LITERATURE REVIEW

COMMUNITY-ASSOCIATED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

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ABSTRACT

Methicillin resistant Staphylococcus aureus (MRSA), is a problematic infection which is becoming more common in a variety of athletic related environments. Early recognition, diagnosis, and timely management of infection can help minimize the severity of infection and decrease the rate of transmission. Since most sports physical therapists typically lack adequate knowledge and ability to identify cases of MRSA infection, the purpose of this review is to provide a background for associated risk factors, recognition, treatment, and prevention of community associated-MRSA in athletic environments.

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PROBLEM

Introduction

The role of the sports physical therapist as a member of the sports medicine team continues to expand, making it critical to increase the knowledge related to non-musculoskeletal issues. Recognition and management of skin infections becomes increasingly necessary in the comprehensive medical care of the athlete. One infection of concern is Staphylococcus aureus, a commonly occurring bacterial species which is moving from nosocomial (hospital based) to community acquired infections found in a variety of athletic related environments.[1,2] Staphylococcus aureus is a naturally occurring bacteria found on skin and can be divided into two categories based on its susceptibility or resistance to beta-lactam antibiotics; methicillin susceptible (MSSA) or methicillin resistant (MRSA).[3]

Methicillin resistant Staphylococcus aureus has been typically associated with healthcare acquired (HA-MRSA) risk factors including recent surgery and hospitalization, indwelling catheter, kidney dialysis, and prolonged stay in long-term care facility (nursing home, hospice).[4,5] In the late 1990's, cases of MRSA began to appear in the community (CA-MRSA)[6] and athletic related environments.[7,8] These athletic related cases had risk factors for acquisition and clinical characteristics distinct from HA-MRSA.[5,9-12] Risk factors included participation in contact sports with repeated close physical contact with other competitors, open abrasions, increased use of antibiotics, and sharing of personal equipment.[7,8,10,12-15]

The medical management of MRSA can be problematic due to low incidence rates, rapid transmission, and limited pharmacological interventions that effectively manage the infection. Low incidence rates of CA-MRSA in athletic environments can delay accurate recognition, diagnosis, and timely management which can increase the risk of infection in other individuals and cause subsequent increase in morbidity. Unrecognized or untreated cases of MRSA can quickly progress to a severe infection requiring hospitalization,[16,17] surgery,[17] and may lead to death. Selection of appropriate treatment strategies can also be further complicated by the characteristic antibiotic resistance patterns associated with CA-MRSA.[5,10] Most sports physical therapists typically lack adequate knowledge and ability to identify cases for physician referral and an understanding of the severity and complications of untreated cases. Since the prevalence of CA-MRSA cases has increased in athletic environments,[12-14,18-20] individuals who provide medical care for athletes should have a basic understanding of CA-MRSA infections. Therefore, the purpose of this review is to provide the sports physical therapist with a background for associated risk factors, recognition, treatment, and prevention of CA-MRSA in athletic environments.

Literature Search

A search of PubMed (1966-2006) and Web of Science (1981-2006) was conducted in December 2006, using combinations of the terms, Staphylococcus aureus, methicillin resistant, MRSA, athletic, and sport. The search was limited to articles written in English using human subjects. Fifty articles were obtained in the original search and titles and abstracts were screened for relevance. References were also cross referenced and other relevant literature was reviewed for identification of studies not found using the original search terms. A total of 13 manuscripts ranging from case reports to retrospective cohort studies related to CA-MRSA in athletics were retained.[7,8,12-14,16-23]

Risk Factors

Staphylococcus aureus is a ubiquitous bacteria which can cause infections in patients of any age, race, gender, or economic background and has been found in a variety of settings, regions, and countries.[24] Community associated-MRSA has been diagnosed in individuals who participate in contact and non-contact sports from high school to professional levels *(Table 1)*. Reported cases have ranged from 1-13 athletes per team and involved up to 25% of the team.[7,8,12-14,16-23] Three common factors implicated in the development of CA-MRSA include exposure to infection, compromised skin integrity, and transmission via direct (person-to-person) or indirect contact (person-to-object).

Exposure to Infection

Swabs from the anterior nares indicate approximately 35% of healthy individuals are colonized with MSSA[3,25] and less than 10% are colonized with MRSA.[12-14,25] Teens, younger adults, males, and individuals with asthma tend to be the most likely carriers.[3,5,25] The high use of antibi-

Table 1. *Community-Associated Methicillin-Resistant Staphylococcus Aureus (CA-MRSA) Cases*

Article	Population	Number of Athletes Infected	Percentage of Team Infected
Stacey et al, 1998[8]	Rugby- club players	5	25%
Lindenmayer et al, 1998[7]	Wrestlers- high school	7	22%
Centers for Disease Control, 2003[20]	Fencers- club team Football- collegiate and high school Wrestlers- high school	3-5 2-10 2	7% unknown unknown
Noe et al, 2004[18]	Football- collegiate	8	unknown
Begier et al, 2004[13]	Football- collegiate	10	10%
Kazakova et al, 2005[12]	Football- professional	5	9%
Nguyen et al, 2005[14]	Football- collegiate	11	11%
Rihn et al, 2005[19]	Football- high school	13	13%
Cohen, 2005[21]	Weight lifters	1	unknown
Cohen, 2005[22]	Weight lifters, volleyball, basketball, football- collegiate varsity and recreational	7	unknown
Kimmel, 2006[16]	Football- collegiate	10	10%
Romano et al, 2006[17]	Football- collegiate	1-11	1-16%
Huijsdens et al, 2006[23]	Soccer- professional	9	21%

otics throughout the year is an additional risk factor for CA-MRSA.[12] Increased antibiotic use alters nasal bacterial flora and thus provides a more conducive environment for MRSA colonization.[26]

Compromised Skin Integrity

Areas of compromised skin integrity serve as an entry portal for a variety of infectious agents. The most common method of transmission is thought to occur when sites of skin injury or abrasion are in direct contact with sites of bacterial colonization.[7,8,12-14] Burns associated with athletic surfaces such as turf or wrestling mats[7,12,13] are associated with CA-MRSA infections, both near[12] and distant to the site of injury.[13] Infection tends to occur most often on extremities which are not covered by athletic apparel (elbow, forearm, knee, lower leg).[1,12] Unfortunately, areas covered by clothing or protective equipment are also at risk following recent skin trauma due to shaving or abrasions.[13] Shaving body areas other than the face (cosmetic shaving) has been shown to increase the risk of infection of areas covered by athletic equipment and clothing.[13]

Transmission via Person-to-Person

The most common method of transmission is direct contact with contaminated individuals.[7,8,12-14] A definitive relationship has been demonstrated between CA-MRSA cases and direct contact with infected individuals.[7,8,12-14] Risk of infection is thought to be proportional to the frequency and duration of exposure to infected individuals. Prolonged contact usually occurs between athletes on the same team, as well as competitors from other teams who participate in sports with a greater duration of competition (football). This risk is less for athletic events with shorter durations (wrestling).[7]

American football,[1,2,12,14,17-20,22] wrestling,[7,20] and rugby[8] have been commonly implicated in increased infection rates due to frequent close contact between athletes. Retrospective analysis of cases indicate the highest infection rates tend to occur with positions such as rugby forwards;[8] American football linemen,[12,14,17] cornerbacks, and wide receivers;[13] and wrestlers[7] where repeated direct contact occurs throughout competition and practices. It should be noted since CA-MRSA has a low incidence rate, analysis of infection rates by position or sport should be interpreted with caution. Community associated-MRSA has been proposed to be more common in individuals with a higher body mass index (BMI).[12] Wrestlers tend to be affected equally across all weight classes[7] which contrasts findings that CA-MRSA has been associated with higher BMI.[12] Findings related to higher BMI in American football may have been due to the association between position (i.e. linemen) and weight. Additionally, wrestlers may be at decreased risk due to strict guidelines involving participation in competition and management of skin infections and open wounds.[27]

Figure 1. *Example of community associated-methicillin-resistant Staphylococcus aureus infection on the posterior aspect of a forearm (photo courtesy of Rod Walters, DA, ATC; University of South Carolina).*

Transmission via Person-to-Object

Identification of the source of infection plays a critical role in limiting the spread and thorough management of CA-MRSA. As CA-MRSA infections become more common in athletics, it is possible that individuals who compete against each other could become independently infected with the same strains,[12] thus increasing the difficulty to identify the source of infections.

Cases reported in non-contact sports have been associated with shared equipment in facilities such as the locker room or athletic training room.[16,20] Even roommates of infected individuals have been shown to be at risk of infection.[14,23,28] Cases of MRSA have also been reported in athletes who shared fencing equipment,[20] razors,[13] towels,[12,20] and bar soap.[14] Staphylococcus aureus has also been shown to be present in whirlpools and taping gel applicators.[12] Common areas in athletic environments which may aid in the transmission of infections include treatment tables, benches, training equipment, and flooring (especially carpeted) in locker rooms, weight rooms, and athletic training rooms. However, other studies have failed to demonstrate a definitive link between MRSA cases and playing surfaces[12] or sharing of equipment or facilities.[1,2,13,19]

SOLUTION

Case Identification/ Differential Diagnosis

The most common presentation of CA-MRSA is in the form of soft tissue lesions such as an abscess or cellulitis.[5,6,12,14,15,29,30] Visual inspection offers several clues for identification of CA-MRSA *(Figure 1 and 2)*. Clinically, the lesion and erythema are associated with pain that is out of proportion for the severity of the lesion. Other characteristic signs and symptoms include redness, swelling, warmth, and possible purulent discharge from the lesion site. Lesions are commonly mistaken for a spider bite,[15,29] pimple, or boil.[12,14] Occasionally, the diameter of the affected area can be as large as 5-7 cm.[12]

Management

The increased prevalence of MRSA soft tissue infections found in the general population necessitates a low threshold for timely physician referral following the appearance of a lesion that presents with the aforementioned characteristics. The sports physical therapist should contact the physician to facilitate and ensure expedited care. Since recurrent infections are common[12,13] the sports physical therapist should have an even lower threshold for physician referral for individuals with a history of CA-MRSA. In the event that the team physician or patient's primary care physician is unavailable for immediate consult, the patient should be directed to an urgent care facility. Until the wound has been formally diagnosed, MRSA should be suspected.

A small, uncomplicated area can be managed using over the counter topical antibacterial ointment (Neosporin), with close monitoring of the concerning lesion for up to 48 hours. The sports physical therapist should be able to gain a sense over 24-48 hours as to whether an area is progressing to something more concerning. Worsening signs or symptoms during the first 48 hours or failure of the lesion to improve within 48 hours warrants immediate physician referral. Any degree of either fluctuance (presence of pus), pronounced erythema, progressive warmth, or increasing pain should raise serious suspicion for infection, particularly in patients who may have been briefly hospitalized during their post-operative period. Surgical wounds should also be monitored in a similar manner.

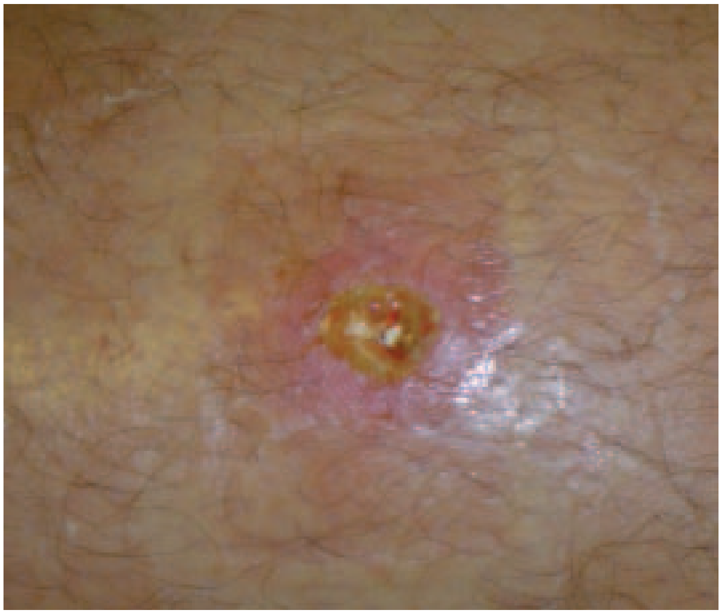

Figure 2. *Example of community associated-methicillin-resistant Staphylococcus aureus infection over the anterior lower leg (photo courtesy of Allen Hardin, PT, SCS, ATC; University of Texas)*

During the initial management period, the sports physical therapist should stress the importance of wound management and suggest strategies to limit opportunities for exposure to other individuals. Education regarding personal hygiene such as frequent hand washing and daily bathing with antibacterial soap[31] or an antimicrobial agent, such as povidone-iodine liquid soap or 4% chlorhexidine, should be encouraged.[12,14,15,20,32] Athletes should be required to keep wounds clean and covered to decrease transmission between individuals.[13,33] Failure to adequately cover wounds[14,15] and multiple contacts with infected individuals further increase risk for infection.[7,12] The sports physical therapist should also utilize frequent hand washing or the use of alcohol based gel before and after individual treatment sessions.

Specific Treatment

A critical step in the specific management of CA-MRSA is recognizing clinical differences between mild skin infections and CA-MRSA infected lesions. Unfortunately, early in the disease process, skin lesions may not present with signs and symptoms suggestive of CA-MRSA. Early intervention is necessary to prevent transmission and progression of infection. The first line of treatment for mild skin infections includes the use of either topical Mupirocin or oral antibiotics directed towards common skin flora.[28,34] Topical antibiotics are useful for less severe infections. However, strains of CA-MRSA are resistant to numerous antibiotics, including commonly used beta-lactams, cephalosporins, macrolides, penicillins, and quinolones.[5,10] Therefore, if the initial treatment with oral antibiotics is unsuccessful in eradicating pain, swelling, or erythema, the lesion should be excised, drained, and the fluid cultured to direct treatment based on antibiotic sensitivity.[5,6,35]

Systemic antibiotic use should be reserved for severe infections or mild to moderate infections that cover a large surface area. Large wounds may require hospitalization, surgical debridement, and intravenous antibiotics.[17] It is recommended to use an antimicrobial agent that successfully treats a variety of gram positive bacterial infections.[28,34] Antibiotics such as clindamycin, sulfamethoxazole/trimethoprim (SMX-TMP), ciprofloxacin, rifampin, vancomycin, or linezolid are appropriate treatment options.[19,28,31,34] However, treatment with SMX-TMP and fluoroquinolones have been shown to be associated with higher recurrence rates and are not recommended as first line therapy.[28] Most infections resolve in 10 days with appropriate treatment.[12] Infections that do not respond to initial treatment regimens or recur should be treated with another group of antibiotics based on antibiotic sensitivity.[12,13,30]

Obtaining nasal cultures from individuals who may have been exposed to CA-MRSA to determine the presence of infection is questionable.[19] Approximately 35% of asymptomatic individuals carry MSSA[3,25] and less than 10% of asymptomatic individuals carry MRSA.[12-14,25] A positive nasal culture may warrant the use of topical mupirocin if deemed necessary by the treating physician. Topical mupirocin is applied to the nares prophylactically for 5 days to 4 weeks to prevent or limit the spread of infection,[19,20,28,30,36] but may not entirely reduce the risk of infection since infection can occur regardless of a positive nasal culture.[19]

APPLICATION IN PHYSICAL THERAPY

Prevention

The management of CA-MRSA begins with prevention. Reports of CA-MRSA cases tend to be relatively isolated, yet individuals have been diagnosed in numerous athletic settings.[7,8,12-14,18-20] Although CA-MRSA cannot be entirely prevented, best practice measures can be employed to reduce the risk among athletes. The National Athletic Trainers' Association (NATA) and the Centers for Disease Control (CDC) have outlined recommendations *(Tables 2 and 3, respectively)* for the prevention and management of CA-MRSA.[20,33] One retrospective study was able to demonstrate a decreased rate of infection after implementing a variety of prevention strategies.[17] Future research should determine the effectiveness of these recommendations.

Table 2. *Official recommendations from the National Athletic Trainers' Association (NATA) regarding Community associated-methicillin-resistant Staphylococcus aureus.*[28]

1. Keep hands clean by washing thoroughly with soap and warm water or using an alcohol-based hand sanitizer routinely.
2. Encourage immediate showering following activity.
3. Avoid whirlpools or common tubs with open wounds, scrapes, or scratches.
4. Avoid sharing towels, razors, and daily athletic gear.
5. Properly wash athletic gear and towels after each use.
6. Maintain clean facilities and equipment.
7. Inform or refer to appropriate health care personnel for all active skin lesions and lesions that do not respond to initial therapy.
8. Administer or seek proper first aid.
9. Encourage health care personnel to seek bacterial cultures to establish a diagnosis.
10. Care and cover skin lesions appropriately before participation.

Athletes should be encouraged to use liquid antibacterial soap (3% hexachlorophene or 4% chlorhexidine) for showers immediately after practice and frequently wash hands. Clinicians should also be encouraged to wash hands thoroughly with soap or utilize an alcohol based hand sanitizer, both before and after treatment sessions. Attention to meticulous wound cleanliness and care should be rigorously emphasized. Universal precautions should be employed when treating open skin wounds, which may serve as a reservoir of infection. All wounds should be appropriately covered during and following competition at all athletic events.

Techniques to reduce the frequency of abrasions should be employed, although the use of elbow and knee pads may facilitate infection due to moisture retention conducive to bacterial growth.[13] It is recommended that disposable, adhesive mesh strips *(Figure 3)* be firmly secured to the skin in order to prevent wounds that occur via contact from either playing surfaces or other competitors. Skin irritation may develop in a subset of athletes with sensitive skin secondary to removal of the mesh on a daily basis. Therefore, the decision to use an adhesive mesh should be made on an individual basis.

Table 3. *Centers for Disease Control (CDC) recommendations for preventing methicillin-resistant Staphylococcus aureus among sport participants*[18]

1. Cover all wounds. If a wound cannot be covered adequately, consider excluding athlete with potentially infectious skin lesions from practice or competitions until the lesions are healed or can be covered adequately.
2. Encourage good hygiene, including showering and washing with soap after all practices and competitions.
3. Ensure availability of adequate soap and hot water.
4. Discourage sharing of towels and personal items (for example, clothing or equipment).
5. Establish routine cleaning schedules for shared equipment.
6. Train athletes and coaches in first aid for wounds and recognition of wounds that are potentially infected.
7. Encourage athletes to report skin lesions to coaches and encourage coaches to assess athletes regularly for skin lesions.

The practice of sharing personal hygiene products (soap, razors) and sports equipment should also be discouraged. Individual or disposable towels should replace community towels commonly used at practices and games. Towels used in the clinic should be washed in hot water (> 71˚C) using detergent and chlorine bleach after each use.[32]

Athletes should be required to shower immediately following practice before entering the sports medicine clinic or athletic training room. Equipment such as treatment tables and weight room benches should be cleaned and disinfected appropriately after use with a commercial product that contains anti-viral and anti-microbial substances, such as CaviCide® (Unimed-Midwest, Inc., Burnsville, MN).[37] Pillow cases and other treatment table linens should be changed following individual treatment sessions and washed in a similar manner as towels. Proper disinfection techniques should also be employed for hydrotherapy (whirlpool) areas. Circulating tubs should be cleaned with an Environmental Protection Agency (EPA) registered disinfectant. The cleaning solution is circulated in the spa for 10 minutes, followed by a rinse with water, and finally air-dried.[37] Non-circulating tubs should be scrubbed, rinsed and drained, sprayed with an EPA registered disinfectant, allow to sit for 10 minutes, rinsed with water, and air-dried.[37] Traditional pools, Jacuzzi spas, and exercise pools (Hydroworx or Swimex) use chlorine or ultrasonics to filter and disinfect the pools. Sports physical therapists should be cautious of an athlete with open skin wounds who uses pools. Turbulence can cause skin debridement and subsequent use of towels can produce a source of cross-contamination.

Figure 3. *Disposable adhesive mesh strip applied to the posterior elbow*

Secondary to the potent virulence and potential adverse sequelae, CA-MRSA is reportable in many states. Outbreaks in confined environments should be closely monitored, but ethical concerns and issues exist when reporting communicable diseases. Individuals and teams may fear the negative stigma associated with a communicable disease.[14] Also, the consequences resulting from restricted participation may have implications on win/loss records, and current and future financial incentives (scholarships and contracts) for participation in athletic events. Ultimately, the sports medicine team should determine the time for return to participation that ensures safety of the athlete with CA-MRSA, teammates, and other competitors.

CONCLUSIONS

Research regarding CA-MRSA in athletic environments is relatively limited and retrospective designs are typically used to describe prevalence rates and treatment strategies. Due to low incidence rates, systematic tracking using large database programs such as the NCAA Injury Surveillance System (ISS) may help determine treatment and prevention effectiveness. Community associated-MRSA is increasingly prevalent in athletic settings. The sports physical therapists must be aware of risk factors, differential diagnosis, optimal timing for referral, and treatment strategies, while working closely with certified athletic trainers and team physicians. Due to rapid transmission and adverse sequelae of infection (hospitalization, surgery, death), cases of CA-MRSA should not be minimized. Ultimately, prevention is the greatest defense and all members of the sports medicine staff should utilize universal precautions when treating athletes, maintain clean environments within commonly shared areas, encourage proper hygiene practices, and discourage sharing of personal items such as razors, soap, and towels.

REFERENCES

1. Bartlett PC, Martin RJ, Cahill BR. Furunculosis in a high school football team. *Am J Sports Med.* 1982;10:371-374.
2. Sosin DM, Gunn RA, Ford WL, Skaggs JW. An outbreak of furunculosis among high school athletes. *Am J Sports Med.* 1989;17:828-832.
3. Kluytmans J, van Belkum A, Verbrugh H. Nasal carriage of Staphylococcus aureus: Epidemiology, underlying mechanisms, and associated risks. *Clin Microbiol Rev.* 1997;10:505-520.
4. Lowy FD. Staphylococcus aureus infections. *N Engl J Med.* 1998;339:520-532.
5. Naimi TS, LeDell KH, Como-Sabetti K, et al. Comparison of community- and health care-associated methicillin-resistant Staphylococcus aureus infection. *JAMA.* 2003;290:2976-2984.
6. Herold BC, Immergluck LC, Maranan MC, et al. Community-acquired methicillin-resistant Staphylococcus aureus in children with no identified predisposing risk. *JAMA.* 1998;279:593-598.
7. Lindenmayer JM, Schoenfeld S, O'Grady R, Carney JK. Methicillin-resistant Staphylococcus aureus in a high school wrestling team and the surrounding community. *Arch Intern Med.* 1998;158:895-899.
8. Stacey A, Endersby K, Chan P, Marples R. An outbreak of methicillin-resistant Staphylococcus aureus infection in a rugby football team. *Br J Sports Med.* 1998;32:153-154.
9. Naimi TS LK, Boxrud DJ, Groom AV, et al. Epidemiology and clonality of community-acquired methicillin-resistant Staphylococcus aureus in Minnesota, 1996-1998. *Clin Infect Dis.* 2001;33:990-996.
10. Weber JT. Community-associated methicillin-resistant Staphylococcus aureus. *Clin Infect Dis.* 2005;41:S269-S272.
11. Chambers HF. The changing epidemiology of Staphylococcus aureus? *Emerg Infect Dis.* 2001;7:178-182.
12. Kazakova SV, Hageman JC, Matava M, et al. A clone of methicillin-resistant Staphylococcus aureus among professional football players. *N Engl J Med.* 2005; 352:468-475.
13. Begier EM, Frenette K, Barrett NL, et al. A high-morbidity outbreak of methicillin-resistant Staphylococcus aureus among players on a college football team, facilitated by cosmetic body shaving and turf burns. *Clin Infect Dis.* 2004;39:1446-1453.
14. Nguyen DM, Mascola L, Bancroft E. Recurring methicillin-resistant Staphylococcus aureus infections in a football team. *Emerg Infect Dis.* 2005;11:526-532.
15. Centers for Disease Control and Prevention. Outbreaks of community-associated methicillin-resistant Staphylococcus aureus skin infections- Los Angeles County, California, 2002-2003. *Morb Mortal Wkly Rep.* 2003;51:88.
16. Kimmel C. When MRSA strikes. *Training & Conditioning.* 2006;16:13-19.
17. Romano R, Lu D, Holtom P. Outbreak of community-acquired methicillin-resistant Staphylococcus aureus skin infections among a collegiate fooball team. *J Ath Train.* 2006;41:141-145.
18. Noe E, Puls A, Bishop D, Hamstra KL. Skin infection within eight collegiate football players. *J Ath Train.* 2004;39:S-86.
19. Rihn JA, Posfay-Barbe K, Harner CD, et al. Community-acquired methicillin-resistant Staphylococcus aureus outbreak in a local high school football team unsuccessful interventions. *Pediatr Infect Dis J.* 2005;24:841-843.
20. Centers for Disease Control and Prevention. Methicillin-resistant Staphylococcus aureus infections among competitive sports participants- Colorado, Indiana, Pennsylvania, and Los Angeles County, 2000-2003. *Morb Mortal Wkly Rep.* 2003;52:793-795.
21. Cohen PR. Community-acquired methicillin-resistant Staphylococcus aureus: Skin infection presenting as an axillary abscess with cellulitis in a college athlete. *Skinmed.* 2005;4:115-118.
22. Cohen PR. Cutaneous community-acquired methicillin-resistant Staphylococcus aureus infection in participants of athletic activities. *South Med J.* 2005;98:596-602.
23. Huijsdens XW, van Lier AM, van Kregten E, et al. Methicillin-resistant Staphylococcus aureus in Dutch soccer team. *Emerg Infect Dis.* 2006;12:1584-1586.
24. Salgado CD, Farr BM, Calfee DP. Community-acquired methicillin-resistant Staphylococcus aureus: A meta-analysis of prevalence and risk factors. *Clin Infect Dis.* 2003;15:131-139.
25. Mainous AG, III, Hueston WJ, Everett CJ, Diaz VA. Nasal carriage of Staphylococcus aureus and methicillin-resistant S aureus in the United States, 2001-2002. *Ann Fam Med.* 2006;4:132-137.
26. Aly R, Maibach H, Strauss W, Shinefield H. Effects of systemic antibiotic on nasal bacterial etiology in man. *Appl Microbiol.* 1970;20:240-244.
27. National Collegiate Athletic Association. Wrestling Rules Committee. NCAA Wrestling Rules. Indianapolis, IN: National Collegiate Athletic Association; 2003.
28. Iyer S, Jones D. Community-acquired methicillin-resistant Staphylococcus aureus skin infection: A retrospective analysis of clinical presentation and treatment of a local outbreak. *J Am Acad Dermatol.* 2004;50:854-858.
29. Dominguez TJ. It's not a spider bite, it's community-acquired methicillin-resistant Staphylococcus aureus. *J Am Board Fam Pract.* 2004;17:220-226.
30. Cohen PR, Kurzrock R. Community-acquired methicillin-resistant Staphylococcus aureus skin infection: An emerging clinical problem. *J Am Acad Dermatol.* 2004;50:277-280.

31. Rihn JA, Michaels MG, Harner CD. Community-acquired methicillin-resistant Staphylococcus aureus: An emerging problem in the athletic population. *Am J Sports Med.* 2005;33:1924-1929.
32. Centers for Disease Control and Prevention. Guidelines for environmental infection control in health-care facilities: Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HIC PAC). *Morb Mortal Wkly Rep.* 2003;52:1-48.
33. National Athletic Trainers' Association. Official statement from the National Athletic Trainers' Association on Community-Acquired MRSA Infections (CA-MRSA). Dallas, TX: National Athletic Trainers' Association; 2005.
34. Draghi DC, Sheehan DF, Hogan P, Sahm DF. Current antimicrobial resistance profiles among methicillin-resistant Staphylococcus aureus encountered in the outpatient setting. *Diagn Microbiol Infect Dis.* 2006; 55:129-133.
35. Bratcher D. Methicillin-resistant Staphylococcus aureus in the community. *Pediatr Infect Dis J.* 2001;20:1167-1168.
36. Maltezou HC, Giamarellou H. Community-acquired methicillin-resistant Staphylococcus aureus infections. *Int J Antimicrob Agents.* 2006;27:87-96.
37. Sehulster L, Chinn RY, CDC, HICPAC. Guidelines for environmental infection control in healthcare facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). *MMWR Recomm Rep.* 2003;52:1-42.

ORIGINAL RESEARCH

CAN SERIOUS INJURY IN PROFESSIONAL FOOTBALL BE PREDICTED BY A PRESEASON FUNCTIONAL MOVEMENT SCREEN?

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ABSTRACT

Background. Little data exists regarding injury risk factors for professional football players. Athletes with poor dynamic balance or asymmetrical strength and flexibility (i.e. poor fundamental movement patterns) are more likely to be injured. The patterns of the Functional Movement Screen™ (FMS) place the athlete in positions where range of motion, stabilization, and balance deficits may be exposed.

Objectives. To determine the relationship between professional football players' score on the FMS™ and the likelihood of serious injury.

Methods. FMS™ scores obtained prior to the start of the season and serious injury (membership on the injured reserve for at least 3 weeks) data were complied for one team (n = 46). Utilizing a receiver-operator characteristic curve the FMS™ score was used to predict injury.

Results. A score of 14 or less on the FMS™ was positive to predict serious injury with specificity of 0.91 and sensitivity of 0.54. The odds ratio was 11.67, positive likelihood ratio was 5.92, and negative likelihood ratio was 0.51.

Discussion and Consclusion. The results of this study suggest fundamental movement (as measured by the FMS™) is an identifiable risk factor for injury in professional football players. The findings of this study suggest professional football players with dysfunctional fundamental movement patterns as measured by the FMS™ are more likely to suffer an injury than those scoring higher on the FMS™.

Key words: Functional Movement Screen, injury prediction

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INTRODUCTION

Participation in football is one of the leading causes of sport related injury with over 500,000 injuries occurring per year in high school and collegiate football.[1] To date, the injury rate for professional football has not been reported in the literature; but, the injury rate for high school and collegiate football ranges from 1.3 to 26.4 per 1000 athletic exposures (18.4-51.7 injuries per 100 players).[1-5] Although limited published reports exist on injury risk factors for professional football players,[6] researchers have prospectively identified risk factors for injury in high school and collegiate levels of competitive football. These risk factors include previous injury,[2,7] body mass index,[7-9] body composition (percent body fat),[9] playing experience,[2] femoral intercondylar notch width,[10] cleat design,[11] playing surface,[6] muscle flexibility,[12] ligamentous laxity,[12] and foot biomechanics.[13,14] Although these risk factors have been examined individually, injury risk is likely multifactorial.[15-17] The dynamic interplay of risk factors during sport and their relationship to injury, needs additional investigation. Furthermore, evaluation of isolated risk factors does not take into consideration how the athlete performs the functional movement patterns required for sport.

Recently, researchers have utilized movement examinations that involve comprehensive movement patterns to predict injury.[18] Pilsky et al[18] hypothesized that tests assessing multiple domains of function (balance, strength, range of motion) simultaneously may improve the accuracy of identifying athletes at risk for injury through pre-participation assessment. The Functional Movement Screen (FMS™) is a comprehensive exam that assesses quality of fundamental movement patterns to identify an individual's limitations or asymmetries. A fundamental movement pattern is a basic movement utilized to simultaneously test range of motion, stability, and balance.[19,20] The exam requires muscle strength, flexibility, range of motion, coordination, balance, and proprioception in order to successfully complete seven fundamental movement patterns. The athlete is scored from zero to 3 on each of the seven movement patterns with a score of 3 considered normal. The scores from the seven movement patterns are summed and a composite score is obtained. The intra-rater reliability of the composite score (which was used in the analysis for this study) for the FMS™ is reported to have an ICC value of 0.98.[21] Additional information regarding the development and uses of the FMS™ was documented by Foran,[22] Cook,[23] and recently published journal articles.[19,20]

Mobility and stability extremes are explored in order to uncover asymmetries and limitations. The scoring system was designed to capture major limitations and right-left asymmetries related to functional movement. Additionally, clearing tests were added to assess if pain is present when the athlete completes full spinal flexion and extension and shoulder internal rotation/flexion.

The seven tests utilize a variety of basic positions and movements which are thought to provide the foundation for more complex athletic movements to be performed efficiently. The Appendix includes pictures of and detailed scoring criteria for each of the seven tests which compose the FMS™. The seven tests are: 1) the deep squat which assesses bilateral, symmetrical, and functional mobility of the hips, knees and ankles, 2) the hurdle step which examines the body's stride mechanics during the asymmetrical pattern of a stepping motion, 3) the in-line lunge which assesses hip and trunk mobility and stability, quadriceps flexibility, and ankle and knee stability, 4) shoulder mobility which assesses bilateral shoulder range of motion, scapular mobility, and thoracic spine extension, 5) the active straight leg raise which determines active hamstring and gastroc-soleus flexibility while maintaining a stable pelvis, 6) the trunk stability push-up which examines trunk stability while a symmetrical upper-extremity motion is performed, and 7) the rotary stability test which assesses multi-plane trunk stability while the upper and lower extremities are in combined motion.

The relationship between the FMS™ score and injury risk has not been previously reported. Therefore, the purpose of this study was to examine the relationship between professional football players' score on the FMS™ and the likelihood of a player suffering a serious injury over the course of one competitive season.

METHODS

The strength and conditioning specialist associated with the team studied had extensive experience (11 years) as a professional football strength and conditioning specialist and utilized the FMS™ as part of pre-season physical performance testing prior to the 2005 season. All players who attended training camp were tested on each of the seven tests of the FMS (as described in the Appendix) each year. The composite score for each player was then variable analyzed in this retrospective study.

In order to protect the identity of the subjects, only limited injury information and FMS™ data were available to the authors for analysis, which is why common demographic data routinely reported in most studies are not included. In addition, the authors agreed with the professional football team not to state the name of the team in any subsequent publications.

The sample included only those players who were on the active roster at the start of the competitive season (n = 46) and the surveillance time for the study was one full season (approximately 4.5 months). Membership on the injured reserve and time loss of 3 weeks was utilized as the injury definition. This operational definition of injury ensured the player was placed on the injured reserve due to a serious injury. The study was approved by the University of Evansville Institutional Review Board.

Data Analysis

To determine if a significant difference existed in composite FMS scores between those injured and those who were not injured, a dependent t-test was performed with significance set at the $p < 0.05$ level. To determine the cut-off score on the FMS™ that maximized specificity and sensitivity a receiver-operator characteristic (ROC) curve was created. In this context, the FMS™ can be thought of as a special test used to determine if a player is at risk for a serious injury. An ROC curve is a plot of the sensitivity (True +'s) versus 1-specificity (False +'s) of a screening test. Different points on the curve correspond to different cut-off points used to determine at what value a test is considered positive.[24] The test value (FMS™ score) which maximizes both True +'s and controls for False +'s is identified on the ROC curve as the point at the upper left portion of the curve. Once the cut-off score was identified, a 2 x2 contingency table was created dichotomizing those who suffered an injury and those who did not, and those above and below the cut-off score on the FMS™.

Simple odds ratios, likelihood ratios, sensitivity and specificity were then calculated. Post-test odds and post-test probability were calculated according to the formula provided, which allowed for the estimation of how much an individual's FMS™ score influenced the probability of suffering a serious injury.

At the start of the season, a probability (pretest probability) existed for suffering a serious injury. To determine how much the probability of serious injury increased when a player's score is below the cut-off score (magnitude of the shift from pre-test to post-test probability), the post-test probability was calculated utilizing a 3-step calculation process as described by Sackett et al.[25] The positive likelihood ratio (+LR) value is the value associated with the special test utilized. In this case, the special test is the FMS™ and is considered negative for a given subject when their score is above the cut-off score determined by the ROC curve. The FMS™ scale is considered positive if a given subject's score is equal to or below the cut-off score determined by the ROC curve. The calculation is as follows:

1. Convert the pre-test probability to odds:

$$\text{Pre-test odds} = \frac{\text{pre-test probability}}{1 - \text{pre-test probability}}$$

2. Multiply the odds by the appropriate +LR value:

$$\text{Pre-test odds X} + LR = \text{post-test odds}$$

3. Convert the post-test odds back to probability:

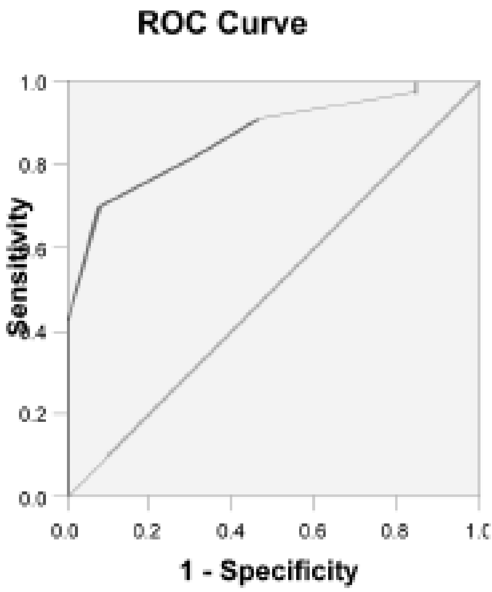
$$\frac{\text{post-test odds}}{\text{post-test odds} + 1} = \text{post-test probability}$$

Pre-test probability is synonymous with the prevalence of the disorder. In this case it would be the probability (at the start of the season) of a player suffering a serious injury as defined. In the absence of published data, an estimation of prevalence was made.[26] Since no injury rate data was available for professional football, a conservative prevalence of 15% was used based on previous high school and collegiate injury surveillance studies and expert opinion.[1-5]

RESULTS

The subjects were professional football players who made the final team roster before the start of a competitive sea-

Figure. Receiver-operator characteristic (ROC) curve for the FMS composite score and injury status.



Coordinates of the ROC Curve

Test Result Variable(s): FMS

FMS	Sensitivity	1 - Specificity
10.0000	1.000	1.000
12.0000	1.000	.846
13.5000	**.970**	**.846**
14.5000	**.909**	**.462**
15.5000	.818	.308
16.5000	.697	.077
17.5000	.424	.000
18.5000	.242	.000
20.0000	.000	.000

Coordinates of the ROC curve showing that the FMS composite score value which corresponds best with the upper left hand portion of the curve is between 13.5 and 14.5 justifying the cut-point determination of 14.

son. The mean (SD) FMS™ score (highest possible score is 21) for all subjects was 16.9 (3.0). The mean score for those who suffered an injury was 14.3 (2.3) and 17.4 (3.1) for those who were not injured. A t-test revealed a significant difference between the mean scores of those injured and those who were not injured (df = 44; t = 5.62; $p < 0.05$).

Upon analysis of the ROC curve (Figure) and corresponding table of sensitivity and specificity values, it was determined that an FMS™ score of 14 maximized specificity and sensitivity of the test. Specifically, the point is chosen so that the test correctly identifies the greatest number of subjects at risk (true positives) while minimizing incorrectly identifying subjects not at risk (false positives). On a ROC curve, this point is usually at the left uppermost point of the graph[27] (the point where the curve turns). Using this value, subjects were dichotomized into groups with a score of 14 as well as by injury status (Table). This cut-off score represents a sensitivity of 0.54 (CI95 = 0.34-0.68) and specificity of 0.91 (CI95 = 0.83-0.96). The odds ratio was 11.67 (CI95 = 2.47-54.52), positive likelihood ratio 5.92 (CI95 = 1.97-18.37), and negative likelihood ratio 0.51 (CI95 = 0.34-0.79).

The odds ratio of 11.67 can be interpreted as a player having an eleven-fold increased chance of injury when their FMS™ score is 14 or less when compared to a player whose score was greater than 14 at the start of the season. The post-test probability was calculated to be = 0.51. That is to say, if an athlete's score on the FMS™ was 14 or less, their probability of suffering a serious injury increased from 15% (pre-test probability of 0.15) to 51% (post-test probability of 0.51; CI95 = 0.25-0.76).

Table. *2x2 contingency table indicating if an athlete's FMS score was above or below the cut-off point and if they had suffered serious injury.*

FMS score ≤ 14?	Serious Injury? YES	NO
YES	7	3
NO	6	30

DISCUSSION

Sports physical therapists, athletic trainers, and strength and conditioning specialists using the FMS™ in professional football have casually observed that players with lower scores were more likely to be injured. Basic statistical procedures were used to test this observation. Those players with a score of less than 14 were found to have a substantially greater chance of membership on injured reserve over the course of one competitive season than those scoring greater than14.

To estimate the value of the FMS™ as a diagnostic test to predict the likelihood of injury, the purpose of the tests such as the FMS™ was considered. It was important to maximize the test's ability to rule in the potential disorder (injury), or in other words, to maximize the test's specificity. Higher specificity increases the ability to use the test to recognize when the disorder is present. That is, a highly specific test has relatively few false positive results and speaks to the value of a positive test.[28] The reverse is true when a given diagnostic test has high sensitivity. Because the FMS™ in this study was shown to be highly specific (0.91) for suffering a serious injury, the test can be used to rule in the condition studied. The sensitivity was 0.54, so the test offers limited capability to rule out the condition.

To consider how this information can be applied to an individual athlete, the shift from pre to post-test probability was calculated. Accurate estimation of the prevalence (pre-test probability) of a given disorder when attempting to determine the magnitude of the shift from pre to post-test probability when using the positive likelihood ratio of a special test is critical. If too high of a value is used, it will artificially inflate the magnitude of the shift and imply the special test (in this case the composite FMS™ score) is more powerful than it really is. A conservative prevalence rate (15%) was used to control for this potential error. In the absence of published data, professional football injury rates were discussed with professional football sports medicine personnel, who indicated that 15% was on the low end of what they would expect over the course of one competitive season.

The findings of this report suggest that athletes with dysfunctional fundamental movement patterns (as measured by lower scores on the FMS™) are more likely to suffer a time-loss injury, but can not be used to establish a cause-effect relationship. Some additional limitations of this study should be noted. Because this review only considered data from one team, selection bias is a limitation. Furthermore, the same data set that was used to determine the ROC curve cut-off score was used to test the cut-off score in the prediction model. Using the same data to determine cut-off score and evaluate those cut-off scores as predictive is more likely to demonstrate meaningful findings than when using cut-off scores determined with different data. Ideally, a cut-off score should be established from a separate prospective study, and then that value is applied to the prediction model to prevent inflation of the post-test probability and odds ratio.

Another limitation of the study was that only those on injured reserve for at least 3 weeks were used as the definition of an injury. These criteria may not have captured injuries that were meaningful, but were not of long enough duration to place the athlete on the injured reserve.

Future research should be conducted in a prospective manner that includes detailed injury surveillance and a more robust injury definition. Having access to data on multiple variables (such as previous injury) not available for this study would allow researchers to build a regression equation that predicts those who will suffer a time loss injury. Based on this retrospective analysis, the authors suggest including the FMS™ score in the model by using the individual test scores in addition to the composite score. With this detailed information, it may be possible to specifically identify factors (previous injury, deep squat score, lunge score) that contribute most to injury risk and then focus injury prevention efforts on modifiable factors such as dysfunctional movement.

CONCLUSION

Fundamental movement patterns such as those assessed by the FMS™ can be easily tested clinically. This retrospective descriptive study demonstrated that professional football players with a lower composite score (<14) on the FMS™ had a greater chance of suffering a serious injury over the course of one season.

REFERENCES

1. Shankar PR, Fields SK, Collins CL, et al. Epidemiology of high school and collegiate football injuries in the United States, 2005-2006. *Am J Sports Med.* 2007; 35;1-9
2. Turbeville S, Cowan L, Owen W. Risk factors for injury in high school football players. *Am J Sports Med.* 2003; 31:974-980.
3. Powell J, Barber-Foss K. Injury patterns in selected high school sports: A review of the 1995-1997 seasons. *J Athl Train.* 1999;34:277-284.
4. DeLee JC, Farney WC. Incidence of injury in Texas high school football. *Am J Sports Med.* 1992;20:575-580.
5. Prager BI, Fitton WL, Cahill BR, Olson GH. High school football injuries: A prospective study and pitfalls of data collection. *Am J Sports Med.* 1989;17:681-685.
6. Powell J. Incidence of injury associated with playing surfaces in the national football league. *J Athl Train.* 1987;22:202-206.
7. Tyler TF, McHugh MP, Mirabella MR, et al. Risk factors for noncontact ankle sprains in high school football players: the role of previous ankle sprains and body mass index. *Am J Sports Med.* 2006;34:471-475.
8. Almeida S, Trone D, Leone D, et al. Gender differences in musculoskeletal injury rates: a function of symptom reporting? *Med Sci Sports Exerc.* 1999;31:1807-1812.
9. Gomez JE, Ross SK, Calmbach WL, et al. Body fatness and increased injury rates in high school football linemen. *Clin J Sport Med.* 1998;8:115-120.
10. LaPrade R, Burnett Q. Femoral intercondylar notch stenosis and correlation to anterior cruciate ligament injury. A prospective study. *Am J Sports Med.* 1994; 22:198-203.
11. Lambson RB, Barnhill BS, Higgins RW. Football cleat design and its effect on anterior cruciate ligament injuries. A three-year prospective study. *Am J Sports Med.* 1996; 24:155-159.
12. Krivickas L, Feinberg J. Lower extremity injuries in collegiate athletes: Relation between ligamentous laxity and lower extremity muscle tightness. *Arch Phys Med Rehabil.* 1996;77:1139-1143.
13. Glick JM, Gordon RB, Nishimoto D. The prevention and treatment of ankle injuries. *Am J Sports Med.* 1976; 4:136-141.
14. Dahle L, Mueller M, Delitto A, Diamond J. Visual assessment of foot type and relationship of foot type to lower extremity injury. *J Orthop Sports Phys Ther.* 1991; 14:70-74.
15. Bahr R, Krosshaug T. Understanding injury mechanisms: A key component of preventing injuries in sport. *Br J Sports Med.* 2005;39:324-329.
16. Meeuwisse WH. Assessing causation in sport injury: A multifactorial model. *Clin J Sport Med.* 1994;4:166-170.
17. Bahr R, Holme I. Risk factors for sports injuries-a methodological approach. *Br J Sports Med.* 2003;37:384-392.
18. Plisky P, Rauh M, Kaminski T, Underwood F. Star Excursion Balance Test as a predictor of lower extremity injury in high school basketball players. *J Orthop Sports Phys Ther.* (In Press).
19. Cook G, Burton L, Hogenboom B. The use of fundamental movements as an assessment of function - Part 1. *NAJSPT.* 2006;1:62-72.
20. Cook G, Burton L, Hogenboom B. Pre-participation screening: The use of fundamental movements as an assessment of function - Part 2. *NAJSPT.* 2006;1:132-139.
21. Anstee L, Docherty C, Gansneder B, Shultz S. Inter-tester and intra-tester reliability of the Functional Movement Screen Paper presented at: National Athletic Training Association National Convention, 2003; St. Louis, MO.
22. Foran B. High Performance Sports Conditioning. Champaign, IL: Human Kinetics; 2001.
23. Cook E. Athletic Body Balance. Champaign, IL: Human Kinetics; 2004.
24. Rosner B. Fundamentals of Biostatistics. 5th ed. Pacific Grove, CA: Brooks/Cole; 2000.
25. Sackett DL, Haynes RB, Guyatt GH, Tugwell P. Clinical Epidemiology; A Basic Science for Clinical Medicine. 2nd ed. Boston, Mass: Little, Brown and Company Inc; 1992.
26. Guyatt GH, Rennie D. Users' Guides to the Medical Literature; Essentials of Evidence-Based Clinical Practice. AMA Press; 2002.
27. Portney LG, Watkins MP. Foundation of Clinical Reserach; Applications to Practice. 2nd ed. Upper Saddle River, NJ: Prentice Hall; 2000.
28. Fritz JM, Wainner RS. Examining diagnostic tests: An evidence-based perspective. *Phys Ther.* 2001;81:1546-1564.

Appendix. *The scoring criteria and descriptions of the 7 tests of the FMS™.*

1. Deep Squat

The subject will assume the starting position by placing his/her feet shoulder width apart with the feet in line with the sagital plane. The dowel will be held overhead with the shoulders flexed and abducted and the elbows extended. The subject will squat down with the heels on the floor and head and chest facing forward. If a score of III is not accomplished, the subject will be asked to perform the test with a 2x6 board under their heels. If this allows for a completed squat a II is given. If the subject still cannot complete the movement a I is scored.

III

II

I

2. Hurdle Step

For the hurdle step the subject will align their feet together with the toes touching the base of the hurdle, which is then adjusted to the height of the subject's tibial tuberosity. The dowel will be positioned across the shoulders, just below the neck. The subject will be instructed to slowly step over the hurdle and touch their heel to the floor while the stance leg remains in extension. The moving leg is then returned to the starting position. A III is scored if one repetition is completed bilaterally, a II if the subject compensated in some way by twisting, leaning or moving the spine, and a I if loss of balance occurs or contact is made with the hurdle.

III

II

I

3. In-line lunge.

The length of the subject's tibia will be measured from the floor to the tibial tuberosity. The subject will then be instructed to place the end of his/her heel on the end of the 2x6 board. Using the tibia length a mark is made on the board from the end of the subject's toes. The dowel is held behind the back in contact with the head, thoracic spine, and sacrum. The hand that is opposite the front foot should grasp the dowel at the cervical spine and the other hand at the lumbar spine. The subject will then place the heel of the opposite foot at the measured mark on the board, and the back knee will be lowered enough to touch the board behind the heel of the front foot. A III is given for a successfully completed repetition, a II for compensation and a I for incompletion or loss of balance.

III

II

I

4. Shoulder Mobility

The subject's hand will first be measured from the distal wrist crease to the tip of the third digit. The subject will then be asked to make a fist with each hand. The subject will be instructed to assume a maximally adducted, extended and internally rotated position with one shoulder and a maximally abducted, flexed and externally rotated position with the other so that the fists are located on the back. The distance between the two fists on the back at the closest point will be measured. A III is given if the fists are within one hand length, a II if the fists are within 1 ½ hand lengths, and a I if the fists fall outside this length. At the end of this test a clearing exam is administered. The subject will place his/her hand on their opposite shoulder and attempt to point the elbow upward. If pain results from this movement using either shoulder a score of zero is given for the entire shoulder mobility test.

III

II

I

Clearing Exam

5. Active Straight Leg Raise

The starting position for the active straight-leg raise requires the subject to lie supine with the arms in an anatomical position and head flat on the floor. The 2x6 board is placed under the knees, and the anterior superior iliac spine (ASIS) and mid-point of the patella are identified. Using those two landmarks a mid point on the thigh is found. The dowel is placed on the ground perpendicular to this position. The subject will be instructed to lift the test leg with a dorsiflexed ankle and extended knee while keeping the opposite knee in contact with the board. If the malleolus of the raised leg is located past the dowel than a score of III is given. If the malleolus does not pass the dowel then the dowel is aligned along the medial malleolus of the test leg, perpendicular to the floor. If this point is between the thigh mid point and patella, a II is scored. If it is below the knee a I is received. The test should be performed bilaterally.

III

II

I

6. Trunk stability push-up

The subject will begin in a prone position with both feet together. The hands will be placed shoulder width apart with the thumbs at forehead height for males and chin height for females. With the knees fully extended and the feet dorsiflexed the subject should perform one push-up in this position with no lag in the lumbar spine. By completing the push-up a score of III is given. If the subject cannot perform the push-up the hands are lowered, with the thumbs aligning with the chin for males and the clavicles for females. If a push-up is successful in this position a score of II is given; if not, a I is scored. At the end of this test a clearing exam is given. The subject should perform a press-up in the push-up position. If there is pain associated with this motion a score of zero is given for the entire test.

III

II

I

Clearing Exam

ORIGINAL RESEARCH

REFERENCE VALUES FOR THE CLOSED KINETIC CHAIN UPPER EXTREMITY STABILITY TEST (CKCUEST) FOR COLLEGIATE BASEBALL PLAYERS

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ABSTRACT

Background. The Closed Kinetic Chain Upper Extremity Stability Test (CKCUEST) is a tool developed and used in the clinic to evaluate progress during upper extremity rehabilitation. A need exists for reference values of CKCUEST for use in a clinical setting.

Objectives. To calculate reference values for the CKCUEST that may assist clinicians in developing goals and objectives for male collegiate baseball players who are recovering from injuries to the upper extremity. To determine if differences exist in scores according to playing position.

Methods. The sample consisted of 77 collegiate, male baseball players between the ages of 18 and 22 who reported no recent history of injuries to the shoulder, elbow, or the hand-wrist complex. The CKCUEST was administered three times to the athletes and the number of touches when performing the CKCUEST during the 15-second test was measured and recorded. An average of the three tests was used for data analysis.

Results. No significant differences existed according to playing position. The data did not differ from the normal distribution; therefore, reference values were calculated and reported for use by clinicians in development of goals and objectives for this population.

Discussion and Conclusion. The CKCUEST appears to be a clinically useful test for upper extremity function.

Key Words: upper extremity, functional testing, closed-kinetic chain

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INTRODUCTION

In the college baseball population, injuries to the upper extremities are very common, as throwing and batting activities place an enormous amount of stress on the joints of the upper extremity.[1] Fifty-eight percent of all injuries in collegiate baseball involved the upper extremity and accounted for seventy-five percent of the total time lost from sport, longer than injuries to other parts of the body.[1] Pitchers sustain the majority of upper extremity injuries, as the intense, repetitive throwing that pitching requires places a greater amount of stress on the upper extremity compared to other positions.[1] Throwing a baseball produces rotational velocities greater than 6000 degrees per second; and, at the point of release, distraction forces at the glenohumeral joint can be one to one and a half times the athlete's body weight.[2] Because throwing places so much stress on the upper extremity, the athlete must have adequate strength, stability, and mobility in order to return to activity after injury. If the athlete returns to activity too soon, re-injury may occur rather easily.

A closed-kinetic chain activity is defined as an activity in which the terminal joint meets considerable external resistance which prohibits or restrains free motion; whereas, an open-kinetic chain activity is defined as an activity in which the terminal joint is free.[3] Most of the activities in baseball are open-kinetic chain movements. However, an increase in the use of closed-kinetic chain activities in clinical rehabilitation has occurred to help return the athlete to their sport. Closed-kinetic chain activities may help improve dynamic stability through joint approximation and co-contraction.[4] Compression from closed-kinetic chain activity also stimulates mechanoreceptors and helps improve proprioception.[4] These improvements may be important when determining if the patient is ready to return to activity.

A need exists to develop tests that provide objective data to help clinicians determine a patient's readiness to return to activity. These tests should be easy for clinicians to use and for patients to understand. The tests should also be cost efficient and require minimal space in the clinic.[4,5] The closed-kinetic chain upper extremity stability test (CKCUEST) is intended for these purposes. The starting position for performing the CKCUEST is a traditional push-up position. The subject maintains this position while touching with one hand the ground on their opposite side. The score on the test is the number of touches completed in 15 seconds.[4,5]

To be useful in the clinical setting, reference values for the CKCUEST are needed to assist the clinician in developing goals and objectives for their clients. The purpose of this study is to establish a set of reference values for the CKCUEST in the collegiate baseball population at a community college or NCAA Division III college level. A secondary purpose was to determine if there were differences in CKCUEST scores based on playing position. Once reference data is developed, clinicians may have a quick and easy method to objectively determine if their patient is progressing in their rehabilitation.

METHODS

Subjects

This study was determined to be safe for human subjects by the Institutional Review Board of Arizona School of Health Sciences, A. T. Still University – Mesa Campus. Informed consent was obtained from each subject prior to data collection. The initial sample consisted of 78 collegiate, male baseball players between the ages of 18 and 22 who reported no recent history of injuries to the shoulder, elbow, or the hand-wrist complex. Subjects were recruited from two community colleges in Arizona and one NCAA Division III college in California. Subjects were excluded if they did not meet the age range, they had surgery on either upper extremity within the last year, were not fully cleared by their team physician to participate in practice or competition, or were experiencing pain or fatigue in either upper extremity from recent activity.

Data Collection Procedures

Subjects completed a screening questionnaire to ensure that no recent surgery or injury existed to the shoulder, elbow, and hand-wrist complex. Each subject was then assigned a number for identification. The weight of each subject was measured (in pounds), converted to metric units, and recorded. The height was measured using a standard 10-foot tape measure (in inches), converted to metric units, and recorded. Each player's position was also recorded. Each subject was then given a brief explanation on how to perform the test.

Two strips of athletic tape with a width of 1.5 inches were placed parallel to each other 36 inches apart on a tile floor as measured with a standard tape measure. The starting position for the test is one hand on each piece of tape while assuming a pushup position (Figure). The subjects were instructed that from the starting position they were to use one hand to reach across their body and touch the piece of tape lying under the opposing hand. After touching the tape line the hand would be returned to the original starting position. The subject would perform the same movement with the other hand. Touches were counted as every time the hand reached across the subject's body and touched the tape. The total time for the trial was 15 seconds. Each subject performed a warm up trial and then three real trials of the test with a rest period of 45 seconds between trials. An average of the three trials was used for data analysis.

Figure: *Set-up and starting position of the CKCUEST.*

Table 1: *Descriptive statistics for all players (n = 7)*

	Age (yrs)	Height (m)	Weight (kg)	BMI*
Mean	19.03	1.83	83.5	24.92
SD	1.22	0.08	12.23	2.91
95% CI	0.27	0.02	2.73	0.65

* Body Mass Index

Data Analysis

Descriptive statistics including mean, standard deviation, 95% confidence interval (CI), kurtosis (with 95% CI) and skewness (with 95% CI) were calculated for the number of touches performed for the CKCUEST. In addition, a Kolmogorov-Smirnov Goodness-of-Fit Test was also performed to insure that the data fit the normal distribution. To ensure no differences in scores on the CKCUEST according to player position, a one way analysis of variance was performed to determine differences in the scores among four difference groups: pitchers, catchers, infielders, and outfielders. An alpha level of 0.05 was chosen as the level of significance.

Table 2: *Scores on CKCUEST according to playing position*

Position	n	Mean *	Standard Deviation
Pitcher	30	30.30	4.82
Catcher	9	30.41	3.52
Infielder	26	30.78	4.02
Outfielder	12	30.30	4.00
All Players	77	30.41	3.87

*The number of touches performed on the CKCUEST

RESULTS

Initially, 79 subjects participated in this study. Two subjects were excluded due to upper extremity pain or discomfort while participating in the actual test. The descriptive statistics for the sample can be found in Table 1.

Scores on the CKCUEST can be found in Table 2. As a result of the analysis of variance, no significant difference was found between the scores on the CKCUEST across position ($F = 0.045$; $df = 3,73$; $p = 0.99$). Therefore, the scores on the CKCUEST are not dependent on the position of the baseball player.

A 95% CI was used to test the null hypothesis that the data fit a normal distribution.[7] If zero is included in the range of the confidence interval, the null hypothesis cannot be rejected. Zero was included in the ranges for both skewness and kurtosis, and it can be concluded the data did not differ from a normal distribution. A Kolmogorov-Smirnov Goodness-of-Fit Test was also performed to insure that the data fit the normal distribution, which revealed the data fit a normal distribution ($p = 0.22$).

DISCUSSION

A proliferation of rehabilitation techniques has occurred for the upper extremity using closed-kinetic chain activities.[8] The use of closed-kinetic chain exercises are beneficial for the lower extremity; therefore, it is reasoned closed-kinetic chain exercise is probably beneficial for the upper extremity.[9] Closed-kinetic chain exercises may increase electromyographic activity, improved joint stability and proprioception, and utilize multiple joint involvement.[10] Closed-kinetic chain exercises may provide large resistance with low acceleration, greater compression forces, increased joint congruency, low shear forces, and enhanced dynamic stabilization.[11] Wilk et al[12] proposed closed-kinetic chain exercise for the upper extremity such as isometric press-ups and isometric weight bearing with weight shift for functional tests. Closed-kinetic chain exercise involving weight bearing and shifting during rehabilitation may enhance muscular co-contraction of the glenohumeral joint through joint compression and approximation.[12] These exercises parallel the demands when performing the CKCUEST.

Most common assessments of the upper extremity are performed in an open-kinetic chain fashion, which measure the patient's pathology and levels of strength, stability, proprioception, and range of motion. Yet, assessment of these variables only test part of the role of the upper extremity in its main function; to place the hand/wrist complex in a position to manipulate the environment. When comparing open- and closed-kinetic chain assessments, no relationships exist between the outcomes, suggesting that a complete and thorough assessment of the shoulder must include more simple open-kinetic chain assessments and more complex closed-kinetic chain assessments.[13-15]

Difficulty arises in trying to describe any closed-kinetic chain test that assesses the independent function of the shoulder complex or the elbow complex, as the function of these complexes are not independent from one another and requires coordination between scapular, glenohumeral, elbow, and forearm muscles.[16] This coordination may explain the need for assessment tools that attempt to measure the function of the wrist/hand complex, the elbow complex, and the shoulder complex simultaneously.

Some tests that have been introduced to assess upper extremity function using closed-kinetic chain activities include curl-ups or partial curl-ups, pull-ups, or push-ups tests.[13,14] However, Goldbeck and Davies[4] suggest that no commonly used tests exist in the literature to identify deficits in upper extremity closed-kinetic chain function, which was their justification for developing the CKCUEST.

Ellenbecker[19] reported reference values for the CKCUEST of 18.5 touches for males and 20.5 touches for females (females used a modified starting position). These numbers are drastically different from the results obtained in this study (30.41 touches for males; SD = 3.87). The number of touches recorded for males in Goldbeck and Davies[4,5] study was 27.8 (SD = 1.77). Using 95% confidence intervals to determine differences, a difference does exist in the data collected by Goldbeck and Davies[4,5] (27.09 – 28.51 touches) and the data from this study (29.55 – 34.28 touches).

A major issue with the CKCUEST is the validity of the test. The authors were unable to assess any studies which evaluated the sensitivity and specificity of the test with any pathological conditions at this time. Nor were the authors able to assess the agreement or relationship with any other test used to evaluate function of the upper extremity. Still, the CKCUEST is a published test in the literature and is probably being used in a multitude of settings. Therefore, more descriptive data is needed for clinicians who use the test, which was a major objective in conducting this study.

The current authors had several concerns with the CKCUEST. One possible problem with the CKCUEST is the test places high loads of force on the wrist, elbow, and shoulder. The starting push-up position is not a position that the general population performs regularly. Patients presenting with co-morbidities of the upper extremities may have difficulties performing the task. The body position when performing the test requires a substantial amount of trunk strength or stability and patients who have compromised trunk strength or impairments may not be good candidates for the CKCUEST. The older geriatric population may not be able to perform the test and patients who are susceptible to fracture may be at increased risk because of the force of impact. For an athletic population or a population of conditioned individuals, the test might be perceived as a good functional assessment.

CONCLUSION

Closed-kinetic chain exercise and testing have become popular as it assesses the upper extremity as a unit. The CKCUEST appears to be a clinically useful test for upper extremity function. Reference values have been developed for the CKCUEST for collegiate-level baseball players. No differences existed in scores by position, and the values found in this sample fit a normal distribution.

REFERENCES

1. McFarland EG, Wasik M. Epidemiology of collegiate baseball injuries. *Clin J Sport Med.* 1998;8:10-13.
2. Newsham KR, Keith CS, Saunders JE, Goffinett AS. Isokinetic profile of baseball pitchers' internal/external rotation. *Med Sci Sports Exerc.* 1998;20:1489-1495.
3. Steindler A. Kinesiology of the Human Body. Springfield, Ill.: Charles C. Thomas; 1955.
4. Goldbeck TG, Davies GJ. Test-retest reliability of the closed kinetic chain upper extremity stability test. *Journal of Sports Rehabilitation.* 2000;9:35-45.
5. Goldbeck TG, Davies GJ. Test-retest reliability of the closed kinetic chain upper extremity stability test: A clinical field test. *Phys Ther.* 1999;79:S79.
6. Portney L, Watkins M. Foundations for Clinical Research: Applications to Practice. 2nd ed. Upper Saddle River, NJ: Prentice Hall Health; 2000.
7. Tabachnick B. Using Multivariate Statistics. New York: Harper Collins Publishers, Inc; 1989.
8. Lephart SM, Henry TJ. The physiological basis for open and closed kinetic chain rehabilitation for the upper extremity. *Journal of Sport Rehabilitation.* 1996;5:71-87.
9. Ellenbecker TS, Cappel K. Clinical application of closed kinetic chain exercises in the upper extremities. Orthopaedic Physical Therapy Clinics of North America. 2000;9:231-245.
10. McGee C, Kersting E, Davies GJ, McLean KP. Standard rehabilitation vs. standard plus closed kinetic chain rehabilitation for patients with shoulder impingement: A rehabilitation outcomes study. *UW-L Journal of Undergraduate Research.* 1998;1:103-113.
11. Heiderscheit BC, Rucinski TJ. Biomechanical and physiologic basis of closed kinetic chain exercises in the upper extremities. Orthopaedic Physical Therapy Clinics of North America. 2000;9:209-218.
12. Hirashima M, Kadota H, Sakurai S, et al. Sequential muscle activity and its functional role in the upper extremity and trunk during overarm throwing. *J Sports Sci.* 2002;20:301-310.
13. The President's Challenge. (homepage on the Internet) The Physical Fitness Test. Bloomington, Indiana: The President's Challenge. (cited July 17, 2006) Available from: http://www.presidentschallenge.org/
14. Tritschler, K. Barrow and McGee's Practical Measurement and Assessment. 5th ed. Philadelphia: Lippincott, Williams, & Wilkins; 2000.
15. Wilk KE, Arrigo CA, Andrews JR. Closed and open kinetic chain exercise for the upper extremity. *Journal of Sport Rehabilitation.* 1996;5:88-102.
16. Ellenbecker TS, Mattalino AJ. Glenohumeral joint range of motion and rotator cuff strength following arthroscopic anterior stabilization with thermal capsulorraphy. *J Orthop Sports Phys Ther.* 1999;29:160-167.
17. Schulte-Edelmann JA, Davies GJ, Kernozek TW, Gerberding ED. The effects of plyometric training of the posterior shoulder and elbow. *J Strength Cond Res.* 2005;19:129-134.
18. Ellenbecker TS. Clinical Examination of the Shoulder. 1st ed. St. Louis: Saunders; 2004.
19. Ellenbecker TS, Manske R, Davies GJ. Closed kinetic chain testing techniques of the upper extremities. Orthopaedic Physical Therapy Clinics of North America. 2000;9:219-229.

ORIGINAL RESEARCH

HIP STRENGTH AND KNEE PAIN IN FEMALES

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ABSTRACT

Background. Poor alignment between the patella, tibia, and femur has been identified as a primary cause of anterior knee pain. More recently, impaired hip strength has been discussed as a possible reason for the onset of knee pain.

Objectives. The purpose of this study was to determine if individuals with knee pain had weakness in the hip muscles.

Methods. Nineteen females between the ages of 18 and 40, experiencing unilateral knee pain for no greater than four weeks, were examined. Bilateral gluteus maximus and medius strength were measured with a MicroFET hand-held dynamometer.

Results. Strength of the gluteus medius and maximus muscles were significantly less in the extremities of patients experiencing knee pain than the extremity without knee pain.

Discussion and Conclusion. Given biomechanical relationships between the hip and knee, examining the entire lower kinetic chain should occur when evaluating patients with knee pain. Using impairment-based interventions, such as addressing hip strength in addition to knee pain, may enhance intervention effectiveness. Results of this study provide data that suggest that individuals with knee pain had weak hip muscles.

Key Words: knee pain, hip strength

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INTRODUCTION

According to the American Academy of Orthopaedic Surgeons,[1] knee pain is a common condition resulting in 19.4 million pain-related visits to a physician's office each year. This number is significantly higher than low back pain (5.9 million visits), hip pain (3.2 million visits), and ankle pain (1.9 million visits). Knee pain impairs function, which can lead to disability.[2]

Historically, causes of knee pain have been related to a structural deformity or malalignment of the patella and its relationship to surrounding structures. Patella and distal femur malalignment have been linked to anterior knee pain.[3] Interventions for such impairments have been to use either surgical or non-surgical strategies to correct the deformity or re-align the patella.[4] More recent information has indicated that improper alignment at the knee may originate proximally and that poor force production at the hip, secondary to muscle weakness, could be a factor that ultimately causes stress to the knee.[5,6] Juhn[7] and Powers[8] hypothesized that knee pain could be caused by a maltracking of the patella on the femur. They proposed that instability of the patella can result from unequal activity in the components of the quadriceps femoris muscle[8] or diminished hip extensor, rotator, or abduction strength.[5] Repetitive contact between the patella and femur, along with maltracking due to muscular imbalance in the hip, can result in anterior knee pain.[7]

It is important to understand the relationship between the different forces acting on the femur when considering the biomechanics of the lower extremity. A number of forces are exerted on the femur during ambulation, and the muscles acting on the femur play an important role in balancing these forces.[9] Duda et al[9] suggest an important relationship between the gluteal muscles and femoral neck loading. Femoral neck loading occurs when the muscle contraction exerts a force on the femur. When femoral loading occurs during weight bearing, the proximal and distal ends of the femur are subject to considerable shear forces. Previous research has suggested that hip strength may be a factor in anterior knee pain, therefore, the purpose of this study was to determine if individuals with knee pain had weakness in the hip muscles.

METHODS

Subjects

A sample of convenience of 24 females between 18 and 40 years of age were recruited for this study. Fulkerson[10] stated that females are more likely to be affected by anterior knee pain and Lephart et al[11] demonstrated that biomechanical differences exist between men and women. Given the effects of gender on biomechanics, females were chosen for this study to decrease the impact of a potentially confounding variable.

According to Berk,[12] bone growth is complete in most girls by age 16, when the epiphyses close completely. Torry and McCaw[13] reported "approximately ninety percent of all persons over the age of 40 will show pathological evidence of osteoarthritis in weight bearing joints." Based on these findings, females within the range of 18 to 40 years of age were recruited in order to minimize the chance of results being skewed by pubertal changes before the age of 18 or the onset of osteoarthritis after the age of 40.

The inclusion criteria were females with unilateral anterior knee pain (intermittent or constant) for no greater than four weeks. Exclusion criteria included (1) back, hip, or foot pain, (2) bilateral knee pain, (3) prior surgery or trauma to the back, hip, knee, or foot, and (4) medical conditions such as pregnancy, cancer, neurological impairment, bone disease, or stomach ulcer. These exclusion criteria were chosen to ensure subject safety and enable proper positioning.

The Utica College Institutional Review Board approved this research proposal. Upon approval, subjects were recruited using general announcements. All subjects signed an informed consent form prior to participation in this study.

Instrumentation

The International Knee Documentation Committee (IKDC) Subjective Knee Form was used to measure participants' knee pain and function in order to further define the sample. This form is knee specific, rather than disease specific, and provides data relative to knee symptoms and function during activities of daily living, sports, and work activities.[14] Irrgang et al[14] studied a group of 533 patients with a variety of knee pathologies to determine the reliability and validity of the IKDC Subjective Knee Form. The results of the study revealed a test-retest reliability coefficient of 0.94 and an internal consistency coefficient alpha of 0.92.

The MicroFET hand-held dynamometer (Noggan Health Industry; South Draper, UT) was used to measure muscle strength on all subjects. A reliability study was performed to determine the testers with the highest intrarater reliability for the hand-held dynamometer in order to maximize reliability. The reliability study took place on a sample of female subjects in the Utica College Clark Athletic Center, Physical Therapy Clinical Laboratory. Intrarater reliability was determined by testing gluteus medius and gluteus maximus muscles bilaterally on five female subjects without knee pain, each measured three times. Data were analyzed using Pearson product-moment coefficient of correlation in order to decipher the two researchers with the greatest interrater reliability. Intrarater reliability for the tester chosen to perform all measurments was r = 0.87 and 0.93 for the gluteus medius and gluteus maximus, respectively, which is consistent with high reliability reported previously by Click et al (0.95).[15]

Procedures

Once recruited, and after the consent form was signed, participants completed an intake form for demographic and subjective information about their knee pain. After reviewing the intake forms, subjects that met inclusion criteria completed the IKDC Subjective Knee Form, answering questions regarding performance during activity. Participants did not need to carry out physical activity while completing this form.

Participants underwent bilateral gluteus medius and maximus muscle strength testing using the MicroFET hand-held dynamometer. Each researcher tested bilateral gluteus medius and maximus muscles using the positions outlined by Bohannon[16] for the gluteus medius and Kendall et al[17] for the gluteus maximus. Hip extension force *(Figure 1)* was measured in prone on the posterior aspect of the thigh, proximal to the knee, with the knee at a ninety-degree angle.[17] Hip abduction force *(Figure 2)* was measured in supine with the knee extended and the hip in neutral relative to extension, abduction, and rotation. Force was measured at the lateral surface of the thigh, just proximal to the lateral condyle of the femur.[16]

The unaffected extremity was measured first. For hip extension, stabilization was given at the lumbar spine and for hip abduction it occurred at the lateral aspect of the contralateral extremity. Subjects performed three trials of one repetition each, with five to seven second holds, for both the gluteus maximus *(Figure 1)* and medius *(Figure 2)*. Twenty to thirty seconds of rest were provided between each trial as recommended by Bohannon.[18] Time for muscle contraction and rest periods was measured with a stopwatch.

Figure 1. *Gluteus maximus muscle strength testing position*

Figure 2. *Gluteus medius muscle strength testing position*

Data Analysis

Descriptive data for the average of the three measurements of the gluteus maximus and medius muscles included means and standard deviations. One dependent t-test was used to compare the strength differences in the extremity with pain and the extremity with no pain for the gluteus maximus and one t-test for the gluteus medius muscle. In order to adjust for possible inflation of the 0.05 alpha level due to multiple t-tests, the significance was adjusted (0.05/2) to the accepted alpha level of $p < 0.025$

RESULTS

Of the 24 subjects volunteering for the study, 19 subjects satisfied the inclusion and exclusion criteria and completed the study (mean age = 24.0 years; SD = 4.8; range = 18-35 years). The mean IKDC Subjective Knee Form score was 66.12 (SD = 15.8; range = 39.09 to 89.66). Of the subjects tested, 52.6% had an affected left knee and 47.4% had an affected right knee. College students comprised 57.9% of the subjects tested.

The difference of 4.39% change (SD = 5.33) between the strength scores for the unaffected gluteus medius and affected gluteus medius was significant (df = 18; t = 3.49; $p < 0.025$). In addition, the difference of 1.9% change (SD = 1.26) between the strength scores for the unaffected gluteus maximus and affected gluteus maximus was also significant (df = 18; t = 6.47; $p < 0.025$).

DISCUSSION

Previous authors have studied the effect that muscles have on the function of the skeletal system. An[19] stated that the mechanical behavior of the skeletal system was directly related to the muscles that were acting on it. Duda et al[9] reported that other structures have to adapt if the muscles are not able to balance forces acting on the skeletal system. The present study suggested that persons with knee pain had weak hip muscles. Therefore, it is possible that weak musculature around the proximal end of the femur may alter the forces acting on the knee and lead to compensatory stress at the distal aspect of the femur. These findings were consistent with previous theories on the effects that muscles have on the skeletal system.[9, 19]

Previous authors have linked hip pathology to problems in other areas of the biomechanical chain, including the back, knee, and foot.[3,6,20-27] Results of these studies further support the need to evaluate the entire biomechanical chain when a problem occurs in one area of the chain, and the results of the present study support this theory, as well. As the results of this study suggested, a relationship appears to exist between knee pain and weak gluteal musculature and clinical interventions focused on restoring proximal weakness may be warranted.

The gluteus maximus is one of the muscles thought to play a role in controlling alignment of the lower extremity and is identified as an important muscle to consider when treating anterior knee pain. The gluteus maximus posteriorly rotates the pelvis and controls limb activities during rotational movements.[28] Weakness of the gluteus maximus could, therefore, alter the rotational forces on the femur during activities and possibly affect the knee joint.

Powers et al[29] provided evidence for a biomechanical theory that the entire lower extremity chain should be considered during patient management. Using kinematic magnetic resonance imaging these authors observed that, during weight bearing conditions, femoral internal rotation was associated with lateral patellar displacement. The authors found that during knee extension in the closed-kinetic chain a measurable femoral internal rotation occurred. These findings are consistent with the idea that forces in an area other than the site of pain may be associated with the problem.

Previous authors have supported the relationship between gluteal weakness and knee pain. Ireland et al[6] tested abduction and external rotation strength of 15 females, between the ages of 12 and 21, who had patellofemoral pain and 15 non-symptomatic subjects matched for age, gender, and body weight. Subjects with knee pain demonstrated less strength in hip abduction and external rotation. Although the research of these authors is valuable, limitations existed that should be identified. All subjects experienced pain for longer than three months, which presents a potential significant confounding variable because it is possible that gluteal weakness, from disuse or guarding, was a result of knee pain. The present study differed in that subject recruitment was limited to subjects who had an onset of pain for less than 28 days; therefore, reducing the potential confounding nature of this variable.

Tyler et al[30] suggested that subjects with anterior knee pain responded favorably to interventions that included hip abduction, adduction, flexion, and extension progressive resistive strengthening strategies. Similar to Ireland et al,[6] Tyler et al[30] included only subjects that had experienced anterior knee pain for four weeks or more.

As the evidence mounts for a relationship between hip weakness and knee pain; causality has not been established. The methodology of Ireland et al,[6] Tyler et al[30] and the present study do not allow for causality to be inferred. Implications for patient/client management, however, should be considered. Patients may benefit from these findings by receiving a comprehensive examination, as well as interventions for deficits along the entire lower extremity kinetic chain during treatment.

Limitations

A small age range hindered the study. Given that females with knee pain were obtained through a sample of convenience from the college community and the surrounding area, this study's age distribution was skewed. The majority of subjects were students in the age range of 18 to 24. For these reasons, study results cannot be considered to represent the general population.

A control group was not considered at the time of data collection. It was not within the scope of the study given limitations on study time and resources. The study could be enhanced by including a control group in future investigations.

Future Studies

Future study on this topic should involve a broader age range in order to illustrate a better representation of the general population with knee pain. A study comparing males and females could also be conducted to compare differences in gluteal strength and knee pain between the genders.

Results of our study suggested that perhaps intervention for weak hip musculature could be useful when treating patients with knee pain. These results, therefore, provide a seed of evidence that could lead to further study. Studying the effectiveness of hip strengthening to address deficits in a randomized controlled manner is a possible outgrowth of this work. Considering the relationship that hip strength has on the entire biomechanical chain, it may also prove beneficial to examine the most reliable means of strengthening the abductors and adductors of the hip.

CONCLUSION

The results of this study suggested that individuals with knee pain had weak hip muscles. Results may have significant clinical implications consistent with the foundational components of physical therapy practice. Physical therapist examination and intervention strategies that consider the entire lower extremity kinetic chain were supported by the study results.

REFERENCES

1. American Academy of Orthopaedic Surgeons. Patient Visits for Selected Conditions, 1998; (2003) Available at: http://orthoinfo.aaos.org/fact/thr_report.cfm?Thread_ID=88&topcategory=Knee.
2. Peat G, McCarney R, Croft P. Knee pain and osteoarthritis in older adults: A review of community burden and current use of primary health care. *Ann Rheum Dis.* 2001;60:91-102.
3. McNally EG. Imaging assessment of anterior knee pain and patellar maltracking. *Skeletal Radiol.* 2001;30:484-495.
4. Dye SF. Patellofemoral pain: A current perspective. *The Journal of Musculoskeletal Medicine.* 2001;18:440-449.
5. Powers CM, Flynn T. Research Forum. Presented at: Combined Sections Meeting of the American Physical Therapy Association; February 2003, Tampa.
6. Ireland M L, Willson JD, Ballantyne BT, et al. Hip strength in females with and without patellofemoral pain. *J Ortho Sports Phys Ther.* 2003;33:671-676.
7. Juhn MS. Patellofemoral pain syndrome: A review and guidelines for treatment. *Am Fam Physician.* 1999; 60:2012-2019.
8. Powers CM. Patellar kinematics, part 1: The influence of vastus muscle activity in subjects with and without patellofemoral pain. *Phys Ther.* 2000;80:956-965.
9. Duda GN, Schneider E, Chao EYS. Internal forces and moments in the femur during walking. *J Biomech.* 1997; 30:933-941.
10. Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. *Am J Sports Med.* 2002;30:447-456.
11. Lephart SM, Ferris CM, Riemann BL, et al. Gender differences in strength and lower extremity kinematics during landing. *Clin Orthop Relat Res.* 2002;401:162-169.
12. Berk LE. Infants, Children, and Adolescents,2nd ed. Needham Heights: Allyn & Bacon; 1996.
13. Torry MR, McCaw S. The bilateral asymmetry of the kinematics and kinetics of osteoarthritic gait. *Gait Posture.* 1996;4:167-208.
14. Irrgang JJ, Anderson AF, Boland AL, et al. Development and validation of the International Knee Documentation Committee Subjective Knee Form. *American J Sports Med.* 2001;29:600-613.

15. Click Fenter P, Bellew JW, Pitts T, Kay R. A comparison of 3 hand-held dynamometers used to measure hip abduction strength. *J Strength Cond Res.* 2003;17:531-535.

16. Bohannon RW. Muscle strength testing with hand-held dynamometers. In LR Amundsen (ed.), Muscle Strength Testing. New York: Churchill Livingstone Inc; 2005.

17. Kendall FP, McCreary EK, Provance PG. Muscles: Testing and Function (4th ed.). Philadelphia: Lippincott, Williams & Wilkins; 1993.

18. Bohannon RW. Test-retest reliability of hand-held dynamometry during a single session of strength assessment. *Phys Ther.* 1986;66:206-209.

19. An K. Muscle force and its role in joint dynamic stability. *Clin Orthop Relat Res.* 2002;403S:S37-S42.

20. Cookson L. Atypical knee pain: The biomechanical and neurological relationship between the pelvis, hip, and knee – A case report. *Clinical Chiropractic.* 2003;6:63-66.

21. Ellison JB, Rose SJ, Sahrmann SA. Patterns of hip rotation range of motion: A comparison between healthy subjects and patients with low back pain. *Phys Ther.* 1990; 70:537-541.

22. Emms NW, O'Connor M, Montgomery SC. Hip pathology can masquerade as knee pain in adults. *Age Ageing.* 2002;31:67-69.

23. Shuler JA, Hart AL, Malone T. Assessment of hip musculature strength in patients with plantar fasciitis. *Phys Ther.* 1999;79:S27.

24. Wilk KE, Davies GJ, Mangine RE, Malone TR. Patellofemoral disorders: A classification system and clinical guidelines for nonoperative rehabilitation. *J Orthop Sports Phys Ther.* 1998;28:307-322.

25. Fairbank JC, Pysant PB, Van Poortvliet JA. Influence of anthropometric factors and joint laxity in the incidence of adolescent back pain. *Spine.* 1984;9:461-464.

26. Mellin G. Correlations of hip mobility with degree of back pain and lumbar spinal mobility in chronic low-back pain patients. *Spine.* 1988;13:668-670.

27. Riegger-Krugh C, Keysor JJ. Skeletal malalignments of the lower quarter: Correlated and compensatory motions and postures. *J Orthop Sports Phys Ther.* 1996;23:164-169.

28. Fulkerson JP, Arendt EA. Anterior knee pain in females. *Clin Orthop Relat Res.* 2000;372:69-73.

29. Powers CM, Ward SR, Fredericson M, et al. Patellofemoral kinematics during weight-bearing and non-weight-bearing knee extension in persons with lateral subluxation of the patella: A preliminary study. *J Orthop Sports Phys Ther.* 2003;33:677-685.

30. Tyler TF, Nicholas SJ, Mullaney, MJ, McHugh MP. The role of hip muscle function in the treatment of patellofemoral pain syndrome. *Am J Sports Med.* 2006; 34:630-36.

EXERCISE RELATED LEG PAIN (ERLP): A REVIEW OF THE LITERATURE

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ABSTRACT

Exercise related leg pain (ERLP) is a regional pain syndrome described as pain between the knee and ankle which occurs with exercise. Indiscriminant use of terminology such as "shin splints" has resulted in ongoing confusion regarding the pathoanatomic entities associated with this pain syndrome. Each of the pathoanatomic entities – medial tibial stress syndrome, chronic exertional compartment syndrome, tibial and fibular stress fractures, tendinopathy, nerve entrapment, and vascular pathology – which manifest as ERLP are each described in terms of relevant anatomy, epidemiology, clinical presentation, associated pathomechanics, and intervention strategies. Evidence regarding risk factors for ERLP general and specific pathoanatomic entities are presented in the context of models of sports injury prevention.

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INTRODUCTION

Leg pain associated with exercise is a common experience among athletes. One of the earliest published descriptions of leg pain was by Hutchins in 1913.[1] He described "spike soreness" as soreness in the medial leg in runners. He stated, "Specifically, the area begins four and a quarter inches above the internal malleolus and extends about an inch along the tibial border."[1] Over time, the term "shin splints" came to be associated with leg pain. Some used the term with regard to medial leg pain,[2-4] while others have used the term more generically. Slocum[5] reported that shin splints "designates a symptom complex characterized by pain and discomfort in the lower part of the leg after repetitive overuse in walking and running."

In 1966, the American Medical Association (AMA) Committee of the Medical Aspect of Sports, Subcommittee on Classification of Sports Injuries published the Standard Nomenclature of Athletic Injuries. This book defined shin splints as "pain and discomfort in the leg from repetitive running on hard surface or forcible excessive use of the foot flexors; diagnosis should be limited to musculotendinous inflammations, excluding fatigue fracture or ischemic disorder."[6] Detmer[7] opined that the AMA definition of shin splints was too restrictive, and suggested that the term include "injuries which are not obvious muscle strains, tendinitis, stress fracture, or compartment problems." Recognizing the confusion in terminology regarding "shin splints," Batt[8] wrote a review of terminology associated with leg pain. He concluded that the term "shin splints" is a generic term which does not refer to any specific pathology, but rather, to the location of pain.

Other generic descriptions of pain syndromes exist based on the location of pain, including anterior knee pain, metatarsalgia, and low back pain. As the term "shin splints" is accompanied by a great deal of confusion as to the type and location of pain, it is reasonable to use a term that more accurately describes the location and association of the pain. Brukner[9] recommended avoiding use of the term "shin splints" and proposed the term "exercise related lower leg pain" to describe shin, calf, ankle, or foot pain associated with or aggravated by exercise. Reinking and Hayes[10] suggested the use of the term "exercise related leg pain" (ERLP) to describe pain between the knee and ankle which is associated with exercise. These authors recommended elimination of foot pain from this syndrome as the foot is anatomically distinct from the leg, and also suggested elimination of the term "lower" from the syndrome name as ERLP can and does occur along the length of the leg. This nomenclature confusion has delayed development of a good understanding of leg pain. The objective of this paper is to provide a literature review of ERLP including epidemiology, pathoanatomic entities, risk factors, and preventative strategies.

METHOD OF REVIEW

Searches of two electronic databases were conducted: the National Library of Medicine (MedLine) and the Cumulative Index for Nursing and Allied Health Literature (CINAHL) over the past 10 years during this ongoing research on exercise related leg pain. Searches have included terms such as shin splints, exercise related lower leg pain, exercise related leg pain, leg pain, lower leg pain, chronic leg pain, medial tibial stress syndrome, stress fracture, tibial stress fracture, fibular stress fracture, chronic exertional compartment syndrome, exertional compartment syndrome, exertional leg pain, tendinopathy, tendinitis, tendinosis, nerve entrapment, arterial occlusive disease, popliteal artery, tibial artery, injury prevention, and sports injury prevention. Searches have been limited to articles written in English using human subjects. Over 350 articles appeared relevant based on titles or abstracts and were screened for inclusion in this review. A total of 111 articles were selected for use in this paper including epidemiological studies, observational studies, randomized controlled trials, literature reviews, systematic reviews, meta-analyses, and case reports. As this is not a systematic review, the intent was not to critically evaluate the evidence but rather to provide a summary of the literature on ERLP to assist the sports physical therapists in better understanding the conditions that present as ERLP.

EPIDEMIOLOGY OF ERLP

While only a few studies have investigated ERLP epidemiology in athletes, the data consistently show it to be a commonly experienced pain syndrome. Running (cross-country and distance track events) has the most common occurrence of ERLP; other sports with reported occurrence of ERLP include soccer, volleyball, field hockey, basketball, gymnastics, and dance.[11] Orava and Puranen[12] reported an 18% incidence of ERLP over a five year period in a group of 2750 athletes presenting to a

Finnish sports clinic with overuse injuries. Researchers at a sports medicine clinic in British Columbia have published three epidemiological reports of overuse running injuries from 1981 to 2002. In those studies, the percentage of total running injuries attributed to ERLP were 13.2% of 1819 injuries,[13] 20.4% of 4173 injuries,[14] and, most recently, 12.8% of 2002 injuries.[15] In a study of 63 collegiate cross-country athletes, 52% reported a history of ERLP which interfered with running.[16] Sallis et al[17] reported the most common site of injury in collegiate cross-country runners was the leg. In 2005, the National Collegiate Athletic Association (NCAA) began to collect overuse injury data for men's and women's cross-country as a part of the NCAA Injury Surveillance System. The data indicated that in men's cross country, ankle sprains and ERLP were the most common practice and race injuries that kept participants out seven days or more. Exercise related leg pain was also one of the most common injuries for women's cross country that kept participants out seven days or more.[18] In a 15-year longitudinal study of high school cross country running injuries, Rauh et al[19] reported that the leg was the most common site of injury and re-injury.

PATHOANATOMIC ENTITIES

Exercise related leg pain includes the pathoanatomic entities of medial tibial stress syndrome (MTSS), chronic exertional compartment syndrome (CECS), tibial or fibular stress fractures, tendinopathies (posterior tibialis, anterior tibialis, peroneals, and Achilles), nerve entrapment syndromes, and vascular syndromes. Of these conditions, MTSS, stress fractures, CECS, and tendinopathies are the most common causes of ERLP.[11] Nerve entrapments and vascular syndromes are very rare entities in athletes.[20-22] A review of the pathoanatomy, clinical signs and symptoms and, as available, specific epidemiological data for six pathoanatomic entities follows.

Medial Tibial Stress Syndrome

Medial tibial stress syndrome presents as pain along the posteromedial border of the distal two-thirds of the tibia, usually most localized at the intersection of the distal and middle thirds.[23] The pain typically intensifies at the initiation of the exercise session, may subside during exercise, and resolves with rest. No neurological symptoms are associated with MTSS.[24] Mubarak et al[3] published the first descriptive paper on MTSS in 1982. These authors reported that Dr. Drez was the individual who coined the term "medial tibial stress syndrome" based on his clinical experience with the presentation of the condition. The investigators studied a group of 12 patients with posterior-medial leg pain and using compartmental pressure studies, ruled out chronic compartment syndrome. They identified the condition as most likely a periostitis along the posterior medial border of the tibia.

Traditionally, the tibialis posterior muscle was considered the anatomic source of medial leg pain associated with MTSS.[25] However, anatomic studies have challenged this hypothesis as the tibialis posterior tibial origin is lateral to the area of symptom identification.[4,26] These studies suggested the soleus muscle and its investing fascia, along with the flexor digitorum longus and the deep crural fascia, are responsible for traction-induced posterior-medial periosteal pain. Bouche and Johnson[27] used fresh frozen cadaver limbs to examine the pathomechanics of MTSS. They concluded that distal tibia fascial traction is generated by contraction of the superficial and deep posterior compartment muscles and this tension contributes to the development of MTSS.

Detmer[28] developed a classification system for MTSS with three types of lesions. Type I involves tibial stress fractures or stress reactions, Type II involves the fascial attachment of soleus to the tibia, and Type III is a chronic deep posterior compartment syndrome. However, this classification system is not recommended[24] as the system combines multiple diagnostic entities, perpetuating confusion regarding the tissue origin of the athlete's pain. In this paper, MTSS will be used to describe pain along the fascial insertion of the soleus muscle on the tibia.

Regarding the epidemiology of MTSS, the terminology confusion in the literature confounds precise identification of the incidence of this condition. An early study of MTSS in runners showed 239 runners of a group of 1650 (14.5%) developed medial tibial pain, but this work was done before clarity existed in the diagnostic parameters of MTSS.[13] James et al[25] reported 13% of 232 injuries to runners was "posterior tibial syndrome," a condition described similar to MTSS. Two studies of high school cross-country runners have shown an overall MTSS incidence of 12%[29] and 15%,[30] with higher incidence in females than males.

Although MTSS is a condition involving the interface of soft tissue and bone, evidence exists that MTSS is associated with decreased tibial bone density. In a study of leg pain in male soccer players, tibial bone density in the MTSS group was 15% lower than a group of non-athletic control subjects, and 23% lower than a group of athletic control subjects.[31] These data should not be misinterpreted that the decreased bone density caused MTSS; in fact, lower bone density may have been a result of chronic MTSS. However, this research is the first to raise the question of the relationship between bone health and MTSS. In a follow-up study, Magnusson et al[32] found that the low regional density of the distal tibial returned to normal following recovery from pain symptoms. The authors concluded that the decreased bone density develops in conjunction with MTSS, but whether a causal relationship exists is unknown.

Treatment for MTSS is largely based on anecdotal reports rather than evidence-based practice. Early management of MTSS pain often includes relative rest from the offending activity, cross-training, and the use of modalities such as icing and ultrasound. Other recommendations include ankle muscle strengthening, stretching, and a progressive return to running. Little, if any, evidence exists to support such recommendations.[23,24] A survey study of the effect of custom foot orthotics on MTSS suggested that most athletes who were prescribed foot orthotics reported that the orthotics helped their condition.[33] In recalcitrant cases, fasciotomy of the superficial and deep fascia of the posteromedial leg may be recommended, but the outcome of this surgery is not always successful in returning the athlete to sport.[34]

Chronic Exertional Compartment Syndrome

The second condition in the ERLP complex is CECS. This conditions was first described 1956 by Mavor[35] in a professional soccer player. The leg has five osseofascial compartments, each bounded by an inelastic layer of fascia.[36] These compartments – anterior, lateral, superficial posterior, deep posterior, and posterior tibialis – enclose the ankle and foot musculature. When the volume of these compartments increases, the compartmental pressure increases, potentially affecting the movement of blood, lymph, and nerve impulses through the compartment and inducing tissue ischemia. This increase in compartmental pressure is referred to as compartment syndrome, and can be caused by either macrotraumatic or microtraumatic events. In the case of a macrotraumatic injury to a limb, acute compartment syndrome may occur as bleeding into the compartment increases the compartmental volume and pressure. This condition is a medical emergency and must be dealt with promptly. Chronic exertional compartment syndrome, on the other hand, is a microtraumatic condition associated with overuse and results from increased muscle volume within a compartment during exercise.

The onset of CECS is usually distinct from MTSS as the athlete describes pain that does not begin at the initiation of exercise but rather begins at a predictable point after exercise initiation. The pain is characterized as "cramping" or "burning" and may or may not subside immediately after exercise. In addition, complaints of numbness and weakness in the lower leg and foot are frequent.[36] The classic diagnostic sign of CECS is elevated intra-compartmental pressure with exercise. The anterior compartment is the most common site of symptoms.[36,37]

The epidemiology of CECS is uncertain as much inconsistency exists in the populations studied with regard to age, sport, and sex. Some authors report CECS is most common among distance runners.[38,39] In their retrospective review of 2002 running injuries, Taunton et al[15] reported 28 cases (1.4%) of anterior compartment syndrome. The anterior compartment is described as the most commonly affected, followed by the lateral compartment.[36,39,40] In a review article, Blackman[36] reported that the preponderance of the literature indicates CECS commonly presents bilaterally with no difference in incidence of CECS between males and females. Styf[38] investigated the cause of ERLP in 98 athletes with purported CECS. He found that 27% of the patients had elevated compartmental pressures with exercise; the other patients had diagnoses including MTSS and peroneal nerve compression.

Little evidence exists to support conservative treatment for CECS. Blackman et al[41] reported that in a pilot study, a combination of massage and stretching intervention increased the amount of work performed prior to onset of symptoms, but no change in the intra-compartmental pressures occurred. Recalcitrant cases may require compartmental fasciotomy for symptom reduction.[36,42]

Stress Fracture

Bony overuse injury is a third category of ERLP. Bone is a dynamic tissue with a mineral component that is constantly being remodeled based on the imposed stresses. In normal bone, a balance exists between mineral deposition and mineral resorption and no net loss of bone mineral content. However, with excessive repetitive stress over time, bone mineral resorption can exceed deposition. This condition causes a net loss of bone mineral content resulting in a fatigue fracture.[43] Unlike the macrotraumatic acute bone fracture injury caused by a large imposed force in a short period of time, the fatigue fracture is a microtraumatic injury representing a maladaptation to smaller repetitive forces. This condition was first described as leg or foot pain in soldiers after marching, and hence, was initially named "march fractures."[44]

The onset of pain is usually gradual, and pain decreases with rest in early stages. As the stress fracture develops, the pain may persist after exercise and occur during daily activities.[45] Tibial stress fractures are more common than fibular, consistent with the fact that the tibia bears greater load in ambulation.[46] As the tibia is the more medial bone of the leg, the symptoms of stress fracture may present indistinguishable from MTSS; the athlete will typically complain of pain along medial leg. In order to make a definitive diagnosis, the gold standard imaging technique is the triple-phase bone scan.[45] On a bone scan, a stress fracture will present as intense uptake in one focal site of the bone, whereas MTSS presents as diffuse uptake along the medial tibial border.

Few studies have been done examining the incidence rates of stress fractures in athletes. Bennell and Brukner[47] stated, "Incidence rates, expressed in terms of exposure, have rarely been reported for stress fractures in athletes. Nevertheless, available data suggest that runners and ballet dancers are at relatively high risk for stress fractures." Johnson et al[48] tracked injuries for all athletes at a Division II institution over a two-year period. During this time, 34 stress fractures in 24 athletes were diagnosed in the 914 athletes. Of the 34 stress fractures, 13 were tibial, more than any other anatomic site. Of those 13 fractures, nine occurred in females and four in males. Goldberg and Pecora[49] studied three years of data on stress fracture occurrence in collegiate athletes at a large private university. They found an annual incidence of 1.9%, but 67% of the injuries were in freshmen. The authors suggested the injuries were a result of the significant increase in training volume between high school and college. The tibia was the second most common region of stress fracture in the study, following the metatarsals of the foot.

Female athletes are at greater risk for stress fractures than male athletes.[43,46,47,50-52] Several hypotheses have been forwarded to explain this increased risk. In a review article, Bennell et al[53] stated, "menstrual disturbances, caloric restriction, lower bone density, muscle weakness, and leg length differences are risk factors for stress fracture." However, these authors note that no evidence exists to support lower extremity alignment issues such as pelvic width and knee valgus as risk factors for stress fracture.

Shaffer and Uhl[54] recently completed a systematic review of the prevention and treatment of stress fractures in athletes. They reported that while no high level evidence supports any prevention strategies, limited evidence exists to support the use of shock absorbing insoles for stress fracture prevention. Likewise, another systematic review of the interventions for prevention and treatment of stress fractures found evidence that the use of shock absorbing inserts in footwear reduced the incidence of stress fractures in military personnel.[55] These authors also found limited evidence to support the use of a pneumatic brace during the rehabilitation period after a stress fracture.

Tendinopathy

The fourth diagnostic entity within ERLP is tendinopathies, or pathologic conditions affecting tendon tissue. Tendons are collagenaceous structures which link muscle to bone. In a normal tendon, the collagen fibrils are arranged in a parallel fashion. In a pathologic tendon, the collagen alignment is disorganized with abnormal intratendinous material. Khan et al[56] described the two primary tendinopathies as tendinitis and tendinosis. These authors used histologic evidence to support their contention that tendinitis is a rare condition and tendinosis is the more common tendon pathology. A key finding in much of the research on tendon pathology is the conspicuous absence of inflammatory cells in involved tendon tissue. In an essay on the use of language in medicine, Bernstein[57] pointed out that the words we use influence how we think. The pervasive and indiscriminant use of the term "tendinitis" to describe any tendon pathology causes health care practitioners and patients to continue to view tendon pathology as inflammatory, which is, in fact, rarely the case.

The pain pattern of an athlete with tendon pain depends on the chronicity of the condition. In an early stage, the athlete may have tendon pain only after exercise. As the condition progresses, the pain may become constant with all daily activities.[58] The pain is typically located along the course of the tendon, in some cases at the enthesis site and in other cases as the tendon passes around a bony prominence. Tendon pain is typically intensified with resisted testing of the involved muscle group.

As with the other ERLP conditions, little accurate epidemiological information is available regarding the tendon conditions. Several authors report tendon pathology is a "common" overuse injury,[59-63] but give no specific incidence of injury. In their study of injuries in runners, James et al[25] reported Achilles tendinitis was the third most common problem behind knee pain and posterior tibial pain, with 11% of the running injuries in this category. Using a population of 2002 injured runners, Taunton et al[15] identified Achilles tendinitis in 96 runners (4.8%), peroneal tendinitis in 13 runners (0.65%), and posterior tibialis tendinitis in 11 runners (0.55%). All tendon pathologies in this study were referred to as "tendinitis."

Interventions for tendon pain also are largely based on anecdotal reports and clinical lore. Such interventions include relative rest, cross-training, stretching, strengthening, ultrasound, iontophoresis, cryotherapy, counterforce bracing, foot orthotics, non steroidal anti-inflammatory medications, extracorporeal shock wave therapy, and surgery. At present, the only conservative intervention that is supported by evidence is the use of eccentric training.[64-70]

Nerve Entrapment

Nerve entrapment is a rare clinical condition in athletes, but one that the clinician must recognize from a thorough examination. The peripheral nerves involved in entrapment syndromes of the leg include the saphenous, common peroneal, superficial or deep peroneal, sural, and tibial nerves. The symptoms of these syndromes may include pain, paresthesia, motor weakness, and decreased coordination, depending on the specific nerve. For example, the saphenous nerve is a sensory nerve only, and entrapment may result in sensory loss or medial leg pain that may mimic vascular claudication type pain. Hirose and McGarvey[71] describe three stages of nerve entrapment: Stage 1 in which patients feel pain and occasional paresthesia, especially at night; Stage 2 in which the paresthesia is more constant throughout the day and muscle weakness may develop; and Stage 3 in which patients experience constant pain, and sensory and motor loss.

Nerve symptoms may occur secondary to CECS and compartmental pressure assessments are necessary to distinguish CECS from nerve entrapment. The etiology of nerve entrapment is not well understood, but is hypothesized to be a result of either blunt trauma or repetitive motion leading to scarring of the nerve sheath.[21] No epidemiological data pertaining to nerve entrapment in the leg were found in the literature. Detail regarding specific nerve entrapments is outside the scope of this paper; the reader is referred to excellent review papers on this topic for more information.[21,22,71]

Treatment of nerve entrapment includes such conservative measures as nerve flossing, padding, orthotic prescription, and corticosteroid injections. Recalcitrant cases may require a surgical decompression of the nerve entrapment.

Vascular Pathology

The last of the clinical entities within the umbrella of ERLP is vascular pathology. Entrapments of the popliteal artery or the anterior tibial artery have been reported in the literature but are extremely rare.[20,72] This condition presents with symptoms very similar to CECS, and diagnostic differentiation requires compartmental pressure testing. Distal pulses are diminished in this condition, and the athlete will describe a deep ache or "cramping" sensation.[20] If identified, this condition requires surgical intervention.

EXERCISE RELATED LEG PAIN RISK FACTORS

Van Mechelen et al[73] proposed a "sequence of prevention" model for sport injury which involved four steps: (a) identifying the extent of the injury problem, (b) understanding the etiology and mechanism of injury, (c) introducing appropriate preventative measures, and (d) assessing the effectiveness of those measures. In a systematic review on the prevention of exercise-related leg pain (ERLP) in athletes,[74] the authors concluded there is "little objective evidence to support the widespread use of any existing interventions to prevent shin splints." A primary reason for this paucity of evidence regarding interventions is that the risk factors for ERLP are not well understood. Multiple factors are hypothesized to contribute to the development of ERLP including intrinsic and extrinsic factors. Intrinsic factors are those contained within a person, including sex, race, bone structure, bone density, muscle length, muscle strength, joint range of motion, diet, and body composition. Extrinsic factors are those outside of a person, including training volume (frequency, duration, and intensity), types of conditioning activities, specific sport activities, training surface, shoes, and environmental conditions.

Extrinsic Risk Factors

Extrinsic factors, including training volume, training surfaces, shoes, and sport activities, are often cited as causes of lower extremity overuse injury. However, consensus evidence is generally lacking to support most of these factors. A preponderance of evidence suggests that excessive training mileage in runners is a risk factor for lower extremity overuse injury.[13,25,75-77] Macera et al[76] reported that the most important predictor of lower extremity overuse injury was running 40 or more miles per week. A study of overuse injuries in 1300 United States Marine Corps recruits indicated that a higher weekly training volume was associated with an increase in lower extremity overuse injuries.[78] However, although excessive training is commonly mentioned as a risk factor for ERLP, no supporting evidence exists specific to ERLP.

Sport type is another purported risk factor for ERLP. Ugalde and Batt[11] stated, "A high incidence of shin pain is associated with running and jumping sports," but offered no specific epidemiological information. In their prospective study of athletes with acute shin splints, Batt et al[79] found that most of the athletes (83%) were cross-country runners or track athletes. In a study of ERLP in collegiate female athletes, Reinking[80] found the highest incidence in cross-country and field hockey athletes.

Intrinsic Risk Factors

More research has focused on intrinsic risk factors associated with lower extremity overuse injuries. Several studies have reported a greater risk of lower extremity overuse injury in athletes with a pronatory foot type.[25,59,81-84] However, conflicting data exist suggesting excessive foot pronation is not an intrinsic risk factor for lower extremity overuse injury.[85-88] Three recent studies have not supported this relationship.[10,30,95] Potential confounding issues in the conflicting results regarding foot pronation and ERLP include the variation in the static and dynamic foot pronation measures, sample populations, and pathoanatomic entity included in the study.

Poor musculotendinous flexibility is another commonly cited intrinsic factor for generic lower extremity overuse injury.[61,96-98] Kaufman et al[59] reported that tight posterior calf muscles as measured by limited ankle dorsiflexion with the knee extended was a risk factor for lower extremity overuse injury, but close examination of his data show that this was only true for the development of Achilles tendon pain. In a review of the relationship between flexibility and sport injury, Gleim and McHugh[99] concluded "There is no scientifically based prescription for flexibility training and no conclusive statements can be made about the relationship of flexibility to athletic injury." This conclusion was supported by a second systematic review on the effect of stretching on sport injury.[100]

Neely[101] conducted a comprehensive review of the literature on intrinsic risk factors for ERLP using studies of both military and civilian populations. The review focused on intrinsic factors including age, somatotype, sex, past history of injury, and physical fitness. From the review, the author concluded that "overwhelming evidence" supports several intrinsic factors as increasing the risk of ERLP. Those factors are female sex, including age > 24 years, a high body mass index (BMI), low BMI in females, poor physical fitness as measured by a 1- or 2-mile timed run, and past history of injury. The BMI data indicated that for both men and women, a higher percentage of body fat resulted in a greater risk of injury. The authors suggested that higher body fat may be a consequence of excessive forces placed on tissues because of additional weight. However, women showed a bimodal relationship between BMI and injury; not only was more body fat a risk for injury but also less body fat than normal.

Intrinsic factors associated with bone-related ERLP (stress fractures) include sex, menstrual function, bone density, and foot type. It is well documented that stress fractures are more common in females than in males.[43,46,49-52,102,103] Barrow and Saha[104] found stress fractures to be more common in collegiate female runners with menstrual irregularity. These authors also found a much higher incidence of disordered eating in the women with menstrual abnormalities. These results were corroborated in a study of 113 track and field athletes, where the authors found not only menstrual dysfunction to be a risk factor for stress fractures in female athletes but also low bone density.[105] Based on anecdotal evidence, foot pronation is considered a risk factor for tibial stress fractures, while others have reported that excessive supination is a causal factor.[106,107] In a literature review on risk factors for stress fractures, Bennell et al[53] stated, "time-honoured risk factors such as lower extremity alignment have not been shown to be causative even though anecdotal evidence indicates they are likely to play an important role in stress fracture pathogenesis."

PREVENTION OF ERLP

As described earlier in this paper, van Mechelen[73] proposed a sport injury prevention model involving four steps: (1) identifying the extent of the injury problem, (2) understanding the etiology and mechanism of injury, (3) introducing appropriate preventative measures, and (4) assessing the effectiveness of those measures. The second step of his model, injury etiology and mechanism, was further developed by Meeuwisse[108] in which he described a multifactorial causation model involving intrinsic and extrinsic factors. In this model, intrinsic factors predisposed an athlete to injury, and subsequent exposure to extrinsic factors resulted in injury to susceptible athletes. Bahr and Krosshaug[109] focused on the complex interaction of intrinsic and extrinsic factors as an inciting event in sports injury etiology. Based on these previous models,[73,108,109] Meeuwisse et al[110] has proposed a new expanded model of sport injury etiology which takes "the cyclic nature of changing risk factors into account to create a dynamic, recursive picture of etiology." This model attempts to take into account both intrinsic and extrinsic risk factors as well as repeated exposures and changing circumstances that combine to cause injury. The current problem with ERLP is that very little is understood about the specific intrinsic and extrinsic factors which may predispose an athlete to injury with repeated exposures.

In a recent systematic review on the prevention of ERLP in athletes, Thacker et al[74] concluded there is "little objective evidence to support the widespread use of any existing interventions to prevent shin splints." In another systematic review focused on the prevention of stress fractures in athletes and soldiers, the authors reported that their comprehensive review of the literature "highlights how little we know about what works to prevent one of the most common and potentially serious sports- and exercise-related overuse injuries."[111] These two systematic reviews on the status of ERLP prevention strategies exemplify the lack of understanding of the etiology of ERLP and highlights the need for future research in this area.

CONCLUSION

In summary, it is evident from this review that in spite of the common occurrence of ERLP in athletes, the lack of success in the prevention and treatment of ERLP is a reflection of the limited understanding of the risk factors which lead to the development of ERLP. In Van Mechelen's sequence of prevention model,[73] development of successful prevention strategies requires development of knowledge of the mechanisms and factors associated with the development of the condition. In addition to a lack of consensus knowledge, another barrier to the development of successful approaches to address this problem is the indiscriminate use of terms such as "shin splints," which can unintentionally lead one to not consider the complexity of the different pathoanatomic entities that manifest as ERLP. Careful and thorough examination identifying the location, nature, and chronology of symptoms as well as static and dynamic neuromusculoskeletal impairments is essential in the development of an appropriate intervention plan. A great need exists for ongoing high quality research both in the identification of extrinsic and intrinsic risk factors and the development of successful intervention programs.

REFERENCES

1. Hutchins CP. Explanation of spike soreness in runners. *American Physical Education Review.* 1913;18:31-35.
2. Holen KJ, Engebretsen L, Grontvedt T, et al. Surgical treatment of medial tibial stress syndrome (shin splint) by fasciotomy of the superficial posterior compartment of the leg. *Scand J Med Sci Sports.* 1995;5:40-43.
3. Mubarak SJ, Gould RN, Lee YF, et al. The medial tibial stress syndrome. A cause of shin splints. *Am J Sports Med.* 1982;10:201-205.
4. Michael RH, Holder LE. The soleus syndrome. A cause of medial tibial stress (shin splints). *Am J Sports Med.* 1985; 13:87-94.

5. Slocum DB. The shin splint syndrome. Medical aspects and differential diagnosis. *Am J Surg.*1967;114:875-881.
6. American Medical Association. Committee on the Medical Aspects of Sports. Subcommittee on Classification of Sports Injuries. Standard nomenclature of athletic injuries. 1966.
7. Detmer DE. Chronic leg pain. *Am J Sports Med.* 1980; 8:141-144.
8. Batt ME. Shin splints–A review of terminology. *Clin J Sport Med.* 1995;5:53-57.
9. Brukner P. Exercise-related lower leg pain: An overview. *Med Sci Sports Exerc.* 2000;32(3 Suppl):S1-3.
10. Reinking MF, Hayes AM. Intrinsic factors associated with exercise-related leg pain in collegiate cross-country runners. *Clin J Sport Med.* 2006;16:10-14.
11. Ugalde V, Batt ME. Shin splints: Current theories and treatment. *Crit Rev Phys Rehab Med.* 2001;13:217-2653.
12. Orava S, Puranen J. Athletes' leg pains. *Br J Sports Med.* 1979;13:92-97.
13. Clement DB, Taunton JE, Smart GW, McNicol KL. A survey of overuse running injuries. *Phys Sportsmed.* 1981;9:47-58.
14. MacIntyre JG, Taunton JE, Clement DB, et al. Running injuries: A clinical study of 4,173 cases. *Clin J Sport Med.* 1991;1:81-87.
15. Taunton JE, Ryan MB, Clement DB, et al. A retrospective case-control analysis of 2002 running injuries. *Br J Sports Med.* 2002;36:95-101.
16. Reinking MF, Hayes AM. Exercise related lower leg pain in collegiate cross country runners [Abstract]. *J Orthop Sports Phys Ther.* 2003;33:A-49-50.
17. Sallis RE, Jones K, Sunshine S, et al. Comparing sports injuries in men and women. *Int J Sports Med.* 2001; 22:420-423.
18. National Collegiate Athletic Association. ISS results available for fall sports. *The NCAA News*; 2006.
19. Rauh MJ, Margherita AJ, Rice SG, et al. High school cross country running injuries: A longitudinal study. *Clin J Sport Med.* 2000;10:110-116.
20. Bradshaw C. Exercise-related lower leg pain: Vascular. *Med Sci Sports Exerc.* 2000;32(3 Suppl):S34-36.
21. McCrory P. Exercise-related leg pain: Neurological perspective. *Med Sci Sports Exerc.* 2000;32(3 Suppl):S11-14.
22. McCrory P, Bell S, Bradshaw C. Nerve entrapments of the lower leg, ankle and foot in sport. *Sports Med.* 2002; 32:371-391.
23. Beck BR. Tibial stress injuries. An aetiological review for the purposes of guiding management. *Sports Med.* 1998; 26:265-279.
24. Kortebein PM, Kaufman KR, Basford JR, Stuart MJ. Medial tibial stress syndrome. *Med Sci Sports Exerc.* 2000; 32(3 Suppl):S27-33.
25. James SL, Bates BT, Osternig LR. Injuries to runners. *Am J Sports Med.* 1978;6:40-50.
26. Beck BR, Osternig LR. Medial tibial stress syndrome. The location of muscles in the leg in relation to symptoms. *J Bone Joint Surg Am.* 1994;76:1057-1061.
27. Bouche RT, Johnson CH. Medial tibial stress syndrome (tibial fasciitis): A proposed pathomechanical model involving fascial traction. *J Am Podiatr Med Assoc.* 2007; 97:31-36.
28. Detmer DE. Chronic shin splints. Classification and management of medial tibial stress syndrome. *Sports Med.* 1986;3:436-446.
29. Bennett JE, Reinking MF, Pluemer B, et al. Factors contributing to the development of medial tibial stress syndrome in high school runners. *J Orthop Sports Phys Ther.* 2001;31:504-510.
30. Plisky MS, Rauh MJ, Heiderscheit B, et al. Medial tibial stress syndrome in high school cross-country runners: Incidence and risk factors. *J Orthop Sports Phys Ther.* 2007; 37:40-47.
31. Magnusson HI, Westlin NE, Nyqvist F, et al. Abnormally decreased regional bone density in athletes with medial tibial stress syndrome. *Am J Sports Med.* 2001;29:712-715.
32. Magnusson HI, Ahlborg HG, Karlsson C, et al. Low regional tibial bone density in athletes with medial tibial stress syndrome normalizes after recovery from symptoms. *Am J Sports Med.* Jul-Aug 2003;31:596-600.
33. Eickhoff CA, Hossain SA, Slawski DP. Effect of prescribed foot orthoses on medial tibial stress syndrome in collegiate cross-country runners. *Clin Kines.* 2000;54:76-80.
34. Yates B, Allen MJ, Barnes MR. Outcome of surgical treatment of medial tibial stress syndrome. *J Bone Joint Surg Am.* Oct 2003;85-A:1974-1980.
35. Mavor GE. The anterior tibial syndrome. *J Bone Joint Surg Br.* 1956;38-B:513-517.
36. Blackman PG. A review of chronic exertional compartment syndrome in the lower leg. *Med Sci Sports Exerc.* 2000; 32(3 Suppl):S4-10.
37. Hutchinson MR, Ireland ML. Chronic exertional compartment syndrome. *Phys Sportsmed.* 1999;27:90-92.
38. Styf J. Diagnosis of exercise-induced pain in the anterior aspect of the lower leg. *Am J Sports Med.* 1988;16:165-169.
39. Hutchinson MR, Ireland ML. Common compartment syndromes in athletes. Treatment and rehabilitation. *Sports Med.* 1994;17:200-208.
40. Qvarfordt P, Christenson JT, Eklof B, et al. Intramuscular pressure, muscle blood flow, and skeletal muscle metabolism in chronic anterior tibial compartment syndrome. *Clin Orthop.* 1983;179:284-290.
41. Blackman PG, Simmons LR, Crossley KM. Treatment of chronic exertional anterior compartment syndrome with massage: a pilot study. *Clin J Sport Med.* 1998;8:14-17.
42. Black KP, Taylor DE. Current concepts in the treatment of common compartment syndromes in athletes. *Sports Med.* 1993;15:408-418.

43. Arendt EA. Stress fractures and the female athlete. *Clin Orthop.* 2000;372:131-138.

44. Verma RB, Sherman O. Athletic stress fractures: Part I. History, epidemiology, physiology, risk factors, radiography, diagnosis, and treatment. *Am J Orthop.* 2001;30:798-806.

45. Brukner P. Exercise-related lower leg pain: Bone. *Med Sci Sports Exerc.* 2000;32(3 Suppl):S15-26.

46. Nattiv A. Stress fractures and bone health in track and field athletes. *J Sci Med Sport.* 2000;3:268-279.

47. Bennell KL, Brukner PD. Epidemiology and site specificity of stress fractures. *Clin J Sport Med.* 1997;16:179-196.

48. Johnson AW, Weiss CB, Jr, Wheeler DL. Stress fractures of the femoral shaft in athletes-more common than expected. A new clinical test. *Am J Sports Med.* 1994;22:248-256.

49. Goldberg B, Pecora C. Stress fractures: A risk of increased training in freshmen. *Phys Sportsmed.* 1994;22:68-78.

50. Nattiv A, Armsey TD, Jr. Stress injury to bone in the female athlete. *Clin J Sport Med.* 1997;16:197-224.

51. Nelson BJ, Arciero RA. Stress fractures in the female athlete. *Sports Med Arthr Rev.* 2002;10:83-90.

52. Zeni AI, Street CC, Dempsey RL, Staton M. Stress injury to the bone among women athletes. *Phys Med Rehabil Clin N Am.* 2000;11:929-947.

53. Bennell K, Matheson G, Meeuwisse W, Brukner P. Risk factors for stress fractures. *Sports Med.* 1999;28:91-122.

54. Shaffer SW, Uhl TL. Preventing and treating lower extremity stress reactions and fractures in adults. *J Athl Train.* 2006; 41:466-469.

55. Rome K, Handoll HH, Ashford R. Interventions for preventing and treating stress fractures and stress reactions of bone of the lower limbs in young adults. *Cochrane Database Syst Rev.* 2005.

56. Khan KM, Cook JL, Taunton JE, Bonar F. Overuse tendinosis, not tendinitis. *Phys Sportsmed.* 2000;28:38-47.

57. Bernstein J. In the beginning was the word. *J Bone Joint Surg Am.* 2006;88:442-445.

58. Curwin S, Stanish WD. Tendonitis: Its Etiology and Treatment. Lexington, MA: DC Heath & Co.; 1984.

59. Kaufman KR, Brodine SK, Shaffer RA, et al. The effect of foot structure and range of motion on musculoskeletal overuse injuries. *Am J Sports Med.* 1999;27:585-593.

60. McKenzie DC, Clement DB, Taunton JE. Running shoes, orthotics, and injuries. *Sports Med.* 1985;2:334-347.

61. O'Toole ML. Prevention and treatment of injuries to runners. *Med Sci Sports Exerc.* 1992;24(9 Suppl):S360-363.

62. van Mechelen W. Running injuries. A review of the epidemiological literature. *Sports Med.* 1992;14:320-335.

63. Hutchinson MR, Cahoon S, Atkins T. Chronic leg pain: Putting the diagnostic pieces together. *Phys Sportsmed.* 1998;26.

64. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med.* 1998;26:360-366.

65. Fyfe I, Stanish WD. The use of eccentric training and stretching in the treatment and prevention of tendon injuries. *Clin Sports Med.* 1992;11:601-624.

66. Ohberg L, Lorentzon R, Alfredson H. Eccentric training in patients with chronic Achilles tendinosis: normalised tendon structure and decreased thickness at follow up. *Br J Sports Med.* Feb 2004;38:8-11.

67. Purdam CR, Jonsson P, Alfredson H, et al. A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. *Br J Sports Med.* 2004; 38:395-397.

68. Rees JD, Wilson AM, Wolman RL. Current concepts in the management of tendon disorders. *Rheumatology (Oxford).* 2006;45:508-521.

69. Wilson JJ, Best TM. Common overuse tendon problems: A review and recommendations for treatment. *Am Fam Physician.* 2005;72:811-818.

70. Young MA, Cook JL, Purdam CR, et al. Eccentric decline squat protocol offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *Br J Sports Med.* 2005; 39:102-105.

71. Hirose CB, McGarvey WC. Peripheral nerve entrapments. *Foot Ankle Clin.* 2004;9:255-269.

72. Korkola M, Amendola A. Exercise-induced pain. *Phys Sportsmed.* 2001;29:35-38.

73. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med.* 1992;14:82-99.

74. Thacker SB, Gilchrist J, Stroup DF, Kimsey CD. The prevention of shin splints in sports: A systematic review of literature. *Med Sci Sports Exerc.* 2002;34:32-40.

75. Marti B, Vader JP, Minder CE, Abelin T. On the epidemiology of running injuries. The 1984 Bern Grand-Prix study. *Am J Sports Med.* 1988;16:285-294.

76. Macera CA, Pate RR, Powell KE, et al. Predicting lower-extremity injuries among habitual runners. *Arch Intern Med.* 1989;149:2565-2568.

77. Walter SD, Hart LE, McIntosh JM, Sutton JR. The Ontario cohort study of running-related injuries. *Arch Intern Med.* 1989;149:2561-2564.

78. Almeida SA, Williams KM, Shaffer RA, Brodine SK. Epidemiological patterns of musculoskeletal injuries and physical training. *Med Sci Sports Exerc.* 1999;31:1176-1182.

79. Batt ME, Ugalde V, Anderson MW, Shelton DK. A prospective controlled study of diagnostic imaging for acute shin splints. *Med Sci Sports Exerc.* 1998;30:1564-1571.

80. Reinking MF. Exercise-related leg pain in female collegiate athletes: The influence of intrinsic and extrinsic factors. *Am J Sports Med.* 2006;34.

81. Wang X, Wang PS, Zhou W. Risk factors of military training-related injuries in recruits of Chinese People's Armed Police Forces. *Chinese Journal of Traumatology.* 2003;6:12-17.

82. Busseuil C, Freychat P, Guedj EB, Lacour JR. Rearfoot-forefoot orientation and traumatic risk for runners. *Foot Ankle Int.* 1998;19:32-37.

83. Messier SP, Pittala KA. Etiologic factors associated with selected running injuries. *Med Sci Sports Exerc.* 1988; 20:501-505.

84. Williams DS, McClay IS, Hamill J. Arch structure and injury patterns in runners. *Clin Biomech.* 2001;16:341-347.

85. Lun V, Meeuwisse WH, Stergiou P, Stefanyshyn D. Relation between running injury and static lower limb alignment in recreational runners. *Br J Sports Med.* 2004;38:576-580.

86. Hreljac A, Marshall RN, Hume PA. Evaluation of lower extremity overuse injury potential in runners. *Med Sci Sports Exerc.* 2000;32:1635-1641.

87. Montgomery LC, Nelson FR, Norton JP, Deuster PA. Orthopedic history and examination in the etiology of overuse injuries. *Med Sci Sports Exerc.* 1989;21:237-243.

88. Wen DY, Puffer JC, Schmalzried TP. Lower extremity alignment and risk of overuse injuries in runners. *Med Sci Sports Exerc.* 1997;29:1291-1298.

89. Sommer HM, Vallentyne SW. Effect of foot posture on the incidence of medial tibial stress syndrome. *Med Sci Sports Exerc.* 1995;27:800-804.

90. Viitasalo JT, Kvist M. Some biomechanical aspects of the foot and ankle in athletes with and without shin splints. *Am J Sports Med.* 1983;11:125-130.

91. DeLacerda FG. A study of the anatomic factors involved in shinsplints. *J Orthop Sports Phys Ther.* 1980;2:55-59.

92. Lilletvedt J, Kreighbaum E, Phillips RL. Analysis of selected alignment of the lower extremity related to the shin splint syndrome. *J Am Podiatry Assoc.* 1979;69:211-217.

93. Willems TM, De Clercq D, Delbaere K, et al. A prospective study of gait related risk factors for exercise-related lower leg pain. *Gait Posture.* 2006;23:91-98.

94. Willems TM, Witvrouw E. Gait-related risk factors for exercise-related lower-leg pain during shod running. *Med Sci Sports Exerc.* 2007;39:330-339.

95. Reinking MF, Austin TM, Hayes AM. Exercise-related leg pain in collegiate cross country athletes: the influence of foot type [abstract]. *J Orthop Sports Phys Ther.* 2007;37:A75.

96. Micheli LJ. Lower extremity overuse injuries. *Acta Medica Scandinavica*, Supplement. 1986;711:171-177.

97. Krivickas LS. Anatomical factors associated with overuse sports injuries. *Sports Med.* 1997;24:132-146.

98. Stanish WD. Overuse injuries in athletes: a perspective. *Med Sci Sports Exerc.* 1984;16:1-7.

99. Gleim GW, McHugh MP. Flexibility and its effects on sports injury and performance. *Sports Med.* 1997;24:289-299.

100. Hart L. Effect of stretching on sport injury risk: A review. *Clin J Sport Med.* 2005;15:113.

101. Neely FG. Intrinsic risk factors for exercise-related lower limb injuries. *Sports Med.* 1998;26:253-263.

102. Shaffer RA, Rauh MJ, Brodine SK, et al. Predictors of stress fracture susceptibility in young female recruits. *Am J Sports Med.* 2006;34:108-115.

103. Hame SL, LaFemina JM, McAllister DR, et al. Fractures in the collegiate athlete. *Am J Sports Med.* 2004;32:446-451.

104. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am J Sports Med.* 1988;16:209-216.

105. Bennell KL, Malcolm SA, Thomas SA, et al. Risk factors for stress fractures in track and field athletes. A twelve- month prospective study. *Am J Sports Med.* 1996;24:810-818.

106. Simkin A, Leichter I, Giladi M, et al. Combined effect of foot arch structure and an orthotic device on stress fractures. *Foot Ankle Int.* 1989;10:25-29.

107. Korpelainen R, Orava S, Karpakka J, et al. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med.* 2001;29:304-310.

108. Meeuwisse W. Assessing causation in sport injury: A multifactorial model. *Clin J Sport Med.* 1994;4:166-170.

109. Bahr R, Krosshaug T. Understanding injury mechanisms: A key component of preventing injuries in sport. *Br J Sports Med.* 2005;39:324-329.

110. Meeuwisse WH, Tyreman H, Hagel B, Emery C. A dynamic model of etiology in sport injury: The recursive nature of risk and causation. *Clin J Sport Med.* 2007; 17:215-219.

111. Jones BH, Thacker SB, Gilchrist J, et al. Prevention of lower extremity stress fractures in athletes and soldiers: A systematic review. *Epidemiol Rev.* 2002;24:228-247.

CLINICAL SUGGESTION

USE OF POOL NOODLES FOR THE SHOULDER AND ANKLE

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ABSTRACT

The purpose of this manuscript is to provide two clinical suggestions that are inexpensive, easy to fabricate, and very user-friendly activities that can be used for most patients and athletes. The first clinical suggestion is a method of restoring stability of the scapular muscles around the shoulder complex. Following a period of disuse, whether from a surgery or an injury, weakness may be present in the shoulder. This suggestion is an easy and inexpensive tool which can be used in restoring stability of the scapula in all planes of movement as well as combinations of these planes. The method can also be used as a progression from gravity assisted to gravity resisted active range of motion. The purpose of the second clinical suggestion is to provide an inexpensive and easy to use method of improving proprioception in the ankle. Ankle sprains are among the most common injuries seen in sports. Proprioceptive activities are used not only in the rehabilitation process following an injury but as a training tool to help prevent ankle injuries. This method can be used in the clinic, in a training facility, or as part of a home exercise program.

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PROBLEM #1-SHOULDER

The most frequent cause of shoulder pain is subacromial impingement syndrome accounting for 44 – 60% of all complaints of shoulder pain during a physician visit.[1] Although the cause of shoulder impingement is often multifactorial, scapular stabilizer weakness is often associated with the impingement. Cools et al[2] found that overhead athletes with impingement syndrome demonstrated strength deficits and muscular imbalances in scapular muscles compared with non-injured athletes. Weakness in scapular stabilizers will create poor length tension relationship between the scapula and the humerus. The length between the scapula and humerus will put the rotator cuff in a more lengthened position. If the scapular stabilizers are weak or fatigued, the result will be a destabilized scapula which will contribute to impingement. Muscular weakness and fatigue in the scapular stabilizers will also lead to a loss of upward scapular rotation during overhead activities referred to as scapular dyskinesis.[3] Scapular squeezes and shoulder shrugs are excellent ways of strengthening the musculature surrounding the scapula. Both of the exercises work in one plane of motion and, therefore, do not prepare the shoulder for many of the functional activities of life or sport. Because the glenohumeral joint is the most mobile joint in the body, the scapula must be able to stabilize that joint in multiple positions.

SOLUTION TO PROBLEM #1

A nice way to train the scapular stabilizers in multiple positions and planes of movement is to perform extreme shoulder ABC's. The only piece of equipment needed for extreme ABC's is a giant marker.

To fabricate a giant marker for the extreme shoulder ABC's take a 1 foot piece of ½ inch piece of PVC pipe and slip the pipe approximately 6 inches into the hole at one end of a pool noodle. The PVC pipe will serve as the handle for the marker. Cut the pool noodle in half making it 3 feet in length. Have the patient hold the PVC pipe handle to draw the alphabet *(Figure 1)*. Very weak or deconditioned patients can start by drawing the alphabet on the floor in an effort to stay below 90° of shoulder flexion *(Figure 2)*. As the patient gets stronger, progress to where they are drawing the alphabet on the wall. Writing larger letters seems to challenge the scapular stabilizers

Figure 1. *Patient holding PVC pipe handle*

Figure 2. *Patient drawing the alphabet on the floor with the shoulder below 90 degrees of flexion*

Figure 3. *Patient writing larger letters above horizontal*

more than smaller letters *(Figure 3)*. Repeating the alphabet two times is quite challenging to the patient.

PROBLEM #2-ANKLE

Another use for a swimming pool noodle is as a device to help a patient to regain kinesthetic awareness after an ankle injury or surgery. As many as 70% of athletes in some sports suffer from recurrent ankle sprains.[4] Following an ankle sprain, many patients will complain of residual symptoms 6 to 18 months after the injury.[5] Research has found that there is a loss of kinesthetic awareness following trauma to the ankle.6 This deficit in kinesthetic awareness or proprioception can lead to a re-injury of the same area, injury to another area, or at the very least a decrease in performance in athletic activities.

The core of ankle training research over the past decade has been directed toward the development of exercise programs aimed at preventing the occurrence and reoccurrence of ankle sprains. These programs have focused on proprioception, strengthening, balance, and coordination exercises.7 Often patients will perform activities on an uneven or unstable surface in hopes of regaining the kinesthetic awareness. Therapists will have patients walk in sand or over rolled towels to challenge the balance of the patient. Sand is often not readily available in a clinical setting. Towels, while readily available, will be compressed after the patient has walked across them several times.

SOLUTION TO PROBLEM #2

A foam pool noodle can be cut in half long ways and laid on the floor with the cut side down. To begin, the patient can simply balance on the foam noodle on one leg *(Figure 4)*. Laying two halves of the noodle end to end will provide a 12 foot length to walk or run depending on where the patient is in the recovery process *(Figure 5)*. Have the patient walk or run across the noodle as if they were walking along a balance beam. The foam will challenge the ankle in a safe manner.

Figure 4. *Patient balancing on a foam noodle on one leg*

Figure 5. *Patient walking on a foam noodle*

REFERENCES

1. van der Windt DA, Koes BW, de Jong BA, Bouter LM. Shoulder disorders in general practice: incidence, patient characteristics and management. *Ann Rheum Dis.* 1995; 54:959-64.
2. Cools AM, Witvruw EE, Nele NM, Danneels LA. Isokinetic scapular muscle performance in overhead athlete with and without impingement syndrome. *J Athl Train.* 2005; 40:104-110.
3. Kibler WB: The role of the scapula in athletic shoulder function. *Am J Sports Med.* 1998;26:325-327.
4. Gerber JP, Williams GN, Scoville CR, et al. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int.* 1998; 19:653-660.
5. Braun BL. Effects of ankle sprain in a general clinical population 6 to 18 months after medical evaluation. *Arch Fam Med.* 1999;8:143-148.
6. Freeman MA, Dean MR, Hanham IW. The etiology and prevention of functional instability of the foot. *J Bone Joint Surg Br.* 1965;47:678-685.
7. Coughlan G, Caulfield B. A 4-week neuromuscular training program and gait training patterns at the ankle joint. *J Athl Train.* 2007;42:51-59.