Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

INFECTIOUS DISEASE IN ATHLETES

Thomas L. Sevier, MD

VIRAL UPPER RESPIRATORY INFECTIONS

One of the most common illnesses encountered in clinical sports medicine is the viral upper respiratory tract infection (URI). These infections commonly occur because they can be caused by so many different viral strains. Understanding the relationship between exercise and URIs has obvious potential public health implications. For the athlete, this knowledge can mean the difference between being able to compete or missing an event because of illness. This section addresses current clinical and scientific evidence supporting guidelines for sports and exercise participation in athletes with a URI.

Acute URIs impose a significant burden each year in terms of days of disability, lost school or work days, and medical costs.[48] The average adult has from one to six episodes of the common cold each year. The Centers for Disease Control has estimated that 429 million URIs occur annually in the United States, resulting in $2.5 billion in medical costs and missed school and work days.[80] It has been proposed that URIs cause more disability among athletes than all other diseases combined.[66] The analysis of disease patterns among Summer and Winter Olympic athletes treated by the medical staff is remarkably consistent, with respiratory infections always heading the list, followed by gastrointestinal disorders and skin infections running a close third.[35] In the 1988 Summer Olympic games, some of the world's greatest athletes were reportedly

From Ball Memorial Hospital Sports Medicine Fellowship Program; Central Indiana Sports Medicine; Human Performance Laboratory, Ball State University; and the Department of Medicine, Indiana University Medical Center and Muncie Center for Medical Education, Muncie, Indiana

unable to compete owing to infectious illness.[26] In the 1992 Winter Olympics, a sizable number of athletes reported that they were unable to compete or had subpar performances because of URI.[55]

Most URIs are caused by rhinoviruses, although many viral strains may cause the common cold. Rhinoviral infections occur year round, with well-defined periods of prevalence in the fall and spring accounting for about 40% of infections in the adult population.[19] Coronaviruses are also considered to be a major cause of winter colds and tend to be more common in the 15- to 19-year-old age group.[11, 38] The enteroviruses, specifically coxsackievirus, occur mostly during the late summer and early autumn months and, even though not a common cause of URI,[51] are of particular significance to athletes engaged in intensive training regimens. Animal studies have confirmed that exhaustive exercise increases susceptibility and severity of these infections.[31, 45] In addition, several coxsackie viral strains have been associated with myocarditis, and exercise during an infection may in fact increase the risk of developing viral cardiomyopathy.[9, 64, 75] Also, Burke et al[10] reported that exercising during subclinical myocarditis was thought to be responsible for some arrhythmia-associated deaths.

Symptoms of URI in the athlete are the same as those in the general population and may vary from case to case depending on multiple factors, such as the type of infecting virus and the response of the host-immune defense mechanisms. Common viral URI symptoms range from runny nose, sneezing, and congestion to sore throat, hoarseness, and nonproductive cough. Patients often feel weak and occasionally have myalgias despite little or low-grade fever.

Epidemiology

URIs are spread from person to person by contact with respiratory secretions allowing the virus to gain entry into a susceptible host's respiratory tract via large or small particle aerosols or by direct or indirect contact with contaminated objects. Dick et al[22] demonstrated that aerosol spray was the most important mechanism of rhinovirus transmission. Other research on URI transmissions indicates that athletes could spread the virus through shared athletic equipment and towels. Hand-to-hand transmission has been demonstrated to occur under certain experimental conditions,[33] but it is questionable how much this contributes to natural transmission. Athletes who belong to teams housed in crowded dormitories, classrooms, and gymnasiums can pass respiratory viruses back and forth to each other, which may account for the higher incidence of URIs during cooler months.[11] Exposure to cold temperatures, damp environments, or drafts does not seem to enhance vulnerability to a URI.[23, 69]

Many athletes believe that exercise improves their resistance to infection and report that they have fewer colds and other respiratory tract infections.[73] There are relatively few studies that have explored the rela-

tionship between physical activity and URI. Considerable anecdotal information from coaches and team physicians supports the belief that severe exertion, especially when coupled with mental stress, places athletes at increased risk for a URI.[27] Evidence from several studies supports this contention.[16, 32, 36] In an epidemiologic study by Nieman et al[57] of runners in the Los Angeles marathon, 12.9% of the participants reported symptoms consistent with a URI during the week following the race, in comparison to 2.2% of similarly experienced runners who registered for the race but did not participate for reasons other than sickness. The author also reported that runners who trained 60 or more miles a week suffered twice as many URIs as those who trained less than 20 miles a week.

Both epidemiologic and clinical data support the concept that heavy exertion increases the risk of URI because of changes in immune function.[7, 46, 58, 59] The relationship between physical activity and URI appears to be modeled in the form of a J-curve[56, 69] (Fig. 1). The risk of respiratory tract infection appears to decrease below that of sedentary individuals when one engages in moderate exercise training but rises above average during periods of excessive exercise. In a randomized controlled study, the effects of walking on immune response and URIs were evaluated using a group of sedentary, mildly obese women, with the exercising subjects experiencing URI symptoms only one-half the days of the control group.[52, 60]

Effects

It is well established that various measures of physical performance capability are reduced during an infectious episode.[20] Significant altera-

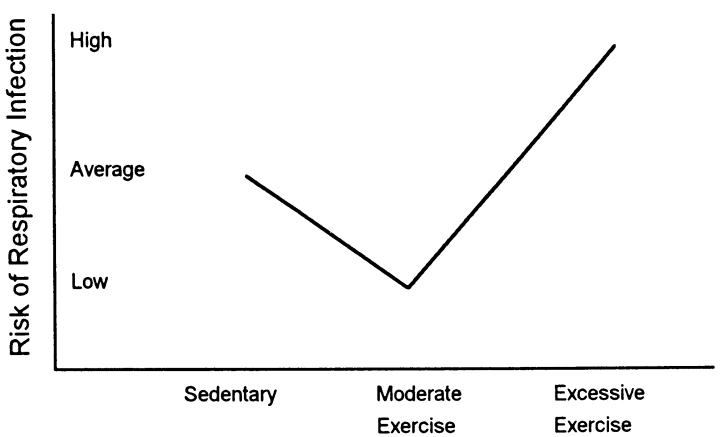

Figure 1. Risk of respiratory infection. J-shaped model of relationship between varying amounts of exercise and risk of respiratory tract infections. This model suggests that moderate exercise may lower risk of respiratory infection while excessive amounts may increase the risk. (*From* Watson RR, Eisinger DC, Nieman DC, et al: Exercise and Disease. Boca Raton, FL, copyright CRC Press, Inc., 1992, p 123; with permission.)

tions in cardiac, respiratory, and skeletal muscle function have been documented, and respiratory tract infections can be associated with muscle aches and a subjective sensation of weakness. Friman[29] assessed isometric strength in subjects with recent viral infections and a matched control group. A 5% to 15% reduction in isometric strength was found in infected subjects that had not fully recovered compared with reference values 1 month after illness. Astrom et al[1] examined muscle tissue obtained from patients recovering from recent viral and mycoplasma illnesses and noted significantly reduced oxidative and glycolytic skeletal muscle enzyme activity in infected patients. Electron microscopy showed focal abnormalities in muscles similar to changes seen in some types of muscular diseases. When muscle biopsies were repeated 3 months after illness, these problems were almost completely resolved. A study by Friman et al[30] in 1985 found that isometric strength and cardiac stroke volume were reduced during the pyrexial phase of sandfly fever. Muscle performance decrements, which were correlated to subjective symptoms such as myalgia, recovered with defervescence; however, stroke volume remained below the preinfection baseline during early recovery. During fever, an increased heart rate maintained cardiac output at preinoculation levels, whereas cardiac output fell in early convalescence. This decrease in cardiac output correlated significantly with the severity of fever. Sandfly fever is an unusual viral infection and may not be representative of typical viral illness in humans, but these findings are disturbing and warrant further investigation.

Sudden death has occurred in young people and is often associated with vigorous exercise. In a study of 78 sudden deaths during or immediately after exercise, Jokl and McClellan[40] found a history of recent URI in 5 out of 78 (6%). Cardiovascular problems accounted for most of the remainder. It has not been proved that intercurrent viral illness increases the risk of sudden death during exercise, but there are a surprising number of anecdotal reports of deaths in young healthy people who had been vigorously exercising during an acute viral illness.[64] Several studies have demonstrated that exhaustive exercise after contracting an infection may be detrimental, possibly owing to the increased virulence of the coxsackievirus, which has a predilection for the heart muscle with intense exercise.[62] The myocarditis and pericarditis that these viruses produce may increase the risk of acute arrhythmias leading to sudden death.[44, 75] In a 1991 report, 2 of 34 sports-related sudden cardiac deaths were from myocarditis: a 14-year-old boy who died while swimming and a 35-year-old man who died while running.[10] In these two deaths, the only cardiac abnormality at autopsy was focal microscopic myocyte necrosis associated with a lymphocytic infiltrate. Neither patient had a history of fever or flu-like illness; however, this study was retrospective. It is possible that a viral infection caused the myocarditis, and exercise, in the face of this myocarditis, triggered an arrhythmic death.[24]

Several investigations have examined the effects of URI on pulmonary function and found detectable abnormalities. Decreases in maximal inspiratory and expiratory flow rates in both children and adults have been demonstrated.[18, 61] Small or peripheral airway abnormalities are also

associated with URI.[8, 12] Respiratory muscle strength was studied in 12 subjects who developed naturally acquired URI, and maximal inspiratory and expiratory mouth pressures fell significantly during these infections.[50] The lowest pressures usually occurred between the third and seventh day of clinical illness, which was often after the peak of clinical symptoms. Full recovery did not take place until the 14th day (Fig. 2). The authors concluded that weakness of the inspiratory muscles may contribute to breathlessness during exertion, which is a common symptom in previously healthy young adults who develop acute URIs. These studies indicate that asymptomatic mechanical dysfunctions of the lungs are a frequent sequelae of acute viral URIs and that deterioration in pulmonary function can occur even after apparently mild common colds with no clinical evidence of lung involvement.

Unexplained deterioration in athletic performance can in some individuals be traced to either recent respiratory tract infections or subclinical viral infections that run a protracted course.[63] Daniels et al,[20] for example, concluded that during a mild febrile state, there is a marked effect on the ability or willingness of some individuals to perform both cardiorespiratory and musculoskeletal exercise.

Recommendations

Infections that are subclinical in the normal population may greatly affect maximum performance in athletes. Exercising during the prodro-

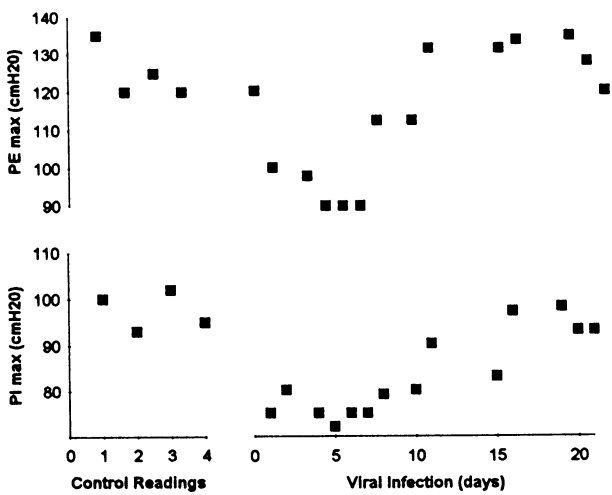

Figure 2. Sequential recordings of maximal static expiratory (PE max) and inspiratory (PI max) mouth pressures before, during, and after an upper respiratory tract infection in one representative subject. (*From* Mier-Jedrzejowicz A, Brophy C, Green M: Respiratory muscle weakness during upper respiratory infections. Am Rev Respir Dis 138:5, 1988; with permission.)

mal and early acute phase of some infections may worsen or prolong the illness and set the athlete up for a greater risk of potential complications.[27] Aforementioned negative alterations in cardiac, respiratory, and skeletal muscle function may individually or collectively hamper performance. Significant biomechanical changes were noted during naturally occurring URI (Weidner T: Unpublished observations, 1993), which could lead to an increased risk of musculoskeletal injury for athletes participating in sports during illness. From a practical standpoint, any workout during a severe viral infection would be suboptimal
in enhancing fitness or sports-specific skills; therefore if signs and symptoms indicate that viral infection is impending, the athlete should greatly reduce volume and intensity of heavy training for 1 to 2 days. Most clinical authorities recommend that if the athlete has symptoms of the common cold with no constitutional involvement, training may be safely resumed a few days after resolution of symptoms.[64, 71] Mild exercise during sickness with the common cold does not appear to be contraindicated, but strenuous exercise should be avoided. For competitive athletes who are afraid to miss any training days even when ill, Eichner[25] recommends athletes do a *neck check.* If symptoms are located "above the neck," such as stuffy or runny nose, sneezing, or scratchy throat with no constitutional symptoms, the athlete should be allowed to proceed cautiously through their scheduled workout at half-speed. After a few minutes, if the head clears and the athlete feels better, he or she can gradually increase the intensity and finish the workout. If the individual feels worse, he or she should rest. The athlete with "below the neck" symptoms, such as fever, aching muscles, hacking or productive cough, vomiting, or diarrhea, should not train.

If a patient has symptoms and signs of systemic involvement, 10 to 14 days should be allowed before resumption of full intensive training and exhaustive exercise to avoid relapse, increased risk of musculoskeletal injury, or potential life-threatening complications, such as cardiomyopathy and arrhythmia. These precautions are advised because of the growing documentation showing a relationship between intensive exercise and the risk of developing viral cardiomyopathy and other severe forms of viral infection.[70] Both epidemiologic and clinical data support the concept that heavy exertion increases the athlete's risk of URI because of negative changes in immune function. Considerable evidence indicates that environmental factors, such as improper nutrition and psychological stress, can compound the negative influence that heavy exertion and training can have on the immune system. Based on current understanding, the athlete is urged to eat a well-balanced diet, keep other life stresses to a minimum, avoid overtraining and chronic fatigue, keep away from individuals who are ill before and after important events, obtain adequate sleep, and space vigorous workouts and competitive events as far apart as possible.

INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis (IM), the clinical and hematologic manifestations of which are most commonly observed in adolescents and young adults, was discovered in 1967 to be caused by the Epstein-Barr virus (EBV). This double-stranded DNA virus of the herpes group displays itself in its host as a self-limiting, lymphoproliferative disease with autoimmune features. Although IM is rarely life-threatening, its management in the patient-athlete should be monitored as well as individualized before training is resumed.

The EBV is transmitted through oral secretions, and following the acute infection, it is excreted in the saliva continuously or intermittently for months.[54] Although this excretion is present for a lengthy period, it is extremely uncommon for IM epidemics to occur as a result of the repeated as well as prolonged exposure of the virus needed to infect an individual. It has even been demonstrated that the college roommates of IM patients have no increased risk of infection.[34, 67] The EBV itself infects the antibody-producing B lymphocytes, which in turn causes the proliferation of atypical B cells. A surge of T lymphocytes, responsible for cell-mediated immunity, follows along with the development of antibodies against the EBV, the combination of which terminates the infection. The intense T cell response produces the characteristic lymphoid hyperplasia, striking lymphocytosis, and appearance of atypical lymphocytes in the peripheral blood.

IM most often occurs between the ages of 15 and 25, with 25% to 50% of these infected individuals developing the classic syndrome. Cases do present in preadolescents and the elderly, but the clinical manifestations commonly differ in these two age groups. By age 30, more than 90% of Americans have been infected with EBV. It has also been noted that 1% to 3% of college students become infected per school year. Males and females have an approximately equal frequency of infection, whereas the rate is 12 to 30 times higher in whites than in blacks. The racial difference has been attributed to more frequent early life experience with EBV in blacks.[47]

The incubation period for primary EBV infection is approximately 30 to 45 days. The prodromal period tends to last 3 to 5 days with symptoms of headache, fatigue, anorexia, malaise, and myalgias. In the following 5- to 15-day period, the clinical manifestations of classic IM include moderate to severe sore throat with tonsillar enlargement (one third of patients have tonsillar exudates), moderate fever, enlarged tender posterior cervical lymph nodes with lymphadenopathy often generalized, petechiae on the soft palate, and a palpable enlarged spleen by the second week in 50% to 70% of patients. Hepatomegaly occurs in approximately 35% of cases, but fewer than 15% present with clinical jaundice, and with even less frequency, a rubellalike rash and periorbital edema are present (Fig. 3). The EBV infection can be severe in older adults, with debilitating malaise, fatigue, and fever, whereas in very young children, the virus produces such minimal symptoms, such as mild tonsillitis, that it is often not recognized as an EBV infection.

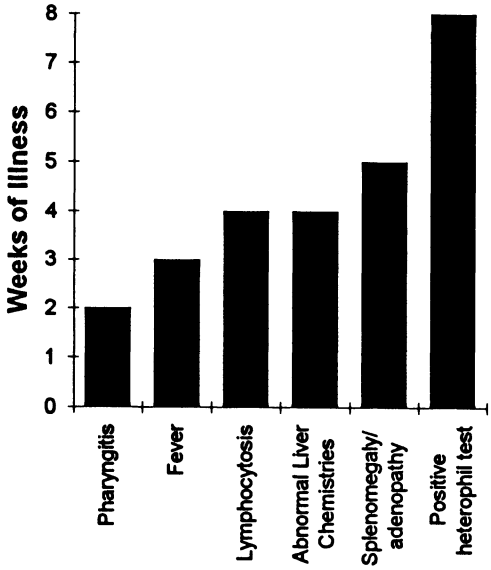

Figure 3. Longest duration of major features of infectious mononucleosis in 90% of patients. In mild cases, the duration is shorter than shown here; in 10% (severe cases), the duration is longer. (*From* Eichner ER: Infectious mononucleosis: Recognition and management in athletes. The Physician and Sportsmedicine 15:12, copyright 1987; with permission.)

The hematologic abnormalities most often noted are modest leukocytosis (10,000 to 20,000/mm^3) during the first week of illness with the most consistent feature being a striking absolute lymphocytosis (50% of the total white blood cell count and 4500 lymphocytes/mm^3) with many atypical lymphocytes (10% to 20% of all leukocytes, 1000/mm^3).[37] A mild reduction in platelet count (140,000/mm^3) is present in approximately 50% of patients. By the second week of illness, approximately 85% of patients with IM show abnormalities in liver function tests that reflect mild hepatitis (modest elevated transaminases, alkaline phosphatase, and lactic dehydrogenase). By the fourth to fifth week of illness, the hematologic values and liver function tests return to normal.

In most cases, clinical suspicion of IM induced by the EBV can be confirmed serologically with a rapid slide test, such as the Monospot (Ortho Diagnostic Systems, Raritan, NJ), which is based on the classic heterophil antibody absorption test. It is extremely sensitive for detection of EBV-induced IM, but repeat testing may be needed because only 60% of patients have heterophil antibodies by the second week of illness. This test is also negative in most children younger than 5 years of age. If the heterophil test is negative, confirmation of IM requires an EBV-specific antibody study. The cytomegalovirus (CMV) and *Toxoplasma gondii* are also causes of heterophil-negative IM but may produce some varied symptoms. Often a precise diagnosis is not needed, but when dealing with the athlete and recommendations regarding physical activity, a firm diagnosis is a necessity.

There seems to be a difference in the clinical course of IM when comparing athletes to nonathletes. To substantiate this theory, it has been shown that the ratio of symptomatic to subclinical cases was far higher

in Yale undergraduates (3:1) than in the physically more active West Point cadets (1:3).[34, 67] Athletes also return to training more quickly after illness than nonathletes return to their usual activities, most likely owing to the increased motivation to return to competition. The athlete may recover quicker but may not be able to compete at his or her preillness level of fitness for as long as 3 months.

Complications

Life-threatening complications of IM are rare, but the most serious include splenic rupture and airway obstruction. Although palpable spleen enlargement occurs in approximately 50% of patients, the prevalence of splenic rupture is less than 0.2%. Nearly all ruptures occur between the 4th and 21st day of symptomatic illness, and the spleen tends to enlarge to 250 to 500 g, or two to three times the normal size. In a 1976 survey, 22 cases of traumatic splenic rupture in athletes were reported, all in white males. Seventeen cases (77%) occurred during participation in football. Nine patients (41%) had IM at the time of the trauma.[28] It should be noted, however, that most ruptures do not occur during athletic activities but while performing daily routines such as lifting, defecating, or getting in and out of bed. Although uncommon, rupture caused from palpation of the spleen by a physician has occurred; therefore care should be given when examining a patient, and percussion instead of palpation may be preferable when assessing splenic size. Pain from splenic rupture begins suddenly in the left upper quadrant of the abdomen, is usually worse with inspiration, and may radiate toward the left shoulder (Kehr's sign). These symptoms may then be followed by generalized abdominal pain and shock. Ultrasound and computed tomography (CT) are useful in diagnosing both splenomegaly and splenic rupture. The treatment of choice is often splenectomy, but nonoperative management has been advocated.

Airway obstruction owing to massive enlargement of tonsils and adenoids has occurred and may require emergency nasotracheal intubation. The use of corticosteroids can shrink the tonsils and adenoids within hours. Group A beta-hemolytic streptococcal pharyngitis is found in 5% to 30% of the patients and is treated with penicillin or erythromycin. Treatment with ampicillin should be avoided because it produces an erythematous maculopapular rash owing to a unique antibody present in IM. Neurologic complications, such as encephalitis and Guillain-Barré syndrome, occur in fewer than 1% of the patients. Also rarely seen are pneumonitis, autoimmune hemolytic anemia, severe thrombocytopenia or granulocytopenia, myocarditis, and pericarditis.

Treatment

Treatment for IM is supportive for the most part, consisting of rest, fluids, and analgesics. Acetaminophen is recommended for fever, head-

ache, and muscle pain along with lozenges, saltwater gargles, or viscous lidocaine for sore throat. Occasionally codeine is prescribed for refractory pain. Stool softeners should be used when codeine is being taken or when splenomegaly is present, to decrease the possibility of constipation and straining, which could lead to splenic rupture. Corticosteroid therapy is recommended in patients with severe hepatitis, immune cytopenia, myocarditis, or neurologic complications. A short course of corticosteroids early in the acute phase of illness with starting doses of 40 to 80 mg of prednisone and tapering over 5 to 12 days has been shown to decrease the severity of fulminant respiratory symptoms. There is no definite evidence demonstrating a reduction in splenic size with corticosteroid use and little or no effect on the duration of illness.[49] The clinical benefit of acyclovir has been shown to be minimal, with no improvement in spleen and liver size or lymphadenopathy.

There have been theories proposed that IM may lead to the chronic fatigue syndrome. Because of the variety of causes associated with the chronic fatigue syndrome, including viral, endocrine, and psychoneurotic, and the lack of a diagnostic test, serious doubts have been raised about this syndrome being caused by a single virus, such as EBV.

Return to Play

After being diagnosed with IM, the patient-athlete is likely to be concerned about the length of time this disease will prevent participation in training and competition. In the initial stages, when fever, pharyngitis, lymphadenopathy, and fatigue are present, even the most committed athlete does not feel like exercising strenuously.[79] There is no need for prolonged bed rest, which can cause deconditioning and possibly slow recovery. The most important consideration in determining when the physician should permit return to play is the possibility of splenic rupture. Because rupture usually occurs during the second or third week of illness, the patient, parents, coach, and trainer should be advised that the patient-athlete will not be able to compete until at least after the third week of illness has passed. On initial physical examination, the spleen size should be noted as well as during weekly follow-up examinations. After the third week when permission for return to play is being considered, noninvasive imaging, such as ultrasonography or CT scanning, is suggested and provides a highly accurate means of determining spleen size. If splenomegaly is present at this time, another imaging study should be done in 1 week, or after 5 weeks from the onset of illness, the patient can be permitted to return to limited activity. If splenomegaly is not present and the patient is afebrile, pharyngitis has resolved, and liver functions if initially elevated have returned to normal, the patient may be allowed to resume easy training without contact, such as jogging, swimming, or cycling, at approximately 50% of maximum (Table 1). If after 1 week, the patient is able to endure this level of activity, full training can be resumed. Above all, treatment must be individualized,

Table 1. CRITERIA FOR RETURN TO ACTIVITY IN AN ATHLETE WITH INFECTIOUS MONONUCLEOSIS

Weeks Following the Onset of Illness*	Criteria for Return to Activity†
Week 1 (day 7)	No return recommended
Week 2 (day 14)	No return recommended
Week 3 (day 21)	No subjective complaints
	Pharyngitis and lymphadenopathy resolved or nearly resolved
	Afebrile
	Bilirubin <3 mg/dL
	Liver enzymes <3-fold
	No complications
	No splenomegaly, with confirmation by radiographic means‡
Week 4 (day 28)	Same as Week 3
>Week 5 (>day 35)	No subjective complaints
	Pharyngitis and lymphadenopathy resolved
	Afebrile
	Bilirubin <3 mg/dL
	Liver enzymes having peaked and returning to baseline
	No splenomegaly by physical examination
	Complications completely resolved§

*Onset of acute phase of illness, not to include prodromal period.

†Return to activity is graded; initial return is at 50% with avoidance of contact for 1 week, then return to full activity as tolerated.

‡Ultrasound, computed tomography, or radionuclide scan.

§If a complication develops, consultation with appropriate specialist recommended.

From McKeag DB, Kinderknecht J: A basketball player with infectious mononucleosis. *In* Smith J (ed): Common Problems in Pediatric Sports Medicine. Chicago, Mosby-Year Book, 1989, p 201; with permission.

and the patient-athlete should return to activity, whether routine or competitive, only when he or she feels physically ready.

GASTROINTESTINAL DISORDERS

Gastrointestinal disorders in athletes vary from viral gastroenteritis, to food poisoning, to traveler's diarrhea. Although the cause and clinical symptoms may differ with all of the following illnesses, steps must be taken to prevent dehydration to enable the athlete to return to participation as soon as possible.

Viral Gastroenteritis

Viral gastroenteritis occurs in two major forms: epidemic and sporadic. The epidemic form is caused by the Norwalk virus, whereas the sporadic form is caused by the rotavirus. The principal difference between the two viruses is that the rotavirus, with an incubation time of 24 to 48 hours, usually infects infants and young children and may also

cause symptoms that range from subclinical illness to life-threatening dehydration. The Norwalk virus typically is seen in adults and older children and seldom produces an illness severe enough to require hospitalization.[42] Both viruses are most commonly transmitted through the fecal-oral route, with respiratory transmission also being possible in the rotavirus.[43] Clinical symptoms of gastroenteritis include nausea, diarrhea, and vomiting, along with fever, abdominal cramps, and myalgias. Although the course of gastroenteritis is normally self-limited, fluid replacement during the illness is essential. Clear fluids, especially those containing electrolytes (Gatorade), are suggested along with caffeine-free soft drinks, fruit juices, broths, and bouillons. If severe dehydration occurs, it may be necessary to hospitalize the athlete so intravenous rehydration can be instituted. Antimotility drugs, such as diphenoxylate (Lomotil) or loperamide (Imodium), can be effective in reducing abdominal cramps but must be used with caution because they can prolong the gastrointestinal infection. Recommendations on return of the athlete to practice are mainly limited to making sure he or she is rehydrated and that the physical symptoms, such as diarrhea, have resolved.

Food Poisoning

Food poisoning is most commonly caused by the *Staphylococcus* bacterium during the warm weather months. Dairy products and meats are frequently contaminated, usually from the infected skin or respiratory tract of the food handler. A toxin is produced when the bacteria is allowed to multiply while in an unrefrigerated environment and causes vomiting and diarrhea 1 or 2 hours after the food is ingested. Symptoms usually subside after a few hours, and treatment is not necessary except for rehydration of the athlete. As with viral gastroenteritis, the use of antimotility drugs is not recommended because they may prolong or worsen the illness.

Traveler's Diarrhea

Athletes going to Latin America, Africa, the Middle East, and Asia are at highest risk for acquiring traveler's diarrhea. Symptoms typically begin during the first week of the stay and usually last 48 to 72 hours, although peak athletic performance may not be regained for a week or more. *Escherichia coli* accounts for approximately 50% of the cases of traveler's diarrhea, but *Salmonella, Shigella, Giardia lambia,* and rotavirus can also produce this illness, which is passed through the fecal-oral route. Symptoms include diarrhea, cramps, nausea, and malaise. For preventive purposes, tap water and iced beverages should be avoided as well as food from street vendors, fresh leafy greens, and fruits that cannot be peeled before eating. Athletes who are not allergic to salicylates can prophylactically use bismuth subsalicylate (Pepto-Bismol) by

taking 2 ounces of liquid or 2 tablets four times a day. The most important treatment once again is rehydration. In the absence of fever or blood in the stool, loperamide may be used to help control the diarrhea in a dosage of 4 mg initially followed by 2 mg after each unformed stool. Patients with moderate to severe traveler's diarrhea can be treated with a combination of 160 mg trimethoprim and 800 mg methoxazole (Bactrim DS) for 3 to 5 days.

OTITIS EXTERNA

Otitis externa, also known as swimmer's ear, is an inflammation of the external auditory canal and is typically seen among swimmers, divers, and surfers during the summer months or when the weather is hot and humid. This condition is most commonly caused by the bacterium *Pseudomonas aeruginosa* but has been related to infection by the fungus *Aspergillus*.[68]

Cerumen, which is normally present in a healthy ear canal, is water repellent and aids in maintaining an acidic pH in the canal, thus preventing bacterial and fungal growth. Athletes who spend time in the water tend to have little cerumen present, causing them to lose their natural defense mechanisms against bacteria and fungus. This lack of cerumen also leads to absorption of the moisture retained in the canal after water exposure, producing a hyperhydrated and macerated canal. Other factors that contribute to an athlete's development of otitis externa are freshwater swimming, swimming in improperly chlorinated pools, scratches caused by insertion of objects to clean or relieve itching in the canal, and the degree to which the athlete's head is submerged in the water.[77]

The patient with otitis externa presents clinically with exudate, edema, and erythema in the canal along with a feeling of fullness, mild itching, and an increase in the level of pain by pulling on the auricle.

Treatment consists of cleansing the ear canal by irrigation with water or hydrogen peroxide, followed by the use of topical antibiotic drops such as polymyxin or 0.25% acetic acid solution. If excessive edema is present, insertion of a wick saturated with 50% Burow's solution may be used. Fungal infections can be treated with 1% tolnaftate solution three times per day for 7 days. Often intense itching is an annoying symptom and can be controlled with corticosteroid drops once the infection is under control.[74] After treatment has started, the athlete may return to water activities within 2 to 3 days, as long as the pain with examination, drainage, and redness has resolved.

The prevention of otitis externa is associated with keeping the ear canals as dry as possible. Suggestions include gentle towel drying of the ears after getting out of the water, avoiding scratching or touching the ears, use of drying agents, and wearing swim caps or using silicone ear plugs.

DERMATOLOGIC INFECTIONS

The dermatologic infections of athletes can be classified as bacterial, viral, or fungal. For the most part, these infections are easily treated, but early diagnosis is important to prevent those disorders that are contagious from being transmitted to fellow athletes.

Bacterial Infections

In general, bacterial skin infections tend to have several causative factors in common, including a history of minor lacerations or skin abrasions, irritation from pads or tape, sweat, dirt, occlusion by equipment as well as contact from infected athletes. The following disorders are those most frequently seen in athletes.

Impetigo

Impetigo presents as a superficial infection of the skin that is caused by either β-hemolytic streptococci or *Staphylococcus aureus* and is most commonly seen in wrestlers, swimmers, and gymnasts. The initial lesions vary from small vesicles to large bullae. These lesions rupture and exude a honey-colored serous fluid, which then forms a crust. Impetigo can occur in children or adults and most often forms on the face but also appears on other body parts. A bacterial skin culture, which confirms the clinical diagnosis, can be performed by removing the crust and swabbing the base of the vesicle. Because of the small potential for associated glomerulonephritis, a urinalysis to examine for red blood cells and protein should also be obtained if a streptococcal infection is found.[41] The lesions should be debrided with hydrogen peroxide then treated with the topical antibiotic mupirocin (Bactroban), three times per day for 7 to 10 days. Because impetigo is contagious, athletes who are infected should not participate in contact or water sports until the lesion has healed. In addition, neither towels nor athletic equipment should be shared.

Furuncles

A furuncle, also known as a boil, is an infection of hair follicles or sebaceous glands and is usually caused by *S. aureus.* The lesions present as warm, tender, inflamed nodules or abscesses on the trunk or extremities and can arise from existing areas of folliculitis. A 7- to 10-day course of oral antibiotics is indicated as well as a stab wound to facilitate drainage if the lesion is fluctuant. An outbreak of furuncles was documented among male athletes in a Kentucky high school during the 1986–87 school year, with 25% of the basketball and football players being affected.[76] As with impetigo, athletes who are swimmers or involved with contact sports must stop participation until the furuncle has resolved.

Folliculitis

Hot tub folliculitis, caused by *P. aeruginosa,* has been recognized as being associated with the use of hot tubs, whirlpools, jacuzzis, and swimming pools.[14] These reddish papulovesicular pustular lesions usually appear on the axillae, breast, or pubic areas within 2 days of exposure. Therapeutic measures are typically not needed owing to the disease being self-limiting, lasting 7 to 10 days. To reduce the presence of the *Pseudomonas* organism, close monitoring of chlorine and disinfectant levels and higher water temperatures at a pH of 7.2 to 7.8 are necessary.

Erythrasma

Erythrasma is an infection caused by *Corynebacterium minutissimum* and is most often seen in the groin folds or axillae. This lesion mimics a fungal infection and appears as reddish brown patches of desquamation. A definitive diagnosis is made by examination with Wood's light (longwave ultraviolet light), which causes a coral red fluorescence to be displayed. Elimination of the infection is usually achieved by use of topical antibiotics or antibacterial soaps, whereas more severe cases may require oral erythromycin or tetracycline at a dosage of 1 g daily for 10 to 14 days.

Pitted Keratolysis

Pitted keratolysis is an infection of the plantar skin caused by *Corynebacterium.* Owing to the strong odor this infection produces, it has been referred to as the "toxic sock" syndrome. It clinically presents as 1- to 3-mm circular areas of erosion with a punched-out appearance and is frequently seen in tennis and basketball players as well as runners. This infection tends to develop in a hyperhydrotic environment and is aggravated by occlusive footwear. Treatment includes agents that promote drying, such as 20% aluminum chloride (Drysol), 5% benzoyl peroxide preparations, or 2% erythromycin solution. Removing the moisture-filled environment by, for example, wearing absorbent cotton socks and changing them frequently is important. For more resistant infections, oral erythromycin can be used.

Viral Infections

Warts

Warts, also known as verrucae, are epithelial tumors caused by many types of the human papillomavirus. Common warts usually develop on the hands and present as cauliflower-shaped, raised areas that are irregular and rough. Plantar warts appear differently because they occur on a weight-bearing surface and develop as a flat lesion extending deep into the skin with a hyperkeratotic surface. A plantar wart can be

differentiated from a callus by trimming the lesion superficially. Pinpoint bleeding points are produced when a plantar wart is present. Although common warts may be a cosmetic problem, plantar warts tend to be the most disabling for athletes. Because of the effects of perspiration on the skin of the feet, athletes seem to have a greater predisposition for plantar warts.[4] The incubation period for warts is approximately 6 months. Although the infectivity rate is low, inoculation may result from exposure to a wart or from contaminated floors, equipment, and clothing.[78] Because calluses are more susceptible to papillomavirus infections than normal skin, gymnasts, football players, and wrestlers commonly develop warts.[5] Although warts are often difficult to treat, their course is self-limiting. Surgery and radiotherapy are no longer recommended because these destructive techniques can compromise athletic performance. Plantar warts are often effectively treated with 40% salicylic acid plasters. Both types of warts tend to respond to topical 17% salicylic acid (Duofilm) as well as compounds of 17% salicylic acid and lactic acid in flexible collodion (Viranol) applied nightly. More painful plantar warts can be treated with a weekly injection of 1% lidocaine into the base of the wart over a period of 1 to 3 weeks. For preventive measures, athletes prone to warts should consider using drying powders on their feet as well as wearing rubber sandals in locker rooms.

Molluscum Contagiosum

Molluscum contagiosum is a viral infection caused by a large pox virus, which most commonly develops on the hands, forearms, and face. It clinically appears as solitary or multiple lesions that are raised, umbilicated, firm, skin-colored papules, ranging from 2 to 4 mm. Frequently lesions are located along a scratch (Koebner's lines) and are characteristic of molluscum contagiosum. This infection is transmitted by personal contact, especially in swimming pools or gymnasiums; therefore swimmers and wrestlers are often affected. Athletes who have this lesion should refrain from practice and competition until it has cleared. Treatment consists of curettage of each papule followed by electrocautery or application of liquid nitrogen.

Herpes

Herpes simplex is a viral infection of the skin and mucous membranes whose clinical course consists of three stages: a primary infection, a latent phase, and then recurrence. There are two types of herpes simplex virus. Type 1 is typically localized above the waist, usually on the face and hands, whereas type 2 affects the genitals. Herpes simplex presents clinically as a group of 1- to 2-mm vesicular bullous lesions on an inflammatory base, which may last 2 to 3 days before the tops come off and a crust forms. The crusted lesions then last 5 to 7 days. Besides the appearance of skin lesions, approximately 25% of those infected develop systemic symptoms, such as fever, myalgia, lethargy, headache,

and sore throat. Events such as trauma, sunlight, illness, surgery, stress, or menstruation may later trigger a recurrence.[78] Because the virus has a high level of infectivity and is transmitted through skin-to-skin contact, athletes in contact sports are especially susceptible. Wrestlers, in particular, are commonly infected with this virus; thus the term *herpes gladiatorum* has been coined. The lesions in wrestlers tend to occur on the right side of the face or body owing to the grappling positions used in practice or competition.

During an outbreak in 1989 at a wrestling camp in Minnesota, out of the 60 wrestlers infected (34% of those attending the camp), 73% had lesions on the head, 42% on the extremities, and 28% on the trunk. Also, 57% had lesions only on the right side of the body, 35% had lesions on both sides, and 8% had lesions only on the left side.[6] A definitive diagnosis of herpes simplex can be made by examining scrapings microscopically using Tzanck stain and looking for multinucleate giant cells. Although intramuscular and topical acyclovir has been used in the past, the treatment of choice is oral acyclovir (Zovirax) with 400 mg given 3 times per day for 7 to 10 days, which tends to reduce pain, duration of lesions, and virus shedding.[53] This simplified regimen produces a similar response to the more traditional oral acyclovir dosing (200 mg, five times per day) and improves compliance.

Those athletes who recognize a definite prodrome before the skin lesions appear can reduce the severity of their symptoms by beginning oral acyclovir at the time of prodrome. Oral acyclovir was used prophylactically in a double-blind, controlled study on immunocompetent individuals with frequently recurrent herpes labialis. Patients placed on 400 mg of acyclovir, twice daily, had a 71% decrease in virus culture-positive lesions as compared with the placebo group.[65] Side effects of oral acyclovir are minimal, although nausea, vomiting, and diarrhea can occur. Although acyclovir is useful in treating herpetic lesions, athletes must not be allowed to participate in practice or competition until the lesions are fully healed. The recognition of this viral infection early and removal of the infected athlete from participation immediately are the most important factors in preventing widespread outbreaks from occurring.

Fungal Infections

Tinea is the name applied to various superficial fungal infections of the skin, which are classified according to the location of the infection. A definitive diagnosis for all fungal infections can be made by KOH preparations of skin scrapings. Although therapy for fungal infections may vary according to the site of infection, there are several contributing factors that they usually have in common, including the presence of increased moisture from sweat, occlusive footwear, shared towels, contaminated locker room floors, and minor cutaneous injuries.

Tinea pedis, also known as athlete's foot, is the commonest fungal infection found in athletes. It is most typically caused by *Trichophyton*

rubrum, which presents as an asymptomatic erythematous area with peripheral scaling, or *Trichophyton mentagrophytes,* which presents with painful, itchy blisters. The most important diagnostic clue is infection of the toe-web between the fourth and fifth toe.[5] A dermatophid reaction often occurs with athlete's foot, appearing as dyshidrotic eczema with pruritic vesicles or annular plaques on the hands; therefore if hand dermatitis is recognized, the athlete should be examined for tinea pedis.[81] Topical treatment with fungistatic antifungals such as clotrimazole (Lotrimin) or ketoconazole (Nizoral) is effective and should be applied several times a day. They often provide symptomatic relief but relapse is common. Terbinafine (Lamisil) offers significant advantages over most topical preparations owing to its fungicidal and anti-inflammatory properties. A 1-week course of terbinafine twice daily has been shown to produce both symptomatic relief and mycologic cure.[6a] The importance of preventive treatment cannot be overemphasized. Precautions such as keeping the foot area dry, wearing absorbent socks and changing them frequently, using drying powder, wearing nonocclusive leather shoes, and wearing sandals in public shower areas are recommended.

Tinea cruris, also known as jock itch, is a fungal infection of the groin and upper thighs, although the scrotum is normally not involved. It is caused by the same fungi that give rise to tinea pedis and often begins on the feet and spreads via clothing or towels to the groin area. Tinea cruris appears clinically as red, scaly patches, with pruritus and itching being common. Treatment is the same as for tinea pedis, and prevention includes good hygiene and wearing clean, loose clothing to help prevent the presence of a moisture-rich environment. In addition, it is recommended that the athlete put on socks before putting on underwear and towel-dry the feet last after showering to reduce the chance of spreading the infection from the feet to the groin.[3]

Tinea corporis is a fungal infection of the smooth skin of the body and is usually caused by *Trichophyton tonsurans* as well as the same organisms responsible for tinea pedis and cruris. The lesions present as annular areas, with the outer edges being erythematous while the center portion is often clear; thus the term *ringworm* has commonly been used. These lesions often appear on the shoulders, neck, and back. Although not as prevalent in athletes as tinea pedis and cruris, an outbreak of tinea corporis in wrestlers, termed *tinea corporis gladiatorum,* has been documented in New York and in Baltimore.[15] The most effective treatment again seems to be topical antifungals and oral fluconazole if necessary.

Tinea of the nails, also known as onychomycosis, tends to be commoner in toenails than fingernails. Although caused by the same fungi as tinea pedis, this infection is not as easily treated. It presents as a distal or lateral detachment of the nail with subsequent thickening and deformity. In the past, total cure of this infection was rare, but fluconazole has now shown positive results. Treatment with 100 mg orally every other day has shown cures of infections in 16 to 32 weeks.[17] An alternative fluconazole regimen of 200 mg given once a week for 16 weeks in fingernail and 32 weeks in toenail infections has produced long-term cures.

Tinea versicolor is a fungal infection common in swimmers and divers caused by *Malassezia furfur.* The upper part of the chest as well as

the back, neck, and arms is typically affected with white or light tan-colored, irregularly shaped scaly patches that are asymptomatic. Inspection under a Wood's lamp reveals a characteristic yellow-orange color. Treatment to kill the fungus is best accomplished by applying 2.5% selenium sulfide shampoo for 15 minutes daily for 3 days then once weekly for prevention. An alternative regimen consists of applying this medication to the affected areas at bedtime then rinsing it off in the shower the next morning. This method may lead to minor skin irritation in sensitive patients, but it is easier to use. The athlete should be reminded that after the tinea is cured, the depigmented spots may remain for 1 to 2 months until gradual repigmentation can occur by natural mechanisms.

IMMUNIZATIONS IN ATHLETES

Guidelines for immunizations in athletes are similar to the guidelines for sedentary people.[72] There are specific situations, however, that deserve additional consideration.

Tetanus

Tetanus immunizations should be kept up-to-date in all adolescents and adults. This is especially important in athletes because of the relatively high risk of trauma that may occur with sports participation. A tetanus diptheria booster every 10 years maintains immunity following a primary series. Traumatic wounds that occur in athletes who are not fully immunized should be managed according to guidelines depending on history of immunization status as well as the type, extent, and contamination of the wound (Table 2).

Table 2. SUMMARY GUIDE TO TETANUS PROPHYLAXIS IN ROUTINE WOUND MANAGEMENT—UNITED STATES

History of Absorbed Tetanus Toxoid (Doses)	Clean, Minor Wounds		All Other Wounds*	
	Td†	TIG	Td†	TIG
Unknown or <3	Yes	No	Yes	Yes
≥3‡	No§	No	No¶	No

*Such as but not limited to wounds contaminated with dirt, feces, soil, saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

†For children under 7 years old, DPT (DT, if pertussis vaccine is contraindicated) is preferred to tetanus toxoid alone. For persons 7 years old and older, Td is preferred to tetanus toxoid alone.

‡If only three doses of fluid toxoid have been received, a fourth dose of toxoid, preferably an absorbed toxoid, should be given.

§Yes, if more than 10 years since last dose.

¶Yes, if more than 5 years since last dose. (More frequent boosters are not needed and can accentuate side effects.)

Td = Tetanus and diphtheria toxoids; TIG = tetanus immune globulin.

From Center for Disease Control: Diphtheria, tetanus, and pertussis: Guidelines for vaccine prophylaxis and other preventive measures. MMWR 34:27, 1985; with permission.

Influenza

Influenza is certainly not a life-threatening illness in young individuals; however, in a top-level athlete, a bout of influenza can mean losing valuable practice and playing time that could diminish a team's competitive edge. Immunization against influenza may be particularly beneficial for athletes who play winter sports in the snowbelt because close contact among team members can result in rapid spread and disrupt a significant portion of the season. In the competitive environment of collegiate and professional athletics, decreased performance secondary to influenza among key participants could have a significant impact on a team's conference and national rankings as well as tournament play. At this level of competition, the loss of a single game can mean the difference between the end of a season and a bid to the national championships.

College athletes are at higher risk because of the special environment the college campus provides. In this setting, athletes move from class to class, travel home for breaks to different parts of the United States, and then return to campus bringing a variety of potentially infectious strains of viruses with them. Many factors affect a decision regarding immunization of athletes against influenza. Some athletes want to avoid an injection even more than they want to avoid a possible illness, and coaches might not want their team immunized because they fear unwanted side effects. Cost may also be a consideration, especially for a large group of athletes. A single dose of influenza vaccine during the 1992–93 "flu" season was approximately $3, which is not prohibitive, but when one takes into account the expense of syringes and the fee for administration, costs can rise rapidly.

Athletes should be offered influenza vaccinations only if supplies are adequate first to meet the needs of the population at high risk for influenza complications, such as the elderly, chronically ill, and providers of medical and essential community services. Lack of an adequate supply has so far not been a problem because unfortunately fewer than 30% of patients who are at high risk receive the vaccine each year.[72]

In advising athletes and coaches on who should be immunized, it is best to consider not only the season of the sport, but also the likely incidence of a particular strain of influenza occurring in each specific circumstance. Rationales for and against immunizations and factual information about specific side effects should be discussed with team coaches as well as the athletes. Influenza vaccines should not be administered to athletes if (1) they are allergic to eggs because the vaccine is produced in egg embryo cultures or (2) they have a current febrile illness.

In contrast to the typical *high-risk* patient receiving an influenza vaccine, healthy young athletes maintain high levels of antibodies for up to a full year, so athletes can be immunized as early as September and still have adequate antibody levels for the peak influenza season of December through February.[39] This way winter sport athletes can be vaccinated in the early preseason period. If there are side effects, such as transient soreness at the injection site (25% to 50%) or fever and general-

ized muscle aches (less than 1%), the athletes will not lose valuable practice or playing time. There is still some misconception that influenza vaccine can cause influenza or Guillain-Barré syndrome. The influenza vaccine contains killed virus and cannot cause infection, and no neurologic complications have occurred with the viral strains used over the past 16 years.

It is a judgment call for the team physician to recommend immunization against influenza. Lack of immunization in this population is not a life and death issue. At worst, influenza may cause one or two missed games or disrupt an athlete's practice for a week; however, if you have a team that competes during the flu season, it is reasonable to offer the opportunity for vaccination to members of the team.

Measles

Measles vaccination is of special importance for young athletes. Besides the medical consequences for infected individuals, a measles outbreak can have deleterious effects on team sports. The incidence of measles has been increasing rapidly in the United States despite wide availability of an excellent attenuated vaccine. Measles have increased from a low of 1497 cases in 1983 to more than 27,786 in 1990.[2] An outbreak of measles followed the 1991 International Special Olympics in Minneapolis, and that same year in Indianapolis, during the World Gymnastics competition, three athletes from the New Zealand team were diagnosed with measles after mingling among most of the international delegations and local community volunteers and visitors. Fortunately, owing to the prompt and coordinated response by the competition officials and local health department, no major secondary outbreak occurred.[13] These two outbreaks associated with mass spectator events as well as multiple reports of sporadic outbreaks around the United States underscore the fact that indoor sporting events offer a prime environment for viral transmission.

Measles outbreaks related to sporting events are potentially serious because of the danger of transmission to susceptible persons in large groups gathered in a confined environment.[21] A packed, humid gym provides the classic conditions for respiratory transmission of this disease. As noted earlier, sports teams can transport the measles virus when they travel on the road, exposing their opponents as well as the local community spectators.

Team physicians can play an important role by promoting proper vaccination of their athletes. Individuals born after 1957 should receive live attenuated measles vaccine if they received measles vaccine before 15 months of age or if they received the less stable measles vaccine available before 1980. Essentially all young adults who have not had a booster since 1980 require revaccination to assure adequate immunity.

SUMMARY

The objective of this article has been to focus on the more common infectious diseases that the physician caring for athletes may encounter in the office practice of sports medicine. Clearly the physician plays a critical role in treatment and prevention through early recognition and intervention when appropriate. Fortunately, being a fairly young, healthy athletic population, these patients respond well to treatment. It is critical that physicians caring for athletes take an active role to educate and counsel their patients on appropriate preventive measures and give clear return to participation recommendations. Advice should be based on consideration of each athlete's unique situation as well as up-to-date knowledge concerning the particular infectious disease process involved and appropriate treatment options.

ACKNOWLEDGMENTS

The author wishes to thank Penny York for her clerical assistance and his wife, Diana K. Sevier, DDS, for her helpful advice and comments during the preparation of this manuscript.

References

1. Astrom E, Friman G, Pilstrom L: Effects of viral and mycoplasm infections on ultra structure and enzyme activities in human skeletal muscle. Acta Pathol Microbiol Scand [A]84:113, 1976
2. Atkinson WL, Hadler SC, Redd SB, et al: Measles surveillance—United States 1991. MMWR 41:SS-6, 1992
3. Bart B: Skin problems in athletics. Minnesota Med 66:239, 1983
4. Basler RS: Dermatologic aspects of sports participation. Curr Concepts Skin Dis 3:15, 1985
5. Basler RS: Skin lesions related to sports activity. Primary Care 10:479, 1983
6. Belongia EA, Goodman JL, Holland EJ, et al: An outbreak of herpes gladitorum at a high school wrestling camp. N Engl J Med 325:906, 1991

6a. Bergstresser PR, Elewski B, Hanifin J, et al: Topical terbinafine and clotrimazole in interdigital tinea pedis: A multicenter comparison of cure and relapse rates with 1- and 4-week treatment regimens. J Am Acad Dermatol 28:4, 1993

7. Berk LS, Nieman DC, Youngberg WS, et al: The effect of long endurance running on natural killer cells in marathoners. Med Sci Sports Exerc 22:207, 1990
8. Blair H, Greenberg S, Stevens P, et al: Effects of rhinovirus infection on pulmonary function of healthy human volunteers. Am Rev Respir Dis 114:95, 1976
9. Burch GE: Viral diseases of the heart. Acta Cardio 1:5, 1979
10. Burke AP, Farb A, Virmani R, et al: Sports-related and nonsports-related sudden cardiac death in young adults. Am Heart J 121:568, 1991
11. Casey JM, Dick EC: Acute respiratory infections. *In* Casey JM, Foster C, Hixson EG (eds): Winter Sports Medicine. Philadelphia, FA Davis, 1990, p 112
12. Cate T, Roberts J, Russ M, et al: Effects of common colds on pulmonary function. Am Rev Respir Dis 108:859, 1973
13. Centers for Disease Control: Measles at an international gymnastics competition—Indiana. MMWR 1992
14. Chadrasekar PH, Rolston KW, Kannangara DW, et al: Hot tub-associated dermatitis due to *Pseudomonas aeruginosa.* Arch Dermatol 120:1337, 1984
15. Cohen BA, Schmidt C: Tineal gladiatorum. N Engl J Med 327:820, 1992
16. Cohen S, Tyrrell DA, Smith AP: Psychological stress and susceptibility to the common cold. N Engl J Med 325:606, 1991

17. Coldiron B: Recalcitrant onychomycosis of the toenails successfully treated with fluconazole. Arch Dermatol 128:909, 1992
18. Collier A, Pimmel R, Hassellblad VC, et al: Spirometric changes in normal children with upper respiratory infections. Am Rev Respir Dis 117:47, 1978
19. Couch RB: The common cold: Control? J Infect Dis 150:167, 1984
20. Daniels WL, Sharp DS, Wright JE, et al: Effects of virus infection on physical performance in man. Milit Med 150:8, 1985
21. Davis RM, Whitman ED, Orenstein WA, et al: A persistent outbreak of measles despite appropriate prevention and control measures. Am J Epidemiol 126:438, 1987
22. Dick EC, Jennings LC, Mink KA, et al: Aerosol transmission of rhinovirus colds. J Infect Dis 156:442, 1987
23. Douglas RG, Lindgren KM, Cough RB: Exposure to cold environment and rhinovirus common cold. N Engl J Med 279:742, 1968
24. Eichner ER: Infection, immunity and exercise. Phys Sportsmed 21:125, 1993
25. Eichner ER: Neck check. Runner's World 27:16, 1992
26. Fitzgerald L: Exercise and the immune system. Immunol Today 9:337, 1988
27. Fitzgerald L: Overtraining increases the susceptibility to infection. Int J Sports Med 1:S5, 1991
28. Frelinger DP: The ruptured spleen in college athletes: A preliminary report. J Am Coll Health Assoc 26:217, 1978
29. Friman G: Effect of acute infectious disease on isometric muscle strength. Scand J Clin Lab Invest 37:303, 1977
30. Friman G, Wright JE, Ilback NG, et al: Does fever or myalgia indicate reduced physical performance capacity in viral infections? Acta Med Scand 217:353, 1985
31. Gatmanitan BG, Ghason JL, Lerner AM: Augmentation of the virulence of murine Coxsackie virus B3 myocardiopathy by exercise. J Exp Med 131:1121, 1970
32. Graham NMH, Douglas RM, Ryan P: Stress and acute respiratory infection. Am J Epidemiol 124:389, 1986
33. Gwaltney J, Moskalski P, Hendley J: Hand-to-hand transmission of rhinovirus colds. Ann Intern Med 88:463, 1978
34. Hallee TJ, Evans AS, Neiderman JC, et al: Infectious mononucleosis at the United States Military Academy. A prospective study of a single class over four years. Yale J Biol Med 3:182, 1974
35. Hanley DF: Medical care of the US Olympic team. JAMA 236:147, 1976
36. Heath WG, Ford ES, Craven TE: Exercise and the incidence of upper respiratory tract infections. Med Sci Sports Exerc 23:152, 1991
37. Hoagland RJ: Infectious mononucleosis. New York, Grune & Stratton, 1967
38. Inhorn SL, Dick EC: Coronaviruses. *In* Feigin RD, Cherry JD (eds): Textbook of Pediatric Infectious Diseases, ed 2. Philadelphia, WB Saunders, 1987, p 1531
39. Johnson CC: Do athletes need influenza vaccines? Phys Sportsmed 18:9, 1990
40. Jokl E, McClellan JT: Exercise and cardiac death. Baltimore, University Park Press, 1971
41. Kantor GR, Bergfeld WF: Common and uncommon dermatologic diseases related to sports activities. Exerc Sport Sci Rev 16:215, 1988
42. Kapikian AZ: Viral gastroenteritis. JAMA 269:627, 1993
43. Kapikian AZ, Chanock RM: Rotaviruses. *In* Fields BN, Knipe DM, Chanock RM, et al (eds): Virology, ed 2. New York, Raven Press, 1990, p 1353
44. Lau RC: Coxsackie B virus infections in New Zealand patients with cardiac and noncardiac diseases. J Med Virol 11:131, 1983
45. Levinson SO, Milzer A, Lewin P: Effect of fatigue chilling and mechanical trauma on resistance to experimental poliomyelitis. Am J Hyg 42:204, 1945
46. Mackinnon LT, Chick TW, van As A, et al: The effect of exercise on secretory and natural immunity. Adv Exp Med Biol 216A:869, 1987
47. Maki DG, Reich RM: Infectious mononucleosis in the athlete. Diagnosis, complications, and management. Am J Sports Med 10:162, 1982
48. Marano M: National Center for Health Statistics. Current estimates from the National Health Interview Survey United States, 1987 Vital and Health Statistics Series 10:166. Public Health Service. Washington, United States Government Printing Office, 1988
49. McKeag DB, Kinderknecht J: A basketball player with infectious mononucleosis. *In* Smith NJ (ed): Common Problems in Pediatric Sports Medicine. Chicago, Year Book Medical Publishers, 1989, p 191
50. Mier-Jedrzejowicz A, Brophy C, Green M: Respiratory muscle weakness during upper respiratory tract infections. Am Rev Respir Dis 138:5, 1988
51. Modlin JF: Coxsackie virus and echovirus. *In* Mandell GL, Douglas RG Jr, Bennett JE (eds): Principles and Practice of Infectious Diseases, ed 2. New York, John Wiley & Sons, 1985, p 814

52. Nehlsen-Cannarella SL, Nieman DC, Balk-Lamberton AJ, et al: The effects of moderate exercise training on immune response. Med Sci Sports Exerc 23:64, 1991
53. Nelson MA: Stopping the spread of herpes simplex. A focus on wrestlers. Phy Sportsmed 20:117, 1992
54. Niederman JC, Miller G, Pearson HA, et al: Infectious mononucleosis. Epstein-Barr virus shedding in saliva and the oropharynx. N Engl J Med 294:1355, 1984
55. Nieman DC: Exercise, immunity and respiratory infections. Sports Science Exchange 4:39, 1992
56. Nieman DC, Johanssen LM, Lee JW, et al: Infectious episodes in runners before and after a roadrace. J Sports Med Phys Fitness 29:289, 1989
57. Nieman DC, Johanssen LM, Lee JW, et al: Infectious episodes in runners before and after the Los Angeles Marathon. J Sports Med Phys Fitness 30:316, 1990
58. Nieman DC, Nehlsen-Cannarella SL: Effects of endurance exercise on immune response. *In* Shephart RJ, Astrand PO (eds): Endurance in Sport. Oxford, Blackwell Scientific Publications, 1991
59. Nieman DC, Nehlsen-Cannarella SL: The effects of acute and chronic exercise on immunoglobulins. Sports Med 11:183, 1991
60. Nieman DC, Nehlsen-Cannarella SL, Markoff PA, et al: The effects of moderate exercise training on natural killer cells and acute upper respiratory tract infections. Int J Sports Med 11:467, 1990
61. O'Connor S, Jones D, Collins J, et al: Changes in pulmonary function after naturally acquired respiratory infections in normal persons. Am Rev Respir Dis 120:1087, 1979
62. Reyes MP, Lerner AM: Interferon and neutralizing antibody in sera of exercised mice with Coxsackie virus B-3 myocarditis. Proc Soc Exp Bio Med 151:333, 1976
63. Roberts JA: Loss of form in young athletes due to viral infection. BMJ 290:357, 1985
64. Roberts JA: Viral Illnesses and sports performance. Sports Med 3:296, 1986
65. Rooney JF, Straus SE, Mannix ML, et al: Oral acyclovir to suppress frequently recurrent herpes labialis-A double-blind, placebo-controlled trial. Ann Intern Med 118:268, 1993
66. Ryan AJ, Dalrymple W, Dull B, et al: Round Table, Upper respiratory infections in sports medicine. Phys Sportsmed 3:29, 1975
67. Sawyer RN, Evans AS, Niederman JC, et al: Prospective studies of a group of Yale University freshman. Occurrence of infectious mononucleosis. J Infect Dis 123:263, 1971
68. Schelkun PH: Swimmer's ear: Getting patients back in the water. Phys Sportsmed 19:85, 1991
69. Schouten WJ, Verschuur R, Kemper HCG: Physical activity and upper respiratory tract infections in a normal population of young men and women: The Amsterdam growth and health study. Int J Sports Med 9:451, 1988
70. Sharp JCM: Viruses and the athlete. Br J Sports Med 23:47, 1989
71. Simon HB: Exercise and infection. Phys Sportsmed 15:135, 1987
72. Simon HB: Immunization and chemotherapy for viral infections. *In* Rubenstein E, Federman D (eds): Scientific American Medicine. New York, Scientific American, 7:33, 1990, p 2
73. Simon HB: The immunology of exercise: A brief review. JAMA 252:2735, 1984
74. Smith PG, Lucente FE: Infections. *In* Cummings CW, Fredrickson JM, Harker LA, et al: (eds): Otolaryngology–Head and Neck Surgery. St. Louis, CV Mosby, 1986, p 256
75. Smith WG: Adult heart disease due to Coxsackie virus group B. Br Heart J 28:204, 1966
76. Sosin DM, Gunn RA, Ford WL, et al: An outbreak of furunculosis among high school athletes. Am J Sports Med 17:828, 1989
77. Springer GL, Shapiro ED: Fresh water swimming as a risk factor for otitis externa: a case-control study. Arch Environ Health 40:202, 1985
78. Stauffer LW: Skin disorders in athletes: Identification and management. Phys Sportsmed 11:101, 1983
79. Strauss RH: Sports Medicine. Philadelphia, WB Saunders, 1991, p 109
80. The Office of Disease Prevention and Health Promotion, US Public Health Service: US Department of Health and Human Services, Disease Prevention/Health Promotion: The Facts. Palo Alto, Bull Publishing Co, 1988
81. Tineal pedis. Sports Med Dig 12:5, 1990

Address reprint requests to

Thomas L. Sevier, MD
Central Indiana Sports Medicine
100 North Tillotson Avenue
Muncie, IN 47304