Penn Medicine

PENN ORTHOPAEDICS

EXCELLENCE *IN MOTION* 2016

DATA SUPPLEMENT

INNOVATORS IN ORTHOPAEDIC CARE

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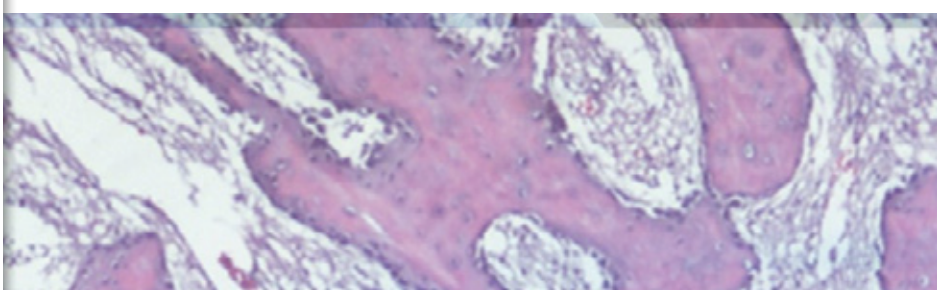

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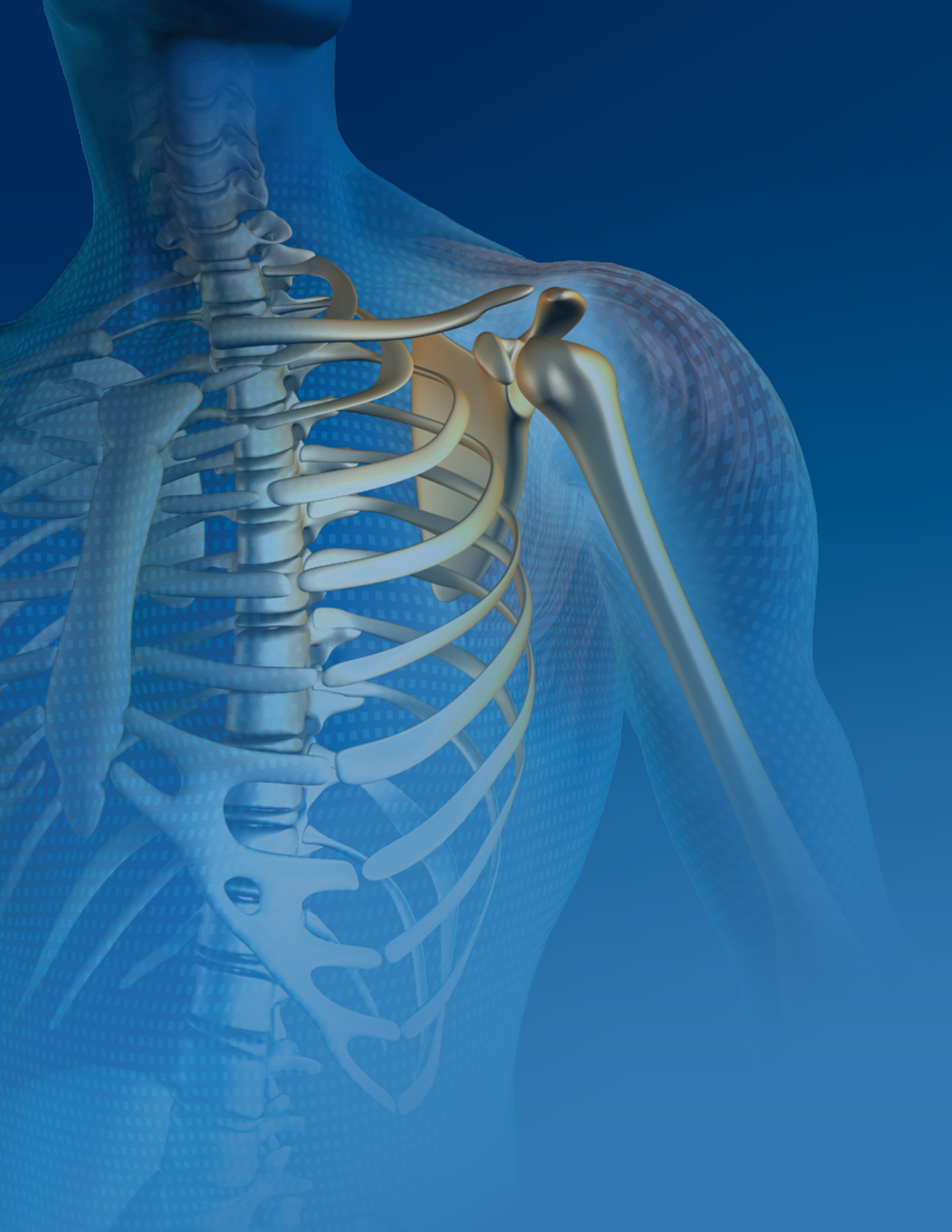


Source: Sabina Louise Pierce

Dear Colleagues,

I'm pleased to bring you the second 2015 issue of Excellence in Motion, the newsletter of Penn Orthopaedics.

The subject of this issue is innovation, a word that today carries the implication of rapid engagement and invention. Developments in orthopaedic surgery and research are rarely quick to arrive, however. The articles in this issue feature the advanced work of Frederick S. Kaplan, MD, and James L. Carey, MD, MPH, one a researcher and the other a surgeon, as well as highlights on the continued advancements made in each of the Penn Orthopaedics services.

Our first article on page four discusses a pioneering cartilage repair procedure called autologous chondrocyte implantation (ACI). One of Dr. Carey's specialties is the cartilage of the knee, perhaps the least hospitable terrain in the musculoskeletal system. Essential to movement, articular cartilage is avascular, aneural and non-regenerative. Director of the Penn Center for Advanced Cartilage Repair and Osteochondritis Dissecans Treatment, Dr. Carey has been performing ACI procedures to restore cartilage in the knee. ACI combines surgery with cells harvested from the knee and amplified in cell culture, a genuinely innovative therapy that is bettering the lives of patients at Penn Medicine.

Dr. Kaplan has devoted his career to the origins and the mechanisms of fibrodysplasia ossificans progressiva (FOP), an incurable autosomal skeletal dysplasia. FOP causes uncontrolled heterotopic bone growth, and was a complete mystery until Dr. Kaplan, his longtime collaborator Eileen M. Shore, PhD, and their colleagues confirmed its genetic source in 2006. Since that time, he has worked tirelessly to parse the genomic profile of FOP and its variants in a search not only for treatments, but for the genetic key to bone growth and its potential for regenerative treatments in the future. A report on Dr. Kaplan's research appears on page seven.

The efforts of Drs. Kaplan and Carey and the highlights of each service presented here are typical of Penn Orthopaedics, where extraordinary things happen every day. It is my hope that you, our peers and partners in care, find this issue rewarding and thought-provoking.

L. Scott Levin, MD, FACS

Chair, Department of Orthopaedic Surgery
Paul B. Magnuson Professor of Bone and Joint Surgery
Professor of Surgery, Division of Plastic Surgery
Medical Director, Penn Musculoskeletal Center

AUTOLOGOUS CHONDROCYTE IMPLANTATION (ACI)

FOR RESTORATION OF ARTICULAR CARTILAGE

James L. Carey, MD, at Penn Sports Medicine, specializes in repairing the most vulnerable component of the body's most vulnerable joint–the cartilage of the knee. This article reviews several of the current methods and approaches for the repair of osteochondral injury, including autologous chondrocyte implantation, an advanced technique Dr. Carey brought to Penn Medicine several years ago. Dr. Carey is the Director of the Penn Center for Advanced Cartilage Repair and Osteochondritis Dissecans Treatment.

When osteochondral injury occurs, it is often the result of trauma, repetitive motion/impact, abrupt abnormal direction of weight loading, fractures, wear/degeneration, friction abrasion or inflammatory conditions. The primary symptoms include pain, functional impairment and a catching sensation during extension.

As a form of connective tissue that originated in primitive vertebrates with bite-sized lives, cartilage may never have been intended to be intrinsically repairable. Evidence of this remains in the articular (hyaline) cartilage of the human knee. All of the elements endemic to intrinsic repair—vascularity; neural signaling; various, numerous, and mobile regenerative cells; sufficient and regulated fluid balance; a rich source of nutrition—are missing from articular cartilage. This means that cartilage repair is a slow, inefficient process, and that the body has few mechanisms to offer assistance.

Despite this drawback, articular cartilage has many of the elements of modern industrial design: a highly specialized, low-friction coating comprised of a dense extracellular matrix that provides lubrication, responsive stress distribution and the capacity to withstand loads up to five times the bearer's body weight. Its materials include collagen, proteoglycans and tissue fluids, and an architecture defined by zones and regions. Articular cartilage is sparsely populated by metabolically active cells, or chondrocytes, that act to maintain and repair the matrix. Unfortunately—and in keeping with the principal flaw of cartilage—chondrocytes do not replicate and cannot migrate, so are active only in their immediate vicinity.

Because articular damage is progressive, it is important to intervene before it becomes widespread. The likelihood of a successful, enduring repair or restoration for isolated focal cartilage injury is greatly diminished once general cartilage deterioration occurs. Unfortunately, early warning isn't a capacity identified with cartilage injury. Articular cartilage is aneural—and because it contains no calcium, it is almost invisible to x-rays. Outside of an acute injury, the first warning sign of significant damage to the cartilage may not appear

SEE OUR **STEP-BY-STEP OUTLINE**
of the ACI procedure on Page 6.

until a fragment splits away and strikes a nerve or impinges movement of the joint.

Repairing the Damage: Autologous Chondrocyte Implantation (ACI)

Although a variety of palliative measures are available to address minor osteochondral injuries, larger cartilage defects (>1cm^2) or defects that extend from the surface to the bone (i.e., full-thickness) can only be addressed by reparative or restorative surgery, as defects of this size have been shown to worsen and increase in size. Defects greater than 3cm^2 likely need ACI or osteochondral allograft (OCA).

Reparative measures involve the arthroscopic penetration of the subchondral plate, either by drilling, abrasion or microfracture. The objective in each of these procedures is to create small openings in the cartilage that, by extending into the subchondral bone marrow below, prompt the release of multipotent mesenchymal stem cells into the cartilage, which then differentiate into articular tissue. Microfracture is now considered a standard of care, though clinical studies suggest that durability is an issue by comparison to newer restorative procedures involving transferred hyaline cartilage.

The restorative procedures at the Penn Center for Advanced Cartilage Repair and Osteochondritis Dissecans Treatment include ACI, OCA, and osteochondral autograft transfer (OAT).

The Procedure

ACI, the most recent addition to the armamentarium at Penn Medicine, has the goal of producing tissue to restore the durability and function of articular cartilage with the patient's own cartilage cells. The procedure is best suited for younger patients with full-thickness lesions. Unlike other cartilage restoration procedures, ACI depends upon endogenous cells amplified *ex vivo* to rebuild and restore the articular surface. The cells are harvested from the patient as plugs during the first step of the procedure from healthy articular cartilage in a minor load-bearing area of the knee. The plugs then undergo *in vitro* chondrocyte amplification in cell culture for three to five weeks in an outside laboratory and are returned to the hospital as vials, each of which contains ~12 million cells. Each patient receives an average of 2-3 vials.

During the second step of the procedure, the chondral defect is first cleaned of all debris and expanded until the surrounding cartilage consists only of healthy tissue. An absorbable cell-free collagen membrane is then sutured over the defect and sealed with fibrin glue. At this point, the chondrocytes are implanted beneath the graft to fill the defect. See our step-by-step outline of the procedure on page six. In the following six weeks, the patient undergoes postoperative rehabilitation involving continuous passive motion and protected weight bearing. Within approximately a year's time, the implanted chondrocytes obtain the consistency of articular cartilage.

"We shall find that an ulcerated cartilage is universally allowed to be a very troublesome disease; that it admits of a cure with more difficulty than a carious bone; and that, when destroyed, it is never recovered."

—**WILLIAM HUNTER**, ON THE STRUCTURE AND DISEASE OF ARTICULATING CARTILAGES. PHILOS TRANS 42(B): 514-521, 1742.

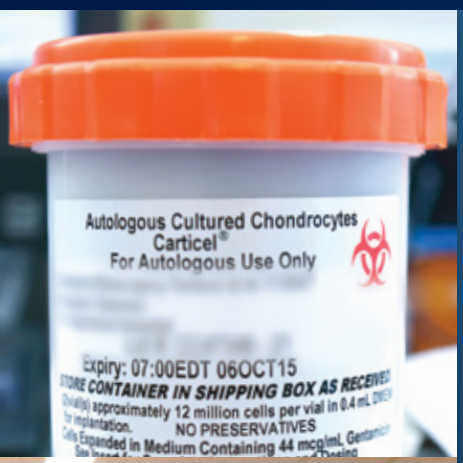
Discussion

For surgeons, ACI possesses a number of advantages. The procedure allows for the treatment of irregular shapes without removing healthy tissue, and permits the treatment of challenging curvatures (trochlea and patella). Patients benefit because ACI permits them to choose the date of implantation and preserves the healthy tissue of the knee. Full recovery for ACI takes approximately 12-18 months while the injected cartilage cells assimilate with the surrounding cartilage. It is important to note, however, that most patients are back on their feet within 4-6 weeks, can resume normal activity and load bearing within 6 months and get back to higher intensity activities within 1 year. In addition, long-term follow-up of ACI has demonstrated durable improvement in pain and function, with graft survivorship of >80% at 10 years. These advantages to both the patient and the surgeon make ACI an appealing option for the treatment of small to moderate size cartilage defects.

Reference // Clin Orthop Relat Res. 2008 Apr;466(4):952-62.

ACI PROCESS: STEP-BY-STEP

Autologous Chondrocyte Implantation (ACI) is a relatively new procedure for patients with small to moderate size cartilage defects in the knee. ACI is a two stage procedure where the Orthopaedic surgeon harvests and grows millions of the patient's own cartilage cells, and then implants these new cartilage cells to fill in the damaged area. Normal cartilage is 1,000 times more slippery than ice on ice, but when that cartilage is damaged it is unable to heal itself and impedes normal movement—causing pain. These types of cartilage defects are common and over time can worsen and lead to disabling joint disease if left untreated.



1

Vile of New Cartilage Cells

Six to eight weeks before implantation, some of the patient's own healthy cartilage cells are harvested and sent to a lab where millions more cells are grown.

2

Preparing for Surgery

James L. Carey, MD, MPH is among the nation's leaders in ACI. Here Dr. Carey and physician assistant Sabrina Fitzig scrub in and discuss preparations for surgery.

3

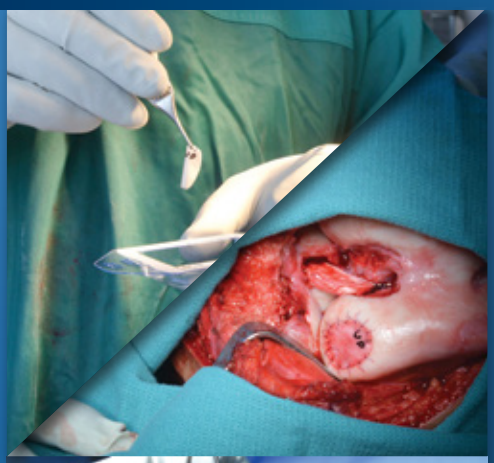
Arthroscopic Examination

Before the initial incision, Dr. Carey examines the cartilage and other structures in the knee using minimally invasive arthroscopy.

4

Site Preparation

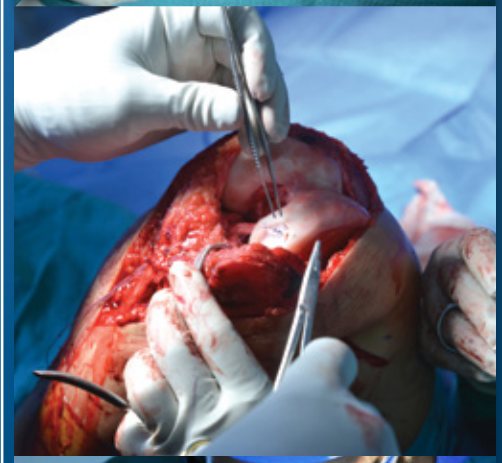
After opening the knee, ACI requires intense precision during the preparation of the damaged area where the new cells will be implanted.



5

Holding the New Cartilage

Before the new cells are implanted, Dr. Carey sizes and places a porcine cover over the defect to hold the new cells in place.



6

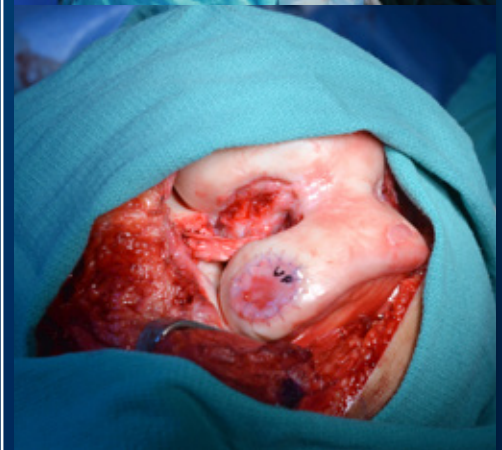
Securing the Cover

The porcine cover is sutured securely around the perimeter of the defect, leaving a small section open for injection of the new cartilage cells.

7

New Cell Injection

Dr. Carey injects the new cartilage cells under the cover where they will change from a liquid when injected to fully assimilate with cartilage by ~1 year post-surgery.



8

Sealing the Cells

After injection, the remaining section of the cover is sutured shut and a sealant is applied to prevent any leakage of the new cells and allow them to assimilate with the surrounding cartilage during recovery.

Advances in Rare Osteopathologies at Penn Medicine

Clinical Presentation of Severe Variants of Fibrodysplasia Ossificans Progressiva (FOP)

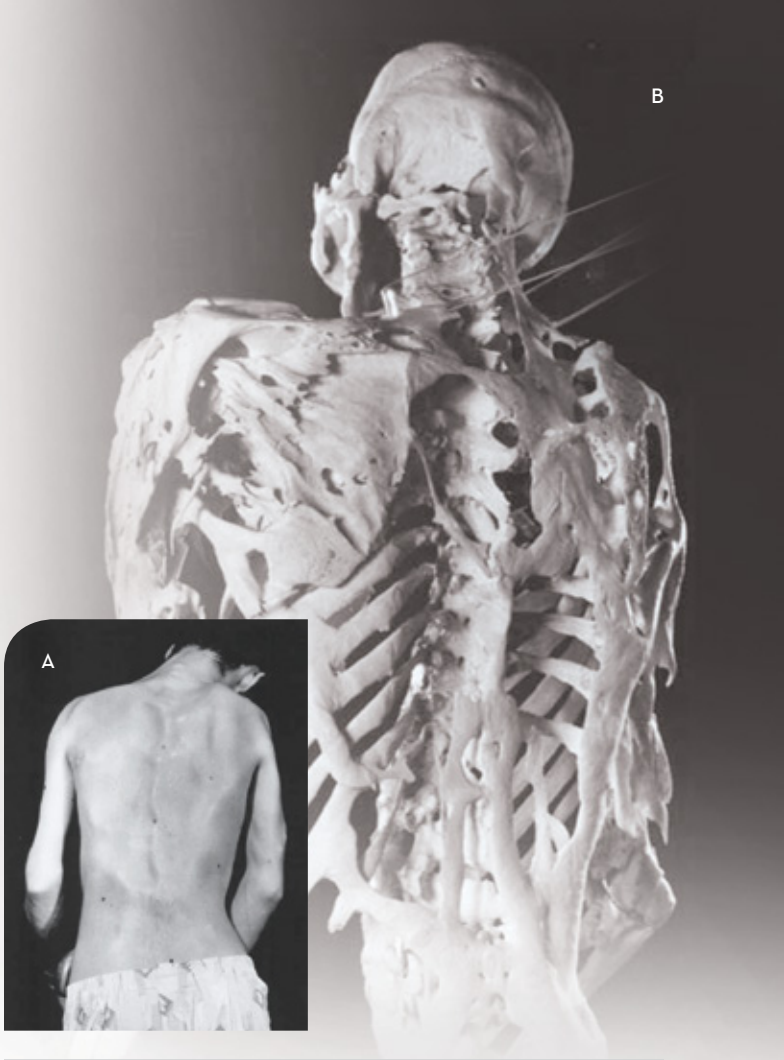
The Isaac & Rose Nassau Professor of Orthopaedic Molecular Medicine and Chief of Molecular Orthopaedic Medicine at the Perelman School of Medicine, **Frederick S. Kaplan, MD,** leads the Center for Research in Fibrodysplasia Ossificans Progressiva (FOP) and Related Disorders at Penn Medicine. Dr. Kaplan and lifelong colleague **Eileen M. Shore, PhD**, have devoted much of their careers investigating the origins and the mechanisms for heterotopic bone formation and skeletal metamorphosis in FOP, among the rarest skeletal dysplasias known.

Frederick S. Kaplan, MD

Eileen M. Shore, PhD

Fibrodysplasia ossificans progressiva (FOP) is considered the most severe and disabling disorder of extraskeletal (heterotopic) ossification in humans. An ultra-rare autosomal disorder (only 800 known patients in the world), FOP almost always presents with two characteristic and unique anatomical features: malformation of the great toes and episodic soft tissue flare-ups resulting in progressive heterotopic ossification. The effects of the disorder are generally first seen in childhood, either without warning or following minor or incidental trauma (injections, for example). From the point of initiation, FOP progresses over time to cause extra-articular fusing of the axial and appendicular joints and ossification of the large striated muscles, until the patient is immobilized in a bony, subcutaneous carapace, often described as a second skeleton.

Clinical interest in ultra-rare, incurable diseases has rarely been prolific, but here FOP deserves a measure of consideration: in the last 60 years, the disorder has evoked eight journal reports a year, on average. By contrast, 1,014 articles about gallbladder stones appeared in the PubMed record in 2014 alone. Until the early 1990s, moreover, when Dr. Kaplan published the first in a series of articles describing the heritable transmission of FOP, no one had touched upon the genetic sources of FOP.



The rigid posture of this 25-year-old man with fibrodysplasia ossificans progressiva was due to ankylosis of the spine, shoulders, and elbows. Plates and ribbons of ectopic bone contour the skin over the back and arms (A), and can be seen directly on the skeleton (B). Courtesy of the Mütter Museum, College of Physicians of Philadelphia.

Fortunately, at about this time, genomic profiling became a practical reality. Within a few years, Dr. Kaplan and colleagues around the world were focusing on variants of the bone morphogenetic protein (BMP) gene family in FOP; the BMPs are identified with limb patterning and ossification. After more than a decade of research, Dr. Kaplan, Dr. Shore, and a team affiliated with the Center for FOP Research at Penn confirmed in 2006 that a mutation at the BMP receptor protein ACVR1 was the source of FOP.

In the report describing this discovery, the genomic profiles of five families with FOP were compared to those 112 controls without the disorder to isolate a single nucleotide mutation within the ACVR1 receptor (called ACVR1 p.R206H) as the cause of FOP. Commonly expressed in skeletal muscles and chondrocytes, ACVR1 is a receptor for BMPs that, when activated, downregulate BMP antagonists, expand cartilage elements, induce ectopic development of cartilage (chondrogenesis), and stimulate joint fusion. In persons with FOP, the R206H mutation causes a change in the receptor, leading to disruption of the BMP signaling pathway and subsequent irregular and uncontrollable osteogenesis.

CASE STUDY

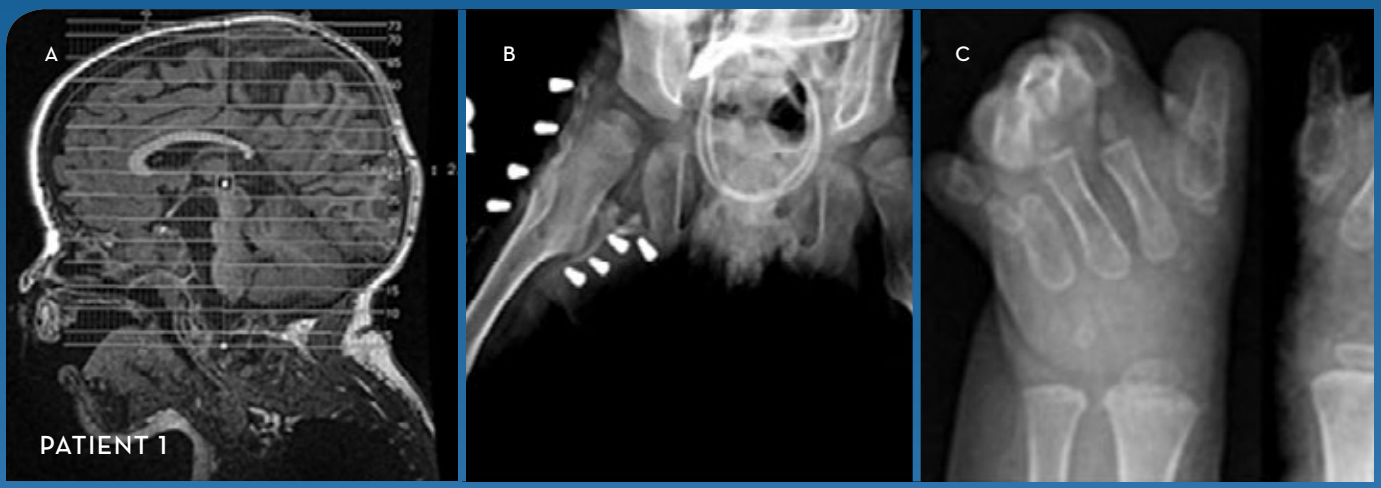
Following up on the decades-long series of investigative reports, in 2015, Dr. Kaplan, Dr. Shore, and their colleagues at the McKay Orthopaedic Research Laboratory published an article that offered revelations on one of the rarest variants of FOP discovered to date.

The team reported in on a pair of unrelated patients who represent genetic variants of the standard FOP phenotype. Patients with ≥ 1 uncommon feature, in addition to the classic anatomic features of FOP, are categorized as "FOP-Plus." Patients with significant deviations from one or both features of the classic anatomic features are categorized as "FOP Variants." Patients with FOP variants are further subdivided into those who have minimal to no obvious malformation of the great toes and/or adult-onset progressive heterotopic ossification, and those who have severe malformations of the great toes and/or widespread reduction deficits of the digits on the hand and the foot. Such abnormalities are extremely rare; fewer than 2% of those thought to have FOP are defined as FOP-Plus and FOP Variants.

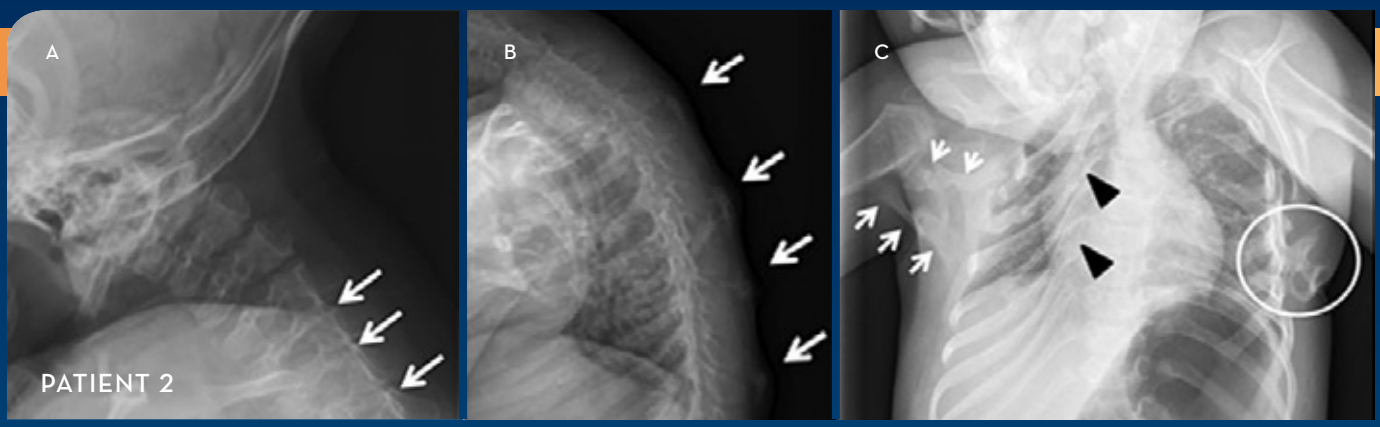
Patient Examination

Patient 1 was a 16-month-old girl who was the result of *in vitro* fertilization, was born at 34 weeks gestation, and had macrocephaly, microretrognathia, dysmorphic facial features, and forelimb reduction with no nail anomalies. She had no family history of congenital anomalies or heterotopic ossification. A biopsy of a soft tissue mass on the back exhibited fibroproliferative neoplasm. In addition to the aforementioned anomalies, she presented with a shunted hydrocephalus; hypoplasia of the brainstem (confirmed by MRI); tethered spinal cord; sparse hair; small malformed teeth; sensorineural hearing loss; dysconjugate gaze; gross developmental motor delay; decreased range of motion of the shoulders, elbows, and hips; and soft tissue masses on the neck, back, and buttocks. Subsequent physical examination revealed additional anomalies including functionally ankylosed shoulders, bilateral proximal medial tibial osteochondromas, and a fixed, rigid neck with chin-on-chest deformity. Radiographs revealed orthotopic fusion of the lower cervical vertebrae and confirmation of the physical examination findings. A severe variant of FOP was suspected. Gene sequencing showed a previously unknown heterozygous mutation in the ACVR1 gene: (ACVR1; R258G).

Patient 2 was an 11-month-old girl who was the result of natural fertilization and was born (by cesarean) at 38 weeks gestation. She previously had heart surgery. There was no family history of congenital anomalies or heterotopic ossification. She had trigonocephaly, scaphocephaly, frontal suture fusion, dysmorphic facial features, microretrognathia, low-set dysmorphic ears, hypertelorism, left renal duplication, 4-limb reduction anomalies (absence of middle and distal phalanges of the hands and feet) with no nails, soft tissue masses on the neck and parietal regions, gross motor delay, and female genitalia with asymmetric labia and hypoplasia of the clitoris (diagnosed as gonadal dysgenesis). Radiographs revealed heterotopic ossification in the area of the previous heart surgery, skeletal muscle atrophy with low-grade fibroblastic proliferation, malformations of the cervical vertebrae and ribs, short broad femoral necks with bilateral dysplasia of the hips, proximal medial tibial osteochondromas, and widespread heterotopic ossification in the neck, back, and shoulders. An MRI showed agenesis of the corpus callosum. Her karyotype was 46, XY. A severe variant of FOP was suspected. Following gene sequencing, this patient, too, exhibited the previously unknown ACVR1; R258G heterozygous mutation.



(A) Hypoplasia of the brain stem (including the pons and cerebellum) as well as a ventriculo-peritoneal shunt (for treatment of congenital hydrocephalus) are noted. (B) Anteroposterior radiograph of the pelvis reveals short broad femoral necks, bilateral dysplasia of the hips, and extensive bridging heterotopic ossification of the right hip (arrows). Also note ventriculo-peritoneal shunt. (C) Anteroposterior radiographs of hands reveal severe reduction deficits of all digits with multiple malformations. Also note osteochondroma of left distal radius.



(A) Lateral radiograph of cervical spine at 13 months of age reveals orthotopic ankylosis of the posterior elements of C4-C5, C5-C6, and C6-C7 (arrows). (B) Lateral radiograph of the thoracic spine at 22 months of age shows multiple areas of heterotopic ossification (arrows). (C) Anteroposterior radiograph of the chest at 22 months of age shows severe right rib malformations (black arrowheads), severe restrictive heterotopic ossification of the right chest and shoulder girdle (white arrows), and osteochondromas of the left chest wall (white circle).

Conclusion

Both patients with severe variant FOP had similar anomalies, suggesting that the single nucleotide substitution mutation, R258G, was associated with dysfunction of the ACVR1 kinase receptor. The association of the R258G of ACVR1 mutation with FOP had never before been described in the literature. The high fidelity phenotype–genotype relationship in these unrelated children with the most severe FOP phenotype reported to date suggests that the shared features are due to the dysregulated activity of the mutant kinase during development and postnatally, and provides vital insight into the structural biology and function of ACVR1 as well as the design of small molecule inhibitors.

Several years after reaching this milestone, however, the Penn team noted that merely knowing the genetic precipitant for FOP was "often insufficient to decipher the pathophysiology of the disorder or to effectively treat those affected." Thus, at the Center for Research in FOP and Related Disorders at Penn, clinical research continues to further elucidate the phenotype–genotype relationships that propel the pathology of FOP. Since 2006, it has become clear that the explanation for FOP involves more than a single aberrant mutation. As of today, 11 variant mutations at the ACVR1 protein have been linked to FOP, as have a number of other factors that contribute to the generation, promotion and episodic progression of the disorder in the setting of genetic mutation. These include inflammatory and immunological factors, vascular-derived mesenchymal stem cells and a hypoxic lesional microenvironment.

Because FOP is extremely rare to begin with, studying its variants provides the opportunity to probe phenotype–genotype relationships that have revelational implications for deciphering the role of the BMP pathway in normal physiology, as well as for understanding how single amino acid substitutions alter the function of a highly conserved protein kinase receptor. Moreover, studies on FOP variants have shown that seemingly small variations in genotype can give rise to large variations in phenotype that provide important insight into the molecular mechanisms of FOP and BMP signaling. Understanding the specific effect of a missense mutation on ACVR1 function could eventually help guide the design of pharmacologic agents that will modify or prevent the postnatal consequences of the disease.

Discussion

All of the classic and common variable features of FOP as well as many, if not all, of the atypical features evaluated in this study could plausibly be ascribed to dysregulation of the BMP signaling pathway. However, further studies of BMP signaling in animal models of classic and variant FOP will be critical to address these questions, and are in development.

At this time, there is no definitive treatment for patients with FOP or its variants, however the continued research conducted by Penn and others is paramount to understanding the role of the ACVR1 gene mutation and its many variants. Pinpointing exactly how these mutations cause the mirad abnormalities associated with FOP will help to guide researchers to possible treatments and perhaps a cure for this severely debilitating disease. Once identified, for example, disease-causing mutations in ACVR1 can be used as pharmaceutical targets for the development of signal transduction inhibitors (STIs) and other therapeutic strategies.

Postnatal inhibition of ACVR1, in addition, could have a significant role in treating other common acquired disorders of orthotopic and heterotopic ossification. Conversely, the mutation(s) of FOP and its variants could be harnessed for tissue engineering to form new bone for therapeutic applications. ■

Reference // Kaplan FS, Kobori JA, Orellana C, Calvo I, Rosello M, Martinez F, Lopez B, Xu M, Pignolo RJ, Shore EM, Groppe JC. Multi-system involvement in a severe variant of fibrodysplasia ossificans progressiva (ACVR1 c.772G>A; R258G): A report of two patients. Am J Med Genet A. 2015 Oct;167A(10):2265-71. doi: 10.1002/ajmg.a.37205. Epub 2015 Jun 11.

PENN ORTHOPAEDICS
SPECIALTY HIGHLIGHTS

Penn Orthopaedics provides patients with the most advanced comprehensive diagnostic, surgical and rehabilitative treatments in nine specialties. The following are recent highlights from each specialty—including the renowned McKay Orthopaedic Research Laboratory and The Children's Hospital of Philadelphia (CHOP).

12 LOCATIONS

72 FULL-TIME CLINICAL/RESEARCH/ CHOP FACULTY

12,204 TOTAL CASES

96,234 PATIENT VISITS

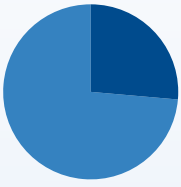
>> Foot and Ankle

- In conjunction with L. Scott Levin, MD, FACS, the Foot and Ankle service continues to utilize vascularized bone grafts and combined bone and myocutaneous flaps for reconstruction of failed procedures referred to Penn from multiple outside institutions.
- In conjunction with the Department of Radiology, the Foot and Ankle service continues to conduct research utilizing the new technology of standing, weight bearing CAT scans for evaluation of the clinical results of surgery.
- The Foot and Ankle section has added Kathryn O'Connor, MD, who will provide a clinical presence at the Penn Musculoskeletal Center at Penn Medicine University City.
- Daniel C. Farber, MD, continues to serve as member at large to the Board of the American Academy of Orthopaedic Surgeons. The faculty has also participated on committees for the American Academy of Orthopaedic Surgeons and the American Orthopaedic Foot and Ankle Society.
- Keith L. Wapner, MD, has presented 21 talks at national and international meetings including the Japanese Orthopedic Foot and Ankle Society, the Brazilian Orthopedic Foot and Ankle Society and the 2nd Qatar Foot and Ankle Conference.

Section Chief:
Keith L. Wapner, MD

2015 PATIENT VISITS: 15,837

2015 PATIENT SURGICAL VOLUMES: 1,039



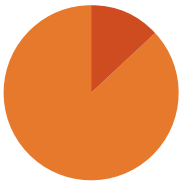
- Inpatient: 275
- Outpatient: 764

Selected Articles // Mao H, Shi Z, Wapner KL, Dong W, Yin W, Xu D. **Anatomical study for flexor hallucis longus tendon transfer in treatment of Achilles tendinopathy.** Surg Radiol Anat. 2015 Aug;37(6):639-47. doi: 10.1007/s00276-014-1399-y. Epub 2014 Dec 27. // Asai S, Otsuru S, Candela ME, Cantley L, Uchibe K, Hofmann TJ, Zhang K, Wapner KL, Soslowsky LJ, Horwitz EM, Enomoto-Iwamoto M. **Tendon progenitor cells in injured tendons have strong chondrogenic potential: the CD105-negative subpopulation induces chondrogenic degeneration.** Stem Cells. 2014 Dec;32(12):3266-77. doi: 10.1002/stem.1847. // Lee DY, Seo SG, Kim EJ, Kim SJ, Lee KM, Farber DC, Chung CY, Choi IH. **Correlation between static radiographic measurements and intersegmental angular measurements during gait using a multisegment foot model.** Foot Ankle Int. 2015 Jan;36(1):1-10. doi: 10.1177/1071100714559727. Epub 2014 Nov 17. // O'Connor KM, Johnson JE, McCormick JJ, Klein SE. **Correlation of Clinical, Operative, and Histopathologic Diagnosis of Interdigital Neuroma and the Cost of Routine Diagnosis.** Foot Ankle Int. 2016 Jan;37(1):70-4. doi: 10.1177/1071100715603118. Epub 2015 Aug 28.

>> Hand and Wrist

Section Chief:
David J. Bozentka, MD

2015 PATIENT VISITS:
12,170



2015 PATIENT SURGICAL VOLUMES: 1,668

- Inpatient: 221
- Outpatient: 1,447

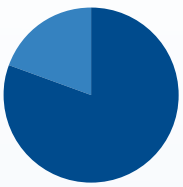
- This summer, Penn Orthopaedics Chair L. Scott Levin, MD, FACS, led a team of Penn and Children's Hospital of Philadelphia surgeons to perform the world's first pediatric bilateral hand vascularized composite allotransplantation in eight-year-old Zion Harvey.
- After completing his hand surgery fellowship at the University of Cincinnati, Benjamin L. Gray, MD, joined the Hand and Wrist service in 2015.
- Dr. Levin is the principle investigator for the multicenter Axogen-sponsored study: A Multicenter, Prospective, Randomized, Subject and Evaluator Blinded Comparative Study of Nerve Cuffs and Advance Nerve Graft Evaluating Recovery Outcomes for the Repair of Nerve Discontinuities currently enrolling patients.
- Penn Hand and Wrist has added a second Hand and Microsurgical fellowship.

Selected Articles // Pulos N, Bozentka DJ. Carpal Ligament Anatomy and Biomechanics. Hand Clin. 2015 Aug;31(3):381-7. doi: 10.1016/j.hcl.2015.04.007. // Pulos N, Bozentka DJ. **Management of complications of flexor tendon injuries.** Hand Clin. 2015 May;31(2):293-9. doi: 10.1016/j.hcl.2014.12.004. Epub 2015 Feb 28. Review. // Silvestre J, Lin IC, Levin LS, Chang B. **Experience in Hand Surgery for Graduates of Three Surgical Specialties.** Plast Reconstr Surg. 2015 Oct;136(4 Suppl):24. doi: 10.1097/01.prs.0000472302.77696.0f. No abstract available. // Silvestre J, Levin LS, Serletti JM, Chang B. **The Plastic Surgery Hand Curriculum.** Plast Reconstr Surg. 2015 Dec;136(6):1239-47. doi: 10.1097/PRS.0000000000001784. // Fisher MB, Belkin NS, Milby AH, Henning EA, Bostrom M, Kim M, Pfeifer C, Meloni G, Dodge GR, Burdick JA, Schaer TP, Steinberg DR, Mauck RL. **Cartilage repair and subchondral bone remodeling in response to focal lesions in a mini-pig model: implications for tissue engineering.** Tissue Eng Part A. 2015 Feb;21(3-4):850-60. doi: 10.1089/ten.TEA.2014.0384. Epub 2014 Dec 11.

>> Joint Replacement

Section Chief:
Charles L. Nelson, MD

2015 PATIENT VISITS:
23,843



2015 PATIENT SURGICAL VOLUMES: 3,861

- Inpatient: 3,117
- Outpatient: 744

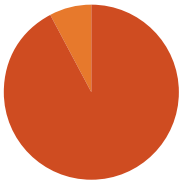
- In May, Penn Orthopaedics held the 3rd Annual Philadelphia Revision Hip and Knee CME Course in partnership with the International Congress for Joint Replacement. Leaders from across the country joined the Penn faculty in a two-day didactic and hands-on cadaveric course at the Penn Human Tissue Lab focusing on delivering key knowledge and skills in caring for patients with failed hip and knee replacements.
- Also in May, 2015, Penn Orthopaedics, in partnership with the International Congress for Joint Replacement, organized the 3rd Annual ORTI—East Orthopaedic Resident and Fellow Skills Course—designed to provide residents with basic concepts and skills for primary hip and knee replacement.
- Gwo-Chin Lee, MD received the James Rand Award for a clinical research study at the American Association of Hip and Knee Surgeons Meeting in November, 2014.
- Penn Joint Replacement has added a second Adult Reconstruction fellowship who cover approximately 3000 joint replacements within the Penn health system.

Selected Articles // Liu, J, Elkassabany, NE, Poultsides, L, Nelson, CL, Neuman, MD, Memtsoudis, SG. **Staging Bilateral Total Knee Arthroplasty During the Same Hospitalization: The Impact of Timing.** J. Arthrop, Epub ahead of print, 2015, Feb 17. // Nelson, CL, Elkassabany, N, Kamath, AF, Liu, J. **Malnutrition more so than obesity increases complications after total knee arthroplasty.** Clin Orthop Rel Res, Epub ahead of print, 2015, May 21. // Nelson, CL, Vanushkina, M, Irgit, K, Strohecker, K, Bowen, TR. **Stemmed femoral implants show lower failure rates in revision total knee arthroplasty.** The Knee, Epub ahead of print, 2015, May 29. // Courtney, PM, Rozell, JC, Melnic, CM, Sheth, NP, Nelson, CL. **The effect of malnutrition and obesity on complication rates following primary joint replacement.** J Surg Orthop Adv, In press, 2015. // Walls, JD, Abraham, J, Nelson, CL, Kamath, AF, Elkassabany, N, Liu, J. **Hypoalbuminemia more than Morbid Obesity is an Independent Predictor of Complications after Total Hip Arthroplasty.** J Arthrop, Epub ahead of print, July, 2015. // Sheth NP Donegan DJ, Foran JRH, and Sugarman J. **Global Health and Orthopaedic Surgery: A Call for International Morbidity and Mortality Conferences.** Int J of Surgery Case Rep. 2015; 6C:63-67. // Evangerlista, P, Kamath, AF, Aversano, FJ, Silvestri, J, Lee, GC, Nelson, C. **Ceramic-Ceramic Hip Arthroplasty for Osteonecrosis: Average 5-Year Follow-up in Patients Less than 50 Years of Age,** Bull Hosp Joint Dis: 73: 42-5, 2015. // Ibrahim, S, Jorgenson, E, Richardson, DM, Nelson, CL, Steinman, M. **Race, Rehabilitation, and 30-day Readmission after Elective Total Knee Arthroplasty,** Geriatric Orthop and Rehabil, 6: 303-310, 2015.

>> Neuro-Orthopaedics

Section Chief:
Keith D. Baldwin, MD, MPH, MSPT

2015 PATIENT VISITS: 3,340



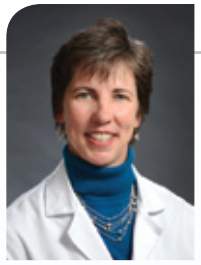
2015 PATIENT SURGICAL VOLUMES: 384

- Inpatient: 352
- Outpatient: 29

- Penn Neuro-Orthopaedics has initiated a strategic transition plan for complex multidisciplinary patients with spina bifida and cerebral palsy to care for their needs into adulthood, both musculoskeletal and non-musculoskeletal. This plan also allows for seamless integration of surgery into recovery from strokes and brain injuries.
- Penn Neuro-Orthopaedics represented Penn Orthopedics at the National Orthopedic Leadership council to help drive health policy in Washington, D.C.
- As part of Penn Orthopaedics' continuing mission to push advanced, multidisciplinary musculoskeletal treatment for patients, treatment plans now integrate gait analysis of complex gait disorders for surgical and non-surgical decision making in collaboration with Penn Physical Medicine and Rehabilitation.
- Our treatment for spastic equinovarus (neuromuscular club foot) has resulted in excellent one year outcomes, from 45.5 on the foot and ankle disability score to 72.9 at a year post-op, with walking speeds of approximately 1.5 times faster and many patients being able to walk independently when they were previously unable.

Selected Articles // Kolman SE, Ruzbarsky JJ, Spiegel DA, Baldwin KD. **Salvage Options in the Cerebral Palsy Hip: A Systematic Review.** J Pediatr Orthop. 2015 Apr 14. [Epub ahead of print] // Morris, Tyler R.; Keenan, Mary Ann; Baldwin, Keith. **Peroneal nerve palsy Current Orthopaedic Practice.** 26(2):155-159, March/April 2015. // Christos D. Photopoulos, MD; Surena Namdari, MD, MSc; Keith D. Baldwin, MD, MPH, MSPT; Mary Ann Keenan, MD. **Decision-Making in the Treatment of the Spastic Shoulder and Elbow: Tendon Release Versus Tendon Lengthening.** JBJS Reviews, 2014 Oct; 2 (10): e3.

>> Orthopaedic Oncology

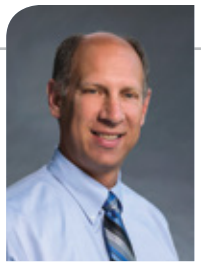


Section Chief:
Kristy L. Weber, MD

- For 2015-16, Penn Orthopaedic Oncology organized and funded three Penn Sarcoma Program Pilot Grants for Research at Penn Medicine, Penn Veterinary Medicine, and the Children's Hospital of Philadelphia (CHOP).
- Starting in February 2015, Penn Orthopaedic Oncology now participates fully in the orthopaedic oncology service at CHOP.
- Penn Orthopaedic Oncology held its inaugural Steps for a Cure event on July 26 as the first event orchestrated by the newly developed Sarcoma Advocacy group led by Penn sarcoma patients and their families.
- Kristy L. Weber, MD, Chief of Penn Orthopaedic Oncology, holds a number of national leadership roles including ORS (Orthopaedic Research Society) Secretary, AOA (American Orthopaedic Association) Critical Issues Committee Chair and RJOS (Ruth Jackson Orthopaedic Society) President.

Selected Articles // Cannada LK, Wolf JM, Lajam C, Weber KL: **Guide for Women in Orthopaedic Surgery (Section 2 Editor).** Ruth Jackson Orthopaedic Society/Zimmer, (2015). // Weber KL: **Interview with Kristy Weber, Orthopaedic Oncologist IN Being a Woman Surgeon: Sixty Women Share Their Stories**, Ed. Preeti R. John., Gordian Knot Books, p.261-263, 2015 // Weber KL and Arkader A: **Sports Injury or Something Worse?**, Children's Doctor (published at CHOP), August, 2015 // Kim EY, Chapman TR, Ryu S, Chang EL, Galanopoulos N, Jones J, Kubicky CD, Lee CP, Teh BS, Traughber BJ, Van Poznak C, Vassil AD, Weber K, Lo SS: **ACR Appropriateness Criteria: Non-Spine Bone Metastases.** J Palliative Med 18:1-7, 2015.

>> Research



Section Chief:
Louis J. Soslowsky, PhD

- Louis J. Soslowsky, PhD, was appointed Associate Dean for Research Core Facilities in the Perelman School of Medicine. Robert L. Mauck, PhD was appointed Director of the McKay Laboratory for Orthopaedic Surgery Research. Ling Qin, PhD became a tenured Associate Professor. Eileen M. Shore, PhD, was appointed to the Strategic Planning Committee of the American Society of Bone and Mineral Research (ASBMR), 2015. Chair, Research Agenda; Member, Global Initiatives.
- The Biedermann Orthopaedic Research Lab opened in June 2015. Penn Orthopaedics Research Engineer Michael W. Hast, PhD, was named the inaugural Director.
- 2015 marked the opening of the Penn Human Performance Lab. Penn has hired Josh Baxter, PhD, as Director of this state-of-the-art facility. The lab will include a team of Penn Musculoskeletal specialists using the most advanced tools and technology to evaluate patients to maximize individualized treatment plans for a variety of musculoskeletal injuries, disorders and diseases.
- Lachlan J. Smith, PhD mapped the transcriptome of intervertebral disc progenitor cells at key stages of embryonic development.
- Bob Pignolo serves as Primary Investigator on the first interventional trial, open-label extension, longitudinal natural history study in FOP, the latter two of which were initiated in 2015.
- Frederick S. Kaplan, MD, and Dr. Eileen Shore were recognized in the 250th Anniversary of the Perelman School of Medicine for discovery of the genetic mutation that causes Fibrodysplasia Ossificans Progressiva (FOP).

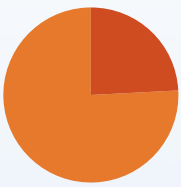
Selected Articles // Driscoll TP, Cosgrove BD, Heo SJ, Shurden ZE, Mauck RL, **"Cytoskeletal to Nuclear Strain Transfer Regulates YAP Signaling in Mesenchymal Stem Cells with Dynamic Loading,"** 2015, Biophysical Journal, 108(12): 2783–2793. // Connizzo BK, Freedman BR, Fried JH, Sun M, Birk DE, Soslowsky LJ., **Regulatory role of collagen V in establishing mechanical properties of tendons and ligaments is tissue dependent.** J Orthop Res. 2015 Jun;33(6):882-8. doi: 10.1002/jor.22893. Epub 2015 Apr 27. // Chandra A, Lin T, Zhu J, Tong W, Huo Y, Jia H, Zhang Y, Liu XS, Cengel K, Xia B, Qin L. J **PTH1-34 blocks radiation-induced osteoblast apoptosis by enhancing DNA repair through canonical Wnt pathway.** Biol Chem. 2015 Jan 2;290(1):157-67. // Gorth DJ, Lothstein KE, Chiaro JA, Farrell MJ, Dodge GR, Elliott DM, Malhotra NR, Mauck RL, Smith LJ. **Hypoxic regulation of functional extracellular matrix elaboration by nucleus pulposus cells in long-term agarose culture.** J Orthop Res. 2015 May;33(5):747-54. // Lindborg, C.M., Propert, K.J., Pignolo, R.J.: **Conservation of pro-longevity genes among mammals. Mechanisms of ageing and development** 146-148C: 23-27, 2015. // de Bakker C. M., Altman A. R., Tseng W. J., Tribble M. B., Li C., Chandra A., Qin L., Liu X. S., **μCT-based, *in vivo* dynamic bone histomorphometry allows 3D evaluation of the early responses of bone resorption and formation to PTH and alendronate combination therapy.** Bone. 2015; 73: 198-207.

>> Shoulder and Elbow

Section Chief:
David L. Glaser, MD

- Close collaboration with researchers from the McKay Research Laboratory has helped the shoulder and elbow service to form one of the largest shoulder research laboratories in the world.
- The shoulder and elbow service received six refereed research grants in 2015, including NIH, Veterans Affairs, health system and the industry grants.
- Penn shoulder and elbow faculty presented eight abstracts at national meetings, giving 15 talks at international, national, and regional meetings in 2015.

2015 PATIENT VISITS:
8,478



2015 PATIENT SURGICAL VOLUMES: 999

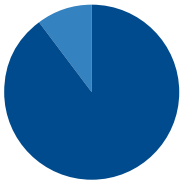
- Inpatient: 242
- Outpatient: 757

Selected Articles // Reuther, K.E., Tucker, J.J., Thomas, S.J., Vafa, R.P., Liu, S.S., Gordon, J.A., Caro, A.C., Yannascoli, S.M., Kuntz, A.F. and Soslowsky, L.J., 2015. **Effect of scapular dyskinesis on supraspinatus repair healing in a rat model.** Journal of Shoulder and Elbow Surgery, 24(8), pp.1235-1242. // Reuther, K.E., Thomas, S.J., Tucker, J.J., Vafa, R.P., Gordon, J.A., Liu, S.S., Caro, A.C., Yannascoli, S.M., Kuntz, A.F. and Soslowsky, L.J., 2015. **Overuse activity in the presence of scapular dyskinesis leads to shoulder tendon damage in a rat model.** Annals of biomedical engineering, 43(4), pp.917-928. // Rooney, S.I., Tobias, J.W., Bhatt, P.R., Kuntz, A.F. and Soslowsky, L.J., 2015. **Genetic Response of Rat Supraspinatus Tendon and Muscle to Exercise.** PloS one, 10(10), p.e0139880. // Tucker, J.J., Riggin, C.N., Connizzo, B.K., Mauck, R.L., Steinberg, D.R., Kuntz, A.F., Soslowsky, L.J. and Bernstein, J., 2016. **Effect of overuse- induced tendinopathy on tendon healing in a rat supraspinatus repair model.** Journal of Orthopaedic Research, 34(1), pp.161-166. // Horneff JG, Hsu JE, Voleti PB, O'Donnell J, Huffman GR: **Propionibacterium acnes infection in shoulder arthroscopy patients with postoperative pain.** J Shoulder Elbow Surg. Elsevier, 2015 Notes: 10.1016/j.jse.2015.03.008. // Jiabin Liu, Kenneth A Richman, Samuel R Grodofsky, Siya Bhatt, George Russell Huffman, John D Kelly, David L Glaser, Nabil Elkassabany. **Is there a dose response of dexamethasone as adjuvant for supraclavicular brachial plexus nerve block? A prospective randomized double-blinded clinical study.** Journal of Clinical Anesthesia, Volume 27, Issue 3, May 2015, Pages 237–242.

>> Spine

Section Chief:
Vincent Arlet, MD

2015 PATIENT VISITS:
2,133



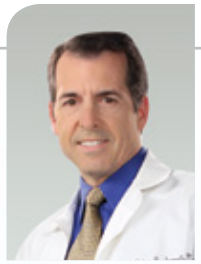
2015 PATIENT SURGICAL VOLUMES: 429

- Inpatient: 386
- Outpatient: 43

- Vincent Arlet, MD, chief of the Spine service, presented at a number of events and courses in 2015 including the French Spine Society; the European Spine Journal/AO Spine Meet the expert forum on Spine Osteotomies in Barcelona, Spain; the Depuy Synthes Spine Course; and the Inaugural Philadelphia Spine Summit where both he and fellow Penn Orthopaedics spine surgeon Harvey E. Smith, MD served as course directors. Dr. Smith also presented at the AAOS instructional course lecture in Las Vegas, NV and was named chair of the Biologics Committee for the Association for Collaborative Spine Research Subspecialty Group Meeting in Miami, FL.
- Dr. Smith was also the recipient of numerous grants in 2015. These include grants to study the Impact of Pre-Culture and In Vivo Remobilization on Engineered Disc Replacement; and the Regeneration of the Intervertebral Disc using Notochord-Derived Cells and Mesenchymal Stem Cells. 2015's grants also included a career development award for Biomedical Laboratory Research & Development Service of the VA Office of Research and Development.

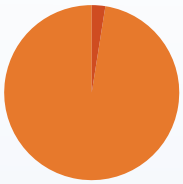
Selected Articles // Martin John T, Collins Christopher M, Ikuta Kensuke, Mauck Robert L, Elliott Dawn M, Zhang Yeija, Anderson D Greg, Vaccaro Alexander R, Albert Todd J, Arlet Vincent, Smith Harvey E: **Population average T2 MRI maps reveal quantitative regional transformations in the degenerating rabbit intervertebral disc that vary by lumbar level.** Journal of orthopaedic research: official publication of the Orthopaedic Research Society Oct 2014 // Nestorovski D, Milby AH, Gopal PP, Smith LJ, Smith HE, Malhotra NR: **Combined Treatment Approach for Rare Occipito-cervical Spinal Lesions: leiomyosarcoma.** Journal of Spine and Neurosurgery 2014 Notes: In Press // Zannikos S, Lee L, Smith HE.: **Minimum Clinically Important Difference and Substantial Clinical Benefit: Does one size fit all diagnoses and patients?** Seminars in Spine Surgery 2014 Notes: In Press. // Woodward CC, Milby AH, Smith HE: **Future Advances for Treating Lumbar Disc Herniation and Degeneration: Nucleus Replacement, Annular Repair, and Biologics.** Seminars in Spine Surgery 2015 // Zhang Y, Chee A, Shi P, Adams SL, Markova DS, Anderson DG, Smith HE, Deng Y, Plastaras CT, An HS: **Intervertebral Disc Cells Produce Interleukins Found in Patients with Back Pain.** AJPMR 2015.

>> Sports Medicine



Section Chief:
Brian J. Sennett, MD

2015 PATIENT VISITS:
13,668



2015 PATIENT SURGICAL VOLUMES: 1,491

- Inpatient: 38
- Outpatient: 1,453

- The inaugural Penn Medicine Advances in Throwing symposium was held on January 31, 2015. The symposium featured a multidisciplinary approach to the evaluation and treatment of pathologies related to the throwing athlete. John D. Kelly, IV, MD, serves as the Director of the Penn Throwing Center.
- John Vasudevan, MD, served as medical director for the Tri-rock Philly Triathlon and Rahul Kapur, MD, served as medical director of the Philadelphia Love Run Half-Marathon held on March 29, 2015. Brian J. Sennett, MD, Chief of Penn Sports Medicine, continues to serve as an Orthopaedic Consultant to the Philadelphia 76ers.
- The OREF awarded a prestigious New Investigator Grant to principal investigator Miltiadis Zgonis, MD. This grant provides funding to pilot several studies investigating novel strain transfer mechanisms within the meniscus and will add to our knowledge of how the native meniscus works within the body and better inform tissue-engineered scaffold designs for meniscus repair or replacement.
- The Penn Cartilage Symposium has become an international course organized and run by Course Directors James L. Carey, MD, MPH and Robert L. Mauck, PhD. This year's course, entitled "New Directions in Osteochondral Repair and Regeneration," was attended by a record 173 participants, including physicians, scientists, mid-level providers, nurses, veterinarians, physical therapists, athletic trainers, and students.

Selected Articles // Opar D, Drezner J, Shield A, Williams M, Webner D, Sennett B, Kapur R, Cohen M, Ulager J, Cafengiu A, Cronholm PF. **Acute injuries in track and field athletes: a 3-year observational study at the Penn Relays Carnival with epidemiology and medical coverage implications.** Am J Sports Med. 2015 Apr;43(4):816-22. PMID:25560540 // Pontillo M, Spinelli BA, Sennett BJ. **Prediction of in-season shoulder injury from preseason testing in division I collegiate football players.** Sports Health. 2014 Nov;6(6):497-503. PMID:25364482 // AAOS Monograph: Osteochondritis Dissecans (published by the American Academy of Orthopaedic Surgeons). **Experts on osteochondritis dissecans explain the best methods for clinical evaluation and diagnosis, as well as surgical treatment options for skeletally immature and skeletally mature patients.** James L. Carey, MD, MPH served as editor of this monograph. // Shea KG, Carey JL, Richmond J, Sandmeier R, Pitts RT, Polousky JD, Chu C, Shultz SJ, Ellen M, Smith A, LaBella CR, Anderson AF, Musahl V, Meyer GD, Jevsevar D, Bozic KJ, Shaffer W, Cummins D, Murray JN, Patel N, Shores P, Woznica A, Martinez Y, Gross L, Severino K; American Academy of Orthopaedic Surgeons. **The American Academy of Orthopaedic eons evidence-based guideline on management of anterior cruciate ligament injuries.** J Bone Joint Surg Am. 2015; 97:672-4.

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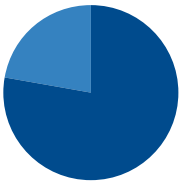
>> Trauma and Fracture

Section Chief:
Samir Mehta, MD

2015 PATIENT VISITS:
7,533



2015 PATIENT SURGICAL VOLUMES: 1,655

- Inpatient: 1,288
- Outpatient: 367

- Samir Mehta, MD, Chief of Orthopaedic Trauma at Penn Orthopaedics, was named Chair of the Education Committee for the Council for Orthopaedic Residency Directors of the American Orthopaedic Association, named clinical advisor to the Biedermann Lab for Orthopaedic Research at the University of Pennsylvania and also joined the AO Foundation Community Development Committee.
- Derek J. Donegan, MD, was named Medical Director of Eastern University Athletic Training Department.
- Jaimo Ahn, MD, PhD, joined the Translational Musculoskeletal Research Center, VA Medical Center, Philadelphia, PA. as an investigator, was elected to the American Orthopaedic Association (AOA)., and is also on the Steering Committee for the Combined Degree and Physician Scholar Programs.
- Drs. Mehta, Donegan, and Ahn have also served as faculty for the AO in several national and international courses. Dr. Ahn also received the Chairman's Award for Extraordinary Service, Orthopaedic Surgery, University of Pennsylvania.
- The Penn Orthopaedics Trauma team were also the recipients of a number of Grants including:
  - » Foundation for Orthopaedic Trauma grant, PI, Acceleration of Geriatric Fracture Healing by Local Activation of NICD
  - » 2015 NIH/NIAMS RO1, Co-I, Regulation of fracture healing by TSP2
  - » FDA-approved DOD grant investigating the use of a novel compound in the treatment of infection after orthopaedic trauma.

Selected Articles // Austin DC, Donegan D, Mehta S. **Low Complication Rates Associated with the Application of Lower Extremity Traction Pins.** J Orthop Trauma. 2015 Mar 14. [Epub ahead of print] // Yoon RS, Donegan DJ, Liporace FA. **Reducing subtrochanteric femur fractures: tips and tricks, do's and don'ts.** J Orthop Trauma. 2015 Apr;29 Suppl 4:S28-33 // Florschutz AV, Donegan DJ, Haidukewych G, Liporace FA. **Plating of femoral neck fractures: when and how?** J Orthop Trauma. 2015 Apr;29 Suppl 4:S1-3. // Ahn J, Achor T. **How to be an effective teacher.** Journal of Orthopaedic Trauma 28 Suppl 9:S15-17. 2014. // Lopas LA, Belkin NS, Mutyaba PL, Gray CF, Hankenson KD, Ahn J. **Geriatric mice demonstrate decreased fracture healing potential associated with compromised callus expansion and resultant bone volume.** Clinical Orthopaedics and Related Research. 472:3523-3532, 2014. // Ashley JW, Hankenson KD, Ahn J(shared). **Notch signaling promotes osteoclast maturation and resorptive activity.** Journal of Cellular Biochemistry, Epub Apr 25 (DOI: 10.1002/jcb.25205), 2015. // Ahn J(shared), Sagi HC, Ciesla D, Collinge C, Molina C, Obremskey WT, Guillamondegui O, and the Orthopaedic Trauma Association Evidence-Based Quality Value and Safety Committee. **Venous Thromboembolism Prophylaxis in Orthopaedic Trauma Patients: OTA Member Practice Patterns and OTA Expert Panel Recommendations.** In press. 2014. // Matuszewski PE, Kim TW, Gay AN, Mehta S. **Acute Operative Management of Humeral Shaft Fractures: Analysis of the National Trauma Data Bank.** Orthopedics. 2015 Jun;38(6):e485-9. // Krueger CA, Mehta S. **Trends in firearm safety-do they correlate with fewer injuries.** Curr Rev Musculoskelet Med. 2015 Sep;8(3):272-5. // Bibbo C, Nelson J, Fischer JP, Wu LC, Low DW, Mehta S, Kovach SJ. **Lower Extremity Limb Salvage After Trauma: The Versatility of the Anterolateral Thigh Free Flap.** J Orthop Trauma. 2015 May 9. // Hannigan GD, Pulos N, Grice EA, Mehta S. **Current Concepts and Ongoing Research in the Prevention and Treatment of Open Fracture Infections.** Adv Wound Care (New Rochelle). 2015 Jan 1;4(1):59-74. // Matuszewski PE, Dombroski D, Lawrence JT, Esterhai JL Jr, Mehta S. **Prospective intraoperative syndesmotic evaluation during ankle fracture fixation: stress external rotation versus lateral fibular stress.** J Orthop Trauma. 2015 Apr;29(4):e157-60.

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