branch of the descending genicular artery and from the popliteal artery via the superomedial genicular artery.[8,9] The success of this graft lies in maintaining the cambium layer of the periosteum which is critical to the osteogenic capacity of the MFC graft.[10] The vascularity of the cortical and cancellous bone of this graft allows for significant shaping of the graft to fit into the scaphoid defect without loss of sufficient blood supply to the graft as seen in other grafts from the fibula or iliac crest.[8,9]

Case Report

A 21-year-old right-hand dominant male college student presented to our clinic with a chief complaint of eight months of right wrist pain without known trauma. At the time the patient reported he had increased his sporting activities including basketball and snowboarding over the past year but could not recall a specific trauma to the right wrist. On exam, he had swelling and tenderness to palpation over the anatomic snuff box. Right wrist range of motion was limited by approximately 10 degrees in flexion and extension due to pain. He had full range of motion in supination, pronation, ulnar deviation, and radial deviation. He was neurovascularly intact. X-rays showed a displaced scaphoid waist fracture with no sign of bridging callus (Figure 1). The patient

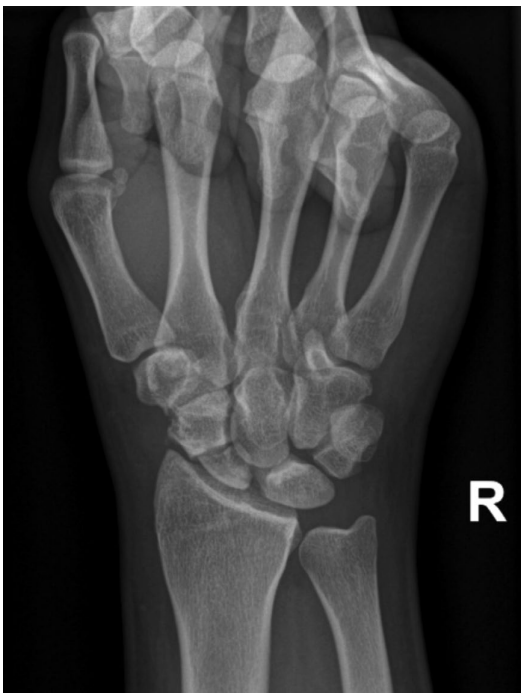

Figure 1. Radiographs at initial presentation demonstrating right scaphoid waist nonunion.

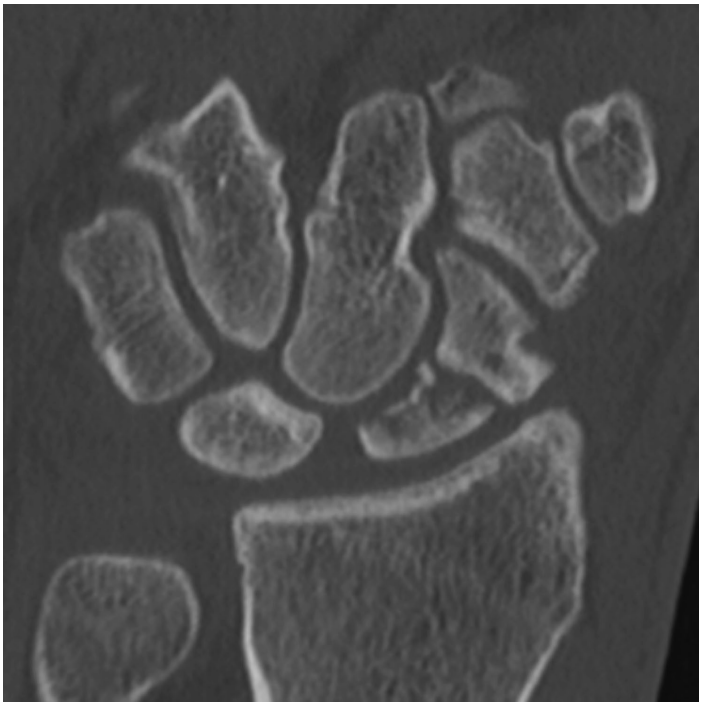

Figure 2. CT at initial presentation demonstrating scaphoid nonunion without callus formation.

was diagnosed with scaphoid nonunion and a CT wrist was ordered which confirmed the diagnosis (Figure 2). The patient was then referred to our orthoplastics service.

The patient was evaluated by the orthoplastics service one month after initial presentation. His exam was unchanged from prior. Non-operative and operative treatment were discussed with the patient. After discussing surgical options the patient agreed with the recommendation to pursue right wrist scaphoid nonunion open reduction internal fixation with non-vascularized corticocancellous bone graft from the distal radius. At this time the patient was informed that if the distal radius bone graft did not go on to persistent union within three months, the next indicated operative intervention would be a vascularized medial geniculate graft.

The patient underwent right scaphoid nonunion repair with distal radius bone graft and plate and screw fixation two months after index presentation (Figure 3). CT imaging was utilized throughout routine follow-up to assess bony healing. Five months after the distal radius bone graft, CT right wrist demonstrated collapse of the severely comminuted fracture of the proximal pole of the scaphoid with no evidence of bony bridging of the fracture fragments or incorporation of bone graft material despite intact hardware (Figure 4). The decision was made to pursue medial femoral condyle vascularized bone flap for treatment of the patient's right scaphoid nonunion.

Eleven months after initial presentation the patient underwent removal of hardware and nonunion repair with medial femoral condyle vascularized bone flap under general anesthesia. The decision was made to harvest the bone graft from the right lower extremity. Two attending surgeons worked simultaneously on the right wrist and right thigh. The right upper extremity procedure was performed under tourniquet at 250 mmHg. A volar incision was made on the right wrist through the previous surgical incision. The superficial branch of the radial artery was identified as it crossed over flexor carpi radialis. This was carefully preserved, clipped, ligated, and dissected out proximally for potential inflow to the bone flap. The FCR tendon sheath was incised and FCR was retracted laterally allowing for identification and inspection of the radial artery. An arthrotomy was performed through the volar aspect of the wrist capsule. The scaphoid plate was identified and a screwdriver was used to remove the screws and free the plate. The plane of the scaphoid fibrous nonunion was then developed with a freer elevator. The proximal pole of the scaphoid was removed with a rongeur, and a small burr was used to debride the nonunion site at the waist until there was punctate bleeding.

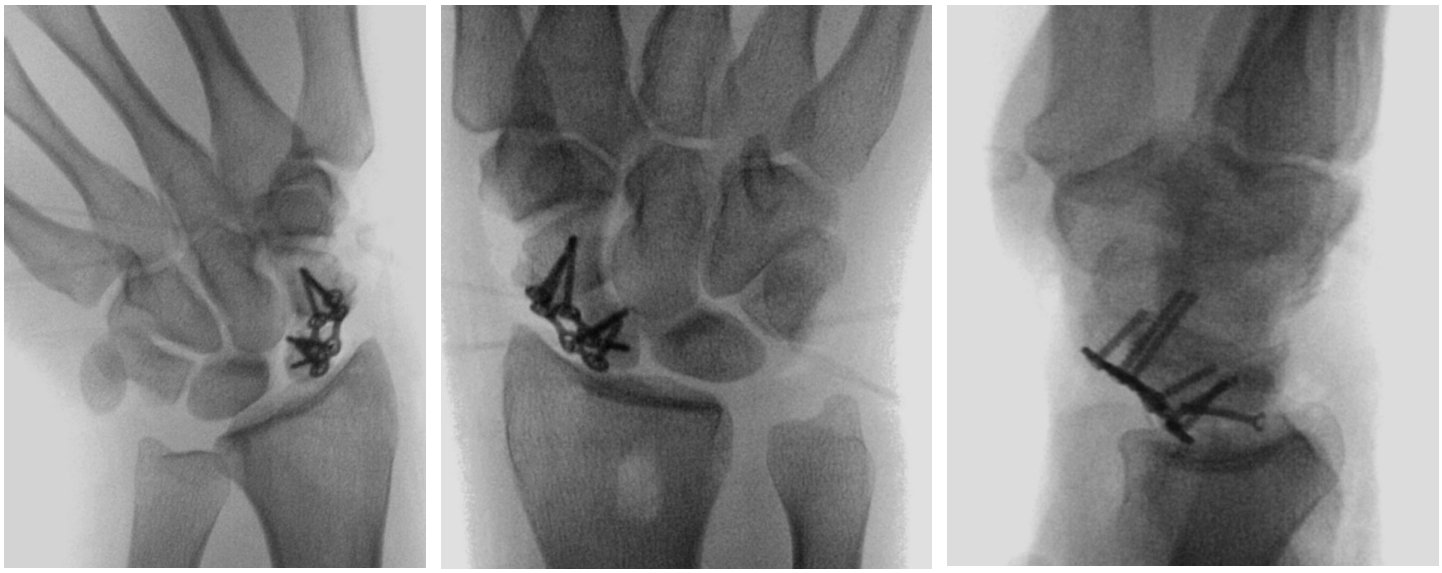

Figure 3. Intra-operative fluoroscopy demonstrating right distal radius nonunion status post right scaphoid nonunion repair with distal radius bone graft and plate and screw fixation.

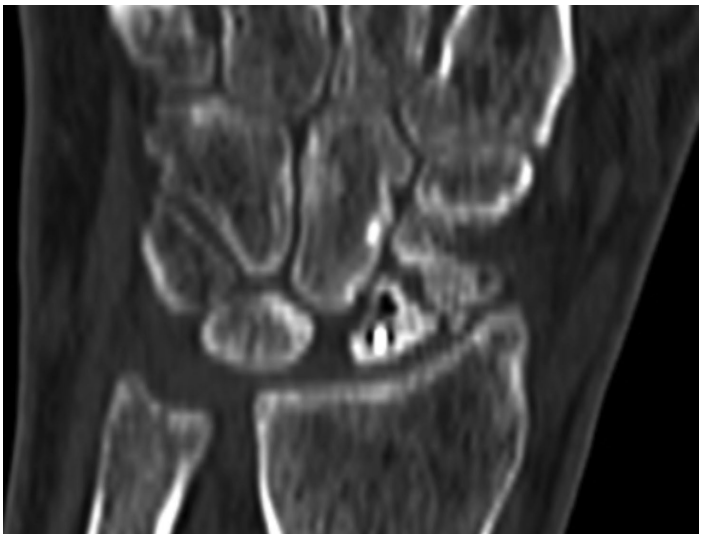

Figure 4. CT R wrist five months status post right scaphoid nonunion repair with distal radius bone graft and plate and screw fixation demonstrating no evidence of bony bridging or incorporation of bone graft.

Simultaneously the right lower extremity tourniquet was inflated to 350 mmHg. A longitudinal incision was made over the medial thigh. Dissection was taken down to the vastus fascia which was incised, allowing the vastus medialis to be reflected anteriorly. The medial geniculate artery was identified and traced retrograde to the takeoff of the superficial femoral artery. A subperiosteal dissection performed at the interface of the geniculate artery and the femoral condyle using the vascular geniculate vessels on a periosteal leash. The vessels were then cross-clamped proximally and the block of corticoperiosteal cancellous bone was transferred to the scaphoid. A drain was placed at the distal thigh prior to closure.

The bone graft was tailored to fit into the defect of the proximal pole of the right scaphoid with a rongeur. A proximal mold was created with bone wax to help define the necessary size and margin of the graft. Additionally, cancellous bone that had been excised from the MFC flap was used also to pack the distal aspect of the scaphoid. A 2.4 24 mm headless cannulated screw was then placed from the distal pole of the scaphoid into the new reconstructed proximal pole of the scaphoid. Placement was confirmed with direct inspection and intra-operative fluoroscopy (Figure 5). Microvascular anastomosis was performed between the dorsal branch of the radial artery and vena comitans using a 2 mm coupler for outflow. An end-to-end anastomosis of 9-0 nylon with spatulated vessels was performed on the arterial side yielding excellent inflow and outflow. The wound was copiously irrigated and closed. Excellent arterial signals were present at the conclusion of the case and a stitch was used to mark the vascular pedicle of the flap for post-operative monitoring. The right wrist was immobilized in a bulky compressive volar splint. The patient was discharged home from the hospital on post-operative day three.

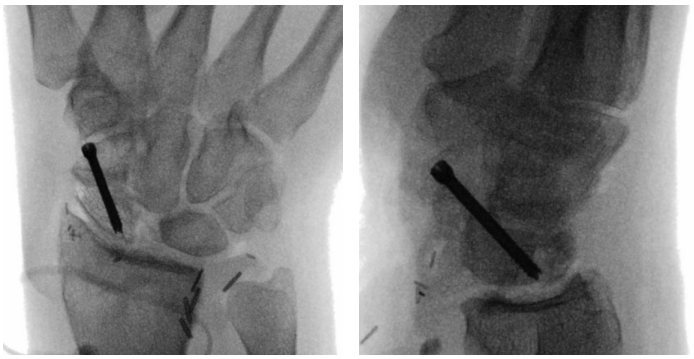

Figure 5. Intra-operative fluoroscopy of the right wrist during medial femoral condyle vascularized bone flap to the right scaphoid.

At the patient's two-week post-op appointment he reported he was doing well and denied new numbness or paresthesias to the hand. The surgical incisions were healing well, and his sutures were removed. The vascularized graft had excellent doppler signals. Radiographic images obtained in clinic demonstrated excellent alignment between the native scaphoid and the geniculate osteocartilaginous graft. The headless screw in place without signs of hardware complication. The patient was placed in a thumb spica cast with a plan to follow-up in two weeks. At four week-follow-up the patient had some painless wrist range of motion and was neurovascularly intact. Radiographs again demonstrated intact fixation of the right scaphoid. The patient was placed in a thumb spica cast and scheduled for a CT right wrist to be obtained eight weeks post-operatively to assess healing. CT right wrist demonstrated progressive healing with sites of early bony bridging adjacent to the surgical screw, though fracture lines were still visible.

At four-month follow-up the patient demonstrated continued increase in painless wrist range of motion. He reported some residual wrist stiffness as well as a painless prominence on the volar aspect of his wrist. He was otherwise neurovascularly intact. CT right wrist performed at that time demonstrated interval bone healing along the scaphoid waist with hardware intact. A hypertrophic spur

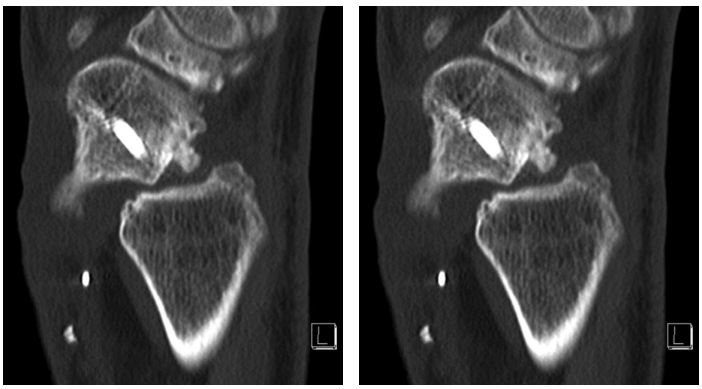

Figure 6. CT right wrist four months status post medial femoral condyle vascularized bone flap to the right scaphoid.

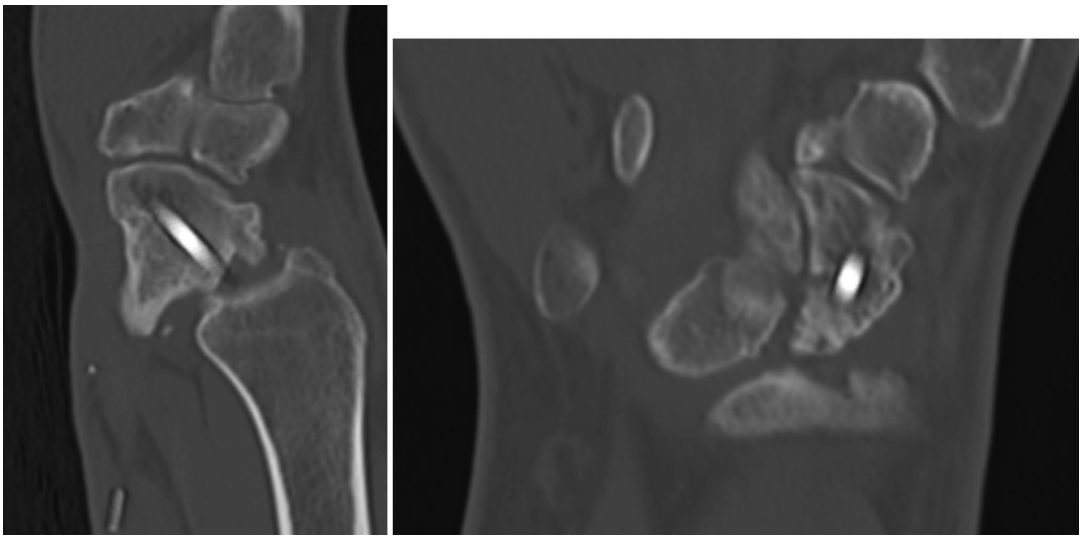

Figure 7. CT right wrist eight months status post medial femoral condyle vascularized bone flap to the right scaphoid.

was notably extended from the scaphoid into the volar soft tissues. Repeat CT right wrist performed eight months post-operatively demonstrated increased healing and callus formation across the fracture site and hardware in place. No evidence of nonunion.

At one year follow-up patient was doing well and had returned to athletic activities including playing football. He denied pain in the wrist but endorsed occasional crepitus. Radiographs demonstrated increased bony bridging of the scaphoid fracture site and a large volar scaphoid osseous prominence (Figure 8). He had a persistent volar wrist excrescence for which he will electively undergo excision of right scaphoid prominence.

Discussion

Scaphoid nonunion is a relatively common but difficult to predict complication of scaphoid fracture and can be a challenging issue to treat. It is essential to recognize scaphoid nonunion early in its course so the appropriate intervention can be completed. As with our patient, CT scan can be used to identify nonunions promptly and allow for operative planning and decreased delays in intervention. In 2009, Geoghegan et al followed scaphoid fractures for four weeks in a thumb spica cast followed by CT scan at the four-week mark. They determined that all scaphoid fractures which appeared nondisplaced and united at four weeks went on to union.[11] In 2018, Bhat et al proposed their own protocol in which nondisplaced and minimally displaced scaphoid fractures were treated conservatively in a cast for six weeks at which time radiographs were obtained to assess fracture healing. Fractures with doubtful union then underwent CT imaging for assessment of healing at the fracture site. Those fractures will more than a 2mm gap were indicated for percutaneous screw

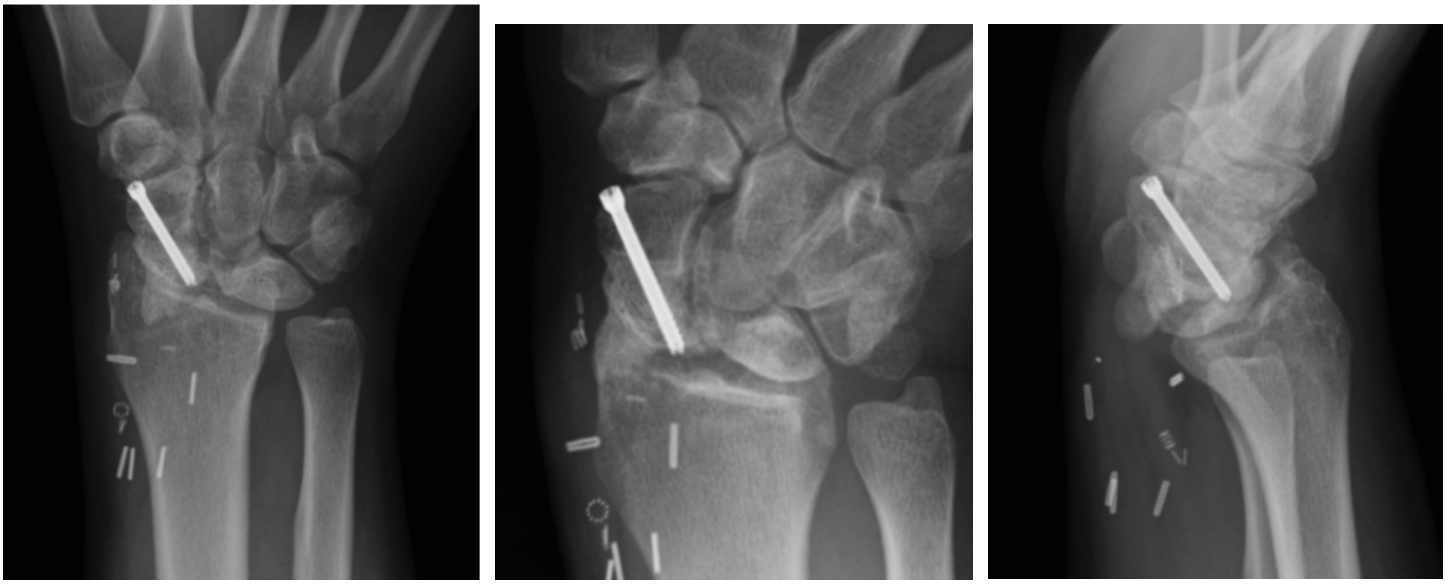

Figure 8. XR right wrist one year status-post medial femoral condyle vascularized bone flap to the right scaphoid.

fixation of the scaphoid whereas those with less than 2mm gap continued immobilization in a cast for an additional two to four weeks.[12] By following this protocol they achieved a 100% union rate at one year. CT imaging at four to six weeks of planned non-operative management of scaphoid fracture may help predict which fractures will ultimately progress to nonunion. These patients can then be assessed for which operative intervention would be most appropriate based on CT findings. Patients with concern for impending or present proximal pole collapse would benefit from early consideration for a vascularized medial femoral condyle bone graft which would decrease their time to definitive union.

Conclusion

The vascularized medial femoral condyle is a reliable option for repair of scaphoid nonunion that allows for treatment of small proximal pole defects. It has particularly dependable blood supply which reduces the risk of bone graft reportion, a common obstacle when treating scaphoid nonunion. We anticipate that our patient will regain full range of motion and grip strength. The use of the medial femoral condyle vascularized bone graft provided our patient with a superior option for definitive surgery to treat their scaphoid nonunion.

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Arthroplasty

U·P·O·J

Arthroplasty Tips and Tricks: Diaphyseal Impaction Grafting and Metaphyseal Cone for Severe Tibial Bone Loss in Revision Knee

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Introduction

With the prevalence of revision knee arthroplasty continuing to rise, it is important to have several techniques available to address bone loss in the revision setting.[1,2] Multiple treatment algorithms have been proposed to help surgeons manage bone loss in the revision knee setting to restore the patient's limb alignment, joint line, soft tissue balance, patellar tracking, and range of motion.[3,4]

One particularly challenging instance is the patient with a previously violated diaphysis, resulting in a sclerotic diaphysis combined with metaphyseal bone loss. The sclerotic diaphysis impairs cemented stem fixation, and that lack of initial stability impairs bone ingrowth onto a metaphyseal cone and subsequent long term fixation.

One solution that has been proposed for this challenging problem is diaphyseal impaction grafting in combination with metaphyseal cone for patients with severely compromised bone. This has been described with promising initial results. The purpose of this paper is to describe a case report utilizing the technique of diaphyseal impaction grafting with a metaphyseal cone for a patient with severe tibial bone loss in a revision knee and review literature regarding this technique.[5]

Case Report

This is a 76 year old female who presented to clinic with several years of worsening right knee pain. She has a history of a right total knee arthroplasty (TKA) in 2000 and subsequent revision TKA in 2005. On physical exam, notable findings are that her BMI is 43, range of motion is 0-95 degrees, and she has an intact extensor mechanism. She also had gross varus and valgus instability throughout range of motion. Her x-rays demonstrate evidence of loosening of her tibial component, marked bone loss, marked shortening of the leg and lowering of the joint line. Preoperatively, an infectious workup was obtained including bloodwork and synovial aspirate to rule out periprosthetic infection and was negative.

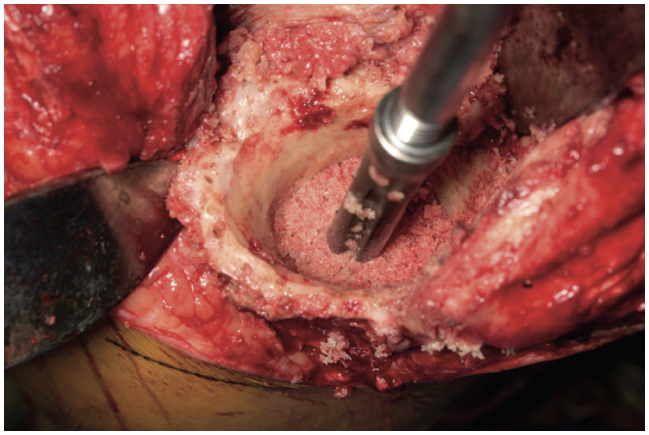
She was indicated for a revision TKA. Plan A was to revise the tibia only with impaction bone grafting and a metaphyseal cone. Plan B was to revise both components with tibial impaction bone grafting and a metaphyseal cone. Intraoperatively, she was found to have an Anderson Orthopedic Research Institute Type IIB AORI IIB bone loss involving both tibial plateaus, which was consistent with preoperative planning. The femoral component was not loose. Therefore, we proceeded with the initial plan of revising the tibial component only, with diaphyseal impaction grafting using a metaphyseal cone, and cementing stem.

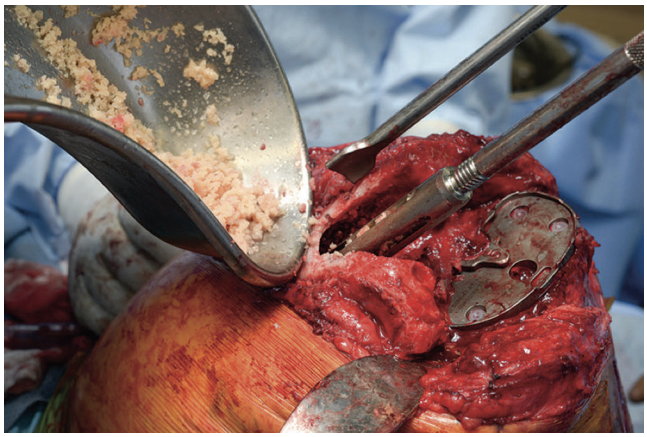
We used the original technique for impaction grafting originally described by Hovelius and modified by Bedard et. al. with the use of cones. Sclerotic canals are irrigated and debrided. Then, the diaphysis is touch reamed and either a bone plug or cement restrictor is placed 2 cm distal to where the tip of the implant will end. A reamer or trial stem is placed centrally in the position of the final implant and to guide the placement of cancellous autograft surrounding the trial stem. Small pieces (no larger than 5mm) are introduced around the stem in batches and densely impacted to fill approximately 1-2 cm at a time. This is repeated until the graft is impacted to the level of the metaphyseal cone. The bone graft should be packed densely enough that after removing the trial stem, no bone graft should fall into the reconstituted diaphyseal canal.

Then, a metaphyseal cone is implanted in standard fashion. Any gaps between the implant and the host bone are supplanted with autograft. Then, the diaphysis is carefully prepared and a cement gun is used to fill the canal and cone in a retrograde fashion. Finally, the implants are implanted.

Discussion

The goals of a revision TKA, like that of a primary TKA, are to restore limb alignment, joint line, soft tissue balance, patellar tracking, and range of motion. This goal can be





complicated by bone loss in the revision setting, making it a challenging endeavor. Patients can have bone loss due to several etiologies including infection, osteolysis, loosening and mechanical bone loss, stress shielding, and iatrogenic loss during revision surgeries. It is important to determine the cause of bone loss as part of the preoperative workup.

Bieganoswki et al give several tips in approaching the revision TKA. The authors state: "rTKA can be broken down into three steps, generalized as follows: (1) reestablish the tibial platform, (2) restore the flexion gap, and (3) reconstitute the extension gap."[3] They also advise regarding the use of intraoperative anatomic landmarks to evaluate for bone loss and estimate the location of the joint line: "One may theoretically estimate femoral bone loss by assuming that the normal distance from the lateral and medial epicondyles to the joint line is 25 mm and 30 mm, respectively. The adductor tubercle is usually 40–45 mm proximal to the joint line. Furthermore, a rough calculation of the depth of tibial bone loss begins with the presumption that the fibular head is 15 mm distal to the joint line."[3]

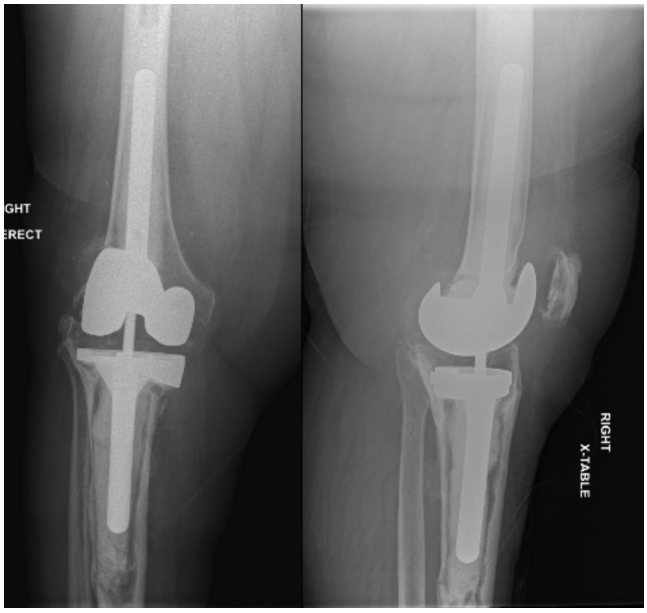
Bone loss requires a systematic approach and multiple techniques based on differing severity and location. It is important to have a preoperative plan based on imaging, but know that final classification of bony defects cannot be done until after implant removal. The most commonly used classification in the revision knee is the Anderson Orthopaedic Research Institute (AORI) classification. Several proposed treatment algorithms propose treatments based on the amount of bone loss and therefore the AORI type. As described above in the case report, this patient was classified as an AORI IIB, in that they had bone loss of both tibial plateaus.

AORI Classification.[7]

Type	Description
I	Minor and contained cancellous bony defects that do not affect implant stability
II	Moderate to severe cancellous and/or cortical bone defects IIA: one tibial plateau or femoral condyle IIB: both tibial plateaus or femoral condyles
III	Massive cavitary and segmental bone loss of both tibial plateaus and/or femoral condyles with/without ligament or tendon involvement

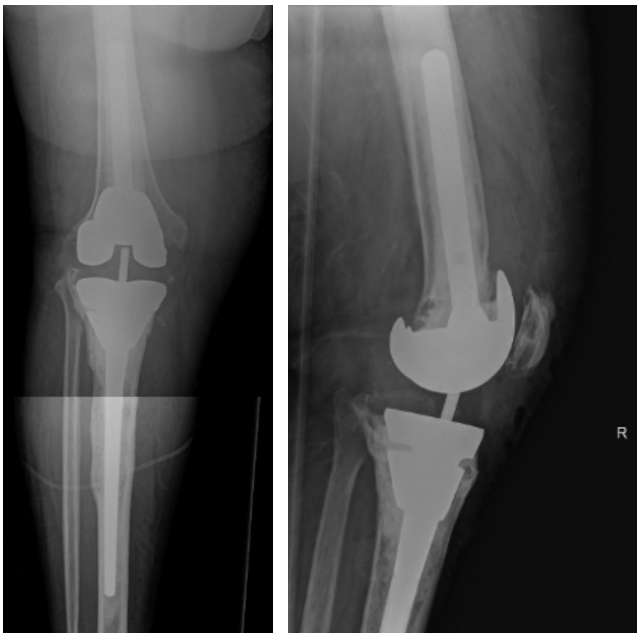
Treatment algorithms have been proposed to suggest management of bony defects. An article by Sheth et. al provides a systematic approach to bone loss in rTKA and a framework for the treatment and implant selection based on available reconstruction options.[4] Bieganowski et al reviews each surgical technique and the AORI type for which it is indicated for. They also review the pros and cons of each technique. This article indicates both impaction bone grafting and metaphyseal cones are indicated for AORI II/III defects. This article does not review the use of impaction bone grafting in combination with metaphyseal cones.[3]

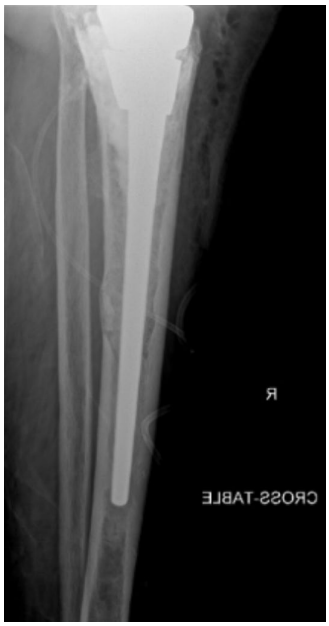
Finally, this article by Bedard et. al. descibes outcomes in the use of metaphyseal cone and diaphyseal impaction grafting in the same technique as described above. 33 revision TKAs underwent this technique and had 100% survival at 5 years from aseptic loosening. 6 knees (17%) required further revision; 4 for infection and two for periprosthetic fracture. 1 unrevised patient had asymptomatic loosening. All unrevised TKAs were incorporated radiologically. This study suggests promising clinical outcomes in patients undergoing this technique.[5]



Conclusion

Bone loss in the revision TKA is a challenging issue, but a sclerotic diaphysis in combination with substantial metaphyseal bone loss is a particularly challenging issue. Diaphyseal impaction grafting with a metaphyseal cone is a promising solution for this issue. Surgeons must have a systematic approach and several techniques ready to address bone loss in the revision knee.





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Pediatrics

Pediatrics Tips and Tricks: Essentials of the Brachial Plexus Physical Examination in Newborns

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Introduction

A perceptive physical examination of the newborn is invaluable in guiding the early diagnostic work up for a wide constellation of congenital and traumatic conditions. The pediatric orthopaedic surgeon should be well acquainted with patterns of injury associated with birth trauma, the intricacies of normal and abnormal neurodevelopmental findings, and recognition of deformity in infants. While decreasing in incidence, brachial plexus birth injury (BPBI) still affects approximately 1 in 1,000 newborns.[1-4] Of these children, 20 to 30% will have residual deficits in upper limb function which may require surgical intervention. Serial neuromuscular examination is the gold standard when determining necessary treatment and future functional outcomes.[3-8]

Broadly, BPBI can be categorized based on upper, middle and lower trunk involvement with isolated upper trunk injuries having increased likelihood of spontaneous nerve recovery and better functional outcomes in the long term. Isolated lower nerve trunk is exceedingly rare given the mechanism of injury for BPBI typically involves shoulder dystocia and traction primarily affecting the upper portion of the brachial plexus. Narakas (1987) published a landmark study classifying BPBI into four categories according to nerve roots and corresponding muscle groups affected (Table 1).[6] Ascending group number has been associated with poorer neurologic prognosis and greater likelihood of requiring surgical intervention.[6,7,8,9] Al-Qattan et al. later expanded upon the classification by accounting for the prognostic value associated with the return of elbow flexion and wrist extension.[9] Initial exam of the newborn should aim to differentiate upper versus lower lesions with subsequent follow-up examination monthly through 6 months of age to assess prognosis and need for surgery.[5,7-9]

History

Brachial plexus birth injury is associated with maternal, gestational, and obstetric risk factors which necessitate targeted history and collaboration with obstetric colleagues. Shoulder dystocia remains the strongest risk factor and increases the risk of BPBI by approximately 100-fold. Other well-known risk factors include use of vacuum or forceps during delivery, breech positioning, birth weight $>$ 4.5 kg, and a baby consistently heavier than expected throughout pregnancy. Hypotonia at birth has also been associated with increased risk for BPBI due to increased susceptibility of the brachial plexus to stretch.[2,4,10] Maternal factors including gestational diabetes and protracted active phase of labor are also pertinent.[4,10] Cesarean delivery, twin or multiple birth mates, and lower birth weight are protective.[2,4]

History obtained from the obstetrician should attempt to quantify strength and directionality of force applied during delivery. Widening of the head-shoulder angle is associated with upper root injury and exceedingly more common than lower root injury which is produced by excessive abduction of the arm.11 In multiple studies, less than 20% of BPBI cases have been total/global or lower plexus injuries with a very small minority being isolated lower.[11,12] Cases of BPBI classified as lower root injury are more commonly total plexus injuries with spontaneous recovery of the upper plexus. Greater magnitude of force equates to higher risk of a global brachial plexus injury and nerve root avulsion.[11] Head-to-body interval of greater than 60 seconds, pubic symphysiotomy, intentional clavicle fracture, Zavanelli maneuver with subsequent C-section, and abdominal rescue maneuvers are all key aspects of the obstetric history.[12]

Physical Exam

The physical exam of the newborn can be mystifying for the orthopaedic consult resident due to challenges with examining a patient not yet capable of verbalizing or following commands. A thorough pediatric brachial plexus exam includes assessment of crepitation or deformity, range of motion (ROM), strength, and neurologic function.[13] A quiet, warm room with dim lighting is

Table 1. Narakas (1987) classification, modified and adapted from Al-Qattan et al (2009).[6,9]

Palsy Group	Nerve Roots	Affected Muscles	Clinical Presentation	Outcome (% recover)
I. Erb's	C5-C6	Deltoid, teres minor (axillary n.) Supraspinatus, infraspinatus (suprascapular n.) Biceps, brachialis (musculocutaneous n.) Brachioradialis, supinator (radial n.)	Shoulder adducted and internally rotated, elbow extended, and forearm pronated	80%
II. Extended Erb's	C5-C7	Group I deficits PLUS Wrist extensors (radial n.)	Same as above with wrist drop	60%
III. Total Plexus	C5-T1	Group I – II deficits PLUS Wrist flexors, hand intrinsics (ulnar n., median n.)	Complete flaccid paralysis of the limb	30-50%, functional hand in many cases
IV. Total with Horner's Syndrome	C5-T1	Group I – III deficits PLUS Ptosis, miosis, anhidrosis (sympathetic chain)	Complete flaccid paralysis with Horner's syndrome	Few recover, lifelong deficits

ideal for infants who are sensitive to light and at risk of exposure. The baby should be laid supine with blankets and clothing removed.[10,11] Feeding the newborn beforehand, using distracting objects to divert attention, and involving parents can reduce irritability.[10]

Observation & Palpation

Over 50% of BPBI cases have no known risk factors, so physical exam remains paramount.[2,4] Due to high incidence of humerus and clavicular fractures with BPBI, exam should begin with observation for deformity and asymmetry of shoulders and upper extremities. Skin examination should include careful assessment for open fractures or pinhole wounds about the clavicle and humerus. Palpate both clavicles for step-offs, "bumps", or subcutaneous crepitus.[13,14]

Abnormal posturing of a newborn can distinguish upper from lower brachial plexus lesions (Figure 1). Upper trunk lesions, also known as an Erb's Palsy (C5-C6) is the most common, are characterized by the classic "waiter's tip" appearance of the upper extremity in which: 1) the shoulder is adducted and internally rotated, 2) elbow extended, 3) forearm pronated, and 4) wrist flexed (Figure 2). Extended Erb's (C5-C7) encompasses diminished strength in elbow and finger extensors as well. While less common, total brachial plexus palsy is a more devastating

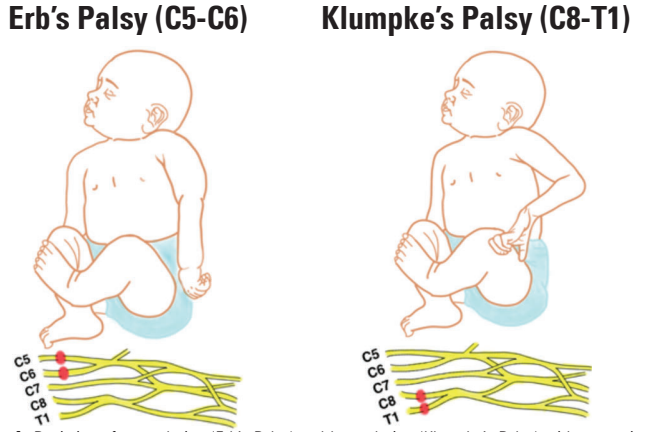

Figure 1. Depiction of upper lesion (Erb's Palsy) and lower lesion (Klumpke's Palsy) with respective nerve roots below.

Figure 2. Picture of infant demonstrating shoulder abduction and internal rotation, elbow extension, and forearm pronation characteristic of an upper trunk injury.

injury in which the affected limb presents as a flaccid, flail extremity. Isolated lower trunk injuries, or Klumpke's Palsy, are rare and produce a "clawed hand deformity" with 1) wrist extension, 2) metacarpalphalangeal hyperextension, and 3) interphalangeal flexion.[6,10] Examiners should also assess for the triad of Horner's Syndrome (ptosis, miosis, and anhidrosis) as injury occurring proximal to white rami communicans hinders the sympathetic output to the head, neck and eye.[6] Respiratory distress may indicate phrenic nerve injury with resultant diaphragm paresis (DP) which occur in up to 2% of brachial plexus injuries. Injury to the phrenic nerve or Horner's syndrome are prognostic indicators as they often as associated with nerve root avulsion.[15]

Range of Motion

Physical exam of the infant involves assessment of range of motion (ROM), active movement, and reflexes. In utero, neonates develop flexion contractures of the elbows, knees, and hips that persist after birth and are a normal finding on

exam.[13] Assessment of neck ROM can lead to identification of torticollis often due to sternocleidomastoid trauma during delivery. Limited upper extremity ROM with fixed adduction and internal rotation of the shoulder and elbow flexion should lead to consideration of arthrogryposis or Sprengel deformity of the scapula. Cerebral palsy should also be included in the differential diagnosis for children with limited ROM or atypical contractures.[10] Gentle ROM exercises may also elicit a painful response if a fracture is present.

Strength Assessment

Motor responses can be elicited in older children by dangling shiny or distracting objects in front of the infant, stimulating a reaching response. Observe shoulder abduction, internal rotation, and external rotation, elbow and wrist flexion and extension, and hand function. The "cookie test" assesses biceps flexion, a critical prognosticator, by requiring the child to bring a treat to their mouth with the affected extremity while the shoulder is restrained in adduction.[9] Strength may be quantified via standard MRC muscle power assessment, or preferentially, using the Active Movement Scale (AMS) which also assigns scores for muscle grade ranging from no contraction to full motion against gravity (Table 2). In comparison to MRC Grades, AMS grades allow for identifying subtle differences in strength in infants and young children with brachial plexus injuries who are not able to cooperate with provider examination or resist force. The AMS is a formal test administered by a trained physician or pediatric physical/occupational therapist with gravity eliminated and against gravity if the infant is capable of the former.[16]

Table 2. Comparison of MRC Grading versus AMS Grading systems.[16]

MRC Grade	Observation	AMS Grade	Observation
Gravity Eliminated			
0	No contraction	0	No contraction
1	Flicker contraction	1	Contraction, no motion
2	Active movement	2	Motion ≤ ½ range
		3	Motion > ½ range
		4	Full motion
Against Gravity			
3	Active movement	5	Motion ≤ ½ range
		6	Motion > ½ range
		7	Full Motion
Against Resistance			
4	Active movement, some resistance		
5	Normal power, full resistance		

Table 3. Primitive reflexes relevant to brachial plexus examination of newborn, modified and adapted from Payares-Lizano and Pino (2019).[12]

Reflex	Age present (mo)	Description	Upper Trunk BPI (C5, C6)	Lower Trunk BPI (C8, T1)
Moro "startle"	< 6	Startle: Loud noise or abrupt change in head position produces abduction of shoulder, extension of elbow and digits. Embrace: Immediately followed by adduction and flexion.	Startle: Weak shoulder abduction, external rotation Embrace: Weak elbow flexion	Startle: Unable to extend IP joints Embrace: Unable to flex wrist or MCP joints
Grasp	< 3	Grasps examiner's finger in palm	N/A	Characteristic claw hand deformity, unable to activate intrinsic hand muscles
Neck righting	< 10	When head is turned to one side, trunk and limbs spontaneously follow to ipsilateral side	Asymmetry may not be apparent, subtle difficulty flexing elbow when reaching across body	N/A
Symmetric tonic neck	< 6*	When neck is flexed, upper limbs flex and lower limbs extend	Weak elbow flexion	N/A
Asymmetric tonic neck	< 6*	When head is turned to one side, ipsilateral "face side" limbs extend and contralateral "skull side" limbs flex	Ipsilateral: Weak shoulder abduction Contralateral:Weak elbow flexion	Asymmetry may not be apparent, subtle contralateral sided weakness with wrist flexion

* May require 1-2 months to develop, disappear up to 10 months later.

Serial examination with reference to previous scores is an objective method of tracking the natural history of an infant's injury.

Neurologic Function

Primitive reflexes are a useful tool when identifying brachial plexus injury (Table 2). Simply observing infants and ranging joints does not result in a consistent, reproducible muscle activation as demonstrated by primitive reflexes. Subtle asymmetries in the Moro, grasp, tonic neck, and neck righting reflexes can help localize injury as upper lesion, lower lesion, or flaccid paralysis (flail limb).[12] For example, a child with unilaterally absent grasp reflex but symmetric Moro reflex may have a lower brachial plexus lesion. When testing primitive reflexes, the Moro reflex may lead to agitation and should be performed last. Note, primitive reflexes may be abnormal in children with concurrent neuromuscular conditions including cerebral palsy.

Differential Diagnosis

Shoulder dystocia, the leading risk factor for BPBI, occurs when maternal pelvic inlet obstructs the fetal shoulder,

which can lead to a traction injury most commonly affecting the upper trunk of the brachial plexus. Clavicle and humerus fractures are associated with shoulder dystocia. The literature is variable on whether clavicular fractures are protective against BPBI. Some studies suggest that timing of the clavicle fracture is pertinent, e.g., whether the fracture occurs prior to extensive traction on upper extremity.[17] Additionally, spiral morphology of clavicular fracture correlates with BPBI. Plain films are sufficient to diagnose the fracture and may also lead to identification of hemidiaphragm position, if visible.[18] Infants with clavicular or humerus fractures often present with pseudoparalysis, or a reluctance to move the affected limb due to fracture-related pain. To distinguish pseudoparalysis from BPBI, repeat exam in two to four weeks is recommended to allow for fracture healing and mitigate the confounding effects of fracture pain.[18]

Radial nerve palsy due to prolonged compression of the upper arm against the maternal pelvic brim or due to humeral shaft fracture should also be on the differential diagnosis list when evaluating a new patient with suspected BPBI. Infants with isolated radial nerve palsy present with inability to extend the wrist, thumb, and metacarpophalangeal joints (Figure 3). Importantly, preserved external rotation of the shoulder and elbow flexion differentiates radial nerve palsy from upper trunk injuries. Bruising along the posterolateral aspect of the affected arm or presence of a subcutaneous nodule representing fat necrosis are skin lesions that imply prolonged radial nerve compression (Figure 3).[19] Radial nerve palsy often resolves spontaneously within months of initial injury and may be treated conservatively with passive stretching and extension splinting to prevent wrist and digital flexion contractures.

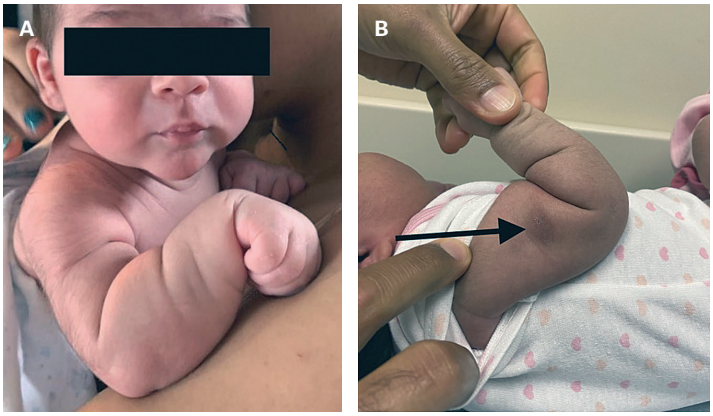

Figure 3. Typical presentation of isolated radial nerve palsy. **(A)** Wrist and finger drop with preserved elbow flexion. **(B)** Subcutaneous nodule with posterolateral bruising.

Conclusion

Workup for suspected brachial plexus birth injury (BPBI) begins during infancy with a thorough orthopedic evaluation. Assess for risk factors and obtain maternal and birth history including birth weight, presence of shoulder dystocia, and any heroic maneuvers employed during delivery. A physical exam incorporating observation of deformity, primitive reflexes, and strength grading systems will help determine extent of BPBI (Erb's, Extended Erb's, Global) and differentiate from birth fracture or neonatal radial nerve palsy. Evaluate for Horner's syndrome and phrenic nerve injury as these may be indicators of nerve root avulsion. Patients with associated upper extremity fractures require repeat examination after fracture healing to rule out pseudoparalysis.

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Post-Traumatic Carpal Tunnel Syndrome in Children

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Introduction

Carpal tunnel syndrome (CTS) is rare in children but can occur after traumatic wrist or forearm injuries.[1-7] Distal radius fractures are the most common cause.[8-12] Patients present acutely in the emergency department after recent injury and urgent carpal tunnel release (CTR) is recommended.[8] Alternatively, those who present later after an injury are treated more like idiopathic CTS cases and may trial more conservative measures before surgery.[10]

In adults with distal radius fractures, studies have showed good outcomes after acute CTR done at the same time as open reduction and internal fixation.[12,13] Given the limited information about CTS in children, the purpose of this study is to describe the presentation and treatment results of post-traumatic CTS in a pediatric population. It should be noted that children with mucopolysaccharide storage disorder such as Hurler syndrome can develop CTS without a history of trauma.[14,15]

Methods

In this retrospective single-center cohort study, all children with post-traumatic CTS from June 2007 to August 2022 were identified by diagnostic codes. Demographic and clinical presentation, treatment, and outcomes data were collected from electronic medical record review and descriptive statistics were calculated. Patients were categorized by time of presentation (Acute < 3 days, Subacute 1-6 weeks, or Delayed > 6 weeks) and whether patients were monitored before surgical treatment. Chi-Square and independent sample t-test were used to compare subgroups. $P<0.05$ was considered statistically significant.

Results

Eighteen patients (16 male) with post-traumatic CTS were identified. The average age at time of presentation was 12.8 ± 3.6 years. Of the associated traumas, 83% (15/18) had a history of a fracture involving the distal radius. Of these fractures, 93% (14/15) were displaced, 40% (6/15) had an associated ulna fracture, 40% (6/15) involved the physis, and 27% (4/15) were open fractures. Most of these patients (10/15) had a closed reduction and 6 required open reductions and internal fixation.

In this group, 72% (13/18) patients presented acutely including 6 with symptoms and clinical concern for CTS on the day of injury. 22% (4/18) presented sub-acutely and one patient had a delayed presentation. For all presentations, the most common symptoms were numbness (78%), paresthesias (33%), swelling (33%), and increasing pain (28%). 17% (3/18) had clinical concern for concomitant compartment syndrome on presentation. 6 patients were initially monitored (2 acute, 3 subacute, 1 delayed). Ultimately, 16/18 of the patients underwent operative CTR (13/13 acute, 2/4 subacute, 1/1 delayed).

At follow-up, 81% (13/16) of surgically treated patients had complete symptomatic relief. After CTR, there was no detectable difference in treatment outcomes between patients who presented acutely versus subacutely or delayed ($p = 0.214$). On average, there was not a significant difference in time to symptom resolution between patients who presented acutely versus those who presented subacutely or delayed (1.73 versus 1.58 months, $p = 0.89$). However, among children that developed symptoms acutely, patients who underwent a brief period of observation before CTR had a significantly longer time to symptom resolution than those who underwent immediate CTR (28.0 versus 2.4 weeks, $p < 0.001$).

Discussion

Given its rare presentation, there are few studies on CTS in a pediatric population. Rusch et al. review the etiology of CTS in a cohort of 38 children with 2 traumatic cases.[3] This study is novel for its in-depth assessment of the presentation and outcomes of trauma associated CTS in children and adolescents.

The clinical presentation of post-traumatic acute CTS can be similar to compartment syndrome including symptoms of pain out of proportion and paresthesia.[16] In our cohort, 17% of patients had clinical concern for compartment syndrome. Each underwent

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forearm fasciotomies in addition to CTR. With the potential for permanent disability from long durations of decreased perfusion, acute compartment syndrome should be treated immediately. Though, given the overlap in symptoms, surgeons should consider the concomitant CTR.

Timing of CTR is important. Patients with acute post-traumatic CTS who were observed to monitor symptom resolution had a prolonged recovery (28.0 versus 2.4 weeks) than patients who had more urgent CTR. This finding is similar to adults, in whom delayed intervention for traumatic carpal tunnel had permanent consequences including irreversible and intraneural fibrosis.[8] These results strongly suggest that surgery should not be delayed in children with acute CTS. Next, our subacute post-traumatic CTS group may be akin to previously described "transient CTS" patients in which CTS symptoms after trauma are temporary from nerve contusion and/or stretch.10 Arguably, transient CTS does not require surgical release of the carpal tunnel as nerve dysfunction may resolve with observation, elevation, and adequate fracture reduction. However, of patients who presented sub-acutely, 2/4 ultimately went on to have CTR and of the two that did not, one still had symptoms at their last follow up 18-months later. While it appears that these patients can be safely monitored, we found that the patient may nonetheless need surgery for complete symptom relief. Similarly, for delayed presentations, it is less likely that non-operative treatment such as orthosis or steroid injections will relieve symptoms in pediatric patients.[17] In Van Meir's review of 163 cases of pediatric carpal tunnel, 89% ultimately underwent CTR.[16] Thus, while acute CTS should undergo urgent CTR, there is a high likelihood later presentations will undergo surgical treatment as well and should be anticipated. Encouragingly, operative treatment tends to result in positive outcomes. In our cohort, 80% of surgically treated patients had symptomatic relief at follow-up. These findings in children match adults, in which the clinical benefit of post-traumatic CTR is as effective as those who underwent elective CTR.[2,8-10,12]

Conclusions

Overall, CTS is a rare but known complication of traumatic forearm injuries, especially distal radius fractures. This diagnosis should not be missed in pediatric patients who present with numbness and tingling along the median nerve following forearm injury. Presentation and symptoms justify timely surgical intervention in the acute setting. Urgent surgery reduces the risk of prolonged neurologic recovery following acute CTS in children. Lastly, with subacute presentation, surgery should be considered for patients whose symptoms do not resolve after monitoring given the symptomatic alleviation after treatment.

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Risk Factors and Outcomes of Neonatal Radial Nerve Palsy

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Introduction

Traumatic peripheral nerve palsies are exceedingly rare injuries affecting the newborn patient.[1] Neonatal radial nerve palsies in particular are scarcely represented in the literature, with only a few case series reporting on the topic.[2-7] Frequently associated with difficult labor and delivery, it has been suggested that prolonged, continuous pressure placed on the radial nerve as it courses down the humerus causes a neuropraxia that is often self-resolving.[6] Given similarities in presentation to brachial plexus birth injury (BPBI), radial nerve palsies can often be misdiagnosed leading to undue distress to patients' families.[8] It is therefore imperative to differentiate these two injuries as the treatment and outcomes differ drastically. Although multiple case reports on neonatal radial nerve palsy note spontaneous resolution of symptoms, no study has objectively reported on the injury's presentation and natural history. This series describes the presentation and relevant risk factors associated with neonatal radial nerve palsy while also quantifying the progression of this condition with a validated measure of active motor function.

Methods

Following institutional review board (IRB) approval, a billing query using ICD-9 and 10 codes was conducted for all patients seen for radial nerve palsy from 2007-2022. All patients presenting with signs and symptoms of isolated radial nerve palsy at less than 12 months of age were included. Patients with other common birth injuries including brachial plexus birth injury, clavicle fracture with pseudoparalysis, or humeral shaft fracture with pseudoparalysis were excluded. After identification, clinic and occupational therapy visit notes were reviewed to abstract demographic data, gestational/birth history, presenting symptoms, physical exam findings, and treatments. Serial Active Movement Scale (AMS) scores, a validated scoring system ranging from 0-7 to assess motor function in infants, were routinely captured at each visit to track injury progression over time (Table 1).[9] Descriptive statistics were used to report demographics, risk factors, symptoms, and treatments while paired student t-tests were used to compare AMS scores at presentation and at final follow up.

Results

Twenty-eight total patients with neonatal radial nerve palsy were included. The male to female ratio was 1:1 with an average age at presentation of 18.1 days (range 6 - 53 days). Prolonged labor was recorded for 16 mothers (57.1%) including 12 that underwent Cesarean section and 1 that required an instrumented delivery. Two additional vaginal delivers were complicated by shoulder dystocia. Fifteen patients (54%) had skin markings overlying the radial nerve, including posterolateral arm ecchymosis or fat necrosis (Figure 1b).

Twenty-four patients had complete wrist and finger drop (Figure 1a), while the remaining four had weakness of wrist and finger extension. Mean initial AMS scores for wrist, finger, and thumb extension were 2.1/7, 1.6/7, and 1.5/7 respectively. Wrist and finger progressive extension splints were used in 22 patients for an average of 7.68 weeks (range = 3-26 weeks). The remaining patients were treated with stretching (n = 1) or monitoring (n = 5). Statistically significant improvements in AMS scores were noted between initial and final evaluations with final mean wrist, finger, and thumb extension scores of 6.7/7, 6.8/7 and 6.8/7 respectively ($p < 0.001$) (Table 2). Of the 26 patients with resolution and normalization of AMS scores at final follow up (2 patients lost to follow-up), 18 (69.2%) had resolution by 4 months and 26 (100%) had resolution by 9 months. The average time to resolution for our entire cohort was 11.8 weeks.

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Table 1. Active Movement Scale (AMS) Scores

Score	Observed Movement
Gravity Eliminated	
0	No response
1	Twitch, without movement
2	< Half range
3	> Half range
4	Full movement
Antigravity	
5	< Half range
6	> Half range
7	Full range

Discussion

Developed for the purpose of evaluating active motor function in patients with brachial plexus birth injury, the Active Movement Scale has been validated and adopted in clinical practice at several institutions.[9] With nearly all patients returning for multiple follow up visits, we were able to see a gradual progression toward resolution in the entire cohort. Compared to AMS scores at initial presentation, statistically significant improvements in radial nerve function were seen across the entire cohort.

Difficult labor and prolonged pressure to the lower humerus remains the most likely etiology for radial nerve palsy. As the fetus descends the birth canal, the maternal pelvic brim exerts pressure on the radial nerve as it courses down the spiral groove and along the posterolateral humerus. In instances of prolonged delivery this pressure is accentuated leading to neuropraxia. Consistent with existing literature, risk factors that predisposed these patients to long periods of radial nerve compression were common in the cohort. This study also identified 15 patients that presented with distinct skin markings at the posterolateral upper arm, such as ecchymosis and subcutaneous nodularity which likely represents fat necrosis. A previous series of 25 patients reported skin markings in up to 70% of newborns presenting with signs and symptoms of radial nerve palsy, suggesting that careful examination of the extremity can be helpful in determining the diagnosis.[2]

This study is not without limitations. Since no specific ICD code for neonatal radial nerve palsy exists, it is possible cases could have been miscoded as another type of nerve injury. To mitigate this, broad ICD codes were used in the search query and charts were reviewed by hand to determine eligibility. Additionally, AMS scores were recorded at follow-up visits only meaning overall time to resolution may be overstated. Finally, given the

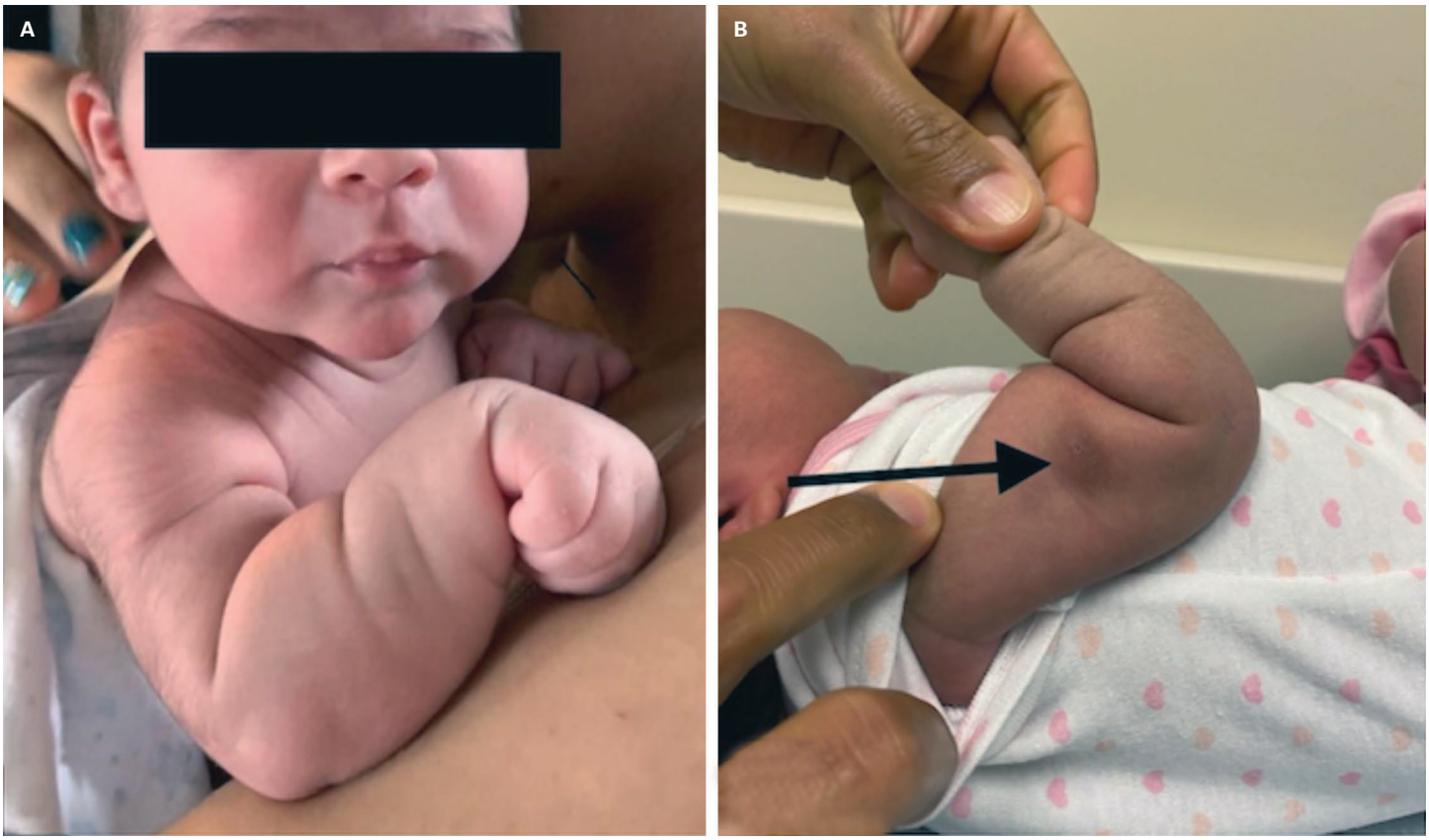

Figure 1. Typical neonatal radial nerve palsy presentation. **(A)** Wrist and finger drop but preserved elbow flexion. **(B)** Ecchymosis and fat necrosis of the posterolateral arm consistent with pressure from prolonged labor.

Table 2. AMS scores and time to resolution for 26 patients with AMS scores at final follow up.

ID	Gender	Time to Presentation (days)	Initial Wrist extension	Final Wrist Extension	Initial Thumb Extension	Final Thumb Extension	Initial Finger Extension	Final Finger Extension	Treatment	Time to Resolution/ Discharge (weeks from presentation)
1	F	6	2	7	0	7	0	7	wrist splint + stretching	16
2	F	7	0	7	0	7	0	7	Stretching	7
3	M	7	2	7	2	7	2	7	wrist splint + stretching	7
4	F	10	0	NA	0	NA	0	NA	wrist splint + stretching	6
5	F	10	0	7	0	7	0	7	wrist splint + stretching	24
6	F	11	2	7	0	7	0	7	wrist splint + stretching	10
7	M	12	0	6	0	7	0	7	wrist splint + stretching	26
8	M	13	0	6	0	6	0	6	wrist splint + stretching	6
9	M	11	1	7	0	7	0	7	wrist splint + stretching	7
10	F	19	2	7	0	7	0	7	wrist splint + stretching	6
11	F	16	7	NA	7	NA	7	NA	none (already resolved)	0
12	M	16	2	7	0	7	0	7	wrist splint + stretching	18
13	M	19	3	7	6	6	3	6	wrist splint + stretching	8
14	F	19	2	7	0	7	0	7	wrist splint + stretching	4
15	F	19	0	7	0	7	0	7	wrist splint + stretching	16
16	F	22	3	7	3	7	3	7	wrist splint + stretching	27
17	M	22	7	NA	7	NA	7	NA	none (already resolved)	0
18	M	25	0	7	0	7	0	7	wrist splint + stretching	24
19	F	32	2	7	0	7	0	7	wrist splint + stretching	24
20	M	11	0	6	0	6	0	6	observation	4
21	M	14	2	6	0	6	2	6	wrist splint + stretching	5
22	F	38	2	6	2	6	2	6	wrist splint + stretching	6
23	F	53	7	NA	7	NA	7	NA	none (already resolved)	0
24	M	26	6	6	3	7	5	7	observation	12
25	M	9	0	7	0	7		7	wrist splint + stretching	35
26	M	20	0	7	0	7	**0**	7	wrist splint + stretching	8
Averages		**17.88**	**2.08**	**6.72**	**1.48**	**6.77**	**1.58**	**6.77**		**11.77**
p-values			**<0.001**		**<0.001**		**<0.001**			

relatively small sample size of this study, it was impossible to perform comparative analyses correlating risk factors to presentation severity and time to resolution. Therefore, large multi-center studies should be considered to better quantify the risk factors of this injury.

Conclusion

Radial nerve palsy is a rare injury in the newborn population. Most of the reported cohort had a history of prolonged labor and physical exam findings consistent with radial nerve compression, supporting the theory that

prolonged compression the radial nerve is responsible for this condition. Nearly universal resolution has previously been reported; however, this is the first study to document the timing and completeness of neurologic recovery with a validated tool for motor function. While this presentation can be distressing to parents and caregivers, a careful history and physical exam can help exclude injuries with worse outcomes. Complete resolution of neonatal radial nerve palsy can be expected within months.

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U·P·O·J

Clinical Relevance of Anterior Cruciate Ligament Tears in Skeletally Immature Patients: Incidence and Co-morbidities

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Introduction

Anterior cruciate ligament (ACL) tears account for 10% of sports injuries in children and adolescents.[1] ACL tears are often accompanied by various co-morbidities,[2-7] which affect surgical decision-making and prognosis.[8, 9] In this review, we discuss the incidence of ACL tears in skeletally immature patients highlighting their co-morbidities.

Incidence of ACL Tears

Incidence of ACL tears varies depending on the age of patients, sex, period of study, study population (general population vs. athlete), type of sports, competitive setting, and geographical region (Table 1). Over the past few decades, the frequency of ACL tears and subsequent reconstructions in the pediatric population has been on the rise, particularly among pre-adolescent and adolescent patients (Table 1).[10-23] One particular study utilized a nationwide database in the U.S. from 2007 to 2011 to reveal a 19% and 18% increase in the diagnosis of ACL tear and a 27% and 16% increase in ACL reconstruction in patients aged 10–14 years and 15–19 years, respectively.[10] However, the change in ACL reconstruction rate in patients aged 5–9 years was similar to that in observed adults although the diagnosis of ACL tears increased by 5%.[10] Another study using a claims and encounter database in the U.S. queried data from 2002 to 2014 and concluded that patients aged 13–17 years had the most significant increase in ACL reconstruction rate over the study period (females: 169.0 to 268.7 per 100,000 person-year; males: 146.8 to 211.7).15 However, the ACL reconstruction rate in patients aged $<$ 13 years was near-zero over the time-period studied.[15] Data from the administrative database of tertiary-care pediatric hospitals in the U.S. between 2004 and 2014 showed that 6.4-fold increase in ACL reconstructions relative to all orthopaedic surgeries in patients aged $<$ 10 years.11 Taken together, the diagnosis of ACL tears in young children is increasing, however, ACL reconstructions for them still seem to be performed mainly only in major children's hospitals, probably due to concerns regarding iatrogenic growth disturbance and deformity.

In a recent meta-analysis by Bram et al, they concluded that the rate of ACL injuries per 1,000 athlete-exposure was 0.069 (95% confidence interval [CI], 0.065–0.074), with a higher rate in females (relative risk, 1.40 [95% CI, 1.25–1.5]).[24] Additionally, they found girls' soccer (0.166 [95% CI, 0.146–0.189]) and boys' football (0.101 [95% CI, 0.092–0.111]) had the highest risk. ACL injuries were over 8 (95% CI, 6.46–11.30) and 6 (95% CI, 5.52–8.49) times more likely to happen in competition versus practice settings for females and males, respectively.[24]

Epidemiologic studies on ACL tears in skeletally immature patients prompted injury prevention programs in young athletes.[25-27] A recent study utilizing the New York statewide database showed the decline in ACL reconstruction rate performed on pediatric patients from 61.0 in 2014 to 51.8 by 2017.13 The authors suggested that the success of injury prevention strategies could be a factor of the recent drop in ACL reconstruction rate.[13]

Co-morbidities of ACL Tears

Incidences of acute lateral meniscal, medial meniscal, and chondral injuries in pediatric ACL tears were reported as 28–56%, 12–38%, and 0–26%, respectively (Table 2).[4-7,28-30] However, a delay in treatment increases the incidence and severity of meniscal and chondral injuries, especially of the medial meniscus (Table 2).[4-7,29] Older age and increased patient weight are also known as a risk factor of associated injuries.[2, 4,6,31-33] The most common associated meniscal tear pattern is a vertical tear.[7,30,34] The root tear, which compromises hoop stresses, was identified in 0.8-38% of the medial meniscus and 9% of the lateral meniscus in adolescent ACL tears.[35,34] Samora et al. found that the most common location and zone of tear were the posterior horn and red-white zone.[30] Due to the high incidence rate of ramp lesions (posterior meniscocapsular tear of the medial meniscus) found in almost 25% of pediatric ACL tears, along with the limited effectiveness of MRI and anterior arthroscopic approaches in detecting

Table 1. Incidence of ACL tears in the general pediatric population of the U.S. and other countries

Author (Year)	Incidence per 100,000 Person-years	Age (years)	Country/ State	Period	ACL Tear Definition
Dodwell[12] (2014)	17.6 in 1990 to 50.9 in 2009	3–20	New York	1990–2009	Reconstructions
Collins[14] (2014)	56.5 in 2006 to 62.8 in 2010	<18	Wisconsin	2006–2010	Reconstructions
Beck[16] (2017)	129 in females and 114 in males over the study period	6–18	U.S.	1994–2013	Tears
Herzog[15] (2017)*	Aged <13 years: 0 in both males and females over the study period Aged 13–17 years: 169.0 in 2002 to 268.7 in 2014 in females; 146.8 in 2002 to 211.7 in 2014 in males	<18†	U.S.	2002–2014	Reconstructions
Brodeur[13] (2022)	49.3 in 2009, 61.0 in 2014, and 51.8 in 2017	3–19	New York	2009–2017	Reconstructions
Gianotti[47] (2009)*	Aged 0–9 years: 0 in both males and females over the study period Aged 10–14 years: 10 in both males and females over the study period Aged 15–19 years: 100 in males and 70 in females over the study period	<20†	New Zealand	2000–2005	Reconstructions
Janssen[21] (2012)*	Aged 5–14 years: 5 in both males and females over the study period Aged 15–24 years: 70 in females and 180 in males over the study period	<25†	Australia	2003–2008	Reconstructions
Lopes[19] (2016)	Aged 0–10 years: 0 in both males and females over the study period Aged 11–20 years: 0.6 in 2008 to 1.2 in 2014 in females; 2.1 in 2008 to 4.0 in 2014 in males	<21†	Brazil	2008–2014	Reconstructions
Shaw[18] (2017)	2.74 in 2005/2006 to 6.79 in 2014/2015	5–14	Australia	2005–2015	Hospital-admitted injuries
Weitz[23] (2020)	17.7 in 1999 to 31.5 in 2011	<18	Finland	1997–2014	Hospital-admitted injuries
Longo[20] (2021)	0.16 in 2001 to 2.04 in 2015	<15	Italy	2001–2015	Reconstructions
Chung[22] (2022)*	10 in 2008 to 22 in 2016	<20	South Korea	2008–2016	Reconstructions

*The approximate incidence was inferred from a figure of the study. †Only data of the specified age group was used in this review.

Table 2. The rate of meniscal and chondral injuries of pediatric ACL tears

Author (Year)	Lateral Meniscus	Medial Meniscus	Cartilage	Time to Diagnosis	Age (years)
Millett[29] (2002)	47%	11%	0%	≤6 weeks	≤14
	36%	36%	0%	>6 weeks	
Lawrence[5] (2011)*	40%	15%	Lateral: 10% Medial: 15%	≤3 months	≤14
	45%	45%	Lateral: 45% Medial: 35%	>3 months	
Samora[30] (2011)	56%	29%	15%	<3 months	<17
Dumont[6] (2012)	56%	38%	Lateral tibia: 8% Medial tibia: 2%	≤5 months	<19
	57%	54%	Lateral tibia: 9% Medial tibia: 8%	> 5 months	
Anderson[7] (2015)	47%	24%	7%	≤3 months	<17
	67%	50%	28%	>3 months	
Newman[4] (2015)	42%	17%	Femur: 13% Tibia: 6%	≤3 months	≤19
	55%	40%	Femur: 39% Tibia: 19%	>3 months	
Dawkins[28] (2022)	49%	28%	NA	>3 months	≤18

*The approximate rate was inferred from a figure of the study. NA, not applicable.

these lesions, clinicians may opt to perform arthroscopic inspection through the intercondylar notch and thorough probing to accurately identify the lesion.[36,37]

Various ligaments around the knee are commonly injured during an ACL tear. The rate of concomitant medial collateral ligament (MCL) injuries is 2–37%.[4,29,38-41] In a study by Sankar et al., 12 of 180 adolescents with ACL tears (7%) had concomitant grade 2 or 3 MCL injuries, however, excellent functional outcomes were achieved at a mean follow-up of 5.3 years with use of a hinged brace to treat the MCL injury.[40] Lee et al. reported that older age and contact injuries were associated with concomitant MCL, lateral collateral ligament (LCL), and/or posterior cruciate ligament injuries.[39]

Additionally, concomitant PLC injuries are not uncommon, particularly in patients with a lateral meniscus tear or Segond fracture and in patients with older age.[38,41] Clinicians should remain vigilant regarding the possibility of such injuries. A retrospective review of MRI studies in adolescents with ACL tears identified a posterolateral corner (PLC) injury in 13% of the patients.[41] In another study evaluating the imaging of preadolescent and adolescent patients, MRI revealed PLC injuries in 52% of ACL tears, with 14% of patients having a complete tear of a structure of the PLC.[38]

Furthermore, the use of advanced, cross-sectional imaging may aid in assessing combined anterolateral ligament (ALL) injury because there is no specific physical examination for evaluating the integrity of ALL injury.[42] The rate of abnormality of ALL on MRI was reported as 60–76% in adolescent ACL tears.[43,44] Helito et al. reported that ALL injuries were associated with MCL, LCL, and iliotibial tract abnormalities and bone contusion on MRI.[43] However, due to the limited reliability in ALL detection with pediatric knee MRIs and due to the limited visualization of ALL on MRI in young children, careful history-taking and physical examination persist as an instrumental component of diagnosing patients with ALL injury.[45,46]

Conclusions

The incidence of ACL tears in skeletally immature patients is rising globally. As a significant proportion of patients have associated injuries and the incidence of these injuries increases without treatment, it is recommended that patients seek orthopaedic care as soon as possible.

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Pediatric Tibial Spine Fractures: Current Research and Debates

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Introduction

Tibial spine fractures (TSFs) are avulsion fractures at the tibial intercondylar eminence.[1] They are relatively rare, occurring in about 3 per 100,000 individuals per year.[2] They are more common in males than females and in children than adults. Common mechanisms of injury include contact, hyperextension, and rotational or pivoting injuries with the knee flexed.[3] Commonly reported injury causes include falls from bicycles and injuries during sports.[4]

The most used TSF classification system is that by Meyers and McKeever, which characterizes nondisplaced fractures as Type I, minimally displaced fractures with an intact posterior hinge as Type II, and completely displaced as Type III.[5] Later, Zaircznyj introduced Type IV to describe comminuted fractures.[5,6] Treatment for TSFs can be operative or nonoperative and depends on factors including fracture severity, pain, range of motion, presence of soft tissue entrapment and/or concomitant injuries, and surgeon and patient/family preference. Research regarding the epidemiology, classification, and optimal treatment for TSFs has increased in recent years.

Epidemiology

Two large epidemiological database studies on TSFs were published in 2021.[7,8] In one, U.S. Healthcare Cost and Utilization Project databases were queried for patients aged 7-18 years with TSFs in 2016 in New York, Florida, or Maryland. They found 185 cases, 69.7% male. The incidence peak was 9.3 per 100,000 individuals at age 14 for males and 3.4 per 100,000 individuals at age 9 for females.[7] In the second study, the U.S. Truven Health Marketscan Database was queried for individuals aged 7-18 with TSFs between 2016-2018.8 They found 878 cases with a male to female case burden ratio of 2.2:1.8 In this study, cases peaked at age 13-14 in boys and 11-12 for girls.[8] Both studies demonstrated that TSFs are more common in males than females and that they peak at a later age for males.[7,8]

Classification Systems

In 2021, Ellis et al. assessed the reliability of the Meyers and McKeever classification system. Deidentified radiographs from 40 patients with TSFs were classified on two separate occasions by pediatric sports medicine surgeons, and intra- and interrater reliability was assessed.[9] They found only fair intra- and interrater agreement ($\kappa = 0.47$ and 0.35, 0.33, respectively) when classifying by Meyers and McKeever type.[9]

Additionally, an alternative classification system was proposed by Green et al. in 2018 which makes use of MRI, rather than radiographs.[10] In this system, non or minimally displaced (< 2 mm) are Grade 1, posterior hinged fractures are Grade 2, and displaced fractures, those that result in meniscus or intermeniscal ligament entrapment, and those that extend to the articular surface are Grade 3. However, this study also showed only fair to moderate inta- and interrater reliability ($\kappa = 0.66$ and 0.34, 0.41, respectively).[10]

Treatment Options

Nonoperative Treatment

There is some debate regarding when nonoperative treatment is indicated for TSFs. An oft-cited convention is that Type 1 fractures are treated nonoperatively, but one multicenter, retrospective cohort study found that 25% (n = 12/48) of patients with reported Type 1 fractures were treated surgically.[11] When the authors reassessed the fracture classification, the number of "true Type 1" fractures was reduced from 48 to 40, but they still found that 15% (n = 6/40) were treated surgically, and 23% (n = 9/40)

had concomitant injuries.[11] Another study by the same group compared outcomes between patients with Type 2 fractures treated operatively (n = 123) and nonoperatively (n = 41).[12] They found that the overall complication and reoperation rates did not differ between groups. However, the nonoperative group had a higher rate of subsequent ACL injury or TSF requiring surgery (4.9% vs. 0.0%), and the operative group had a higher rate of arthrofibrosis (8.9% vs. 0.0%).[12]

A systematic review identified 18 studies regarding the nonoperative treatment of 369 TSFs (n = 173 Type 1, n = 124 Type 2, n = 72 Type 3). The most common complications were persistent stiffness (33.9%) and persistent instability (13.4%). The authors note that few studies stratified complications by fracture grade, but there was a "modestly higher rate" of arthrofibrosis and persistent laxity with higher grade fractures.[13] Taken together, this research suggests that more information is needed to determine when nonoperative treatment should be pursued.

Suture vs. Screw Fixation

In one laboratory study on Yorkshire porcine knees, screw and suture fixation constructs were compared between skeletally mature and immature groups.[14] A standardized TSF was created in 8 mature and 8 immature knees. Then 4 knees in each group underwent fixation with 2 screws, and 4 underwent fixation with a dual-suture technique. The knee specimens then underwent 200 cycles of cyclic loading, followed by load-to-failure testing. In the immature knees, load to failure was not significantly different between groups. In the mature knees, load to failure was 1.9 times higher in the suture group. Displacement and stiffness were also not different between the fixation groups after cyclic loading. The authors' conclusion was that the two fixation constructs appear equivalent in the skeletally immature, and suture fixation may be superior for those skeletally mature.[14]

Open vs. Arthroscopic Fixation

Both open (ORIF) and arthroscopic (ARIF) fixation methods have been used for TSF treatment. In one multicenter, retrospective cohort, 420 patients treated with ARIF and 57 treated with ORIF were compared.[15] They found that ARIF was most often performed by surgeons with sports subspecialty training (85% of cases) and ORIF was more often performed by surgeons without sports subspecialty training (56.1% of cases). Importantly, they found similar rates of short-term complications and return to the operating room between the two groups.[15]

Conclusion

Despite an increase in recent research, there remains no true gold standard for TSF treatment, likely due to their rare nature. Many additional controversies exist as well, including the use of pretreatment MRI, preoperative mobilization, rehabilitation protocols, and the amount of acceptable residual displacement. Prospective research is needed to answer these questions, and CHOP serves as the data coordinating center for a prospective cohort study with 15 other institutions with the objective of beginning to answer these questions.

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U·P·O·J

Tibial Tubercle Osteotomy in the Management of Patellofemoral Instability—A Review of Radiographic Indications at Two Children's Hospitals

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Introduction

Patellofemoral instability (PFI) is a common condition affecting adolescents with an estimated annual incidence up to 77.4 per 100,00 that is highest among females.[1,2] The Tibial Tubercle Osteotomy (TTO) is a corrective surgical technique utilized in management of PFI for medialization and/or distalization of the knee's extensor mechanism using a variety of described techniques.[3] However, due to the potential risk of physeal injury resulting in growth disturbance, its use is largely limited to those at or approaching skeletal maturity.[4] As such, few large adolescent patient series have been reported in the published literature. Traditionally, a Tibial Tubercle-Trochlear Groove (TT-TG) distance greater than 20mm has been utilized as a threshold for indicating the use of TTO in the adult population, but differential anatomic and environmental risk factors for recurrence among adolescents may drive alternative decision-making in younger patients.[5,6] The purpose of this study was to retrospectively review a large cohort of adolescent PFI patients treated with TTO at 2 large pediatric orthopedic surgery centers and examine actual use patient characteristics for this procedure.

Methods

Following institutional review board approval, a retrospective chart and imaging review of adolescent patients with PFI undergoing TTO from 2007-2021 was conducted at 2 large pediatric orthopedic centers. Inclusion criteria identified all adolescent and young adult patients who presented for surgical treatment of PFI and had operative notes available for review. Patients were excluded if they had history of congenital patellar dislocation or had a previous ipsilateral knee surgery, trauma, or other diagnosis that disrupted the natural anatomy of the knee. Demographics such as age, sex, race, and number of preoperative patellar dislocations were recorded. Preoperative knee MRIs were reviewed to evaluate TT-TG distance, Caton-Deschamps index (CDI), as well as Dejour and Oswestry Bristol classifications of trochlear dysplasia. Operative notes were reviewed to determine if TTO was done to achieve anteromedialization of the extensor mechanism in isolation (AM) or if it was combined with distalization (AMD). Descriptive statistics were used to summarize study variables while bivariate analysis were used to compare traditional surgical indications.

Results

A total of 125 patients (74.8% female) with a mean age of 16.25 years were identified. The median number of patellar dislocations prior to surgery was 3. Anteromedialization (AM) was done in 75 patients while anteromedialization plus distalization (AMD) was done in 50 patients. MPFL reconstruction was performed concurrently in 115 patients (92%) with the remaining 10 (8%) having their reconstruction as a secondary, staged procedure. The mean preoperative TT-TG distance and CDI for the entire cohort were 18.66 mm and 1.33, respectively. Patella alta, defined as a CDI > 1.3, was present in 43 patients (34%). Trochlear dysplasia was prevalent (93.1%) in our cohort with 21% having Dejour D morphology and 31% having OBC Severe dysplasia. TT-TG values were similar ($p > 0.05$) across Dejour and Oswestry-Bristol Classifications and among those with and without patella alta (CDI > 1.3). Significant differences in patellar height were appreciated between AM and AMD groups with a mean CDI of 1.29 and 1.31 for AM and AMD groups respectively ($p = 0.023$). Cohort characteristics are further summarized in Table 1.

Discussion

A lateralized extensor mechanism insertion of the tibia and patella alta are two of the leading modifiable risk factors for recurrent PFI.[6-9] This is one of few studies examining

Table 1. TTO Demographics and Radiographic Summary (N = 125)

Demographic or Radiographic Parameter	Value
Mean Age (SD)	16.25 (2.51)
Sex	
Male	32 (25.2%)
Female	93 (74.8%)
Race	
White	94 (74%)
Black	17 (13.4%)
Asian	6 (4.7%)
Hispanic	2 (1.6%)
Other	8 (6.3%)
Number of Dislocations Prior to Surgery (IQR)	3 (2, 5)
Median Length of Follow Up in Months (IQR)	13.92 (7.56, 28.92)
TTO Type	
AM	75 (60%)
AMD	50 (40%)
TT-TG distance in millimeters (SD)	18.66 (4.29)
Caton-Deschamps index (SD)	1.33 (0.20)
Dejour Classification	
Normal	8 (6.9%)
A	27 (23%)
B	35 (27%)
C	24 (23%)
D	22 (21%)
Oswestry-Bristol Classification	
Normal	8 (6.9%)
Mild	31 (26.7%)
Moderate	41 (35.3%)
Severe	36 (31%)
MPFL Reconstruction	
Concurrent	115 (92%)
Staged	10 (8%)

TTO utilization in the management of PFI in a majority adolescent population. Our study found mean TT-TG distances to be less than the 20mm threshold often cited as an indication for TTO, with no identified differences between trochlear dysplasia classification groups or between patients with and without patella alta.

Normative data in adults suggests that TT-TG distances over 20 mm may indicate surgical correction in symptomatic patients.[5] However, like many radiographic measures, values in adult populations are not directly applicable to pediatric patients who may have anatomic differences due to remaining growth or who may exhibit different risk factors than their adult counterparts. Dickens et al. validated TT-TG using MRI in skeletally immature patients and developed a percentile-based growth curve showing that TT-TG gradually increases with age until reaching normal adult values by 15 years of age.[10] While the aforementioned growth curves may not have been utilized for surgical decision making, it is possible that symptomatic treatment via TTO corresponded to the upper percentiles of TT-TG distances in this largely adolescent population. Previous adult-based studies with similar trochlear dysplasia and patella alta rates demonstrated that combined MPFL reconstruction and TTO is an effective alternative option for the treatment of PFI with satisfactory patient outcomes.[11] Given that greater than 60% of this study's cohort had moderate to severe trochlear dysplasia suggests a complex interaction exists between anatomic risk factors and shows surgical decision making is not based on one singular measure.

Due to its retrospective nature, this study has inherent limitations. Surgical indications were at the discretion of the treating surgeon, each of whom may have integrated other clinical and radiographic factors into their decision-making for TTO use. While TT-TG, CDI, and trochlear dysplasia classification are the most commonly cited factors impacting surgical decision-making for PFI, the application of such measures is varied across surgeons and institutions with many surgeons relying on their experience and comfort level when pursuing surgical intervention.[12,13] Novel methods of measuring patellar lateralization and trochlear morphology were not included in this study, but may have impacted surgical decision making in this cohort.[6,14,15]

Conclusion

In this large, multicenter cohort of adolescents and young adults undergoing TTO, the mean preoperative TT-TG distance was observed to be less than traditional indications of 20mm. TT-TG distances were not significantly different between different trochlear dysplasia classifications or patellar heights implying a multidimensional decision-making process. Further work is needed to determine the patient characteristics and other radiographic factors contributing to surgeons utilizing a lower radiographic threshold for TTO in adolescents through comparison with patients treated with MPFL reconstruction alone.

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Bone and Development

Injectable Radiopaque Hyaluronic Acid Granular Hydrogels for Intervertebral Disc Repair

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Intorduction

Injectable hydrogels have the potential to increase minimally-invasive treatment options for degenerative disc disease, a condition that affects millions of Americans each year.[1] However, many hydrogels for intervertebral disc (IVD) repair suffer from weak mechanical integrity, expulsion from the injection site, and gel migration within the disc.1 To overcome these limitations, we developed an injectable hyaluronic acid granular hydrogel with encapsulated radiopaque zirconium oxide nano-powder for IVD repair. The granular structure imparts both injectability and enhanced biomaterial integrity when compared to previously investigated polymer solutions, while the radiopacity allows for direct visualization of gel injection and retention at the injection site. The purpose of this study was to characterize the material properties of the radiopaque granular hydrogel, determine its capacity to restore disc mechanical properties ex vivo, and demonstrate feasibility of in vivo minimally invasive delivery.

Methods

Norbornene-modified hyaluronic acid (NorHA. 2 wt.%, 25% degree of modification of norbornene groups), dithiothreitol (DTT, 6mM), zirconium oxide nano-powder (ZrO_2, 30 wt.%), ammonium persulfate (APS, 10 mM), and tetramethylethylenediamine (TEMED, 10mM) were thoroughly mixed in phosphate-buffered saline (PBS) and allowed to form a bulk hydrogel over 30 min. The bulk hydrogel was then fragmented by extrusion fragmentation to obtain microgels.[2] Microgels were then jammed by vacuum-driven filtration to form an injectable radiopaque granular hydrogel (Figure 1a). Extrusion forces were measured using a custom-built setup consisting of an Arduino Uni Rev 3, round force-sensitive resistor (Interlink), and

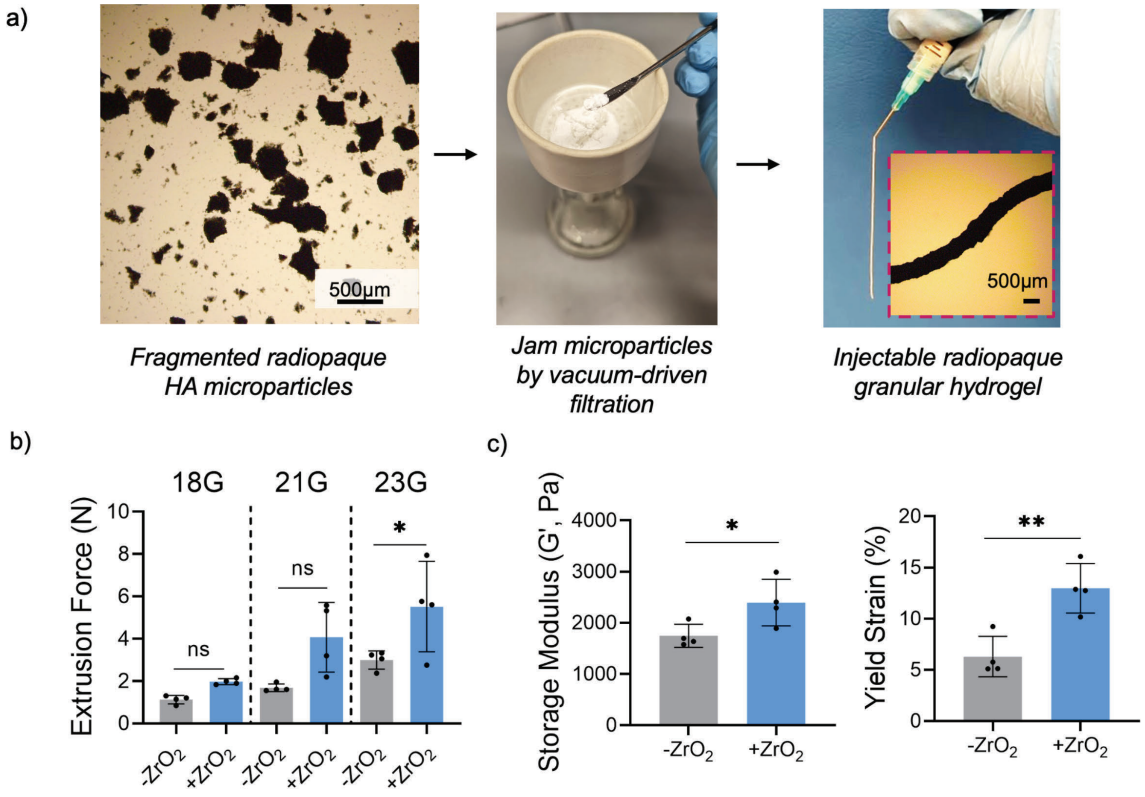

Figure 1. Radiopaque granular hydrogel **(A)** fabrication; **(B)** extrusion force; **(C)** mechanical characterization.

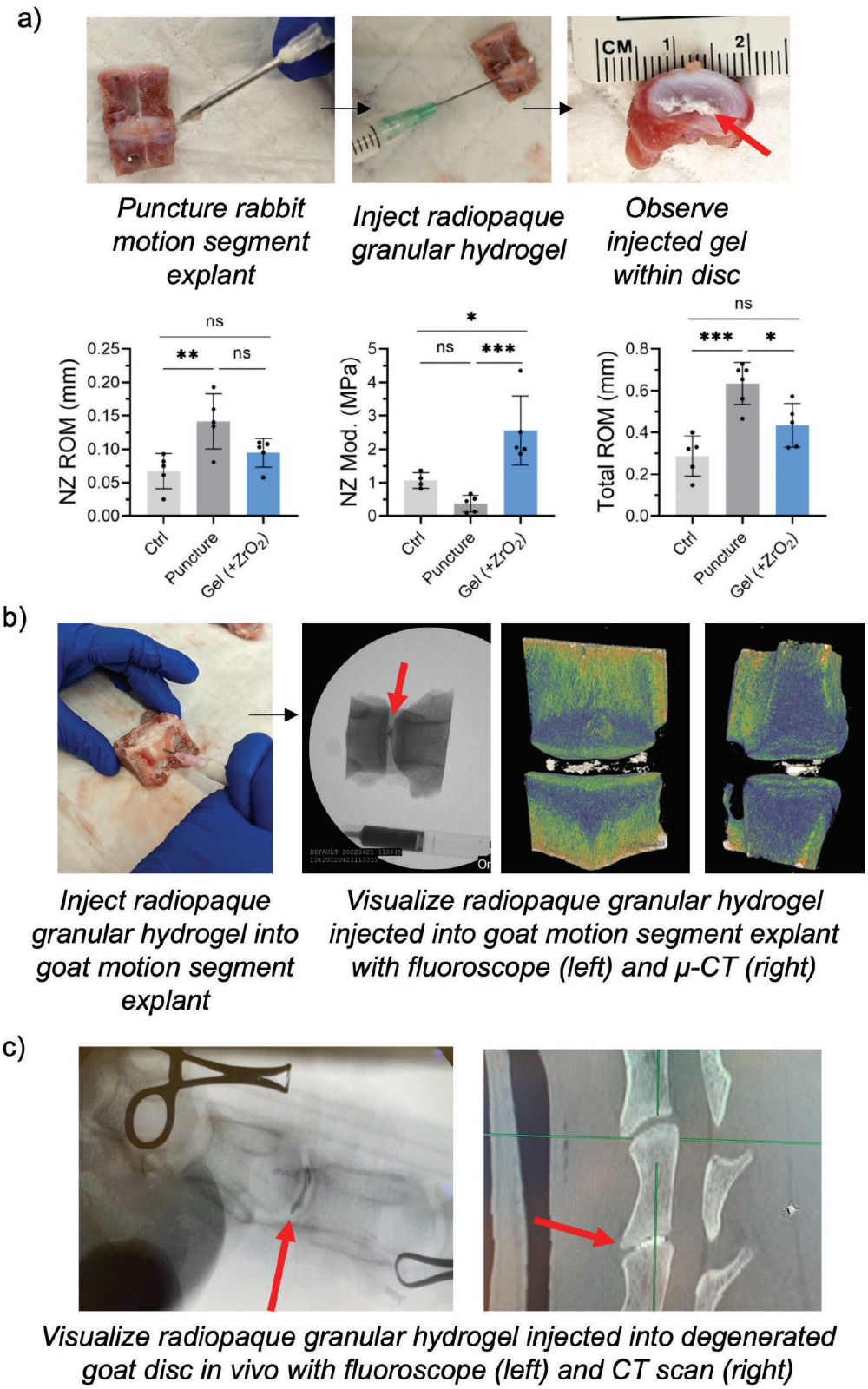

Figure 2. Radiopaque granular hydrogel evaluation for **(A)** restoration of mechanics, **(B)** imaging ex vivo and **(C)** imaging in vivo.

syringe pump.[3] Rheological properties were assessed using an oscillatory shear rheometer (AR2000, TA Instruments). Storage modulus (G') and yield strain were determined from oscillatory shear time sweeps (1% strain, 1 Hz). To mimic the loss of disc mechanical function associated with disc degeneration ex vivo, rabbit lumbar spinal motion segments were punctured with a 16G needle, and nucleus pulposus tissue was removed (Figure 2a). Radiopaque gel was injected into punctured rabbit motion segment explants using a 21G needle, and compressive mechanical properties of disc explants (i.e., neutral zone range of motion [NZ ROM], neutral zone modulus [NZ Mod.], and

total range of motion [Total ROM]) were determined using mechanical testing methods previously described.[4] To confirm adequate visualization on radiographic imaging, radiopaque gel was injected ex vivo into goat cervical motion segments using an 18G needle (Figure 2b). The goat explant was imaged using a fluoroscope as well as µCT scan to visualize the radiopaque gel within the disc. Finally, as an in vivo proof of concept, following IACUC approval disc degeneration was induced in one goat at the C4-C5 level of the cervical spine via injection of 200µL of 2U ChABC, as previously described.[5] 14 weeks following ChABC injection, a second procedure was performed to inject 500µL of the radiopaque granular hydrogel percutaneously into the C4-C5 disc space under fluoroscopic guidance using an 18G spinal needle (Figure 2c). Data is presented as mean ± standard deviation, unless otherwise indicated. Statistical analysis was conducted in GraphPad Prism 8 using ANOVA and a Tukey's post hoc comparison. For all quantified samples, $n \geq 3$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$, ns = not significant.

Results

ZrO_2 nano-powder was encapsulated within fragmented hyaluronic acid microgels (average ~100 µm diameter), which were then subsequently assembled into a jammed state to form a microporous injectable radiopaque granular hydrogel (Figure 1a). The inclusion of zirconia oxide nano-powder significantly increased the extrusion force of the granular gel through 21G and 23G needles, but not 18G needles (Figure 1b). The radiopaque granular gel also had a significantly higher storage modulus and yield strain compared to the granular gel without the encapsulated zirconia oxide (Figure 1c). In ex vivo mechanical studies, punctured rabbit motion segments (mimicking degenerative conditions) exhibited increased neutral zone and total range of motion as well as decreased neutral zone compressive moduli (Figure 2a). Upon ex vivo injection of the radiopaque granular hydrogel, both the neutral zone and total range of motion, as well as neutral zone modulus, were returned to healthy (control) levels (Figure 2a). Due to the high encapsulated ZrO2 content, the injected radiopaque granular hydrogel could be visualized within the goat disc ex vivo as seen by both fluoroscopy and µ-CT imaging (Figure 2b). The in vivo proof of concept study demonstrated that the radiopaque granular gel could be successfully delivered to a degenerative disc in a minimally invasive fashion under fluoroscopic control and visualized on post-operative CT imaging (Figure 2c).

Discussion

In this work we developed an injectable hyaluronic acid granular hydrogel for IVD repair. The injectable gel restored disc mechanics in punctured rabbit discs ex vivo. Though limited to an n = 1 proof-of-concept study, the injectable hydrogel was injectable percutaneously and visible through clinically-relevant imaging in a degenerated disc goat in vivo model. Future directions will include long-term in vivo outcomes in a goat model such as restoration of disc height, mechanics, and MRI signal after hydrogel injection.

Significance

The radiopaque injectable granular hydrogel explored in this study has the potential to advance injectable hydrogel biomaterials for minimally-invasive degenerative disc disease treatment.

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Functionally Graded Lattices in Additively Manufactured Rib Implants Result in Similar Biomechanics

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Introductions

Rib fractures are difficult to treat surgically due to large variability in bone size, curvature, and mechanical properties of the natural bone. Titanium alloy (Ti6Al4V) rib implants are used when open reduction internal fixation is required for reconstruction. Undesirable clinical outcomes such as chest tightness, component loosening, and secondary fracture at the ends of implants are often reported, with revision surgery required in up to 15% of patients.[1] Rib implants may be improved with 3D printing or additive manufacturing (AM), because this process allows for creation of patient-specific geometries and tuning of mechanical properties of implants. In a previous study, we printed 100 x 10 x 1.5 mm Ti6Al4V beams, and decreased bending stiffness up to 15% by uniformly changing the internal lattice structure.[2] Decreasing the mechanical stiffness towards the ends of rib implants may reduce incidence of secondary fracture, but interfragmentary fixation strength cannot be compromised. In the current study, we sought to characterize the biomechanics of functionally graded lattice designs by testing implants in a biofidelic synthetic bone model. We hypothesized that, by making gradient-based changes in internal lattice architecture, we could maintain requisite construct rigidity at the fracture line and decrease the likelihood of secondary fracture at implant ends.

Methods

This study included 6 implant designs (n = 6), with 5 custom AM implants, and a traditionally manufactured titanium control group (MatrixRib, DePuy Synthes, Figure 1A). Custom implants were manufactured with variable rectilinear infill patterns between the 0.5 mm floors and roofs of the plates. Groups included a sparse infill group, 3 functionally graded infill groups (Gradients 1, 2, and 3) and a solid infill group. Custom implants had a similar width (10 mm), thickness (1.5 mm), and length (70 mm) as control implants, but did not include notches to allow for maximization of infill volume. All custom implants were additively manufactured from Ti-6Al-4V powder via laser-powder bed fusion and heat-treated using standard processing conditions. Custom 4th generation Sawbones were designed and manufactured to represent a transverse fracture at the apex of curvature on a 50th percentile male's 4th rib on the left side. Components were implanted with six 2.7 mm diameter nonlocking cortical screws by fellowship-trained orthopaedic surgeons. To simulate post-operative respiration, ribs were compressed in a two-point bend test to generate flexural moments between 0 – 0.5 Nm at the fracture line. Tests were run at 3 Hz for 360,000 cycles to simulate 17.5 days of post-operative breathing (Figure 1B).[3] 3D motion capture was performed at 100 Hz to measure 6 degrees of freedom of interfragmentary motion during cyclic loading. Following cyclic testing, the plates were subjected to ramp-to-failure tests at a rate of 1 mm/s and failure mechanisms were recorded. Differences in cyclic stiffness, 3D interfragmentary motions, ramp-to-failure stiffness, maximum load, and work to failure were tested for significance using one-way ANOVA tests with post-hoc Holm-Sidak pairwise comparisons.

Results

During cyclic testing the Control group demonstrated significantly decreased construct stiffness, as measured by the load cell (0.76 ± 0.28 N/mm), compared to all AM implant designs (means between 1.35 – 1.61 N/mm) (Figure 2A). 3D motion capture indicated that the Control group bone fragments rotated significantly more about the bending axis (2.7 ± 1.3 deg) than the other

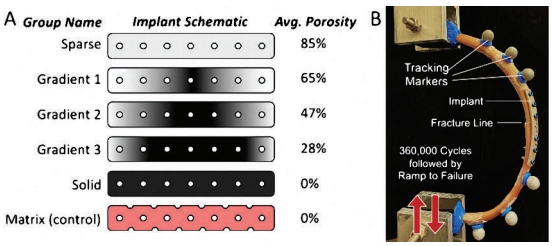

Figure 1. (A) Schematic diagram outlining the functionally graded implant designs tested in the experiment, along with average porosities of the infill for each design; **(B)** Photograph of a synthetic rib undergoing 2-point bend testing. 3D motion tracking markers were used to quantify interfragmentary motions.

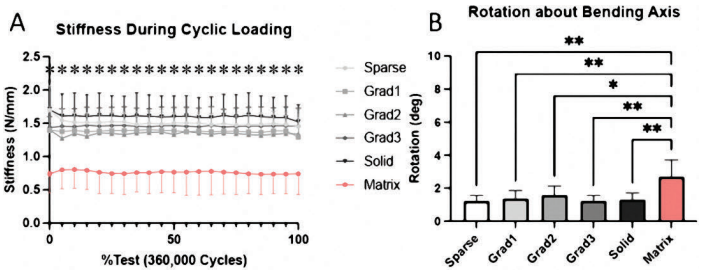

Figure 2. (A) Load-cell-measured stiffness throughout cyclic loading, with significant differences between the control (pink) and experimental (gray) groups. **(B)** Mean interfragmentary rotations about the bending axis during cyclic loading. * = $p < 0.05$; ** = $p < 0.001$.

groups (means between 1.2 – 1.6 deg) (Figure 2B). There were no significant differences between groups for the other 5 degrees of freedom. Ramp to failure testing showed that Control group bending stiffness (0.84 ± 0.28 N/mm) was significantly lower than the stiffness of the Sparse group (1.40 ± 0.07 N/mm), but not other groups (Figure 3A). Ultimate force values were highest for the Gradient 2 group (35.8 ± 5.6 N, which was significantly different from the Gradient 1 (25.8 ± 4.2 N) and Control (25.9 ± 2.7 N) groups (Figure 3B). There were no significant differences between groups for work prior to failure. All constructs failed via bone fracture at the most posterior screw hole.

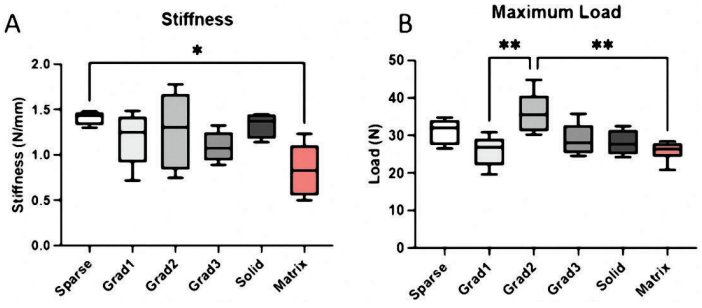

Figure 2. (A) Load-cell-measured stiffness throughout cyclic loading, with significant differences between the control (pink) and experimental (gray) groups. **(B)** Mean interfragmentary rotations about the bending axis during cyclic loading. * = $p < 0.05$; ** = $p < 0.001$.

Discussion

Results from this study only prove a portion our initial hypothesis. Using Control implant behavior as a gold-standard, our implant designs were largely successful in controlling interfragmentary motion and showed no signs of permanent deformation during the cyclic load test. The lack of notches in AM designs likely caused increased resistance to bending. Interestingly, changes in functionally graded lattice architecture did not lead to many significant differences in AM implant performance throughout the experiment. Our designs did not result in predictable stepwise changes to stiffness or maximum load, and functional grading of the lattice did not change the failure mechanism, which was the same for all test specimens. These results are likely confounded by the thin cross-sectional area of the implants and small volume of bone available for screw purchase. Additional studies are ongoing to map changes in mechanical behavior of functionally graded implants.

Significance/Clinical Relevance

Additive manufactured orthopaedic implants may benefit from the use of functionally graded lattice structures. In the case of rib implants, it is likely that the screw hole—not implant stiffness—creates the stress riser responsible for secondary fractures at implant ends.

Acknowledgements

This study was supported by the Manufacturing Pennsylvania Innovation Program and NIH/NIAMS P30AR069619. DePuy Synthes provided materials and technical support.

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Cartilage, Meniscus and Disc

Nanoparticles 'Clicked' onto Nanofibrous Scaffolds for Meniscal Repair

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Click chemistry is a class of reactions wherein molecular pieces are easily joined together, like buckling two pieces of a seat belt.[1] In the context of tissue engineering, click chemistry can serve to fabricate innovative delivery mechanisms capable of regenerating damaged or diseased tissues via spatiotemporal-controlled release. Dense connective tissues, such as knee menisci, sustain demanding load-bearing functions via a complex arrangement of extracellular matrix proteins that surrounds and protects tissue specific cell types. Following injury, endogenous cells within these tissues are unable to promote tissue regeneration and instead form less functional scar tissue. Thus, therapies that activate and recruit endogenous cells to promote tissue regeneration may significantly improve treatment and quality of life for millions of patients annually. Click chemistry-based delivery systems present a novel way of achieving these therapeutics. We previously showed that activation of Hedgehog signaling via a small molecule agonist, Purmorphamine (Pur), accelerated wound closure and attenuated cartilage erosion that results from meniscus injury.[2] Here, we overcome challenges to Pur delivery, such as rapid clearance and off-target toxicity, via delivery from polymeric nanoparticles (NPs) localized to the injury site by immobilizing NPs on a nanofibrous repair scaffold via click chemistry.

Rather than employing copper-catalyzed azide-alkyne cycloaddition as done by Lancuski et. al.[3], we utilized strain-promoted azide-alkyne cycloaddition (SPAAC) to functionalize electrospun polycaprolactone (PCL) nanofibrous scaffolds with azide groups that click with PEG-PCL NPs (prepared via oil-in-water emulsion) containing the alkyne counterpart (DBCO). These azide-modified scaffolds, when compared to electropsun PCL controls lacking azide groups, selectively reacted with DBCO-conjugated fluorophores, validating the successful fabrication of a 'clickable' scaffold. Scratch assays using murine and porcine meniscus fibrochondrocytes revealed that Pur-loaded, DBCO-conjugated NPs increased the migration of meniscus fibrochondrocytes compared to free Pur delivery. We then implanted NP-conjugated scaffolds in vivo using a nude rat xenotransplant model. For this, adult porcine meniscal explants were incised to create a horizontal defect that was either left unfilled or filled with NP-conjugated scaffolds (Pur-loaded and empty). All groups were evaluated at 3 and 14 days and analyzed for cell invasion via staining of nuclei and matrix deposition with H&E. We observed increased cell infiltration from day 3 to 14. More notably, there was a marked increase in cell number with implantation of Pur-loaded NPs compared to empty controls.

This study validates a new approach to produce nanofibrous azide-functionalized PCL scaffolds via SPAAC click chemistry. The reaction between azide-modified PCL nanofibers and fluorophore-DBCO was highly specific, fast, repeatable, and stable over the long-term after repeated washes. This indicates that azide-modified PCL scaffolds may be used to immobilize DBCO-modified NPs. When tested in vivo, Pur delivery via NP-conjugated scaffolds accelerated meniscal repair via increased cell infiltration. These data support the novel use of click chemistry-based approaches for sustained and localized delivery of small molecule drugs for dense connective tissue repair. Clinically, this delivery method has the potential to overcome many challenges in musculoskeletal tissue repair by providing sustained and local release. More generally, this study demonstrates the applicability of click chemistry-based mechanisms in biomedical therapeutics.

Acknowledgements

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Muscle, Tendon, and Ligament

U·P·O·J

Inducible Rosa, but not αSMA or Scx, Cre driven excision achieves substantial *Col12a1* knockdown in tendon healing

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Introduction

Tendon healing follows a typical wound healing process involving transient and heterogeneous cell populations. Collagen XII, a fibril-associated collagen, regulates tendon cell and matrix organization,[1] and *Col12a1* expression increases post-injury. Tamoxifen-inducible Cre mouse models permit spatial and temporal knockdown, and these models are advantageous for investigating the specific role of collagen XII in de novo tissue formation following injury. However, these models may also target other intrinsic or extrinsic cell populations that do not contribute to the healing response, such as vascular cells and cells in the adjacent tissue, and the efficiency of different spatial Cre drivers for collagen XII knockdown are unknown. Therefore, the objective of this study was to evaluate the efficiency of *Col12a1* knockdown in the healing tissue versus native tendon using three tamoxifen-inducible Cre mouse models (1) Rosa-CreERT2 model to ubiquitously target cells contributing to the healing response; 2) αSMA-CreER^{T2} model to target peritenon-derived progenitor cells that infiltrate into the injury; and 3) Scx-CreER^{T2} to target tendon-derived cells) and two tamoxifen dosage protocols (short- and long-dose). We hypothesized that 1) the Rosa-CreER^{T2} model would result in the greatest knockdown independent of region, 2) the αSMA-CreER^{T2} model would exhibit increased knockdown in the healing tissue compared to the native tissue, and 3) the Scx-CreER^{T2} model would show greater knockdown in the healing tissue with the long-dose tamoxifen protocol.

Methods

Male Rosa-CreER^{T2};*Col12a1*$^{flox/flox}$ (RosaKO), αSMA-CreER^{T2};*Col12a1*$^{flox/flox}$ (αSMAKO), and Scx-CreER^{T2};*Col12a1*$^{flox/flox}$ (ScxKO) mice with their respective Cre- littermate controls were used (n = 8/group, IACUC approved). At 90 days old, mice underwent bilateral patellar tendon injury surgery as described,[2] and Cre excision of the conditional alleles was induced via four IP injections of tamoxifen (100mg/kg body weight). The short-dose group received tamoxifen at days −1, 0, 1, and 2 days post-injury, and the long-dose group received tamoxifen at −3, 0, 3, and 6 days post-injury, where day 0 is the day of surgery. Mice were sacrificed two weeks later, and left knees were fixed for three hours in 4% paraformaldehyde prior to cryo-embedding. Injured patellar tendons were sectioned axially at a thickness of 40μm, and sections were microdissected using a 25G needle to ensure isolation of the healing tissue and the adjacent, native tendon struts. The tissue was digested, RNA was isolated as described,[3] and qPCR was performed for *Col12a1* expression. ΔCt values were normalized to the housekeeper gene, *Abl1*.

Statistics

Two-way ANOVAs for genotype and tamoxifen dosage protocol were conducted within each region, and paired t-tests were conducted to compare *Col12a1* expression in the native and healing regions. Significance was set at $p<0.05$.

Results

Supporting our hypothesis, the RosaKO model demonstrated the highest knockdown compared to control regardless of region and tamoxifen dosage protocol with substantial decreases in *Col12a1* expression (~117-2,200-fold decrease, Figure 1A). The short-dose protocol in RosaKO mice also resulted in greater knockdown compared to the long-dose protocol in both the native (~22-fold decrease) and healing (~5.5-fold decrease) regions. Contrary to our hypothesis, *Col12a1* expression surprisingly increased in the healing region of αSMAKO mice with the short dose protocol (~2.5-fold, Figure 1B), and no differences between ScxKO and control mice were observed in any region or tamoxifen dosage protocol (Figure 1C). However, expression was increased in the long dose group in the healing region of ScxKO compared to the short dose group. Finally, *Col12a1* expression was elevated across several groups in the healing region compared to its respective native region.

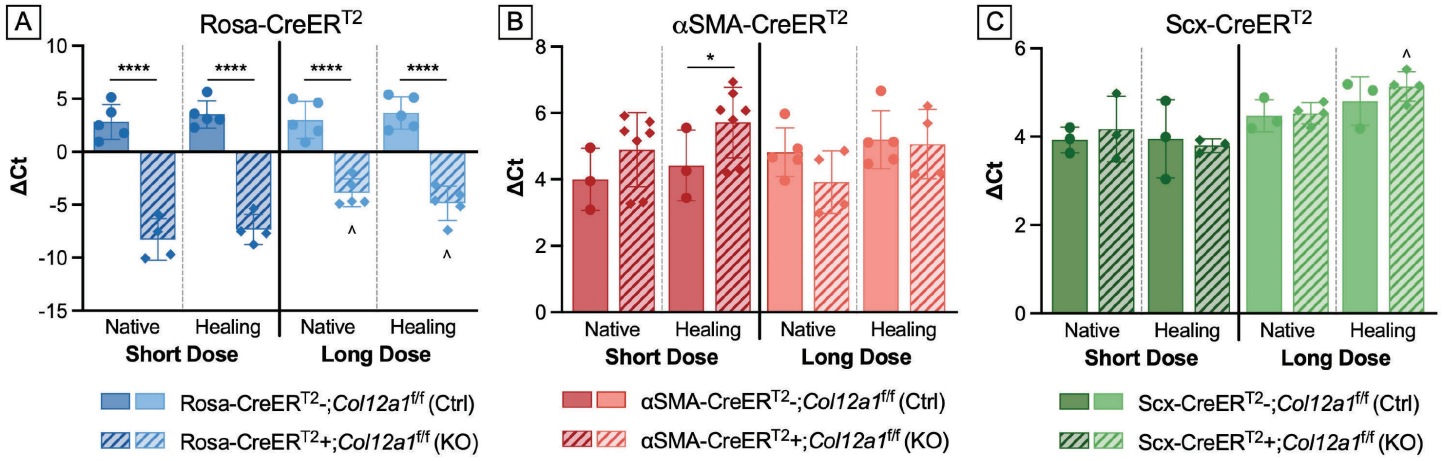

Figure 1. (A) The Rosa model achieved dramatically reduced expression of *Col12a1* expression in both the native and healing regions in mice administered the short and long dose tamoxifen protocols. The short dose groups also demonstrated greater knockdown compared to their respective long dose group; **(B)** In the αSMA model, *Col12a1* expression was surprisingly increased in the short dose, healing region of KO mice, while no other differences were observed; **(C)** In the Scx model, *Col12a1* expression was higher in the long dose, healing region of KO mice compared to its respective short dose group. (*$p < 0.05$, ****$p < 0.0001$, ^$p < 0.05$ compared to short dose protocol)

Discussion

During patellar tendon healing, infiltrating peritenon-derived αSMA+ cells are the primary contributors to the healing response prior to differentiating into Scx+ cells by two weeks post-injury.[4] Our previous data showed that *Col12a1* expression is increased one-week post-injury and returns to uninjured levels by six-weeks post-injury. Therefore, we expected that targeting *Col12a1* knockdown to αSMA+ cells during early healing would result in knockdown within the healing, de novo tissue. However, no knockdown was demonstrated in the αSMA model, even when tamoxifen was administered through 6 days post-injury, and this suggests that this Cre driver may not be sufficient in this model. Similarly, while no knockdown in the healing region of the ScxKO model may be attributed to the timing of Scx expression during tendon healing, there was no significant knockdown in the native region either, and this model also may not be effective in *Col12a1* knockdown. Additionally, both short and long-dose protocols were tested to consider the metabolism rate of tamoxifen in maximizing knockdown to the healing tissue, and in the RosaKO model, greater *Col12a1* knockdown with the short dose protocol indicates that administering tamoxifen on consecutive days before and after injury is more efficient. Finally, as expected, we observed that *Col12a1* expression was generally greater in the healing tissue than the native region, suggesting that collagen XII plays a critical role in tendon healing. Future studies will investigate this role by inducing knockdown of *Col12a1* at the time of tendon injury in a Rosa-CreER^{T2} model.

Significance

Compared to αSMA-CreER^{T2} and Scx-CreER^{T2}, the Rosa-CreER^{T2} model achieves substantial *Col12a1* knockdown when induced at time of tendon injury. Our results highlight the importance of carefully considering an appropriate tamoxifen-inducible Cre model when targeting specific genes.

Acknowledgements

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Scleraxis Targeted Deletion of Collagen XI Impairs Tendon Mechanical Function During Postnatal Development

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Introduction

Mutations in the *Col11a1* gene are implicated in Type II Stickler Syndrome and result in joint problems including hypermobility and early arthritis.[1] Beyond its role in Stickler Syndrome, collagen XI is highly expressed during tendon postnatal development and interacts with collagen I and II during heterotypic fibril formation.[2] We previously showed that during patellar tendon postnatal development, tendon-targeted (ScxCre) collagen XI deficiency disrupts tendon structure, resulting in tenocyte nuclear disorganization, larger diameter collagen fibrils, and increased tendon length.[3] However, whether these structural findings result in alterations in tendon mechanical function is unknown. Therefore, the objective of this study was to define the role of collagen XI in the acquisition of tendon mechanical function during postnatal development using tendon-targeted collagen XI knockout mice. We hypothesized that tendon-targeted collagen XI knockout would result in inferior tendon mechanical properties.

Methods

Tendon-targeted ScxCre;*Col11a1*$^{flox/flox}$ (KO), ScxCre;*Col11a1*$^{flox/wt}$ (HET), and Cre-littermate control (CTRL) mice were used (IACUC approved). Patellar tendons were harvested at postnatal days (P) 10, 20, and 30 for mechanical testing (n = 12/group). The tibia-patellar tendon-patella complex was dissected, tendon cross-sectional area was measured, and the tibia was potted in polymethylmethacrylate. The patella was gripped with sandpaper (P10 and P20) or clamped directly (P30) using custom fixtures, and tendons were subjected to a protocol consisting of preconditioning, stress relaxations at 3, 4, and 5% strain each followed by a dynamic frequency sweep (0.1-10 Hz), and a ramp to failure at 0.1% strain/s. For each age, genotypes were compared using a one-way ANOVA with Tukey post-hoc tests. Significance was set at $p \leq 0.05$.

Results

Tendon-targeted collagen XI knockout resulted in substantial alterations in patellar tendon mechanical properties. Cross-sectional area (Figure 1A) of KO tendons was greater only at P10, while KO tendons (Figure 1B) were significantly longer than CTRL tendons at all ages, consistent with previous histological findings. Stiffness (Figure 1C) and failure load (Figure 1D) of KO tendons were dramatically reduced compared to CTRL and HET tendons. Material properties of KO tendons showed similar trends, with marked reduction in modulus (Figure 1E), failure stress (Figure 1F), and failure strain (Figure 1G) at all ages compared to CTRL and HET tendons. Dynamic modulus was similarly reduced in KO tendons at all ages (data not shown).

Discussion

Previous work evaluating the role of collagen XI in postnatal development found that tendon-targeted knockout disrupts tendon structure, resulting in tenocyte nuclear disorganization, larger diameter collagen fibrils, and increased tendon length.[3] Results of the present study further elucidate the importance of collagen XI in regulating tendon structure-function during early postnatal development. The mechanical differences in KO mice were particularly striking, with KO patellar tendons demonstrating substantial reductions in modulus of 45%, 78%, and 60% at P10, P20, and P30, respectively. These findings are consistent with previous mechanical findings in mature (day 60) KO tendons[4,5] but the emergence of these stark mechanical differences as early as postnatal day 10 point to a critical and previously undescribed role for collagen XI in the initial establishment of tendon hierarchical structure. In tendon, collagen XI is most highly expressed during embryonic development with minimal expression during adulthood,[2] and therefore, inferior mechanical function in KO tendons may be due to alterations in fibril nucleation and assembly. Additionally, motivation for this study was primarily driven by the observation of significant patellar tendon lengthening and

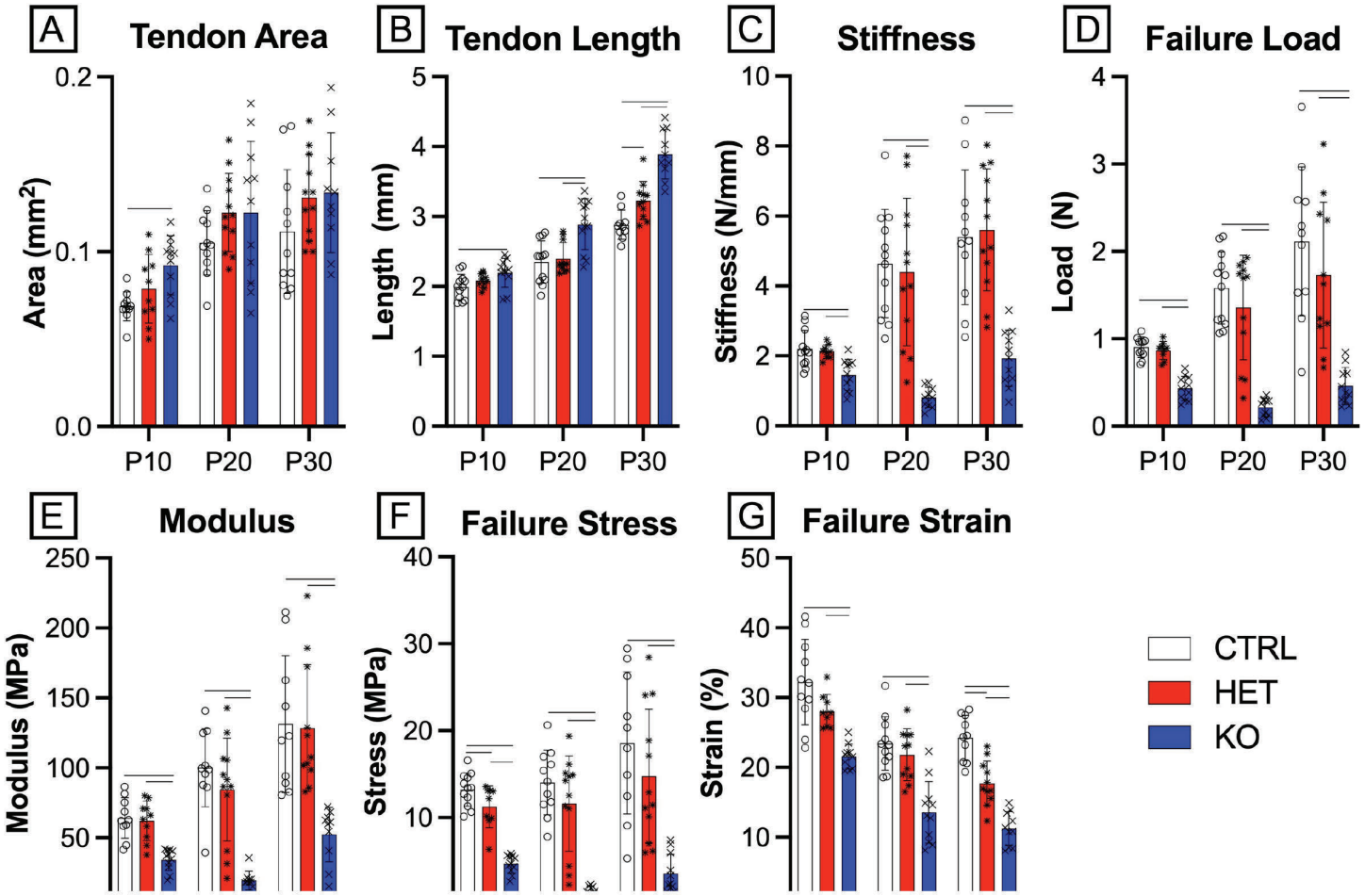

Figure 1. Patellar tendon. **(A)** cross-sectional area was higher in KO tendons at p10; **(B)** tendon length was greater at all ages; **(C)** Stiffness and **(D)** failure load were significantly reduced in KO tendons at all ages compared to CTRL and HET tendons; **(E)** Elastic modulus; **(F)** failure stress; and **(G)** failure strain were significantly reduced in KO mice compared to CTRL and HET tendons at all ages. (—p<0.05)

the presence of granulation tissue in mature tendons. Our findings suggest the absence of collagen XI leads to the deposition of substantially weakened matrix, resulting in injury and elongation following the initiation of ambulation around postnatal day 10. Preliminary gene expression findings at P10 (not shown) support a pathological response with increased expression of genes associated with non-collagenous matrix proteins and remodeling, and studies are ongoing to further elucidate the mechanisms underlying the role of collagen XI in tendon development.

Significance

Collagen XI is critical in the establishment of tendon structure and mechanical function during postnatal tendon development. These findings highlight the need to further define the regulatory role of collagen XI in tendon development and healing, which could lay the foundation for future therapeutic applications.

Acknowledgements

This study was funded by NIH/NIAMS R01AR073231 and the Penn Center for Musculoskeletal Disorders (P30 AR069619).

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Tendon-Targeted Collagen XII Knockout Attenuates Male and Female Achilles Tendon Function Similarly

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Introduction

Collagen XII is a Fibril-Associated Collagen with Interrupted Triple Helices (FACIT) that regulates collagen fibril assembly and is primarily expressed throughout tendon growth and development.[1] Mutations in the *Col12a1* gene result in myopathic Ehlers-Danlos syndrome, a connective tissue disorder in which patients exhibit weakness at birth, absence of deep tendon reflexes and distal joint hypermobility and contracture.[2] Our novel tendon-targeted collagen XII mouse model demonstrated that flexor digitorum longus (FDL) tendons exhibited reduced elastic mechanical properties in both male and female mice.[1] However, the role of collagen XII on the elastic, viscoelastic, and dynamic response to load in the Achilles tendon, which experiences a complex and distinct mechanical loading environment, remains unknown. Therefore, the objectives of this study were to (1) elucidate the role of collagen XII on Achilles tendon mechanics and dynamic response to load using tendon-targeted (Scleraxis-Cre) collagen XII knockout mice and (2) understand whether the role of collagen XII on Achilles tendon biomechanical function and dynamic response to load is comparable between sexes. We hypothesized that tendon-targeted knockout of collagen XII would result in consistent reductions in elastic and viscoelastic function and collagen fiber realignment in both sexes.

Methods

Achilles tendons from male and female, day 30 tendon-targeted collagen XII knockout (KO) mice (ScxCre;*Col12a1*$^{f/f}$, n = 4/group) and wild-type (WT) control mice (Cre-littermates, n = 4/group) (IACUC approved) were subjected to our mechanical testing protocol[3]: stress relaxation testing at 3, 5, and 7% strain each with subsequent dynamic frequency sweeps at 0.1, 1, 5, and 10 Hz, followed by a quasistatic ramp-to-failure. During the ramp-to-failure, dynamic collagen fiber realignment was quantified using cross-polarization imaging,[3] and alignment data was interpolated with a polynomial fit as a function of strain from the load-displacement data. For each sex, comparisons between genotypes were conducted using two-tailed, t-tests with significance set at $p \leq 0.05$ and trends at $p \leq 0.1$.

Results

Cross-sectional area (CSA) was not different across genotypes for both male and female tendons (data not shown). Consistent with our hypothesis, structural properties, maximum load and linear stiffness (Figures 1A, B), and material properties, maximum stress and linear modulus (Figures 1C, D), were significantly decreased in both male and female KO tendons. Further, genotypic differences were observed in viscoelastic properties in both sexes. Percent relaxation was significantly increased in KO tendons at all strain levels (7% strain shown in Figure 2A). Additionally, dynamic modulus was significantly decreased in male and female KO tendons, while phase shift was significantly

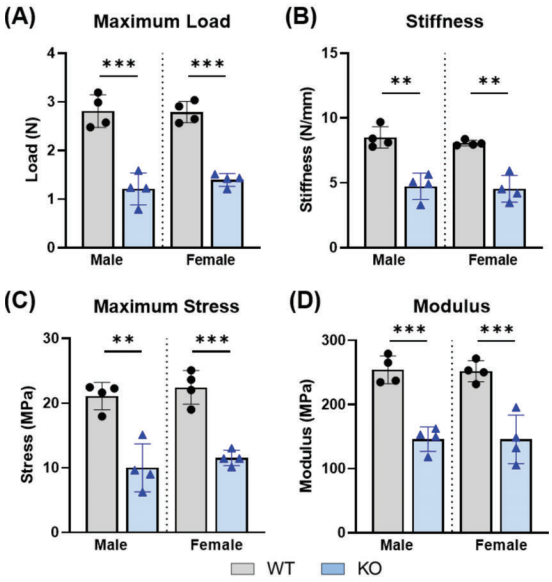

Figure 1. Male and female KO Achilles tendons demonstrated significant reductions in structural (A-B) and material (C-D) properties relative to WT controls. Data as mean ± standard deviation (*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$).

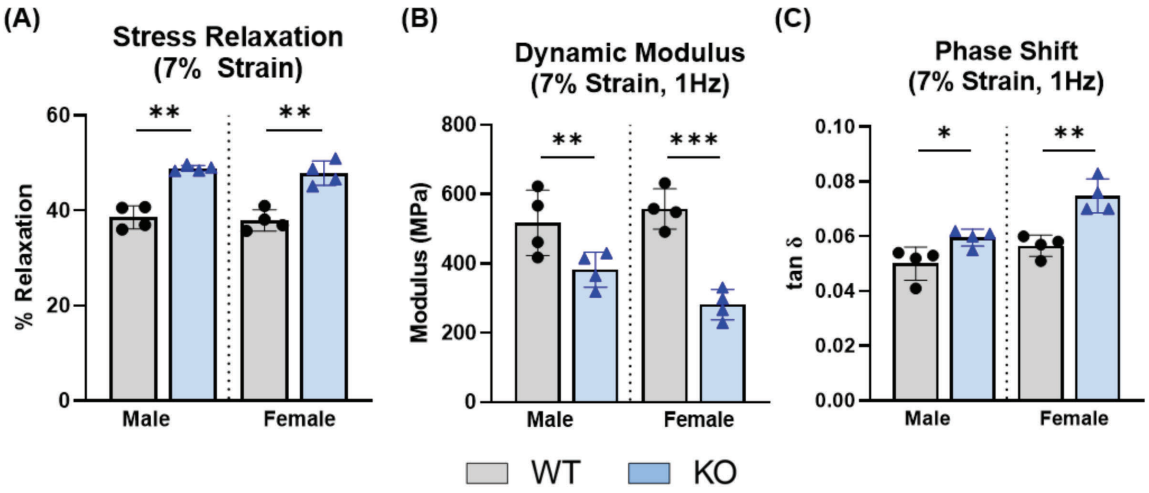

Figure 2. Male and female KO Achilles tendons had increased stress relaxation **(A)** and significantly decreased dynamic modulus **(B)** and increased phase shift **(C)** relative to WT controls. Data as mean ± standard deviation (*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001).

increased in male and female KO tendons across at all strain levels and frequencies (7% strain at 1Hz shown in Figures. 2B and 2C, respectively). These results are supported by reductions in collagen fiber realignment in both male and female KO tendons, as demonstrated by significantly greater normalized circular variance values from 3-9% strain (Figures. 3A, B), encompassing the toe and linear elastic regions of these tendons.

Discussion

This study investigated the role of collagen XII on Achilles tendon elastic and viscoelastic mechanics and dynamic collagen fiber realignment using tendon-targeted male and female ScxCre;*Col12a1*$^{f/f}$ mice. Consistent with previous data from collagen XII knockout FDL tendons [1], we showed that tendon-targeted collagen XII knockout resulted in striking reductions in Achilles tendon elastic and viscoelastic mechanical properties and collagen fiber realignment in both sexes. These mechanical deficits could be attributed to the improper hierarchical assemblies of KO tendons resulting in disorganized tendon matrices with an inferior ability to quasi-statically and dynamically respond to load. This was evidenced by marked reductions in the KO tendons' responses to realign resulting in inferior mechanical properties, especially maximum load, modulus, and dynamic modulus. Although similar differences in elastic and viscoelastic mechanical properties were present in both male and female tendons in response to collagen XII knockout, more genotypic differences were present in female mice. Genetic variations in the *Col12a1* gene have been associated with an increased risk of ACL ruptures in women,[4] implicating potential sex-specific effects of collagen XII knockout. Our results demonstrate that decreased collagen XII expression detrimentally affects male and female Achilles tendon elastic and viscoelastic properties and dynamic collagen fiber realignment in response to load.

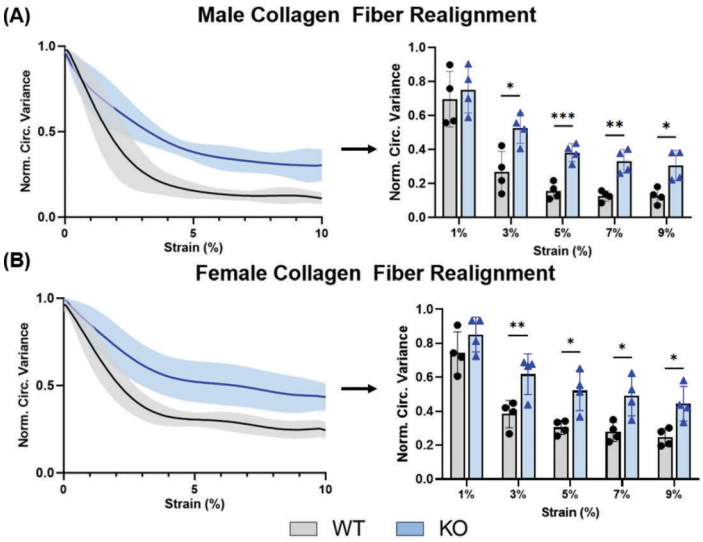

Figure 3. Male **(A)** and female **(B)** KO Achilles tendons exhibited significantly reduced collagen fiber realignment with increasing strain. Decreased normalized circular variance is indicative of increased collagen fiber realignment. Data as mean ± standard deviation (*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001).

Significance

This study further elucidates the role of collagen XII in regulating tendon function. Future studies will evaluate the mechanisms that contribute to these results. Understanding the effects of collagen XII in both male and female tendons can be used to develop potential treatments modalities for myopathic Ehlers-Danlos syndrome.

Acknowledgements

We thank Ashley Fung for her assistance. This study was supported by NIH/NIAMS R01AR078790 and Penn Center for Musculoskeletal Disorders (NIH/NIAMS, P30AR069619).

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Collagen XII Regulates Cell and Matrix Organization and Structure During Postnatal Tendon Development

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Introduction

Collagen XII is a fibril-associated collagen with interrupted triple helices (FACIT), and mutations in the *Col12a1* gene are associated with myopathic Ehlers-Danlos syndrome (mEDS), a connective tissue disorder resulting in symptoms such as joint hypermobility and contractures[1]. Collagen XII interacts with type I collagen to mediate fibrillogenesis and has also been shown to regulate tendon cell organization and the formation of interacting cellular processes[2]. In mature tendon-targeted collagen XII knockout (ScxCre;*Col12a1*$^{flox/flox}$) mice, we previously showed that patellar tendons demonstrated reduced viscoelastic properties and collagen fiber realignment[3], further suggesting a critical role for collagen XII in establishing matrix organization for proper mechanical function. However, whether these mechanical differences in the absence of collagen XII are due to the initial deposition of disorganized matrix or disordered cell organization early in development leading to disorganized matrix is still unknown. Therefore, the objective of this study was to evaluate the role of collagen XII in regulating cell and matrix organization and structure during postnatal tendon development, prior to the establishment of significant matrix deposition. We hypothesized that collagen XII disrupts cell organization, resulting in alterations in tendon structure and organization.

Methods

Postnatal day 10 tendon-targeted collagen XII knockout (KO, ScxCre;*Col12a1*$^{flox/flox}$) and control (CTRL, Cre- littermates) mice were used (IACUC approved). Tendon morphology: Knees (n = 4-6/group) were fixed, paraffin processed, and sectioned in the sagittal plane. Sections were stained with toluidine blue to measure tendon length and quantify cell density. Matrix & cell organization: Knees (n = 5-6/group) were fixed, and patellar tendons (PTs) were dissected from the joint. PTs were blocked and permeabilized, stained with AF 647 phalloidin and Hoechst, and optically cleared using increasing fructose concentrations (20-115% wt/vol) [4]. Z-stacks were acquired (40μm thickness) using a multiphoton microscope to visualize collagen with second harmonic generation (SHG) imaging, actin, and nuclei. Collagen density (SHG intensity), matrix alignment, and nuclear shape (sphericity) were calculated. Matrix alignment was quantified using a fast Fourier algorithm to calculate circular standard deviation of the fiber direction distribution. Fibril structure: PTs (n = 4/group) were fixed, processed, sectioned, stained, and imaged via transmission electron microscopy (TEM) as described[5]. Gene expression: RNA was extracted from PTs (n = 4-6/group), converted to cDNA, pre-amplified, and loaded into a Fluidigm Dynamic Array. Target genes included those of collagens, non-collagenous matrix, remodeling, cell-ECM, and cell markers. Statistics: Nuclear shape and fibril diameter distributions were compared using Kolmogorov-Smirnov tests, and all other parameters were compared using two-tailed, Student's t-tests with significance set at $p \leq 0.05$.

Results

Tendon Morphology: PTs in KO mice were significantly longer than CTRL (Figure 1A-B). Matrix & cell organization: KO tendons demonstrated higher average forward SHG signal (Figure 1C), indicative of greater collagen density, and increased circular standard deviation of fiber directions, signifying greater collagen matrix disorganization in KO tendons (Figure 1D-F). Additionally, cell density was higher in KO tendons (Figure 1G), and nuclei were rounder (Figure 1H). In contrast to CTRL tendons, where actin filaments were arranged parallel with the long-axis of the tendon, actin appeared disordered and less aligned in KO tendons, consistent with matrix disorganization (Figure 1I-J). Irregular cell shape was also observed in TEM tendon cross-sections (Figure 2A). In CTRL tendons, cell protrusions interacted with those of neighboring cells towards establishing defined fibril bundles. In KO tendons, however, cell protrusions were fragmented with abundant fibripositors (white arrows). Fibril structure:

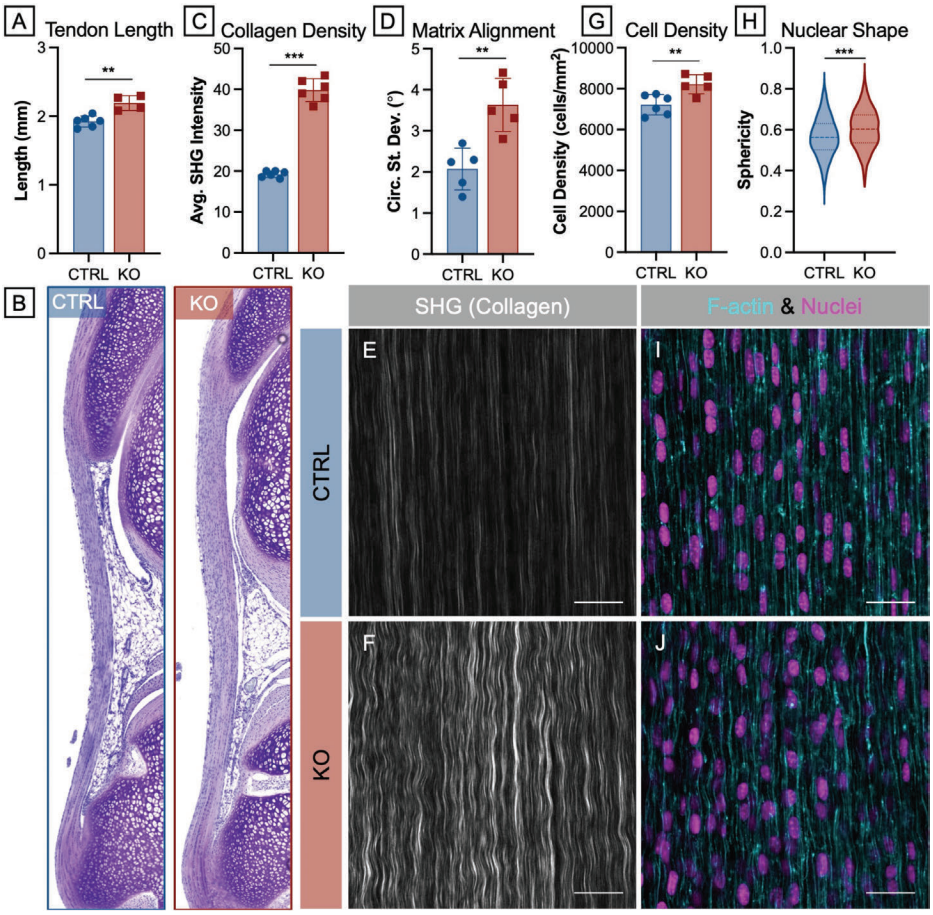

Figure 1. (**A-B**) KO patellar tendons are longer than CTRL. SHG imaging revealed that **(C)** collagen density was higher, and **(D)** the matrix was more disorganized in **(E)** KO tendons compared to **(F)** CTRL. **(G)** Cell density was higher and **(H)** nuclei were rounder in KO tendons. **(I-J)** Actin staining also revealed that cells were less aligned. (**p < 0.01,***p < 0.001, scale bar = 25μm).

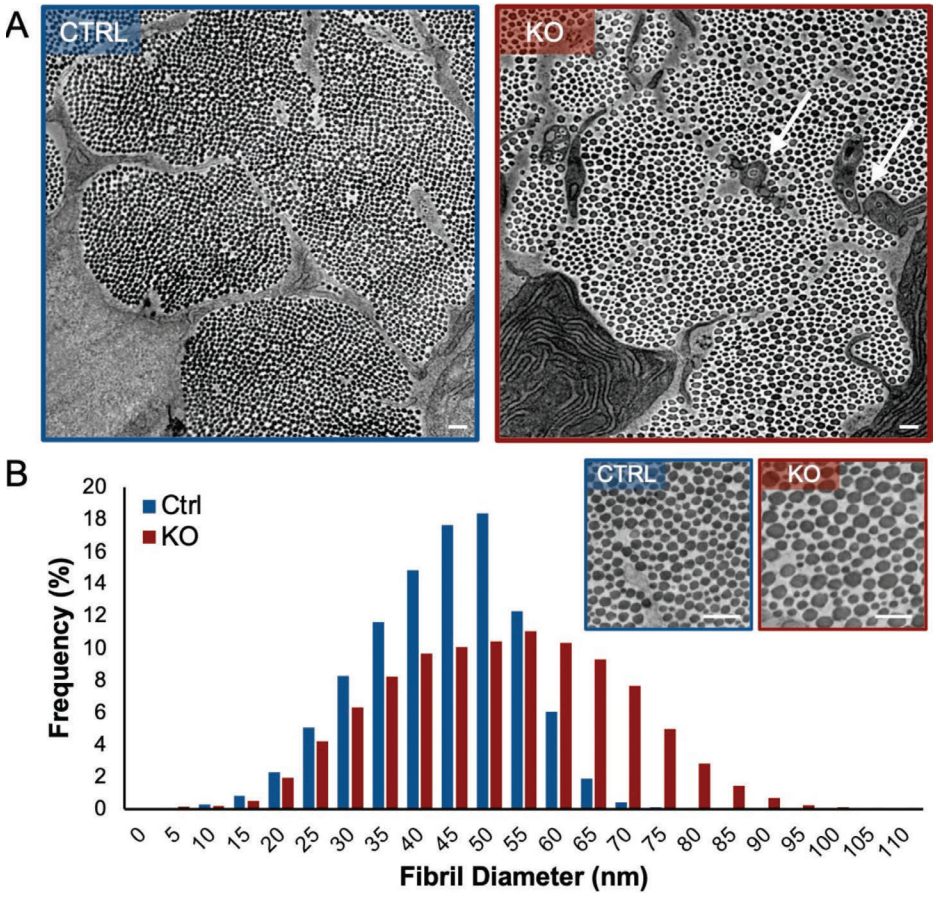

Figure 2. (A) Cell protrusions in CTRL tendons interact with neighboring cells and form fibril bundles, while those in KO tendons are fragmented and contain several fibripositors (white arrows); **(B)** KO tendons have a heterogenous collagen fibril diameter population with a greater percentage of larger fibrils. (scale bar = 200nm).

The collagen fibril diameter distribution in KO tendons was more heterogeneous with a greater percentage of larger diameter fibrils compared to CTRL (Figure 2B). Gene expression: As expected, Col12a1 expression was significantly reduced (Figure 3A), while expression of *Fn1, Mmp2, and Serpine1* (Figure 3B-D) was increased in KO tendons. Expression of tendon-related genes (*Col5a2, Dcn, Bgn, Tnc, Tnmd*) and those associated with cell-cell and cell-matrix interactions (*Cdh11, Cdh2, Cd44, Itgb1*) were also increased in KO tendons (data not shown).

Discussion

During tendon development, proper cell and matrix organization is essential for establishing tendon hierarchical structure and function, and our findings indicate that collagen XII is critical in this process. In the absence of collagen XII, postnatal day 10 patellar tendons have disrupted matrix and cell organization, altered cell and nuclear shape, increased fibril diameter, and increased expression of tendon and cell-matrix related genes.

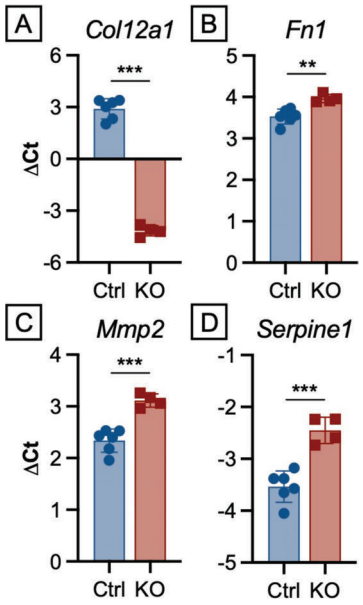

Figure 3. (A) *Col12a1* expression is significantly reduced in KO mice, while expression of **(B)** Fn1; **(C)** *Mmp2*; **(D)** *Serpine1* is increased. (**p < 0.01, ***p < 0.001).

Though findings support previous work in mature mice[2], interestingly, our results during early postnatal growth suggest that alterations in cell organization may precede or result in disorganized matrix deposition. Preliminary findings at postnatal day 30 show similar but less striking alterations in matrix organization and fibril diameter, further supporting a more prominent cell-mediated mechanism during early development. Additionally, increased tendon length, collagen and cell density, and fibril diameter point to a hypertrophic phenotype due to collagen XII knockout. Cornea and skin studies showed that collagen XII may be necessary for storing latent TGF-β, and its absence increased TGF-β activity[6,7]. Gene expression findings in this study support a similar mechanism in tendon, where TGF-β responsive genes such as *Serpine1* are upregulated despite no changes in *Tgfb1*, *2*, or *3* expression. Studies are ongoing to explore this mechanism and further elucidate the role of collagen XII in regulating initial cell organization during embryonic tendon development.

Significance

Collagen XII regulates cell and matrix organization and structure during postnatal tendon development, highlighting its importance in the establishment of tendon hierarchical structure and function.

Acknowledgements

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Focal Adhesion Kinase Regulates Mechanosensitive Gene Transcription and Tendon Maturation

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Introduction

Throughout development and postnatal growth, resident tendon cells respond to mechanical cues from the nascent tendon extracellular matrix (ECM) to regulate tissue properties. Focal adhesion kinase (FAK, gene: *Ptk2*) is an intracellular protein kinase that regulates the actin cytoskeleton and cell-ECM adhesions. In *in vitro* tendon cell culture, FAK inhibition alters cell morphology and attenuates the cell's tenogenic gene expression response to growth factor stimulation and mechanical stretching.[1–4] In addition, FAK inhibitor treatment of explanted tendons attenuates ECM to nuclei strain transmission, indicating that FAK plays an active role in regulating cell-ECM attachment within the native tendon ECM.[5] Finally, tendon-targeted FAK conditional knockout mice demonstrate altered development of tendon structure compared to wildtype (WT) mice at 30 days of age (P30), specifically exhibiting reduced cross-sectional area (CSA) yet mostly normal mechanical properties.[4] Despite these known roles for FAK in tendon, the degree to which FAK regulates mechanotransduction within the native tendon ECM as well as the regulatory role of FAK-dependent mechanotransduction in maintaining tendon homeostasis throughout postnatal growth remain unknown. Therefore, the objectives of this study were to define the role of FAK in (1) regulating mechanotransductive gene expression in response to de-tensioned free-floating explant conditions and (2) regulating tendon homeostasis during postnatal growth. We hypothesized that (1) FAK inhibition will attenuate the tendon gene expression response to free-floating explant conditions and that (2) tendon-targeted FAK conditional knockout will negatively impact tendon maturation and maintenance of homeostasis during postnatal growth, specifically by eliciting a reduction in tendon CSA and mechanical properties.

Methods

Free-Floating Tendon Explant

Flexor digitorum longus (FDL) tendons from male and female P30 WT mice were freshly dissected and randomized to receive FAK-inhibitor (FAK-I; 10 M PF-573228; Tocris; Minneapolis, MN) or control (DMSO) media (DMEM supplemented with 5% FBS and 25 mM HEPES). Tendons were maintained in explant culture conditions at 37°C for 1.5, 4, or 12 hours to assess the initial mechano-response to acute de-tensioning (n = 5/treatment group/timepoint). *Gene Expression:* Following explant treatment, RNA was immediately isolated from all tendons to quantify mechanotransductive gene expression using Taqman assays (*Acta2*, *Cyr61*, *Mmp3*, *Mmp13*, *Ptk2*; Housekeeper control: *Abl1*).

In Vivo Mouse Model

We utilized tendon-targeted FAK knockout (Scx-Cre;FAK$^{F/F}$; FAK-KO) mice,[6] in which we validated reduced *Ptk2* expression.[4] Achilles tendons (ATs), FDL tendons, and patellar tendons (PTs) from P60 FAK-KO and WT littermate controls were used for viscoelastic mechanical testing (males only) and histology (males and females). *Viscoelastic Mechanics:* Tendon CSAs were measured (n = 11-13/genotype), and tendons were subjected to a viscoelastic mechanical testing protocol (preconditioning, viscoelastic stress relaxation and dynamic frequency sweep, and a quasi-static ramp to failure). *Histology:* Whole ankle and knee joints were fixed, decalcified, paraffin embedded, and sectioned in the sagittal plane (n = 3-5/genotype). Additional knee joints were sectioned in the transverse plane to visualize the PT cross-section (n = 4-7/genotype). Overall tissue morphology was visualized via toluidine blue staining.

Results

Free-Floating Tendon Explant

In DMSO-treated tendons, we observed substantial increases in *Mmp3* (~90-fold) and *Mmp13* (~15-fold) gene expression with time under de-tensioned free-loading conditions, while expression of genes involved in cell contractility (*Acta2*, the Yap/Taz target gene *Cyr61*, and *Ptk2*) were not dramatically affected at these timepoints (Figure 1A-

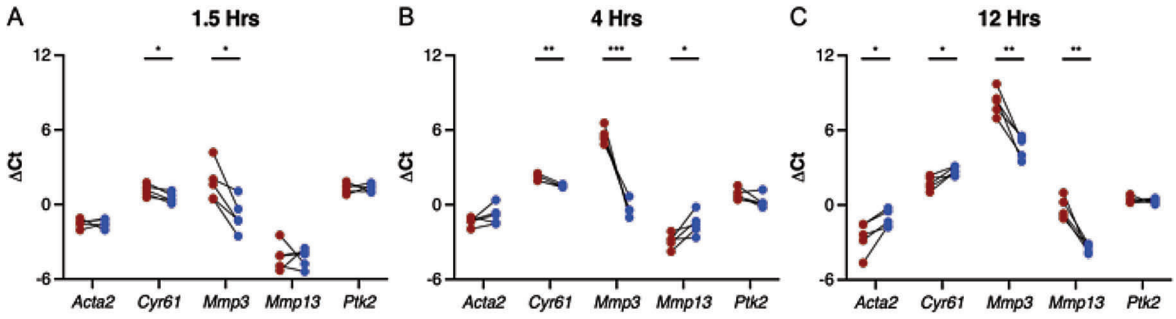

Figure 1. Mechanotransductive gene expression response to de-tensioning was significantly affected by FAK-I treatment. Gene expression levels (represented as "ΔCt") for DMSO and FAK-I treated tendons following **(A)** 1.5; **(B)** 4; and **(C)** 12 hours of free-floating explant conditions. Data represented as mean "±" standard deviation with plotted individual datapoints. Connected datapoints indicate contralateral FDLs. Bars indicate significant differences (t-test: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$).

C). Strikingly, FAK-I treatment resulted in differences in mechanotransductive gene expression at all timepoints evaluated. Specifically, compared to DMSO treated tendons, FAK-I treated tendons demonstrated reduced Mmp3 expression at all timepoints and reduced Mmp13 expression at the 12 hour-timepoint (Figure 1A-C). In addition, FAK-I treatment modulated the expression levels of Acta2 and *Cyr61* throughout the experiment (Figure 1A-C).

In Vivo Mouse Model

For tendon viscoelastic mechanical properties, we observed reduced CSA in all FAK-KO tendons, with the greatest effects in the ATs and PTs (Figure 2A). Structurally, FAK-KO resulted in reduced stiffness in the ATs (Figure 2B) and reduced maximum load in the FDLs (Figure 2C) compared to WT tendons. Interestingly, material properties including modulus and maximum stress were increased in FAK-KO ATs and PTs (Figure 2D-E). Finally, dynamic moduli were increased in the FAK-KO ATs and PTs at all frequencies evaluated (Figure 2F).

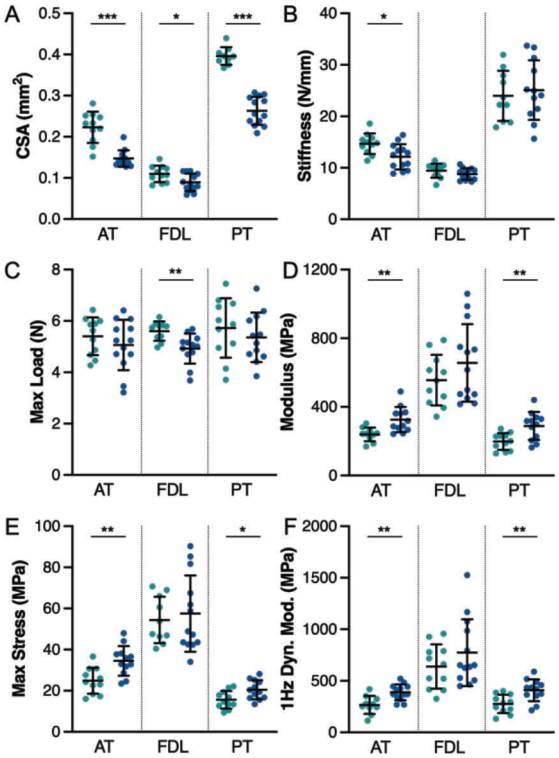

Figure 2. FAK-KO tendons demonstrated reduced size yet increased material properties at P60. **(A)** CSA; **(B)** stiffness; **(C)** max load; **(D)** modulus; **(E)** max stress; and **(F)** 1 Hz (shown as representative of all frequencies) dynamic modulus values for WT and FAK-KO tendons. Data represented as mean "±" standard deviation with plotted individual datapoints. Bars indicate significant differences (t-test: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$).

Discussion

This study investigated the regulatory roles of FAK on cell mechanotransduction within the native tendon ECM and on maintaining tendon homeostasis during postnatal growth. Consistent with our first hypothesis, FAK inhibition affected mechanotransductive gene expression levels in response to de-tensioned, free-floating explant conditions. Previous experiments demonstrated that tendons respond to de-tensioning by increasing expression of matrix remodeling genes such as *Mmp3* and *Mmp13* and decreasing expression of mechanotransductive genes such as *Acta2* and *Cyr61*.[7–10] In the present study, we observed similar severe increases in *Mmp3* and *Mmp13* with increased duration of explant treatment. Interestingly, FAK-I treatment suppressed this catabolic response to de-tensioning. In addition, we observed that FAK-I alters *Acta2* and *Cyr61* expression throughout our experimental timepoints. Taken together, these results indicate that FAK regulates the sensation of and response to changes in mechanical tension in the tendon *in situ* ECM.

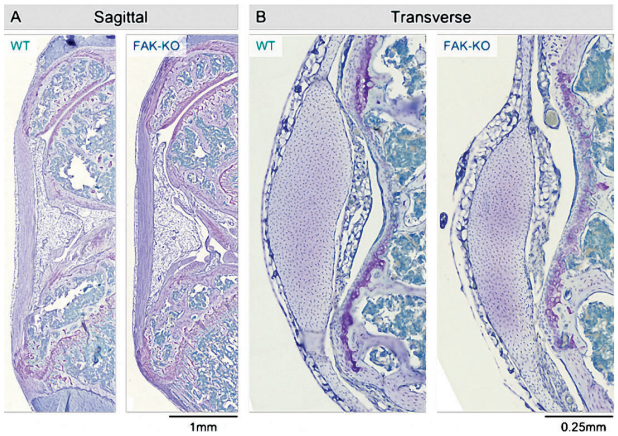

Figure 3. Paraffin histology confirmed the reduced size of FAK-KO tendons at P60. **(A)** Sagittal; **(B)** Transverse sections of patellar tendons from WT and FAK-KO mice.

Consistent with our second hypothesis, we observed substantially reduced CSA with FAK-KO in all tendons evaluated. Interestingly, despite their reduced size, FAK-KO tendons generally demonstrated increased material properties compared to WT tendons. Collectively, these results indicate that FAK regulates tendon size throughout postnatal development and that FAK-KO ultimately yields a tendon with increased material properties. Ongoing work will define the mechanism by which FAK regulates tendon size during development and postnatal growth. Specifically, we will investigate the regulatory role of FAK on tendon cell proliferation and ECM deposition. In addition, future work will investigate how mechanical loading regulats FAK-dependent mechanotransduction and tendon response *in vivo*.

Acknowledgements

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U·P·O·J

Collagen XII is a Critical Regulator of Supraspinatus Tendon Mechanics and Collagen Fiber Realignment Across Sex

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Introduction

Collagen XII is a Fibril-Associated Collagen with Interrupted Triple Helices (FACIT) that regulates collagen fibril assembly and is primarily expressed throughout tendon growth and development. Mutations in the *Col12a1* gene result in myopathic Ehlers-Danlos syndrome, a connective tissue disorder in which patients exhibit weakness at birth, absence of deep tendon reflexes and distal joint hypermobility and contracture[1]. Our novel tendon-targeted collagen XII mouse model demonstrated that patellar tendons exhibited reduced elastic, viscoelastic, and dynamic collagen fiber realignment properties across sex[2]. However, the role of collagen XII on the supraspinatus tendon, which experiences a complex, region-specific (insertion and midsubstance) loading environment within the rotator cuff of the shoulder, remains unknown. Therefore, the objectives of this study are to (1) elucidate the regulatory role of collagen XII on supraspinatus tendon whole-tissue and regional mechanics and dynamic response to load in mature mice using tendon-targeted (Scleraxis-Cre) collagen XII deficient and knockout mice and (2) understand whether the role of collagen XII on supraspinatus tendon biomechanical function and dynamic response to load is dependent on sex. We hypothesized that tendon-targeted deficiency and knockout of collagen XII would result in decreased supraspinatus tendon whole-tissue and regional elastic mechanics, whole-tissue viscoelasticity, and regional collagen fiber realignment across sex.

Methods

Supraspinatus tendons from male and female, day 60 tendon-targeted collagen XII heterozygous (HET) mice (ScxCre;*Col12a1*$^{f/wt}$, n = 8-9/group), knockout (KO) mice (ScxCre;*Col12a1*$^{f/f}$, n = 6-9/group) and wild-type (WT) control mice (Cre- littermates, n = 7-9/group) (IACUC) were subjected to our established mechanical testing protocol and collagen fiber realignment method. Tendons underwent stress relaxation testing at 3, 5, and 7% strain each with subsequent dynamic frequency sweeps at 0.1, 1, 5, and 10 Hz, followed by a quasistatic ramp-to-failure. Throughout the ramp-to-failure, dynamic collagen fiber realignment was quantified using cross-polarization imaging, and regional fiber alignment data was interpolated with a polynomial fit as a function of strain from the load-displacement data. Images were also used to optically measure strain to calculate regional moduli (insertion and midsubstance). For each sex, comparisons between genotypes were conducted using one-way ANOVAs followed by Bonferroni post-hoc tests. Significance was set at $p \leq 0.05$ and trends at $p \leq 0.1$.

Results

Cross-sectional area (CSA) was not different between male tendons, while female KO tendons exhibited a trending decrease in CSA relative to WT (data not shown). Consistent with our hypothesis, linear stiffness was significantly decreased in KO mice across sex and between female HET and KO mice (Figure 1A). Moreover, insertion modulus was significantly reduced in HET and KO tendons across sex whereas midsubstance modulus was significantly reduced in male KO tendons and female HET and KO tendons (Figures. 1B, C). Further, genotypic differences were observed in viscoelastic properties across sex. Percent relaxation was significantly increased in KO tendons across sex at all strain levels (5% strain shown in Figure 1D). Additionally, dynamic modulus was significantly decreased in male KO tendons and in female HET and KO tendons, while phase shift was significantly increased in KO tendons across sex across at all strain levels and frequencies (5% strain at 1Hz shown in Figures. 1E and 1F, respectively). These results are supported by reductions in collagen fiber realignment in HET and KO tendons across region and sex, as demonstrated by significantly greater normalized circular variance values for insertion and midsubstance regions from 3-7% strain (Figures. 2A-D), encompassing the toe and linear elastic regions of these tendons.

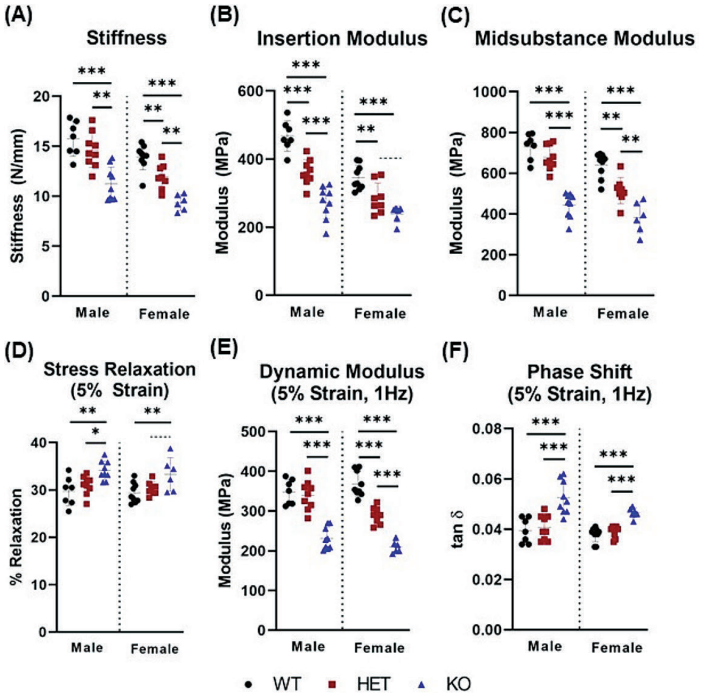

Figure 1. Differences between male and female elastic **(A-C)** and viscoelastic **(D-F)** mechanical properties of WT, HET, and KO supraspinatus tendons. Data as mean ± standard deviation (---$p \leq 0.1$ *$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$).

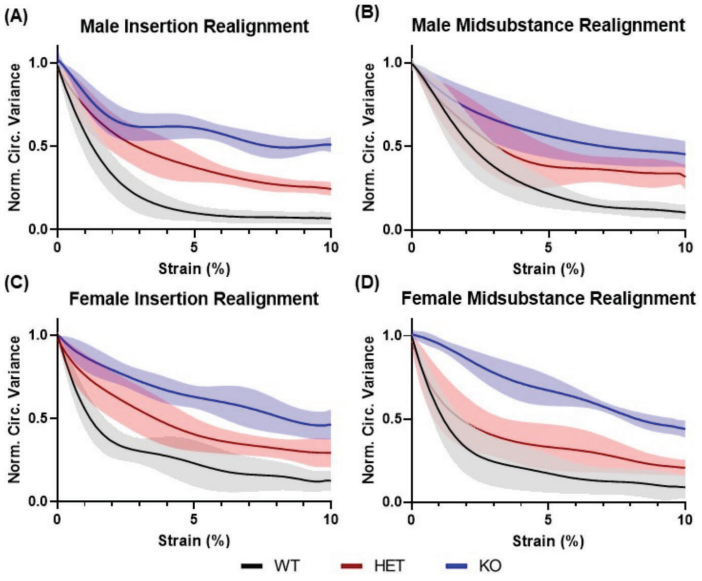

Figure 2. Collagen fiber realignment distribution differences for male **(A-B)** and female **(C-D)** WT, HET, and KO supraspinatus tendon insertion and midsubstance regions. Decreased normalized circular variance is indicative of increased collagen fiber realignment.

Discussion

This study investigated the role of collagen XII on supraspinatus tendon elastic and viscoelastic mechanics and dynamic collagen fiber realignment using tendon-targeted male and female ScxCre;*Col12a1*$^{f/wt}$ and ScxCre;*Col12a1*$^{f/f}$ mice. Consistent with previous data [2], we showed that tendon-targeted collagen XII knockout resulted in striking reductions in regional and whole-tissue elastic and viscoelastic mechanical properties and regional collagen fiber realignment. Further, reductions in these properties in our collagen XII deficient HET tendons, highlight the allele-dependency of collagen XII on tendon mechanical function and dynamic collagen fiber realignment. These mechanical deficits could be due to the improper hierarchical assemblies of HET and KO tendons resulting in disorganized tendon matrices with an inferior ability to respond to load. This was evidenced by marked reductions in the HET and KO tendons' responses to realign resulting in inferior mechanical properties, especially whole-tissue stiffness, regional moduli, and dynamic modulus. Although similar differences in elastic and viscoelastic mechanical properties were present across sex in response to collagen XII deficiency and knockout, more genotypic differences were present in female mice. Genetic variations in the *Col12a1* gene have been associated with an increased risk of ACL ruptures in women[4], implicating potential sex-specific effects of collagen XII deficiency and knockout. Overall, our results demonstrate that decreased collagen XII expression detrimentally affects supraspinatus tendon mechanical properties and dynamic collagen fiber realignment across sex.

Significance

This study elucidates the critical role of collagen XII in regulating male and female supraspinatus tendon regional and whole-tissue mechanics and dynamic structural response to load within the complex loading environment of the rotator cuff of the shoulder. Clinically, understanding the effects of collagen XII in tendon across sex can be used to develop and evaluate potential treatments modalities for myopathic Ehlers-Danlos syndrome.

Acknowledgements

We thank Ashley Fung for her assistance. This study was supported by NIH/NIAMS R01AR078790 and Penn Center for Musculoskeletal Disorders (NIH/NIAMS, P30AR069619).

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The Mechanosensor Focal Adhesion Kinase Regulates Cell Shape and Tendon Development

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Introduction

Throughout development and postnatal growth, resident tendon cells respond to mechanical cues from the nascent tendon extracellular matrix (ECM) to regulate tissue properties. Focal adhesion kinase (FAK, gene: *Ptk2*) is an intracellular protein kinase that regulates the actin cytoskeleton and cell-ECM adhesions. In tendon cells, FAK activity is required for tenogenic gene expression in response to growth factor stimulation and mechanical stretching.[1–3] In addition, pharmacological inhibition of FAK in explanted tendons significantly attenuates ECM to nuclei strain transmission.[4] Despite these known roles for FAK in tendon, the mechanism by which FAK activity regulates cell mechanotransduction as well as the role of FAK-dependent mechanotransduction in tendon development remain unknown. Therefore, the objective of this study was to evaluate the regulatory role of FAK in (1) tendon cell-ECM mechanical interactions and in (2) tendon development. We hypothesized that (1) FAK activity regulates tendon cell *in vitro* focal adhesion morphology and cell spreading behavior, and that (2) reduced FAK expression will negatively impact tendon development.

Methods

In Vitro Cell Culture

Tail tendon cells were isolated from P30 WT male and female mice (3 mice in 2 independent experiments; n = 30 cells/treatment/mouse). Cells were cultured on fibronectin-coated coverslips and treated with a FAK inhibitor (10μM PF-573228; FAK-I) or vehicle (DMSO) control. Immunofluorescence staining was performed to quantify cell morphology and pFAK localization 6 hours post-treatment.

In Vivo Mouse Model

Tendon targeted FAK knockout (Scx-Cre;$FAK^{F/F}$; FAK-KO) mice were generated.[5] Achilles tendons (ATs), flexor digitorum longus tendons (FDLs), and patellar tendons (PTs) from P30 male and female FAK-KO and WT littermate controls were used for gene expression analysis, paraffin histology, and viscoelastic mechanical testing. *Gene Expression:* RNA was isolated from tendons to evaluate *Ptk2* expression using Taqman assays, with *Abl1* as a housekeeping control (n=6/genotype/sex). *Paraffin Histology:* Whole ankle and knee joints were fixed, decalcified, paraffin embedded, and sectioned in the sagittal plane (n=5/genotype/sex). Hoechst nuclear staining was used to quantify cell density and nuclear aspect ratio (nAR). Overall tissue morphology was evaluated via toluidine blue staining. *Viscoelastic Mechanics:* Tendon cross-sectional areas (CSAs) were measured (n=7-9/genotype/sex), and tendons were subjected to a viscoelastic mechanical testing protocol (preconditioning, viscoelastic stress relaxation and dynamic frequency sweep, and a quasi-static ramp to failure).

Results

In Vitro Cell Culture

FAK-I treated cells developed pronounced cell protrusions compared to DMSO treated cells (Figure 1A). While cell area was not different between groups (Figure 1B), FAK-I treated cells had significantly higher cell compactness values relative to DMSO treated cells (Figure 1C), which is indicative of the increased protrusion phenotype. Focal adhesions were closer to the cell periphery and colocalized less with pFAK staining in FAK-I treated cells relative to DMSO treated cells (Figure 1D-E).

In Vivo Mouse Model

Ptk2 expression was reduced in all FAK-KO tendons relative to WT tendons, thereby validating our conditional knockout mouse model (Figure 2). FAK-KO tendons were not remarkably distinct from WT tendons histologically, and there were no differences between groups in quantified cell density or nAR (data not shown). Interestingly, FAK-KO tendons were consistently smaller compared to WT tendons (Figure 3A), while there was

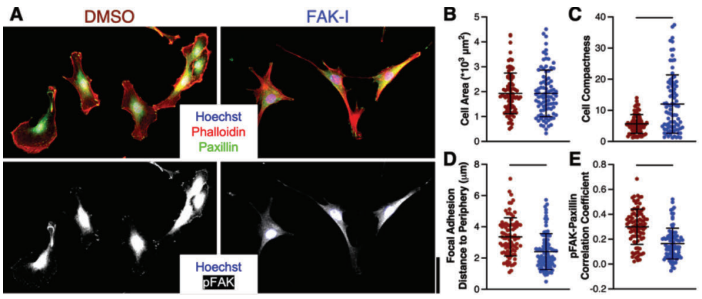

Figure 1. (A) Representative images of DMSO and FAK-I treated tail tendon cells. Scale: 50 μm; **(B)** Cell area; **(C)** cell compactness, **(D)** focal adhesion distance to cell periphery, and **(E)** pFAK-Paxillin correlation coefficient quantifications of DMSO and FAK-I treated tail tendon cells. Data was analyzed with t-tests comparing groups. Bars represent sig. diff. between groups ($p < 0.05$).

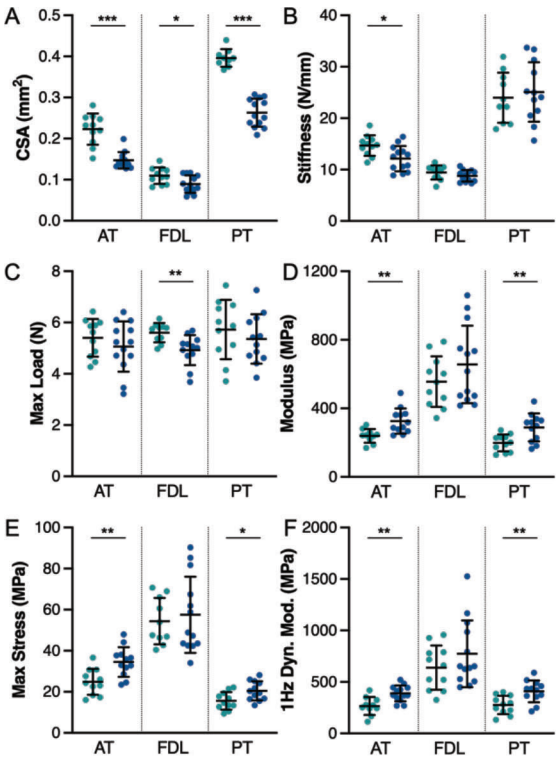

Figure 2. *Ptk2* expression for ATs, FDLs, and PTs from WT and FAK-KO tendons. Data was analyzed with t-tests comparing groups. Bars represent sig. diff. between groups ($p < 0.05$).

no difference in animal body weight (body weight data not shown). Despite the decreased size in all FAK-KO tendons, stiffness was only decreased in PTs (Figure 3B), and modulus was generally comparable and, in fact, superior in ATs (Figure 3C). Failure properties of FAK-KO tendons demonstrated decreased maximum load in the AT and PT, while maximum stress was increased in the FDL (Figure 3D-E). Viscoelastic stress relaxation was not different between groups (data not shown), though dynamic modulus was increased in all FAK-KO tendons relative to WT tendons at all frequencies evaluated (1Hz dynamic modulus data shown as representative in Figure 3F).

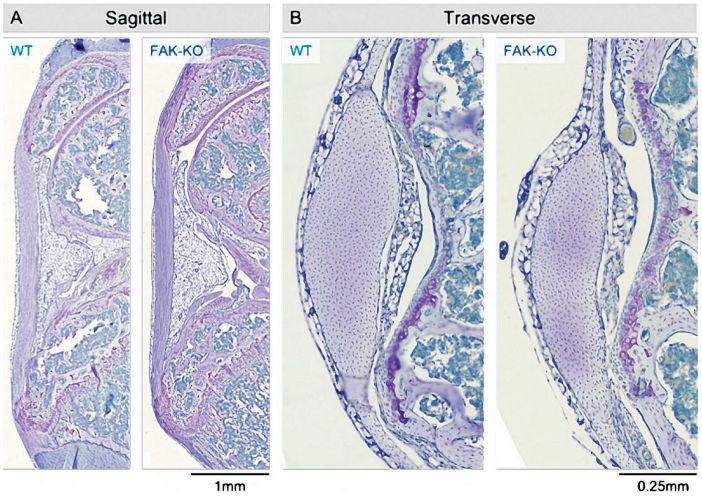

Figure 3. (A) Cross-sectional area (CSA); **(B)** stiffness; **(C)** modulus; **(D)** maximum load; **(E)** maximum stress; and **(F)** 1Hz dynamic modulus measurements for WT and FAK-KO tendons. Data was analyzed with t-tests comparing groups. Bars represent sig. diff. between groups ($p < 0.05$).

Discussion

Consistent with our hypothesis, inhibition of FAK activity in tendon cells significantly affected focal adhesion morphology and cell spreading behavior. Taken together with our previous results demonstrating attenuated ECM to nuclear strain transmission with reduced FAK activity,[4] these findings help explain FAK's regulatory role on tenogenic gene expression.[1–3] In our in vivo model, all tendons from FAK-KO mice were smaller, which is consistent with our hypothesis and indicates that FAK plays an essential role in tendon development. Interestingly, structural properties were not consistently reduced, and material properties were comparable or increased in FAK-KO tendons relative to WT tendons, potentially indicating changes in matrix assembly.

Significance

Due to the mechanical role and mechano-responsiveness of tendons, defining the key transductive pathways that regulate cell and tissue properties will be critical to better understand disease and to develop improved therapies nmnm . Our results indicate that FAK-dependent tendon cell mechanotransduction may drive tissue assembly during growth and development.

Acknowledgements

We acknowledge financial support from NIH/NIAMS (T32AR007132 and P30AR069619).

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Collagen III Haploinsufficiency Alters Fibril Size but Not Mechanical Properties in Uninjured, Young Adult Male Murine Tendons

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Introduction

Clinically, *Col3a1* mutations present as vascular Ehlers-Danlos syndrome (vEDS), a rare but life-threatening condition due to abnormalities in the matrix of the vasculature and hollow viscera. Additional patient morbidity from tendon pathology[1,2] indicates a consequential role for type III collagen (Col3) in tendon. While a role for Col3 in matrix homeostasis has been established in the vasculature,[3] cutaneous skin,[4] articular cartilage,[5] meniscus,[6] and bone,[7] the involvement of Col3 in tendon structure and function is poorly understood. Early investigations focused on understanding the role of Col3 in female murine tendon,[8] but biological sex is known to influence tendon health, Col3 levels,[9] and vEDS presentation,[10] motivating investigation of the role of Col3 in male tendon. Therefore, the objective of this study was to elucidate the role of Col3 in tendon homeostasis using a murine model of male vEDS. We hypothesized that Col3 haploinsufficiency would alter fibrillogenesis and fibril maintenance yielding decreased large diameter fibrils and mechanically inferior tendons.

Methods

Tendons from male wild-type (WT) Balb/cJ and heterozygous *Col3a1*$^{+/-}$ mice at 90 days of age were assessed (IACUC approved). Patella-patellar tendon-tibia complexes were dissected and prepared as described[11] for mechanical testing (n ≥ 9/group). Tendons were assessed with a viscoelastic testing protocol consisting of: 1) preconditioning, 2) stress relaxation at 3% and 6% strain with a subsequent sinusoidal frequency sweep (10 cycles at 0.1, 1, 5, and 10 Hz) at each strain level, 3) return to gauge length, and 4) ramp to failure at a strain rate of 0.1% strain/s. Images were captured during the ramp to failure for elastic modulus measurement. Patellar tendons were fixed, processed, and imaged using transmission electron microscopy as described[12] to measure collagen fibril diameters (n = 3/group). T-tests were used to determine the impact of genotype on mechanical properties. A Kolmogorov-Smirnov test was used to assess the effect on collagen fibril diameter distributions. Significance was set at $p \leq 0.05$.

Results

Compared to WT tendons, *Col3a1*$^{+/-}$ tendons were not significantly different in any quasistatic or viscoelastic mechanical property including stiffness (Figure 1A), elastic modulus (Figure 1B), dynamic modulus (Figure 1C), phase shift (Figure 1D), and percent relaxation (data not shown). In contrast, collagen fibril size distributions were significantly different between genotypes (Figure 2, $p < 0.0001$). WT tendons had a characteristic bimodal fibril diameter distribution (Q1: 70.0 nm, Q2: 116.5 nm, Q3: 154.2 nm). *Col3a1*$^{+/-}$ tendons had a tighter fibril diameter distribution (Q1: 80.6 nm, Q2: 125.0 nm, Q3: 154.0 nm) with a smaller proportion of small diameter fibrils (< 70 nm) and a greater proportion of intermediate and large diameter fibrils (> 110 nm).

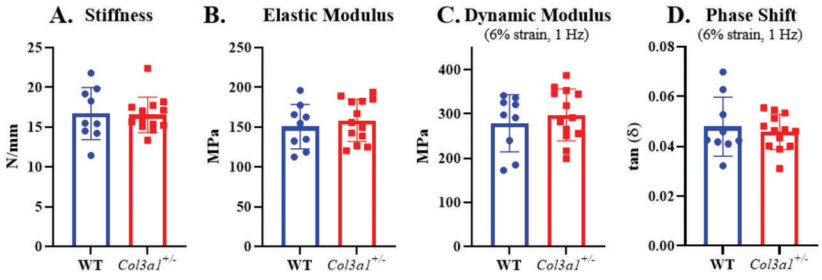

Figure 1. Collagen III haploinsufficiency does not impact male tendon mechanical properties. No differences in stiffness **(A)**; elastic modulus **(B)**; dynamic modulus **(C)**; phase shift **(D)**; or percent relaxation (data not shown) were observed between genotypes. No differences were seen in other strain or frequency levels (data not shown).

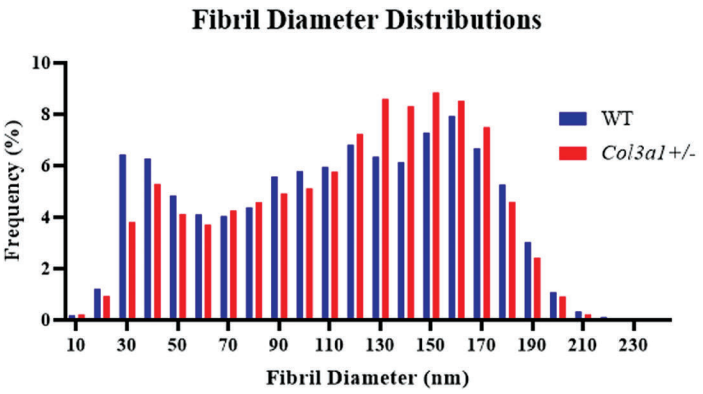

Figure 2. Collagen III haploinsufficiency alters collagen fibril size. Collagen fibril diameter distribution was characteristically bimodal in WT tendons. *Col3a1*$^{+/-}$ tendons had significantly different fibril distribution ($p < 0.0001$) with a lower proportion of smaller fibrils (< 70 nm) and a higher proportion of intermediate and large fibrils (> 110 nm).

Discussion

We studied the role of Col3 in tendon homeostasis using a murine model of male vEDS. In contradiction to our hypothesis, Col3 haploinsufficiency in young adult male mice did not alter uninjured patellar tendon quasistatic or viscoelastic mechanical properties. However, in support of our hypothesis, Col3 haploinsufficiency did significantly alter the fibril diameter distribution. This alteration in matrix structure may have mechanical consequences in settings where fibrillogenesis and maintenance are altered, such as in injury and/or aging. Importantly, genotype-dependent changes to fibril diameter in male mice differ from those observed in female mice. In developing female mice, Col3 haploinsufficiency resulted in a decrease in large diameter fibrils, 8 in contrast to the increase in large diameter fibrils observed in the present study. Interestingly, this indicates a differential influence of biological sex on tendon matrix structure in the context of Col3 haploinsufficiency. Conclusions from this study should be interpreted in the context of a conventional haploinsufficiency mouse model which conflates developmental and regulatory effects. Moreover, dose-dependent effects of Col3 insufficiency cannot be investigated due to perinatal lethality of *Col3a1*$^{-/-}$ mice. Further, in recognition of the critical importance of Col3 in the provisional healing matrix, subsequent investigations will define the effects of Col3 knockdown in injury and advanced aging contexts. Future studies will leverage the power of inducible Col3 knockdown to further delineate the sex-, dose-, and age-dependence of the tendon response to Col3 knockdown in homeostatic and injury environments.

Significance

In addition to direct implications for patients with Col3a1 mutations, insights from this study reveal contributions of Col3 to tendon structure which serve as an important foundation for future investigations of sex-based differences in the regulatory role of Col3.

Acknowledgements

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Loading Levels in Patellar Tendon, Quadriceps Tendon, and Patellofemoral Joint Across 35 Rehabilitation Exercises

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Introduction

Patellar and quadriceps tendinopathy and patellofemoral pain are prevalent knee joint pathologies, particularly affecting physically active individuals. Yet state-of-the-art knee rehabilitation protocols often rely on clinician perspectives, potentially leading to suboptimal recovery outcomes. Progressive loading promotes tissue healing, but the loading levels in knee tendons and the patellofemoral joint have not been quantified for most common rehabilitation exercises. As such, clinicians need more quantitative evidence to refine knee rehabilitation plans that progress therapeutic loading and suit patient-specific recovery goals. We recently developed a "Loading Index" based on loading peak, impulse, and rate to define overall loading "tiers" in the Achilles tendon during various exercises.[1] In this study, our goal was to establish a Loading Index to quantify, compare, rank, and categorize loading levels in the patellar tendon, quadriceps tendon, and patellofemoral joint across 35 common clinical knee rehabilitation exercises and routine activities.

Methods

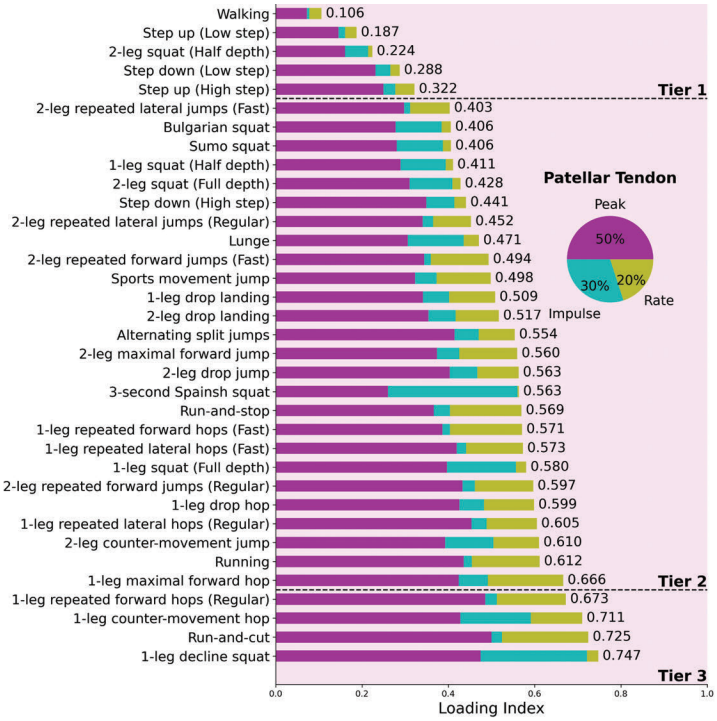
We recruited 20 healthy adult subjects (10F, 25.9 ± 5.7 years, body mass index = 24.1 ± 2.6 kg/m2) who had no self-reported knee pain or injury, and obtained their informed consent for this IRB-approved study. We recorded motion data of each subject performing 35 knee rehabilitation or routine exercises (Figures 1-3), and calculated knee flexion angles and moments using an inverse dynamics biomechanical model. Next, we estimated load in the patellar tendon, quadriceps tendon, and patellofemoral joint based on the knee flexion angles and moments, using effective tendon moment arms and force relationships among the 3 knee structures defined in literature.[2-4] We normalized each knee structure load by subject weight, zeroed negative force components to account for physiologically one-way loading, then calculated their peak, impulse, and rate metric during each exercise. We calculated the Loading Index of each exercise as a weighted sum of the group-average of each loading metric.[1] For knee tendons, we set 50% Loading Index weight on loading peak, 30% on impulse, and 20% on rate, similar to our Achilles tendon study.[1] For patellofemoral joint, we set 50% weight on loading peak and 50% on impulse based on our perspective on their relative clinical importance. To compare the overall loading levels in each knee structure across all 35 exercises, we ranked their Loading Indices in an ascending order, then categorized them into 3 equally-divided tiers, from Tier 1 (low, i.e. < 0.333) to Tier 3 (high, i.e. > 0.667).

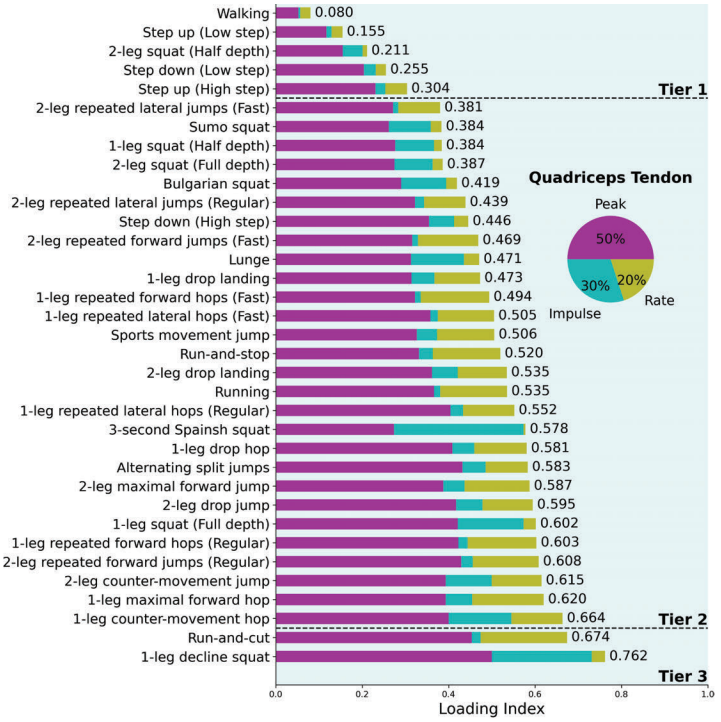
Results

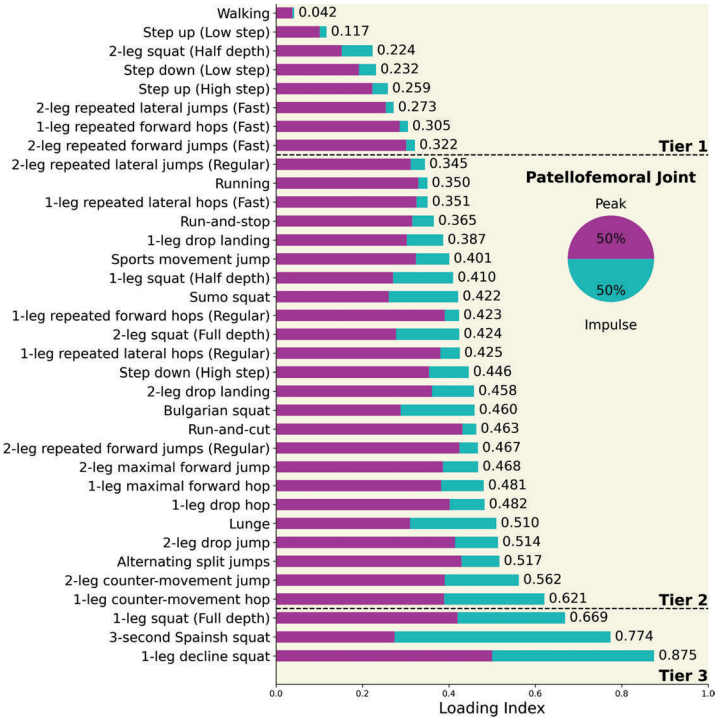
Overall loading levels in each knee structure varied substantially across exercises (Figures 1-3). Most rehabilitation exercises fell into Tier 2 ($0.333 <$ Loading Index < 0.667), suggesting they provide moderate knee loading. Few exercises provide low-level loading (Tier 1) that resembles routine walking, while single-leg decline squat was the only exercise that generate highest-level loading in all 3 knee structures (Tier 3). For many exercises with moderate or high loading levels, loading peak, impulse, and rate varied between fast-speed exercises (e.g. run-and-cut) and long-duration exercises (e.g. Spanish squat). Loading ranks were generally similar among the 3 knee structures, yet for quadriceps tendon and patellofemoral joint forces, there is a notable bias towards higher Loading Index for high knee flexion exercises. Notably, exercises with long duration and high knee flexion (e.g. squats) saw a relative shift up the ranks while many fast-speed but short-duration exercises (e.g. running and jumping) moved down the ranks substantially.

Discussion

Our results show that most rehabilitation exercises feature a moderate level of knee structure loading, while few exercises provide low level of loads that resemble walking, or







Figures 1-3. Loading Indices ranked for patellar tendon, quadriceps tendon, and patellofemoral joint forces across 35 rehabilitation exercises, categorized low (Tier 1), moderate (Tier 2), and high (Tier 3). Loading Index weights for patellar and quadriceps tendons are 50% on loading peak, 30% on impulse, and 20% on rate. Weights for patellofemoral joint are 50% on loading peak and 50% on impulse.

loading with both high magnitude and long duration. Exercises with a moderate level of knee structure loading provide the most diverse opportunities for patient-specific rehabilitation planning, as clinicians can simplify protocols and choose mechanically similar exercises that best suit patient needs and abilities during rehabilitation. Conversely, the scarcity of low-loading exercises suggest that clinicians should use caution when prescribing early-stage exercises to avoid tissue over-loading that poses risks of healing complication. While many fast exercises feature high level patellar tendon loading, exercises with high knee flexion often generate higher quadriceps tendon and patellofemoral joint loading. We thus recommend clinicians differentiate loading profiles among the 3 knee structures and adjust rehabilitation strategies to progress therapeutic loading on the healing knee structure.

Significance

To our knowledge, this study is the first to quantify loading in three knee structures across a large collection

of exercises in the same cohort. Our findings provide straightforward tools for clinicians to design rehabilitation protocols that prescribe progressive therapeutic loading while suiting patient-specific recovery needs, and set a benchmark for future research on complex and chronic knee pathologies.

Acknowledgments

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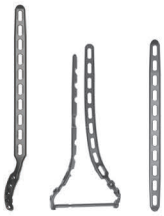

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