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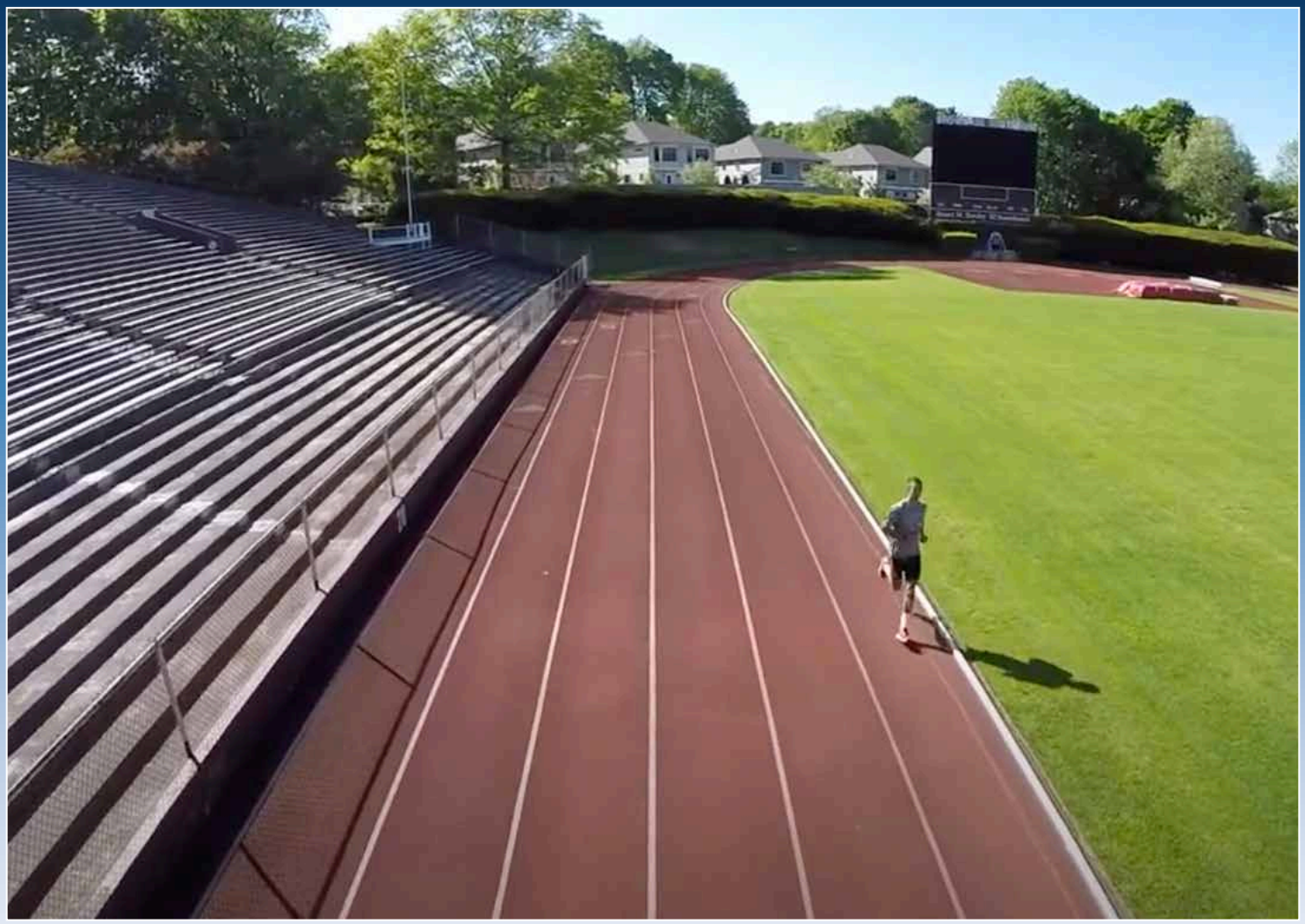

SPECIAL SECTION

SPORTS MEDICINE: THE BIGGER PICTURE

GUEST EDITORS: BRETT D. OWENS, MD; RAMIN R. TABADDOR, MD

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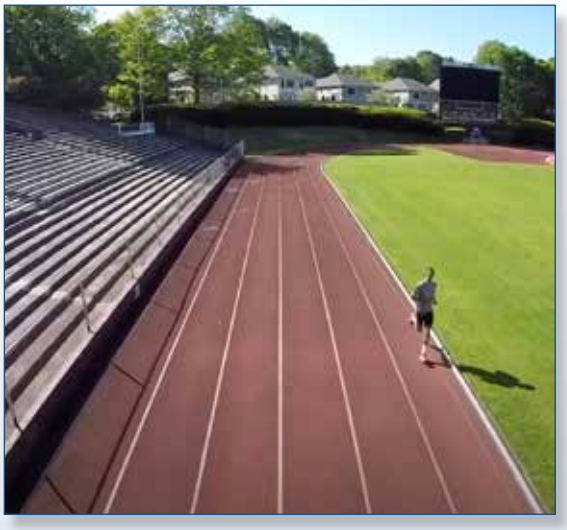

On the cover: Former Brown University runner and patient of Dr. Ramin Tabaddor as seen in a video describing his return to sports after hip surgery. https://www.youtube.com/watch?v=LJPHA5OkXRQ [COURTESY OF UNIVERSITY ORTHOPEDICS]

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"Presence Hallucinations" in PD Patients

JOSEPH H. FRIEDMAN, MD

The behavioral aspects of Parkinson's disease (PD) are as fascinating as they are problematic. Until the late 1980s the only non-motor problem in PD that attracted any significant interest from physicians was depression. Even neurologists could see that a lot of their PD patients were depressed. But, of course, why shouldn't they be? But some raised the question of whether the depression was reactive, that is, a normal response to a progressive, incurable, potentially disabling disorder, or whether it was intrinsic to the disease, a foreordained process directly related to the loss of neurons in a particular location in the brain. At that time, some attention, albeit very little, was paid to the problem of medication-induced psychosis, dementia and a host of other problems, most of which were not recognized.

Hallucinations occur in about 20–30% of medication-treated PD patients, and these tend to be fairly stereotypic. They are usually visual, and half as often auditory, mostly in the context of co-existent visual, and considerably less often tactile, olfactory or gustatory. They are generally without emotional content, unlike hallucinations that occur in schizophrenia and other primary psychotic disorders. Some are pleasing, like the baby my patient saw every evening, "who was such a good baby. He never made a sound," or the tiny people another patient saw who looked like small Christmas ornaments. Some authorities have employed the label of "minor" hallucinations to those that occur only transiently in the peripheral visual field, but are never seen directly. These are usually perceptions of shadows, cats or dogs running by, but disappear when looked at directly, and to a peculiar sensation, called "presence hallucinations." These are not really hallucinations, as they do not occur as a false perception in one of the five special senses. These are the feelings that there is someone or something behind or to the side. They are never sensed as threats. The patient does not worry about being attacked. The person or animal is never hiding. The patient turns to see who or what is there, but there is nothing. Hence there is a strong feeling of a "presence." As best I know, this is transcultural, although I am not aware that it has been studied, so I am unsure. The feeling resolves if the medications are reduced or an antipsychotic medication is given.

As noted above, the sense of presence is not an hallucination. It is classified with the actual hallucinatory phenomena, but in the category of "minor" hallucinations. It is a unique phenomenon. Although there may be occasional patients with a psychotic delusion that they have an angel, or devil, watching over them, or an invisible twin, in PD, the patients don't know who is present, and are neither frightened, worried or pleased. The presence is neither reassuring, nor threatening. And, although I believe it may be unique to PD, there are two settings in which a sense of presence has been described, but the phenomena are quite different. A sense of presence is well described in the recently bereaved, who continue to feel the presence as if the person was still alive. This sensation may last a long time, but apparently is most commonly present early on. The second scenario, sometimes called "the third person factor," occurs in people who have had near-death experiences, and have been led by an unseen person to safety. The survivor may consider the unseen presence an angel or not, but generally not a psychotic experience. The presence is not felt again but is credited with the successful overcoming of a life-threatening event.

The perception of a presence, like the perception of an illusion, or even a fleeting hallucination, are experiences that we've all had. We've all turned to see who was in the room with us when there was no one there. We've seen an image at a distance that we think is a boy walking a dog but it turns out to be fire hydrant as we get closer. Or we've answered a door, having heard a knock,

but there is no one there. These are all part of normal existence and only become medical phenomena when they become overly common, or overly real. In PD, "presence hallucinations" have been identified in untreated patients in a single report. They are otherwise associated with use of the medications for treating motor dysfunction in PD, and are considered a milestone on the road to the development of PD psychosis. Yet, these patients always know this is unusual, never think these are "real" experiences, and are never bothered by the experience. It's simply an odd experience.

I have no idea what the underlying pathophysiology is. This is not a process that has been studied. I recently encountered a PD patient who reported that she had a feeling of presence, but only on her left side, just out of view. This is interesting, suggesting a degree of focality to the pathophysiology. I am fascinated by this phenomenology but, in truth, I don't actually think it is a very important clinical observation, other than for reassuring patients and their doctors that it is a not uncommon experience for treated PD patients.

One of the many rewards of practicing medicine is to share the world of our patients, often quite different in so many ways than our own. ❖

Author

Joseph H. Friedman, MD, is Editor-in- chief Emeritus of the *Rhode Island Medical Journal,* Professor and the Chief of the Division of Movement Disorders, Department of Neurology at the Alpert Medical School of Brown University, chief of Butler Hospital's Movement Disorders Program and first recipient of the Stanley Aronson Chair in Neurodegenerative Disorders.

Communicating Through COVID-19: Keeping Patients Connected to Loved Ones

ANITA KNOPOV, MD; KELLY WONG, MD; RORY MERRITT, MD, MEHP

Notice went out hospital-wide: dying patients with COVID-19 could have up to two visitors for only fifteen minutes. As hospitals confronted the dual realities of PPE shortages and that anyone could be a silent carrier of the novel coronavirus, a new dictum went out: no patient – ambulatory or dying, with or without COVID 19 – could have any visitors. No exceptions.

Suddenly, nurses, residents and attendings would find themselves breaking bad news through telephone. Patients would now be surrounded by healthcare workers separated by layers of PPE rather than the embraces of their loved ones. How would patients respond? Would they avoid the hospital until too late? Would the patients who were already hospitalized rush to leave the hospital to avoid an isolating hospitalization?

Hospital visitation is essential. It provides comfort to patients, promotes family-driven patient advocacy, prevents sundowning and most important of all, chaperones patients and their most cherished from living through dying. The heightened anxieties around the pandemic in light of societal upheaval and real concerns about the disease's lethality make patient visitation even more important.

With so many pressing priorities of hospital systems – obtaining PPE, developing infection control protocols, preparing for the surge, flattening the curve, up-training staff – who would worry about patient visitation? The emotional well-being of our patients was lagging behind loads of new policies to address their physical well-being.

Technology was an intuitive workaround, one that many providers started to use on their own, with their own devices. We shucked the 'protective' Ziploc bags from our cell phones, and placed them on speakerphone next to an intubated patient's head, promising family members, "They can hear you. I promise." We learn early in medicine to be leery of making promises, especially when it pertains to death and dying. But we promise that a family member's last words to their dying loved one are heard and that they matter. "I'm going to walk away to give you a moment alone. Just hang up when you feel you are finished."

Although the voices of family members is certainly better than no contact at all, the voices themselves could be overwhelmed by the whirring of high-flow oxygen and the incessant cacophony of I.V. pumps. We sought a way to allow patients to connect face-to-face with their loved ones and for the critically ill and intubated, a way for their families to truly see their condition. Video-conferencing via tablets was the agreed upon solution, a technique that has been tried at the institutional scale at other hospitals settings.[1,2]

We learn early in medicine to be leery of making promises, especially when it pertains to death and dying. But we promise that a family member's last words to their dying loved one are heard and that they matter.

Given the small geographical nature of the State of Rhode Island – it takes barely an hour to drive from top to bottom – we envisioned a statewide solution. Partnering with the Rhode Island Medical Society, a group of committed individuals, physicians and non-physicians, a website was created to solicit donations of used tablets. The message resonated and within one week we had obtained seven hundred devices, with a large contribution from Amazon. We reached out to every hospital in the state, reformatted each device thanks to the support of information technology (IT) wizards and distributed devices based on the percentage of hospital beds a hospital had of total Rhode Island hospital beds.

Each hospital has implemented the devices in various ways. At one, social work took on the task of distributing the devices, scheduling meeting times with families, and setting up the device for patients; at other hospitals, IT staff, palliative care and chief internal medicine residents led the effort. In an era marked by profound stress and competing priorities one would think families would not find time to thank hospitals for their efforts to connect loved ones. And yet, the thank yous are pouring in. "Letting them see each other on the iPad before [she] died is so special. Thank you."

Thank you for letting us care for your loved one. We will do everything we can to help you say goodbye. We promise. ❖

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Sarah Stevens, RIMS Office Manager, adheres to best social distance practices at Scarborough Beach while checking RIMJ on her phone. Sarah, who started at RIMS in May 1996, has been an indispensable member of the team, working with the leadership, membership, and specialty societies, and as a valuable liaison to the journal for the past eight years. She retired on August 31. Best wishes to Sarah in all her future endeavors.

NARRAGANSETT PHOTOS: JOSEPH MIELE

PROVIDENCE, RHODE ISLAND

Michael E. Migliori, MD, Ophthalmologist-in-Chief at RI Hospital, and RIMS Public Laws Chair, pauses to access the journal on Blackstone Boulevard.

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Sports Medicine: The Bigger Picture

BRETT D. OWENS, MD
RAMIN R. TABADDOR, MD
GUEST EDITORS

When we initiated the plan for this special issue in October 2019, fall sports were in full swing. Our professional lives were packed with our clinical sports medicine practices and our sideline and training room work for the high school, collegiate, and professional teams we cover. How things have changed in the past year…

Since March 2020, COVID-19 has completely redefined our professional and personal lives more than any event in our lifetimes. Spring sports were abruptly ceased, and schools moved to online education. Our practices have scrambled to care for patients through telehealth visits. There are few aspects of our professional lives that have not been impacted.

Historically, in times of despair, tumult and crisis, this country has looked to sports to inspire, distract, harmonize and connect us. Bob Costas stated, "The best thing about sports is the sense of community and shared emotion it can create." In these difficult and challenging times, we have deeply felt the absence of sports and the positive effects it has on our lives. Yet the loss of human life due to this illness has also brought reckoning with the sometimes oversized role sports plays in our community and suspending athletics seems like a small sacrifice in the face of the mortality and morbidity witnessed.

For many, the question remains, "When will sports return?" And how can we return to sports in a way that protects our athletes, as well as team staff, families and spectators? Will the incidence and type of injuries be influenced by the deconditioning athletes may have experienced during this shutdown or by the way athletes are reintroduced to training or competition? How will our responsibilities and scope of care for athletes change as it pertains to COVID-19 prevention? For those in the sports medicine field, these are just some of the unanswered questions we face.

While the current pandemic has had an overwhelming impact on sports medicine, the downstream effects of this contagion and our response will be studied for years to come. For the current issue, we have assembled an esteemed group of sports medicine experts to produce a collection of (non-COVID) topics relevant to the care of athletes. We hope that as athletes are able to return to their fields, this issue will help us provide optimal care. We have also provided a brief summary on the state of sports participation during this pandemic, though the situation may change much by the time this issue is published. While there is much uncertainty about the return of sports, we may all have a renewed appreciation for the positives they impart to society.

Guest editors

Brett D. Owens, MD, Professor of Orthopaedic Surgery, Department of Orthopaedic Surgery, Alpert Medical School of Brown University, Providence, RI.

Ramin R. Tabaddor, MD, Assistant Professor of Orthopaedics, Department of Orthopaedic Surgery, Alpert Medical School of Brown University, Providence, RI.

COVID-19 and Review of Current Recommendations for Return to Athletic Play

JAMES DOVE, MD; ANDREW GAGE, MD; PETER KRIZ, MD; RAMIN R. TABADDOR, MD; BRETT D. OWENS, MD

ABSTRACT

In December 2019 a respiratory illness known as Coronavirus 2 (SARS-CoV-2, COVID-19) broke out in a region in China and rapidly spread to become a pandemic affecting all sporting events worldwide. The Summer Olympics scheduled to be held in Tokyo were postponed until 2021, and all professional leagues in the United States postponed or canceled events. As the United States has begun to open up, there remains uncertainty of when sporting events can safely be held. Many professional leagues and the National Collegiate Athletic Association have established guidelines and recommendations for their athletes to compete safely. In this article, we review the protocols that have been established to allow athletes to return to play, and we review briefly the effects COVID-19 infection may have on athletes.

INTRODUCTION

In December 2019 a severe acute respiratory syndrome later known as Coronavirus 2 (SARS-CoV-2 or COVID-19) broke out in a region of Wuhan, Hubei Province, China.[1] The virus rapidly spread worldwide, and by March 2020 the World Health Organization (WHO) designated it a pandemic.[2,3] The global crisis affected every aspect of life, including sports. In an historic manner, major local and international sporting events were affected, including the Olympic Games in Tokyo, which were postponed until summer 2021.[4] In the United States, all professional, collegiate, and organized sporting events were postponed or canceled.[17,18] The effect on athletes has been devastating as all formal training and practices have been banned. As we begin to emerge from the effects of COVID-19, we enter a new uncertainty in the world of sports. As athletes gradually return to training and practices and eventually to games again, it will become important for trainers, team physicians and all providers to take the proper precautions and recommendations to ensure athletes may continue to participate and compete at high levels amidst the concern of virus spread. The purpose of this article is to review the current recommendations and models being developed to allow for a healthy and safe return to sport for all those involved and to briefly review COVID-19 infection in athletes.

INITIAL COVID-19 RESPONSE

The risk of COVID-19 spread, or infectivity, appears to be very high.[5] Much like influenza, the coronavirus spreads from person-to-person in close contact by respiratory droplets.[5,6] Because of the high infectivity, significant measures were taken by countries, including suspending all immigration and travel and ordering mandatory quarantining and self-isolation.[7,8] As countries begin to reopen and as athletes return to training and practices, it is important to mitigate and limit the risk of spread of the virus. Each sport has its own unique risks to the spread of the virus, and this fact should be taken into account. Some sports, such as golfing and time trial cycling, can realistically socially distance throughout competition. Other sports, however, such as football and soccer, cannot practically socially distance.[9] A distinction should be made for those sports that are low risk and high risk for spreading the virus. Travel also increases the risk of viral spread.[6-9] Athletes that travel across the country have the potential to increase the spread, sometimes unknowingly, unless proper precautions are maintained. In regards to spectators or non-athletic participants, they are at risk of contracting or spreading the virus as well. One of the first documents to address the issue of risk and mitigating the risk of viral spread for mass gatherings was published by the WHO in March 2020.[10] In an effort to categorize the risk of mass gatherings for sporting events, the WHO added an addendum that included a tool that may be completed by organizers of events to assess accurately the overall risk of spread.[15] Those events with high to very high risk need to make significant efforts to mitigate the risk or consider postponing or canceling the event.[9,15] In the United States, professional sports and collegiate athletic organizations have developed their own safety and health protocols to limit the risk of viral spread.

COLLEGIATE ATHLETICS

The National Collegiate Athletic Association (NCAA) recently released the third publication on "the Resocialization of Collegiate Sport" established by the NCAA COVID-19 Advisory Panel led by NCAA Chief Medical Officer Brian Hainline. The goal of the report is to provide new guidance to prevent community spread of COVID-19 in the athletics

setting.[11] The first publication provided guidance for phasing in sports and the second publication emphasized personal and institutional considerations to prevent spread of COVID-19.[11,13,14] The newest report emphasizes the point that the first two publications were written with the assumption that nationally we would see a decline in the rate of COVID-19 infection. Upon examining the data provided by the CDC, however, the 7-day moving average has continued to increase.[12] Despite the current trends we may be observing with COVID-19 cases, the third publication provides guidance and recommendations to help mitigate the risk of spread, including daily self-health checks, universal masking on all sidelines, and testing to be implemented for all athletic activities including pre-season, regular season and post-season.[11] The athletic season will certainly look different with the implementation of these recommendations, but it will help to reduce the risk of further spread of the virus among athletes.

Finally, the latest publication provided by the NCAA updates the risk assessment and risk categorization for each sport. Low contact risk sports include bowling, equestrian, golf, swimming, tennis and track and field. Medium contract risk sports include baseball, softball, cross country, and gymnastics, while high contact risk sports include basketball, football, ice hockey, lacrosse and volleyball[11]. Within this framework, the publication offers recommendations for testing strategies based on the risk. For instance, they recommend that for a high contact risk sport all student-athletes and "inner-bubble" personnel (coaches, medical staff, officials, other essential personnel) should be tested upon arrival to campus, every two weeks for surveillance during off-season and weekly PCR testing during in-season (pre-season, regular season, post-season).[11] In contrast, for low contact risk or medium contact risk sports, testing is recommended for student-athletes and inner-bubble personnel upon arrival to campus but no more than every two weeks for the off-season and in-season.[11] **Table 1** depicts the testing strategies recommended by the NCAA. Furthermor, the guidelines recommend that for high contact risk sports, testing should be performed and results available within 72 hours of competition. If PCR testing cannot be performed within 72 hours the competition should be postponed or canceled.

Even with these guidelines and recommendations from the NCAA, individual conferences have developed their own ways to mitigate the risk of virus spread among their scholar athletes by planning to play a conference-only schedule[19] while other conferences have canceled all fall sports[20,21] and other conferences have delayed fall sports until the spring for possible competition at that time.[28] Ultimately, the decision about return to play will fall upon the individual institutions with guidance from their conferences, but with these recommendations and protocols, the NCAA has provided schools an opportunity to return to sports with ways to limit virus spread and mitigate risk. The team physician will play a vital role to ensure athletes can compete in a safe environment.

PROFESSIONAL SPORTS

Professional sports will offer a unique look into the protocols and procedures used to help athletes return to play. In the United States, all professional leagues have provided guidelines for physicians, providers, and trainers to help limit the risk of COVID-19 spread. Some leagues have taken unique measures to protect their athletes that will be competing.

In June, the National Basketball Association (NBA) developed and released to teams a 113-page document detailing the procedures and protocols that will take place in the "bubble" in Orlando, Florida to complete the 2019-2020 season for 22 of the 30 teams. Very similar to the recommendations put forth by the NCAA and the WHO, it is expected for players to social distance as much as possible; however, the rules in Orlando are strict as people will be wearing a device that sets off an audible alarm if they are within 6 feet of other people for more than 5 seconds; players and personnel are also likely to be wearing monitors to assess body temperature and blood oxygen levels.[16] Testing

Table 1.

Scenario	High Contact Risk	Medium Contact Risk	Low Contact Risk
Arrival on Campus	All athletes and "inner bubble" personnel tested	All athletes and "inner bubble" personnel tested	All athletes and "inner bubble" personnel tested
Summer Athletic Activities (Voluntary)	Surveillance PCR testing	Surveillance PCR testing	Surveillance testing in accordance to university plan for all students
Summer Athletic Activities (Required)	Surveillance PCR testing	Surveillance PCR testing	Surveillance testing in accordance to university plan for all students
In-Season (Pre-season, Regular season, Post-season)	Weekly PCR testing	Surveillance PCR testing	Symptomatic testing
Out-of-Season Athletic Activities	Surveillance PCR testing	Surveillance PCR testing	Surveillance testing in accordance to university plan for all students

• Surveillance PCR testing = testing 25-50% athletes and inner bubble every two weeks if physical distancing, masking, and other protective features are not maintained
• Symptomatic and high contact risk individuals should have additional testing regardless of scenario or contact risk sport

will be implemented every other day through October 13th, and athletes who test positive must quarantine and likely leave the bubble.[33] Creating an isolated bubble will certainly lower the risk of COVID-19 spread, and as of now, the NBA appears to have the resources to provide players with all testing and monitors that they require. The approach taken by the NBA appears to be the most stringent requiring all games to be played in Orlando and all players to remain in the bubble for the duration of the season.

In similar fashion, the National Hockey League (NHL) has established guidelines to return to play with two "hub" cities to host 24 of the 31 teams: 12 from the Eastern Conference in Toronto and 12 from the Western conference in Edmonton.[39] League commissioner Gary Bettman and the Return to Play Committee established phases to its reopening: Phase 2 began on June 8 and allowed limited workouts at team facilities, Phase 3 began on July 13 and included training camp in the two hub cities, and Phase 4 began on August 1 for the Stanley Cup Qualifiers.[38] Players and personnel will undergo daily testing with results provided within 24 hours while in the hub cities; the NHL has also designated Secure Zones which include rinks, hotels, bars, restaurants, and entertainment options.[40] The models established by the NBA and NHL are unique and historic. Their guidelines and protocol may influence further practices from other leagues. At this time however, the other major professional leagues will allow their teams to travel, but they also have provided their own protocols and procedures to be followed to protect their athletes.

For Major League Baseball (MLB), COVID-19 interrupted spring training when commissioner Rob Manfred postponed the season on March 12 following increased concern and spread of the virus across the United States.[34] As he and medical personnel including Dr. Gary Green, the MLB Medical Director, began plans to return to the season, they considered establishing a bubble situation in a few cities much like the NBA and NHL, but MLB Senior Vice President and Deputy General Counsel Patrick Houlihan said that plan lacked practicality as housing for over 1,800 players and just as many staff would be difficult to obtain and players did not like the idea of the bubble for the entire 60-game season.[35] The NBA and NHL are both finishing a season that was well under way before the pandemic hit. With only a few months left to play, a bubble situation is feasible for these leagues while others have to handle the issue of travel and playing in multiple cities. At the time of this writing, following opening weekend, the Miami Marlins organization was found to have as many as 17 members, including 15 players, test positive for the virus.[37] That weekend the Marlins played in Philadelphia against the Phillies. The MLB has decided to revise the schedule and isolate the Marlins and Phillies for a period of time; other teams are continuing play.[36] The MLB is hit with its first major blow, and how they decide to move forward might influence how other leagues handle an increase in COVID-19 spread among their athletes.

In the National Football League (NFL), players have begun reporting to training camps, and in preparation for their arrival, the NFLPA (Player's Association) and league have agreed to protocols to manage and mitigate the risk of viral spread. Established by Dr. Allen Sills, the Chief Medical Officer of the NFL, and other providers, these recommendations and regulations include testing of all players before arriving to camp and testing every day for two weeks or testing until positive cases are below 5% league-wide.[22] Along with testing, the league recommends to continue social distancing, wearing masks, and maintaining healthy hygiene habits. With the update, Dr. Sills included a look at the adjustments made to the training facilities by teams to handle the new reality with COVID-19.[23,24]

In the recent Virtual AOSSM 2020 Annual Meeting, President James Bradley, MD, spoke with both Dr. Sills and NFL Commissioner Roger Goodell. During the discussion, Dr. Sills made the point that the NFL's protocols to mitigate risk of viral spread could help provide a template for other aspects of society to open including schools and businesses.[44] The data that they obtain will offer a glimpse into which procedures and protocols are most successful to limit community spread of the virus. All eyes will be on the NFL and other professional leagues as they begin to return to play to evaluate their successes or failures.

All professional leagues that have returned to play have done so without fans in the stands. It is unclear when we might see the return of fans at sporting events, but for now, this measure will help to reduce community spread. Dr. Anthony Fauci, the Director of the National Institute of Allergy and Infectious Diseases and lead member for the White House Coronavirus Task Force, spoke on this idea during an interview with the Wall Street Journal. As other countries are moving towards having fans return to sporting events, Dr. Fauci recommended for events in the US, "they should mandate…to have a mask on" for fans who want to enter the stadium, and they "should have a considerable degree of distancing."[45] Obviously, competing without fans creates its own bubble among the players and essential personnel. This measure allows sports to continue without a direct threat to the community. However, in regards to collegiate sports, it does not appear that everyone has agreed with this practice, as some universities are considering 25% attendance at their college football games.[46,47] The other concern, regardless of fans in the stands, would be the tailgates and fraternity parties that will occur surrounding football games and other events.[48] All of these events are major risk factors for community spread in the fall. At this point, many questions still remain as to the best way to assimilate fans back into the sporting experience, both in and out of the stadium.

YOUTH AND HIGH SCHOOL SPORTS

Specific recommendations and protocols for return to sport in youth leagues and high schools are lacking in the literature. This fact is likely due to the intrinsic nature of these sports to be regulated locally by the state, cities, and communities. In May 2020, the National Athletic Trainer's Association (NATA) released recommendations for communities that will begin returning to play with emphasis on establishing a COVID-19 response team, preparing athletes with detraining concerns to return to sport, and providing risk mitigation strategies.[42] These recommendations are very broad, however, and lack any comment on frequency of testing for athletes. Due to limited availability, testing among secondary school athletes will not occur at the frequency as it does at higher levels of competition. This is concerning as asymptomatic participants competing will increase the risk for community spread. Also in May 2020, the National Federation of State High School Associations (NFHS) under the Sports Medicine Advisory Committee (SMAC) provided recommendations to state associations for opening school activities and sports. They recognize that testing availability for high school sports will be limited but strongly encourage for each phase of opening that all students and coaches undergo screening including a temperature check before being allowed to participate.[43] They also include a categorization of risk for each sport, much like the NCAA's report on the Resocialization of Collegiate Sport. The SMAC acknowledges that local and state associations will be responsible for establishing the protocol and procedures to return to play.

In the United States, different regions have been more affected than others by COVID-19. Each community should consider the risk of spread based on their prevalence of the virus before returning to play. For event coordinators and athletic associations responsible for deciding guidelines in their communities, they should use objective criteria such as the WHO risk assessment tool for mass sports gatherings to help mitigate the risk of community spread. As stated, the NATA and SMAC have also provided guidelines which should be followed. It is understood that sports are a very important aspect of life, and we are at a time to begin returning to play, but the proper precautions must be taken to limit virus spread.

THE NEW TRAINING ROOM WITH COVID-19

COVID-19 may change the interactions between athlete and provider, but the role of the training room to provide complete care to the athlete should not be altered. As we have seen in the NFL, strong measures have been put into effect to protect those entering training facilities.[23,24] The senior authors of this review are team physicians for NCAA collegiate teams in the Northeast region, and below we have included the protocols issued by our institutions and conferences for interactions in the training room. Both the Ivy League and Atlantic-10 Conference have delayed fall sports for possible spring competition.

Education is the first step to help protect athletes and providers. All athletes should be aware of the signs and symptoms of COVID-19 infection. Many programs will begin their season with a brief review of COVID-19 and how it is spread. The sports physicians will likely be called upon to help educate the athletes.

Before any athlete or essential personnel enters the training room or training facility, a screening assessment should be performed. This evaluation should include a brief questionnaire and a temperature check. Any patient who screens positively for symptoms or who has a fever should be quarantined and tested for COVID-19. Isolating COVID-19-positive patients is the initial step to limit spread of the virus. If possible, programs should perform contact tracing as well for those that have interacted with COVID-19-positive athletes or personnel.

Inside the training room, everyone should wear masks. Appropriate personal protective equipment (PPE) should be provided. Seeing athletes at staggered times will help limit numbers and allow for appropriate social distancing. Lockers, supplies, and training beds need to be sanitized after each use. Disinfectants and sanitizers should be available. Programs need to take the initiative to develop these protocols with the recommendations provided by the NCAA and their respective conferences.

COVID-19 IN THE ATHLETE

Finally, a review of COVID-19 and its effects on the athlete should be discussed. Although the majority of severe cases of COVID-19 appears to affect those adults greater than 60 years old with co-morbidities,[25-27] the sports physician should be cognizant to recognize those athletes or personnel that exhibit signs or symptoms concerning for complications related to the virus. Toresdahl et al. give a brief review on how to manage the athlete with COVID-19, including when in-home isolation can be discontinued and an emphasis on mental health support.[41] As athletes return to play, it is likely that an increase of cases will be seen by the sports physician and (s)he should be able to triage and manage non-severe cases.

Early research on COVID-19 revealed that angiotensin-converting enzyme 2 (ACE2) is a receptor for possible viral entry of the virus.[29] This association is significant, as ACE2 is found throughout the gastrointestinal system, the heart, the kidney, and type II alveolar cells in the lungs.[30] In regards to the athlete, physicians must be comprehensive in their evaluation for those that are infected and are looking to return to play following resolution of their infection. Specifically, athletes should have a thorough cardiovascular exam as patients with COVID-19 infection have shown increased

troponin levels above the 99th percentile, suggestive of significant myocardial damage.[31] The effects of having athletes return to play without proper cardiac clearance would be devastating and Baggish, et al. provided initial guidance for the cardiac evaluation of previously infected athletes.[32]

Though many athletes may test positive during the course of competition and even prove to be asymptomatic, it will take a multidisciplinary approach to ensure these athletes are safe enough to continue to compete at high levels. In a panel discussion led by Dr. Rick Wright at the Virtual AOSSM 2020 Annual Meeting, all participants agreed that athletes must come first.[44] COVID-19 has had an immeasurable effect on sports, including economic devastation, but we must remember that before returning to play we must provide a safe and healthy environment for all those competing.

SUMMARY

COVID-19 has drastically changed the world of sports. In historic fashion, international events, including the 2020 Summer Olympic Games and all professional leagues worldwide, have been postponed or canceled. The risk of virus spread was unacceptably high for sporting events to continue until we knew more about the disease and how to limit risk of community spread. As more information has become available worldwide, society is beginning to reopen. In regards to sports, multiple guidelines and recommendations have been established with input from medical professionals involved in all professional leagues and medical directors at the CDC and WHO to help mitigate the risk of spread. For professional leagues, many people will be observing the successes or failures they may experience as their guidelines may offer the framework to allow other parts of society to reopen. The sports medicine doctor, athletic trainer, physical therapist and all providers will have very significant roles to ensure that all athletes are educated and healthy to compete when they return to play.

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Shoulder and Elbow Injuries in the Adolescent Throwing Athlete

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ABSTRACT

Shoulder and elbow injuries in the adolescent population can be generally divided into skeletally immature and skeletally mature. Skeletally immature injuries refer to damage to the open growth plate (physis) in the young athlete, which have distinct differences in long-term risks if not managed correctly due to the potential for growth disturbance. Skeletally mature injuries occur in athletes with closed growth plates and are less likely to limit growth potential. It is important to recognize these different types of injuries, as well as the patients most at risk for each type because treatment may vary significantly between the two groups. The main skeletally immature injuries covered by this review will include: medial epicondyle apophysitis ("Little Leaguer's elbow), medial epicondyle fractures, olecranon stress fractures, capitellar osteochondritis dissecans (OCD), and proximal humeral apophysitis ("Little Leaguer's shoulder"). The skeletally mature injuries discussed will include: valgus extension overload syndrome (VEOS), ulnar collateral ligament (UCL) tear, shoulder instability, and superior labral anterior-posterior (SLAP) tears. We will review the history and presentation of the injuries as well as different treatment strategies and return to play guidelines for both primary care sports physicians as well as orthopedic surgeons.

INTRODUCTION

Youth sports are ubiquitous across America, and on any given weekend, millions of adolescents participate in baseball and softball. An estimated 2.4 million children participate in baseball and softball through the Little League organization alone.[1] Children and families devote significant time, money, and effort into children's participation in youth sports. According to the Aspen Institute's 2019 state of play report, 71.8% of all kids between the ages of 6–12 participated in some type of sport in 2018, with families spending an estimated $693 on sports per child, with baseball specifically costing families $660 per child.[2] With high participation rates as well as increasing trends towards sport-specific specialization, there is also a non-trivial amount of pain and injury associated with overhead youth throwing sports such as baseball and softball. In 2019 it was reported that children on average play 1.8 different sports, versus 2011 when this number was 2.1, indicating that single sports specialization continues to be an ongoing problem.[2]

A recent study found that the most commonly diagnosed injury in baseball sustained in both practice and competition were strains/sprains, accounting for 39% and 44% respectively of the 52,889 total estimate of national injuries.[3] Hand/wrist (14.6%), shoulder (13.9%), and arm/elbow (11.6%) injuries accounted for a significant burden. Ultimately, 11.0% of injuries sustained in competition required surgery, compared to 2.8% of injuries sustained in practice. Demographics and injury breakdown were similar for softball injuries; however, total softball injury rates were significantly greater than baseball injury rates (RR 1.38, 95% CI 1.12–1.71).[3] Additionally, Trofa et al published data from baseball injuries presenting to US emergency rooms between 2006–2016 revealing the overall incidence to decrease by 11.7% during that time period. However, these injuries still accounted for an estimated 54,777 injuries per year.[4] The most common sites of injury were the upper extremity (36.3%) and face (26.2%), and the most common diagnoses were contusion (26.8%), fractures (23.6%), and strains/sprains (18.7%). The data revealed elbow injuries to have increased over that time period, accounting for 17.7% of total injuries. The only mechanism of injury to have increased in correlation with the increase in elbow injuries in this data set was throwing, further emphasizing the role of throwing as a source of potential injury in youth athletes.

Multiple epidemiologic studies published within the last 2 years reveal the increasing role of overuse in shoulder and elbow injuries in youth throwing sports. Previous publications estimated overuse in adolescent athletes to be the cause of 50% of all injuries in this population.[5] Saper et al's 2018 publication of injuries from 2005–2015 in high school baseball players, showed that shoulder and elbow injuries in this population were caused by overuse in 71.3% and 73.9% of cases, respectively.[6] Factors associated with increased risk for shoulder and elbow injury included: increased height, increased pitch velocity, pitching for multiple teams, and pitching through fatigue, with factors related to overuse and fatigue having the strongest contribution to risk.[7,8] This is consistent with the current body of literature surrounding burnout and overuse injuries in youth sports and

the relationship to early sports specialization and excessive sport volumes.[9] Even "healthy" young baseball players report arm or shoulder pain at alarmingly high rates; in a 2013 survey of summer league players with an average age of 15.2 years, 74% reported playing with arm pain or fatigue.[10] In the same survey of summer league youth baseball players, 46% reported being encouraged to play even though they were having pain.[10] A 2010 prospective cohort study from Lyman et al of 298 youth league pitchers conducted over two seasons revealed 26% and 32% of players reported elbow and shoulder pain respectively.[11] Risk factors for reporting elbow pain included playing baseball in multiple leagues, decreased self-satisfaction, arm fatigue during play, and throwing fewer than 300 or more than 600 pitches during the season.[11] Risk factors for reporting shoulder pain included decreased self-satisfaction, arm fatigue during pitching, throwing greater than 75 pitches in a game, and throwing fewer than 300 pitches during the season.[11] Additionally, Major League Baseball (MLB) has funded a large amount of research to form the "Pitch Smart" guidelines to help prevent youth injury with regards to innings pitched and rest times after throwing (**Table 1**).[12] Additionally, this research has shown that pitchers who exceed 100 innings in one season are up to 3.5 times more likely to sustain injury.[13]

Table 1. MLB Pitch Smart Guidelines for Youth Pitchers[12]

Age	Daily Max (Pitches in Game)	0 Days Rest	1 Days Rest	2 Days Rest	3 Days Rest	4 Days Rest	5 Days Rest
7–8	50	1–20	21–35	36–50	N/A	N/A	N/A
9–10	75	1–20	21–35	36–50	51–65	66+	N/A
11–12	85	1–20	21–35	36–50	51–65	66+	N/A
13–14	95	1–20	21–35	36–50	51–65	66+	N/A
15–16	95	1–30	31–45	46–60	61–75	76+	N/A
17–18	105	1–30	31–45	46–60	61–80	81+	N/A
19–22	120	1–30	31–45	46–60	61–80	81–105	106+

HISTORY AND PHYSICAL EXAM

Obtaining a thorough history and physical exam can accurately diagnose many of the pathologies noted above. Patient sex, age, sport and position(s) played, number of teams, seasons played, and pitch counts are all crucial for formulating a differential. Obtaining a thorough "throwing history" of when the athlete experiences pain is also crucial to making the correct diagnosis. For players who are pitchers, it is important to inquire about what types of pitches they throw ((2-seam/4-seam FB, curve, changeup, other off-speed pitches), as well as when the pain occurs, where the pain occurs, how often they pitch, and how many months of the year they pitch. (**Appendix 1.** Sample patient intake form used in our practice to obtain a throwing history)

SKELETALLY IMMATURE INJURIES

Medial epicondyle apophysitis

Also known as Little Leaguer's elbow, medial epicondyle apophysitis is an example of a traction apophysitis which occurs over the medial aspect of the elbow. The medial epicondyle apophysis is the last primary ossification center to close in the elbow, closing between the ages of 15–16 in most individuals.[14] This is significant because the ulnar collateral ligament (UCL) complex of the elbow which provides primary stabilization in the elbow during throwing originates here.[15]

Medial epicondyle apophysitis occurs due to chronic valgus forces during throwing, with the greatest prevalence occurring in players aged 11 to 12 years old.[16] Sixty-eight percent of players who develop this injury report a history of elbow pain.[16] Although it may not always be symptomatic, a 1965 radiographic study of 162 baseball players aged 9–14 by Adams et al reported all 80 pitchers included displayed some degree of traction related change to the medial epicondyle.[17]

Medial epicondyle fractures

Fractures to the epiphysis of the medial epicondyle lie on the extreme end of the spectrum of traction injuries to the area and have a characteristic acute presentation. Osbahr et al described the presentation in a 2010 case series detailing 8 skeletally immature baseball players, all of whom described an acute pop with significant medial elbow pain while throwing.[18] Median time to return to play was 7.6 months, and 3 of 8 players had greater than 5mm of displacement and underwent operative management with open reduction and internal fixation. Medial epicondyle fractures are typically diagnosed on radiographs of the elbow after this characteristic presentation, and treatment depends on the degree of displacement seen on imaging, the mechanism of injury, and the patient's overall activity level (**Figure 1**).

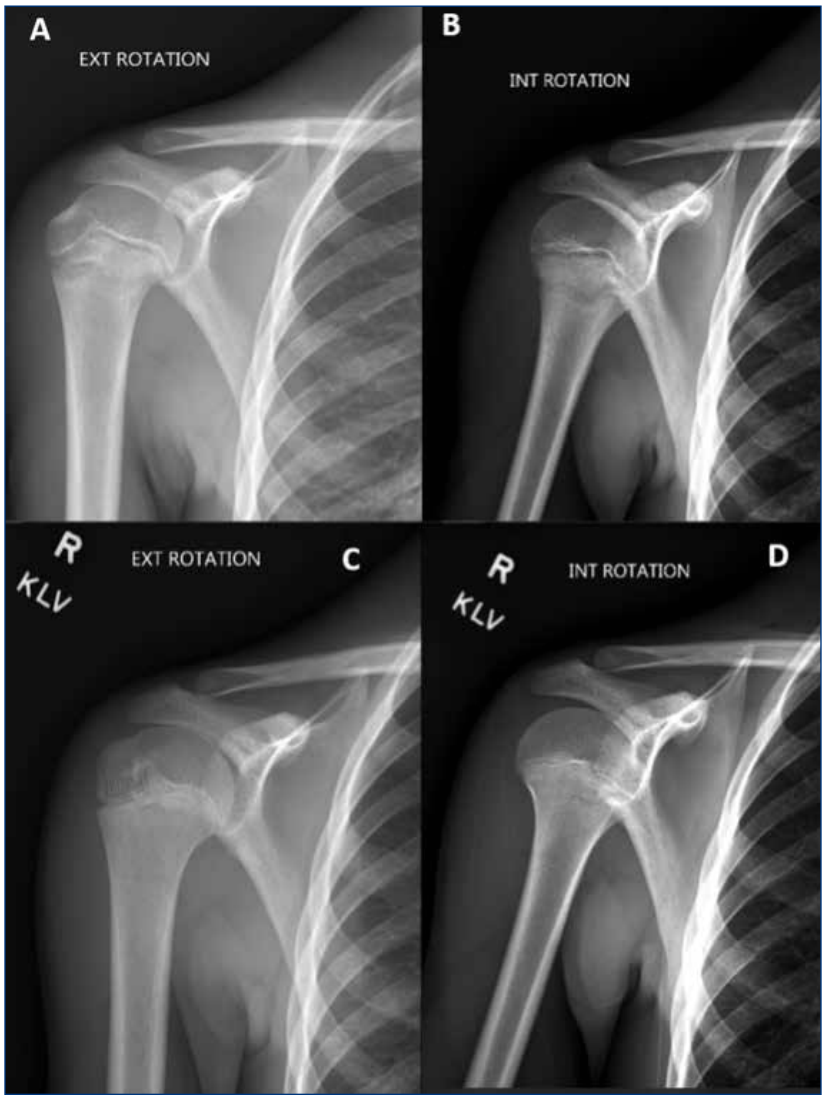
Olecranon apophysitis and stress fractures

The olecranon is also exposed to the stress of throwing and triceps contraction during the acceleration phase generates force directly perpendicular to the olecranon apophysis (**Figure 2**).[19] Chronic forceful contractions due to throwing lead to a similar spectrum of injury as medial epicondyle apophysitis, depending on the degree of stress overload and degree of epiphyseal closure. In adolescents with an immature apophysis, olecranon apophysitis may develop. As the apophysis closes, the risk of avulsion fracture and stress fracture increases. Concurrent ulnar collateral ligament (UCL) injury has been identified as a risk factor for olecranon stress fractures in throwers and there is a significant overlap with medial epicondyle injuries[20] – both can occur with the common pathophysiological driver of chronic overuse. Patients classically present with pain specifically over the olecranon, worsened during the follow-through phase of throwing, and associated with posterior elbow swelling and decreased range of motion.

Figure 1.
[**A**] AP and [**B**] Internal oblique view of displaced medial epicondyle fracture. The internal oblique view allows for determination of the maximal amount of displacement. [**C**] Post-operative image following open reduction internal fixation of the fracture with screw and washer.

Figure 2. T2 weighted MRI in a 15-year-old catcher showing enhanced signal within the olecranon representing olecranon stress reaction (arrow).

Figure 3. [**A,B**] External and internal rotation anteroposterior views demonstrating widening of the proximal humeral physis consistent with Little Leaguer's shoulder at the time of diagnosis, and [**C,D**] following 10 weeks conservative management with throwing holiday.



Little Leaguer's shoulder: Proximal humeral epiphysiolysis

First described as "Little Leaguer's Shoulder" (LLS) by Dotter in 1953[21], LLS refers to traction apophysitis of the proximal humeral growth plate, also associated with overuse (**Figure 3**). Maximal external rotation torque occurring during the arm-cocking phase of throwing leads to shear stress on the proximal humeral epiphyseal cartilage leading to the development of LLS[22]. Patients typically describe chronic worsening pain in the throwing shoulder, occasionally even with simple lifting of the arm, which is improved by rest. Physical exam reveals tenderness over the posterior aspect of the proximal humerus in congruence with the location of the growth plate, with or without swelling.

Figure 4.
[**A**] AP X-ray demonstrating lucency (row) in the capitellum consistent with capitellar OCD. [**B**,**C**] Sagittal cuts from T2 weighted MRI demonstrating increased uptake in the subchondral bone of the capitellum seen in patients with OCD lesions.

Patients may have decreased range of motion and muscle weakness due to pain. Radiographs demonstrate widening of the proximal humeral epiphysis consistent with a traction apophysitis.[23]

Capitellar Osteochondritis Dissecans

Osteochondritis dissecans (OCD) of the radiocapitellar joint is a distinct clinical entity, likely related in pathophysiology to other disorders of endochondral ossification, most notably Legg-Calve-Perthes disease. Capitellar OCD refers to the non-inflammatory degeneration of subchondral bone most commonly in the capitellum, related to excessive compressive forces and repetitive microtrauma most common in overhead and throwing athletic endeavors.[24] Patients with capitellar OCD typically present with a history of chronically worsening lateral elbow pain (relieved by rest), clicking, and flexion contractures in severe cases. Physical exam most commonly finds tenderness over the capitellum and lateral elbow with the elbow maximally flexed to best palpate the capitellum. Imaging of suspected OCD should include X-ray of the elbow and/or advanced imaging modalities such as MRI if a high clinical suspicion, as early OCD is commonly missed on plain radiographs (**Figure 4**).[25]

SKELETALLY MATURE INJURIES

Valgus extension overload syndrome

Valgus extension overload syndrome (VEOS) refers to a constellation of injuries in all three compartments of the elbow (medial, lateral, and posterior) directly related to the repetitive stress of the throwing action. The most notable finding is the development of posteromedial osteophytes, osteochondritis, and chondromalacia within the olecranon fossa.[26,27] VEOS typically presents with posteromedial elbow pain, possibly preceded by a decrease in pitch velocity and accuracy. Pain is typically worse during the follow-through phase of throwing when the elbow reaches terminal extension, and in cases with significant osteophyte burden, patients can report the sensation of locking or catching.

Physical exam typically reveals pain over the posteromedial portion of the olecranon and is sometimes associated with crepitus. Provocative maneuvers can reveal pain with terminal extension, and the elbow must also be examined for valgus instability and UCL injury. The valgus extension overload test is performed by holding a valgus force on the elbow and rapidly extending the elbow. If this produces posteromedial pain about the olecranon, this is deemed a positive test. Plain radiographs are typically diagnostic, revealing osteophytes and loose bodies.

Ulnar collateral ligament tear

The ulnar collateral ligament (UCL) complex of the elbow is the primary static stabilizer against the extreme valgus stresses occurring in the overhead throwing motion, and thus is prone to overuse injury.[28] The UCL is subject to chronic microtrauma from throwing, leading to inflammation and structural weakening, which can predispose to an acute tear. Acute UCL tear classically occurs during play with an acute popping sensation and the development of pain and ecchymosis over the medial elbow. Physical exam is generally notable for tenderness over the medial elbow and valgus instability in complete tears. Increased valgus laxity can be indicative of injury to the UCL when compared to the contralateral side. It is important to assess for medial epicondyle apophysitis or fracture, which can be ruled out with absence of pain on resisted wrist flexion and normal X-rays. MRI is the most sensitive and specific imaging modality (**Figure 5**), although ultrasound can also be useful in initial assessment.[29]

Shoulder Instability

Shoulder instability is a spectrum ranging from frank dislocation to repetitive subluxation events, and is often more associated with collision sports such as American football,

Figure 5.
T2 MRI indicating chronic UCL deficiency and thickening in the setting of an ulnar collateral ligament tear.

and rugby. However, the repetitive nature of pitching in baseball puts players at risk for shoulder instability due to microtrauma and microinstability.[30] Injuries to the labrum, both anterior-inferior (Bankart lesion), and superior (SLAP lesion), are common among baseball players, especially pitchers.[31] It can also occur in a traumatic fashion after a headfirst slide, or dive for the baseball while fielding when the arm is in the vulnerable position of abduction an external rotation.

SLAP tears (superior labral anterior posterior)

Superior labral anterior-posterior (SLAP) tears in throwing athletes are thought to be due to the extreme external rotation of the throwing movement, with a "peel-back" mechanism involving the biceps-labral junction.[32,33] SLAP tears cause a vague aching pain localized to the posterosuperior joint line. Patients typically report a loss of velocity on pitches, and pain worse in the late-cocking phase of throwing. SLAP tears can be very challenging to diagnose on exam. Multiple maneuvers have been described, including the Jobe, O'Brien, Yergason, active compression, shear and Speeds tests. However, no single test has proven reliable in diagnosing a SLAP tear and therefore a battery of tests should be performed during evaluation.[34,35]

IMAGING

Plain radiographs should be obtained as part of the standard workup. Three views (true anteroposterior, scapular Y, and axillary lateral) of the shoulder and two views (anterior-posterior and lateral) of the elbow are adequate for most shoulder and elbow injuries, respectively. An anteroposterior (AP) of the shoulder may show humeral physeal widening on internal and external rotation views, which is indicative of LLS (**Figure 3**). Comparison views of the contralateral asymptomatic shoulder can be useful to confirm the diagnosis.[23]

Elbow radiographs may reveal olecranon stress fractures, olecranon apophysitis, or medial epicondyle fractures. Displacement of medial epicondyle fractures can be determined by an internal rotation view; however, this is subject to interpreter variability (**Figure 1**).[36] Similar to the shoulder, comparison views may be indicated if the diagnosis is in question. A capitellar OCD may also be visible on radiographs, but this is not often the case and typically requires advanced imaging for diagnosis and management.[25] Computed tomography (CT) is not frequently obtained in the workup for adolescent thrower pathologies. In the case of posteromedial impingement from VEOS, a CT scan of the elbow with 3D reconstruction can be useful to identify osteophytes or fragments from the olecranon tip for surgical planning of debridement.

Magnetic resonance imaging (MRI) is indicated in certain situations. When there is high suspicion for UCL injury, an MRI of the elbow should be obtained. OCD lesions of the capitellum can be graded based on MRI findings, and treatment is directed based on the size and stability of the lesion seen on MRI. Stress fractures of the olecranon may also be identified on MRI. Compared to a conventional MRI, an MR arthrogram increases the sensitivity and accuracy of diagnosing SLAP tears, as well as Bankart lesions and other labral pathology associated with shoulder instability.[37]

TREATMENT

Nonoperative treatment with temporary cessation of aggravating activities is the mainstay of treatment for the majority of overuse injuries, especially in the skeletally immature athlete. Treatment is primarily non-operative, involving rest from throwing. Players affected by medial epicondyle apophysitis are often those who are playing the most and at the highest level in youth sports, and may need an enforced rest period of 4–6 weeks, as well as symptomatic treatment with non-steroidal anti-inflammatory drugs (NSAIDS), ice, and/or work with a physical therapist to improve biomechanics[38].

Medial epicondyle avulsion fractures, particularly those

with intraarticular incarceration, ulnar nerve entrapment, open fracture, or instability associated with a dislocation are managed operatively. However, there is significant debate regarding the degree of displacement that indicates operative intervention.[39–41] Some of this can be accounted for by the poor interobserver reliability in measurement on radiographs.[42] Classically, displacement up to 5 mm can be treated nonoperatively. Some authors advocate for stricter indications for surgery in the overhead athlete, yet no prospective randomized study has validated appropriate indications.[39]

Whether simple apophysitis or stress fracture, treatment is generally non-operative with rest from throwing, NSAIDs, ice, and physical therapy for a period of 4–6 weeks. Olecranon stress fractures should be immobilized for 4 weeks in 20° of flexion, with rehabilitative exercises starting at 6 weeks.[43] Olecranon stress fractures that fail to improve with nonoperative measures may benefit from stabilization and can result in >90% return to play rates.[44] High rates (>70%) of concomitant UCL injuries have been noted in these patients.[20]

In the case of LLS, treatment is exclusively initiated with non-operative management focused on rest of the throwing shoulder and successfully results in full return-to-play in 90–95% of players.[23,45,46] A longer time from onset of pain until rest initiation and the presence of range of motion deficits is associated with worse outcomes.[47,48] One study found that for patients with LLS, a 3-month period of eliminating throwing, coupled with physical therapy for strengthening has been shown to return 91% of patients to play.[23]

Stable capitellar OCD lesions should initially be managed conservatively in skeletally immature athletes, as these may heal during maturity of the epiphysis.[49] However, a skeletally mature athlete with an unstable symptomatic OCD lesion can benefit from early arthroscopic debridement, microfracture, and in some cases osteochondral grafting depending on size of the lesion.[50]

While skeletally mature injuries may be more resistant to non-operative management, it often is still the first line of treatment in the youth athlete. Posterior impingement caused by valgus extension overload is typically seen in older athletes, as younger patients have not yet formed osteophytes. Non-operative management, with a prescribed decrease in throwing activity and physical therapy is paramount, and symptom control achieved via ice, NSAIDS, and intra-articular glucocorticoids is used as needed. Arthroscopic or open debridement can be indicated in patients who fail non-operative management. Park et al. identified a high rate of posterior olecranon tip fractures in adolescents with VEOS and treated them successfully with arthroscopic debridement after a trial of nonoperative management.[51]

Management of UCL tears in the adolescent athlete can be controversial; however, initial management for young athletes is generally non-operative, with rest, ice, bracing, and physical therapy for 6 weeks before a gradual return to activity.[52] Elite athletes and those athletes who fail non-operative management can be considered for orthopedic referral and UCL repair or reconstruction.[53] Although the gold standard surgical management for UCL tear in competitive throwers is reconstruction, as initially described by Jobe, recent reports of successful repair have shown promising results in high school and college athletes.[53,54] The indications are limited to young patients with acute simple tears, but this technique may allow faster return to sport with no donor site morbidity.

Athletes who experience first time instability in season will often desire to return to play following non-operative management.[55] The literature has shown that frequently an athlete may return to play following approximately 3 weeks of rehabilitation; however, it is crucial to counsel these athletes regarding the risk of recurrence.[55] Recurrence can be especially high in younger athletes. Gigis et al. compared treatment of first-time dislocaters aged 15–18 that underwent conservative versus arthroscopic management.[56] Among the 27 patients managed conservatively, 19 (70.3%) experienced recurrent dislocation.[56] It is important to note that this study included patients with traumatic dislocation and their individual sport was not specified. While bracing may help limit the risk of repeat instability events, it can be severely limiting, especially in the case of throwers.[57] If non-operative management does not provide symptomatic relief and return to the same level of play, operative treatment should be performed to address the underlying pathology (either arthroscopic or open, based on the extent of the pathology present).

Likewise, SLAP tears should initially managed with focused rotator cuff and periscapular strengthening. In general, operative management is utilized for patients that fail to improve with nonoperative treatment. However, there are specific indications for more acute operative intervention for some of these injuries. SLAP tears are uncommon in adolescents, yet when they occur, nonoperative management with rotator cuff strengthening and scapular stabilization is successful in returning the majority of patients to sport. When arthroscopic repair is performed for those failing nonoperative treatment, the successful return to overhead throwing is highly variable, ranging from 22-64%, which is significantly worse than other athletes.[58]

PREVENTION

Prevention of adolescent throwing injuries may be the most important aspect of managing these patients. The majority of these injuries occur from repetitive overuse and throwing while fatigued.[8,59] Youth pitchers carry a 5% chance of sustaining a serious throwing injury with 10 years of competitive throwing. Literature suggests that pitching at high velocity, arm fatigue, pitching on multiple teams, increased pitch count per game, and participation in showcases are risks factors for injuries that require surgical intervention.[8,60,61]

Pitching more than 100 innings per year is also associated with a 3.5 times increased risk of injury. Position played has been shown to be a risk factor for injury, as pitchers have increased incidence of injuries compared to position players. It was also shown that pitchers who also played catcher were injured more frequently.[62] A nationwide survey study of youth and adolescents found that certain factors, including pitching on consecutive days, pitching on multiple teams with overlapping sessions, and pitching multiple games in one day increased odds of experiencing pitching-related arm pain.[63] Overall, overuse, pitching at high velocity or in a more competitive environment, and pitching while fatigued increase the injury risk for these young athletes. It has been shown that athletes with throwing-related pain have weakened posterior shoulder musculature, especially in their trapezius and supraspinatus, compared to increased strength in their internal rotators.[64] In professional pitchers, preseason weakness in external rotation, specifically in the supraspinatus, was associated with increased risk of throwing-related injury that ultimately necessitated operative intervention.[65] Posterior shoulder girdle muscle strengthening may be a way to help prevent throwing-related injury, especially in young athletes.[64] Working on neuromuscular control and strength of an athlete's core, scapula, and lower extremity is important in prevention of injury and postoperative rehabilitiation.[66,67] A focus on developing a balanced kinetic chain during the throwing motion to promote an effective and fluid coordination of the entire body is extremely important to offload excessive stress on the shoulder and elbow joints and prevent injury while enhancing performance.[68] The American Academy of Orthopedic Surgeons and Little League has made recommendations for the maximum amount of innings pitched in one game and one week based on age, the number of days of rest needed based on the amount of pitches thrown in the previous game, the maximum number of pitches in one game, and the appropriate age when different pitches should be learned (change-up, curveball, slider, etc.).[67] It is also important to educate players, parents, and coaches, and conduct regular screening and monitoring of those with at risk.[63,69]

CONCLUSION

Overuse injuries in the adolescent throwing athlete can be very common, and typically affect the shoulder and elbow. Providers of all types are likely to see these injuries, but primary care physicians are often the first line of defense in the prevention and diagnosis of this long list of potential issues. A thorough throwing history and physical exam are extremely important in making the correct diagnosis, with early referral to physical therapy and orthopedic surgery for proper management. By better educating players, coaches, and parents, these injuries can potentially be avoided, helping maximize playing time and minimize the time needed for operative intervention.

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Management of Anterior Shoulder Instability for the In-Season Athlete

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ABSTRACT

Management of in-season anterior instability poses a unique challenge to providers as they are faced with the conundrum of helping an athlete return to play as quickly as possible, while minimizing the risk of recurrent instability and progressive damage to the glenohumeral joint. The decision for early return to play versus in-season surgery ultimately is a collective decision-making process between the athlete, provider and training staff. However, it is the physician's obligation to properly counsel the athlete on the risks of early return to play following conservative management. Apart from athletes who are in the last season of their career or have other extenuating circumstances, requiring return to play (RTP) in the same season (i.e. upcoming championship or combine), given the high risk of recurrence in athletes managed conservatively, physicians should strongly encourage early surgical stabilization. Surgical management of instability most commonly includes arthroscopic Bankart repair and capsulorrhaphy, however open Bankart repair should be considered in high-risk athletes (i.e. contact athletes, recurrent instability, sub-critical glenoid bone loss). In athletes with critical glenoid bone loss an osseous augmentation procedure should be performed, such as the Latarjet procedure.

INTRODUCTION

Traumatic anterior shoulder instability is a common injury in athletes at all levels, and represents a spectrum of injury ranging from micro-instability to subluxation to dislocation.[1,2] Anterior displacement of the humeral head most commonly results in partial loss of the native glenohumeral contact surfaces, also referred to as a subluxation which comprises up to 85% of primary instability events. Glenohumeral dislocations, however, result in complete loss of contact between the glenoid and humeral head articular surfaces, requiring reduction. Dislocations are less frequent when compared to subluxation and only comprise 15% of new instability events.[3,4] In-season instability poses a unique challenge given athletes' desires to expeditiously return to play (RTP); however, such a vulnerable population must be counseled on the risk of recurrent instability and further damage to the joint, with up to 73% of athletes able to RTP in the same season experiencing a recurrence of instability.[5] Despite the ubiquity and gravity of these injuries, the management of in-season athletes currently lacks consensus. Currently, most clinicians agree that each athlete requires an individualized approach with a particular emphasis placed on completion of pain-free, sport-specific activities prior to resuming competition.[6] Regardless of one's management preferences, the formulation of an appropriate strategy demands an intimate understanding of pathoanatomy, an ability to assess the risk of recurrence, and familiarity with the various treatment options available to the athlete. This review aims to provide insight into current trends in the management of in-season athletes with anterior instability while simultaneously serving as a guide for team physicians tasked with caring for these athletes.

HISTORY

The evaluation of anterior shoulder instability begins with a thorough history, with particular attention paid to prior instability events which could include dislocation, subluxation or both. Additionally, it is important to determine the athlete's type and level of sports activity, time in their competitive season, and future expectations for sports participation. Individuals presenting after a traumatic event should be asked about the mechanism (contact vs non-contact), the severity (subluxation vs dislocation), and the direction of displacement. For those presenting in the absence of a traumatic event, it is important to determine the context in which their sensation of instability occurred. Anterior instability is most often symptomatic when shoulder abduction is coupled with external rotation. Given the shoulder's proximity to the brachial plexus, physicians should also inquire about neurologic symptoms. While sensorimotor deficits are present in less than 6% of all anterior shoulder dislocations, this risk increases to almost 8% if the injury is associated with a fracture of the greater tuberosity or a tear of the rotator cuff. When a neurological injury is present, the structure most commonly involved is the axillary nerve.[7]

PHYSICAL EXAM

Examination of a patient on the field or in the locker room with suspected acute instability should always begin with

observation. An acute anterior shoulder dislocation will typically demonstrate a loss of shoulder contour, a palpable anteroinferior humeral head, and the arm held in adduction and internal rotation. Next, the patient's neurovascular status and range of motion should be assessed. It is critical to note the importance of performing a complete neurovascular exam before and after any reduction maneuver as there is a risk for iatrogenic injury. If at this time the athlete's presentation is concerning for an acute dislocation, it is not unreasonable to acutely attempt a reduction. However, it is important to note that if there is any concern for concomitant fracture, reduction should be delayed until appropriate radiographs have been obtained and the reduction can be performed in a controlled environment under sedation to prevent further displacement of the fracture. Additionally, an on-field reduction should not be attempted more than once if not initially successful as such patients likely will require analgesia and/or sedation. Following reduction, active and passive range of motion as well as a neurologic exam should be re-evaluated

On the other hand, patients presenting to the office with a history of recurrent instability may have more subtle shoulder asymmetry and physical exam findings. In addition to the typical physical exam which should include inspection, palpation, ROM and strength testing, in this population, provocative tests are frequently performed to assess the type and degree of instability including but not limited to, the apprehension test, relocation test, sulcus sign and the surprise test. The apprehension test can be performed with the patient laying supine at the edge of the bed to support the scapula. The arm is brought into 90 degrees of abduction and subsequently external rotation. As the arm is brought into increasingly more external rotation the patient, if positive, will have subjective feelings of instability and become apprehensive with further external rotation. The relocation test can be performed in this position by placing a posterior directed force on the proximal humerus. If the patient endorses relief of the apprehension with this maneuver, then the test is considered to be positive. The surprise test then includes abrupt discontinuation of the posterior directed force on the proximal humerus, as performed during the relocation test resulting in immediate return of apprehension if positive. Of note, when apprehension, rather than pain, is used as the criteria for these special tests the positive predictive value of each tests increases. While the surprise test is considered to be the most accurate special test, when the apprehension, relocation, and surprises tests are all positive, the positive predictive value for anterior instability approaches 94%.[9] Finally, patients should be assessed for generalized hyperlaxity using the Beighton Scale. Patients with scores of ≥2 are 2.5 times more likely to experience shoulder instability and may be at risk for recurrent dislocations.[10,11]

PATHOANATOMY

Athletic injuries to the shoulder can cause derangement to the normal glenohumeral anatomy. Dislocation or subluxation events can harm the bony, ligamentous and other soft tissue structures of the shoulder. The most common pathology to the shoulder after a first-time instability event is a tear of the anterior inferior glenoid labrum, also known as a Bankart lesion. This is found in 97% and 96% of all patients sustaining a first-time anterior dislocation and subluxation event, respectively.[12] Furthermore, anterior shoulder dislocations can cause fracture of the anterior-inferior glenoid, commonly termed a "Bony Bankart Lesion."[13,14] Similar to Bankart lesions, the anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesion can occur which involves stripping and displacement of the anterior scapular periosteum in a sleeve-like fashion. This differs from a Bankart lesion, which is a tearing of that scapular periosteum below the labrum with the lesion scarring in a medialized position. ALPSA lesions have been found to have a higher rate of failure after surgical repair.[15,16] While a Bankart lesion occurs when the labrum is torn from the glenoid with concomitant glenohumeral ligament stretching, the glenohumeral ligaments may be torn from the humerus and are referred to as HAGL lesions (humeral avulsions of the glenohumeral ligament).[6] While HAGL injuries were previously thought to be rare, occurring in less than 10 % of patients sustaining an anterior instability event, certain populations may be at greater risk. Recent data by Owens et al. demonstrates that female athletes may be at increased risk for HAGL lesions, with 25% of female collegiate athletes suffering from anterior instability having evidence of a HAGL lesion on post-injury imaging.[17,18] Vigilant surveillance for HAGL lesions is extremely important, as they can often be missed, and have been shown to be a culprit for recurrent instability.

As mentioned previously, anatomic changes in the bony architecture can result from anterior instability and increase one's risk for recurrent shoulder instability. This can occur with acute fracture of the anterior inferior glenoid at the time of the index instability event or can be a more chronic, progressive process due to recurrent subluxation/dislocation or possibly recurrent micro-instability leading to attritional glenoid bone loss (GBL). Bone defects in the humeral head can also occur after dislocation or subluxation events. These humeral head injuries are termed Hill-Sachs lesions, and occur with impact of the posterolateral humerus into the glenoid. As expected, patients with complete dislocation, when compared to subluxation, are at an increased risk for osseous defects to the glenoid and humeral head. For example studies of first-time dislocators have shown 22% of patients to have evidence of osseous glenoid defects and 90% with Hill-Sachs lesions following an anterior dislocation.[19] Conversely, in a study of patients with first-time subluxation events, only 11% and 7% of patients have evidence

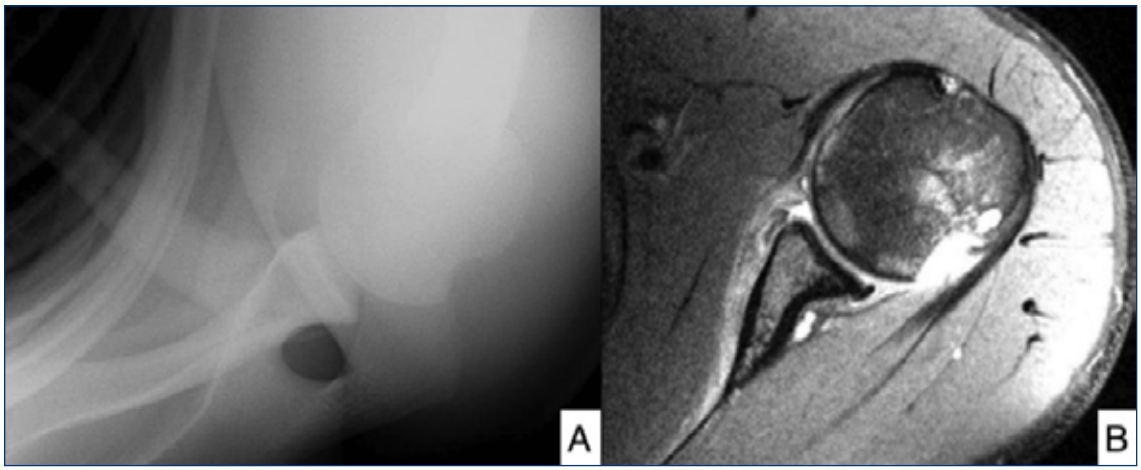


Figure 1. Axillary lateral radiograph [**A**] and axial MRI [**B**] demonstrating a large Hill-Sachs lesion with concomitant anterior glenoid bone loss.

Figure 2. Anteroposterior view of the left shoulder with evidence of anteroinferior dislocation concomitant bony Bankart and Hill-Sachs lesions.

of glenoid and humeral head osseous defects, respectively.[12] Bipolar bone loss, which is a term used to describe an injury when there is simultaneous GBL and a concurrent Hill-Sachs lesion, can also be an important cause of recurrent anterior stability, and must be appropriately identified and addressed. **(Figure 1)**. Furthermore, when a Hill-Sachs lesion is deemed to be "off-track", resulting in engagement of the humeral head defect with the glenoid, the risk of instability substantially increases.[20] Those which are "on-track" may override the glenoid surface and be at less risk of an instability moment.[6,20]

IMAGING

Imaging should begin with plain radiographs of the shoulder after the injury. Three views of the shoulder are obtained, with a true anteroposterior, an axillary or West Point view, and a scapular Y view **(Figure 2)**. Attention should be paid to ensuring a concentric reduction of the glenohumeral joint and observing for any fractures. Computed tomography (CT) scans are useful to assess the bony architecture of both the glenoid surface and the humeral head. More recently 3D CT scans have been helpful in more precisely measuring GBL.[21] Magnetic resonance imaging (MRI) is the best study for evaluating the soft tissue structures in the shoulder joint, including the rotator cuff, labrum, other ligamentous structures, as well as looking at bony edema. MRI arthrogram is more sensitive in evaluating and characterizing labral tears and for visualizing HAGLs.

IN-SEASON MANAGEMENT

The management of shoulder instability in an in-season athlete is highly individualized to the athlete and situation and involves a collective-decision making process with the athlete (and often parents), the provider, and the training staff. When counseling an athlete, it is important to take into consideration many factors including whether the athlete has a history of previous dislocations, the timing of the in-season injury, the sport and position played, the level of play, the handedness of the athlete, the risk of recurrence, and the long-term goals of the athlete. It is paramount that the athlete balances their desire to RTP as soon as possible with the high risk of recurrent instability and potential for further damage to the glenohumeral joint. Given this risk of cumulative damage to the joint with subsequent instability events, the current authors strongly encourage in-season surgery even for first-time dislocators, especially for athletes early or in the middle of their career. However, early return is not unreasonable under certain circumstances that would compel an athlete to desire rapid RTP during the same season, such as the injury occurring during the last season of their career or prior to an upcoming championship.

NONOPERATIVE MANAGEMENT

Depending on the pathology of the dislocation, nonoperative management may be desired for the in-season athlete

as it allows for quicker RTP. This is a more reasonable trial if advanced imaging reveals no osseous abnormalities and is limited to only labral injury. The goal of conservative treatment is a functional shoulder with equal rotator and scapular strength, and the ability to perform sport-specific activities without pain.

There is no standard post-reduction course, but in general there is a period of immobilization, followed by supervised rehab protocols. Immobilization can be in either internal or external rotation, and there is some debate as to the best position. A 2019 Cochran review of seven trials and 704 patients showed no conclusive benefit of immobilization in internal versus external rotation in regards to re-dislocation risk.[22] The length of immobilization is variable and oftentimes is physician and patient dependent; active in-season athletes may prefer a shorter period of immobilization to allow for quicker RTP. Paterson et al. concluded no statistical difference in instability for younger patients less than 30 years old, reporting the rate of recurrent instability to be 41% in athletes immobilized for 1 week or less and 37% for athletes immobilized >3 weeks(p=0.52).[23] Hovelius et al. looked at 257 patients prospectively after anterior traumatic shoulder dislocation and found no difference in recurrence rates between early mobilization versus immobilization for 3–4 weeks.[24] Following a period of immobilization, rehab treatment is generally aimed at strengthening peri-scapular muscles in addition to those involved with shoulder internal rotation and adduction, with the goal of an athlete being able to perform sport-specific activities with no apprehension or pain.[25]

Multiple studies have found nonoperative management to have a quicker RTP, with the cost of a high likelihood of recurrent instability events. Buss et al found 87% of athletes RTP with a mean of 10 days; however, they reported a significant recurrence rate of 1.4 per athlete-season.[26] A prospective study of 27 nonoperative versus 38 arthroscopically stabilized adolescent shoulders found a 70.3% recurrent instability rate compared to 13.1% in the operative group.[27] With such high recurrence rates associated with early RTP following non-surgical management of anterior instability, it is important that athletes fully understand the reparations of recurrent instability, which includes cumulative damage to the glenohumeral joint, specifically GBL. Alarmingly, a prospective study of collegiate athletes demonstrates an average GBL of 6.8% after a first-time dislocation which increased to a mean of GBL of 22.8% after a single episode of recurrence, suggesting critical GBL after only two episodes of instability.[28] With this amount of GBL after only 1 additional instability event, athletes must understand they are at risk for inferior outcomes after arthroscopic stabilization surgery and may require a more complex surgery to address the osseous defects.[29]

SURGICAL MANAGEMENT

Indications

Early surgical intervention, even in first-time dislocators, is highly recommended in athletes without an extenuating circumstance requiring rapid RTP. Furthermore, if an athlete is not able to perform sports-related activities after a period of brief immobilization and rehabilitation, immediate surgery should be recommended. Apart from athletes with a simple instability event, there are some associated injuries that are considered absolute and relative indications for immediate in-season surgery. These injuries include large rotator cuff tears, GBL (13.5% in collision athletes and 25% in non-contact athletes), bony pathology such as a proximal humerus fracture, and off-track Hill-Sachs lesions (**Table 1).**

Table 1. Indications for early surgery in athletes with anterior shoulder instability events

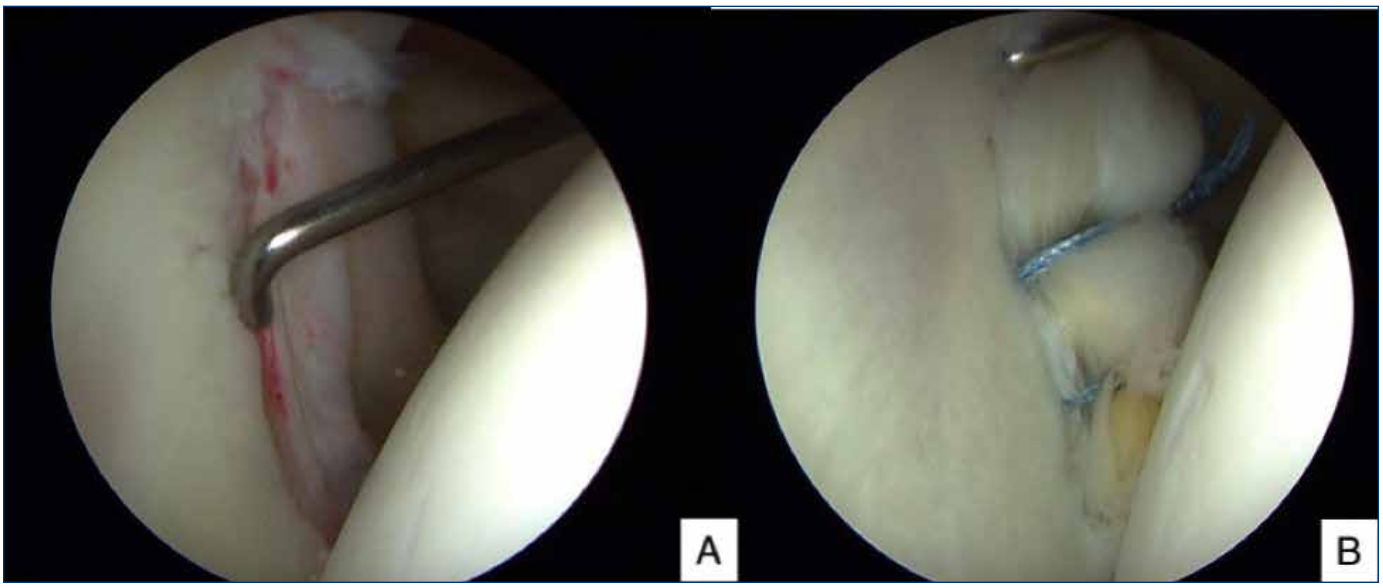
Indications for Early Surgery
Absolute Indications
• Glenoid bone loss >25%
• >50% rotator cuff tear
• Off-track Hill Sachs Lesions
• Irreducible dislocation
• Failed trial of rehabilitation
• Inability to tolerate shoulder restrictions
• Inability to perform sport-specific drills without pain or apprehension
Relative Indications
• Recurrent dislocations in same season
• Collision/contact athlete
• End of the season injury
• <20 years old
• Subcritical Glenoid bone loss > 13.5%

If surgery is deemed to be indicated for an in-season athlete, the surgical options that exist include arthroscopic versus open capsuloligamentous repair +/– a bony augmentation procedure. Conditions that are considered when determining the best method of shoulder stabilization include whether the athlete is a collision or contact athlete, history of recurrent instability and the presence of bony defects (i.e., GBL or Hill-Sachs lesion)

Capsulolabral Repair

Repair of the anterior-inferior labrum (Bankart repair) and capsulorrhaphy can be performed open or arthroscopically **(Figure 3)**. Historically, open repair was the procedure of choice due to early data which demonstrated decrease recurrence rates; however, as surgeons have become more facile at arthroscopy and there has been an improvement in both instrumentation and surgeon repair techniques, this belief has been disproved.[30–33] Given these recent findings,

Figure 3. Arthroscopic view from posterior viewing portal demonstrating a Bankart lesion before [**A**] and after [**B**] repair/capsulorrhaphy.



arthroscopic stabilization has increased dramatically, as it has the advantages of decreased pain, improved postoperative range of motion and faster rehabilitation allowing expeditious RTP.[32–34] More recent data has demonstrated low recurrence rates following arthroscopic Bankart repair with recurrence rates as low as 5% and RTP rates exceeding 90%.[35–37]

Despite this data, it is imperative to consider patient selection, as open Bankart repair may be favorable in a select cohort of patients who are at increased risk for recurrent instability after arthroscopic repair. This includes high-risk contact/collision athletes, athletes under 20 years old, athletes with known ligamentous laxity, subcritical GBL (13.5%–25%), or athletes that require a revision procedure.[29,37,38] Recent data has demonstrated recurrence rates of >50% in contact athletes undergoing arthroscopic stabilization procedures.[39] Conversely, Hennrikus et al. demonstrated only a 5% recurrence rate after open Bankart repair in 21 adolescent contact athletes.[40] Despite these findings, further research must be performed elucidating the role of open versus arthroscopic Bankart repair in high-risk athletes as the current studies which exist are limited by their small sample sizes and lack of prospective data.

Addressing Bony Defects

As discussed above, in addition to capsulolabral injury, anterior instability can also be associated with bony defects including both Hill-Sachs lesions and anterior GBL. It is imperative to address each of these pathologies to prevent recurrent instability. There has been a recent interest in the role that Hill-Sach's lesions, specifically those that engage with the glenoid (off-track lesions), may play in increasing the rate of recurrence with recent data demonstrating off-track HS lesions increasing one's risk of recurrent instability following arthroscopic Bankart repair by a factor of eight.[41] To address this, the arthroscopic *remplissage* was introduced which consists of capsulotenodesis of the posterior capsule and the infraspinatus into the HS defect which prevents engagement between the glenoid and humeral head defect.[42]

Following an anterior dislocation event, up to 90% of athletes may experience associated GBL, with the risk and amount of bone loss increasing with every episode of recurrence.[13] It is imperative that surgeons address GBL to decrease the risk of recurrence. While early data suggested glenoid augmentation when GBL exceeded a critical level of 25%, more recent data the critical level of GBL may be even lower in athletes at 13.5 %.[38] To address GBL, various autogenous and allogenic bone-grafting techniques exist. The most commonly performed technique is transfer of the coracoid to the anterior-inferior glenoid, restoring the osseous anatomy of the glenoid, acting as a bony buttress preventing anterior dislocation.[43] More recent techniques involve reconstruction using distal tibia or iliac crest allograft. However, these procedures are typically reserved for revision surgeries and are still lacking long-term prospective data.[44,45]

CONCLUSION

Anterior shoulder instability is extremely common in athletes and requires an in-depth knowledge of the pathoanatomy, natural history and the various management options related to these injuries. The optimal in-season management of athletes with a first-time shoulder instability event continues to be controversial. Ultimately the decision to perform immediate surgery versus RTP following a short course of rehab is a collective decision-making process and it is up to the physician to properly counsel the patient. If the athlete is under extenuating circumstances that require

rapid RTP (i.e., upcoming combine, post-season play) or is in the last season of their career, it is not unreasonable for the athlete to RTP during the same season. For all other athletes, especially athletes early in their career, and those at high risk for recurrence (young contact/collision athletes) or have a history of recurrent instability, immediate surgery is recommended to prevent further damage to the shoulder joint, which includes GBL and humeral head defects. In patients that do elect to have surgery, it is very important to look closely at the surgical candidate, including their risk for recurrent instability, whether they have associated defects about the glenoid and/or humeral head to determine the most suitable procedure. In athletes with pure capsuloligamentous injury and no history of previous instability or who are low risk for recurrent instability, arthroscopic Bankart repair with capsulorrhaphy is the procedure of choice. If an athlete is at high risk for recurrence, open Bankart repair should be considered. Additionally, if a patient has an off-track HS lesion, a simultaneous remplissage procedure should be performed. Finally, in the setting of critical (>25% GBL) or sub-critical (>13.5%) GBL in contact athletes, a glenoid osseous augmentation procedure should be performed to decrease the risk of recurrent instability.

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Bridge-Enhanced Anterior Cruciate Ligament Repair: The Next Step Forward in ACL Treatment

ERIN K. HAGGERTY, MD; STEPHEN E. MARCACCIO, MD; PAUL D. FADALE, MD; MICHAEL J. HULSTYN, MD; BRETT D. OWENS, MD

ABSTRACT

Anterior cruciate ligament (ACL) injuries are common in young and active patients. In this patient population, surgical treatment with an autograft tendon is recommended to reconstruct a new ACL. ACL reconstruction has a high patient satisfaction, improved patient reported outcomes and allows young patients to return to an active lifestyle, including sports. However, long-term follow-up shows these patients are at higher risk for degenerative arthritis, frequently at a young age. Recent research has focused on re-investigating the utility of performing an ACL repair rather than a reconstruction in the hopes that maintaining a patient's native ligament may not only restore knee stability, but provide improved knee kinematics and lessen the risk of late osteoarthritis and also limit donor site morbidity from autograft harvests. Historically, patients undergoing ACL repair suffered poor outcomes due to issues with intra-articular healing of the ligament; but now, with new bioengineering techniques, bridge-enhanced ACL repairs may provide a feasible alternative in the treatment of ACL injuries.

INTRODUCTION

ACL Tears and the Evolution of Surgical Treatment

Anterior cruciate ligament injuries are common and their incidence continues to increase.[1] The literature reports that approximately 200,000 ACL injuries occur per year in the United States[2,3,4] and that the incidence of reconstructions has increased from 86,687 in 1994 to 129,836 in 2006.[5] Unlike the medial collateral ligament (MCL), the ACL is unlikely to heal on its own due to both its intra-articular location and differences in stem cell characteristics.[6]

Because of this poor healing potential, it has consistently been shown that nonsurgical management of ACL tears in active patients results in recurrent episodes of knee instability and poor clinical outcomes. In fact, a delay of more than twelve months before reconstruction is associated with meniscal and chondral injuries, and a relatively longer time from injury to surgery results in the development of radiographic knee osteoarthritis.[7]

The first surgical treatment for ACL tears began in the early 1900s with attempted open primary repair in which surgeons sutured the torn ends of the ACL back to one another.[11] However, initial follow-up studies demonstrated both high failure rates (as high as 50–90%), recurrent instability, and low-patient satisfaction, therefore pushing surgeons to pursue ACL reconstruction rather than direct surgical repair.[8,9,10] ACL reconstruction has become an effective method for restoring knee stability in athletes and has demonstrated excellent clinical results, with some studies showing that over 89% of athletes return to previous level of competition.[11]

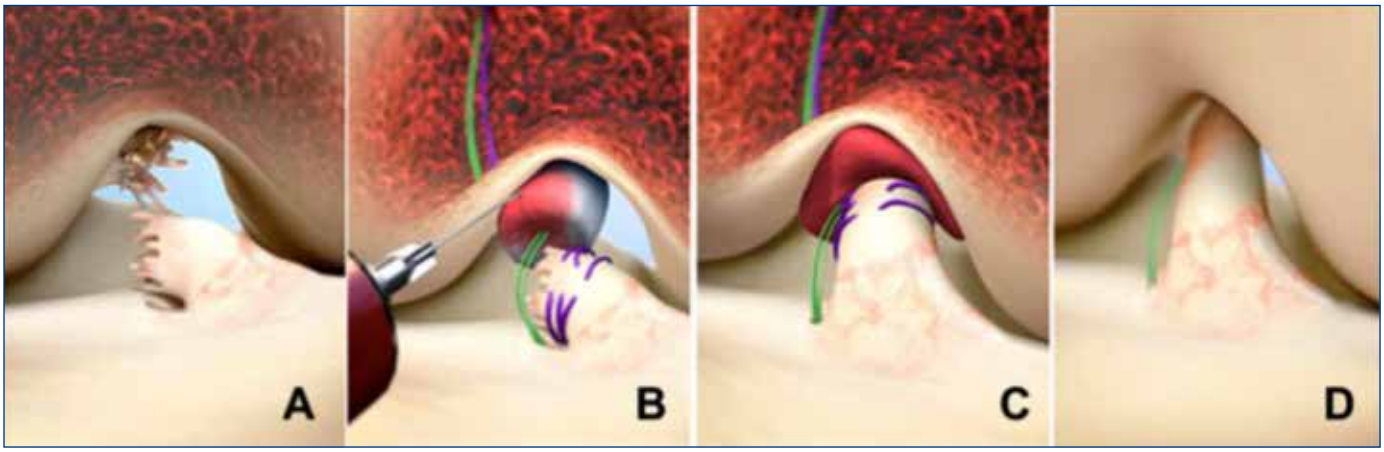
Furthermore, patients experience a significant improvement in patient reported outcome measures after ACL reconstruction with an autograft. Despite these successes, issues following ACL reconstruction remain and some patients' knees do not return to normal even two years after surgery.[12] Patients often experience quadriceps weakness and anterior knee pain from the patella tendon harvest or hamstring weakness if the hamstrings are the chosen donor tissue. Moreover, long-term follow-up of patients with anterior cruciate ligament tears show a significant risk of developing osteoarthritis, with and without ACL reconstruction.[13]

A plethora of research has been conducted over the past several decades on ways to improve the results of ACL reconstructions, but there has been very limited improvement in outcomes over the past twenty-five years. This has led to a resurgence in interest in primary repair techniques. The potential advantages of primary repair include preservation the native ligament tissues, its complex anatomy, and its proprioceptive capacity, while also being less invasive – thus eliminating complications from the graft harvest such as pain and weakness.[14]

Revisiting Repairs

The work of a group of researchers at Harvard and Brown University challenged the abandonment of ACL primary repair as an effective treatment option. Assuming that preservation of a patient's native ACL tissues and anatomy, rather than reconstruction, would lead to improve long-term knee function, they studied the science of ligament healing and developed a new alternative technique. Murray et al. determined that ACL repairs failed initially because intra-articular plasminogen in the knee joint breaks down any early clot that forms.[15] Typically, early clot forms to help heal other tissues, and without the ability to form this provisional scaffold in the knee joint to bridge the wound, there

Figure 1. Stepwise demonstration of the bridge-enhanced anterior cruciate ligament repair (BEAR) technique using the scaffold.
[**A**] The torn anterior cruciate ligament (ACL) tissue is preserved. A whipstitch of No.2 absorbable suture (purple) is placed into the tibial stump of the ACL. Small tunnels (4mm) are drilled in the femur and tibia, and a cortical button with two No. 2 nonabsorbable sutures (green sutures) and No. 2 absorbable sutures attached to it is passed through the femoral tunnel and engaged on the proximal femoral cortex. The nonabsorbable sutures are threaded through the BEAR scaffold and tibial tunnel and secured in place with an extracortical button. [**B**] The scaffold is then saturated with 5 to 10 ml of the patient's blood, and [**C**] the tibial stump is pulled up into the saturated scaffold. [**D**] The ends of teh torn ACL then grow into the scaffold, which is gradually replaced by healing ligament tissue.[17]



is no ability of the ACL to heal itself.[15] Tissue engineers then sought a way to implant a stable bridge by designing a collagen "bridge" that would be resistant to enzymes in synovial fluid that cause degeneration while at the same time maintaining the ability to stimulate cellular ingrowth and proliferation.[15,16]

This new technique is called bridge-enhanced anterior cruciate ligament repair (BEAR). With BEAR, a suture repair of the torn ACL is combined with a specific extracellular matrix scaffold (the BEAR scaffold). This scaffold has been designed from bovine tissue and includes proteins and collagen which have shown the ability to stimulate ACL healing.[16] The scaffold is placed in the space between the two torn ends of the ACL and activated with the patient's own blood (**Figure 1**).[17] The scaffold is used to bridge the gap between the two torn ends of the ligament, minimizing the necessity of absolute re-approximation of the torn ACL ends to promote ligament healing. The scaffold protects the ACL repair by keeping the blood between the torn ends of the ligament and the suture stent across the knee helps to stabilize the knee early on while the ligament begins healing itself.

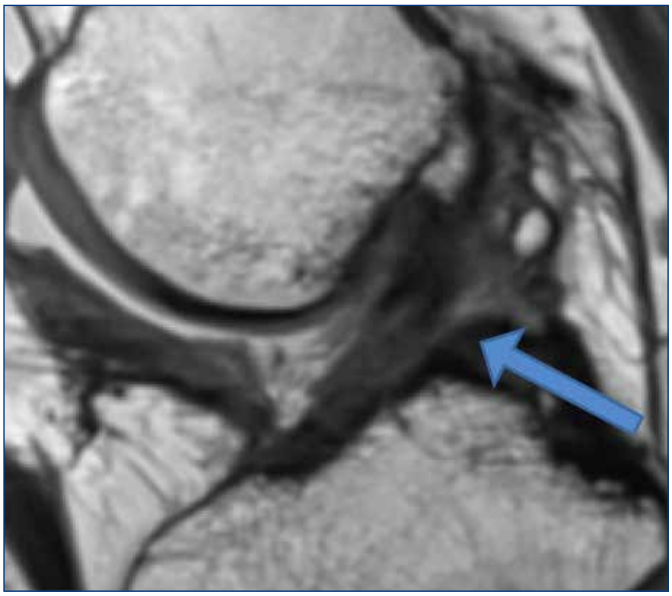
The BEAR implant has demonstrated equivalent rates of both deep joint infection and serious inflammatory reactions when compared to ACLR.[17] In addition, the BEAR technique does not require the compromise of other healthy tissues around the knee, as is required with an ACL reconstruction with an autograft. Thus, one possible benefit of the BEAR technique may be less loss of hamstring strength when compared with ACLR with a hamstring autograft, and less postoperative anterior knee pain that has typically been associated with ACLR using the classic bone-patellar tendon-bone autograft.[11]

BEAR Success thus Far

Initial research showed promising results for the BEAR implant. In animal models, implants were shown to be completely populated with cells as soon as one week after surgery, and by four weeks they contained new vessel and nerve ingrowth.[18] Subsequent large animal porcine models had promising results as well, as pigs with the BEAR implant showed a fibrovascular bundle at the healing ACL tear site via histological analysis three months after surgery when compared to pigs having undergone suture repair alone.[20] Animal models have also shown the BEAR technique has shown comparable mechanical properties when comparing BEAR to ACL reconstructions both at three months and twelve months post-operatively.[22] Additionally, the BEAR technique has shown a lower incidence of posttraumatic osteoarthritis at one year when compared to those pigs who underwent reconstructions.[19,20,21,22]

The first human study, the BEAR 1 Trial, which sought to assess safety in humans, consisted of a cohort of ten patients age 18–35 who underwent the BEAR procedure and ten patients who underwent an ACL reconstruction using hamstring autograft.[17] There were no joint infections or signs of significant inflammation in either group. There were no differences between groups in effusion or pain, and no failures by Lachman examination criteria. No patients required revision ACL surgery at two years.[17] Magnetic resonance images from all of the BEAR and ACL-reconstructed patients demonstrated a continuous ACL or intact graft (**Figure 2**). In addition, hamstring strength at three months was significantly better in the BEAR group than in the hamstring autograft group.[17] These favorable results in the first human study led to further trials in a larger group of patients.

Figure 2. T1-weighted MRI of a patient's knee 9 months post-op after undergoing BEAR procedure. Visible in center of image is reconstituted ACL (blue arrow).



The BEAR 2 trial was a double-blind randomized control trial including 100 patients randomized into either the BEAR procedure (n = 65) or an ACL reconstruction with autograft hamstring tendon (n=35). A 2:1 enrollment scheme was used to help detect for rare negative events. The study included younger adolescents with patients ranging in age from 14–35 years old, with patients having to undergo surgery within six weeks of injury. Again, there were no issues with safety after BEAR implant use. The findings at two years show patient-reported outcome scores between the BEAR group and reconstruction group to be similar. There was also comparable knee stability using the KT-1000. The BEAR cohort had improved hamstring muscle strength at two years when compared to the ACL reconstruction group, and the reinjury rate was found to be similar to that of ACL reconstructions (paper currently in press at AJSM).

Future Directions

The BEAR procedure already appears to be a safe and effective surgery for ACL tears.[17,23] The BEAR technique does not require the compromise of other healthy tissues around the knee, as is required with ACL reconstruction with an autograft. This new technique provides promise that soon surgeons will be able to repair and regenerate the ACL instead of replacing it. Further work is planned for the future to better examine how outcomes and surgical techniques can be improved. For example, the BEAR III trial is a current FDA-approved cohort study that has been approved for up to 250 patients and will be performed by seven surgeons across two centers: University Orthopedics group in Rhode Island and Boston Children's Hospital. Those eligible for recruitment include patients between the ages of 12–80 years-old who experienced an acute ACL tear within the past 50 days. In addition to the BEAR III trial, Rhode Island Hospital is one of six planned sites for an NIH-funded, multicenter, randomized controlled trial of BEAR versus patella tendon autograft reconstruction for acute ACL tear (https://clinicaltrials.gov/ct2/show/NCT03776162?term=MOON&cond=acl&draw=2&rank=1).

SUMMARY

There has been a resurgence of work on ACL repair and excitement for its potential. With the addition of a basic science and translational approach, the BEAR technique offers the potential to change the current treatment paradigm for ACL injury.

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A Clinician's Guide to Femoacetabular Impingement in Athletes

KEVIN DiSILVESTRO, MD; MATTHEW QUINN, MD; RAMIN R. TABADDOR, MD

ABSTRACT

Femoroacetabular impingement (FAI) is the most common cause of hip pain in both professional and recreational athletes. It is caused by abnormal bone development on both the acetabulum and proximal femur as a result of genetic factors and in reaction to high-volume athletics participation. Athletes typically become symptomatic after reaching skeletal maturity and commonly describe deep groin pain that worsens with activities such as squatting, cutting, or pivoting motions. For this reason, sports such as hockey, football, and soccer can be particularly irritating to an athlete with FAI. Moreover, the athlete with FAI often presents with contaminant hip and pelvis pathologies such as athletic pubalgia and iliopsoas tendinopathy that must also be addressed. While this pain often limits performance or participation in sports, perhaps the most significant ramification of FAI is the role it plays in driving early onset osteoarthritis. Fortunately, FAI can be reliably diagnosed through careful history taking, appropriate provocative physical exam maneuvers, and familiarity with hallmark radiographic features. The aims of this review are to provide clinicians with information regarding the pathogenesis of FAI, to thoroughly describe the classic history and physical exam elements, and to introduce various management strategies for athletes suffering from FAI.

KEYWORDS: hip, femoacetabular impingement (FAI), athletes, sports medicine

INTRODUCTION

Femoroaceabular impingment (FAI) is a dynamic pathoanatomic relationship between the acetabulum and the femoral head that results in abnormal contact between the two surfaces. FAI is the most common cause of hip pain in both professional and recreational athletes and is most commonly associated with sports that demand repetitive bursts of acceleration, twisting or cutting motions.[1-3] FAI consists of 3 sub-types of lesions; cam, pincer, and combined (**Figure 1**).[4-8] Cam lesions are the result of the non-spherical shape of the femoral head, often referred to as a "pistol-grip" deformity, which cause abrasions to the acetabular cartilage and subsequent avulsion from the labrum and subchondral bone.

Figure 1. Primary subtypes of FAI. [8]

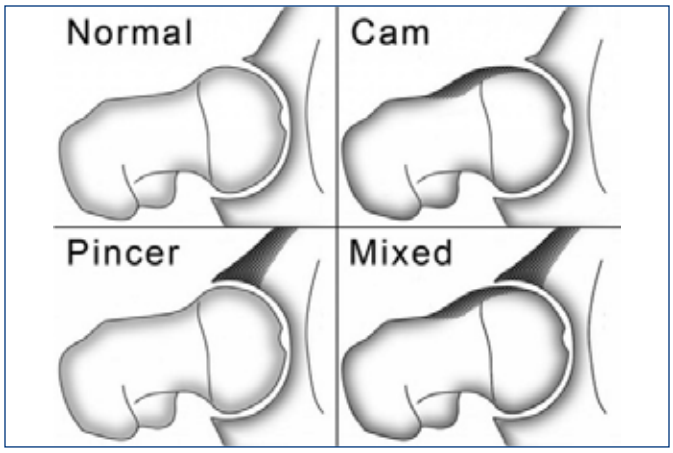
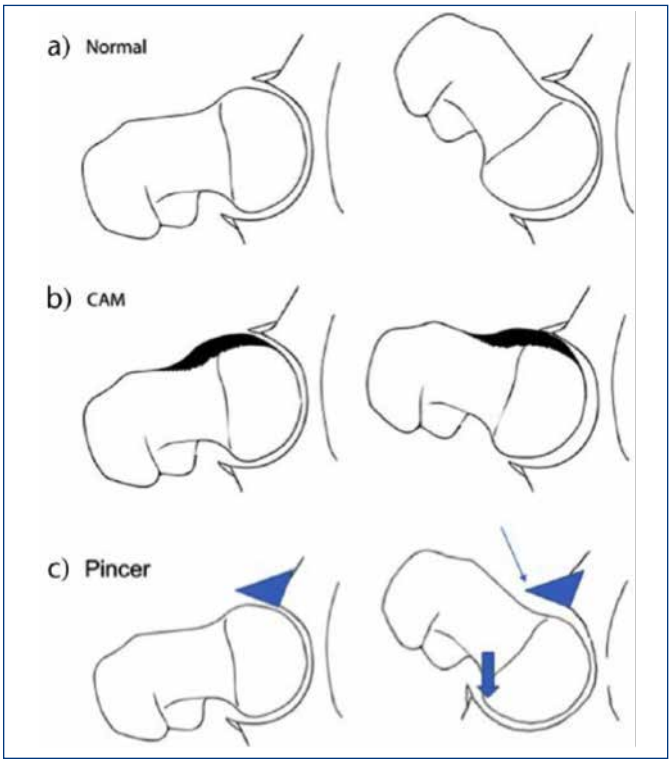


Figure 2. a) Hip demonstrating normal clearance. b) Cam lesion pushes past labrum resulting in damage to the chondrolabral junction. c) Pincer lesion generates crush injury of the labrum by the femoral neck (thin arrow) and contre-coup lesion (thick arrow).[9]



Pincer lesions, on the other hand, are the result of excessive coverage of the femoral head-neck junction by the acetabular rim and can lead to labral tearing (**Figure 2**).[9] The combined subtype incorporates elements of both cam and pincer type morphology.[4]

Determining the true prevalence and specific mechanisms that drive the development of FAI remains a challenge primarily because many patients with FAI morphology are asymptomatic.[10] However, epidemiologic studies have been able to establish that cam lesions demonstrate a predilection for male sex, whereas pincer lesions are more commonly found in females. In patients with symptomatic FAI, combined lesions are the most common subtype and represent up to 85% of cases.[4,10-12] Currently, it is believed that FAI begins with a congenital or acquired bony deformity that is subsequently exacerbated by repetitive microtrauma to the proximal femoral physis and epiphysis. As a result of the increased loading forces over several years there is an accumulation of abnormal bone as the athlete approaches skeletal maturity.[2,11,13]

This hypothesis is supported by the following facts:

1. Individuals with parents or siblings with FAI are more than twice as likely to suffer from FAI.[2,11]
2. There is a higher incidence of FAI in athletes who participate in sports that require repetitive hip motion (i.e. hockey) and enter competitive levels at an early age.[14-16]
3. That radiographic findings of FAI typically begin shortly after physeal closure. [2,11,14,15]

Although the etiology of FAI is not entirely understood, there is a significant amount of literature describing the role of FAI in other sports-related injuries such as ACL tears, athletic pubalgia, and iliopsoas tendinopathy. It is believed that FAI increases the risk for these types of injuries because it restricts internal rotation of the hip which leads to altered biomechanics and increased strain on soft tissue structures.[17–21] Additionally, current data suggests that FAI, more specifically cam and combined type lesions, serve to accelerate the progression to osteoarthritis.[4,10,11] In review of 121 patients under the age of 50 presenting for total hip arthroplasty, only 3 did not have radiographic findings of FAI.[22] Despite the growing recognition of FAI and its implications in long-term joint health, many patients still fail to receive a timely diagnosis with an average time delay of 1–2 years from initial presentation.[10,14]

PRESENTATION AND PHYSICAL EXAM

The evaluation of a patient with suspected FAI must begin with a thorough review of the patient's history, as many of the risk factors or predisposing conditions previously noted are either present in childhood or develop in early adulthood. While FAI can present within a heterogenous population, patients are most often young athletes participating in sports such as hockey, soccer, football, and lacrosse.[13,23] This emphasizes the importance of ascertaining the patient's typical activity level, the ways in which their pain has limited this activity, and any specific return to activity goals they may have. Patients with FAI most commonly describe hip pain or stiffness during or after activity.[13] Their discomfort is typically described as "deep" and located along the hip above the greater trochanter and traversing anteriorly and inferiorly towards the inguinal crease. This is known as the "C-sign". This pain is often exacerbated by deep hip flexion, particularly when coupled with rotation which can occur with physical activity or prolonged sitting.[23,24]

As with most other disorders of the hip, the exam findings and provocative maneuvers used to elicit the presence of FAI are sensitive but nonspecific. For this reason, it is imperative to not only confirm the presence of intra-articular pathology,

Figure 3. The anterior impingement, or FADIR test, consists of flexion, adduction, and internal rotation that results in pain or clicking.

Figure 4. FABER test may produce discomfort in various locations, but is only considered positive for FAI if it elicits anterior hip pain.

but also to rule out concomitant extra-articular pain generators such as tendinopathy, bursitis, and lumbopelvic dysfunction when examining these patients.[3,23,24] Observation of gait and seated posture can provide helpful clues as 34.5% of patients experience discomfort even with light walking and 25.5% experience pain when seated for more than 15 minutes. Additionally, almost 45% of patients with FAI report having pain when transitioning from a seated to standing position.[23] Passive range of motion (ROM) should be assessed next while bearing in mind that FAI morphology commonly occurs bilaterally.[25] Patients with FAI routinely demonstrate decreased ROM in flexion, abduction, and internal rotation. Interestingly, when the hip is flexed to 90°, the loss of internal rotation becomes profound. A study by Wyss et al[26] found that at 90° of flexion, patients without FAI morphology had on average 28° of internal rotation whereas those with FAI morphology had only 4°.

While there are several provocative maneuvers that can be used to evaluate for FAI, only two tests have demonstrated clinical efficacy. The first maneuver involves flexion, adduction, and internal rotation (FADIR) and is also referred to as the anterior impingement sign **(Figure 3**).[9] The second maneuver uses flexion, abduction, and external rotation (FABER) to assume a figure-4 like position (**Figure 4**). The FADIR test assesses for impingement in the anterior portion of the joint and is considered positive if pain or clicking is elicited. The FABER may produce discomfort in various locations, but is only considered positive for FAI if it elicits anterior hip pain or if there is an increased distance from the lateral femoral condyle to the edge of the examination table when compared to the contralateral side. While largely nonspecific, both the FADIR and FABER tests have demonstrated excellent sensitivity at approximately 97%.[3,23,24,27,28] Although the diagnostic accuracy has not been as extensively evaluated, impingement along the posterior aspect of the joint may be tested by extending the hip in an abducted and external rotated position known as the posterior impingement test.[3,24]

RADIOGRAPHIC EVALUATION

Plain Films

If concern for FAI exists after completing a history and physical exam, then weight-bearing plain film imaging should be obtained. This series should include a standard anterior posterior (AP) pelvis, an AP of the symptomatic hip, and a frog lateral. Additionally, clinicians may choose to include a Dunn view or false profile in order to more thoroughly assess the contour of the femoral head and neck. In general, the true AP, Dunn, and modified Dunn are the most useful in assessing pincer morphology while the cross table, and false profile are most useful for cam subtypes.

The AP, Dunn and modified Dunn should first be evaluated for any overt signs of FAI such as egregious over-coverage of the femoral head-neck junction by the acetabulum seen with pincer lesions or the classic pistol grip deformity associated with cam lesions. Additionally, the presence of more subtle findings such as a crossover sign should be explored. The crossover sign is present when the outlines of the anterior and posterior walls intersect and it is indicative of a prominent anterior wall from either overgrowth or excessive retroversion of the pelvis.[4,29,30] Specific measurements that can be made using the AP Pelvis view are the lateral center edge angle (LCEA) and the Tonnis roof angle (TRA) (**Image 1**). The LCEA is formed by a line from the center of the femoral head to the lateral edge of the acetabular sourcil and a vertical line through the center of the femoral head. While there is debate regarding exact cutoff values, an angle of >35-40° is considered to be consisting with pincer morphology. The TRA, also known as the acetabular index, is defined by a line connecting the medial and lateral limits of the sourcil and a horizontal reference line. A TRA of <3° is indicative of pincer morphology.[29,30]

Image 1. AP pelvis demonstrating a LCEA that is within normal limits (31.4°), an abnormal TRA (9°), and evidence of CAM deformity (arrow).

Image 2. Frog leg lateral demonstrating an alpha angle of 93.1° consistent with CAM deformity (>55°).

As previously stated, the most useful information for assessing cam lesions can be found using laterally oriented projections.[4,29,30] The senior author's preference is the frog leg lateral. Here, the sphericity of the femoral head-neck junction, or lack thereof, can be appreciated by calculating the alpha angle. The alpha angle consists of a line connecting the center of the femoral head to the point where the femoral head begins to flatten and a reference line through the axis of the femoral neck (**Image 2**). Angles >55° are considered to be consistent with cam lesions.[4,29,30]

MRI

Although one's history, physical exam, and plain films can establish a diagnosis of FAI, they are unable to adequately assess the articular cartilage and soft-tissue structures surrounding the hip. Magnetic resonance imaging/arthrogram (MRI/A) should be obtained to investigate the condition of these tissues.[4,29,31,32] Traditional teaching had been that 1.5T MRA was the imaging modality of choice, but more recent data has shown equivalent or superior performance of 3T MRI images.[31] 3T MRI not only provides optimal spatial resolution, but also eliminates the need for intra-articular hip injections.[29,31]

Using these images, clinicians are able to thoroughly assess any lesions or alterations to both the labrum (**Images 3 and 4**) and underlying cartilage as well as identify common conditions associated with FAI such as athletic pubalgia (**Image 5**) and hip flexor tendinopathy.[31] However, it important to remember that not all radiographic findings have clinical implications, particularly when it comes to labral tearing as it has been found in up to 69% of asymptomatic hips.[33] This underscores the importance of one's history and physical exam as the primary driver of diagnostic probability. Although debate exists around the role of the capsule in hip stability and FAI, most authors agree that MRI provides a reasonable qualitative assessment of capsular defects.[31] As an added benefit, 3T MRI can also provide further insight into osseous configurations pertaining to acetabular depth and width, femoral and acetabular version, and femoral head that may not be apparent on plain films.[29,31]

Computed Tomography (CT)

While the previously mentioned imaging modalities are incorporated in the standard workup of FAI patients, the role of CT is far more limited. CT is generally reserved for cases in which complex deformities are present or plain films and MRI do not provide sufficient information for proper surgical planning. In these cases, CT scans can be used to not only map the hip and pelvis, but also to produce a 3-dimensional reconstruction that predicts specific positions of discomfort that will help guide operative resection.[4,29]

Image 3. Coronal T2 MRI demonstrating a tear in the superior portion of the labrum associated with FAI.

Image 4. Proton denisty weighted, fat suppressed sagittal oblique MRI demonstrating a tear in the anterior portion of the hip labrum in assocition with FAI.

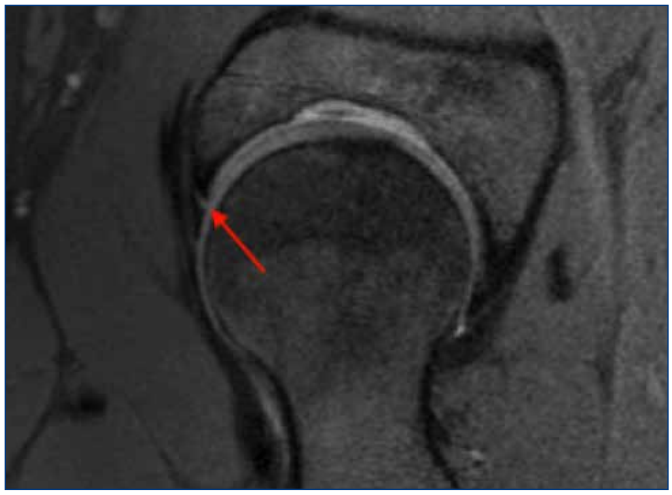
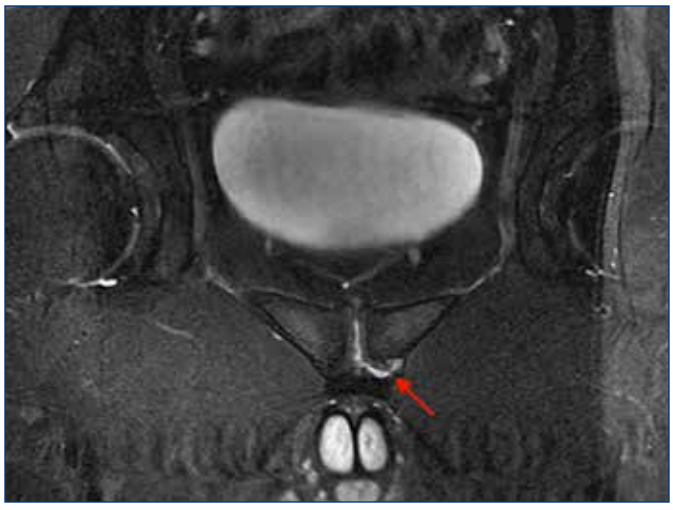


Image 5. T2 axial oblique MRI demonstrating tearing of the rectus aponeruosis from the pubic tubericle.



TREATMENT AND OUTCOMES

Conservative

The first line treatments for femoroacetabular impingement are conservative measures. This includes physical therapy, anti-inflammatory medications, and activity/lifestyle modifications. Studies have shown that good outcomes can be achieved with nonoperative management. One publication showed that 93 hips in 76 patients were successfully managed with physical therapy, rest and activity modifications.[7] It is recommended that athletes initially discontinue participation in their offending sport.[7,34] Physical therapy regimens should focus on core strengthening and stabilization as well as postural retraining with normalization of the dynamic relationship between the hip and pelvic muscles.[7,34,35] Activities such as deep flexion, positions that provoke symptoms, squats and heavyweight strength training should be avoided.[7,34,36,37] Physical therapists and athletic trainers can help athletes work on correcting their movements within the limits of pain and ensure they have appropriate pelvic tilt.[36] For high-level athletes, if the patient can continue to function at a high level despite their pain, then they can be managed with exercises and non-opioid analgesics in season.[35] Physicians can additionally consider local injections to manage symptoms during the season.[34]

Steroid injections can be used to obtain a faster effect in pain relief while hyaluronic acid injections can obtain a more delayed effect in functional improvement.[38] Pain relief from intra-articular injections support the diagnosis of FAI, but a negative response to the injection may predict poor short-term outcomes from surgical interventions.[39] When focusing on non-operative management, another study showed that there is no difference when groups were randomized to receive manual therapy and supervised exercise in addition to a regimen of standard advice and home exercise.[40] Medical and conservative management should be the focus when there are already degenerative changes to the hip joint, as joint-preserving operations such as arthroscopy are no longer indicated.

Many patients have successful outcomes with conservative management. One study of random allocation to arthroscopy versus physical therapy showed no significant difference between the groups at two years of treatment.[41] Another study treated their entire cohort of FAI patients with physical therapy initially. They demonstrated that 70% were successfully managed with conservative measures, while 12% required a steroid injection and 17% progressed to surgery.[7] The authors identified that hips with cam or combined cam-pincer impingement were 4.4 times more likely to progress to surgery than those with isolated pincer deformities.[7] Although conservative measures are often effective as described above, it is important to consider surgical management for FAI patients, especially those with cam lesions. Cross-sectional and longitudinal natural history studies have shown that cam lesions are associated with developing osteoarthritis.[22]

Surgical

Hip arthroscopy is the mainstay of surgical treatment for femoroacetabular impingement. It is most frequently performed with the patient in the supine or lateral position with peritrochanteric, midanterior, and anterior portals. Traction is used to access the central compartment within the acetabulum, where pathology to the articular and labral components can be addressed. The peripheral compartment is also accessed to address any pathology or deformity of the proximal femur. During arthroscopy, the labrum, acetabulum, femur, and capsule can all be addressed. Labral repair is recommended as this leads to greater improvements in postoperative functional scores when compared to labral debridement.[42] If a labral repair is not possible, a labral reconstruction is then employed. Acetabuloplasty can be performed to address the pincer lesion, while femoral osteotoplasties can be performed to address the cam lesion.[43] It is crucial for surgeons to perform an adequate bony resection intra-operatively. Unaddressed or undertreated bony impingement lesions have been found in 79% of revision cases, according to one study.[44]

Athletes with FAI managed surgically can have very high rates of returning to play. One study of 66 athletes showed that 94% of recreational athletes returned to play while 88% of higher level amateur athletes returned, with a significant improvement in all patient reported outcome measures in both groups.[45] Another study analyzing higher level athletes showed that 74% returned to play at preinjury level after surgery for FAI, with professional athletes demonstrating a higher return to sport rate than collegiate athletes.[46] A publication on a Danish registry had less promising results, with only 57% of athletes returning to preinjury level after arthroscopy.[47]

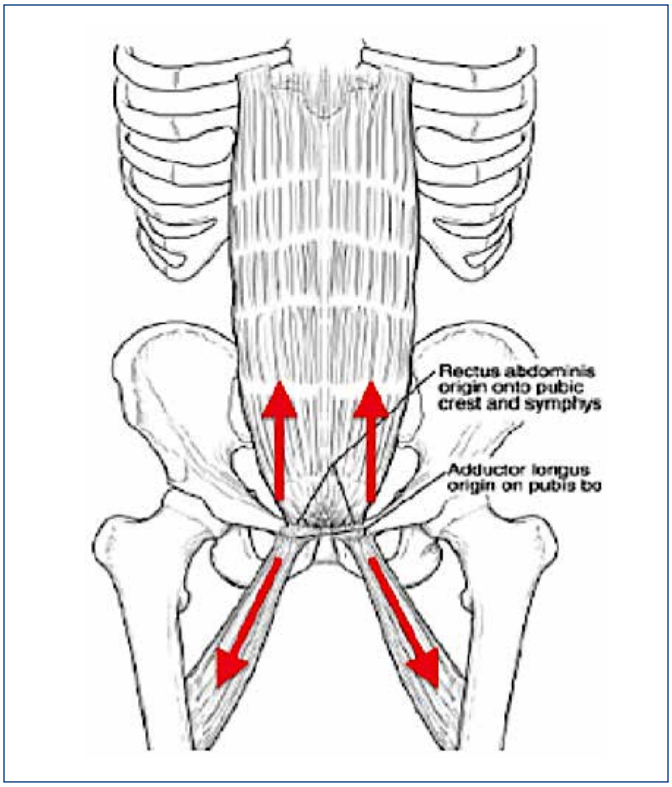
Postoperatively, patients treated with arthroscopy for FAI showed a significant improvement in hip flexion and extension strength; however, this measure still remained lower compared to control groups.[48] Regarding long-term outcomes from arthroscopic management of FAI, a study analyzing patients 7–10 years postoperatively demonstrated a significant decrease in VAS pain scale with significant increases in patient-reported outcome measures.[49] Soccer players have a high rate of return to sport, with the current literature suggesting 96–100% at 9–10 months postoperatively.[50,51] One study showed that 100% of basketball players returned at an average of 7.1 months.[52] Football players had a slightly lower rate of return, with a reported range of 87–92.5% at a mean 6.0 months postoperatively.[53,54] Hockey players had a much broader reported range of return with 67–100% returning to skating/hockey drills at an average of 3.8 months.[55,56]

ASSOCIATED CONDITIONS

Athletic Pubalgia

In addition to femoroacetabular impingement, athletic pubalgia, or sports hernia, is a well-known cause of groin pain in young athletes. The syndrome is described as having exertional inguinal and adductor pain (**Figure 5).** This pain is potentially caused by the disruption of the insertion of the rectus abdominis muscle or the internal oblique muscle from the pubic tubercle, or potentially an abnormality in the external oblique aponeurosis.[18] There is likely a connection between the pathology of athletic pubalgia and FAI. Impingement in the hip joint will lead to altered or restricted movements of the pelvis, which can result in higher stress on compensatory regions such as the pubic symphysis.[18,21] The increased stress can lead to injury to the posterior inguinal wall, resulting in athletic pubalgia. This connection would explain the high prevalence of FAI in patients with athletic pubalgia. One study showed radiographic evidence of impingement in at least one hip in 86% of patients having surgery for athletic pubalgia.[57] Another series of athletes undergoing surgery for femoroacetabular impingement showed that 32% previously had surgery for athletic pubalgia and another 39% complained of athletic pubalgia symptoms at the time of arthroscopy.[58] Combing these subgroups suggest that athletic pubalgia can be present in up to 71% of patients with impingement.[58] As patients often present with both pathologies, intra-articular and extra-articular injections can be used to determine the degree of pain coming from each pathology.[35] Persistent pain on physical exam/exercise challenge after image guided intra-articular injection is consistent with athletic pubalgia.[35]

Figure 5. Restricted range of motion about the hip increases work requirements, and therefore strain, at the insertion of the rectus abdominus and origin of the adductor longus.[21]



In those athletes whose abilities are limited by their symptoms, earlier surgical intervention can be considered. Generally, both conditions are addressed simultaneously as this has historically produced optimal results. Athletes who undergo athletic pubalgia surgery alone generally have a return to play rate of 25% whereas those who undergo FAI surgery alone have a return to play rate of 50–60%.[58,59] However, those who had both conditions corrected at the time of surgery have a return to play rate of 89%.[59] For this reason, when evaluating high level athletes with FAI and athletic pubalgia, physicians should consider simultaneous surgical treatment in order to obtain a more predictable return to sport and minimize time lost from training.[35,59]

Iliopsoas Impingement/Tendinopathy

Another condition associated with femoroacetabular impingement is iliopsoas impingement and tendinopathy. This condition is also referred to as internal snapping hip syndrome. It can imitate joint pain, so it is important to distinguish this diagnosis with careful history and physical exam. The condition stems from a decrease in range of motion, specifically external rotation and abduction. Patients develop a contracture of the iliopsoas and feel a pop over the pectineal eminence. Current literature indicates that iliopsoas fractional lengthening for painful internal snapping is the second most common concomitant procedure performed with FAI surgery, with 73% of the patients in the cohort having both procedures.[60] Fortunately, patients with this associated condition benefit from surgical intervention. Patients with combined FAI and painful snapping hip who had iliopsoas lengthening at the time of treatment had similar improvements and complication rates compared to patients who only had arthroscopy for isolated FAI.[61]

Iliopsoas tendinitis can be a postoperative issue for patients who underwent arthroscopy for FAI. It was diagnosed in 24% of a cohort of 252 postoperative patients.[20] Within this subgroup, 47% improved with activity modification/NSAIDs/PT, 53% required a corticosteroid injection, and 12% required revision arthroscopy and iliopsoas release.[20] A different study looking at revision hip arthroscopy found that 29% of patients had a tight psoas tendon and corresponding labral impingement, for which a partial psoas tendon release was performed.[44] This data stresses the importance of assessing the iliopsoas during the index procedure as these patients do well if it is appropriately addressed, but can have issues postoperatively if it is not.

CONCLUSION

FAI results from a deformity of the proximal femur and/or acetabulum. It is a common cause of hip pain in athletes. Patients should be evaluated with history, physical and the appropriate imaging. Patients can often be managed with conservative measures such as physical therapy. However, patients have high returns to sport with hip arthroscopy. Patients with FAI should also be evaluated for athletic pubalgia and iliopsoas impingement, as they are frequent concomitant conditions.

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The Weekend Warrior: Common Hand and Wrist Injuries in Athletes

JOSEPH A. GIL, MD; ARNOLD-PETER C. WEISS, MD

ABSTRACT

Weekend warriors are individuals who condense their weekly physical activity into extended intervals over one or two days.[1] Excessive physical activity can result in a multitude of overuse and traumatic upper extremity injuries. The purpose of this review is to highlight the etiology and management of the more common hand and wrist injuries in athletes.

INTRODUCTION

The Centers for Disease Control and Prevention and the American College of Sports Medicine recommend at least 30 minutes of physical activity on most days of the weeks in order to optimize the health benefits achieved from an exercise regimen.[2] Weekend warriors are individuals who condense their weekly physical activity into extended intervals over one or two days.[1] Excessive physical activity can result in a multitude of overuse and traumatic upper extremity injuries.[3] Overuse injury is defined as repetitive microtrauma that occurs at a rate that exceeds the tissue's capacity to adapt and recover.[4] Of the 1–3% of adults in the United states classified as weekend warriors, 65% participated in sports or an exercise regimen.[1] While lower extremity injuries are the most common in sports-related injuries, upper extremity injuries account for approximate 22% of these injuries.[5] The purpose of this review is to highlight some of the common hand and wrist injuries in athletes.

HAND

Phalangeal and metacarpal fractures and joint dislocations

The pattern of **phalangeal and metacarpal fractures** depends on the position of the bone and the external force applied. Transverse fractures from result from direct blows and distal phalanx fractures occur from crush injuries and may be associated with a nail bed laceration. Sudden radial or ulnar deviation forces result in a spiral fracture of the proximal phalanx or the metacarpal.[6]

Metacarpal and phalanx fractures that occur in athletic injuries generally result from lower energy trauma in comparison to the high energy trauma such as those that occur in a motor vehicle accident.[7] Given the lower level of energy, a majority of these fractures are stable and could successfully be managed nonoperatively for short period (i.e. 2 to 6 weeks) of immobilization in a splint, cast, or buddy tape.[8,9] In cases where the fracture remains unstable or there is unacceptable articular displacement, the fracture should be reduced through a closed or open approach and secured with either Kirschner wires, independent lag screws, or a plate.[10,11]

The most common **hand joint injures** in athletes include injuries to the proximal interphalangeal (PIP) of the digits and the metacarpophalangeal (MP) joint of the thumb.[7] PIP joint injuries include dislocations, fracture-dislocations, and collateral ligament injuries. Collateral ligament injuries occur when the digit suddenly sustains an axial load and dorsiflexion force. In most circumstances, the collateral ligament injuries are incomplete and can be effectively treated with buddy taping, splinting and early range of motion. With proper immobilization, the athlete could return to sport immediately.[7]

PIP joint dislocations account for the majority of dislocations in the hand. These are most commonly caused by impact in ball handling sports (e.g. basketball, football, etc.). Dorsal dislocations result from sudden hyperextension that results in rupture of the volar plate. A majority of these are successfully managed with closed reduction and a short period of immobilization in a dorsal blocking splint or buddy tape.[7] Immediate return to sport with a stable joint results in no impact on the final outcome.[12] Volar dislocations result from a sudden radial or ulnar deviation force combined with a volarly directed force.[6,13] These dislocations are associated with central slip, collateral, and transverse retinacular ligament injuries. These should be close reduced and immobilized to protect the central slip to prevent subsequent boutonnière deformity. In case where close reduction is not readily achieved, open reduction reveals that the proximal phalanx head is buttonholed between the central slip and the lateral band.[7] Dislocations are frequently associated with articular fractures.[7] In those circumstances, the objective is to restore a concentric and stable joint with minimal intraarticular displacement, which could sometimes be achieved with a closed reduction but may require percutaneous fixation or in some cases even open reduction and internal fixation. This injury may keep an athlete out of sport for at least 6 to 8 weeks.[9,14]

Thumb MP joint injuries in sports are common and

primarily include dislocations, fracture-dislocations, and collateral ligament tears.[7,15] **Dorsal thumb MP dislocation** is generally caused by a sudden hyperextension force that results in rupture of the volar plate. These are often readily reduced and immobilized for up to 4 weeks. In cases where reduction is not readily achieved, open reduction usually reveals that the metacarpal head is trapped between the two heads of the flexor pollicis brevis or the reduction is block because of volar plate, collateral or flexor pollicis longus (FPL) interposition.[7,16]

Campbell first reported gamekeeper's thumb as mechanism for chronic **thumb ulnar collateral ligament** injury in 1955.[17] Subsequently acute thumb UCL injuries were found to occur commonly in skiers who sustain a sudden hyperextension and hyperabduction force to the thumb MP joint resulting in avulsion of the ligament off of the base of the proximal phalanx.[18] Patients present with pain at the thumb UCL that is exacerbated with gripping. Assessment of stability in full extension and 30 degrees of flexion is critical so that both the accessory and proper bundles are examined, respectively.[15] Nonoperative managed is reserved to partial and nondisplaced avulsions.[19] Operative repair is indicated in circumstances of gross instability, displaced avulsion fracture or in cases where the adductor aponeurosis interposes between the torn ligament and the bone (i.e. Stener lesion).[15,19]

Closed tendon injuries

Mallet finger is caused by disruption of the terminal slip of the extensor mechanism resulting in characteristic extensor lag.[7] Mallet finger injuries have been classified into 5 types: 1) tendon attenuation, 2) tendon rupture, 3) tendon avulsion fracture, 4) fracture, and 5) physeal fracture.[20] Extension splinting remains the gold standard in circumstances where the joint remains concentric.[21] Comparison of splinting versus pinning mallet injuries demonstrated excessive risk of long-term complications after pinning.[22] In the setting of joint subluxation, there may be value of operatively reducing and pinning the joint.[23]

Flexor digitorum profundus (FDP) tendon avulsion injuries occur when the finger is forcefully extended while the profundus tendon is contracting.[24] This injury, also referred to as jersey finger, commonly occurs when a player grabs the jersey of another player resulting in avulsion of the tendon. FDP avulsion injuries have been classified into 3 types: 1) tendon retracted into palm, 2) tendon retracts to the level of the PIP joint and could occasionally avulse a small piece of bone, and 3) avulsion with large osseous fragment that prevents retraction beyond the middle phalanx. While all 3 warrant reinsertion, type 1 injuries are the most time-sensitive and should be repaired within one week because a substantial portion of the blood supply is compromised as the viniculae are torn.[24]

WRIST

Wrist fractures

While **distal radius fracture** most commonly occur in the pediatric and elderly populations, sports-related distal radius fractures in the young adult population remain the most common cause of distal radius fractures in this population.[25] These fractures primarily occur from a fall onto an outstretched wrist. According to the American Academy of Orthopedic Surgeons (AAOS) clinical practice guideless, distal radius fractures should undergo operative fixation if post-reduction radial shortening is >3 mm, dorsal tilt is > 10 degrees, or intra-articular displacement is >2 mm.[26] Currently, fixation of distal radius fractures with volar-locking plates through a modified Henry approach or extended flexor carpi radialis (FCR) approach has become the most common method for surgical fixation of these fractures.[27] While the introduction of the volar plate introduced an effective approach to managing these fractures, subsequent recognition of problems associated with volar plating spearheaded investigations that demonstrated the signification of plate placement and screw lenths.[28,29] Although optimal outcome could be achieved with restoration of alignment within these parameters, it is important to recognize that even optimally reduced and nonoperatively managed fractures could be associated with complications such as extensor pollicis longus (EPL) tendon ruptures and immobilization-related complications such as stiffness.

Scaphoid fractures result from falls on the outstretched, hyperextended, and radially deviated wrist.[15] Scaphoid fractures account for 70% of all carpal bone fractures and most commonly occur in young males.[30,31] The majority of the blood supply to the scaphoid enters dorsally and distally at the dorsal ridge.[32] A lesser degree of blood supply enters the scaphoid volarly at the distal tuberosity.[32] This predominately retrograde blood flow corresponds to the healing potential of scaphoid fracture. Distal pole fractures heal reliably whereas proximal pole fractures are predisposed to delayed healing and nonunion. Nondisplaced scaphoid fractures present with predictable tenderness on exam; however, radiographs are often negative.[33] These cases were once routinely managed with a period of immobilization and repeat imaging. Only 20% of these cases subsequently develop radiographic evidence of fracture.[33] Advanced imaging with a magnetic resonance imaging (MRI) or commuted tomography (CT) scan has been demonstrated to have a higher sensitivity and specificity for detecting occult scaphoid fractures.[33,34] Obtaining these studies has been demonstrated to accelerate time to diagnosis and reduce the duration of unnecessary immobilization.[35] The majority of acute nondisplaced scaphoid fractures are treated nonoperatively with cast immobilization.[36] Although several scaphoid fracture classifications have been described, the four fracture characteristics that generally provide insight into the optimal strategy are anatomic location (70–80% occur at the waist,

10–20% at the proximal pole, and least frequently at the distal pole), displacement, fracture stability, and chronicity.[37,38] For instance, distal pole fractures have been demonstrated to consistently heal (100% union) in 7–8 weeks and nondisplaced scaphoid waist fractures heal in 9–10 weeks with a nonunion rate of 6%.[38] In contrast, nondisplaced proximal pole fractures required 4–8 months of immobilization and had a 14% nonunion rate.[37,38] Operative reduction and fixation of scaphoid fractures is indicated for displaced or unstable fractures, delayed fracture presentation, proximal pole fractures, open fracture, and established nonunions.[39,40] Currently, scaphoid fractures are most commonly stabilized with a single headless compression screw (HCS) placed along the longitudinal axis of the scaphoid.[41,42]

Other commonly encountered carpal fractures in athletes include hook of hamate and triquetral avulsion fractures.[43] **Hook of hamate fractures** account for 2% of carpal bone fractures and are associated with racquet sports, golfers and baseball players and are a result of direct impact.[44,45] The fracture is associated with pain at the hypothenar eminence that is exacerbated with gripping or direct palpation.[44,45] A carpal tunnel view could detect the fracture; however the fracture is best delineated with a CT scan.[44,45] Acute nondisplaced fractures should be immobilized in a short arm cast resulting in an approximately 50% healing rate.[46,47] Nonunions or displaced fractures that are symptomatic or present in associated with ulnar nerve compression are effectively managed with excision with no sequelae on grip strength.[44,45]

Triquetral fractures are the second most common carpal fractures accounting for approximately 15% of all carpal fractures.[48,49] Dorsal triquetral fractures are caused by an axial load applied to the dorsiflexed and ulnarly deviated wrist.[48] The most common type of dorsal triquetral is an avulsion of the dorsal radiocarpal or dorsal intercarpal ligament from its insertion onto the triquetrum.[48,50,51] Dorsal avulsion fractures present with tenderness at the dorsum of the triquetrum and radiographically detected on the lateral radiograph. Given that the this fracture represents avulsion of the critical dorsal extrinsic wrist ligaments, the wrist should be casted for at least 3 weeks followed by progressively working on regaining range of motion and strength.[51]

Scapholunate ligament injuries

The **scapholunate (SL) ligament** is the most commonly injured ligament in the carpus. The injury is caused by excessive wrist hyperextension, ulnar deviation and intercarpal supination.[52] Based on the degree of energy, the ligament may be partially or completely torn.[52] When the ligament is completely torn, the scaphoid flexes and the lunate and triquetrum extend, resulting in a dorsal intercalated segment instability pattern (DISI).[53] Unrecognized injuries result in abnormal cartilage wear patterns and subsequent arthrosis that progresses through a pattern known as scapholunate advanced collapse.[54]

SL ligament injuries initially present with radial sided wrist pain, diffuse swelling, and diminished range of motion. The scaphoid shift test is generally not tolerated acutely.[13] In cases of complete disruption, there may be widening (>3 mm) evident on the posteroanterior radiograph. In some circumstances, subtle widening of the SL interval may be accentuated with a power grip view. It is critical to compare the injured wrist to the contralateral wrist as there may be a physiologic degree of widening that is normal in some individuals.[7,55] Magnetic resonance imaging could further characterize the extent of injury to the ligament and identify concomitant injures.[56] Wrist arthroscopy remains the gold standard diagnostic tool for carpal ligament injury.[55] Management (i.e. immobilization, arthroscopic debridement, repair, reconstruction, or salvage procedure) depends on the timing, extent of ligament injury, carpal alignment, reducibility of malalignment, and the status of the cartilage.[57]

Distal radioulnar joint and Triangular fibrocartilage complex injuries

The triangular fibrocartilage complex (TFCC) includes the volar and dorsal distal radioulnar ligaments, central disc, meniscus homolog, ulnocarpal collateral ligament, ulnolunate ligament, ulnotriquetral ligament, and the extensor ulnaris tendon subsheath.[58,59] The TFCC is the primary stabilizer of the distal radioulnar joint (DRUJ).[60] **TFCC injuries** in athletes can occur from an acute traumatic event or repetitive microtrauma that results in degenerative tears.[60] Acute TFCC typically result from a concomitant axial load and rotational stress at the TFCC.[60] TFCC injuries in athletes can be distinguished from other ulnar-sided wrist injuries by preforming a careful history and physical examination.[7] TFCC pain localizes to the depression between the ulnar styloid and the pisiform.[7] Radiographs are often normal in patients with isolated TFCC injuries.[7] MRI (3Telsa) has been demonstrated to effectively detect TFCC injuries with a sensitivity and specificity of 86% and 100%, respectively.[56] In cases where imaging is negative but history and exam are consistent with TFCC injury, wrist arthroscopy can result in a definitive diagnosis and allow for simultaneous management of TFCC injuries.[3] Degenerative or acute central tears are successfully managed with debridement, while peripheral tears are often repaired given the vascularity and healing potential at the periphery.[61–63]

Overuse conditions

Dequervain's, or first dorsal compartment tendonitis, is the most common overuse related tendonitis of the wrist among athletes.[7] This condition is caused by the cumulative microtrauma that results from repetitive shearing of the abductor pollicis longus and extensor pollicis brevis at the undersurface of the sheath of the first dorsal compartment at the radial styloid.[7] Patients report pain at the radial styloid process that is exacerbated with twisting and grasping activities.[64,65]

Tenderness localizes to the first dorsal compartment over the radial styloid. Finklestein test is performed by placing the patient's thumb in a flexed position and concomitantly ulnarly deviating the wirst.[66] Conservative management includes splinting, steroid injection and therapy.[67] In cases where conservative management fails, first dorsal compartment release is associated with optimal results.[68]

Intersection syndrome results from the repetitive shearing that occurs between the first and second dorsal compartments. Pain and occasionally crepitus localizes to this region approximately 4 cm proximal to the level of the wrist. The primary etiology of the pain is suspected to be inflammation with the 2nd dorsal compartment. Conservative management includes splinting, steroid injection and therapy. In cases were conservative management fails, the second dorsal compartment could be surgically released.[69]

Extensor carpi ulnaris (ECU) pathology is the second most common overuse injury in athletes.[70] Microtrauma to the ECU tendon that occurs with repetitive activity in sports such as tennis can result in ECU tendonitis.[71] In contrast, abrupt supination of the flexed and ulnarly deviated wrist can result in rupture of the ECU subsheath and associated ECU instability.[71] ECU injuries are associated with pain and swelling along the course of the ECU tendon.[72] Conservative management includes splinting, nonsteroidal anti-inflammatories (NSAIDs), steroid injection and therapy.[71] ECU instability could be initially managed with a course of immobilization of the wrist in a pronated, extended and radial deviated position.[73] If ECU instability does not resolve with immobilization, several ECU stabilization techniques have been reported.[72,74,75]

CONCLUSION

Weekend warriors are individuals who condense their weekly physical activity into extended intervals over one or two days.[1] Excessive physical activity can result in a multitude of overuse and traumatic upper extremity injuries. Recognizing common hand and wrist injuries in these athletes can help clinicians effectively diagnose and manage these patients.

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Foundational Health for Runners: Is it the Key to Minimizing Injury?

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ABSTRACT

BACKGROUND: Injury rates in runners are as high as 80%. Here, we focus on the concept of foundational health including sleep, recovery, nutrition, stress and physical health and how it can reduce injuries.

METHODS: The literature was reviewed to find papers linking running injuries and athletic performance to the foundational health topics discussed.

RESULTS: There are many factors that can improve athletic performance and reduce injuries in runners other than the often-discussed topics: training philosophies, footwear, and running form. This paper shows how a multidisciplinary approach including education on sleep, rest, stress, nutrition, strength, and mobility all can improve performance and reduce injuries.

CONCLUSIONS: The care and management of an injured runner is multifactorial and the treatment should be as well. By optimizing foundational health, the sports medicine professional will not only reduce injury risk, but also improve performance and overall health.

KEYWORDS: foundational health, running, injuries, recovery, strength

INTRODUCTION

Injuries are the bane of runners' existence and with injury rates as high as 79%, it is of no surprise the struggle runners have with pain and recovery.[1] Why are the injury rates so high? Why are these injuries so difficult to prevent and treat? What can runners do about them? Let's explore.

The high mileage of distance runners, especially marathon distances, requires huge time commitments and demands on the body. Running is a repetitive activity that takes place in one plane, going in one direction, with high ground reaction forces, one foot striking in front of the next for approximately 1,200 steps per mile. The inherent nature of the activity is a true example of a cumulative trauma injury, so it's no wonder the injury rates are so high. Let's take this example to shed more light:

Jane is a 140 lb. runner who takes 1,200 steps per mile and runs 25 miles per week. Jane also endures ground reaction forces of 2x's her body weight with each step (hypothetical and realistic assumption).

- 140 lbs. x 2 (twice her body-weight per step) = 280 lbs. of force per step
- 1,200 steps per mile x 25 miles per week = 30,000 steps per week
- 30,000 steps per week x 280 lbs. per step = 8,400,000 lbs. of force entering her body weekly

Resiliency can be defined as the ability to recover from and adapt to stimuli. From the example above you can see the stimuli is massive with runners. If a runner lacks resiliency and proper biomechanics, then injuries will occur. Injury risk can be lessened with good foundational health (physical, emotional, and mental), a symmetrical and efficient gait pattern, and adequate motion and strength. Here we will focus on foundational health factors rather than the popular, but rarely agreed upon, topics such as running form, training philosophies, or footwear.

Minimizing injury and improving performance can be aided by all foundational health topics discussed in this article: improved sleep, better recovery, better nutrition/hydration, less stress, better strength. Most runners reach a point where the body can not manage the demands of running and can not recover adequately, which is when injury usually occurs. A more holistic approach to these athletes may be the key.

RESULTS

Sleep

We are a nation of sleep-deprived people from teens to adults. The National Sleep Foundation recommends that teens get 8–10 hours of sleep and adults 7–9 hours nightly. Our teens are sleeping an average of 5–6 hours per night and adults are not doing much better. For optimal recovery from exercise and for optimal health, adequate sleep is crucial. What happens to us when we deprive ourselves of the needed sleep?

Increased injury risk:

- Athletes who sleep on average <8 hours per night have a 1.7 times greater risk of being injured than athletes who obtain ≥8 hours of sleep per night.[2] If we're not giving our bodies enough time to recover and heal post workout, it is no wonder we are susceptible to injury.[3,4]

Decrease in athletic performance:

- Without enough sleep our speed, endurance, reaction time, focus, and motor skills all suffer.[5] Sleep has been shown to improve motor skills by 15–20% which can be essential to establish efficient running form and cadence.[6,7]

Decreased recovery, muscle growth and repair:

- During sleep and leading up to sleep, there are a number of hormonal responses that take place to allow for optimal recovery after exercise.[4]
- Decrease in neurocognitive functions like memory and attention.[4,5]

Increased risk of illness:

- Exercise is a physiological stressor that activates hormones regulating the immune system and metabolic functions.[4,5,8] Lack of sleep lowers immune protection, making one more susceptible to sickness or injury.
- Increased risk of depression and anxiety disorders has been linked to lack of sleep as well.

Difficulty maintaining a healthy body weight:

- Less sleep has been associated with a higher BMI.[5] Altered sleep patterns can affect metabolic hormones that relate to appetite and food consumption. This can lead to changes in food intake and processing, making weight maintenance difficult.[8]

It is safe to say that every major body system does better with sleep and nothing improves with less sleep. So, how do we get more sleep? Well, it comes down to why we are not getting a good night's sleep. If you struggle to "shut down" and fall asleep, here are a few tricks to try.

Brain dump: Before bed, write down what is on your mind (i.e. worries, concerns, to-dos). By writing these down, you can decrease the mental burden and promote relaxation.

Screen time: Stop screen-use approximately one hour before bedtime. The light from the screens stimulates our brains and does not promote restfulness. What you do on your screen (work, social media, paying bills, etc.) may also add to your mental burden, shifting you further from relaxation.

House lighting: Our natural circadian rhythm directs us toward sleep at sunset. The invention of light bulbs has caused a disruption in that natural cycle. By limiting the bright light in our home close to bedtime we may be able to encourage a rest state and decrease stimulation.

Hot baths: Our circadian rhythm leads to a drop in body temperature as we approach time for sleep. After a hot bath, you will encourage a cooling effect. This drop in body temperature could help mimic the natural rhythm and encourage somnolence.

Bedroom is a resting place: Keep the bedroom for sleeping and do not bring work, electronics, or clutter into your room. Dark, quiet, and cool (65–68 °F) will enhance the resting state.

Rest Days

Rest days are crucial and can vary depending on the athlete. An *absolute* rest day means the runner is not partaking in any physical activity at all and will have a more sedentary day. While this can allow an athlete to recover and remain below the injury threshold, absolute rest, if done too often or for too long, can eventually reduce resiliency. It is the authors' recommendation to have one absolute rest day per week.

Active recovery/cross-training days are another form of rest day. Performing an activity at a low/moderate intensity without the same physical demands and stressors of running can aid in recovery. Some examples of good active recovery/cross-training include swimming, yoga, rowing and hiking. Remember, the purpose of rest days is to aid recovery from the running and to rebuild what was depleted and broken down; therefore, a minimal to moderate intensity is ideal. It is the authors' recommendation to have at least one active recovery/cross-training day per week. Some runners, such as fast-growing teens and aging runners, may require more absolute and active rest days.

Nutrition And Hydration

Nutrition and hydration requirements can vary depending on the runner and the level of exercise. Recommendations for loading before exercise, fueling during exercise, and recovery post-exercise may be slightly different per individual. Yet, there is an overall theme that awareness of what an athlete is putting into his or her body can help or hinder exercise goals and health. A proper balance of nutrient intake and water consumption is essential. Think about incorporating as many natural whole foods in their diet as possible and limiting processed foods. Whole foods are foods that rot and rotting is a natural process. If a food item has a shelf life of two years, it is most likely highly processed and could lead to increased rates of inflammation in the body.[9] Ingesting enough nutritious calories ensures that one has the energy available to fuel the high demands of distance running. Although nothing will replace the personal advice of a nutritionist or registered dietitian, here are some evidence-based guidelines:

Carbohydrates: Runners cannot afford to "cut carbs" as they are a primary fuel source. In addition to being vital for endurance performance, carbohydrates can slow the release of stress hormones in the body.[10] Look to take in approximately 6–10 grams of carbohydrates per kilogram of body weight daily.

Proteins: Protein intake allows for accelerated muscle growth and accelerates recovery by rebuilding the muscle fibers stressed during a run. The amino acids found in proteins help build the body's cells, including stimulating

white blood cells of the immune system, which can protect the body from illness.[10,11] Look to take in approximately 1.2–1.7 grams of protein per kilogram of bodyweight daily.

Fats: Fats should not be avoided or excluded from the diet.[10] Restricting fats and overall caloric intake can actually be detrimental to athletic performance and recovery. It has been linked to increased injuries in female athletes and directly related to disorders like Relative Energy Deficiency Syndrome in Sport.[10,12] Twenty to thirty-five percent of daily caloric intake should be comprised of healthy fats such as olive oil, fish, avocados, seeds, and nuts.

Hydration: A personalized hydration plan can be useful due to individual differences in sweat content. In general, runners should aim to drink consistently throughout the day to maintain a baseline hydrated state.[10,13] Examining the color of one's urine is a simple way to assess fluid intake. Adequately hydrated urine should be clearer. Additionally, one must be careful with supplements or over-hydrating before running. Try to attain the standard eight 8-ounce glasses per day.

Vitamin and mineral supplements: If a diet is lacking in essential components then supplementation may be beneficial. One should consult with a registered dietician or nutritionist for guidance. It is not uncommon for female runners to be deficient in calcium, vitamin D, and iron. Supplementation may assist these runners in remaining healthy and warding off injuries.

- Calcium plays an important role in bone building. Vitamin D is also important as it aids in calcium absorption. Daily recommendations of calcium and vitamin D for adults are 1000–1500 mg/day and 600–2000 IU/day respectively. These levels can maximize bone health and muscle function.[14,15]
- Iron is an essential mineral that helps with oxygen transport in the blood, energy metabolism, and thermoregulation. Optimal intake is 8–18 mg/day. Women should aim for the higher end: 15–18 mg/day.[16] Consuming leafy green vegetables, lean red meat, or beans ensures adequate iron intake.

The calories burned will depend on the size, gender, and the intensity of the workout. It is necessary to replace these calories with nutrient-rich foods to help recover and prepare for the next run. Any diet that restricts a food group or limits caloric intake to an arbitrary number should be avoided.

Mental & Emotional Health

Mental and emotional health are important aspects of our overall well-being that can be overlooked in clinical and athletic settings as we focus on physical health of the runner. Stress is prevalent across our population, especially high schoolers and young adults. We are seeing alarmingly high rates of anxiety, depression, and suicide. While there are numerous theories on this trend, many point to the increase in social media and smartphone use.[17] If stress levels are elevated for too long, it can negatively impact physical performance and resistance to injury. In a high-stressed state, the body breaks down. By then introducing the demanding physical nature of distance running, it can be too much for a runner to tolerate, leading to injury.[18]

The topic of mental health is gaining traction in the media. In this way, it is becoming more socially acceptable and integrated into our daily dialogue. Hopefully, this will further reduce the stigma and lead more people to seek professional help. Coaches and healthcare providers should be aware of the signs of anxiety, depression, or other emotional stressors; and then encourage professional help.

There is a correlation between poor running performance, lack of sleep, increased screen time, and increased stress. By encouraging proper rest and lessening screen-time use we may be able to keep stress down and enhance the overall health of runners thus improving resiliency. Mindfulness-based interventions have been shown to help manage negative emotions and improve overall athlete well-being. Some literature supports mindfulness-based interventions for reducing injury, but further research is warranted.[19] Other simple techniques to help manage stress might include meditation, breathing exercises, yoga, tai chi, or other restorative activities to complement a rigorous training schedule.

Pre-Run Warm-Up And Post-Run Cool-Down

Pre-run and post-run activities help reduce injury rates. Performing techniques like myofascial release and massage, either with a professional or with a device like a foam roller, help when executed pre and post-run.[20,21] When performed prior to running, these techniques can improve range of motion and muscle activation. After running, they can reduce soreness and augment recovery.[20,21]

In addition to tissue preparation, a dynamic warm-up performed immediately before running can prepare the body, which reduces injury and enhances recovery.[22] Dynamic warm-ups should include activities emphasizing the muscles needed for running such as the calves, gluteals, quadriceps, and hamstrings. These activities should have slightly less impact and slightly more range of motion than actual running. Activities like jumping jacks, squats, lying leg raises, and standing leg swings are some that would suffice.

Passive stretching, foam rolling/massage after a cool down activity (i.e. walking) are examples of effective post run activities.

Strength

Lack of adequate strength is a modifiable intrinsic risk factor that can increase the likelihood of a running injury. It is widely known that strength-training can improve performance in athletes and can help reduce injury.[23] Therefore, it is extremely important to ensure that runners are participating

in a running-specific strengthening routine that emphasizes the physical demands of running. A detailed assessment by a professional is often helpful in addressing the needs of the individual.

Some general key points for strengthening are:

- Most runners would benefit from exercises activating the gluteal muscle group. Exercises that involve hip extension, abduction, and external rotation are crucial. Some examples of common exercises are bridges, clamshells and squats.
- Most runners would benefit from an intrinsic foot-strengthening program. Barefoot exercises help make a runner aware of toe placement and arch height. At its simplest, barefoot walking in a grassy field or barefoot balance exercises are a good foundation.
- Most runners would benefit from a strengthening program incorporating core and pelvic stabilizing muscles. Some examples of common exercises are planks, bird-dogs and weighted farmer's carries.
- Running-specific strengthening should include single-leg standing exercises. These should only be performed once the athlete is strong and stable enough for these balance-based positions. Bilateral simultaneous two-legged strengthening is not as effective, as the single leg positions that mimic running. To prepare for single-leg activity, a runner can transition from squats to partial range single leg squats or shift from deadlifts to partial range single leg deadlifts.
- A combination of body weight exercises, plyometric exercises (jumping), and weight lifting should be incorporated into most running programs.

Range of Motion

Flexibility/stretching (which falls under many names such as passive stretching, active stretching, dynamic stretching...) is another modifiable intrinsic risk factor for injuries. Studies published over the past decade have changed the general perception of stretching. As with most research, we find conflicting results, with some concluding that stretching can prevent injuries and the majority concluding it doesn't change the incidence. Although most of the evidence is not there to support stretching as an injury-preventative modality, when asked, "not stretching" was the number one reason runners thought their injuries occurred.[24] There is obviously a gap in the overall consensus of research and the knowledge base of recreational runners.

Adequate flexibility, which can be assessed by a healthcare or exercise professional, may reduce the likelihood of certain muscle and tendon injuries. Becoming hypermobile or stretching beyond what is considered a normal range does not provide further benefit. Many muscles can become restricted by running. Some common areas where flexibility is needed are the calves, iliopsoas (hip flexors), hamstrings and Iliotibial band. It is important to note that the runner with a chronically tight muscle that does not respond to stretching may not have a tightness issue. There may be an underlying problem associated with weakness or dysfunction of the muscle leading to a strain. In this case, a strengthening/muscle activation intervention can reduce the tightness and associated pain whereas stretching would not help. This emphasizes the need for a detailed assessment of a runner's function – strength, mobility, motor control, posture – to get to the source of an injury.

CONCLUSION

The strongest predictor of running injury is prior injury.[1] A possible cause of this would be runners returning to the sport before fully recovering or before the causes related to the injury are fully addressed. Is this a failure of the patient or of the sports medicine professional? For this reason, healthcare professionals – especially those involved in rehabilitation – need to ensure that the sources of a runner's injury are identified and addressed properly rather than merely attending to pain relief.

Running injuries are multifactorial and the care and management of injured runner should be as well. There is no perfect exercise, no perfect running form, and no perfect running shoe that we can recommend for all runners. What we can universally recommend is the concept of optimal foundational health, which should supersede any specific running strategy. As sports medicine professionals, we should never let the metrics of athletic performance outweigh the health and wellbeing of the runner. Promoting good foundational health will not only reduce injury risk, but also improve performance and overall health which are essential metrics of sports medicine.

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Athletic Pubalgia

WILLIAM BINDER, MD; RAMIN R. TABADDOR, MD; JEFFREY FEDEN, MD

PRESENTATION

A 32-year-old man presented to the emergency department with complaint of pain in his hips and his pelvis. The patient stated he normally plays soccer, but can no longer play due to the pain. He reported that he had this pain previously, and rested for about 6 months, but he returned to play as a goalie the day prior to presentation. He said that his pain was so severe in the suprapubic area that he could not play his position.

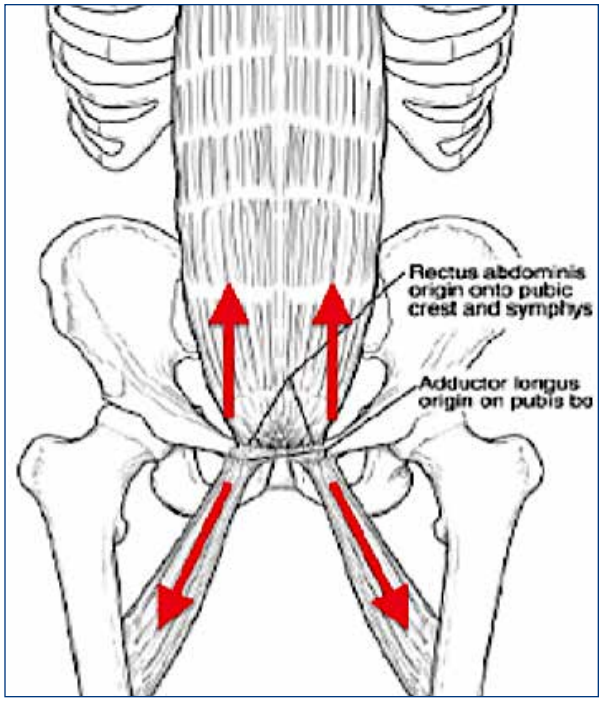
The patient was originally from Brazil, and had been in the United States since age 14. He had no recent travel. He denied shortness of breath, chest pain, fever, or any constitutional symptoms. He was able to ambulate with minimal discomfort, but could not jog, pivot, or sprint without significant pain.

On exam, the patient's vital signs were normal: BP 122/64 mm Hg, pulse 64, RR 14, T 98.2, and pulse oximetry 98% on room air oxygen. The patient had a soft abdomen but was tender over the pubic symphysis. He was non-tender in all quadrants, and there were no masses, n guarding, or rebound. On musculoskeletal exam, there were no deformities. The patient was able to fully range his hips bilaterally with external and internal rotation. He had pain with resisted leg adduction, and he was tender with sitting up.

A plain film of the pelvis was obtained (**Figure 1**).

Figure 1. (blue arrow) Large bony excrescence arising from the superior aspect of the right pubic symphysis extending to the left, consistent with chronic rectus abdominis insertional tendonopathy/tear (athletic pubalgia).

Figure 2. Pathoanatomy of Athletic Pubalgia
The rectus abominus and adductor longus muscles pull in the opposite direction. With injury to the rectus an imbalance in muscle forces occurs causing groin pain.



DISCUSSION

Athletic Pubalgia, originally referred to as Gilmore's groin, and commonly called Sports hernia or core muscle injury, is an injury to the structures comprising the pubic aponeurosis. (**Figure 2**)[1] During athletic movements (pivoting, cutting, kicking and twisting, explosive turning), significant but unequal forces by the rectus abdominus and adductor longus muscles are exerted on the pubic aponeurosis which overlies the pubic symphysis.[1] The rectus can be weakened by athletic movement, leading eventually to tearing in the rectus

and unopposed adducter forces, which may cause groin pain and the bony excresence seen in our patient.

The diagnosis of athletic pubalgia is challenging. It is often misdiagnosed, and is often associated with femoral acetabular impingement.[1,2,3] Plain films can be revealing but an MRI should be performed when athletic pubalgia is considered. MR arthrogram of the hip should be obtained if intraarticular hip pathology is suspected.[4] Management of athletic pubalgia depends on pain and severity. Mild symptoms can be treated with rest, NSAIDs, and physical therapy. In moderate to severe injury, steroid injections can help alleviate pain, but surgical repair is eventually required.[5]

Our patient was referred to orthopedics for further outpatient diagnostic evaluation and management. Unfortunately, he did not follow up and repeated attempts to contact him were unsuccessful.

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Hypothyroidism-induced Acute Kidney Injury and Hyponatremia

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ABSTRACT

Thyroid hormones affect every organ system in the body including renal development and physiology, and electrolyte and water homeostasis. These effects happen as a consequence of the combination of direct effects of thyroid hormones on renal tubules and hemodynamic effects of thyroid hormones. As a consequence, both hypothyroidism and hyperthyroidism significantly affect renal function. This case describes a patient with hypothyroidism-related acute kidney injury without rhabdomyolysis, and no additional precipitating factor. While there are many case reports describing hypothyroidism-related rhabdomyolysis leading to acute kidney injury, there are only a handful of case reports on hypothyroidism-related acute kidney injury without rhabdomyolysis.

KEYWORDS: hypothyroidism, acute kidney injury, rhabdomyolysis, hyponatremia

INTRODUCTION

Hypothyroidism affects every organ system in the body, including the central nervous system, cardiovascular system, gastrointestinal system, musculoskeletal system, and the metabolic rate.[1] Hypothyroidism is associated with an underappreciated but clinically significant alteration of renal physiology.[1-2] While there are many case reports describing hypothyroidism-related to rhabdomyolysis leading to acute kidney injury (AKI),[3-9] there are only a handful of case reports on hypothyroidism-related AKI without rhabdomyolysis.[10-14] This case describes a patient with hypothyroidism and AKI without rhabdomyolysis, and no additional precipitating factor.

CASE PRESENTATION

An 82-year-old male with past medical history of colorectal cancer (in remission for 15 years), hypertension, hypothyroidism and hyperlipidemia, presented from home with a change in mental status. According to family, the patient lived alone but his daughter visited frequently. At his baseline, he was fully oriented and able to take care of himself. Two days prior to presentation, his daughter noted that the patient complained of lethargy and lower back pain, with poor appetite and decreased oral intake. His mental status continued to worsen, and so his daughter brought him to the emergency department. Upon presentation, the patient was unable to recall life events and did not know why he was in the hospital. He denied fever, chills, chest pain, shortness of breath, cough, nausea, vomiting, abdominal pain, diarrhea, constipation, dysuria, urgency, peripheral edema or any focal neurological complaint.

Daily medications included levothyroxine, aspirin, fenofibrate, lisinopril, omeprazole, and vitamin D supplements. The patient had a two-pack-year smoking history but quit smoking decades prior to presentation. The family reported he ingested 5–7 standard alcoholic drinks per week. He was retired and spent most days at home.

Initial vital signs were the following: temperature 97.2°F, heart rate 48 regular beats per minute, respiratory rate 20/min, blood pressure 152/63 mmHg and oxygen saturation was 97% on room air. The patient was alert but oriented only to self. Cardiac exam revealed bradycardic S1S2. On neurological exam he was able to follow commands and had no focal deficits. The rest of his physical examination was within normal limits.

Laboratory results are included in **Table 1**. Urinalysis was unremarkable. EKG showed decreased voltage in precordial leads. TSH was 132.994 uIU/mL (normal range 0.350–5.5 ulU/ml), with free T4 low at 0.16 ng/dl (normal range 0.8–1.8 ng/dl) and T3 low at <20.0 pg/dL (normal range 230–420 pg/dl). CT of the brain without IV contrast, renal ultrasound, chest x-ray and CT of the abdomen and pelvis without IV contrast were unremarkable. Due to the hyponatremia, acute kidney injury, and profound hypothyroidism, the patient was admitted to the medical service.

Both nephrology and endocrine were consulted for acute kidney injury, hyponatremia, and hypothyroidism. The patient received 500mL of intravenous 0.9% normal saline and was then fluid restricted to 1L/day. He was not given any maintenance intravenous fluids. He was also started on intravenous levothyroxine. His serum creatinine and serum sodium level normalized to 1.1 mg/dl and 138 meq/L, respectively, over the next 4–6 days. His mental status also improved by the time of discharge and remained stable at a 3 month follow-up visit. As a result of his rapid improvement with thyroxine and fluid restriction alone, his AKI and hyponatremia were thought to be directly related to myxedema.

Table 1. Laboratory results

Test Name	Results	Reference Range
White Blood cells	6.4 x10^9/L	3.5-11 x10^9/L
Hemoglobin	12.3 g/dl	13.5-16 g/dl
Hematocrit	36.1%	37%-47%
Platelets	228 x10^9/L	150-400x10^9/L
Sodium	125 meq/L	135-145 meq/L
Potassium	3.6 meq/L	3.5-5.1 meq/L
Chloride	93 meq/L	98-110 meq/L
Bicarbonate	18 meq/L	22-32 meq/L
Creatinine	4.06 mg/dl	0.6-1.2 mg/dl
Blood Urea Nitrogen	50 mg/dl	6-24 mg/dl
Glucose	104 mg/dl	67-99 mg/dl
AST	30 IU/L	10-42 IU/L
ALT	9 IU/L	6-45 IU/L
ALP	28 IU/L	34-104 IU/L
Albumin	3.6 g/dl	3.5-5 g/dl
Total Bilirubin	0.6 mg/dl	0.2-1.3 mg/dl
Total Protein	6.2 g/dl	6-8 g/dl
Ammonia	40 µmol/L	2-50 µmol/L
CPK	319 IU/L	20-210 IU/L
Serum Osmolality	281 mOsm/Kg	290-300 mOsm/Kg
Urine Osmolality	516 mOsm/Kg	

Figure 1. Effects of Thyroid hormone on Hemodynamics

Genomic effects
Upregulation of α-myosin heavy chain genes (fast myosin) → Increased contractility and hypertrophy
Downregulation of β-myosin heavy chain genes (slow myosin) → Increased contractility
Upregulation of SR Ca2+-ATPase (SRCa2) → Increase rate of myocardial relaxation in diastolic phase (Lusitropic effect)
Downregulation of phospholamban (PLB) → Inhibits SRCa2 → Increase in Lusitropic effect
Upregulation of b1-adrenergic receptors → Increase heart rate
Upregulation of Na+-K+-ATPase → Chronotropic effect??
Downregulation of Na+/Ca2+ exchanger (NCX1) → Inotropic affect
Upregulation of TRα1 → Increased eNO which leads to vasodilatation
Downregulation of angiotensin receptors → Vasodilation
Effect depends on type of voltage gated potassium channels
Increase in VEGF → Angiogenesis
Increase FGF → Hypertrophy
Nongenomic effects
Increases HIF-1 protein synthesis → Increase in blood volume
Activation of TRα1 through nongenomic pathways → Increased eNO which leads to vasodilatation
Thyroid hormone also activates multiple Ion channels (Na, K, Ca) and regulates multiple specific signal transduction pathways
Modulates sensitivity to adrenergic vasoconstrictors and endothelium-dependent vasodilators.

DISCUSSION

The principal mode of AKI in myxedema is thought to be due to the reduced renal blood flow (RBF) and glomerular filtration rate (GFR), but the exact pathogenesis remains unclear and is thought to be multifactorial.[1] Studies have described both genomic and nongenomic signaling by thyroid hormones.[15,16]

Effect of hypothyroidism on hemodynamics

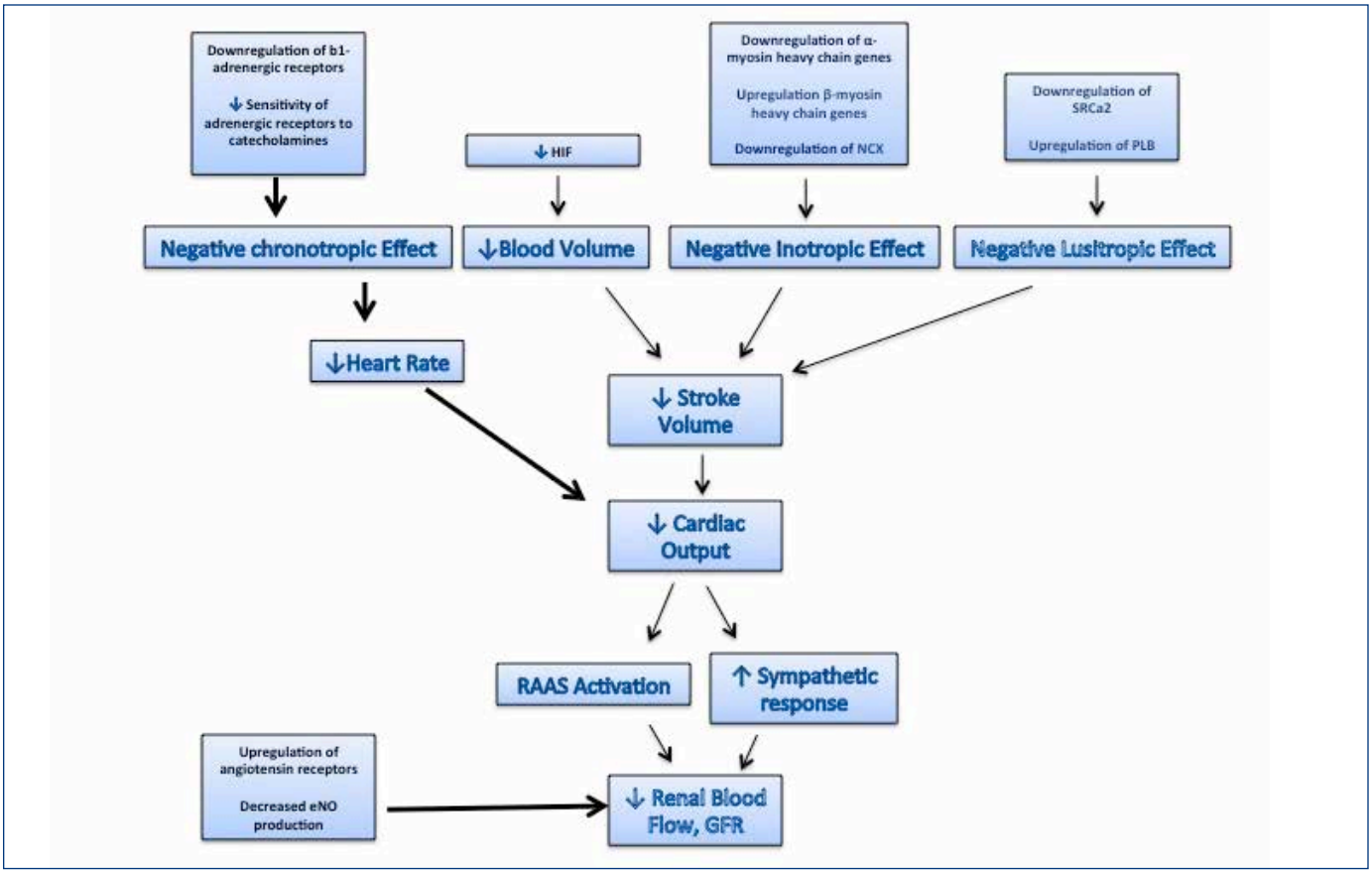
The effects of hypothyroidism on the cardiovascular system has been extensively studied and well documented. Thyroid hormones affect the cardiovascular system by both genomic and non-genomic pathways which widely overlap. (**Figure 1.**)[15,16] Thyroid hormones regulate positive and negative expression of genes for structural and regulating proteins of cardiomyocytes, thus affecting both contraction and electrochemical signaling pathways. Thyroid hormone fine-tunes the expression of genes for myosin heavy chain, which is the main structural part of the sarcomere.[16,17] Thyroid hormone regulates the expression of sarcoplasmic reticulum Ca2+-ATPase (which plays a role in relaxation time during diastole), b1-adrenergic receptors (increase receptor function and density), guanine nucleotide regulatory proteins, Na+-K+-ATPase, voltage gated potassium channels and Na+/Ca2+ exchanger.[18-22]

Thyroid hormones downregulate the expression of angiotensin receptors in vascular smooth muscles (leading to vascular relaxation).[23] Thyroid hormones also activates endothelial nitric oxide synthase through alpha 1 thyroid receptor (TRα1), which in turn leads to vasodilation.[24] Thus, thyroid hormones leads to decreased vascular resistance, which leads to increased vascular compliance, decreased blood pressure, and increased cardiac output (**Figure 2**).

Effect of Hypothyroidism on GFR

Subclinical and clinical hypothyroidism is a common problem in CKD patients. Elevated creatinine can be seen within two weeks of severe hypothyroidism. Creatinine often improves very quickly after appropriate treatment of hypothyroidism but sometimes patients can have slower

Figure 2.



response or incomplete renal recovery with prolonged and severe hypothyroidism.[25-29] Hypothyroidism may also causes increased glomerular capillary permeability which leads to proteinuria. Proteinuria often precedes the reduction in GFR in hypothyroidism.

Because creatinine-based equations are used to estimate GFR, it is unclear how much of an elevated creatinine level reflects the true drop in GFR. Creatinine levels can be altered by myopathy, decreased tubular secretion or creatinine metabolism. Cystatin C blood level cannot be used as a marker of GFR in this patient population as the levels are generally low in hypothyroid patients and elevated in hyperthyroid patients.[30] The exact mechanism is unknown but it is thought to be related to direct effects of thyroid hormone on cystatin C production. In one study, Villabona and colleagues noted increased renal plasma flow (from 542.8ml/min to 717 ml/min) and GFR (from 99.6 to 125.7 ml/min measured with Cr-EDTA clearance) after thyroid hormone replacement in overt hypothyroidism patients. [31]

Effect of hypothyroidism on Tubular function

Thyroid hormones affect renal tubular function by both genomic and nongenomic pathways.[1] Hypothyroidism affects renal tubular secretory and reabsorptive processes by upsetting the expression and activity of various ion channels/transporters. Thyroid hormones also influence the renal tubular physiology by alternating the responses to different hormones (Increased ADH sensitivity).

Hypothyroidism causes renal tubular down regulation of Na+-K+ ATPase, H+-ATPase, Na+-HCO3 exchanger, Na+-H+ exchanger, Na+-Pi IIa exchanger, Na+-sulfate exchanger, Na+-K+-2 Cl2 cotransporter, Na+-Ca2+ exchanger, Cl2 channel and up regulation of aquaporin (AQP) 1 and 2.[1,32,33] As one can imagine, hypothyroidism will lead to impaired urinary concentrating ability, increased urinary sodium excretion, increased fractional excretion of sodium and decreased free water clearance. Hypothyroidism results in low cardiac output which triggers the carotid baroreceptors and consequently increases the non-osmotic ADH secretion. All of these effects of hypothyroidism on sodium and water homeostasis will lead to hypothyroidism-induced hyponatremia.[33]

Effect of hypothyroidism on Skeletal muscle

Hypothyroidism causes rhabdomyolysis by inducing structural abnormalities in the setting of metabolic impairment. Structural abnormalities include decline in fast-twitch type II muscle fiber mass, increase in slow-twitch type I muscle fiber mass, decrease in muscle carnitine and glycosaminoglycan deposition. Metabolic abnormalities include inhibition of mitochondrial activity, decreased protein turnover,

impaired carbohydrate metabolism, low myosin ATPase activity and low ATP turnover. [34.] In turn, rhabdomyolysis causes AKI through intraluminal obstruction by myoglobin, uric acid casts, direct myoglobin toxicity and production of free radicals. [35]

CONCLUSION

This case illustrates how severe symptomatic hypothyroidism can lead to severe renal dysfunction without rhabdomyolysis. Clinicians should be aware of this rare association and patients with unexplained AKI should be worked up for hypothyroidism.

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A Case of Acute Thrombotic Myocardial Infarction in Polyarteritis Nodosa

RIMMY GARG, MD; DEANNA JOE, MD; J. DAWN ABBOTT, MD

ABSTRACT

This is a case of a 33-year-old male with acute myocardial infarction from complete thrombotic occlusion of the distal left anterior descending and 1st obtuse marginal artery secondary to polyarteritis nodosa. This case highlights the treatment course and need for continued awareness of vasculitis as a cause for myocardial infarction.

KEYWORDS: acute myocardial infarction, polyarteritis nodosa, vasculitis

CASE REPORT

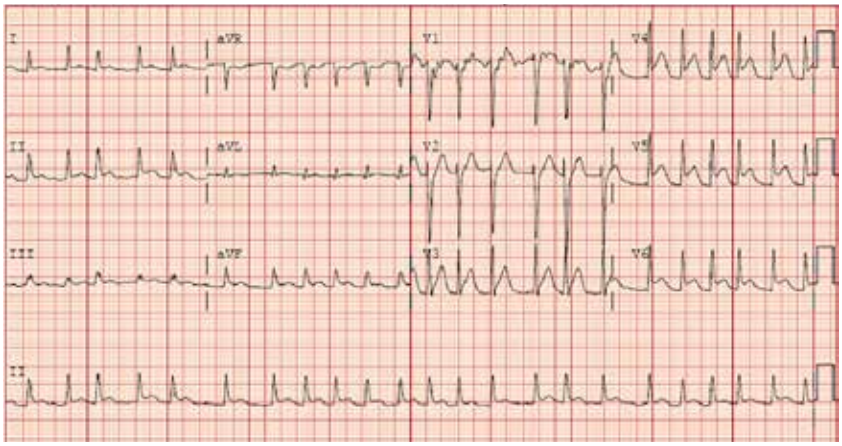
A 33-year-old man with a history of biopsy-proven testicular vasculitis and myopericarditis diagnosed 3 weeks prior presented with acute, left-sided, non-radiating, pleuritic chest pain worse with exertion. He also endorsed night sweats and unintentional weight loss of 20 lbs. in 4 months. Due to the recent inflammatory disorders, he had been treated with colchicine and a taper of ibuprofen, which had helped relieve his symptoms earlier, but were no longer effective for his chest pain.

On physical exam, the patient had a BP of 133/83 with a HR of 114 bpm. He was afebrile with an SpO2 of 99% on room air. He was noted to have bilateral conjunctivitis. He otherwise had clear lungs, normal heart sounds with no murmurs or rubs, soft and non-tender abdomen, and no skin lesions or rashes. Labs noted CRP 91 ml/L, ESR 130 mm/hr, and troponin 0.174. The patient had followed-up with outpatient rheumatology prior to the current presentation; however, autoimmune work-up involving ANA, ANCA, IgA, Anti-dsDNA antibody, rheumatoid factor, and hepatitis B surface antigen was negative. Admission ECG showed sinus tachycardia.

During the hospitalization, the patient was initially treated for recurrent myopericarditis with ibuprofen and colchicine. However, his chest pain persisted with development of shortness of breath. On hospital day 2, troponin increased to 10.5 without ECG changes, but with cardiac MRI showing focal areas of myonecrosis and normal biventricular function with an ejection fraction (EF) of 64%. He was switched from ibuprofen to methylprednisolone due to concern for systemic vasculitis. Three days later, his chest pain recurred in association with ST segment elevations in leads I, II, V3-V6 with new atrial fibrillation (AF) [**Figure 1**].

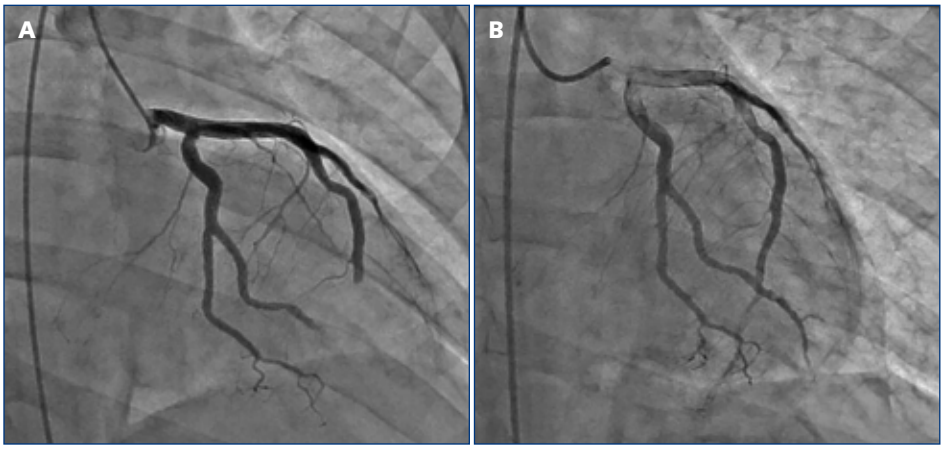
Emergent transthoracic echocardiogram (TTE) demonstrated an EF of 35% with extensive regional dysfunction of the apical and anterior walls. Cardiac catheterization demonstrated diffuse ectasia of the coronary arteries with thrombotic occlusion of the distal left anterior descending (LAD) artery and the first obtuse marginal (OM) branch of the circumflex. (**Figures 2A, 2B**) He underwent primary percutaneous coronary intervention (PCI) to both culprit vessels with balloon angioplasty and thrombectomy with a bare metal stent (BMS) in the LAD. These cardiac findings were consistent with vasculitis and coronary thrombosis or embolism.

Figure 1. ECG showing new ST segment elevations in leads I, II, V3-V6 as well as new atrial fibrillation.



The diagnosis of systemic polyarteritis nodosa (PAN) was made based on his history of testicular vasculitis, weight loss, episcleritis, and acute thrombotic MI. The patient was started on pulse dose steroids with transition to oral prednisone and cyclophosphamide. For the myocardial infarction and subsequent PCI, he received aspirin, clopidogrel, atorvastatin, and metoprolol. For paroxysmal AF, he was anticoagulated with unfractionated heparin until therapeutic on coumadin and started on digoxin for better rate control.

Figure 2. [**A**] Cardiac Angiography during catheterization showing 100% thrombotic stenosis in the distal Left Anterior Descending (LAD) and 1st Obtuse Marginal (OM) branch artery. [**B**] Revascularization of the distal LAD and 1st OM branch artery.



By time of discharge, he had converted to a normal sinus rhythm on ECG.

At two months post-discharge, the patient had no recurrent chest pain and continued to attend cardiac rehabilitation. However, TTE from 10-month follow-up shows a severely reduced EF of 25% with severely dilated left ventricle.

DISCUSSION

Polyarteritis Nodosa

PAN is an ANCA-negative necrotizing vasculitis of small- and medium-sized arteries, not associated with glomerulonephritis. It can result in a multitude of disorders and can be either idiopathic or triggered by an agent, i.e. hepatitis B.[1] While it can affect many organ systems, PAN is a rare disease with a prevalence of about 31 cases/million affecting individuals of any gender, age, and race.[1]

Diagnostic Criteria

Systemic PAN can be diagnosed based on the American College of Rheumatology criteria,[2] which requires 3 out of the following:

- weight loss ≥ 4 kg
- livedo reticularis
- testicular pain or tenderness
- myalgias
- mono- or polyneuropathy
- diastolic blood pressure >90 mmHg
- elevated blood urea nitrogen or serum creatinine levels
- presence of hepatitis B reactants in the serum
- arteriographic abnormality
- presence of granulocyte or mixed leukocyte infiltrate in an arterial wall biopsy

With weight loss, testicular pain with testicular mass status post orchiectomy, biopsy proven testicular vasculitis, and arteriographic evidence during cardiac catheterization, this patient fulfilled four criteria.

Rates of Complication

Cardiac complications are known sequalae of some vasculitides like Kawasaki's, Takayasu's, and esosinophilic granulomatosis with polyangiitis,[3] but are not as well recognized with PAN vasculitis. A recent pathological study of 37 autopsy cases of PAN showed 81% had cardiac vascular involvement, most commonly with panarteritis;[4] however, clinically significant cardiac manifestations are rarely seen pre-mortem. Cardiac involvement has been reported to occur in 4–30% of PAN cases, usually manifesting as congestive heart failure.[1] The occurrence of acute MI is rare and has been described to stem from aneurysmal, thrombotic, or vasospastic events caused by widespread inflammation from PAN.[5] As these manifestations can be silent in early stages of vasculitides, heart function should be evaluated by an ECG or TTE. If these reveal abnormalities or the patient develops symptoms, further investigations, such as cardiac MRI or more invasive procedures can be pursued.[6]

Treatment

Symptomatic cardiac involvement requires aggressive treatment given poor prognosis with 13% five-year survival.[7] Those with evidence of cardiac, gastrointestinal, neurological, or renal involvement are categorized as having moderate to severe PAN and should be treated with both glucocorticoids and cyclophosphamide, as the combination has demonstrated better outcomes including rates of survival.[8,9] In recalcitrant cases of PAN, biologic therapies such as tumor necrosis factor antagonists (i.e rituximab and infliximab) have been shown to be potential alternative options.[10] Various case reports have also identified tocilizumab, a humanized monoclonal anti-interleukin-6 receptor antibody, as a treatment option for those who do not respond to the glucocorticoid-cyclophosphamide combination.[10]

Along with glucocorticoids and cyclophosphamide, our patient also required balloon angioplasty and thrombectomy with placement of a BMS. A stent had to be placed in this case because despite multiple passes with a thrombectomy catheter and balloon angioplasty, the flow in the LAD remained slow due to the thrombus. When vessel size is adequate, trapping the thrombus behind the stent scaffold can be beneficial for limiting the extent of the infarction.

As to why a BMS was used instead of a drug eluting stent (DES), the operator did not specify. However, one possible reason could be that while we do not have data on endothelial healing in patients with vasculitis, DES have delayed healing compared to BMS, which can increase the risk of late stent thrombosis, especially after discontinuation of dual antiplatelet therapy (DAPT). Furthermore, given that this patient had developed new atrial fibrillation necessitating oral anticoagulation, BMS may shorten the duration of triple therapy of DAPT and anticoagulation as that has a high risk of bleeding.

CONCLUSION

Patients with PAN occasionally present with cardiac manifestations, but rarely do they present with acute coronary syndrome (ACS). Multiple case studies have been reported of patients with PAN presenting with ACS, but there is still no consensus on how to treat cardiac stenotic lesions in patients with PAN. In a case of acute MI due to PAN in a young female with myocardial SPECT with minimal reversible ischemia and no recurrent chest pain, the patient had no additional events with standard cardiac medical management and immunosuppressive therapy.[11] In another case, a patient with a BMS followed by placement of DES and another BMS on a separate occasion, underwent a coronary artery bypass graft after a repeat coronary angiography found mildly aneurysmal left coronary vessels with multiple stenoses.[12] While clinically significant cardiac involvement in PAN is rare, it can be life threatening. It is not only important to recognize PAN early, but also to evaluate cardiac function soon after the diagnosis of PAN is made. We believe this patient was appropriately treated in a timely manner, though with the subacute presentation of his chest pain in setting of a recent diagnosis of myopericarditis, ACS superficially may have seemed less likely the cause of his symptoms. As such, thrombotic occlusion and possible acute coronary syndrome should always be maintained in the differential in any PAN patient with chest pain.

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Disclosures

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Investigation of the Injury Rate of Female Fitness Competitors

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ABSTRACT

BACKGROUND: Female fitness competitions are increasing in popularity. Athletes are participating in weight-cutting protocols to help reduce body fat percentage to improve muscle definition and physique.

METHODS: The goal of the study was to investigate weight-cutting practices and determine if these practices were associated with increased injury rates. A survey was distributed at a New England fitness competition.

RESULTS: Thirty-five female fitness competitors participated in the survey at a single competition. The calculated injury rate for female fitness competitors is 0.18 injuries per 1000 hours of training. Age over 35 (p=0.014) and a history of or current eating disorder (p=0.005) were significant risk factors for sustaining an injury. Menstrual cycle abnormalities were present in 11 of 35 individuals (31.4%).

CONCLUSIONS: Female fitness competitor injury rates are low; however, injuries were more common in athletes over age 35 and those with either a history of or a current eating disorder.

KEYWORDS: female athlete triad, bone health, injuries

INTRODUCTION

Female fitness competitions are an emerging form of physique competitions with distinct goals in comparison to female bodybuilding competitions. Competitors are graded on their physiques with hopes to obtain professional status and endorsements. There are different categories to compete, which vary between organizations but generally are based on the participants' muscle mass. Multiple organizations around the world sponsor these competitions. Preparing for these competitions requires rigorous discipline in both exercise and diet, possibly predisposing these athletes to the female athlete triad: decreased energy availability, abnormal menstrual function, and decreased bone mineral density.[1] Female bodybuilders and fitness competitors are also at risk for developing addictive behaviors characterized by exercise dependence and muscle dysmorphia.[2]

Female athletes participating in activities, such as ballet or gymnastics, or those who exhibit prolonged restrictive eating are especially vulnerable to developing conditions of the female athlete triad.[1] Females with these physiologic sequelae have a higher risk for sustaining an injury. A recent prospective multicenter study suggested that in young girls participating in prolonged exercise and demonstrating risk factors related to the triad had a 30%–50% increase in the incidence of bone stress injuries.[3]

To date there is no literature available on the increasing popular activity of female fitness and physique competitions. The safety of their practices has yet to be evaluated because there is currently no research investigating how females are preparing and training for the events. During the weight-cutting phase of training prior to an event, these competitors will restrict their caloric intake, and to date there is no evidence of how low a caloric intake they will consume and how often they exercise. In our study, we sought to evaluate the rate of injuries in females competing in fitness modeling and to identify potential risk factors associated with these injuries so that healthcare professionals and coaches can better counsel this athletic population.

METHODS

A written fill-in-the-blank style survey was designed to investigate female fitness competitor demographics, dietary habits, eating disorder history, and injuries sustained during the last year of training. (**See Appendix Questionnaire**) The survey was done in United States customary units and mathematically converted to SI units. Our first step to understanding this problem was to use the survey to characterize the injuries that women develop during active participation in training for these competitions. The survey was designed by the authors and distributed to amateur and professional fitness competitors in person at a USA Northeast regional fitness competition. Informed consent and risks of participation in the survey were discussed with the participants. Participation in the study was voluntary and anonymous. The study was reviewed by the Institutional Review Board at Lifespan/Rhode Island Hospital.

Statistical analysis was performed utilizing Microsoft Excel (Microsoft Corporation, Redmond, WA) and StatPlus: LE (AnalystSoft Inc, Walnut, CA). A P value of <0.05 was defined as statistically significant. Analysis of the injury

rates over the defined time period allowed calculation of incidence. Fischer Exact tests were performed to identify differences between subgroups. The age subgroup calculations were performed by categorizing the age based on categories 20–25, 25–30, 30–35, 35 and older. Overall injury rate per athlete per year and number of injuries per 1000 hours of exercise were calculated.

RESULTS

Thirty-five surveys (31 amateur and 4 professionals) were distributed and completed (100% response rate) by female fitness competitors at single fitness competition (**Table 1**). The average age was 26.9±3.8 (range 22-38). The average height was 63.3±2.1 inches (range 60-68) [160.8 cm (range 152.4–172.72)] and the average precutting weight was 131.1±8.7 lbs. (range: 120-160) [59.3 kg (range 55.4-72.6)].

Table 1. Demographics of Female Fitness Competitors

Best describes you?	35 fitness competitor (100%)
Amateur or professional?	31 amateur, 4 professional
Average Age	26.9±3.8 yr.
Average Height (in.)	63.3±2.1 in.
Average pre-weight cutting weight (lb.)	131.1±8.7 lb.
Competition weight (lb.)	109.4 lbs. ±20.6

There was a significant decrease in average weight at time of competition (109.4 lbs. ±20.6 [range: 100–145]) [8.5 kg (range 2.7–13.6)](p<0.0001). The average lowest calorie consumption during weight cutting was 1137.6±137 (range: 800–1450). Eleven (31.4%) reported abnormal menstrual cycles. Two (5.7%) reported an eating disorder (1 bulimia nervosa, 1 binge eating). Calcium and vitamin D supplements are taken by 62.9% of all competitors. No competitors reported ever using anabolic steroids. The average hours of training per day were 2±0.62 (range: 1–3). The total hours of training for the group (n=35) was 625.3 hours per competitor and the total number of injuries reported by all the participants in the last year was 4 (1 rotator cuff tear/tendonitis, 1 shin splints, 2 low back strains) resulting in incidence of 0.18 injuries per 1,000 hours of training. No fractures or stress fractures were reported. Fischer Exact tests revealed that eating disorders are associated with injuries (P=0.014) and age older than 35 is associated with injuries (P=0.005).

DISCUSSION

Injury rate studies have been performed on a variety of fitness related competitors and participants. Siewe et al surveyed 71 competitive and elite male bodybuilders and found they had 0.24 injuries per 1000 training hours.[4] Our reported rate of injury in female fitness competitors is 0.18 injuries per 1000 hours. Our female fitness competitors surveyed had a lower injury rate than other competitive fitness and sporting activities, but we recognize that we only had a survey size of 31 athletes. Siewe surveyed 245 competitive and elite powerlifters and found a 0.3 injuries per 1000 training hours.[5] Winwood surveyed 213 strongman athletes and found 5.5 injuries per 1000 hours.[6] Australian competitive calisthenics was found to have an injury rate of 1.1 per 1000 training hours in a prospective 12 month study by Leaf et al in 550 elite and non-elite participants.[7] The calculated injury rate per 1000 hours of training in our group of female fitness competitors was lower than the previous studies of powerlifters, strongmen, and competitive calisthenic participants. [5-7]

Risk factors for sustaining an injury in female fitness competitors were found to be athletes with either a history of an eating disorder or age over 35. However, due to the nature of the study design, we were unable to delineate if the injuries were related to overuse or the direct result of a traumatic event. There was not a statistical significant difference with the presence of a regular menstrual cycle and injury rates. None of the athletes included in the study had a history of fracture in the past year. The injuries found in this study were similar to other injuries described in other studies involving a resistance training population.[5,8,9]

The bodybuilding culture is predominated by muscle size which inevitably has a relationship to anabolic steroid use.[10] The prevalence of steroid use among female fitness competitors and bodybuilders is not truly known as there is little evidence of testing these competitors in the literature and on competition websites. Male steroid users have been noted in the literature to have an increased rate of cardiac complications including early heart failure and an increased risk of tendon ruptures.[11,12,13] Our study on female fitness competitors did not identify any anabolic steroid use among the participants surveyed.

The range of lowest caloric intake during competition preparation was 800 to 1450 kcal with the average being 1138.3 kcal. Given the retrospective nature of the survey, we relied on estimation by the athlete and did not inquire how they calculated these numbers. This represents a severe caloric restriction during preparation. The athletes on average lost 18.7 lbs [8.5 kg] from pre-competition weight to competition weight. We did not inquire about the number of weeks preparing for the competition or types of supplements used to obtain this weight. Further studies should be done prospectively to specifically evaluate how these competitors lose weight for competition and how they gain weight after competition in hopes to learn more about the physiology and if there is a propensity towards developing an eating disorder.

There are several limitations to our study, which may explain why our injury rate was lower than other studies. Participants may not have admitted to having injuries or answered other questions in a biased way. There is an inherent recall bias when asking athletes to remember how

many injuries they suffered in the past year. The survey was anonymous to attempt to achieve the most accurate responses from the participants. Our survey was designed by this research team and has not been formally validated in any previous studies. Trends in training and philosophy on preparation for the competitions can lead to inherent limitations of the study results. Our study participants were from a regional competition where there are a limited number of coaches for these athletes. Furthermore, long-term risks of competing in multiple competitions also needs to be evaluated in regards to long-term safety, injury risk, and possibility of developing an eating disorder as this is unknown to date in this population of athletes. Finally, 31 surveys are not enough to generalize causality between training hours and injury rates among female athletes. A larger national study should be performed to better characterize the population of female fitness competitors in regards to injury rates and training/competition preparation characteristics. However, this survey acts as the starting point to understanding training patterns, diet, and physique characteristics among a specific population of female athletes that will guide subsequent surveys and even potential interventions.

CONCLUSION

Female fitness competitors have a low injury rate of 0.18 injuries per 1000 hours of training in the short-term. Long-term injury risk and health complications were not evaluated as part of this study. The risk factors associated with injury in our study were age over 35 and athletes either with an active eating disorder or history of an eating disorder. Coaches and healthcare professionals should be aware of the unique training and nutrition characteristics of this group of athletes to improve the safety of this sport. Coaches can use the data on average amount of weight lost per competition to help improve the protocols they give to athletes preparing for competition to ensure that weight loss is not too rapid. Further studies need to be performed to improve practices and learn from these competitors to better provide coaching support and appropriate healthcare recommendations.

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Female Fitness Questionnaire

http://rimed.org/rimedicaljournal/2020/09/2020-09-68-contribution-waryasz-appendix.pdf

Concussion: Mechanisms of Injury and Trends from 1997 to 2019

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ABSTRACT

BACKGROUND: There is limited long-term epidemiological data focused on concussions in the United States.

METHODS: The National Electronic Injury Surveillance System was queried from 1997 to 2019 for concussion diagnoses. National incidence rates, stratified by age and sex, were estimated. Injury mechanisms were ranked.

RESULTS: From 1997 to 2019, there was a 3-fold increase in the diagnosis of concussion from 82,103 (95% CI 77,650–86,555) in 1997 to 261,722 (95% CI 212,156–311,288) in 2019 ($p<0.001$). Fall-related head-injury mechanisms were most common in very young (<5 year old) and older (>65 year old) patients. Sports-related injuries were most common in those age 5–24 years old.

CONCLUSIONS: It remains unclear if the observed two-decade rise in reported concussions represents a true increase in incidence or is indicative of improvements in early detection, diagnosis, and treatment during this time period. Common injury mechanisms described highlight the need for improved age-specific safety recommendations.

KEYWORDS: concussion, head injury, epidemiology, fall, sports

INTRODUCTION

Concussions are mild traumatic brain injuries (TBI) caused by force to the head or neck area, either with or without the loss of consciousness.[1,2] Symptoms include headache, amnesia, sleep disturbances, and emotional lability in addition to behavioral and cognitive changes.[3] A substantial proportion of concussion patients continue to demonstrate persistent neuropsychiatric sequelae even up to one year after injury.[4] Despite the financial and health strains imposed by concussion, there have been relatively few long-term epidemiological studies on concussions in the U.S. Furthermore, the studies available often focus on rates of concussion in narrow age ranges or specific sports. Such a limited focus on young athletic groups may neglect important mechanisms of injury such as non-sport related falls causing concussions in the elderly, which may be on the rise.[5]

This study seeks to better characterize the epidemiology and population trends of concussions in the U.S using the NEISS database over a 23-year period (1996–2019). Additionally, we aim to compare the rates of concussion between individual sports, organized team sports, and non-recreational activities. Understanding long-term epidemiologic trends in incidence and mechanism of injury can guide patient education, optimize age-appropriate prevention, and inform activity-specific outreach strategies.

MATERIALS AND METHODS

The National Electronic Injury Surveillance System (NEISS) database is managed by the Consumer Product Safety Commission.[6] It comprises a sample of approximately 100 hospitals of varying sizes throughout the United States and its territories. The database comprises a nationally representative probability sample of all U.S. hospitals that have at least 6 beds and provide 24-hour emergency department (ED) services. Most, but not all U.S. states and territories, are represented. All major regions of the continental United States are represented. Hospitals are divided into 5 stratum, with 4 strata representing adult emergency departments segregated by the number of annual visits (small, medium, large, very large) and 1 strata for pediatric EDs. Individual EDs are assigned probability weights based upon the inverse probability of selection for hospitals in each stratum. These weights are updated every year to adjust for non-response, hospital mergers, and changes in NEISS sampling frame.

At each institution, trained data abstractors review clinical records for physician-diagnosed injuries and report associated products or activities. Data available in the NEISS database includes patient demographic data, diagnosis, body part injured, involvement of a product or activity, location of injury, and ED disposition. For this study, the NEISS database was queried from January 1, 1997 to December 31, 2019 for all ED visits coded as "concussion" (NEISS code 52). Using NEISS probability sampling weights in order to account for the complex sample design, national estimates for concussion, and the corresponding 95% confidence intervals, were calculated for each year from 1997-2019. All of our national estimates were evaluated for statistical reliability and determined to be at low risk of instability based on the Consumer Product Safety Commission guidelines (number

of records over 20, estimates over 1,200, and coefficient of variation over 33%). Corresponding 2019 U.S. Census intercensal estimates[7] were utilized to calculate the most recent national incidence rate estimates and associated 95% confidence intervals stratified by age and sex.

Utilizing the NEISS coding manual as well as the 2018 NEISS Product Code Comparability table,[8,9] all applicable activities and product codes were recorded over the 23-year study period. For improved comprehensibility of the data, categories of some sports injury mechanisms were created by combining appropriate NEISS codes. (**Appendix 1**)

After stratification of patients by age, specific mechanisms of injury were evaluated and the most common mechanisms according to the nationally-weighted estimates were ranked for each age group. Additionally, the team and individual sports most commonly associated with concussions were ranked. For improved comprehensibility, only sports responsible for at least 1% of the total concussions within each category were reported. Survey-adjusted Student's t-test and Rao-Scott modified chi-squared analysis were used for direct comparisons of means and proportions, respectively. Trends in hospital admission rates from 1997–2019 were evaluated using linear regression models.

Microsoft Excel version 16.11.1 (Microsoft Corporation, 2017, Redmond, Washington) was used for data collection and visualization. All statistical analyses were performed using Stata 15.0 (StataCorp., 2017, College Station, TX). Statistical significance was defined as $p < 0.05$ *a priori*.

All data analyzed has been previously de-identified and is made publicly available by the Consumer Product Safety Commission. As such, no Institutional Review Board approval was required for this study.

RESULTS

Demographics, Epidemiology, and Trends Over Time

From 1997 to 2019, 136,592 patients presented to EDs with concussions, representing a national weighted estimate of 4,471,431 (95% CI 4,323,218–4,619,644). There was a 3-fold increase in the estimated incidence of concussions presenting to EDs from 82,103 (95% CI 77,650-86,555) in 1997 to 261,722 (95% CI 212,156-311,288) in 2019 (R^2= 0.9036, p<0.001, **Figure 1**). When stratified by age, this increase was most pronounced in those age 5-24, but occurred among all age groups (all p>0.05, **Figure 2**). Males were significantly more likely to be affected in both of these age groups than females (p<0.05, **Table 1/Figure 3**). Males age 5-14 represented the highest-risk group, presenting with 233.9 concussions per 100,000 person-years (95% CI 219.8-248.1).

In 2019, the estimated yearly incidence rate for concussions was 81.0 per 100,000 person-years, 95% CI 78.6–83.3 (**Table 1**). Overall, males presented more often (86.0 concussions per 100,000 person-years, 95% CI 82.5–89.5) than females (76.2 per 100,000 person-years, 95% CI 72.–79.4, p<0.001); however, this varied by age group (**Table 1**). In those younger than 25, males were more at risk, making up 58.7% of all concussions, whereas females were more at significantly increased risk among those 25 or older, making up 64.3% of all concussion in this group (p<0.001). The highest incidence rate of concussion was in children age 5–14 (186.4 per 100,000 person-years, 95% CI 177.3–195.4), followed by young adults age 15-24 (170.4 per 100,000 person-years, 95% CI 161.6-179.2), (**Figure 2**).

Figure 1. Yearly Concussion Estimate, 1997–2019

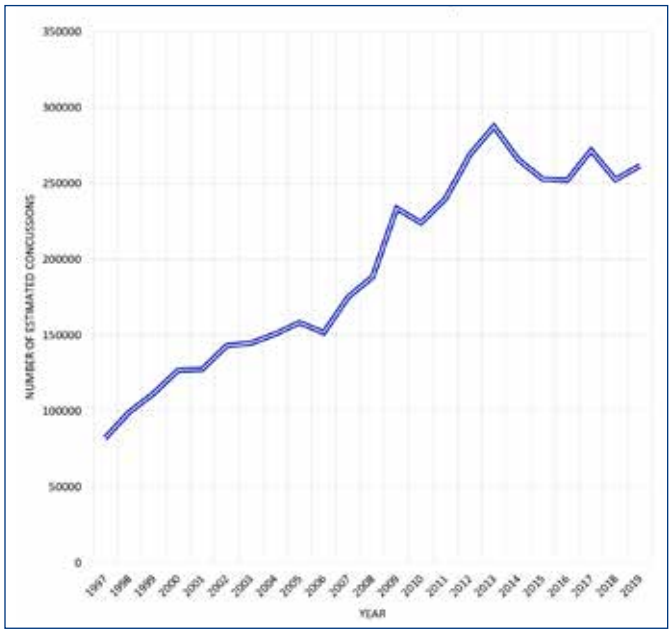


Figure 2. Yearly Concussion Estimates, Stratified by Age, 1997–2019

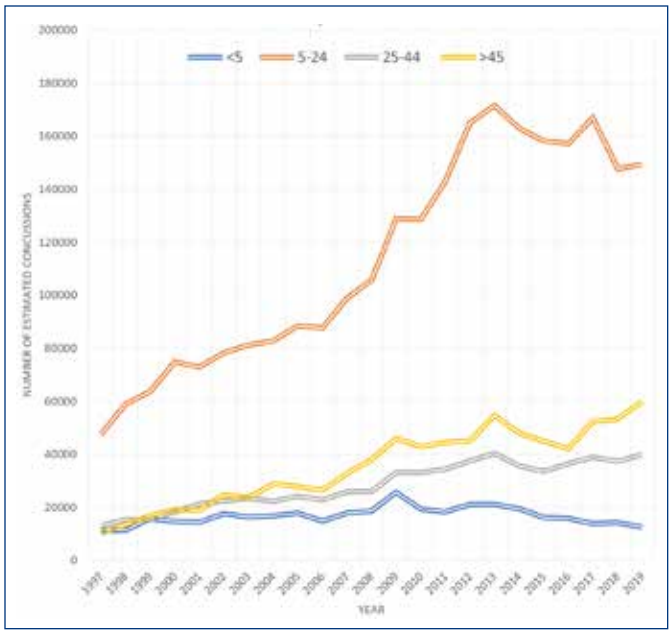
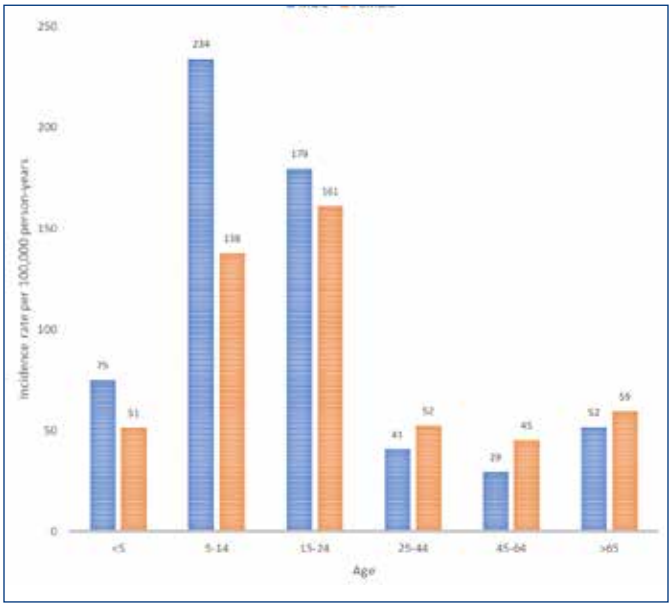


Table 1. Number of Cases, National Weighted Estimates, and Incidence Rate of Concussion Stratified by Age and Gender in 2019

Age/Sex	n (2019)	Estimate (2019)	Incidence/100,000 Person-Years
<5	656	12,615	63.3
Male	389	7,642	75.0
Female	267	4,999	51.3
5–14	3,559	77,234	186.4
Male	2,255	48,994	233.9
Female	1,304	28,240	137.8
15–24	2,324	72,157	170.4
Male	1,222	38,447	179.4
Female	1,102	33,710	161.2
25–44	928	31,276	36.6
Male	414	17,378	40.8
Female	514	22,482	52.3
45–64	709	31,276	37.6
Male	278	11,830	29.4
Female	431	19,446	45.2
≥65	565	28,554	55.9
Male	220	11,908	51.6
Female	345	16,646	59.4
ALL	8,741	261,722	81.0
Male	4,778	136,226	86.0
Female	3,963	125,496	76.2

Figure 3. Incidence Rate for Concussion, Stratified by Age and Gender, 2019



Most Common Mechanisms of Injury

The most common mechanisms of injury varied substantially by age (**Table 2**). Fall-related head-injury mechanisms such as floors, stairs, beds, and chairs/couches were most common in very young (<5 year old) and older (>65 year old) patients. Conversely, sports-related injuries were especially common in children, adolescents and young adults (age 5–24). Football was the most common cause of concussion in this population, accounting for 16.4% of all concussions sustained by 5–14 year olds and 18.3% of all concussions in 15–24 year olds.

Table 2. Most Common Injury Mechanisms Leading to Concussion by Age 1997–2019

Rank	Age					
	<5	**5 to 14**	**15 to 24**	**25 to 44**	**45 to 64**	**≥65**
1st	Floors (13.1%)	Football (16.4%)	Football (18.3%)	Stairs (11.5%)	Stairs (14.2%)	Floors (22.7%)
2nd	Stairs (11.4%)	Bicycles (8.3%)	Basketball (8.8%)	Floors (8.0%)	Floors (11.4%)	Stairs (14.6%)
3rd	Beds (8.4%)	Basketball (7.6%)	Soccer (8.2%)	Bicycles (7.2%)	Bicycles (8.3%)	Beds (6.0%)
4th	Couches (5.2%)	Floors (6.1%)	Bicycles (4.6%)	Baths/ Showers (4.0%)	Ladders (4.7%)	Chairs (3.9%)
5th	Tables (4.9%)	Soccer (5.4%)	Stairs (3.9%)	Horseback Riding (3.2%)	Horseback Riding (4.0%)	Baths/ Showers (3.4%)

Some injury mechanisms were unique to specific age groups. For example, injuries from beds were only commonly seen in those <5 years old and >65 years old. Injuries from tables and couches were only seen in those <5 years old. Concussions cases by horseback riding were only seen commonly in those 25–64. Concussions related to wheelchairs (2.4%), crutches, canes, and walkers (2.1%) were only seen in those > 65 years old.

Sports-Related Concussions

From 1997 to 2019, there were an estimated 753,295 (95% CI 743,151–763,438) concussions caused by individual sports and an estimated 1,139,529 (95% CI 1,128,781–1,150,270) injuries related to team sports that resulted in an ED visit. The most common individual sport responsible for ED visits was cycling, representing 292,111 (95% CI 284,720–299,501) concussions or 38.8% of all individual sport-related injuries during the study period. Additional common individual sports leading to concussion were horseback riding (11.3%), skateboard/scooter riding (10.3%), and snowboarding (8.9%). The most common team sport that led to an ED evaluation for concussion was football, which was responsible for an estimated 458,613 (95% CI 449,683–467,543) cases, or 40.3% of all team sport-related injuries. This was

Table 3. Most Common Team and Individual Sports Associated with Concussions

	Estimated Frequency 1997–2019	Percent
Individual Sport		
Cycling	292,111	38.8%
Horseback Riding	85,285	11.3%
Skateboard/Scooter	77,671	10.3%
Snowboarding	67,169	8.9%
Snow Skiing	60,262	8.0%
Wrestling	40,979	5.4%
Exercise (without equipment)	22,848	3.0%
Martial Arts	12,549	1.7%
Boxing	11,313	1.5%
In-line Skating	11,252	1.5%
Exercise equipment (excl. weight lifting and gymnastic equipment)	10,761	1.4%
Gymnastics	10,356	1.4%
Dancing	9,136	1.2%
Swimming	8,824	1.1%
Other	32,777	4.3%
Team Sport		
Football	458,613	40.3%
Basketball	211,139	18.5%
Soccer	184,385	16.2%
Baseball/Softball	146.405	12.9%
Hockey	42,261	3.7%
Cheerleading	30,968	2.7%
Volleyball	25,556	2.2%
Lacrosse	20,962	1.8%
Rugby	18,351	1.6%

followed by basketball (18.5%), soccer (16.2%), baseball/softball (12.9%), and hockey (3.7%). Patients presenting with concussions from team sports were younger than those injured during individual sports (mean age 16.1 versus 22.8, p<0.001). (**Table 3**)

DISCUSSION

This investigation utilized the NEISS database to identify the national incidence of concussions presenting to EDs in the U.S. from 1997 to 2019. The reported incidence has been rising over the last two decades, reaching a peak in 2013. The observed 3-fold rise over the 23-year study period is particularly pronounced in the 5- to 24-year-old age group, but occurred across all ages. It remains unclear whether this represents a true increase in concussion rate, the effects of greater public awareness and diagnosis, or a combination thereof.

The Centers for Disease Control launched the *Heads Up* initiative in 2003, a program aimed at raising concussion awareness in healthcare providers, youth sports' coaches, and athletes. A 10-year analysis of the program demonstrated better identification of concussive symptoms by coaches, as well as better management of concussion by healthcare professionals.[10] Similarly, the modern surge in social networking has been shown to increase concussion awareness and peer-to-peer support, particularly in young adults.[11] Regardless of the cause, this upsurge in diagnosed concussions over the last 2 decades indicates a need for further understanding of the demographics most affected and the mechanisms that lead to concussions. Better epidemiologic data may lead to improvements in preventative measures, diagnosis, treatment strategies, and utilization of finite resources.

The highest rate of ED visits for concussions was found in the 5- to 24-year-old age range, with football being the most common mechanism of injury. Other significant mechanisms of injury in this age range included bicycles, basketball, and soccer. This closely coincides with trends described by Buzas et al.[12], who reported a rank order of concussion in 4-to 13-year-old children including football, followed by basketball, soccer, and baseball. Their study did not stratify by sex or include concussions that resulted from individual sports or non-sport mechanisms of injury. The rise in sport-related concussions in this age demographic parallels increased participation rates in these sports.[13] When comparing sports-related concussions in males versus females, it is important to note that some sports, such as ice hockey, have different contact rules for men's and women's competition. It has been noted that even in sports that limit or restrict contact, such as soccer, basketball, and women's ice hockey, the majority of injuries still occur via player contact.[14] Based on this data, targeted outreach to prevent concussions in this age group could include initiatives to increase bicycle helmet use, promote head protection or limit head contact in youth soccer and basketball, and eliminate or reduce full contact football practices.[14–16]

Females sought medical attention at EDs for concussions at significantly greater rates than males in the 45–64 and >65 age groups, with the most common mechanisms including falls on floors, stairs, beds/bedframes, and bathtubs/showers. Interventions in this age group designed to reduce the rate of concussion may include appropriate patient and caregiver education, gait assessment to identify those who may benefit from physical therapy, balance and strength exercises, and removal of home hazards such as loose rugs and bathmats.[17] Appropriate discontinuation of psychotropic medications and reduction in polypharmacy have also been associated with decreased falls in the elderly.[17] Similar mechanisms of injury related to falls in the home were observed in the age group <5 years. Prevention in this group may include parent education, improvement in home safety measures, limiting use of bunk beds, maintaining playground equipment, and

use of stationary activity centers in place of infant walkers.[18]

Strengths of this study include the ability to calculate accurate nationwide estimates for concussions presenting to EDs through NEISS probability-sampling weights. Furthermore, the study encompasses a relatively long 23-year study period, which allows us to analyze trends over time. However, there are several potential study limitations that should be noted. This study only evaluates the incidence of patients presenting to EDs for concussive symptoms, thus the true incidence of concussion may be higher than our estimates. Patients may self-treat their symptoms or be assessed in a non-emergency setting by primary care physicians, pediatricians, athletic trainers, or at urgent care centers. These patients would not have been captured in this database. Additionally, beyond basic demographic data, the NEISS database does not offer specific patient characteristics which may have added another layer of sophistication in the analysis. Furthermore, the database also does not provide specific narrative data regarding the injury mechanism, method of treatment undertaken, or patient-specific subjective and objective outcomes measures. Finally, the accuracy of the data included in NEISS is ultimately dependent upon accurate coding. While the NEISS coding manual utilized a single code for concussion throughout the study period which may result in improved coding consistency, as with any large coding-reliant database, variations in interpretation of diagnostic criteria among physicians and coders may still affect the underlying accuracy of the data.

CONCLUSIONS

This study underscores the importance of concussions as an increasingly prevalent and important clinical problem affecting all age groups with substantial implications for public health. We observed a significant increase in the incidence of concussions presenting to EDs between 1997 and 2019 in all age groups. It remains unclear whether this is secondary to a true rise on concussion rates or more likely due to increasing public awareness and earlier presentation, diagnosis, and treatment of patients following concussion. This investigation highlights common age-specific injury mechanisms and may inform possible future avenues for age-specific concussion prevention measures. Further research is necessary to evaluate the effectiveness of such interventions.

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Rhode Island Monthly Vital Statistics Report
Provisional Occurrence Data from the Division of Vital Records

VITAL EVENTS	REPORTING PERIOD		
	MARCH 2019	12 MONTHS ENDING WITH MARCH 2020	
	Number	Number	Rates
Live Births	948	11,188	10.6*
Deaths	957	10,566	10.0*
Infant Deaths	5	60	5.4#
Neonatal Deaths	3	47	4.2#
Marriages	208	6,555	6.2*
Divorces	194	2,830	2.7*

* Rates per 1,000 estimated population

Rates per 1,000 live births

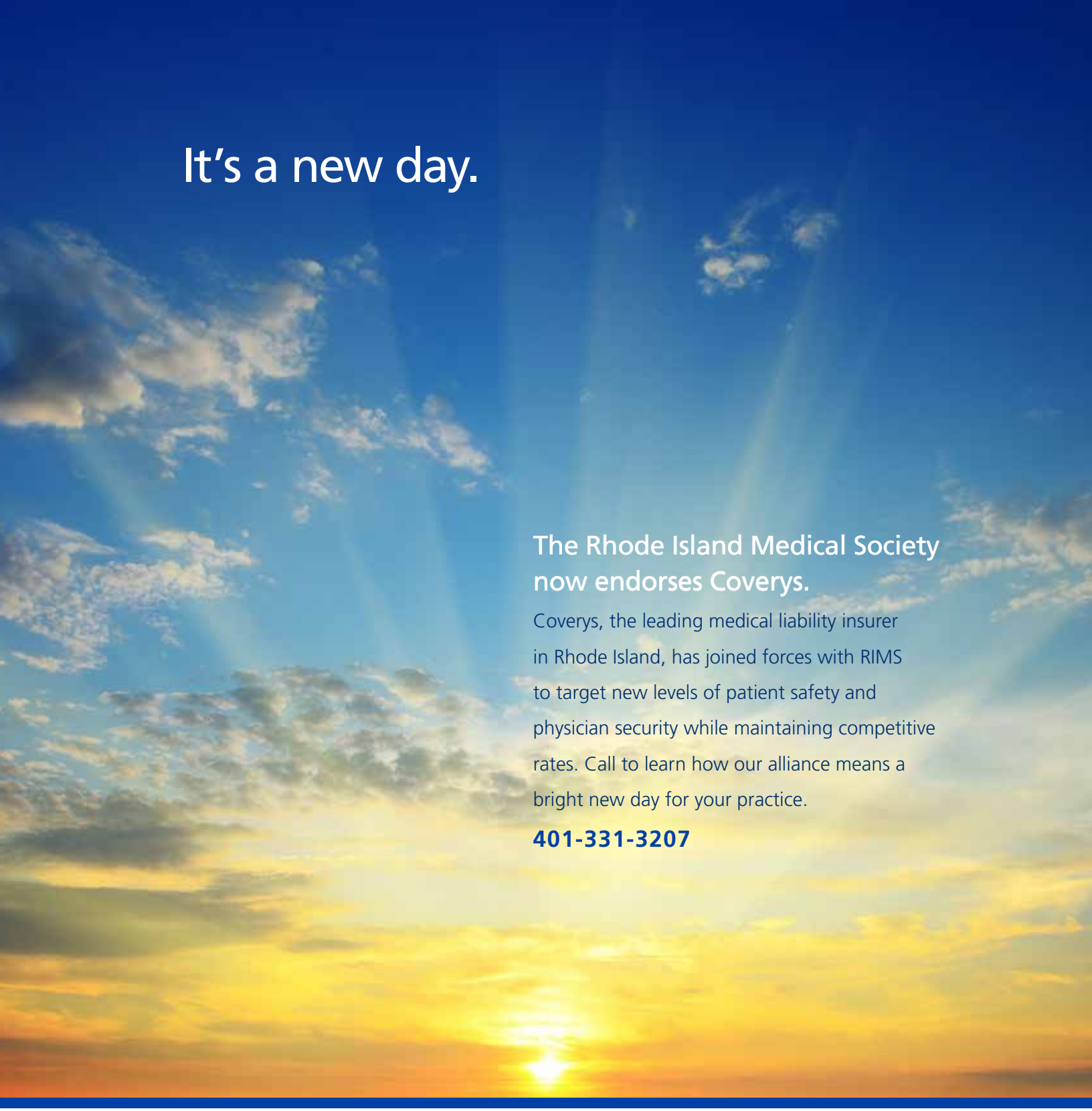
Underlying Cause of Death Category	REPORTING PERIOD			
	SEPTEMBER 2019	12 MONTHS ENDING WITH SEPTEMBER 2019		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	188	2,492	235.2	3,384.5
Malignant Neoplasms	197	2,266	213.9	4,882.0
Cerebrovascular Disease	32	467	44.1	420.0
Injuries (Accident/Suicide/Homicide)	64	910	85.9	12,663.5
COPD	29	492	46.4	550.0

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,056,298 (www.census.gov)

(c) Years of Potential Life Lost (YPLL).

NOTE: Totals represent vital events, which occurred in Rhode Island for the reporting periods listed above.
Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.



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RIMS NOTES: *News You Can Use*

RHODE ISLAND MEDICAL SOCIETY

Friday, June 19, 2020

RIMS NOTES: NEWS YOU CAN USE

Welcome to the current issue of *RIMS Notes*, an exclusive member benefit. At this time, we are focusing our efforts on delivering timely information on COVID-19. Please check our website for up-to-the-hour updates, and your inbox for breaking news.

Volume 8 - Number 13

This issue sponsored by HUB

For more information about our Sponsors, please visit here.

A Message from RIMS President

MEDICAL SOCIETY STATEMENT ON DEATH OF GEORGE FLOYD Attached is a statement of the Rhode Island Medical Society regarding recent events.

Advocacy

June is Pride Month. Click here for a brief reflection on the significance for organized medicine.

GENERAL ASSEMBLY: Both chambers returned to the State House this week to pass a supplemental budget for the current state fiscal year that ends on June 30, and bills specific to certain cities and towns. The State House remained closed to the public. Leadership is targeting the week of July 13 to re-convene to finish crafting a new state budget for FY 22 and to deal with some of the large amount of proposed legislation that was left up in the air when the Governor declared the COVID-19 public health emergency in mid-March.

Physician Wellness and Stress

Physician Support Line for physicians and other healthcare workers:

- **Care Now England/Lifespan Support Hotline for ANY healthcare worker: 401-606-6000**
- **National Physician Support Line: 888-409-0141 (9:00 AM to 3:00 AM next day EST)**

https://www.physiciansupportline.com/

The Physician Support Line was created in March to provide confidential peer-to-peer support for MD's/DO's working on the frontlines of COVID-19. The Line is staffed by over 700 volunteer psychiatrists from across the country seven days per week.

What's New from the AMA

US Health and Human Services to distribute $15 billion to eligible Medicaid and CHIP physicians and organizations. Webcasts on 6/22 and 6/25 for interested physicians. Registration is required.

Q: Why is there a new Provider Relief Fund Payment Portal?
A: Portal will initially be used for new submissions from Medicaid and Children's Health Insurance Program (CHIP) providers seeking payments under the Provider Relief Fund starting Wednesday, June 10, 2020. At this time, this portal will serve as the point of entry for providers who have received Medicaid and CHIP payments in 2017, 2018, 2019 or 2020 and who have not already received any payments from the $50 billion Provider Relief Fund General Distribution.

What's New

Kathleen Boyd, MSW, LICSW, Director of RIMS' Physician Health Program, has been elected to the Board of Directors of the Federation of State Physician Health Programs (FSPHP). The FSPHP is an independent, non-profit, professional organization that supports education and research and promotes best practices to enhance the effectiveness of its 40 state physician health program members. Ms. Boyd also serves on the Performance Enhancement and Effectiveness Review (PEER) Committee of the FSPHP.

The biweekly e-newsletter exclusively for RIMS members.

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RIMS NOTES
is published electronically on alternate Fridays.

Contact Dulce Cosme if you've missed an issue, dcosme@rimed.org.

Working for You: RIMS advocacy activities

August 4, Tuesday

Health Rosetta Summit
"Re-localizing Health Care" webinar

RIMS Physician Health Committee:
Herbert Rakatansky, MD, Chair
(via teleconference)

August 5, Wednesday

Governor's Overdose Prevention and Intervention Taskforce, Racial equity work group

August 6, Thursday

Governor's Legal Counsel regarding immunity (via teleconference)

August 7, Friday

Meeting with Legal Counsel regarding RI Attorney General's pharma lawsuit

Alpert Medical School Student Activity Fair (via Zoom)

Meeting with Physician Health Program consultant

August 10, Monday

Consultations with Accreditation Council for Continuing Medical Education

August 11, Tuesday

COBRE webinar regarding stigma and People Who Use Drugs (PWUD)

AMA Federal Advocacy call regarding Centers for Medicare & Medicaid Services (CMS) proposed Medicare fee schedule (via teleconference)

Call with AMA regarding RI Attorney General's pharma lawsuit

August 12, Wednesday

Board of Medical Licensure and Discipline

Governor's Overdose Prevention and Intervention Task Force:
Sarah Fessler, MD, Past President

August 18, Tuesday

Office of the Health Insurance Commissioner (OHIC), Health Insurance Advisory Committee

Donna Stetson joins RIMS' Physician Health Program team

The Rhode Island Medical Society recently welcomed **DONNA STETSON, LMHC**, to its staff as Clinical Associate. Ms. Stetson brings a wealth of education and experience to this new position, which the Society was able to create under a grant from the Rhode Island Foundation. Ms. Stetson will work exclusively for the Society's Physician Health Program under the direction of Kathleen Boyd, MSW, LICSW. The new position permanently expands the Program's capacity to serve the community.

A licensed psychotherapist, Donna Stetson is a Wellesley College alumna with a master's degree in psychology from Northeastern University and a graduate certificate in Human Resources Management from Bryant University. She is a seasoned Employee Assistance Professional (EAP) who provided case management, counseling and coaching services to staff at a variety of organizations, including New England Medical Center, Mount Auburn Hospital and Massachusetts Medical Society. Her clinical focus includes cognitive behavioral therapy and mindfulness techniques to help clients better manage anxiety, depression, stress and grief. Prior to moving into the mental health field, Donna spent 10 years in management consulting conducting primary research, facilitating focus groups and providing corporate training and public speaking.

August 19, Wednesday

Department of Health (DOH) Diabetes Prevention Program Stakeholder Network (via teleconference)

August 20, Thursday

RI Orthopedic Society (RIOS) teleconference with US Senator Jack Reid

Providence Business News recognition of RIMS' Board Chair, Dr. Peter Hollmann, as "Leader and Doer"

August 24, Monday

AMA/State Medical Society Public Health LEADS regarding COVID-19

August 25, Tuesday

AMA Advocacy Resource Center:
The need for better drug overdose surveillance data

August 26, Wednesday

Meeting with Elizabeth Samuels, MD, subject matter expert Governor's Overdose Task Force regarding Overdose Prevention Centers a.k.a Safe Injection Facilities (SIF)

Meeting of RIMS ad hoc Committee on Governance: Yul D. Ejnes, MD, Chair

August 27, Thursday

OHIC Telemedicine Advisory Group, Peter Hollmann, MD

Racial Equity Work Group
(via teleconference)

RIMS CORPORATE AFFILIATES

The Rhode Island Medical Society continues to drive forward into the future with the implementation of various new programs. As such, RIMS is expanded its Affinity Program to allow for more of our colleagues in healthcare and related business to work with our membership. RIMS thanks these participants for their support of our membership.

Contact Marc Bialek for more information: 401-331-3207 or mbialek@rimed.org

Neighborhood Health Plan OF RHODE ISLAND™

www.nhpri.org

Neighborhood Health Plan of Rhode Island is a non-profit HMO founded in 1993 in partnership with Rhode Island's Community Health Centers. Serving over 185,000 members, Neighborhood has doubled in membership, revenue and staff since November 2013. In January 2014, Neighborhood extended its service, benefits and value through the HealthSource RI health insurance exchange, serving 49% the RI exchange market. Neighborhood has been rated by National Committee for Quality Assurance (NCQA) as one of the Top 10 Medicaid health plans in America, every year since ratings began twelve years ago.

RHODE ISLAND PHYSICIANS CORPORATION PRIMARY CARE

www.ripcpc.com

RIPCPC is an independent practice association (IPA) of primary care physicians located throughout the state of Rhode Island. The IPA, originally formed in 1994, represent 150 physicians from Family Practice, Internal Medicine and Pediatrics. RIPCPC also has an affiliation with over 200 specialty-care member physicians. Our PCP's act as primary care providers for over 340,000 patients throughout the state of Rhode Island. The IPA was formed to provide a venue for the smaller independent practices to work together with the ultimate goal of improving quality of care for our patients.

RIMS gratefully acknowledges the practices who participate in our discounted Group Membership Program

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For more information about group rates, please contact Marc Bialek, RIMS Director of Member Services

Q&A with Dr. Ashish K. Jha, New Dean of the Brown School of Public Health

MARY KORR
RIMJ MANAGING EDITOR

Today, September 1, **ASHISH K. JHA, MD, MPH,** assumes the leadership of the Brown University School of Public Health as Dean. He most recently served as Director of the Harvard Global Health Institute, K.T. Li Professor of Global Health at the Harvard T.H. Chan School of Public Health, and is a Professor of Medicine at Harvard Medical School and a practicing internist at the V.A. Boston Healthcare System.

Dr. Jha will be the third person to hold this position, selected after a national search. He succeeds Bess H. Marcus, PhD, who announced in December 2019 she would be stepping down from the position to resume fulltime research and teaching.

The School opened in 2013 under the leadership of Terrie "Fox" Wetle, PhD. It was accredited in 2016 during her tenure, and now has approximately 250 faculty members and 400 undergraduate and graduate students and more than a dozen research centers.

In an effort to introduce Dr. Jha to RIMJ readers, the editors and several physicians compiled the following questions for his response on a host of issues – from his early influences, to public health during the pandemic, and last but not least, given the theme of this issue is sports medicine – a question about the New England Patriots!

RIMJ: You came to this country as a child, born in India and immigrating with your family to Canada and then the US. The immigrant experience is formative and often a powerful one. Can you describe its impact on your own life and your career in medicine and public health?

DR. JHA: My immigrant experience shaped my path toward a career in medicine and public health deeply. Growing up, I was often excluded from opportunities, treated as an outsider. So over time, as I have come into more leadership positions, I have made creating an inclusive environment a central part of my mission. I believe that, as the old saying goes, talent is evenly distributed but opportunity is not. It is critical for leaders to create opportunities for those who do not readily have them.

RIMJ: You are assuming the leadership at the Brown School of Public Health during a pandemic. What are your top priorities as you begin your new position in a time of crisis, when you have to address current conditions yet keep an eye to the future?

DR. JHA: There has never been a more difficult set of challenges facing public health – but also, a greater set of opportunities. We often say in public health that you never notice our successes. You never notice when the air is clear, when the water isn't contaminated, when a disease outbreak isn't happening. But this moment, when our nation's long-standing under-investment in public health has laid bare the importance of public health, it has, for me, raised several key priorities for Brown SPH.

First, we must create a deep and concerted effort to prevent and more effectively respond to pandemics. I do believe that this will be an important area of work.

PHOTO COURTESY OF HARVARD GLOBAL HEALTH INSTITUTE

Brief Bio: Ashish K. Jha, MD, MPH

EDUCATION/TRAINING

MPH, Harvard School of Public Health, 2004

MD, Harvard Medical School, 1997

AB, Economics, Columbia College, 1992

Fellow, Internal Medicine, Brigham & Women's Hospital, Harvard Medical School, 2002–2004

Chief Resident, University of California, San Francisco, 2000–2001

Resident, University of California, San Francisco, 1997–2000

LEADERSHIP

Member, Institute of Medicine (IOM) at the National Academies of Sciences, Engineering, and Medicine, 2013

Special assistant to the Secretary, Department of Veterans Affairs, 2009– 2013

RESEARCH AREAS

Improving the quality of health care systems with a specialized focus on how national policies impact care, population health.

Co-chaired an international commission on the global response to the 2014 Ebola outbreak in West Africa, and examining/developing strategies to strengthen pandemic preparedness and response.

Written extensively on the roles of international agencies such as the WHO and how they can be made more effective in ID outbreaks, such as Ebola, Zika and now SARS-CoV-2.

PHOTO: RIMJ

In a virtual forum hosted by the Brown University School of Public Health on Aug. 7th, **Dr. Anthony Fauci**, director of the National Institute of Allergy and Infectious Diseases at the National Institutes of Health, joined **Dr. Ashish K. Jha** to address the challenges public health leaders face in addressing the pandemic. Several thousand joined the session and Q&A which followed. It can be viewed here: https://www.brown.edu/news/2020-08-07/fauci-sph

Second, climate change, which is accelerating disease outbreaks, is going to have a profound impact on the health of the world, including right here in Rhode Island. I believe Brown is uniquely poised to lead that effort.

Third, it is clear to me that the health care systems of Rhode Island are undergoing profound changes. In that light, this is a unique opportunity to make Rhode Island a model for how tying together clinical, public health, and other forms of data can profoundly improve the health of the people of this state. And Brown University is the natural partner for that. So, I see a big push towards data science at the university with a key role for the public health school.

Finally, I think it is critical that we as a school directly address the systemic racism that plagues our nation. There are many facets of that work but creating a greater level of diversity and ensuring a more inclusive environment are critical to that. More on that to come.

RIMJ: Given the recent *Nature Medicine* articles suggesting the resolution of neutralizing antibodies within the early convalescent phase, do you think COVID-19 will remain an epidemic/pandemic for the foreseeable future (3–5 years)?

[Long, Q., Tang, X., Shi, Q et al. Clinical and immunological assessment of asymptomatic SARS-CoV-2 infections. *Nat Med* (2020). https://doi.org/10.1038/s41591-020-0965-6]

DR. JHA: While this is an important study, I don't think that it tells us that immunity will wane quickly. All of the evidence so far suggests that there is also an important cellular immune response to SARS-CoV-2 and in many diseases, antibody levels can wane but rise up again when confronted with the pathogen.

We are 7 months into this outbreak and have not seen many convincing cases of re-infections. I remain hopeful that immunity will last long enough that a vaccine can be a feasible approach to raising population immunity and bringing the outbreak under control, ideally sometime in 2021.

RIMJ: How much time do you think will be required to assure a fair modicum of safety for a COVID-19 vaccine and what sort of risks can be "reasonably" accepted with each shortening of a testing period during the clinical trials and what sort of risks can be "reasonably inferred," if any, to a vaccine, from the characteristics of the virus or the vaccine?

DR. JHA: The most optimistic estimates are that it will take 12 more months to develop a vaccine. Obviously, speed is important here, but safety is just as important. As we enter phase 3 trials, I will be paying close attention to things like antibody-mediated disease enhancement and other effects. I suspect we will have reasonably good safety data within about 6 months. Obviously, it will not be long-term data – for that, we will have to wait years, but given that the disease is killing so many Americans and people around the world, my hope is that by the end of 2020, we will have the kind of data we need to feel comfortable getting vaccinated.

RIMJ: If/when a vaccine or vaccines are available for large-scale public uses, what are likely to be major barriers to uptake? Do you think there will be those who refuse to be vaccinated? (E.g., science deniers, mistrust of government, conspiracy theories, vaccine unsafe/rushed/won't work?)

DR. JHA: Anti-vaccine sentiment will definitely be a barrier to uptake. Some of the polls have shown about 20–25% of Americans would not want to get a SARS-CoV-2 vaccine so that will be a barrier. Public health officials will have to think about effective messaging around the vaccine, including being transparent

and open with people about what the vaccine data does and does not show. Equitable distribution, both domestically and globally, will also be an important issue to confront. We have to do some advance planning – and organizations like the WHO and others are working hard to ensure that the vaccine is able to get out to much of the world, and not just to those who are wealthy in wealthy nations.

RIMJ: Why do you think a percentage of Americans are unwilling to accept scientific principles and observations and is this different than in other countries?
DR. JHA: There are science skeptics in other nations too – but the sheer amount of misinformation, spread through Facebook and other social media channels, is profound and deeply disturbing here. Also, in many other nations, that misinformation is largely ignored by political leaders, whereas, in the US, many political leaders use and amplify that misinformation.

RIMJ: For many physicians the pandemic has provided an opportunity to speak about public health, but there are growing concerns that people are "over" the pandemic. There has also been a backlash against public health officials advocating for safety measures such as wearing masks, social distancing, and phased-in re-openings. How can we continue to hold interest in public health issues and remain part of the public conversation about health and policy?
DR. JHA: I appreciate "quarantine fatigue" – and it's true that there are considerable economic and mental health effects of the public health measures we have taken. I do believe that at this point, we don't need total lockdowns and instead, need nuanced approaches, acknowledging side effects.

I think physicians as public health advocates are uniquely situated to deliver this information. We deal with uncertainty and trade-offs all day in clinics and wards. But uncertainty does not paralyze us – we know that as clinicians, we still have to act.

The patient is the population struck by and susceptible to COVID-19. We need to communicate openly and honestly about what we know, what we don't know, and how to get through the next year in a way that maximizes health while preserving livelihoods.

RIMJ: What would you like to see change in medicine to improve inclusiveness in light of the recent national conversation surrounding dismantling racism and what do you see as the biggest opportunity as we reimagine medicine after the pandemic? (E.g., focus on mental health, housing for homeless individuals, more efficient and widespread public health screening, telehealth, overcoming structural inequities in the healthcare system that disproportionately affects marginalized and minority communities, as data has shown in the current COVID-19 crisis.)
DR. JHA: There are so many opportunities here. The recent national conversation around racial justice, combined with the pandemic, has left the door wide open for critical and necessary reforms. We need more medical and public health professionals from underrepresented groups – we need to make changes to the pre-health pipeline to bring in underrepresented minorities, and we need to build an environment in academic medicine that makes people feel included in a way that creates belonging.

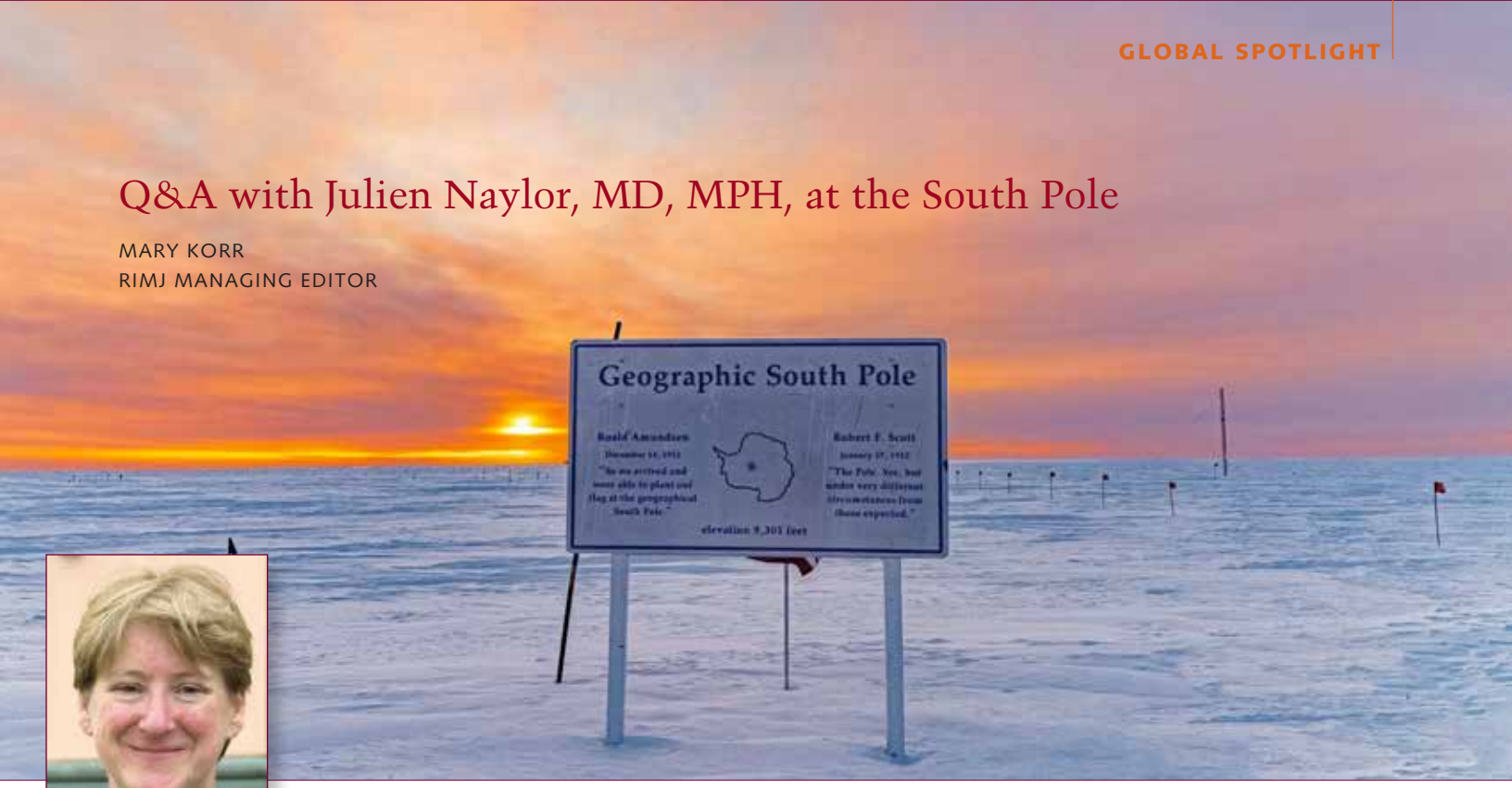
There are many strategies. Training is one important one, but it is not enough. We need greater diversity in our leadership, and we need to speak openly about policies that directly harm our patients. These are natural roles for physicians – as advocates for their patients – and I believe we can and should engage in those discussions.

RIMJ: What do you feel will be the most reasonable approach toward mitigating viral transmission while providing a proper education at the university and K–12 level? Remote learning was useful, but not deemed to be adequate in the long term by most educators.
DR. JHA: I'm a big believer in getting schools open. It's good for kids; it's good for parents. But we have to be smart about it. First, we need to ensure that we open up schools only when community transmission is low enough to warrant it. Second, we need to make important changes to the spaces where people will be – classrooms, hallways, etc., focusing on better ventilation and so forth. Finally, we need to ensure we have some sort of a surveillance strategy, ideally, with testing. If we base our decision on data and science, I believe in many parts of the country, we can get schools open safely for much of the academic year ahead.

RIMJ: And finally, how do you think the Patriots will do without Tom Brady?
DR. JHA: This is a provocative and upsetting question. To lose the greatest quarterback is painful – to have him gone in the middle of the pandemic is awful. Now we get to figure out what the magic formula was – was it Belichick, Brady, or the combination? Sadly, I suspect it was the combination and we will be in the wilderness hunting for the next Lombardi trophy for some years to come. ❖

Q&A with Julien Naylor, MD, MPH, at the South Pole

MARY KORR
RIMJ MANAGING EDITOR



The sun hovers on the horizon a few days before it set for the next six months at Amundsen-Scott South Pole Station. The South Pole has one sunset and one sunrise every year. The sun will appear above the horizon again in late September. Photo taken March 15, 2020. [DANNY HAMPTON, NSF]

The austral winter in Antarctica extends from mid-February through October. **JULIEN NAYLOR, MD, MPH,** is currently serving as physician at the Amundsen-Scott South Pole Station, managed by the National Science Foundation (NSF) during this period of complete isolation, along with 42 other researchers, scientists, and supply and operational personnel. Currently Antarctica is the only continent on Earth without a confirmed case of COVID-19.

Contact with the outside world is mostly limited to about nine hours a day when communications satellites are visible, allowing Internet and telephone connectivity. Iridium telephone is available for use 24-hours a day. There is no television or radio reception, other than that available via the Internet. Through the NSF, RIMJ was able to reach out to Dr. Naylor, to ask her about her experiences thus far wintering-over at the South Pole.

RIMJ: What drew you to work in Antarctica and how long is the usual tenure?

DR. NAYLOR: I had considered applying for the job soon after finishing residency but was not an American citizen, so I was unable to do so. Now well into my medical career as an internist, I was asked by my family nurse practitioner (FNP) friend/colleague if I would be interested in wintering with her at the South Pole: a 10-month commitment. I initially hesitated, given the length of the commitment; however, after careful consideration and discussion with my husband, I decided to apply. I have worked during my career in Alaska with the Alaska Native population and always enjoyed the challenges of rural practice. I was intrigued by the idea of work in the most isolated practice under USA management.

RIMJ: Describe your typical daily/weekly schedule.

DR. NAYLOR: The South Pole Station has a crew of 42 relatively healthy people who have been screened carefully before being allowed to spend the winter at the station. Once the station closes to any outside traffic in mid-February, we become self-sufficient. The medical clinic – myself and an FNP – provide day-to-day general medical care to the crew. We see a very small number of patients during the week. Despite the low-patient census, we are tasked with making sure that the clinic is prepared to deal with an emergency that might happen. We train our Emergency Medical Team (a volunteer group) to assist in emergencies and be able to provide basic first aid support if needed.

Inside the South Pole Station medical clinic. [USAP, NSF]

A newborn seal pup bonds with its mother. The seal pup will stay with its mother for a little over a month until it learns to swim and is weaned from its mother's milk. [MIKE LUCIBELLA, NSF]

RIMJ: What is the spectrum of medical conditions, illnesses and injuries seen at the medical facility there?

DR. NAYLOR: Most of our visits are related to sports injuries, insomnia, headaches, GI issues, and dermatologic conditions. Due to the closed station, we have no viral illness.

RIMJ: What is the capability to respond to emergencies such as appendectomy or acute MI?

The clinic's two-bed in-patient area. [NSF, USAP]

DR. NAYLOR: We are fully prepared to respond to most emergencies and have distance support from the University of Texas Medical Branch (UTMB) as needed. We have the capacity to "admit" a patient who might need medical observation/in-patient care. Our clinic has a 2-bed in-patient area. We can manage a critically ill patient with intubation and cardiac issues. Performing a surgery would be a huge challenge as this winter's staff (me and the FNP) has no surgical experience. We do have the capability to set up an emergency OR area.

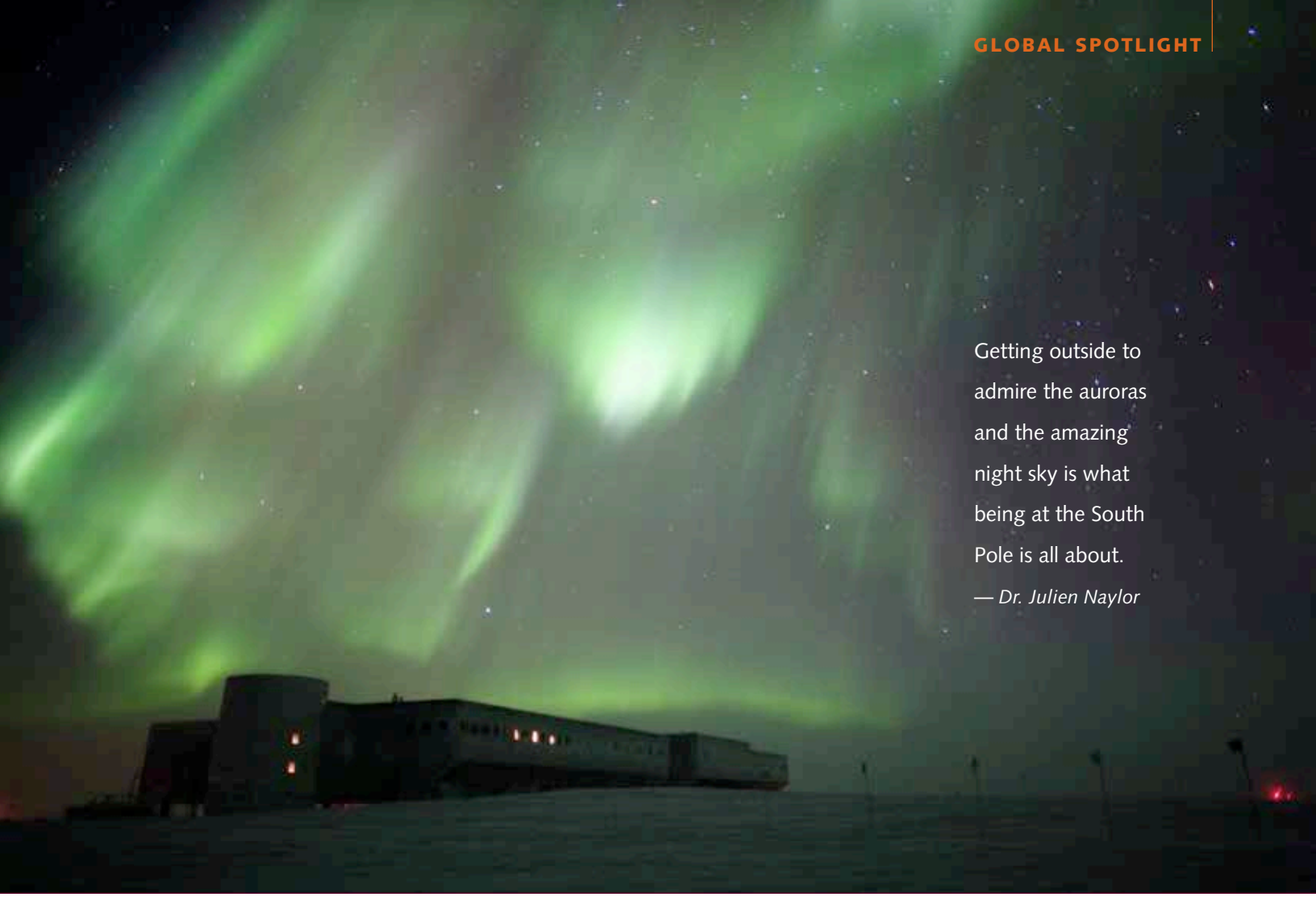
FAQs: U.S. Antarctic Program

The U.S. Antarctic Program is managed by the National Science Foundation. The program comprises research by scientists selected from universities and other research institutions and operations and support by a contractor and other agencies of the U.S. Government. The National Science Foundation (the U.S. Government agency that promotes the progress of science) funds and manages the program. Approximately 3,000 Americans are involved each year.

The program has three year-round research stations - McMurdo Station, Palmer Station, and Amundsen-Scott South Pole Station. Each facility has a medical clinic.

Link to program: https://www.nsf.gov/geo/opp/support/southp.jsp

The elevated station at Amundsen-Scott South Pole Station has lodging for 140 people, offices, a cafeteria, a gymnasium and a medical clinic. [COLIN WHITMORE, NSF]



The aurora australis over Amundsen-Scott South Pole Station. [PATRICK CULLIS, NSF]

RIMJ: What are the diagnostic imaging capabilities at the Clinic?
DR. NAYLOR: We are able to do X-ray, ultrasound, and a wide variety of lab work.

RIMJ: How far are you from full-service back-up and what is the protocol to Medevac a patient? Are they transported to a vessel or flown to New Zealand? And how challenging is that, especially in winter?
DR. NAYLOR: At the South Pole Station, a Medevac is an almost impossible challenge due to the cold temperatures and the ability for a plane to land safely during the winter months. Faced with a critically ill patient who needed a higher level of care, a Medevac would be coordinated through consultation with NSF and UTMB. All crew who work at the South Pole during winter are aware that a critical medical illness does not guarantee that they will be moved to a higher level of care. They have signed statements of understanding. This is why UTMB/NSF screen potential crew vigorously during medical evaluation. However, we would do everything we could to move a patient to a higher level of care if needed.

RIMJ: Are there disaster preparedness drills that occur?
DR. NAYLOR: We have a drill every month testing the station's ability to deal with a disaster scenario. We have an Emergency Response Medical Team of nine non-medical crew members. Every week we have training sessions with them to help them develop basic skills – CPR, evaluating a victim, helping medical staff with patients in the clinic.

RIMJ: What do you do in your off time?
DR. NAYLOR: My time off is very busy! I have many interests – knitting, sewing, reading, watching movies, and catching up on all the great TV series that I have missed. Getting outside to admire the auroras and the amazing night sky is what being at the South Pole is all about. We have a young crew this year that is always coming up with fun activities – many are sports-related. We have a nice gym that is always busy. I volunteer in our hydroponic greenhouse to help put some fresh greens on the cafeteria menu. I help wash dishes five days a week for a couple of hours to help our steward. On my day off, I bake bread or make cookies for the crew. I have been doing an exercise challenge called Couch to 5K on the treadmill. There is no reason to be bored at the South Pole station. ❖

Dr. Americo A. Savastano, Early Sports Medicine Specialist

From an Italian hilltop to Federal Hill

MARY KORR
RIMJ MANAGING EDITOR

Figure 1. Dr. Americo A. Savastano [URI]

The late **AMERICO A. SAVASTANO, MD**, (1906–1987), (**Figure 1**), Chief of Orthopedics at Rhode Island Hospital from 1965–1978, was an early leader in sports medicine. His story is as American as baseball; in fact, he served on President Lyndon Johnson's Council on Physical Fitness and Sports, when it was co-chaired by baseball legend Stan Musial.

As a young boy of about 6 years old, Americo left for America, leaving his family home in the hill town of Orchi, Italy, north of Naples, where his father Carmine was a blacksmith, to cross the North Atlantic alone circa 1912 to live with family friends in Providence, on Federal Hill.

In a Maine newspaper article in 2017, his son, Jeff Savastano, said his dad worked as a 12-year-old shining shoes on the street. It was not an unusual thing to do, as many Providence youth found work on the streets, in their homes, and in factories, as photos by Lewis W. Hine documented in Providence in 1912–1913. (**Figures 2–4**)

Life was tough, at times, but the food was fabulous, with the smell of fresh-baked bread wafting through the streets, and newly rolled pasta hanging in the kitchens.

Figure 2. Boy peddling bills on Atwells Avenue.

Figure 3. Delivering bread on Spruce Street, Providence.

Figure 4. (Left) Children worked alongside family members. This photo was taken next door to a cigar store on Atwells Avenue, and shows the workers and children stripping tobacco leaves for the cigars.

[PROVIDENCE AND FEDERAL HILL, 1912, PHOTOS: LIBRARY OF CONGRESS, LEWIS WICKES HINE]

The young Americo attended public schools, followed by the Rhode Island State College, later called the University of Rhode Island. His yearbook shows "Savvy" majored in general science. The honors student was on the track team, and a member of the glee club, band, and Chemical Society. Upon graduation in 1928, (**Figures 5 and 6**), he attended Harvard Medical School, graduating in 1932.

Perhaps his interest in orthopedics and sports medicine stemmed from his post-graduate training at the New York Rehabilitation Hospital. In addition, from 1936 to 1945, he was as an instructor in orthopedic surgery at the Polyclinic Hospital and Medical School in New York City.

In the December 1962 edition of the *Rhode Island Medical Journal* (RIMJ), he looked back on those years, specifically treating boxers. He stated that the chief argument against boxing is that the contents of the skull (the brain and its appendages) are the chief target. Severe brain damage and death are not uncommon in the sport and he hoped that the Boxing Education and Research Foundation would develop some sound ideas regarding safety, and that an insurance, welfare, and pension plan can be established, such as existed in other sports.

During the 1960s, Dr. Savastano chaired the Rhode Island Medical Society's Sports Medicine Committee, which co-sponsored an annual sports medicine conference at his alma mater, URI, bring in experts from around the country to address sports safety advances across the spectrum – geared to athletes, coaches, parents, schools, leagues and physicians. (**Figure 7**)

His interest in the field took him far and wide. In 1967, he served as team physician at the Pan American Games in Winnipeg, Canada. The following year he was a member of the Exchange Orthopedic Program to Russia. In 1968 he was cited for leadership by the American College for Sports Medicine.

In 1970, Dr. Savastano was inducted into the Rhode Island Heritage Hall of Fame, which cited him as "a specialist in the medical aspects of sports…who organized and conducted orthopedic clinics in many parts of the world."

Dr. Savastano passed away at the age of 80 on April 4, 1987. His obituary in RIMJ stated his medical accomplishments: "He scored first in several treatments never before attempted in Rhode Island, such as reconstruction or replacement of arthritic hips and knees, and the use of surgically implanted steel rods to straighten the spines of scoliosis victims. He developed the Savastano Vitallium, a total knee replacement, and was the editor/author in 1980 of "Knee Joint Replacement Surgery." The obituary also stated he pushed for legislation to require motorcyclists to wear helmets and protective gear.

Dr. Savastano's journey, which began on a hilltop in a small Italian town, is truly the story of an American dream realized – through study, hard work (even if it meant shining shoes), and securing the ties that bind, within and beyond the surgical arena. ❖

Figures 5 and 6. Rhode Island State College yearbook, 1928, portrait of A. A. Savastano (top), and Class of 1928

2nd Postgraduate Conference—

MEDICAL ASPECT OF SPORTS

Sponsored by the University of Rhode Island and the Rhode Island Medical Society
At the F. W. Keaney Gymnasium, U. of R.I., Kingston, on August 22-23, 1963
For Physicians, Trainers and Physical Education Instructors

THURSDAY MORNING — AUGUST 22, 1963

PRESIDING: Dr. A. A. Savastano — Chairman, Committee on Medical Aspects of Sports, Rhode Island Medical Society; Orthopedic Surgeon, Department of Athletics, University of Rhode Island

9:00	REGISTRATION
9:30	WELCOME — Dr. Ernest W. Hartung, Vice President, University of Rhode Island; Dr. Thomas Perry, Jr., President, Rhode Island Medical Society
9:45	Panel Discussion: ADMINISTRATIVE SPORTS MEDICINE FROM THE ADMINISTRATOR'S VIEWPOINT — Professor Maurice Zarchen, Director of Athletics, University of Rhode Island FROM THE COACH'S VIEWPOINT — Ralph Cordisco, Director of Athletics, Haverstraw-Stony Point High School, Haverstraw, New York FROM THE TEAM PHYSICIAN'S VIEWPOINT — Dr. G. Edward Crane, Athletic Surgeon, Brown University
10:45	Panel Discussion: PRESIDING: Jack Zilly, Head Coach of Football, University of Rhode Island QUALIFICATIONS FOR PARTICIPATION IN SPORTS — Dr. A. A. Savastano MEDICAL PROBLEMS AND THE ATHLETE — Dr. S. J. P. Turco, Director of Student Health, University of Rhode Island FIELD DIAGNOSIS AND DECISIONS — Kenneth Rawlinson, Head Trainer, University of Oklahoma
11:30	PHYSIOLOGICAL RESPONSES IN ATHLETES — Dr. Warren Guild, Harvard Medical School

THURSDAY AFTERNOON

PRESIDING: Dr. Joseph E. Donohue — Assistant Director of Student Health, Providence College

1:45	DIAGNOSIS AND MANAGEMENT OF COMMON ANKLE INJURIES — Dr. Thomas B. Quigley, Chairman, Committee on Medical Aspects of Sports, The American Medical Association; Assistant Clinical Professor of Surgery, Harvard Medical School; Surgeon, Peter Bent Brigham Hospital; Surgeon, Harvard University Health Services
2:15	DIAGNOSIS AND MANAGEMENT OF COMMON KNEE INJURIES — Dr. Frederick vom Saal, Chief, Department of Orthopedic Surgery, St. John's Hospital, Yonkers, N.Y.
2:45	DIAGNOSIS AND MANAGEMENT OF COMMON FACIAL INJURIES — Dr. Armand Versaci, Assistant Surgeon, Rhode Island Hospital
3:30	DIAGNOSIS AND MANAGEMENT OF COMMON ELBOW AND SHOULDER INJURIES — Dr. Thomas B. Quigley
4:00	QUESTION AND ANSWER PERIOD

FRIDAY MORNING — AUGUST 23, 1963

PRESIDING: Carl V. Slader — Associate Professor of Physical Education, University of Rhode Island

9:00	CONDITIONING DRILLS . . . Before and During Football Season — Kenneth Rawlinson
9:30	PREVENTION OF CERTAIN KNEE INJURIES — Dr. Daniel F. Hanley, Director of Student Health Services, Bowdoin College; Physician to the 1960 U.S. Olympic Team in Rome
10:00	PREVENTION OF HEAD AND NECK INJURIES Head Gear Problems Neck Strengthening Exercises Kenneth Rawlinson

Figure 7. This program for Dr. Savastano's sports medicine conference at URI, co-sponsored by the Rhode Island Medical Society, appeared in the June 1963 issue of the *Rhode Island Medical Journal*. In the same issue, he contributed an article on the high school athlete.

VA's Dr. John McGeary receives $200K for study on Synchronized Transcranial Magnetic Stimulation for Substance Use Disorder

PROVIDENCE – **DR. JOHN McGEARY**, a research scientist and staff psychologist at the VA RR&D Center for Neurorestoration and Neurotechnology at the Providence VA Medical Center, received an award July 1 for a two-year research study from the VA Office of Rehabilitation Research and Development.

Photo demonstrating the use of a Synchronized Transcranial Magnetic Stimulation device in a home setting.
[COURTESY OF WAVE NEUROSCIENCE INC.]

Dr. McGeary's project, titled "Synchronized Transcranial Magnetic Stimulation for Substance Use-Disordered Veterans," will evaluate the acceptability, tolerability and safety of synchronized TMS as a potential treatment for substance use disorders.

The nearly $200,000 study will allow McGeary and his team at the Providence VA Medical Center, which includes leaders in neurostimulation – **DR. NOAH PHILIP**, Chief of Psychiatric Neuromodulation and Addiction Treatment and **DR. ROBERT SWIFT**, Chief of Mental Health and Behavioral Science Services – to take key first steps to developing a potential new treatment for opiate use disorder, cocaine use disorder, and alcohol use disorder.

"As the response to the COVID-19 pandemic created challenges for traditional forms of addiction treatment, it is more important than ever to develop new treatments, particularly ones that could be used in a home setting," said Dr. McGeary. "There are currently no FDA-approved treatments for cocaine use disorder, so this technology could be a critically important tool for treatment in these cases as well, if the research supports its use." ❖

FDA issues Emergency Use Authorization to Yale School of Public Health for SalivaDirect, which uses a new method of saliva sample processing

On August 15 the U.S. Food and Drug Administration issued an emergency use authorization (EUA) to Yale School of Public Health for its SalivaDirect COVID-19 diagnostic test, which uses a new method of processing saliva samples when testing for COVID-19 infection.

"Providing this type of flexibility for processing saliva samples to test for COVID-19 infection is groundbreaking in terms of efficiency and avoiding shortages of crucial test components like reagents," said FDA Commissioner **STEPHEN M. HAHN, MD**.

SalivaDirect does not require any special type of swab or collection device; a saliva sample can be collected in any sterile container. This test is also unique because it does not require a separate nucleic acid extraction step. This is significant because the extraction kits used for this step in other tests have been prone to shortages in the past. Being able to perform a test without these kits enhances the capacity for increased testing, while reducing the strain on available resources. Additionally, the SalivaDirect methodology has been validated and authorized for use with different combinations of commonly used reagents and instruments, meaning the test could be used broadly in most high-complexity labs.

Yale intends to provide the SalivaDirect protocol to interested laboratories as an "open source" protocol, meaning that designated laboratories could follow the protocol to obtain the required components and perform the test in their lab according to Yale's instructions for use. Because this test does not rely on any proprietary equipment from Yale and can use a variety of commercially available testing components, it can be assembled and used in high-complexity labs throughout the country, provided they comply with the conditions of authorization in the EUA.

This is the fifth test that the FDA has authorized that uses saliva as a sample for testing. Testing saliva eliminates the need for nasopharyngeal swabs, which have also been prone to shortages, and alleviates the patient discomfort associated with these swabs. Since the saliva sample is self-collected under the observation of a healthcare professional, it could also potentially lower the risk posed to healthcare workers responsible for sample collection. While FDA has seen variable performance in tests using saliva, Yale School of Public Health submitted data with its EUA request from which the FDA determined that Yale's test meets the criteria for emergency authorization when used to test saliva samples for SARS-CoV-2, the virus that causes COVID-19 infection. ❖

RIH Alzheimer's researchers present at virtual international meeting

PROVIDENCE – Researchers from the Alzheimer's Disease and Memory Disorders Center at Rhode Island Hospital presented five abstracts at the annual Alzheimer's Association International Conference held virtually recently.

JONATHAN DRAKE, MD, associate director of the center, presented an abstract which assessed whether molecular blood biomarkers of vascular inflammation could be a useful tool in assessing risk for Alzheimer's disease. Results showed that a blood-based protein called Vascular Cell Adhesion Molecule–1 (VCAM–1), an indicator of active inflammation of the body's blood vessels, was higher in people who were further along in the Alzheimer's disease spectrum than those who are less affected. Alzheimer's disease detected early in life may be altered over time with effective interventions. "Developing blood-based biomarkers for Alzheimer's disease is an important milestone that needs to be achieved," said Drake. "This is especially important in the current era given recent advances in the field identifying midlife vascular risk factors as representing between 30 to 60% of one's risk for later-life Alzheimer's disease. Importantly, vascular risk factors are theoretically modifiable, meaning that important steps toward optimal health taken early in life may decrease your risk for succumbing to this devastating disease as you get older."

BRIAN OTT, MD, center director, presented results from a study to determine if in-car video technology can effectively detect unsafe driving events in cognitively impaired older adults, and if providing feedback about these events to drivers and their family members can lead to a reduction in the frequency and severity of unsafe driving behaviors. Unsafe driving events were captured from in-car video recorders and later analyzed, categorized and scored. Half of the participants in the study were monitored but received no feedback. The other half of the participants and their family members were sent a weekly report by mail along with a DVD of recorded unsafe driving events with recommendations. Those in the group who received feedback had 21% fewer unsafe driving events (UDE) compared to the group who receive no feedback. The feedback group also saw a 48% decrease in severity of UDE while the non-feedback group saw a 37% increase. "Results of the study suggest that it may be possible to improve driving safety among older drivers with cognitive impairment using video technology and a behavior modification approach aimed directly at problem behaviors that cause unsafe driving events," said Ott.

LAURA KORTHAUER, PhD, and Ott presented findings on the value of primary care providers (PCPs) screening for early detection of cognitive impairment. The study examined the case histories of 100 local patients referred by their PCPs for genotyping and telephone screening. "Primary care is an important gateway for screening patients for risk for cognitive decline," said Korthauer. "This study shows that implementing a cognitive and genetic screening program for AD risk in a primary care setting is feasible and well-received by patients. The program may provide added value to PCPs in advising patients about risk factor modification."

LORI DAIELLO, PharmD, ScM, presented information on the protocol for an upcoming study of older adults undergoing elective surgery and the role of impaired blood-brain barrier (BBB) function as a pre-surgery indicator of cognitive problems in those with and without risk factors for Alzheimer's disease. "BBB dysfunction is an indicator of brain vulnerability and neurodegeneration in Alzheimer's disease that may also be an important risk factor for postoperative delirium and delayed or incomplete cognitive recovery," said Daiello. "CREATES is the first study of perioperative cognitive outcomes to investigate BBB function with this brain imaging approach. The results will provide important insights into the underlying mechanism(s) of memory impairments that may follow major surgery."

GEOFFREY TREMONT, PhD, presented evidence that yoga may improve some aspects of brain health in individuals living with Mild Cognitive Impairment, a condition often seen as a precursor to Alzheimer's disease. His study included 12 weeks of twice weekly yoga classes, while his colleague, Dr. Jennifer Davis conducted classes involving interactive discussion and presentations addressing healthy living topics relevant to aging and cognitive impairment. "The results of this study showed no statistical differences in specific areas of thinking between the yoga classes and the healthy living classes," said Tremont. "However, we did see signs that yoga participation was associated with improvements in participants' ability to process visual information and engage in planning, organization and holding information in short-term memory." The study also found that the yoga group had a greater decline in perceived stress, whereas the healthy living classes showed a greater reduction in depressive symptoms when compared to the other group. ❖

Appointments

Todd Conklin named Lifespan Executive Vice President and Chief Operating Officer

PROVIDENCE – Lifespan has appointed **TODD CONKLIN, BS, MBA, CPA**, as its executive vice president and chief operating officer, a newly established position for Rhode Island's largest provider of health care.

Conklin will oversee Lifespan's five hospitals and its Gateway Healthcare programs and will be responsible for the system's operational performance, including patient satisfaction, quality of care, and effective service delivery.

He has over 20 years experience in the health care field and joins Lifespan from Ascension Health System, one of the nation's largest Catholic and non-profit health systems, where he served as chief operating officer and oversaw 31 hospitals across Wisconsin and Kansas. Under Conklin's leadership, Ascension improved its operating performance and patient satisfaction levels, and expanded its clinical services and teaching programs. Previously, Conklin was senior vice president of operational finance for the Catholic Health Initiatives system, encompassing 109 hospitals across 19 states.

"Todd is a welcome addition to our team at a critical time for health care both in Rhode Island and nationally,"said Lifespan President and CEO **TIMOTHY J. BABINEAU, MD**. "Todd has a strong record of engaging teams in improving operations, efficiency, and care delivery across the health systems he has helped lead and is well-positioned to serve as Lifespan's first executive vice president and chief operating officer."

"In light of the impact of COVID-19 on public health, the workforce, and state and local economies, ensuring that quality healthcare is accessible to all individuals is now more important than ever,"said Conklin. "I'm looking forward to working with a great team in navigating these challenges, and continuing to innovate and transform Lifespan's operations for the better."

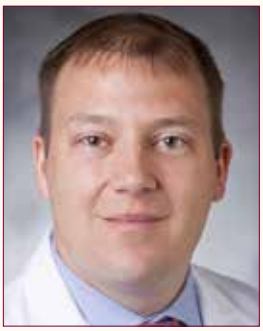
Conklin holds a Bachelor of Science degree in Accounting from Illinois State University, and a Master of Business Administration degree from the Kellogg School of Management at Northwestern University. He is a certified public accountant. ❖

Gerald Colvin, DO, joins CharterCARE

DR. GERALD COLVIN joined CharterCARE Medical Associates in July. Formerly, he was an active member of the staff and cancer program from 2001 thru 2006.

Board-certified in Internal Medicine and Hematology & Oncology, Dr. Colvin is a graduate of Philadelphia College of Osteopathic Medicine and completed his residency in internal medicine at Lehigh Valley Hospital in Pennsylvania. He completed his fellowship training in hematology/oncology at the University of Massachusetts Memorial Medical Center in Worcester.

Dr. Colvin is the former Director of Hematology/Oncology at South County Medical Group, and has served on the faculty of both the Alpert Medical School of Brown University and Boston University School of Medicine. ❖



Samuel H. Eaton, MD, joins Brown Urology in Newport County

PROVIDENCE – Brown Urology's newest physician, **DR. SAMUEL H. EATON**, is now seeing patients at its new location on Aquidneck Island, across from Newport Hospital.

Dr. Eaton sub-specializes in endourology. Some of the procedures and treatments in which he specializes include: robotic and laparoscopic surgery, prostate cancer, kidney cancer, upper tract reconstruction, and comprehensive stone management and BPH surgery.

"I'm excited to work with Newport Hospital and Brown Urology to help expand the reach and breadth of what the practice has to offer," said Dr. Eaton, who added he plans to bring his holistic approach to patient care to the practice. "When treating patients, not only do I want to understand what brought them to me medically, but I also want to understand where they're coming from more generally in life. This way we can work together on whatever problem they're facing in a way that is not only medically appropriate but also in a way that fits with their goals."

Dr. Eaton comes to Brown Urology from Duke University Medical Center. He is a graduate of the University of Illinois-Urbana/Champaign and earned his MD from Columbia University College of Physicians and Surgeons. He completed his urology residency at Boston University Medical Center and subspecialty training in endourology at Northwestern University in Chicago. ❖

Appointments

Pulmonologist James M. Ferguson, MD, joins Newport Hospital

Newport Hospital and Lifespan Physician Group, Inc. recently announced that pulmonologist **JAMES M. FERGUSON, MD**, who specializes in the treatment of ailments of the lung and respiratory system, has joined the hospital's medical staff. He began on July 27 and is now welcoming new patients.

Dr. Ferguson is a graduate of the University of Massachusetts Medical School. He completed both his residency and fellowship in pulmonary and critical care at The Warren Alpert Medical School of Brown University, where he is now an associate clinical professor. Prior to his fellowship, he spent one year as an intensive care hospitalist at The Miriam Hospital, an affiliate of the Lifespan health system. He is board certified in pulmonary and internal medicine.

Dr. Ferguson has a special interest in lung cancer screening and identifying and improving disparities in health and health care. He is a member of the American College of Chest Physicians, American Thoracic Society, and American College of Physicians. ❖

Dr. Eren Kuris returns to University Orthopedics

EAST PROVIDENCE – University Orthopedics is happy to welcome **DR. EREN KURIS** back into the UOI family after a year away completing a Spine Surgery Fellowship.

Dr. Kuris spent the last year at the University of Colorado as a Spine Surgery Fellow, concentrating specifically on complex spinal conditions. As part of the UOI team, he will treat all aspects of spinal care, including degenerative conditions such as disc degeneration, disc herniations, and spinal arthritis; spine deformities; spine trauma; and spine infections.

"Spine surgery is a very dynamic field. We have made significant advances in recent years with regard to our overall understanding of spinal pathology and alignment and the methods and technologies we have to treat spine conditions, which will lead to improved patient outcomes," Dr. Kuris said.

Dr. Kuris completed both his Orthopaedic Surgery Residency and an Orthopaedic Trauma Fellowship at the Alpert Medical School, where he currently serves as an Assistant Professor of Orthopaedics. He holds both an undergraduate and medical degree from Tulane University. Additionally, he is a member of the American Academy of Orthopaedic Surgeons (AAOS), the North American Spine Society (NASS), and the Orthopaedic Trauma Association (OTA). He serves on two committees for NASS (the SpinePAC Advisory Committee and the Political Engagement Committee) and is the Spine Section Editor for the 5th edition of the OTA's Core Curriculum. ❖

Andrew Chen, MD, joins University Orthopedics' Sports Medicine team

EAST PROVIDENCE – University Orthopedics (UOI) recently announced the addition of **DR. ANDREW CHEN** to its Sports Medicine team.

Dr. Chen's specialty is Primary Care Sports Medicine. He treats a wide range of sports-related injuries including finger, hand, and wrist injuries; elbow issues; shoulder issues and pathologies; hip pain and injuries; knee pain and injuries; and ankle and foot problems. Dr. Chen will also treat chronic tendinopathies and injuries that have failed conservative management.

Dr. Chen recently completed a fellowship with Case Western Reserve University at University Hospitals Cleveland Medical Center. He holds an undergraduate degree from Carnegie Mellon University and an MD from Jefferson Medical College. Additionally, he is board-certified in family medicine through the American Board of Family Medicine. He has served as team physician for Kent State University, Lake Erie College, and Hawkin Upper School, which is a college prep school in Ohio.

"I take pride in not only helping my patients recover from an injury but also returning them to optimum health so they excel in the sports and activities they love," Dr. Chen said. "I am honored and humbled to be joining such an experienced and well-regarded team like the one at University Orthopedics."

Dr. Chen is joining UOI September 1 and will eventually be located at UOI's new Mansfield, Massachusetts location when it opens in January 2021. ❖

Appointments

CNE CEO James E. Fanale, MD, to chair American Diabetes Association® event, Ocean State Tour de Cure

PROVIDENCE – Care New England and The American Diabetes Association® (ADA) recently announced that **JAMES E. FANALE, MD**, Care New England Health System president and CEO, will chair the 2020 Ocean State Tour de Cure on Saturday, October 3, 2020, for the second year in a row. In light of the COVID-19 pandemic, this year's event will be held virtually, offering participants an opportunity to join the event wherever they are.

Every dollar raised through the Ocean State Tour de Cure plays an important role in supporting the ADA's mission: to prevent and cure diabetes and to improve the lives of all people affected by diabetes. This year, Care New England will serve as the lead community partner for the Tour event. CNE has made a commitment to the ADA to work together to raise awareness and provide much needed education in order to prevent and assist those in the community affected by diabetes in Southern New England.

Tour de Cure brings together volunteers and participants invested in bending the curve on the diabetes epidemic and funding research to find a cure.

"Care New England cares about the community and everyone affected by, or at risk of developing diabetes. As this year's virtual chair, I look forward to, once again, rallying our employees and all Rhode Islanders, to come together to educate the community about COVID-19 safety, diabetes management, and how to make behavioral and lifestyle adjustments to help fight diabetes," said Dr. Fanale. "Diabetes affects many people on a personal, as well as a community level. Unfortunately, the prevalence of diabetes continues to grow, affecting more than 106,000 individuals here in Rhode Island. Another 200,000 are undiagnosed or living with prediabetes. Attention and action is necessary to help us advance the pace of progress in the fight against diabetes."

The ADA's New England Executive Director, Susan Sarro, thanked Dr. Fanale and Care New England for their continued commitment. "Support from community leaders such as Dr. Fanale make a critical difference in our efforts to engage the business community and other organizations in coming together behind the ADA mission. With CNE's involvement as a leading health system, we can reach deeper into the community to address the needs of those who live with diabetes."

Dr. Fanale and Care New England would like to invite Southern New England participants to join the celebration and be part of the movement to fight diabetes by taking part in Tour de Cure. This virtual fitness and community event will take place Saturday, October 3 to help raise funds to support the ADA's mission. Registration is free with no fundraising minimums. Participants are encouraged to donate and raise funds to the extent they are able. Individuals can join the Care New England Team, called Team CNE, by visiting http://main.diabetes.org/goto/CareNewEngland. ❖

Robert H. Schwengel, MD, to lead the Southern New England Board of Directors

PROVIDENCE – **ROBERT H. SCHWENGEL, MD, FACC**, of Southcoast Health, has been appointed President of the Association's Board of Directors in Southern New England.

Serving for several years on the Southern New England American Heart Association Board of Directors, Dr. Schwengel has been instrumental in the Association's local influence towards funding cutting-edge research and promoting quality systems of care.

"It is a great honor to be chosen as President of the American Heart Association's Board of Directors in Southern New England," said Dr. Schwengel. "I am passionate about helping the American Heart Association continue to raise awareness about cardiovascular and neurovascular health and reduce the health disparities that exist in our communities. I thank my fellow volunteers and Board Members for this opportunity."

"Volunteers are the lifeblood of the American Heart Association and Dr. Robert Schwengel's unwavering commitment, time and compassion inspires endless possibilities to further our mission in Southern New England," said **ALISSA MEMOLI**, Regional Vice President of the Southern New England American Heart Association. "During these unprecedented times, the work of the Association is more important than ever, and we are honored to have him at the helm as we pioneer innovative solutions to build a world of healthier and longer lives." ❖

Recognition

James Taylor, RN, receives 2020 Clare Sullivan Award for Nursing Leadership

Each year, Butler Hospital presents the Clare Sullivan Award for Nursing Leadership, to one of its own for outstanding compassionate care. The award is named in honor of Clare Sullivan, who was the Butler Hospital director of nurses from 1964 to 1992. The first award was given out in 2005.

This year's Clare Sullivan Nursing Leader is **JAMES TAYLOR, RN**. He has been with Butler since starting as an LPN in 2001. ❖

Recognition

South County Hospital receives two 5-Star ratings from CMS

South County Hospital has received a Double 5-Star rating from the Centers of Medicare and Medicaid (CMS) in the recent Hospital Compare report.

The CMS Hospital Quality Star Rating was created by Medicare to help healthcare consumers make informed decisions by simplifying complex criteria that measures healthcare quality.

As a result of data collected by CMS, South County Hospital measured the highest rating of 5-Stars in overall Hospital Quality and Patient Experience in the ratings released in July.

Patient Experience is measured using a national survey that asks patients about their experiences during a recent hospital stay. The survey is administered by HCAHPS (Hospital Consumer Assessment of Healthcare Providers and Systems).

The overall hospital rating includes a variety of measures calculated by CMS, the federal agency that runs the Medicare program, including the Hospital Inpatient Quality Reporting (IQR) Program and the Hospital Outpatient Quality Reporting (OQR) Program.

South County Hospital measured above the national average in:

- Safety of Care (hospital acquired infections, complications of hip/knee surgery, and other patient safety indicators)
- Low Readmission Rates (patients readmitted within 30 days of inpatient discharge)
- Patient Experience (HCAHPS survey)

South County Hospital is accredited by The Joint Commission and is the recipient of numerous accreditations, awards and recognitions for high quality care and excellence in patient satisfaction.

Results of the Hospital Compare are listed and explained on the Official U.S. Government Site for Medicare. ❖

Kent Hospital's Women's Care Unit scores big for maternity practices in infant nutrition

Following the completion of the Centers for Disease Control and Prevention's (CDC) 2018 Maternity Practices in Infant Nutrition and Care (mPINC) survey, Kent Hospital's women's care unit score was 94. The average score in the US is 79. And, for hospitals the same size at Kent Hospital, the score is 78.

Kent Hospital was one of 2,045 US hospitals that responded, demonstrating its commitment to quality improvement in maternity practices that support optimal infant nutrition. The mPINC is a national survey of newborn feeding practices in hospitals in the United States. ❖

Fatima Hospital receives Gold Achievement Award for stroke care

NORTH PROVIDENCE – Fatima Hospital, an affiliate of CharterCARE Health Partners, has received a prestigious national award for quality in stoke care that has been jointly presented by the American Heart Association (AHA) and the American Stroke Association (ASA).

Fatima received the highest level of this award, the Gold Achievement, for achieving compliance with AHA's and ASA's seven stroke achievement measures for 24 consecutive months.

These achievement measures are:

- Percent of acute stroke patients who arrive at the hospital within 2 hour of occurrence and for whom care was initiated at this hospital within t3 hours of occurrence of time last known well. Corresponding measure available for inpatient stroke cases.
- Percent of stroke patients who receive antithrombotic therapy by the end of hospital day two
- Percent of stroke patients who receive VTE prophylaxis the day of or the day after hospital admission.
- Percent of stroke patients who are prescribed antithrombotic therapy at discharge.
- Percent of stroke patients with an ischemic stroke or TIA with atrial fibrillation/flutter discharged on anticoagulation therapy. Corresponding measures available for observation status only as well as inpatient stroke cases.
- Percent of stroke patients with a history of smoking cigarettes, who are, or whose caregivers are, given smoking cessation advice or counseling during hospital stay.
- Percent of stroke patients who are discharged on statin medication.

CharterCARE CEO Jeff Liebman stated "While we are proud to receive any national quality recognition, I am especially proud that this award recognizes consistent quality performance for stroke patients over a sustained period. This reflects that our commitment to quality performance is a constant mission, every day of every year".

The AHA-ASA award is an advanced level of recognition acknowledging hospital for consistent compliance with stringent quality measures on which that the two organizations have collaborated to develop. ❖

Recognition

Lifespan hospitals honored for excellence in stroke care

PROVIDENCE – Three Lifespan hospitals – Rhode Island Hospital, The Miriam Hospital and Newport Hospital – have received the American Heart Association/American Stroke Association's Get With The Guidelines®-Stroke Gold Plus Quality Achievement Award. The award recognizes commitment to ensuring stroke patients receive the most appropriate treatment according to nationally recognized, research-based guidelines based on the latest scientific evidence.

The hospitals earned the award by meeting specific quality achievement measures for the diagnosis and treatment of stroke patients including use of medications and other stroke treatments aligned with the most up-to-date, evidence-based guidelines with the goal of speeding recovery and reducing death and disability for stroke patients.

Rhode Island Hospital was additionally recognized with the Target: Stroke Honor Roll Elite Advanced Therapy designation. To qualify for this recognition, hospitals must meet quality measures developed to reduce the time between the patient's arrival at the hospital and treatment with the clot-buster tissue plasminogen activator, or tPA, the only drug approved by the U.S. Food and Drug Administration to treat ischemic stroke. Also, "door to device times" must occur within 90 minutes for direct arriving patients and within 60 minutes for transfers (for at least 50% of applicable patients).

"Our staff who care for stroke patients are part of a highly skilled, specialized team trained to provide rapid assessment and treatment for the acute stroke patient," said **JO-ANN SARAFIN, MS, APRN, CNP, FNP-BC, SCRN**, stroke program manager for Rhode Island Hospital. "It takes quick recognition of stroke in the community, including astute assessment by EMS with appropriate knowledge of stroke triage protocols. It entails efficient management in the Emergency Department encompassing clinical assessment, imaging, and state-of-the-art treatment in Neurointerventional radiology if indicated. It involves frequent monitoring, education, early rehabilitation and support to assist the stroke patient in achieving the best possible outcome. We are honored to receive this award reflecting on the entire spectrum of stroke care we provide, allowing as many patients as possible to regain their best quality of life."

Newport Hospital was additionally recognized with the Target: Type 2 Diabetes Honor Roll award. To qualify, hospitals must meet quality measures developed with more than 90% of compliance for 12 consecutive months for the "Overall Diabetes Cardiovascular Initiative Composite Score."

"This is wonderful news. It takes a team to be able to achieve these awards and it's due to the efforts of everyone across the hospital – nurses, doctors, rehabilitation specialists, and more. It's exciting that Newport Hospital earned the Target: Type 2 Diabetes Honor Roll Award in the first year it was available," said **KAREN SCHAEFER, MSN, APRN, AGCNS-BC, ASC-BC**, stroke program manager for Newport Hospital and The Miriam Hospital.

Newport Hospital and The Miriam Hospital have met specific criteria to each be designated as a Primary Stroke Center while the Rhode Island Hospital is designated as a Comprehensive Stroke Center. Primary Stroke Centers are equipped to stabilize and provide emergency care for patients with acute stroke, and Comprehensive Stroke Centers can provide more specialized care for patients with complex strokes. ❖

Westerly Hospital receives Get With The Guidelines – Stroke silver quality achievement award

WESTERLY – Westerly Hospital has received the American Heart Association/American Stroke Association's Get With The Guidelines® – Stroke Silver Quality Achievement Award. The award recognizes the hospital's commitment to ensuring stroke patients receive the most appropriate treatment according to nationally recognized, research-based guidelines based on the latest scientific evidence.

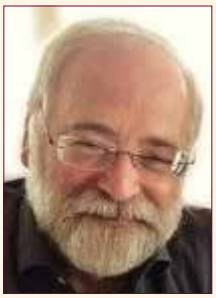
Westerly Hospital earned the first time award by meeting specific quality achievement measures for the diagnosis and treatment of stroke patients at a set level for a designated period. These measures include evaluation of the proper use of medications and other stroke treatments aligned with the most up-to-date, evidence-based guidelines with the goal of speeding recovery and reducing death and disability for stroke patients. Before discharge, patients should also receive education on managing their health, get a follow-up visit scheduled, as well as other care transition interventions.

The award reflects the investments Westerly Hospital has made in attracting high caliber providers and enhancing capabilities at its certified stroke center. As part of Yale New Haven Health, Westerly augments its stroke care through a Telestroke program that provides 24/7 communication with stroke-trained neurologists, so patients can be treated quickly to preserve brain function in the critical early onset of stroke.

"For stroke patients, time is critical and this award recognizes Westerly Hospitals' commitment to ensuring that our patients receive the best care that adheres to nationally-respected clinical guidelines," said chief medical officer **OLIVER MAYORGA, MD**. "The tools and resources help us track and measure our success in meeting evidenced-based clinical guidelines developed to improve patient outcomes."

In 2019, Westerly Hospital became certified as an Acute Stroke Ready hospital by The Joint Commission on Accreditation of Healthcare Organizations. The certification allows smaller and rural hospitals to demonstrate their ability to comply with standards of care for the initial treatment of stroke patients when prompt action and proper medications can save lives and limit the long-term disabling effects of strokes. ❖

Obituaries



JOHN N. BERGERON, MD, 66, of Westerly, passed away on July 30, 2020. He graduated magna cum laude with a Master's in Science from Brown University in 1977, later earning his medical degree from Brown University in 1983.

He was a committed community doctor, caring for the underserved at Wood River Health Services for 37 years. His colleagues described him as "an astute physician and a tremendous diagnostician."

His fascination with understanding how all things worked drove his skills in construction and carpentry, renovating the family home and engineering creative solutions that rivaled those of experts. He raised his sons with his shared interests in technology, computers, and engineering.

Throughout his life he remained a gentle spirit – modest, selfless, and dedicated to the service of others.

He was the loving husband of Joanne (Catanzaro) Bergeron with whom he shared 38 years of marriage. In addition to his wife, he is survived by his son Stephen (Gretchen Taubl) and their children Charlotte, Theodore, Griffin, Max; his son Thomas (Katherine Casey) and their children Ryan, Henry; his siblings Celine Bergeron (Jim Morris), Roberta Landry (Dave), David (Lydia), Claude (Geralyn), Denise Tiemissen (Tim), Paul (Theresa), Anne Martin (Greg), Norman (Susan), Daniel (Cheleasa), Philip Joel; and numerous nieces and nephews.

A donation in his memory may be made to Wood River Health Services, 823 Main St., Hope Valley, RI 02832. ❖

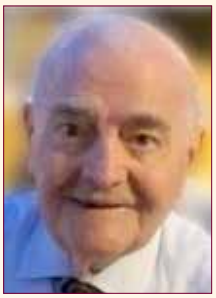
DR. VINCENT F. P. MARCACCIO III, 67, of Cranston, passed away peacefully on August 17, 2020. He was the beloved husband of Anne Marcaccio (Schavone).

Born in Providence, he was a graduate of Hope High School and the University of Rhode Island. He earned his medical degree from the University of Rome, Italy, and completed his residency at Roger Williams Hospital.

He worked as a general practitioner for many years and then returned to Roger Williams specializing in behavioral medicine. He was a parishioner of the former St. John's Church in Providence and, subsequently, of Holy Name Church in Providence.

Dr. Marcaccio is survived by his two loving sons, Andrew S. Marcaccio (Amanda), and Vincent F. Marcaccio. He was blessed with two beautiful granddaughters, Hallie and Hannah. He was the dear brother of Michael and Mary-Ann Marcaccio.

Donations in his memory may be made to: Church of the Holy Name of Jesus, 99 Camp St., Providence, RI 02906, or the charity of your choice. ❖



ROCCO MARZILLI, MD, 91, of East Providence and Jamestown, passed away peacefully on August 18, 2020.

He graduated from Providence College and served in the United States Army for two years during the Korean Conflict. He was a member of the Seventh Army stationed in Stuttgart, Germany, and received two letters of commendation for acts considered above and beyond the call of duty.

He was the husband of the late Kathryn (Vucci) Marzilli. Soon after their marriage, he enrolled at the University of Bologna School of Medicine in Bologna, Italy, where he and Kathryn would spend the first several years of their marriage. Upon returning, they settled down in East Providence where they would remain for over fifty years.

Dr. Marzilli completed his residency at the V.A. hospital in Providence and remained on staff until 1968 when he decided to enter private practice specializing in gastroenterology. He was on staff at St. Joseph's Hospital and Rhode Island Hospital, the latter for 37 years until 2008. He was the first Medical Director for Waterview Villa Rehabilitation and Health Care Center and continued in that role until 2010. He was also on the clinical staff at the Warren Alpert Medical School of Brown University.

He was a member of many medical associations including the RI Division of the American Cancer Society. He also served as Medical Advisor for the Rhode Island Chapter of the National Foundation of Ileitis and Colitis (NFIC) while also serving on the board of directors of the Colitis and Ileitis Association. Dr. Marzilli was also the President of the Rhode Island Gastroenterological Society.

Apart from his work, he was an adoring husband to Kathryn for fifty-seven years. They spent many of those years traveling around Europe, including several trips to Italy. Still, nothing was quite as peaceful and enjoyable as the view from their home in Jamestown.

He is survived by his sister, Rita Perrino of Warwick, his children, Stephen Marzilli of Lansing, MI; Lori Kahler and her husband Jeffrey of Marion, MA, as well as his grandchild, Raymond Leonardo and his wife Olivia of Rockland, MA. Memorial service will be held at a later date. Please visit rebellofuneralhome.com for online condolences. In his memory, please consider making a donation to the Make-A-Wish Foundation by visiting wish.org. ❖