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LOWER EXTREMITY REVIEW

April 16 / volume 8 / number 4

CONFERENCE COVERAGE:
2016 AAOP Annual Meeting & Scientific Symposium

Shoring Up the Rotation:

The importance of hip mechanics in pitching

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GAIT ANALYSIS
ACHILLES EXPERTS PONDER EFFECTS OF HEEL ELEVATION

FOOT HEALTH
INFLUENCE OF ESTROGEN ON THE PLANTAR FASCIA

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18 COVER STORY

Shoring up the rotation:

THE IMPORTANCE OF HIP MECHANICS IN PITCHING

Over the course of a season, collegiate pitchers undergo changes in hip rotational range of motion and hip strength—changes that could increase injury risk or negatively affect performance.

By Giorgio Zeppieri Jr, MPT, SCS, CSCS

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Lower Extremity Review informs healthcare practitioners on current developments in the diagnosis, treatment, and prevention of lower extremity injuries. LER encourages a collaborative multidisciplinary clinical approach with an emphasis on functional outcomes and evidence-based medicine. LER is published monthly, with the exception of a combined November/ December issue and an additional special issue in December, by Lower Extremity Review, LLC.

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out on a limb: Ingenuity targets activity

Diabetic foot specialists have a range of options for managing diabetic foot ulcers—from offloading devices to skin substitutes to surgical procedures. But sometimes the most effective treatment is the one that compels an active patient to simply slow down.

Monitoring and managing patient activity levels was a recurring theme at this year's Diabetic Limb Salvage (DLS) Conference, held in late March in Washington, DC. Given that this event is primarily about vascular and orthopedic surgical techniques, the fact that the discussion circled back to patient activity levels multiple times underscores its clinical significance.

It's no secret that the effects of weightbearing—and dynamic weightbearing in particular—undermine the wound healing process in a patient with a diabetic ulcer on the plantar surface of the foot. But, because patients are notoriously noncompliant with instructions to stay off their feet, ulcer healing efforts have focused on designing devices that will offload the ulcer site even under weightbearing conditions. And the total contact cast (TCC) has always been considered the gold standard for offloading because it is associated with better ulcer healing than other offloading techniques.

But experts say offloading is only part of why TCCs are effective. Another reason is that they make it difficult for patients to walk. With a cast on one leg, patients are forced to walk more slowly, with shorter, less frequent steps. This decrease in activity is, essentially, another way of offloading the ulcer.

Lower extremity professionals have long known this to be true, dating back half a century to diabetic foot care pioneer Paul Brand, MD, who died in 2003.

"Paul Brand used to say if you could tether people's feet to alter their gait, you would get effective offloading," Lawrence A. Lavery, DPM, MPH, a professor of plastic surgery at the University of Texas Southwestern Medical Center in Dallas, said during a DLS presentation.

Now, with the availability of activity monitoring technologies, experts say it's time to once again start emphasizing that aspect of the offloading equation.

"Activity monitors used to be exotic," said DLS presenter David G. Armstrong, DPM, MD, PhD, a professor of surgery at the University of Arizona in Tucson, gesturing at his smart phone. "Now they're things we use every day."

Hardly limited to virtual pedometers, this new generation of monitoring systems includes instrumented insoles or mats that detect increases in plantar pressure or temperature—both of which are associated with excessive activity—in at-risk patients and wirelessly send that information to clinicians. Armstrong described such systems as being "like OnStar for your body."

> Armed with new ways to monitor patient activity, diabetes experts say it's time to revisit that aspect of the offloading equation.

A DLS poster described a study done at the VA Medical Center in Phoenix, AZ, involving a foot mat designed to allow in-home foot temperature monitoring. In patients with recently healed ulcers, the mat detected a temperature difference between the affected and unaffected feet several weeks before the ulcers actually recurred.

A number of questions about such technologies remain unanswered, not least of which being who will pay for them. But the renewed focus on activity monitoring is one I think Brand would appreciate. It sure beats tethering a patient's feet together.

Jordana Bieze Foster, *Editor*

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Get stronger, live longer

But few older adults meet US guidelines

By Katie Bell

Strength training twice weekly, as recommended by two national organizations, can help reduce all-cause mortality in older US adults, but fewer than 10% meet that recommendation, according to a study from Pennsylvania State University.

Motivating older adults to participate in strength training is especially challenging in those with functional limitations—including difficulty standing for two hours, stooping, bending, or kneeling—who may benefit the most, said lead author Jennifer Kraschnewski, MD, MPH, an associate professor of medicine and public health sciences at Penn State College of Medicine in Hershey, PA.

"Most clinicians believe exercise is a good thing for nearly every patient. Unfortunately, it is more difficult to convince patients who have challenges being active, such as those with functional limitations, to do so," Kraschnewski said. "Strength training, for example, is excellent therapy following a fall. Unfortunately, people who have suffered a fall are less likely to be engaged in physical activity due to fear of falling."

Kraschnewski's team analyzed National Health Interview Survey (NHIS) data from 1997–2001, which were linked to death certificate data in the National Center for Health Statistics National Death Index, for more than 30,000 adults 65 years and older.

During the trial period, 9.6% of adults reported meeting the twice-weekly strength training guideline recommended by the

Continued on page 14

Soccer study finds steady rate of ACL injuries, disappointing 3-year outcomes

Despite advances in prevention and intervention, anterior cruciate ligament (ACL) injuries are no less common among professional soccer players now than in 2001, according to research from Sweden.

Perhaps even more concerning, the study found that, although 134 players who completed rehabilitation after ACL reconstruction returned to training, 4% suffered a re-rupture before returning to match play, and only 65% of the 104 players with complete follow-up data were still playing at an elite level three years later.

Investigators from Linkoping University analyzed ACL injury data for 78 men's professional soccer clubs between 2001 and 2015; players who suffered ACL injuries were tracked for three years after returning to training.

During the study period, 157 ACL injuries were recorded; the annual injury rate increased on average by 6%, although that increase was not statistically significant. Players who had ACL reconstruction took a median of 6.6 months to return to training and 7.4 months to return to play. The findings were epublished in late March by the *British Journal of Sports Medicine*. ler

–Jordana Bieze Foster

Source:

Walden M, Hagglund M, Magnusson H, Ekstrand J. ACL injuries in men's professional football: a 15-year prospective study on time trends and return-to-play rates reveals only 65% of players still play at the top level 3 years after ACL rupture. Br J Sports Med 2016 Mar 31. [Epub ahead of print]

Minimalist footwear, shortened stride affect loads differently during running

Running in minimalist footwear and running with a shorter stride length are both associated with reduced knee loads, but the two approaches differ in their effects on loading at the ankle, according to research from the University of Calgary in Canada.

In 14 healthy young adult men, investigators assessed the effect on joint loading of using minimalist versus control footwear or using a stride 10% shorter than the runner's preferred stride length.

Mean knee loads during running were 7.4% lower for minimalist shoes than for control shoes, and 15.7% lower for the shortened stride length than the preferred stride length. Ankle loads, however, were slightly higher for minimalist shoes than for control shoes but slightly lower for a shortened stride length than the preferred stride length. Load at the metatarsophalangeal joint was 10.2% higher for minimalist shoes than for control shoes. Combining minimalist shoes with a shortened stride length was associated with a 22% mean reduction in knee joint load.

The findings were epublished in March by the *Journal of Science and Medicine in Sport*. ler *–Jordana Bieze Foster*

Source:

Firminger CR, Edwards WB. The influence of minimalist footwear and stride length reduction on lower-extremity running mechanics and cumulative loading. J Sci Med Sport 2016 Mar 17. [Epub ahead of print]

Continued from page 13

American College of Sports Medicine (ACSM) and the American Heart Association (AHA), and 31.6% died. Participants who reported guideline-concordant strength training had a 46% lower likelihood of all-cause mortality than those who did not. Further, the association between strength training and mortality remained after adjustment for past medical history and health behaviors (which include level of physical activity). The findings were epublished in February by *Preventive Medicine*.

In a 2014 *Preventive Medicine* study, the same group analyzed NHIS data for 6763 older adults and found that those who complied with ACSM/AHA strength training guidelines were significantly less likely to have functional limitations than those who were noncompliant.

Nine functional limitations were studied, of which the most common were difficulty with standing for two hours, stooping, and bending or kneeling. Kraschnewski suggested that an inability to perform these actions can encourage sedentary behaviors.

Participants who met the strength training guidelines were less likely to have functional limitations. When each of the nine limitations were summed, 21.7% of participants with no limitations, 15.9% of participants with one to four limitations, and 9.8% of those with five to nine limitations met the guidelines.

The study authors concluded the potential for strength training to improve health is substantial, as those who have the most to gain from strength training participate the least.

"Often, the functional limitations can be improved with increased physical activity, although patients may need to start with physical therapy before advancing to a strength training program," she said.

However, Kraschnewski noted that barriers to compliance other than functional limitations may also exist.

"Other factors may include a fear of being active, which can be overcome with support. Additional barriers include disinterest in going to a gym, particularly for older adults, suggesting innovative approaches to provide exercise programming are needed," she said.

Wayne Westcott, PhD, instructor of exercise science at Quincy College in Quincy, MA, agreed that, as an older adult's strength increases, he or she will be able to perform related physical activities with less musculoskeletal stress. Westcott also noted that, because strength training lends itself to customization, it may actually be particularly well suited for older adults with functional limitations.

"Unlike performing bodyweight supporting activities, resistance exercise may be performed with external loads that may be adjusted to match the individual's present level of muscular strength," he said. ler

Sources:

Kraschnewski JL, Sciamanna CN, Poger JM, et al. Is strength training associated with mortality benefits? A 15 year cohort study of US older adults. Prev Med 2016;87:121-127.

Kraschnewski JL, Sciamanna CN, Ciccolo JT, et al. Is exercise used as medicine? Association of meeting strength training guidelines and functional limitations among older US adults. Prev Med 2014;66:1-5.

Stability for CP

AFO use benefits diplegic children

By Jordana Bieze Foster

In children with diplegic cerebral palsy (CP), wearing bilateral ankle foot orthoses (AFOs) that promote heel-first ground contact helps to improve stability and may reduce the risk of falls, according to research presented in March at the annual meeting of the American Academy of Orthotists and Prosthetists (AAOP) in Orlando, FL.

Photo courtesy of Cascade DAFO

In 191 children with diplegic CP, investigators from Gillette Children's Specialty Healthcare in St. Paul, MN, found that walking in bilateral AFOs was associated with a significantly greater margin of stability (MoS) in the backward direction—which is important when transitioning from one limb to another—compared with walking barefoot.

Importantly, the mean backward MoS for the children with CP while wearing AFOs did not differ significantly from the mean value for 76 typically developing children.

"This is where I started jumping up and down," said Michelle

Continued on page 16

Shifting position of shoe heel rocker affects ankle mechanics during gait

Varying the placement of a shoe heel rocker affects walking gait kinematics, even when the toe rocker placement is kept constant, according to research presented in March at the annual AAOP meeting in Orlando, FL.

Investigators from Georgia State University in Atlanta analyzed 18 healthy young women as they walked at a self-selected speed under three footwear conditions. For all conditions, the toe rocker apex was positioned at 63% of foot length and angled at 25°. The heel rocker apex was positioned at the medial malleolus, 1 cm anterior to the medial malleolus, or 1 cm posterior.

Knee and hip kinematics did not differ significantly between shoe conditions. However, significant differences between shoe conditions were observed with regard to ankle range of motion.

Most notably, the most anterior heel rocker position was associated with less ankle plantar flexion at heel strike and more dorsiflexion at midstance than the other two conditions, according to Zahra Safaeepour, PhD, a postdoctoral research associate in the university's Center for Pediatric Locomotion Sciences, who presented the findings. ler

Source:

Safaeepour Z. The effect of different placement of heel rockers on kinematics of lower-limb joints in healthy subjects. Presented at the 42nd annual meeting of the American Academy of Orthotists and Prosthetists, Orlando, FL, March 2016.

Kinematics at foot, not shank, respond to increases in heel-sole differential

Elevating the heel of a shoe significantly affects foot kinematics but not shank kinematics during gait, according to research from the UK presented in March at the annual AAOP meeting in Orlando, FL.

Investigators from the Child Development Center in Bangor, North Wales, retrospectively analyzed gait data for 10 women from a 2004 study conducted at Northwestern University in Chicago, which found that shoe heel height did not significantly affect rollover shapes during walking. The study participants walked while wearing shoes with a flat heel, a 37-mm heel, and a 71-mm heel.

The current analysis found that shank-to-vertical angle during the first half of the gait cycle did not differ significantly with shoe heel height, but that foot-to-horizontal angle increased with increasing heel-sole differential, or pitch.

"Shank kinematics do not change with the pitch of the footwear, but actual foot kinematics do change," said Elaine Owen, MSc, MSCP, a pediatric physiotherapist at the center, who presented the findings at the AAOP meeting. ler

Sources:

Owen E. The effect of walking in footwear with varying heel sole differentials on shank and foot segment kinematics. Presented at the 42nd annual meeting of the American Academy of Orthotists and Prosthetists, Orlando, FL, March 2016.

Hansen AH, Childress DS. Effects of shoe heel height on biologic rollover characteristics during walking. J Rehabil Res Dev 2004;41(4):547-554.

Continued from page 15

Hall, MS, CPO, a prosthetist-orthotist at Gillette, who presented the results of the Thranhardt Award-winning study at the AAOP meeting. "This is a clinically meaningful change, and I think as a clinician it makes intuitive sense."

The MoS measure is the distance between an individual's base of support and their extrapolated center of mass (which accounts for both position and velocity). During forward walking, a negative mediolateral MoS will cause the individual to deviate from a straight line; a negative backward MoS will interrupt forward progression.

"Margin of stability helps to explain why balance can be maintained, even if the center of mass is outside the base of support, as long as it's inside the extrapolated base of support," Hall said.

The children with CP, whose ages ranged from 6 to 18 years, all wore matching bilateral thermoplastic custom-molded AFOs with their own footwear. Forty-six of the children wore hinged AFOs, 90 wore posterior leaf spring devices, and 35 wore solid-ankle devices. All but two were community ambulators based on the parent-reported Gillette Functional Assessment Questionnaire.

All of the AFOs were designed with a plantar flexion stop to position the foot for heel-first contact while walking, rather than the toe-first contact that is typical of children with diplegic CP, Hall said. MoS was normalized to leg length.

Mean mediolateral MoS for the group of children with CP was about 7% lower when they wore the AFOs than when they walked barefoot, which Hall said she did not think was a clinically meaningful difference, particularly since the mean mediolateral MoS associated with AFO use still differed considerably from the mean mediolateral MoS for the typically developing children.

Mean backward MoS for the children with CP was about 20% higher when they wore the AFOs than when they walked barefoot. As mentioned earlier, the mean backward MoS for AFO use did not differ significantly from the backward MoS for the typically developing children.

The backward MoS findings suggest AFO use helps to improve balance in children with diplegic CP and may, in turn, help to reduce the risk of falls, Hall said.

In future work, the researchers would like to look at the effects of stride length—which has been associated with MoS in other populations—and optimizing the footwear-shank angle during AFO use for each child, she said.

Hall noted, however, that a single stability measure is unlikely to wholly reflect the complexities of dynamic balance in children with diplegic CP.

"Probably more than one type of balance assessment is necessary," she said. ler

Sources:

Hall M, Schwartz M. Margins of stability in children with cerebral diplegia: barefoot vs AFO comparison. Presented at the 42nd annual meeting of the American Academy of Orthotists and Prosthetists, Orlando, FL, March 2016.

Hof AL, Gazendam MGJ, Sinke WE. The condition for dynamic stability. J Biomech 2005;38(1):1-8.

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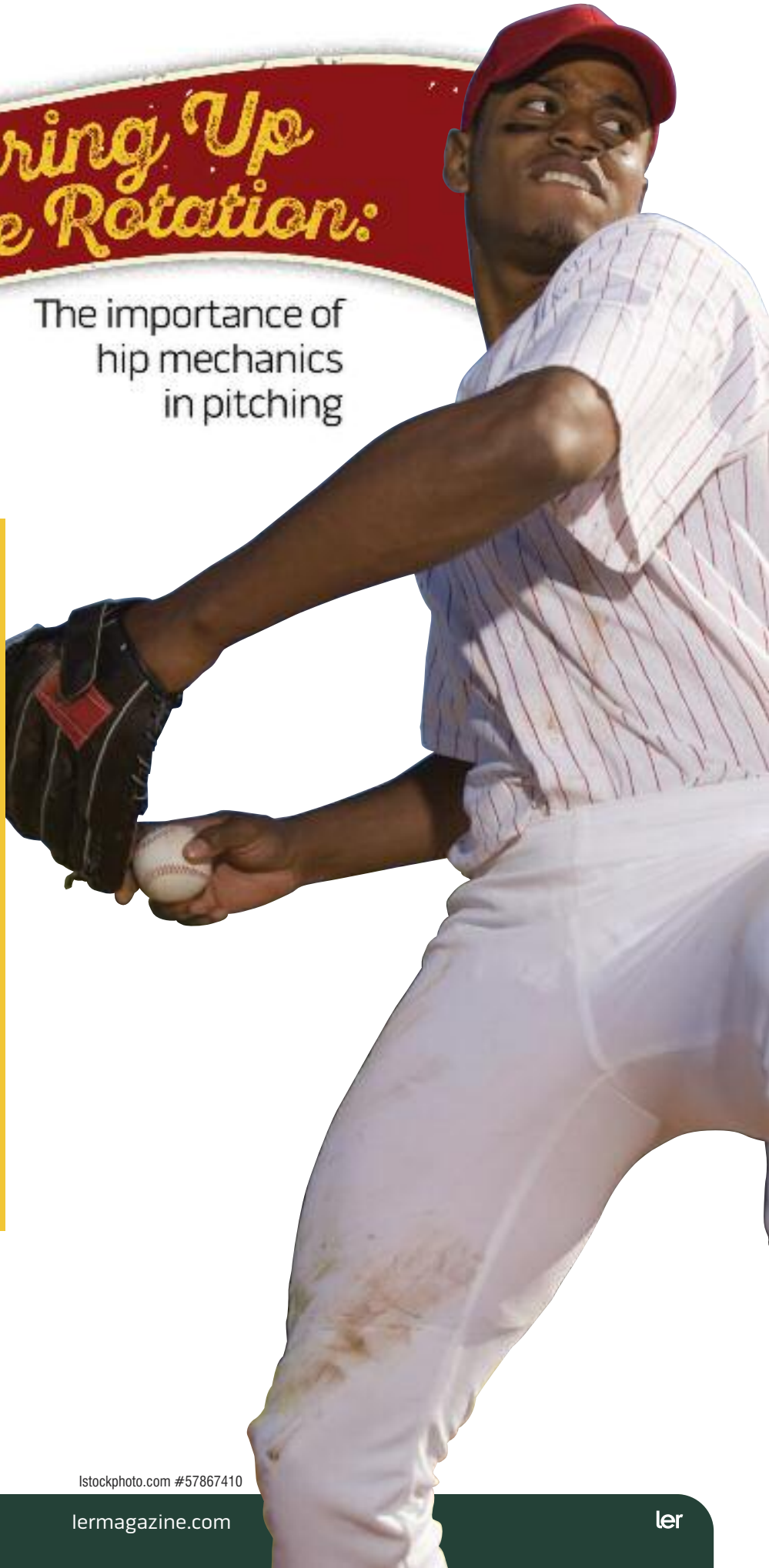
Shoring Up the Rotation:

The importance of hip mechanics in pitching

By Giorgio Zeppieri Jr, MPT, SCS, CSCS

Over the course of a season, collegiate pitchers undergo changes in hip rotational range of motion and hip strength—changes that could increase injury risk or negatively affect performance.



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The baseball pitching motion is a multifaceted sequence of movements that requires the production and transfer of energy from the lower extremities through the trunk and upper extremity to achieve ball velocity.[1-5] Due to the repetitive nature of this motion, pitchers are highly susceptible to injury during the course of a competitive season. Specifically, decreases in hip range of motion (ROM) and strength can alter pitching mechanics so that abnormal stresses act on the trunk, glenohumeral joint, and elbow.[1,3,6-8] These stresses can influence pitching performance and increase injury risk in this patient population.

Mechanics of pitching

The pitching motion and its biomechanical components have been widely defined and are divided into six phases: windup, early cocking/stride, late cocking, arm acceleration, arm deceleration, and follow-through.[9-14] Efficient maximization of this kinetic motion involves the proper utilization of the lower extremities and core in conjunction with the upper extremities to reduce primary reliance on the dominant shoulder to generate ball velocity.[9-14] Operationally, we define the leg ipsilateral to the throwing shoulder as the "trail leg" and the leg contralateral to the throwing shoulder as the "lead leg."

The pitching motion is initiated with the windup (setting phase) and occurs when the pitcher's weight (center of gravity) is transferred toward and over the trail leg as the lead leg is flexed at the knee and hip, initiating pelvic rotation and lumbar flexion.[3,9,14,15]

The early cocking/stride phase occurs as the throwing shoulder begins to maximally externally rotate. The lead knee and hip begin to extend and medially rotate while the trail knee and hip flex to allow for pelvic rotation, trunk stabilization, and lowering the body's center of gravity.[9,14]

During the late cocking phase, the lead foot lands in slight internal rotation (IR) and in line with the trail foot, the throwing shoulder is maximally externally rotated and in line with the nonthrowing shoulder, the trunk is tilted toward the nonthrowing shoulder and slightly hyperextended, and the pelvis is rotated.[9,14,16,17]

After the shoulder achieves maximum external rotation (ER), the pitcher accelerates the throwing arm forward by either driving off the trail leg by extending and externally rotating the trail hip or rotating around the lead leg similar to a tethered ball.[3]

When the ball is released, the lead hip flexes and internally rotates to decelerate the throwing arm and move the body forward, as well as to absorb the distraction forces occurring at the throwing shoulder.[3,9,14,16-18]

Lastly, during the follow-through, the trunk, lead knee, and lead hip continue to flex to disperse the forces created during ball acceleration.[9,14,16,19,20]

Given the important role of the lower extremities in all phases of the pitching motion, a disruption in any of these lower extremity biomechanical contributions, specifically hip ROM and strength, will cause an accumulation of abnormal stress along the throwing shoulder, elbow, and lumbar spine.

Pitching and hip rotation

Restrictions in hip rotational ROM (IR and ER) in both the lead and trail hip have been shown to influence pitching mechanics and injury.[1-3,20] For instance, decreased hip IR and subsequent increased ER in the trail leg during arm cocking and acceleration are associated with increases in lumbar extension, glenohumeral ER, and elbow valgus, which may lead to increased stress along the pars interarticularis, labrum, and ulnar collateral ligament.[1,3,8,15,20,21]

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Continued from page 19

Additionally, inadequate or excess lead hip ROM (restriction or surplus of IR or ER) during arm acceleration through ball release will cause early pelvic rotation, attenuating force production and transfer, as well as decreasing the ability of the lower extremities to absorb forces.[1,3,15,21]

Furthermore, restrictions in lead hip ROM (IR or ER) will change lead-leg foot placement, causing the lead foot to land either "open" or "closed." This will attenuate the force transfer from the lower to upper extremities and cause a decrease in velocity, increased valgus load on the elbow, and increased stress on the glenohumeral joint.[22] The inability of the lower extremities to absorb force will cause the rotator cuff to contract eccentrically to absorb those forces in order to decelerate the arm; this may cause stress at the glenohumeral joint and medial elbow.[3,8,15,20,23-25]

The majority of collegiate pitching-related injuries occur at the shoulder, with rotator cuff tendonitis documented as the most common upper extremity injury.[26-28] Additionally, at this level, upper extremity injuries are responsible for the most time lost from sports participation.[28] Most injuries occur during the season and may result from a breakdown in proper pitching mechanics due to inadequate recovery, increased pitching frequency, or increased pitching workload.[29] Regardless, it is imperative to monitor pitching mechanics during the season. Pitchers are vulnerable to the increased demands of the competitive season, which may fracture the timing pattern of the pitching motion, negatively increasing the stress on upper extremity structures.[29,30]

Prior studies have examined changes in hip ROM and isometric strength as potential injury risk factors, but it is not known if these changes occur over the course of a season and whether they are a result of pitching workload.[4] Despite the evidence describing the importance of hip rotation ROM and strength while pitching, there are few descriptive data detailing the change of hip rotation and strength profiles due to pitching workload over the course of a season in Division 1 baseball pitchers; we conducted a study to address this information gap.[4]

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Our research

The primary goal of our study was to identify changes in hip ER ROM, hip IR ROM, total rotational arc of motion, isometric hip abduction, and hip extension strength in pitchers during a competitive season and to determine the association among changes in hip ROM, strength, and pitching volume (number of pitches during a season).[4]

Fourteen Division I collegiate baseball pitchers consented to participate in our study. Nine were right-hand dominant and five were left-hand dominant. We tested all participants twice: once prior to the beginning of the season before preseason workouts and again at the end of postseason (Super Regional) play, which totaled 66 games over five months.[4] The University of Florida coaching staff in Gainesville documented the individual pitching workload of each participant (calculated by the number of pitches thrown during each game) over the course of the season.[4]

Our study showed pitchers' trail and lead hip ER, hip IR, and total rotational ROM decreased over the course of a competitive season, but only trail and lead hip ER and total rotational ROM were statistically significant.[4] We hypothesize that the repetitive landing with the lead hip internally rotated (causing restrictions in lead hip ER that may alter stride length, lead foot placement, and lead knee flexion angle) in the late cocking phase and posting over the trail hip in IR in the windup phase may result in an overall decrease in bilateral hip ER, which would contribute to the overall decrease in total hip rotational ROM.[4,22] The excessive positioning of the trail hip into IR while driving and initiating the pitching motion may be the cause of adaptations restricting hip ER during the acceleration phase.

In addition to changes in hip rotational ROM, a decrease in hip abduction strength and hip extension strength may lead to faulty sequencing of force from the lower extremities to the upper extremities, which may place an increased load on the upper extremity to produce force when pitching.[1,4,31] This essentially causes the pitcher to become more of an upper-body thrower rather than a whole-body pitcher, leading to an increased risk for upper extremity injuries and diminished performance.[1,4,31,32]

To further illustrate, during the wind-up and arm cocking phase,

Continued on page 22

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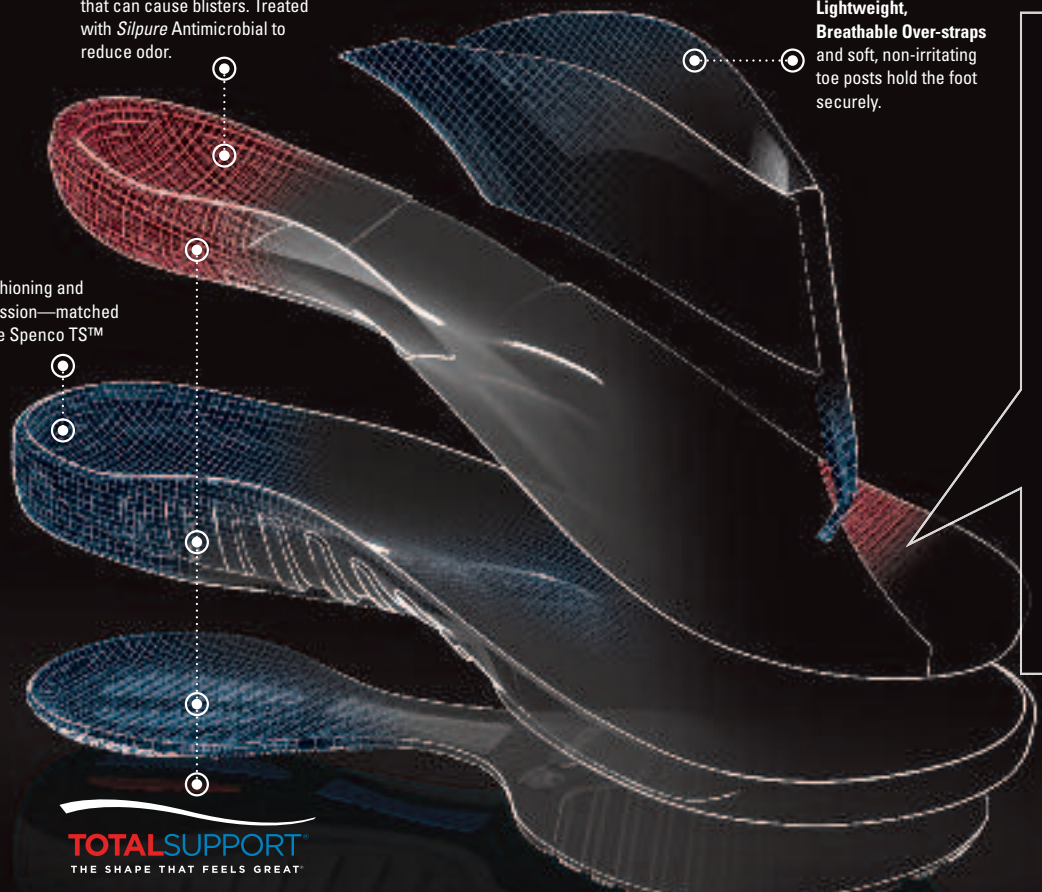

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Continued from page 20

trail hip abductors will fix the pelvis and, along with the hip extensors, initiate the forward movement of the pitcher while he is striding toward the batter.[1,4,7,11,21,25,33,34] An inability to do this will cause the pelvis to drop, impeding the forward movement of the pitcher and reducing the length of the pitching stride.[1,4,11,21,33] This unfavorable consequence will shift the burden of generating force from the lower extremity to the upper extremity, negatively influencing ball velocity and increasing stress on the labrum, posterior capsule, rotator cuff, and medial elbow.[1,4,20,31,32] Additionally, during the late cocking and acceleration phase, lead hip abductors and extensors act to stabilize the lead leg to increase the forward motion and rotation of the trunk and upper extremity.[22,32] An inability to do this will cause a break in the transfer and development of force due to a decrease in trunk rotation, causing a decrease in velocity, as well as an increase strain on the anterior shoulder, rotator cuff, and medial elbow.[22,32]

Our findings demonstrated that both lead and trail hip abduction strength and extension strength decreased during the season; however, those decreases were statistically significant for only lead and trail hip abduction strength.[4] Although these changes may have occurred because of the repetitive nature of pitching and the associated muscular fatigue that occurs over the course of a game or season, we cannot discount other possible contributing factors, such as the decrease in time spent weight training during a season or anthropometric factors.

The changes that occur over the course of a season may be attributed to many causes. We hypothesized that one explanation may be pitching workload (number of pitches thrown over the course of a season). To date, no study had looked at the influence of pitching workload on changes in hip ROM and strength; however, differences between pitchers and position players have been looked at cross-sectionally by Laudner et al.[1] In their study, pitchers were shown to have less hip rotation ROM and strength than their position-player counterparts.[1]

There could be many explanations for these differences; however, one obvious difference between pitchers and position players relates to overall throwing volume. The repetitive motion of pitching—which has been reported to occur between 200 to 1500 times over the course of a season[35] in addition to normal flat-ground long tosses—was hypothesized to predispose pitchers to lower levels of hip ROM and muscular fatigue. Our finding that changes in hip ROM and strength were not significantly associated with pitching workload does not support this hypothesis.[4] However, we considered only in-game pitching volume and not overall pitch count, which includes pitching in practice and before games. Therefore, we may have underestimated total seasonal pitching volume.

Clinical implications

Our findings indicate that, over the course of a season, collegiate pitchers are undergoing changes in hip ROM and strength—specifically decreases in hip ER, hip IR, total rotational ROM, hip abduction strength, and hip extension strength. Although we are not implying a cause-and-effect relationship, these changes may be an indicator of increased injury risk or decreased performance. However, future research is needed to determine if these changes are predictive.

Based on our findings and the emerging literature regarding the importance of lower extremity strength and ROM in pitching performance and injury prevention, it is essential to implement an off-season and in-season training program directed at increasing lower extremity strength and power, while preserving and enhancing lower extremity flexibility. Anecdotally, we find that incoming college players tended to demonstrate poorer strength and flexibility measures compared with their upper-classman counterparts. This, combined with the increased performance demands at the collegiate setting and increased game and practice schedules, increases this population's susceptibility of injury. Additionally, college pitchers with poorer lower extremity strength measures demonstrate lower ball velocity on average. This is not only evident in our population, but in other college pitching populations and settings.[33]

An awareness of lower extremity function during the pitching motion as well as subsequent changes that can occur over a season can aid in developing preventive, rehabilitative, and performance programs to improve athletic performance and screen for potential injury risk in this population.[1,9] Specifically, we recommend routine in-season monitoring of hip ROM and strength by physical therapists, trainers, and strength and conditioning coaches to maintain preseason values.

Giorgio Zeppieri Jr, MPT, SCS, CSCS, is the UF Health Rehab Center–OSMI clinical research chair and a physical therapist specializing in sports and overhead throwing athlete rehabilitation at the University of Florida Orthopaedic and Sports Medicine Institute in Gainesville.

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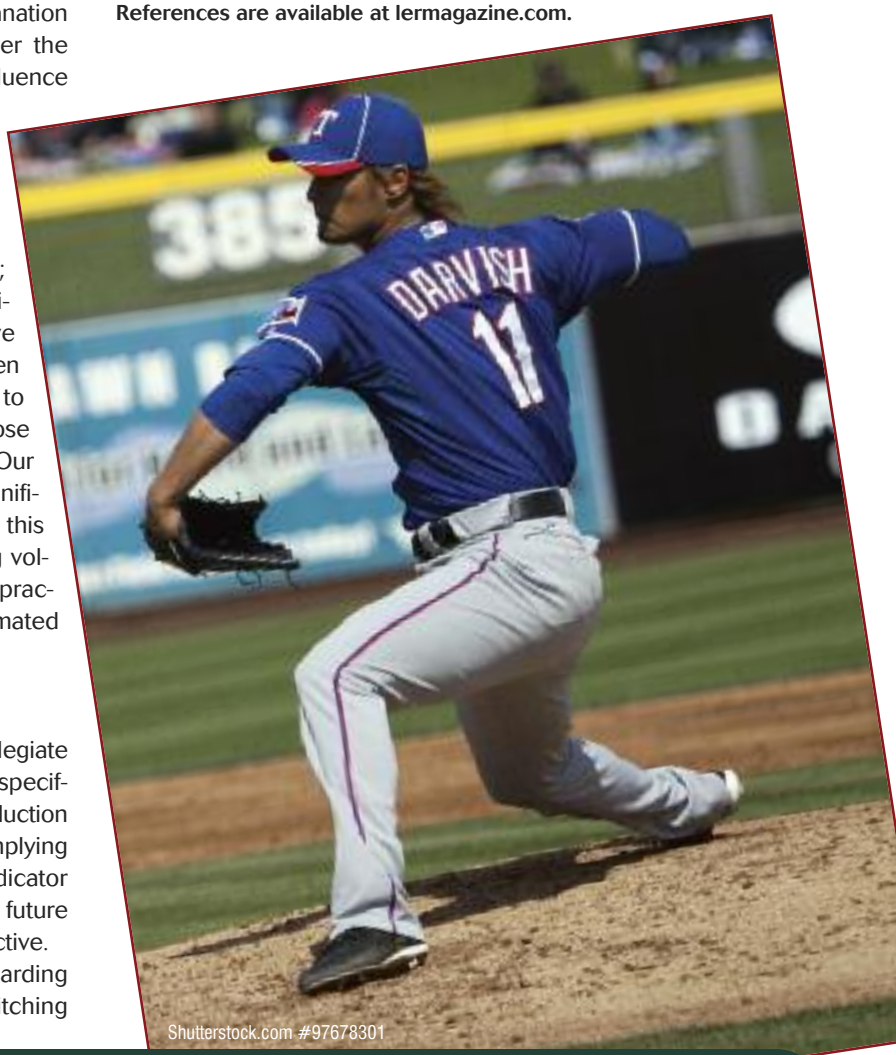

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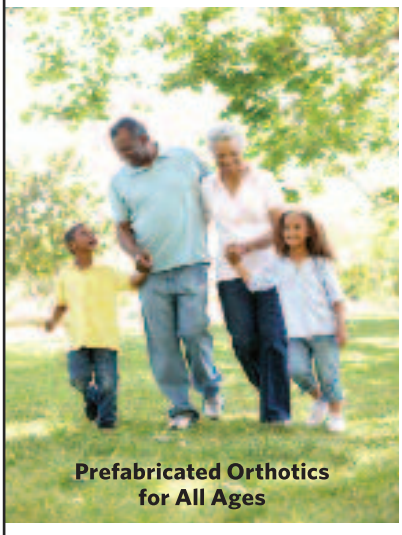
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Managing hallux limitus and rigidus in athletes

By Howard Kashefsky, DPM, FACFAS

The terms "hallux limitus" and "hallux rigidus" refer to a degenerative process of the great toe joint that was first described by Davies-Colley[1] in 1887 and termed hallux flexus. Cotterill later coined the term hallux rigidus.[2] The two terms represent a progression in the spectrum of the disorder, which is characterized by gradual loss of joint range of motion (ROM) in conjunction with joint degeneration, with hallux rigidus typically defined as complete loss of motion. Athletes who experience joint loading that is both elevated and repetitive may be at risk for this problem.

Most of the current literature on hallux limitus and rigidus has focused on surgical management, especially for hallux rigidus; conservative care, however, including orthotic intervention strategies, should always be exhausted prior to surgery. In some cases a trip to the operating room seems unavoidable. Once surgery has been done, however, lower extremity clinicians may be called upon to manage the athlete postoperatively, protect a healing area, and even manage progression.

Risk factors

Hallux rigidus is the most common form of arthritis in the foot, and its incidence is increasing with an aging population.[3] In the general population, hallux limitus and rigidus are more prevalent in women than in men and more likely to be bilateral than unilateral.[4-6] Hallux rigidus is seen commonly in athletes, especially runners, due to repetitive stress at the great toe.[7] Sports that may cause extra stress on the first metatarsophalangeal joint, including those associated with turf toe injuries such as football, basketball, and soccer (especially when played barefoot in sand), and sports that require squatting, such as the catcher's position in baseball, set the stage for injury.[8,9] In general, a soft shoe on a hard surface seems to be a risky combination for athletes where this disorder is concerned.[8,9]

The cause of hallux rigidus is not well understood, despite a plethora of possible causes. The most commonly reported cause is trauma—both acute trauma and repetitive microtrauma.[4,10] Metatarsus primus elevates (elevation of the first ray) was first reported by Lambrinudi,[11] but the question of whether this is a cause or effect of hallux rigidus remains controversial.[11-15] Excessive pronation[16] and internal rotation of the lower limb[17] have also been linked to hallux limitus, but again, causation is difficult to determine.

Hallux biomechanics

The accepted normal ROM of the great toe joint is about 110°, and it is believed that a minimum of 65° of dorsiflexion is needed for gait.[18,19] The measurement is done with the individual supine with the foot in a relaxed position and the hip and knee neutral. Passive measurement is taken with a goniometer.[20] More recently, x-rays have been used to assess ROM, which may be more accurate but not always more practical.

With hallux limitus/rigidus, the proximal phalanx moves into a relative plantar position to the first metatarsal.[21] The sesamoids may become retracted proximally and hypertrophied and have visible osteophytes, joint degeneration, and ankylosis.[22]

Clinical manifestations

Early stages of hallux limitus injury may present as swelling, pain, edema, and limited ROM.[23] Compensatory lateral foot pain and ipsilateral hip pain from external rotator muscle tightness may occur secondary to lower limb external rotation.[7,24]

There are several classification systems for staging.[24-28] The system proposed by Coughlin and Shurnas includes grading based on actual ROM, the relative loss of ROM as a percentage of the ROM on the unaffected side, and clinical symptoms.[25]

In contrast, Hattrup and Johnson have developed a simple three-grade system based on radiographic findings:[24]

Grade 1: Mild subchondral changes with maintained joint space and minimal bony spurring.

Grade 2: Moderate subchondral changes with evidence of joint space narrowing, more bony spurring, and subchondral sclerosis or cyst.

Grade 3: Severe subchondral changes with significant narrowing, spurring, and the presence of loose fragments of cartilage or bone (see Figure 1). This is typically associated with a complete loss of range of motion.

Other classification systems for hallux limitus and rigidus have been described by Roukis et al[26,27] and by Regnauld.[28]

Management options

In this author's experience, surgical management of hallux rigidus in the active athlete typically involves a joint-preserving procedure

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rather than a fusion of the joint. A cheilectomy procedure is often done to remove bone spurs; according to Mulier, athletes in high-level sports such as judo, track and field, soccer, and skating do well with the procedure and are able to return to activity after surgery.[29]

However, hallux limitus can often be successfully treated conservatively. Nonsurgical options outside of shoe gear and orthoses include, rest, nonsteroidal anti-inflammatory drugs, and injections of corticosteroids or sodium hylauronate.[30] In a retrospective analysis of 772 patients with symptomatic hallux limitus, 55% were successfully treated—returning patients to previous activity levels with less discomfort—with conservative care alone. Of those, 84% were treated with foot orthoses, 6% with corticosteroid injections, and 10% with a change in footwear.[31] Joint manipulation and other physical therapy interventions also have been reported to have benefits.[32,33]

Orthotic management

When it comes to managing this problem conservatively, orthotic devices and appropriate shoe gear play a significant role. There is in general a paucity of high-level evidence related to foot orthoses, and hallux limitus/rigidus is no exception; most studies involve small case series.

The goal of an orthotic device for this condition is to provide either an improvement in the postural position of the foot for mild cases or protection of the affected area for severe cases. An ideal orthotic device for the athlete will also be lightweight and low volume.

In addition, there is no clear evidence that an orthotic device for hallux limitus/rigidus needs to be a custom device. In a case series of 32 patients with first metatarsophalangeal joint pain of mechanical origin, modified prefabricated foot orthoses were associated with significantly reduced pain at 24 weeks; however, this study did not include a control group, and further analysis of a subset of patients found no association between change in pain and change in first metatarsophalangeal joint dorsiflexion.[34]

In this author's opinion, an orthotic device for this patient population needs to be full contact to fit the foot appropriately, and at times this may require a custom device.

In mild cases of hallux limitus, an orthotic device may work to enhance the windlass effect, as described by Hicks.[35] This effect involves shortening of the plantar fascia with dorsiflexion of the hallux and elevating the arch of the foot; conversely, it follows that a lowered arch can reduce dorsiflexion of the hallux. Hallux limitus has also been associated with pronation and plantar fasciitis, which further suggests that tension on the plantar fascia under certain conditions may influence hallux limitus.[20,21] Therefore, in theory, a functional foot orthosis designed to support the mid arch in mild cases may improve symptoms.[31,36]

Scherer et al used this approach in fabricating custom orthoses with a 4-mm medial skive for 27 patients with functional hallux limitus, casting the feet with the first ray plantar flexed.[36] They found that the mean maximum first metatarsophalangeal joint dorsiflexion during the stance phase of gait and mean maximum subhallux pressure were significantly improved when patients wore the foot orthoses compared with a no-orthosis condition.

Other options that may be helpful include a first ray cut-out, designed to allow plantar flexion of the first ray and promote further dorsiflexion of the great toe.[9] Further modification to the first ray cut-out design includes a dynamic wedge inclined to 1.5 to 3 mm, elevating the second through fifth metatarsals, and a separate wedge under the distal tip of the great toe, elevating it by 1.5-3 mm, as well.[37] For this modification, shoe fit in the toe box with athletic shoes may become a consideration in some circumstances.[37]

In contrast, in moderate to severe cases of hallux limitus/rigidus, limiting movement of the great toe may be required to reduce symptoms. This limitation of movement could be implemented on a short-term basis to overcome acute inflammation or as a longer-term management plan. The Morton's extension is used to extend material under the great toe to limit movement. It can be used to allow slight movement and protection when made of soft EVA (ethylene vinyl acetate). In severe cases, however, use of a much more rigid fiberglass-plate Morton's extension may be needed (see Figure 2).[9]

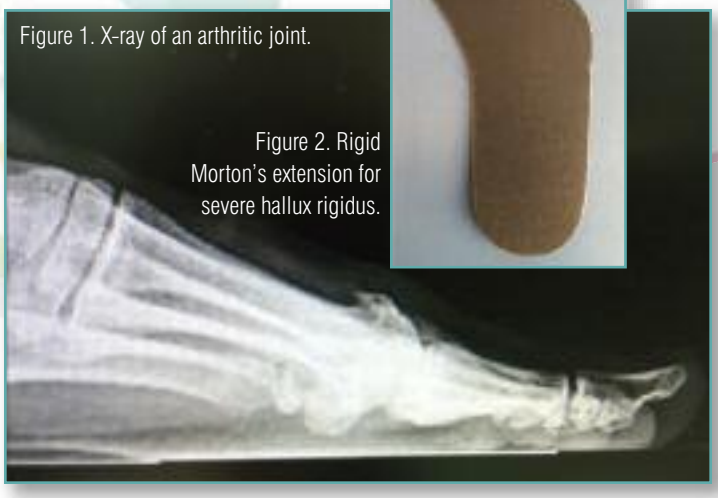

Figure 1. X-ray of an arthritic joint.

Figure 2. Rigid Morton's extension for severe hallux rigidus.

When managing hallux limitus/rigidus, shoes and orthotic devices must work well together. The ideal shoe will have a rocker sole, be stiff enough to protect the great toe joint, and also allow for shock absorption. It should have a removable liner or allow room for an orthotic device. In the athletic population, with sport-specific shoes, this can be a challenge. Clinicians should take the time to familiarize themselves with the shoe designs and brands for each sport. Working with a team of trainers, sports medicine physicians, physical therapists, and the staff of a specialty athletic shoe store is invaluable.

Conclusion

Both hallux limitus and hallux rigidus are common injuries affecting the athletic population. Hallux limitus is a term for staging of hallux rigidus. The process is typically from trauma, either acute or repetitive microinjury. Elevation of the first ray as a contributing factor remains controversial. Depending on the severity of injury, different types of orthotic prescriptions may be appropriate. In cases of mild injury, athletes may benefit from orthotic devices that allow for protected movement. In cases of severe injury, orthoses that block movement of the first metatarsophalangeal joint may be the best option. Foot orthoses are a great tool, and in most cases may be helpful in reducing symptoms. However, more research is needed.

Howard Kashefsky, DPM, FACFAS, is the director of podiatry services at the University of North Carolina Hospitals in Chapel Hill.

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Achilles experts ponder effects of heel elevation

After an Australian study raised questions about the conventional practice of elevating the heel to decrease Achilles tendon load, researchers and clinicians have been revisiting what is known about the many factors—including pain—that can affect tendon load and function.

by Cary Groner

Two years ago, researchers at the Queensland University of Technology (QUT) in Brisbane, Australia, published a study that startled the sports world.[1] Using a custom ultrasound device to measure the propagation of acoustic waves in tendon tissue, the team found that walking in standard running shoes with a typical cushioned heel appeared to increase loads on the Achilles tendon compared with barefoot walking.

The findings produced consternation among other researchers and clinicians, given the long-held belief that modest heel elevation decreases Achilles loading by slightly plantar flexing the ankle and shortening the muscle-tendon unit during gait.[2]

The stakes are fairly high: In athletes, Achilles tendinopathy has a prevalence of 19% and a cumulative lifetime incidence of 24%.[3] Achilles tendon pathology accounts for 8% to 15% of injuries in recreational runners, in fact.[4] But the condition isn't limited to athletic populations; a third of people diagnosed are sedentary.[3] The crucial question is what to do about it, and if heel raises may actually increase tensile loading, there's some important rethinking to be done.

But other researchers and clinicians say wide variability among tendinopathy patients, and the long list of factors other than heel elevation that can affect tendon loading, are also important to consider.

"If you think of the Achilles as a spring, we know that if you use your spring at a shorter length, you may put less load on it," said Jill Cook, PhD, a professor and deputy director of the Australian Centre for Research in Injury in Sport and its Prevention at La Trobe University in Melbourne, Australia. "In theory, a heel raise should take some load off the Achilles, but it may not be effective, depending on a range of other things such as how strong your calf is. The heterogeneity of the population you're investigating is going to complicate your outcomes, and in practice it's really variable."

Since the publication of its initial study, the QUT team has published further research that sheds some light on the situation.[5]

The range of findings reported by studies of heel-elevating interventions—most of which have been conducted in healthy people—underscores the challenges involved.

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Concurrently, other experts have had time to gather their wits, write papers of their own, and try to make sense of it all.

Heel lifts and heterogeneity

An important aspect of the QUT study was that the shoes were associated with changes in several spatiotemporal gait parameters, including lower cadence (five fewer steps/min), greater stride and step length (5% each), and longer step duration (12%), even though both conditions involved walking at identical speeds. Such changes could themselves affect tendon loading, of course, and lead author Scott Wearing, PhD, acknowledged this. Wearing, who is a professor of clinical sciences in the Institute of Health and Biomedical Innovation at QUT, told *LER* after that study's publication that it was unclear which aspects of the shoes contributed to the increased loading effects—the heel raise itself or some other variable.[6]

The study participants, moreover, were healthy—without tendon pain or pathology—and, as noted, the test involved walking rather than running. Research has shown that gait in runners with chronic tendon complaints often differs from healthy controls,[7] however, and it's such patients that primarily concern clinicians.

Complicating matters further, the concept of load has different clinical implications depending on whether one's objective involves alleviating painful symptoms or treating a patient's underlying issues.

"I might tell a patient, ok you can put in a heel lift, but that's only for symptomatic relief," said Karin Silbernagel, PT, PhD, ATC, an assistant professor in the Department of Physical Therapy at the University of Delaware in Newark. "But to treat the problem, we want to load the tendon."

Cook agreed, noting that tendon loading can't necessarily be quantified in a clinically relevant way.

"Tendons are very sensitive to how you load them, and it's difficult to evaluate pathology or structural change and make inferences about pain; they don't necessarily correlate," she continued. "We're getting better at measuring load, but it's hard to do, and I'm not sure that absolute load is a factor. If you run two hundred kilometers a week, your tendon is going to be much more resilient than that of a couch potato, so you can't say, 'This much load makes a difference.' What you can say is that the tendon has to adapt to a certain percentage above the [usual] load. That's where the heterogeneity in populations becomes tricky. As soon as you get people with pathology and pain—different ages, genders, loading environments, biomechanics, strengths, kinetic chain function—it just becomes a minefield to get anything out of it."

The range of findings reported by studies of heel-elevating interventions—most of which have been conducted in healthy individuals—underscores the challenges involved. As far back as 1995, Canadian researchers found that heel lifts didn't decrease Achilles tendon loading at a statistically significant level across their study population—but in two of the five runners studied, it did.[8] Then in 2002, British researchers reported in the *Journal of Applied Biomechanics* that heel lifts were associated with both increases and decreases in peak Achilles tendon force, depending on the runner being tested.[9] They also noted that treatment success might be due to heel raises causing a later tendon force peak, which reduced the average rate of loading. A 2005 paper with the same lead author reported similar findings in soccer players: A 10-mm heel lift had no significant effect on peak Achilles tendon force, peak plantar flexion moment, or corresponding

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Continued from page 29

loading rates across the group; but loading did, in fact, significantly increase with the heel lift in some players and decline in others.[10] Another 2015 paper found that walking with heel wedges didn't reduce Achilles tendon load, but did redistribute it from the medial to the lateral triceps surae during inclined walking.[11]

"There are very individual responses to shoes and heel lifts, and some people respond totally the opposite of what you expect," Silbernagel said.

Reconnaissance and strategy

Cook and Silbernagel point out that the Achilles tendon isn't a monolith, and the part of the tendon where pain or pathology occurs affects diagnosis and treatment strategies.

"Sliding and rotation give the tendon its springlike behavior," Cook said. "But there's the insertion point, there's the midtendon, and there's the peritendon, which is more about friction loading. So there are actually three pathologies producing different signs and symptoms, caused by different loads, that require different interventions."

Silbernagel described how such complexities affect her therapeutic decisions.

"If there's insertional injury, I won't have patients exercise barefoot," she said. "In those cases, I want to start them in various degrees of plantar flexion to minimize the pain. You get compression on the tendon, over the bursa, and onto the bone, when you go into more dorsiflexion. If you put in a heel lift to unload that, you get a great effect. Someone with midportion tendinopathy, but no insertional pain, can often start exercising barefoot, though never on the stairs."

Cook agreed that effective therapy typically entails increasing the loading capacity of the tendon.

"If change in load is a critical factor that induces pain, then you have to adapt the musculotendinous unit to tolerate higher levels of load," she said. "Loading is always critical—tensile loading; heavy, slow resistance training; and then training the tendon to do the energy storage loading that's by far the best thing for your mid-Achilles and insertional Achilles. The difference between those is that with insertional Achilles, you must keep people in a heel lift until symptoms start to resolve. For the peritendon, it's about reducing movement, so heel lifts can help there for a different reason, because you stop the friction between the tendon and the underlying tissue. The treatment can be similar but for completely different reasons."

Cook stressed that heterogeneity in the patient population profoundly affects such decisions, as well.

"My seventy-year-old golfing lady is going to be completely different from my twenty-five-year-old sprinter, and I'll recommend different treatments because of who they are, what factors are contributing to their pain, and what they want to achieve," Cook said. "It's about modifying training so that their load stays under the tendon's capacity, but at the same time trying to increase that capacity. That means modifying how many times a week you train, what sort of drills you do, and addressing pain, strength, and endurance levels with an exercise program."

At Silbernagel's University of Delaware clinic, clinicians use a return-to-sport model premised on individual needs. One issue has to do with how patients experience and respond to pain.

"There are habitual overloaders, who should fear pain more than they do," she said. "Then there are underloaders who are more

Continued on page 32

Continued from page 31

fearful of the pain. If you can move people progressively over time and follow their training diaries, it solves a lot of these sensitivity issues."

Silbernagel and her colleagues pay attention to foot-strike patterns, too, since they affect tendon loading.

"If you're a heel striker I can allow you to run longer than if you're a forefoot striker, which puts more load on the tendon," she said.

Indeed, a 2013 paper reported that female runners with a forefoot or midfoot strike experienced an 11% greater Achilles tendon impulse with each step—which added up to a whopping 48 additional body weights for each mile run—compared with rearfoot-strike runners.[12]

It's complicated

Recent research has more deeply explored the levels of complexity that affect the web of biomechanics related to Achilles function—and dysfunction.

For example, in a 2015 paper, researchers at the University of Southern California in Los Angeles found that tendinotic Achilles tendons are more compliant than healthy ones, and that this affects central nervous system control, which then alters muscle activation patterns in the lower leg.[13]

British investigators, too, have begun to describe the complex relationship between the Achilles tendon and surrounding musculature. In a study presented in 2014 at the International Scientific Tendinopathy Symposium in Oxford, UK,[14] researchers from the University of Leicester reported that runners with Achilles tendinopathy had weaker plantar flexors than healthy runners, and that the soleus muscles, rather than the gastrocnemius, appeared chiefly responsible for the deficits. They speculated that the problem could be due to central motor inhibition or preexisting weakness.

Lead author Seth O'Neill, MSc, a physiotherapy lecturer and PhD candidate at the university, told *LER* that he and his colleagues suspected the role of the soleus in Achilles tendinopathy partly because the condition so often affects endurance athletes.

"In any event longer than about fifteen hundred meters, the soleus is the most important force producer,[15] at about eight times body weight," he said.

He and his team designed their experiment to test isokinetic strength using a dynamometer, with the knee both extended and in 80° of flexion.

"In the extended position, both the gastrocs and soleus worked, but in the flexed position only the soleus can produce any force. The deficit in strength was very similar in both test positions, which suggests it's the same muscle in each case—and that has to be the soleus," O'Neill said. "Our hypothesis is that if the muscle is weak, and not producing force appropriately, it cannot absorb shock and protect your Achilles. That may be why the tendon starts to break down. We think treatment interventions need to target the muscle so your tendon can repair itself and recover."

O'Neill believes, moreover, that therapeutic benefit may not require massive interventions.

"Altering the muscle's motor skill, or the person's control of the muscle, improves how it absorbs shock," he said. "We're finding that patients with Achilles problems tend to be uncoordinated—the tendon jitters as it eccentrically loads—and we think that's a key component. You can do what we do, which is strength training and load monitoring on the model of Karin Silbernagel's work. You could also change the runner's gait with footwear, orthotics, or reeducation.

It's not just about pain; it's about tendon structure, and whether that changes. The studies have shown how variable people's responses are, so the key is to individualize therapy."

Orthoses and shoes

Researchers have studied foot orthoses in this context; perhaps not surprisingly, results have been varied.

For example, a 2007 study reported that just four weeks of either physiotherapy or wearing custom foot orthoses reduced pain by half in 341 male runners with Achilles tendinopathy.[16]

More recently, a 2014 study reported that orthoses were associated with significant reductions in Achilles tendon load in healthy runners—though the researchers used polyurethane insoles rather than prescription devices, and hypothesized that the load reduction was related to the midsole cushioning effect.[4]

Jill Cook and her colleagues studied custom versus sham orthoses in 140 individuals with midportion Achilles tendinopathy, all of whom also did eccentric calf muscle exercises.[3] The authors found that, after a year, the custom devices had been no more effective than the sham ones for reducing symptoms and improving function; nevertheless, all participants reported significant improvement in symptoms.

It's easier on clinicians when everything works rather than when nothing works, of course, but it does make it harder for researchers to parse results. Other studies have shown benefits from certain types of eccentric calf-muscle training, in fact, suggesting that it's a valuable aspect of therapy with or without orthoses.[17]

Not surprisingly, podiatrists and orthotists have their own take on the situation. Bruce Williams, DPM, director of gait analysis studies at the Weil Foot and Ankle Institute in Chicago, said that if he can't stretch tendinopathy patients to an increased range of ankle joint motion, he adapts by raising the heel.

"I think Achilles issues are usually more related to tension loading than impact, though it can be both if the patients have issues with ROM and hit the end of that range," Williams said.

He added that he rarely uses heel lifts alone because they don't allow enough flexibility to treat the range of individual pathologies he sees.

"I use lifts in combination with a prescription for an orthotic device," he explained. "I'll varus or valgus post, or use a wedge under the hallux to engage the third rocker. All of these are meant to treat the whole, because heel lifts just don't work all the time."

Range of motion, foot type, and other variables that Williams has found to be important clinically are often not reported in Achilles studies, he said.

"Was it a high-arched foot in the subject?" he asked. "Was it low-arched? What was the resting calcaneal stance position? How was their ankle range of motion—do they have dorsiflexion issues, and to what degree?"

All such factors, he said, could affect results and partly explain the wide variation in responses.

Harvey Johnson, CO, the owner of Eno River Orthotics in Hillsborough, NC, agreed that the "to-lift-or-not-to-lift" discussion oversimplifies the problem.

"Heel lifts do reduce tensile load on the Achilles," he said. "But when you build stability into the calcaneus with a custom orthotic, the patient isn't sliding around as much as on a flat heel raise."

Shoe design matters too, Johnson said.

"Tibial progression is critical, and if the shoe lacks an anterior

Continued on page 34

Continued from page 33

rocker, it restricts that progression, which increases the need to dorsiflex at the ankle. That loads the Achilles as the body mass moves over the knee."

Johnson echoed Williams' concerns about foot type, as well.

"If studies don't take that into account, their results are skewed," he said. "A twelve-millimeter raise in a high-arched foot doesn't affect Achilles/ankle dorsiflexion nearly as much as twelve millimeters in a low-arched or flat foot."

The view from Oz

Seth O'Neill criticized the Wearing paper for failing to account for the variables its two testing conditions.

"When they compared barefoot to shod, there were differences in cadence; they're taking more steps [when barefoot], each stride is shorter, so effectively you're expecting less Achilles load. It's not a matter of heel rise, it's a matter of absorbing shock differently."

Interestingly, a 2016 follow-up paper by Wearing and his colleagues compared shod walking with or without an added 12-mm in-shoe heel lift, and found the added lift reduced tensile load in the Achilles.[5]

Wearing spoke softly from his office in Brisbane and seemed relatively calm about all the controversy.

"In the first study, what we were actually saying is that shoes modify your gait pattern, and that this change results in greater Achilles tendon loading, despite the shoes already having a heel raise," he said. "When people put on the shoes, they had greater step lengths, lower cadences, and higher ground reaction forces. I think all of those things, either individually or combined, could lead to higher Achilles tendon loading."

His team wasn't oblivious to expert blowback, as it turns out.

"We had a lot of discussions with clinicians who pointed out that heel lifts should lower forces, so then we took the same shoes and added a twelve-millimeter heel lift," he continued. "In that study [the 2016 paper noted above], there were no changes in gait parameters, so we concluded that heel raises themselves do slightly reduce Achilles loading."

Wearing said that all the participants in the 2014 study were rearfoot strikers whether barefoot or shod, so he didn't think foot strike pattern was a major variable in that case. However, his team is currently conducting a study to compare Achilles tendon loading in rearfoot-strike runners and forefoot-strike runners during barefoot running.

"It's a little premature to discuss what we're finding, but from early data, I will say that it looks as if foot-strike patterns do influence Achilles tendon loading," he said.

As for the future, his team is interested in further analyzing which parts of a given shoe contribute to gait changes and the associated increase in Achilles loading.

"We know that heel elevation tends to lower those values, but not to the levels encountered during barefoot walking," he said. "One of the great unknowns for us is how important increasing or decreasing load is in the tendon, because loading is important in homeostasis and maintaining tendon properties, but is also linked to injury. That's the future research: What is a detrimental load?" ler

Cary Groner is a freelance writer based in the San Francisco Bay Area.

References are available at lermagazine.com.

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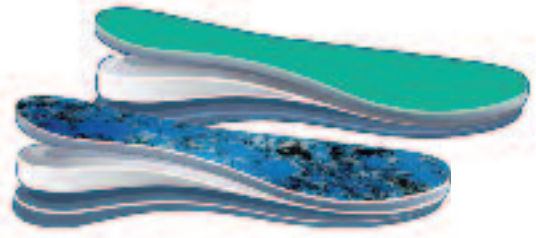

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Influence of estrogen on the plantar fascia

Reported effects of estrogen on knee ligaments and the Achilles tendon raise questions about how the plantar fascia might be altered during the menstrual cycle and how those changes might affect injury risk. This original study was designed to address that question.

By Jerrold Petrofsky, PhD, JD; and Haneul Lee, PT, DSc

The plantar fascia serves as an elastic cushion for weight applied to the foot and also helps increase stability in the ankle.[1,2] It is a thick fibrous connective tissue that originates at the medial tuberosity of the calcaneus and inserts into the proximal phalanges.[2] The central portion is the thickest; it attaches at the posterior aspect of the medial tuberosity of the calcaneus posterior to the origin of the flexor digitorum brevis tendon, and is 1.5 to 2 cm in width, distally, at the level of the metatarsophalangeal joints. The central portion of the plantar aponeurosis divides into five fascicles, one for each of the toes.[1,3] The lateral portion of the plantar aponeurosis is 1 to 1.5 cm in width; it arises from the lateral aspect of the medial tuberosity of the calcaneus, and its distal medial and lateral bands attach to the plantar plate of the fourth toe and to the base of the fifth metatarsal, respectively.

Although the plantar fascia does not differ anatomically between men and women, comparing lower extremity function for both sexes produces a number of interesting statistics. Men experience more plantar loading than women, especially at the midfoot, during a cutting task.[4] Women have significantly higher rates of anterior cruciate ligament (ACL) injuries and anterior knee pain than men.[5-7] Previous studies have shown that ACL injuries are associated with changes in anterior and posterior cruciate ligament laxity due to changes in body temperature during the menstrual cycle and the effect of beta estrogen receptors on these two ligaments.[8-11]

Increased tissue temperature increases the laxity of ligaments in the body.[12,13] Such increases in core and shell temperature are associated with the latter half of the menstrual cycle in women not taking oral contraceptives.[14-16] The increase in estrogen just before ovulation at midcycle also increases laxity of the anterior and posterior cruciate ligaments.[9] This increase in laxity makes the knee unstable, which can lead to compensatory increased activity of the medial and lateral quadriceps muscles.[8] In spite of compensation, knee injuries in runners peak at ovulation.[10] Ankle injuries are also more common in women than men, and presumably involve a similar mechanism.[17]

Although a higher rate of plantar fasciitis in women than men has been reported in the military, little has been done to examine the effect of estrogen on the plantar fascia.

Continued from page 37

Although more running-related injuries are seen in men than in women,[18] and a significantly higher rate of plantar fasciitis in women than men has been reported in a military population,[19] little has been done to examine the effect of estrogen on the plantar fascia. Greater postural sway in the early follicular phase of the menstrual cycle has been reported.[20] This could be due to a complex interaction between knee and ankle laxity, as cited above. In another study, Ericksen and Gribble found more inversion-eversion laxity in women than in men, but no effects of the menstrual cycle.[21] However, they examined their study participants five days before and five days after ovulation; estradiol peaks at ovulation and then falls. Other research has found that center of pressure path length and velocity were significantly higher at ovulation than at menstruation, suggesting altered neuromuscular control strategies; this was not seen in birth control users.[22] In a similar manner, for the Achilles tendon, strain of the tendon was greater in women with a normal menstrual cycle and abolished when women used birth control pill.[23]

Our research

These investigations into the effect of estrogen on the knee ligaments and the Achilles tendon raise questions about how the ligaments in the foot, such as the plantar fascia, might be altered during the menstrual cycle. It stands to reason that, if these same estrogen receptors are found in the plantar fascia, the ligament will be most flexible at ovulation. This, combined with the known effects of estrogen on more proximal aspects of the kinetic chain, should have the added effect of impairing postural control at ovulation.

To test this hypothesis, we examined plantar laxity, elasticity, and thickness in women compared with men over the same time period. The men were controls to account for measurement variation due to environmental factors.

Methods

The participants in this study were 15 healthy women with a regular menstrual cycle and 15 men, all aged between of 18 and 30 years. Participants did not have any orthopedic abnormalities or injuries to the knee or foot and were screened to confirm normal arches and exclude those with flat feet. Basic characteristics are described in Table 1; the only statistically significant difference between genders was for body weight ($p < .01$). All participants signed a statement of informed consent as approved by the institutional review board of Loma Linda University in California.

All participants were advised of the study goals, protocol, and inclusion and exclusion criteria. We made two different estimates of plantar fascia elasticity based on the assumption that plantar fascia elasticity would affect foot length and fascia thickness. First, the foot was loaded at two different weights (half body weight and full body weight) and foot length from the longest toe to the heel was measured on a powdered polypropylene sheet. We also measured plantar fascia thickness with no load and full body weight to assess the elasticity of the plantar fascia. We measured all participants at two time points, 14 days apart, to correspond with the early luteal phase and ovulation in the female participants.

We measured foot length with the participant standing on a talcum-powdered polypropylene sheet. This allowed the foot to have a natural shape while we measured its length, since the friction is low. We measured the length of the foot with a digital caliper that could be extended up to 300 mm under two conditions, unilateral stance and bilateral stance.

Musculoskeletal ultrasound is a useful imaging tool for confirming a diagnosis of plantar fasciitis and for measuring plantar fascia thickness before and after a treatment regimen to gauge the treatment's efficacy. The standard "normal," or asymptomatic, thickness value reported for the plantar fascia is 2.3 to 4 mm, averaging 3.4 mm.[24] Acoustic coupling gel was applied to the plantar surface of the foot, and each foot was evaluated sonographically with a L14-6 MHz linear array transducer. The plantar fascia was examined with the patient in two positions: prone, with the foot hanging over the edge of the examination table and the ankle in a neutral position; and while standing on a platform. The ultrasound probe was applied vertically to the plantar aspect of the heel. The sagittal thickness of the proximal insertion of the plantar fascia was measured at a standard reference point 5 mm from the proximal insertion at the anterior aspect of the inferior border of the calcaneus.

We analyzed data using SPSS for Windows version 22.0. Characteristics of the participants were summarized using mean and standard deviation. T-tests and ANOVA (analysis of variance) were used to compare means. The level of significance was set at less than .05.

Table 1. Patient demographics

Women	Age(years)	Height(cm)	Weight(kg)	BMI
mean	25.7	1.6	59	22
SD	2.1	.1	7.5	2.7
Men				
mean	27.3	1.7	73.4	24.4
SD	2	0	5.5	1.7

Results

The results of the experiments are shown graphically in figures 1 and 2. As shown in Figure 1, foot length was significantly greater in men than in women ($p < .01$). The change in the length of the foot with weight while standing was also different in men and women. For the women, the change in foot length with weightbearing varied during the menstrual cycle; in the early luteal phase, the average change in length from partial to full weightbearing was 3.5 ± 1.4 mm, whereas at ovulation it was 5 ± 2.5 mm. This difference was significant ($p < .01$).

When indexing the foot length change from partial to full weightbearing, an elasticity index can be derived: $.12 \pm .04$ mm/kg body weight for the early luteal phase and $.17 \pm .08$ mm/kg body weight for ovulation. This difference was about 50% at ovulation and under the influence of estrogen. For the men this elasticity index averaged $.07 \pm .003$ mm/kg body weight and was the same at the two time points.

Plantar fascia thickness helps explain some of this. As shown in Figure 2, plantar fascia thickness was greater in men than women ($p < .01$), and women showed a significant reduction in thickness with weightbearing during the early luteal phase.

Discussion

It has been well established that there are estrogen receptors on the anterior and posterior cruciate ligaments, the Achilles tendon, skeletal muscle, and the nervous system.[10,13,23] There are both alpha

Continued on page 40

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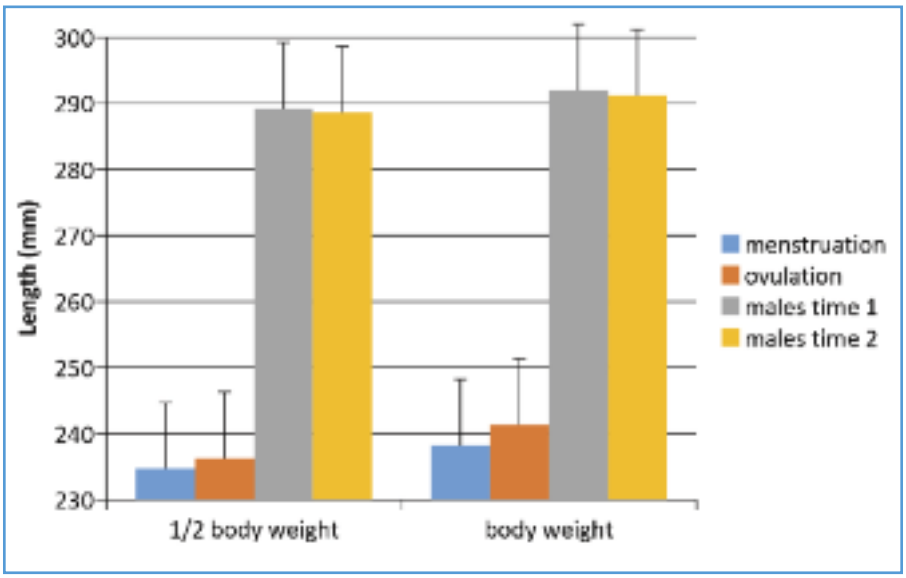
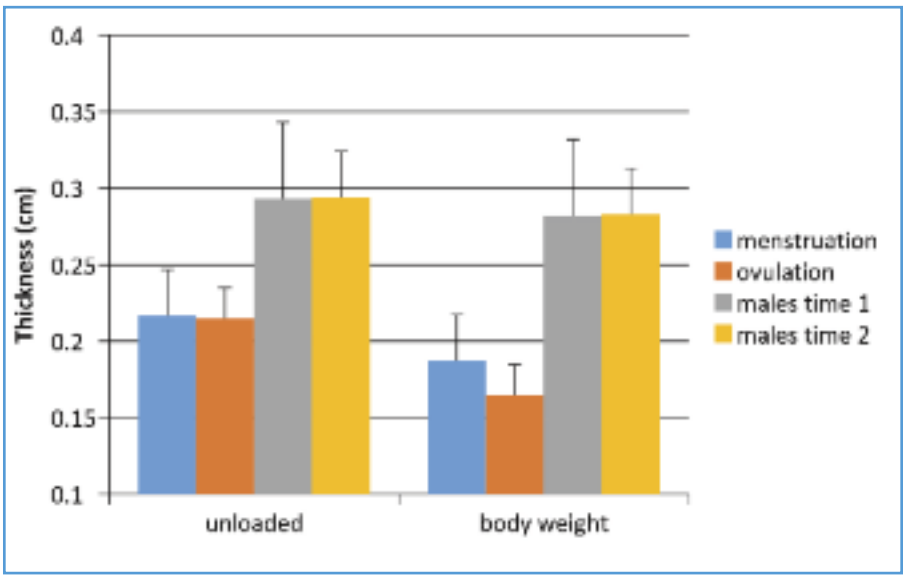
Figure 1. Change in foot length for each gender over time.



Average foot length in 15 female and 15 male participants taken when standing on both legs (half body weight) or full body weight (standing on one leg). Foot length was measured in women at menstruation and ovulation and in men 14 days apart (males time 1 and 2).

Figure 2. Plantar fascia thickness for each gender over time.



The average thickness of the plantar fascia in 15 female and 15 male participants when standing on both feet (full body weight) or lying down (unloaded) at two time points 14 days apart.

and beta receptors in tendons, ligaments, and skeletal muscle altering neuromuscular control and myofascial force transmission pathways during the menstrual cycle.[25-27] There are 17-β estradiol receptors in human connective tissues[28,29] that cause relaxation of connective tissues such as the ACL at the time of ovulation, when estradiol concentration peaks.[10,13] The effect of estrogen on these receptors affects the female neuromuscular system both directly, by altering motor control, and indirectly, by altering elasticity at ovulation.[30]

In our investigation, we found that, as others have shown in the Achilles tendon and knee ligaments, there was a significant increase in elasticity of the plantar fascia at ovulation. Men demonstrated thicker plantar ligaments than women, with no change in elasticity over a two-week time period. These data on the men clearly show the change in elasticity seen in the women is not an artifact of the measurements.

Since central body (core) temperature also increases at ovulation[31-33] some of the elasticity effect may be due to an increase in tissue temperature. However, we are not aware of any reports of deep tissue temperature measurements in the foot during the menstrual cycle, and this will require further study. Further, if environmental temperature changes due to the season, this may impact the measurements. Body temperature changes throughout the day, presenting another confounding variable. Both of these variables would lead to variation in the measurements. Another limitation of the study is the small number of participants. It would be good in the future to examine three populations of women: one with a normal menstrual cycle, one on the birth control pill (in whom, presumably, the variation in elasticity would not be seen), and finally a group of postmenopausal women.

It seems likely that laxity fluctuations in women affect motor control and injury risk in the foot. We have previously reported that static balance is reduced at ovulation in women,[34] which may be due to the increase in laxity since the plantar fascia plays a role in balance during gait.[35] Chronic ankle instability, which has been reported to be more common in female athletes than male athletes at both the high school and collegiate levels,[17] is also associated with postural control deficits. Ericksen and Gribble found better postural control in men than women, but found no association between postural control and the menstrual cycle.[21] However, no studies have yet looked at this possible association in patients with chronic ankle instability.

Conclusion

In female athletes, foot and ankle injuries occur much more frequently than ACL injuries,[36] but the medical literature on hormonal changes with the menstrual cycle and injury risk factors in women has focused primarily on the knee. Because ligament laxity in women can be managed clinically using oral contraceptive pills, this potential risk factor for foot and ankle injuries deserves clinical consideration and further research.

Jerrold Petrofsky, PhD, JD, is a professor of physical therapy at Touro University in Henderson, NV, and is on the pain management advisory board for Pfizer Pharmaceuticals. Haneul Lee, PT, DSc, is assistant professor and director of research in the Department of Physical Therapy at Gachon University in Incheon, Korea.

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The clinical implications of accelerated knee OA

Accelerated knee osteoarthritis (OA), defined by very rapid radiographic disease progression, is also associated with earlier and more severe symptoms than traditional OA. Identification of these symptoms can facilitate early intervention in this high-risk patient population.

By Jeffrey B. Driban, PhD, ATC, CSCS

Osteoarthritis (OA) is organ failure of a diarthrodial joint, defined as a disease by structural changes (eg, cartilage loss, osteophyte formation, synovitis) and defined as an illness by patient-reported symptoms.[1] More than 27 million Americans have OA,[2] and this number is rising. Globally, it's one of the top 15 causes of disability.[3,4] Unfortunately, we lack structure-modifying therapies that slow, prevent, or reverse OA progression, and our symptom-modifying therapies are far from optimal.

One challenge of treating individuals with OA is that there are many paths to joint failure, and intervention strategies may need to be tailored to account for specific paths. For example, someone who develops OA after a meniscal injury and someone who develops OA secondary to repetitive overloading from obesity compounded by malalignment may need different intervention strategies to delay or prevent joint failure.

Patients with OA progress at varying rates. For example, individuals with a history of knee injury tend to be diagnosed with knee OA 10 years earlier than those without a history of injury.[5] Furthermore, one in four patients who experience an anterior cruciate ligament injury has radiographic OA within five years,[6] and two in three patients who suffer a lateral tibial plateau fracture have radiographic OA within nine years.[7] More recently, clinical trials[8-10] and prospective cohort studies[11-15] have helped investigators identify a subset of individuals with accelerated knee OA, in whom the disease progresses even more quickly than post-traumatic knee OA.

Quantifying accelerated knee OA

We recently started studying accelerated knee OA within the OA Initiative, a cohort study primarily of individuals with or at risk for knee OA. The OA Initiative is an ongoing multicenter longitudinal study at four clinical centers in the US: Memorial Hospital of Rhode Island in Pawtucket, The Ohio State University in Columbus, University of Maryland in College Park, Johns Hopkins University in Baltimore, and the University of Pittsburgh in Pennsylvania. The study

> Clinicians should be concerned about patients without radiographic OA who report knee pain, because it may be an early symptom of accelerated knee OA.

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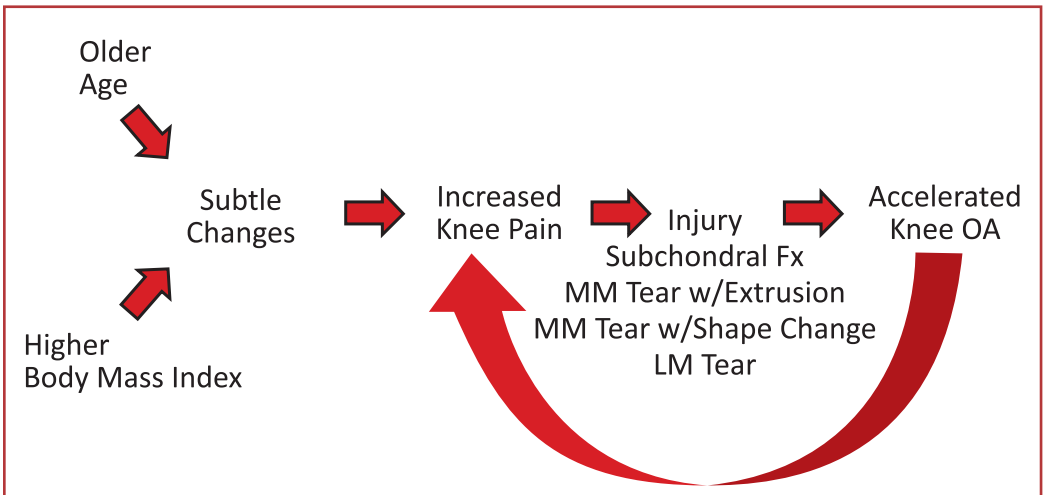

Figure 1. A conceptual model for the origin of accelerated knee OA.

staff recruited three groups of participants between 2004 and 2006: 1390 individuals with symptomatic knee OA; 3284 individuals without symptomatic knee OA but with risk factors for knee OA; and 122 individuals with no knee OA and no risk factors for OA. Overall, the OA Initiative includes 4796 individuals (58% women) aged between 45 and 79 years (average body mass index = 28.6 ± 4.8 kg/m^2).

Although the study population is not representative of the US population, it is a valuable cohort because most participants completed annual evaluations. The annual evaluations typically included an array of patient-reported outcomes, physical performance tests, imaging (radiographs and magnetic resonance images [MRI]), and biospecimen collection. All of the data and manuals from the OA Initiative are publicly available for free.[16]

Within the OA Initiative, 1566 individuals had no radiographic knee OA at baseline and sufficient follow-up data to assess their rate of OA development. We found that 3.4% developed accelerated knee OA, which is defined as having no baseline radiographic knee OA in either knee and developing advanced-stage disease (definite osteophyte and joint space narrowing) within four years.[13] Furthermore, 63% experienced this dramatic progression within 12 months.[17] Adults who develop accelerated knee OA are typically older, overweight, and more likely to have a history of a recent knee injury than those with a slower onset of knee OA or no knee OA.[13,15] Within this subset, our preliminary findings indicated that accelerated knee OA may be related to a history of injuries characterized by subchondral damage, medial meniscal pathology with extrusion (displacement), and lateral meniscal tears; these findings need to be confirmed in larger studies that are underway.[14]

Assessing symptoms

Although individuals with accelerated knee OA may be characterized by unique risk factors, it's unclear if their symptoms differ from other adults with a more gradual onset of OA. If adults with accelerated knee OA experience more symptoms and impaired function than those with a gradual onset of knee OA, particularly if symptoms occur earlier in the former, that would underscore the clinical relevance of this subset of people with knee OA and highlight the need to recognize those at risk and intervene if possible to address those symptoms.

We conducted longitudinal analyses among participants in the OA Initiative who had no radiographic knee OA at baseline to determine if accelerated knee OA was associated with greater pain and other clinical outcomes and if outcomes varied over time between those with accelerated knee OA and those with a more gradual onset. We considered participants to have accelerated knee OA if one or more knees developed a definite osteophyte and joint space narrowing within 48 months of baseline. We defined a gradual onset of OA as someone who had one or more knees increase in radiographic scoring within 48 months; this would include someone who developed a possible or definite osteophyte. We defined the index visit as the study visit when a participant met the criteria for accelerated or gradual onset of knee OA. Our study period included up to three years before and after the index visit.

Our primary outcome variable was self-reported knee pain, based on the WOMAC (Western Ontario and McMaster Universities Arthritis Index) pain score.[18] We explored 11 other secondary outcome measures: knee-specific disability (WOMAC function score), global impact of arthritis (single question with a 0 [very good] to 10 [very poor] response), walking pace, chair-stand pace, maximum isometric knee extension force, maximum isometric knee flexion force, physical activity level (Physical Activity Score for the Elderly), physical health (Short-Form 12 Physical Component Score), mental health (Short-Form 12 Mental Component Score), depression (Center for Epidemiologic Studies-Depression Scale), and use of prescription medicines. To determine the number of prescription medications a participant used in the 30 days prior to a study visit, study staff asked participants to bring in all of their prescription medications, regardless of reason prescribed.

We found that adults who developed accelerated knee OA had greater knee pain, knee-specific disability, and greater global impact of arthritis and slower walking and chair-stand pace compared with those with a more gradual onset of knee OA.[17] These findings were true regardless of the study time point (before or after index visit). Hence, people with accelerated knee OA experienced greater pain and functional limitations up to three years before and after they developed accelerated knee OA. We found no differences for maximum isometric knee extension or flexion force, physical activity level, physical or mental quality of life, depression, or use of prescribed medicines.[17]

Despite the possibility of an interaction between group and time for knee pain, it was not statistically significant. However, we did find differences between groups related to changes in certain outcomes over time. We found that both groups started with similar chair-stand pace, but individuals with a gradual onset of knee OA subtly and gradually improved over time, while adults who developed accelerated knee OA gradually slowed until their index visit, then fluctuated over time.[17] Furthermore, individuals with a more gradual onset of knee OA were steadily less likely to report greater

Continued on page 46


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Continued from page 44

global impact of arthritis at each visit before the index visit, while those who developed accelerated knee OA were more likely to report greater global impact of arthritis during the same time period.[17]

We found that adults with accelerated knee OA are more likely to report greater pain severity and experience more functional limitations than those with a more gradual onset of knee OA, regardless of time. We also found that adults with accelerated knee OA often report being more affected by their knee symptoms and have diminished performance on a functional test (chair-stand) years before developing advanced-stage knee OA. We found a similar, though not statistically significant, trend for self-reported knee pain.

Our findings underscore that accelerated knee OA is a painful and disabling type of OA that warrants more attention. Clinicians should be concerned about patients without radiographic OA who report knee pain, because it may be an early symptom of accelerated knee OA.

Early MRI evidence

An important question for clinicians is why someone might experience knee pain years before they develop accelerated knee OA. It is possible that adults who develop accelerated knee OA are initially developing subtle changes that fail to appear on conventional knee x-rays but are related to knee pain.[14,19] In our preliminary analyses, we found that 18 adults with incident accelerated knee OA often had cartilage damage and meniscal pathology that was apparent on MRI before any radiographic evidence of accelerated knee OA.[14]

These findings agree with those of other investigators who have used MRI to show that adults with no radiographic OA had a high prevalence of cartilage damage (76%), bone marrow lesions (61%), and meniscal pathology (>20%).[19] In that study sample, these

lesions were associated with having knee symptoms and developing new symptoms.[19] Furthermore, the presence of effusion or synovitis and meniscal pathology may predict radiographic OA two years later.[20] Therefore, clinicians may be able to use MRI to identify high-risk adults who report knee pain without radiographic OA. This is a particularly appealing option if a patient has already had a recent knee MRI, if the clinician was already planning to order an MRI to rule out other pathologic findings (eg, acute meniscal tear), or if the cost of MRI decreases.

We used the findings from this study to clarify a conceptual model for the origin of accelerated knee OA (Figure 1). As mentioned earlier, we found in our prior work that being older and overweight are key risk factors for accelerated knee OA.[13] Furthermore, aging[19,21,22] and being overweight or obese[19,23,24] are associated with cartilage damage, bone marrow lesions, and meniscal pathology, which are related to knee symptoms among adults without radiographic OA.[19] Knee pain among those without radiographic OA is particularly concerning because knee pain is a risk factor for a new self-reported knee injury[25] that is severe enough to limit a person's ability to walk for at least two days. This new injury could be a trigger for accelerated knee OA.[13]

Once someone with accelerated knee OA develops advanced-stage knee OA, they are likely to report greater pain and experience more functional limitations for at least the next three years compared with individuals who have had a more gradual onset of knee OA. To prevent this sequela, it is vital that clinicians recognize the need to be more proactive with patients without OA who report knee pain because their pain could be early evidence of accelerated knee OA and will need to be managed accordingly.

Once a patient is considered high risk for accelerated knee OA, clinicians should treat the patient based on knee OA treatment guidelines. Most OA treatment guidelines[26] advocate managing symptoms not just with pharmacological interventions, but also with exercise programs, which could include goals for pain modification and injury or fall prevention. This treatment strategy is low risk and could help prevent a new injury that may trigger the onset of accelerated knee OA; however, this is not yet proven.

Conclusion

Accelerated knee OA is a painful and disabling type of OA in which symptoms and functional limitations may precede the rapid development of advanced-stage knee OA. Furthermore, the effect of accelerated knee OA may linger for at least three years after the onset of advanced-stage knee OA. Clinicians should be concerned about adults without radiographic OA who report knee pain because it may be an early sign of accelerated knee OA.

Jeffrey B. Driban, PhD, ATC, CSCS, is an assistant professor at Tufts Medical Center and Tufts University in Boston.

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Emerging treatments for diabetic neuropathy

Strict glycemic control is the most reliable treatment for diabetic peripheral neuropathy, but expanding knowledge of central and peripheral nervous system processes may help identify therapeutic methods that can effectively target the underlying mechanisms of pathogenesis.

By Sarnarendra Miranpuri, DPM, MD; Kush Patel; Gurwattan Miranpuri, PhD; Abhishek Chopra; and Ravinderjit Singh

The dramatic rise in the incidence of diabetes worldwide can be attributed to human behavior and lifestyle changes over the last century. In 1864, Marchel de Calvi described diabetic peripheral neuropathy (DPN) as a consequence rather than a cause of diabetes.[1] Neuropathy is a common complication of type 1 and type 2 diabetes, with a prevalence of nearly 8% in newly diagnosed patients with diabetes and more than 50% in patients with long-standing disease.[2] Several risks factors are associated with DPN, including hyperglycemia, older age, tobacco use, hypertension, obesity, alcohol consumption, and taller height.[1,2] The landmark Diabetes Control and Complication Trial demonstrated that proper glycemic control can reduce the development and progression of neuropathy significantly, by 64%.[3]

The presentation of diabetic neuropathy characteristically involves a burning or tingling sensation, hyperesthesia, sensory loss in the feet or hands, or combinations of these factors.[1] Patients may also experience cold or numb sensations. Sensorimotor and autonomic neuropathies are the most common diabetic neuropathies. Sensorimotor neuropathy presents with pain, paresthesia, and sensory loss, while autonomic neuropathy can contribute to myocardial infarction, malignant arrhythmia, and sudden death.

Distal sensory diabetic polyneuropathy affects up to 50% of patients.[2] The symptoms frequently affect patients' ability to perform daily activities. Up to half of these patients, however, may be asymptomatic, and an incidental finding of a painless foot ulceration can confirm the diagnosis.[2]

Pathophysiologically, DPN involves metabolic and vascular dysfunction. Hyperglycemia, paramount to the pathogenesis of DPN, plays a central role in the cascade of nerve damage, which is described below:[1]

1) Increased intracellular glucose and nerve and vascular tissue increases sorbitol and fructose and decreases myo-inositol. It also reduces nicotinamide adenine dinucleotide phosphate, which leads

As therapeutic approaches to diabetic neuropathy have evolved, researchers' understanding of the mechanisms underlying its pathogenesis has expanded.

Continued from page 49

to impairment of the primary vasodilatory system. The impaired vascular supply to the nerves ultimately plays a role in DPN.

2) Advanced glycation end products (hemoglobin, plasma albumin, lipoproteins, fibrin, and collagen) cause tissue damage.

3) Oxidative stress is the main pathological process that induces nerve damage by a direct toxic effect or by inhibition of nitric oxide production, thereby leading to nerve ischemia.

4) Intracellular glucose activates protein kinase C, decreases nerve growth factor, and decreases gamma-linolenic acid (subsequently reducing prostacyclin, a vasodilator), which is necessary for ensuring blood flow.

Currently, strict glycemic control is the most tangible treatment for DPN. Expanding knowledge of central and peripheral nervous system processes may help identify therapeutic methods that effectively target the underlying mechanisms of pathogenesis. An improved therapeutic approach may include using protective and regenerative factors to enhance the action to insulin-regulated genes in the endothelial cells to promote antioxidant and anti-inflammatory factors.[4]

A large number of neuroanatomical, neurophysiologic, and neurochemical mechanisms are thought to contribute to the development and maintenance of DPN.[1] Pathogenetic-oriented treatment overlooks the role of glycemic control and cardiovascular risk factors. Combination treatments involving pathogenetic and symptomatic drugs, however, are the most effective treatments for DPN.[5]

Long-standing therapies

Restoration of function and improved pain control are the treatment goals for DPN. Prior to initiation of medicinal therapy, clinicians can help manage risks of DPN with optimal glycemic control, improved lipid levels, blood pressure regulation, smoking cessation, and reduction of alcohol consumption.[2] There are several established modalities used in clinical practice for the treatment of DPN and improving patient quality of life.

First-line therapy with tricyclic antidepressants (TCAs, including amitriptyline, nortriptyline, imipramine, and desipramine)[6-12] in appropriately selected patients has been used widely without approved labeling from the US Food and Drug Administration (FDA). TCAs are thought to relieve pain by blocking neuronal reuptake of norepinephrine and serotonin, thereby inhibiting neurotransmitters in nociceptive pathways.[7] In particular, TCAs are used for deep pain ("pins and needles," "electric," or numb, achy pain).

A recent Cochrane review assessed five studies on the use of TCAs in the treatment of DPN and revealed overall effectiveness with a number needed to treat (NNT) of 1.3.[12] Of the TCAs, amitriptyline has the most published data in support of its use as a treatment for DPN; therefore, it should be recommended as the initial therapy

Table 1. Emerging approaches for gene therapy for DPN.

Target Agent	Key Milestones/Inquiry/Findings
NGF, BDNF, NT3, GDNF[38]	Proper prophylactic treatment of nerve growth factor (NGF) and increasing mRNA expression of NGF, brain-derived neurotrophic factor (BDNF), and neurotrophin-3 (NT3), and glial cell line-derived neurotrophic factor (GDNF) may be a viable treatment to prevent diabetic foot ulceration.
T-channels[39]	T-type calcium channels are implicated in peripheral nociception as it pertains to painful diabetic neuropathy.
Liver X receptor[40]	Activation of liver X receptor (LXR) increases levels of neurosteroids in the sciatic nerve, neuroprotective effects on thermal nociceptive activity, nerve conduction speed, and Na(+) K(+)-ATPase activity: a strong therapy for prevention of DN.
Ca2+ channel[41,42]	Modulation of neuronal Ca2+ may be a feasible therapeutic intervention for painful and degenerative aspects of peripheral neuropathies.
Nrf2[43]	Targeting Nrf2, a transcription factor modulating oxidative stress, may be a useful therapy to curb DPN pain.
Synthetic exendin-4[44]	A glucagon-like peptide-1 receptor agonist may be a useful therapeutic agent to prevent diabetes-induced peripheral nerve degeneration.
KU-32[45]	Administering KU-32 improved sensory neurons, possibly by enhancing mitochondrial bioenergetics; positive therapeutic effects on DPN.
KU-596[46]	An alternative to KU-32; improved diabetes-induced hypoalgesia.
HGF (hepatocyte growth factor) gene VM202[47]	VM202 is safe, well-tolerated and effective nonviral gene therapy for DPN and provided symptomatic relief with improvement in quality of life for three months. VM202 may be particularly beneficial for patients not taking gabapentin or pregabalin.
p2x3r[48]	p2x3r gene promoter DNA demethylation and enhanced interaction with p65 contributes to p2x3r sensitization and DPN.
AAV1.NT-3[49]	scAAV1.tMCK.NT-3 was delivered to the gastrocnemius muscle of mice. Gene therapy attenuates spontaneous autoimmune peripheral polyneuropathy.
VEGF/FLT-1[50]	SC-derived VEGF/FLT-1 (vascular endothelial growth factor receptor 1) signaling impairment affected neurite growth and the neutralization of SC-secreted VEGF was protective both in vitro and in vivo models.
p-CREB, NR2B[35]	Baclofen activated GABAB (γ-aminobutyric acid B) receptor attenuates diabetic neuropathic pain, partly via down-regulating the expression of p-CREB (phospho–cyclic AMP–responsive element binding protein) and NR2B (N-methyl-d-aspartate receptor 2B).
(rAAV)-shRNA-Nav1.3 vector[51]	Three weeks of viral vector administration in diabetic rats showed significant transduction in dorsal root ganglia, reduced neuronal excitability of dorsal horn neurons, and declines in tactile allodynia.

Continued on page 52

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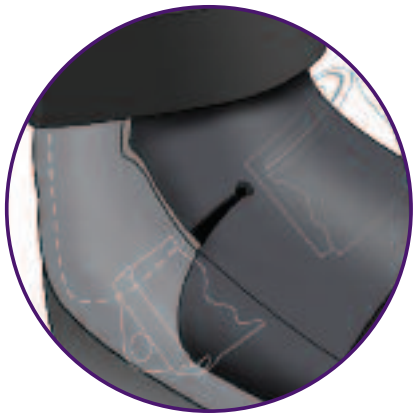

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Continued from page 50

in young patients. Desipramine is recommended for elderly patients and patients who are at risk for anticholinergic adverse effects.[7] These adverse effects often lead clinicians or their patients, particularly older adults, to discontinue use despite the overall effectiveness and affordability of this group of medications.

Although used to treat anxiety disorders, serotonin and norepinephrine reuptake inhibitors (SNRIs, including duloxetine, venlafaxine, and milnacipran)[2,12] are better tolerated and have fewer drug interactions than the TCAs typically used for DPN. A 2007 Cochrane review revealed an NNT of 3.1 for venlafaxine and 6 for duloxetine.[13,14] The selective serotonin reuptake inhibitors paroxetine and citalopram can also be used to treat DPN, but have a limited role.[12]

Anticonvulsants (eg, gabapentin, pregabalin)[14] can be used for DPN if there is inadequate response to TCAs. Gabapentin and pregabalin bind to the alpha-2-delta subunit of the calcium sensitive channels, modulating release of neurotransmitter. The FDA first approved gabapentin in 1994 for use in adult patients with partial epilepsy. In 1998, animal and human studies showed gabapentin has a possible analgesic action.[5] However, up-titrating the dosing to the level necessary for achieving a therapeutic effect is commonly needed.[5] Pregabalin, conversely, has a narrow therapeutic dosing range, which ensures a predictable response and enables an easy dosing process. Pregabalin is also FDA approved for treatment of DPN pain.

Another anticonvulsant, carbamazepine, has been used in treating neuropathy since the 1960s. It requires laboratory monitoring (renal function, liver function, reticulocyte count, platelet count).[15] Topiramate and lamictal are other anticonvulsants that lack evidence for their use in treating DPN.

Opiate (eg, methadone, levorphanol, morphine, oxycontin)[1,12] monotherapy, although controversial, is used to treat DPN in patients who fail other therapies. Nine studies showed a consistent benefit with this group of medications, with an approximately 20% to 30% reduction in pain.[16] Despite the concern of dependency with chronic opiate therapy, study guidelines do suggest a benefit to patients with DPN.[17] Tramadol, a synthetic opiate-like centrally acting narcotic analgesic, acts at mu-opioid receptors and inhibits reuptake of norepinephrine and serotonin. With a lower abuse potential than true opiates, the NNT with tramadol is 3.8.[18]

Topical therapies (capsaicin cream and lidocaine 5% patches)[19,20] have been used to treat neuropathic conditions. Capsaicin, an extract from chili pepper, stimulates depletion of substance P, the most common neurotransmitter in the pain transmission pathway. This cream has been specifically targeted for patients with superficial pain (eg, burning, allodynia, tingling). Most patients experience an initial burning sensation that may persist for several weeks. Lidoderm patches block neuronal sodium channels and have shown limited effectiveness in recent trials. The prime benefit of topical therapy is that it can be used in conjunction with systemic treatment.

Other therapies include N-methyl-D-aspartate (NMDA) antagonists, aldose reductase inhibitors, neurotropic factors, vascular endothelial growth factor, gamma linolenic acid, protein kinase C beta inhibitors, immune therapy, and hyperbaric oxygen therapy.[1,12,21,22] L-carnitine and alpha-lipoic acid are alternative therapeutic agents that are available over the counter, but more long-term data are

Table 2. Emerging stem cell therapies for DPN.

Cell therapy	Key Milestones/Inquiry/Findings
EPC[52]	AMD3100, a rallying mediator of EPC (endothelial progenitor cells), used in combination with stromal-cell-derived factor, performed successfully in making new endothelial cells.
Adult stem cells (vascular progenitors or adipose stem cells)[53]	Adult and induced pluripotent stem cells from cord blood; endogenous progenitor cells to regularize dysfunctional diabetic bone marrow and residing endothelial progenitors using nitric oxide donors, peroxisome proliferator-activated receptor (PPAR)-δ and -γ agonists, or inhibition of transforming growth factor beta (TGF)-β; advances in stem cell therapies for diabetic retinal microangiopathy may form the basis of first clinical trials in the near future.
Fetal liver-derived cell suspension transplantation[54]	Neither life-threatening complications nor significant differences in terms of evaluated diabetes complications (retinopathy, neuropathy, nephropathy, and cardiovascular diseases) between the case and control groups. However, one case of meningioma was reported.
hMSCs-UC[55] (human mesenchymal stem cells-umbilical cord blood)	Moderately reverses the neuronal degeneration of femoral nerve (FN), which might be contributed to the upregulation of nerve growth factor (NGF) with dramatic angiogenesis in FN-innervated gastrocnemius, consequently reversing neuronal structure and function, and preventing or curing foot ulceration.
Mesenchymal stem cells (MSCs)[56]	Cell transplantation might have therapeutic effects on DPN through secreting angiogenic/neurotrophic factors and differentiation to Schwann-like cells.
AMD3100/EPC/SDF-1α[57]	Injection into the sciatic nerve of mice with DPN suggests AMD3100 might be an effective EPC mobilization agent in diabetes, with local SDF-1α injection synergistically increasing vascularity in diabetic nerves.
MSCs[58]	Therapy improves diabetic cardiac autonomic neuropathy and decreases the inducibility of ventricular arrhythmias.
Hematopoietic cells[59]	Emerging evidence that diabetes compromises the function of the bone marrow (BM), producing a stem cell niche-dependent defect in hematopoietic stem cell mobilization; cells are not only victims but accomplices in diabetic complications.
BM-derived stem or progenitor cells[60]	These cells contain various angiogenic and neurotrophic factors and have been studied for treating experimental DPN; this is a novel option because of the effects on both vascular and neuronal components.
BM-MSCs, EPCs, bone marrow mononuclear cells (BMNCs)[61]	Introduced cellular mechanism of the diabetic ulcer. Discussed various stem cell therapies for lower extremity diabetic ulcers and their advantages and limitations.

Continued on page 54

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Continued from page 52

required to demonstrate their efficacy. Despite limited clinical data, these alternative options are still being used in patients with DPN.

Emerging therapies

Gene therapy and growth factor treatment have also been studied as viable treatments for DPN.[26] Creation of excess matrix metalloproteinase (MMP)-9 and MMP-2 causes deterioration in the extracellular matrix of blood vessels, which in turn can cause sensory or neuropathic difficulties for patients with diabetes.[4] Using minocycline and aspirin as nonselective cyclooxygenase inhibitors can be a novel method of decreasing diabetic neuropathy and restoring motor and sensory nerve conduction velocity.[25]

Resveratrol, a natural phenol, may play a significant role in treating the diabetic foot.[26] It may reinstitute insulin sensitivity, cytokine formation, tissue reconstruction, microcirculation, and peripheral nerve function.

Ghrelin, a small peptide, has been shown to have a relevant effect in multiple tissue systems when used to treat DPN due to its anti-inflammatory and regenerative abilities within neural tissues.[27]

ABT-594, a neuronal nicotinic acetylcholine receptor ligand, is extremely potent in animal models with neuropathic and nociceptive pain. In a placebo-controlled, double-blinded, seven-week study, individuals with painful diabetic neuropathy were randomized into four groups.[28] They received twice-daily treatment of either placebo or ABT-594. The experimental treatment was associated with improved pain scores in 50% of patients, but there were adverse effects, including headache, vomiting, dizziness, nausea, and abnormal dreams. Users of nicotine were less sensitive to these adverse effects.

Oxidative and nitrosative stress are key factors to consider when creating potential therapies for DPN.[29,30] Coenzyme Q10 acts as a free radical scavenger and an antioxidant to decrease oxidative stress within the peripheral and central nervous system.

Puerarin, one of several known isoflavones, has been found to alleviate the mechanical and thermal nociceptive response triggered by diabetes without interfering with the normal nociceptive process. Furthermore, it reduces levels of nuclear factor-κB and other proinflammatory cytokines, such as interleukin (IL)-6, IL-1β, and tumor necrosis factor (TNF)-α.[31]

Lipoic acid has therapeutic value as a nutritional supplement for DPN. Because of lipoic acid's lack of bioavailability due to its accelerated metabolism and instability, pharmacophores in conjunction with lipoic acid hybrids are being tested.[32]

Animal models

The streptozotocin diabetic rat has, to date, been the most commonly used model of DPN.[33] A systematic review of possible natural drugs to combat chronic neuropathic pain include: carotenoids (10%), phenols (10%), alkaloids (14%), terpenes (17%), flavonoids (28%), and others (21%).[34] One quarter of these studies looked at streptozotocin-induced diabetic neuropathy.

Baclofen, a gamma aminobutyric acid B (GABAB) receptor agonist, is a novel treatment for DPN that has been tested in animal models.[35] The attenuation of DPN can be accomplished by downregulating the expression of N-methyl-D-aspartate receptor subunit 2B (NR2B) and phosphorylated (p)-cyclic AMP response element-binding protein (CREB) through the activation of the GABAB receptor.

Table 3. Underlying mechanisms of DPN pathogenesis.

Target Agent	Key Milestones/Inquiry/Findings
NGF, BDNF, NT3[38]	Proper prophylactic treatment of nerve growth factor (NGF) and increasing mRNA expression of NGF, brain-derived neurotrophic factor (BDNF), and neurotrophin-3 (NT3) may be a viable treatment to prevent diabetic foot ulceration.
T-channels[39]	T-type calcium channels are implicated in peripheral nociception as it pertains to painful diabetic neuropathy.
LXR[40]	Activation of liver X receptor (LXR), which regulate adrenal steroidogenesis, results in increased levels of neurosteroids in the sciatic nerve, neuroprotective effects on thermal nociceptive activity, nerve conduction speed, and Na(+) K(+)-ATPase activity, a strong therapy for the prevention of DPN.
Ca2+ channel[41,42]	Modulation of neuronal Ca2+ may be a feasible therapeutic intervention for painful and degenerative aspects of peripheral neuropathies.
Spinal cord stimulation[62]	Implications of spinal cord stimulation should be considered as an alternative therapeutic approach to relieve diabetic neuropathy.
Nrf2[43]	Targeting Nrf2, a transcription factor modulating oxidative stress, may be a useful therapy to curb DPN pain.
Synthetic exendin-4[44]	Glucagon-like peptide-1 receptor agonist, may be a useful therapeutic agent to prevent diabetes-induced peripheral nerve degeneration.
B1R [63]	Key mediators in microglia activation, such as the inhibition of kinin B1 receptor (B1R), may provide preliminary evidence for treating DPN pain.
PARP 1/2[64]	PARP (poly-ADP-ribose polymerases)-1 and -2 inhibitor attenuates vincristine-induced mechanical allodynia in a diabetic rat model. Thus, PARP inhibition may be a valuable therapeutic approach.
TNF-α[65]	Thalidomide, a tumor necrosis factor (TNF)-α inhibitor, decreases neuropathic pain and may be able to effectively manage painful DPN.
Spinal microglia[66]	Activated spinal microglia as compelling target for the treatment of DPN.
PGC-1α[67]	Overexpressing PGC-1α (peroxisome proliferator-activated receptor-gamma γ coactivator 1α) in neurons to prevent oxidative stress associated with increased glucose levels.
IRS1, IRS2[68]	The role of IRS(insulin receptor substrate)1, IRS2, and associated signaling pathways that couple to Akt (protein kinase B) and the forkhead/winged helix transcription factor Foxo1.

Continued on page 56

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Continued from page 54

> Stem cell therapy is continually being investigated worldwide in hopes of developing safer and more efficacious treatment options for diabetic neuropathy.

Combination therapies—which include neutral endopeptidase inhibitors, aldose reductase inhibitors, lipoic acid supplementation, and insulin therapy with antioxidants—have been shown to prevent the loss of nerve conduction velocity in a diabetic rat model.[36] These therapies decrease levels of neuropeptide Y, tyrosine hydroxylase, somatostatin, IL-1β, and MMP-2 in streptozotocin-induced diabetic bone marrow supernatant.

Treating diabetic rats with a combination of enalapril, alpha lipoic acid, and menhaden oil ameliorated diabetes-induced steatosis, and elevated serum lipid levels and improved motor and sensory nerve conduction and corneal sensitivity of epineurial arterioles of the sciatic nerves.[37] These findings suggest the combination therapy's potential for treatment of vascular and neural complications caused by type 2 diabetes.

Gene and stem cell therapy

Gene therapy has been a high-yield research focus in the past few years with identification of various well-tolerated and effective gene therapy options for painful diabetic neuropathy. Various emerging gene therapy approaches for DPN[35,38-51] are shown in Table 1.

Clinical and basic science studies are beginning to highlight that stem cell therapies may be a suitable option in the near future for regenerative medical therapy. Indeed, stem cell therapy is continually being investigated worldwide in hopes of developing safer and more efficacious treatment options for diabetic neuropathy. Emerging stem cell therapies for DPN are shown in Table 2.[52-61]

Future perspectives

As therapeutic approaches to DPN have evolved, researchers' understanding of the mechanisms underlying DPN pathogenesis has expanded (Table 3).[38-44,62-68] In addition, recent clinical trials are evaluating the efficacy of multiple proteins involved in the treatment of DPN, such as erythropoietin analogs, angiotensin II receptor type 2 antagonists, and sodium channel blockers.[69]

This knowledge will be instrumental for the development of gene and cell therapies that will provide a foundation for the treatment of future generations. ler

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3 pair for $65

ECON II

A tri-laminate custom orthoses featuring 35 Tan EVA base, 1/16" PPT mid-layer and Blue plastazote top cover

3 pair for $75

Levy Rappel

800/564-LEVY (5389)
www.levyandrappel.com

ler new p

WalkingHealth Fall Risk Analysis

WalkJoy has updated the software used with its namesake gait restoration product to help clinicians assess a patient's risk of falling. WalkingHealth is a portable gait analysis tool to identify a patient's risk of falling as low, medium, or high, along with tracking functional outcome data for physical therapy G-Code documentation. Together, WalkJoy and WalkingHealth provide a hardware-plus-software gait analysis solution for use in physical therapy, podiatry, oncology, and independent and assisted living settings for patients with diabetes, neurological disorders, orthopedic injuries, and fall risks.

WalkJoy
855/WALKJOY (925-5569)
walkjoy.com

Tendon Trak For Tendinopathy

Created by a physical therapist for treating lower extremity tendinopathies, the Tendon Trak from Innovative Bracing supports the tendon to decrease pain and improve function by facilitating anatomically efficient muscle contraction without excessive compression. The Tendon Trak's low profile promotes a comfortable fit and increases compliance in patients suffering from Achilles tendinopathy, patellar tendinopathy, Sever disease, and Osgood-Schlatter disease. Manufactured in the US, the Tendon Trak device comes with an 18" strap or a 23" strap for use at the ankle and knee, respectively.

Innovative Bracing
860/529-800
tendontrak.com

FootFidget Exercise Device

Designed by physical therapists, the FootFidget is a versatile exercise device for health and fitness that can be used as a dynamic footrest when sitting or standing for long periods of time. "Fidgeting" (pressing the foot against the device's flexible resistance bands) while sitting or standing can help improve lower extremity strength, endurance, and circulation; other potential benefits include improved balance, ergonomic positioning, comfort, and focus. The FootFidget comes with one set of medium resistance bands; the FootFidget Pro comes with three sets of bands and can be set at one of three heights.

FootFidget
224/622-3154
footfidget.com

3DSizeME with Color Detection

The 3DsizeME system from TechMed 3D—including the Structure Sensor, 3DsizeME free iPad app, and MSoft software—is a compact, versatile, and affordable scanning solution that now offers a new color detection feature. Operators can now use colored positioning targets to identify anatomical landmarks or to align the 3D form automatically. The operator can use blue or green anatomical positioning targets on the body that are detected during the scan and appear automatically on the 3D form. When scanning a foot or device, the operator can use five positioning targets to immediately obtain 3D alignment.

TechMed 3D
855/TECHMED (832-4633)
techmed3d.com

roducts

TheraSport Leg Sleeves

Therafirm's new TheraSport brand introduces TheraSport Athletic Leg Sleeves to round out a product line engineered to improve circulation and help provide energy for greater endurance, enhanced performance, and a speedy recovery. The soft, lightweight sleeves feature a comfortable band with a nonbinding grip to help the socks stay up. High-tech, moisture-wicking yarns, as well as breathable mesh paneling and moisture-wicking fibers, keep legs cool and control odor. In addition to the existing athletic sock colors (black, white, red, blue, and pink), TheraSport Leg Sleeves are also available in lime.

Therafirm, a Knit-Rite Company
866/842-0984
therafirm.com

SportsAT 2.0 Software

New from Tekscan, the SportsAT 2.0 software provides essential information for complete balance and concussion assessments. Sports medicine professionals can use SportsAT 2.0 to complete objective baseline and postinjury tests, quickly assess injury risk, identify asymmetries, and evaluate progress. Initiate balance training exercises using the Balance Compass to quantitatively measure postural stability and control. Simplify chronic ankle instability assessments with no need to export center of force (CoF) data. CoF movement and velocity provide insights into postural stability and fall risk assessments.

Tekscan
800/248-3669
tekscan.com

Orpyx SurroSense Rx

Orpyx's flagship product, the SurroSense Rx, is a wearable tool to help manage and track complications associated with diabetic peripheral neuropathy. The SurroSense Rx system alerts the user via a smartwatch when plantar pressures are detected above a threshold level for a prolonged period, so the user can take action before damage occurs. In addition to the direct feedback provided by the system, it tracks metrics over time. Data can be viewed easily using a personalized Orpyx Connect account, which the patient can share with healthcare providers to track outcomes and compliance.

Orpyx Medical Technologies
855/996-7799
orpyx.com

FS6 Compression Foot Sleeve

Allied OSI Labs has added the FS6 Compression Sleeve to its growing catalog of products for foot and ankle practices. The FS6 Compression Sleeve is designed to relieve the pain and swelling associated with plantar fasciitis by offering six zones of compression and support: firm compression at the Achilles tendon and midfoot, moderate compression at the heel and forefoot, and mild compression at the ankle and toes. The thin, light FS6 Compression Sleeve conveniently fits under normal socks and in shoes, and is easy for patients to put on and take off. The moisture-wicking fabric helps keep feet cool and dry.

Allied OSI Labs
800/444-3632
alliedosilabs.com

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market mechanics

By Emily Delzell

Lakers celebrate Vitti, retiring head AT

The Los Angeles Lakers honored retiring head athletic trainer Gary Vitti, MS, ATC, during the first quarter of the Lakers' April 3 home game against the Boston Celtics. Vitti, who will retire from his full-time role this summer, has been with the Lakers for 32 seasons and is the longest-tenured trainer in the National Basketball Association (NBA). He'll continue part-time as a Lakers consultant for two more years.

During a video tribute Vitti was presented with a framed Lakers jersey and two first-class tickets to Italy, where he has a vacation home.

Vitti began his career as an assistant trainer with the Utah Jazz (1981-1982). He then served as head athletic trainer at the University of Portland in Oregon before joining the Lakers prior to the 1984-1985 season. The Lakers have won eight NBA championships since Vitti joined the organization.

The NBA Trainers Association named Vitti its Trainer of the Year in 1991, partially due to his philanthropic work. Additional honors have included Southern Connecticut State University's 2001 distinguished alumni award and the 2011 "Local Hero Award" presented by the Los Angeles-based Team Heal Foundation, which works to bring injury prevention, medical care, athletic training, and mentoring to high school athletes in underserved communities. ler

Reichel forms sales rep group Medlutions

Patrick Reichel, a podiatry-focused medical, surgical, and pharmaceutical brand management expert, announced in March that he has formed Medlutions, a contract sales representative group and consulting firm for the Midwest region.

The LaGrange Park, IL-based company has partnered with Amerx Health Care, Anodyne, and 4Path Pathology Services as their Midwest sales force and will work directly with physician offices as a contracted sales associate for manufacturers of medical products, devices, and equipment and medical service providers, according to a company release. ler

Tekscan updates SportsAT software

Boston-based Tekscan in February released its SportsAT 2.0 for balance assessments.

SportsAT, which includes a built-in balance compass to aid in rehabilitation and track progress, helps lower extremity practitioners quickly quantify gait asymmetries and evaluate rehabilitation progress, and features automated time-to-boundary calculations.

Researchers from University of Delaware in Newark in 2014 published data in the *Journal of Sport Rehabilitation* validating that the Balance Error Scoring System (BESS) portion of Tekscan software was comparable to traditional BESS scoring using trained testers. ler

Nike releases three new easy-don shoes

Beaverton, OR-based shoe giant Nike in March released three athletic shoes newly fitted with its Flyease entry system, designed for children and adults with disabilities (See "Nike launches easy-don Flyease high-tops," August 2015, page 66).

The LeBron Soldier IX Flyease and Pegasus 32 Flyease are available now in retail stores and at nike.com, and the Flex Run Flyease will be available beginning May 1. ler

Radius Health drug cuts fracture risk

Waltham, MA-based Radius Health's investigational drug abaloparatide-SC (subcutaneous) increases bone mineral density in the spine and hip in postmenopausal women with osteoporosis and may reduce their risk of vertebral and nonvertebral fractures, according to research presented April 1 at Endo 2016, the annual meeting of the Endocrine Society, in Boston.

Researchers investigated patients enrolled in the randomized, double-blind, comparative, multicenter international phase 3 ACTIVE clinical trial to evaluate the efficacy and safety of 80 µg of abaloparatide-SC for preventing fractures in otherwise healthy ambulatory postmenopausal women with osteoporosis. Overall, 2463 patients aged between 49 and 86 years were randomized to one of three treatment arms for 18 months: double-blind 80 µg of abaloparatide-SC, blind-matched placebo, or open-label 20 µg of subcutaneous teriparatide.

Abaloparatide-SC significantly increased bone mineral density in both the spine and hip and significantly reduced the risk of new vertebral and nonvertebral fractures compared with placebo regardless of baseline patient characteristics, including age, bone mineral density, and history of prior fractures.

The drug also reduced major osteoporotic fractures by 55% compared with teriparatide and increased bone density to a greater extent in the hip region compared with teriparatide.

Radius Health funded the study. ler

Foot Management honors LA Rams ATs

Baltimore, MD-based Foot Management hosted the Ed Block Courage Award dinner on March 11 to honor the St. Louis/LA Rams athletic trainers as the Ed Block NFL Athletic Training Staff of the Year.

The Professional Football Athletic Trainers Society membership recognizes one National Football League athletic training staff annually for distinguished service to their club, community, and athletic training profession. The awards are dedicated to Ed Block, who was the head athletic trainer for the Baltimore Colts from 1954-1977 and was inducted into the NATA Hall of Fame in 1974. ler

Patients give custom devices high marks

The Winnipeg-based Pedorthic Association of Canada (PAC) on April 11 released results of a patient-centered study showing six weeks of custom foot orthosis wear reduced foot and ankle pain and upped activity levels in most people who wore the devices.

The Patient Outcome Measure Evaluation Strategy (POMES) study surveyed 217 patients from 23 Canadian pedorthic clinics over 12 months about their perspectives on custom orthoses before and after using the devices. All were aged between 18 and 65 years and presented with a below knee musculoskeletal condition.

Survey results showed that 91% of patients reported reduced pain after wearing custom orthoses for six weeks; 81% said the orthoses met their goals; 83% said they were very satisfied with how often they wore their orthoses; 93% felt the advantages of the devices outweighed the disadvantages; 77% said their orthoses were very comfortable; and 87% would recommend custom orthoses to family and friends.

Visit pedorthic.ca for more information. ler

Continued on page 62

Continued from page 61

Ongoing Care OA brace improves gait

Ongoing Care Solutions' pneumatic unloader brace with extension assist improved various gait parameters in people with knee osteoarthritis (OA) compared with standard conservative care, according to a study published in the April issue of *The Journal of Knee Surgery*.

Investigators at Rubin Institute for Advanced Orthopedics in Baltimore randomized 24 people with knee OA to treatment with the OA Rehabilitator brace made by the Pinellas Park, FL-based company and 12 to standard care.

After wearing the brace for at least three hours a day for three months, braced participants had significant improvements in walking speed, knee extension at heel strike, total range of motion, knee joint forces, and rate of knee flexion from midstance to terminal stance compared with the matched cohort.

These results confirm those of a pilot study published in 2013 in the same journal. ler

15 high schools get $50K each for ATs

The NFL Foundation, the National Athletic Trainers' Association (NATA), Gatorade, and the Professional Football Athletic Trainers Society (PFATS) announced on March 14 the winners of the Athletic Trainer Initiative at the seventh-annual Youth Sports Safety Summit, held in Alexandria, VA, and hosted by NATA and the Youth Sports Safety Alliance.

The national grant contest expands access to athletic trainers in underserved high schools to improve youth athlete safety.

The 15 winning schools received $50,000 each to develop athletic training programs that will provide nearly 5000 student athletes with consistent access.

The grant contest, which launched in October 2015, is an extension of the partners' athletic training outreach program, which has provided more than $3 million to help fund athletic trainers in communities nationwide. ler

Amniox allografts help heal DFUs

Atlanta-based Amniox's umbilical cord and amniotic membrane (UC/AM) wound allograft product helped promote healing in chronic diabetic foot ulcers (DFUs), according to findings of a retrospective study presented on March 23 at the Superbones Superwounds East 2016 Conference in Teaneck, NJ.

After wound debridement, clinicians applied Neox Wound Allograft (cryopreserved UC/AM) directly to 32 DFUs in 29 patients, securing grafts with sutures. Complete healing was achieved in 28 wounds, or 87.5%.

The initial wound area was an average of 10.6 cm^2, average time to healing was 13.79 weeks, and the average number of Neox applications was 1.68. ler

BOC wins fifth Stevie award in 4 years

The Board of Certification/Accreditation (BOC) won a bronze Stevie on March 4 at the annual business awards ceremony, held in Las Vegas.

The Owings Mills, MD-based BOC won the E-Commerce Customer Service award in recognition of its innovative customer service support and social media efforts. This is BOC's fifth Stevie award win in four years, four of which were for customer service. ler

ACSM debuts translational journal

The Indianapolis, IN-based American College of Sports Medicine (ACSM) on April 5 epublished the first issue of its new peer-reviewed journal, *Translational Journal of the American College of Sports Medicine (TJACSM)* at journals.lww.com/acsm-tj.

The new online journal publishes original research, clinical trials, systematic reviews, meta-analyses, and policy research addressing the translational implications of basic, clinical, and policy science to everyday, real-world practice in sports medicine and exercise science.

"This new journal will place ACSM in the forefront for translational and policy science related to exercise," said *TJACSM* Editor-in-Chief Joseph E. Donnelly, EdD, FACSM, a professor of internal medicine and director of the Energy Balance Laboratory and Center for Physical Activity and Weight Management, Cardiovascular Research Institute at the University of Kansas Medical Center in Kansas City.

Plans call for publishing new articles every other week. Donnelly and the editorial board also envision periodic special issues focusing on current topics in translational sports medicine and exercise science. Content will be free for a time, then will be provided as an ACSM member benefit and available for individual and institutional subscriptions. Wolters Kluwer, headquartered in Alphen aan den Rijn, the Netherlands, publishes *TJACSM*. ler

Leipzig to host 2016 OTWorld in May

The German Association of Orthopaedic Technology in March announced dates for OTWorld, an international trade show and world congress that this year will focus on 3D printing.

Attendees and presenters at OTWorld 2016, scheduled for May 3-6 in Leipzig, will consider choices of 3D materials, design, and manufacturing methods, while the trade fair will demonstrate the latest 3D printing solutions, including the FabBus from the Goethe Lab project for additive manufacturing at Aachen University of Applied Sciences. This mobile laboratory is installed in an adapted double-decker bus, which was once part of the Berlin traffic network.

Go to ot-world.com for more information. ler

ASB holds first National Biomechanics Day

The American Society of Biomechanics (ASB) on April 7 had its first National Biomechanics Day, a nationwide, synchronized event held at labs and other biomechanics facilities around the nation to educate high school students and their teachers about the field.

Paul DeVita, PhD, current ASB president and professor of kinesiology at Eastern Carolina University (ECU) in Greenville, NC, established the event to raise awareness of biomechanics, which is almost always first introduced at the university level. The long-term goal of the annual event, he said, is to make biomechanics a part of high school curricula.

ECU faculty and students were among 160 university and commercial biomechanists in 30 states to participate, and its event hosted 50 local high school students at the biomechanics lab in the Department of Kinesiology and the human movement analysis lab in the Department of Physical Therapy. ler

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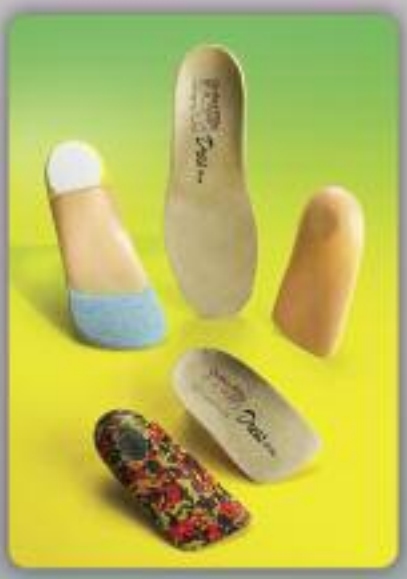

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