Chapter 4

IMMERSION FOOT SYNDROMES

JOHN ADNOT, M.D.* AND CHARLES W. LEWIS, M.D.†

*Private Practice of Dermatology, Overton Park Building, 4200 S. Hulen Drive, Fort Worth, Texas 76109; formerly, Dermatology Service, Fitzsimons Army Medical Center, Aurora, Colorado 80045

†Private Practice of Dermatology, Dermatology Clinic, Regenstrief Health Center, 1050 Walnut Street, RG524, Indianapolis, Indiana 46202; formerly, Chief, Dermatology Service, Brooke Army Medical Center, Fort Sam Houston, Texas 78234

INTRODUCTION

Since the founding of this country, American soldiers have been fighting wars wearing a wide variety of shoes and boots to protect their feet from the environment. Soldiers of the Continental Army, 1775 to 1781, wore simple low-cut leather shoes with the rough side out and cloth leggings laced over the lower leg.[1] Joseph Lovell, a surgeon general in the 1800s, noted the importance of enabling soldiers to keep their feet warm and dry with wool socks and laced shoes reaching at least to the ankle. He also observed that letting the feet remain wet and cold for any length of time led to constitutional illnesses.[2]

In 1861, Union and Confederate soldiers wore any type of personally owned boot or shoe, but most used a simple, laced, ankle-high brogan. During the Civil War, Union troops were issued the first mass-produced shoes that distinguished between the left and right foot. Up to this time, most shoes were made to be worn on either foot. Many Confederate troops were barefoot or used canvas and wood to fashion crude walking shoes. Officers and mounted troops typically wore leather boots.[2]

Ankle-high, heavy leather shoes continued to be manufactured and were issued to soldiers during World War I. Wool wraps called puttees were wound around the lower leg from the knee to the ankle to protect the leg. It was not until the spring of 1918, however, that the Pershing boot, a heavier shoe with more waterproof construction, was developed.[3] It was effectively designed for the demands of trench warfare. After the war, modifications on footwear reflected garrison life and the need for economy.[3]

Ankle-high shoes and canvas leggings were initially worn in World War II. Because of material shortages and lack of preparedness for the footwear needs of wartime, despite the experience available from World War I, it was not until the end of 1943 that the first combat boot appeared.[3] It was a brown laced boot with a leather flap on the upper. Because leather is a permeable material, all leather boots leak to some extent. It was not until 1944 that the M-1944 Shoepac, with a moccasin-type rubber boot, was approved for distribution. It was the best available modification for the cold, wet conditions of trench warfare in Europe.[3] The first jungle boots made of canvas and rubber were used in the South Pacific.[3] A modified jungle boot, the tropical combat boot, was designed and tested during the latter part of World War II. It consisted of spun nylon, a leather midsole, and a full-length rubber outer sole. Production was not started until the summer of 1945.[3]

During the Korean conflict, a special brown leather jump boot that laced all the way up the front was issued to paratroopers and became popular throughout the army. Except for switching to a black color, these boots remained mostly unchanged through the Korean and Vietnam conflicts. In the mid-1960s a black leather and olive drab nylon-webbing jungle boot with a cleated sole became the favorite footwear of the American soldier.[4]

The type of footwear worn by the soldier in combat, along with environmental conditions and preventive hygiene measures, has played a crucial role in producing a variety of cutaneous disorders of the feet. These "disease, nonbattle injuries" range from minor inconveniences to very significant conditions that may result in hospitalization.

Based on methods of clinical diagnosis and laboratory confirmation of soldiers evacuated from war zones with inflammatory conditions of the feet, the following list was presented by Pillsbury and Livingood:

> At Fitzsimons General Hospital, after classification and appropriate studies, patients referred with this diagnosis were divided into the following categories and proportions:
>
> 1. The hyperhidrosis (dyshidrosis) syndrome, 51 percent.
> 2. Pyoderma secondary to trauma, maceration, or hyperhidrosis (dyshidrosis) syndrome, 14 percent.
> 3. Dermatophytosis, 20 percent.
> 4. Dermatitis venenata produced by medication (which had usually been prescribed for the treatment of the presumed fungal infections), 11 percent.
> 5. Other dermatitis venenata, 2 percent.
> 6. Resistant pustular eruptions (the so-called bacterid of Andrews), 1 percent.
> 7. Pustular psoriasis, 0.5 percent.
> 8. Acrodermatitis continua of Hallopeau, 0.5 percent.[5(p593)]

Controlled studies demonstrating effective methods for preventing foot diseases in military popula-

tions are rare. However, two studies are well documented:

> A convincing controlled study along these lines was conducted by Maj. (later Lt. Col.) Laurence Irving, Chief, Physiology Section, Headquarters, Eglin Field, Fla. Sandals were issued to approximately 1,000 men, who were permitted to wear them on the post as much as they wished; most of them practically gave up wearing shoes. A similar number of men wore shoes as usual. Within a month, the proportion of severe dermatophytoses in men wearing sandals fell from 30 to 3 percent, while in the control group, the disease remained as troublesome as usual.
>
> A similar study was conducted in New Guinea, while the 43d Infantry Division was in a rest area. Some 300 men with unclassified skin diseases, many of whom undoubtedly had dermatophytosis of the feet, were kept on the beach for 4 hours daily, without clothing or shoes. They bathed, exercised, or just lay in the sun as they wished. Within a month, the majority of infections had cleared without any other treatment.[5(pp602–603)]

During the conflict in Vietnam, one of the authors (CWL) convinced the Commander of the Second Brigade, First Infantry Division, to direct the purchase of 5,000 pairs of rubber shower thongs for use after combat operations in the rice paddies. By allowing soldiers to use these open rubber thongs upon return to base camp, and limiting the continuous wet exposure to not more than 72 hours, the rate of tropical immersion foot problems was generally kept to a level of 10% or less. Prior to institution of these policies, a combat unit could experience 70% to 75% loss of personnel due entirely to inflammatory skin diseases of feet that had been continuously wet more than 72 hours (Exhibits 4-1 through 4-3).

While it is often impossible to determine the exact role of diseases limited to the feet in overall effectiveness of a combat unit, rates of sick call and hospitalization always increased during periods of combat operations in the rainy seasons and decreased in dry seasons.[4] However, the fact that many combat units lost hundreds to thousands of man-days due to large numbers of individuals placed either on quarters or on light, noncombat status because of their skin diseases, was rarely included in statistical reports.

OVERVIEW

Injuries to the feet from prolonged immersion in water or contact with dampness, in a range of environmental temperatures, may be collectively referred to as "immersion foot syndromes." These syndromes have been referred to as trench foot, swamp foot, tropical jungle foot, paddy-field foot, jungle rot, sea boot foot, bridge foot, and foxhole foot. Although most common during wartime, they also appear with occupational and recreational activities.

Injuries related to simultaneous exposure to cold temperatures and a wet environment are subdivided into trench foot and immersion foot. Those involving warmer temperatures include tropical immersion foot and warm water immersion foot.

This review describes each condition to alleviate confusion over nomenclature and to aid in recognition and treatment (Table 4-1). To keep the focus narrow, the extremes of the temperature injury spectrum (ie, true frostbite and burns) are not specifically addressed, but referred to as necessary for clarification.

INJURIES IN COOL OR COLD CLIMATES

All four of the immersion foot syndromes discussed in the next two sections are characterized by pain that is continuous over several days to weeks. They may incapacitate large numbers of troops in a unit simultaneously because of common exposure to the harsh environment. Commanders may prevent these injuries by being aware of the hazards and preventing long-term exposure to the predisposing conditions. In this section the two syndromes resulting from cold, wet conditions will be discussed.

Trench Foot

"Trench foot" refers to injury resulting from prolonged exposure to wet conditions, *without* immersion, in cold weather. The term probably originated in World War I, when many men were confined to trenches in cold, damp weather for prolonged periods. The condition was recognized as a cause of considerable loss of manpower as far back as the Greek Campaigns[6] and the Napoleonic and Crimean

EXHIBIT 4-1

ARMY REGULATION 40-29: PREVENTION OF SKIN DISEASE AMONG TROOPS OPERATING IN INUNDATED AREAS

HEADQUARTERS
UNITED STATES ARMY VIETNAM
APO SAN FRANCISCO 96375

REGULATION NUMBER 40-29 — 10 January 1968

MEDICAL SERVICE
Prevention of Skin Disease Among Troops
Operating in Inundated Areas

1. PURPOSE: To outline policy and procedures for the prevention of disabling skin conditions, which may occur when troops are required to conduct field operations in flooded rice paddies and other inundated areas.
2. SCOPE: This regulation is applicable to all units assigned or attached to this command.
3. GENERAL: Fungus infection of the foot is probably the most common skin disease causing disability among troops in RVN. The common athlete's foot with involvement of the toe webs and soles of the feet does not occur frequently in Vietnam; or if it does, it is relatively mild. The most severely affected areas have been the top of the feet and legs under the boots, the groin, and the buttocks. The lesions often spread to produce bright red rings which may run together. Although not so common, immersion foot is also a potential hazard.
4. RESPONSIBILITIES: Commanders are responsible for implementing measures outlined below when, in their opinion and upon the advice of their surgeon, they are considered necessary and practical.
5. PREVENTIVE MEASURES:
   a. Limiting the duration of operations in watery terrain. The tactical situation permitting, a 48 hour limit (2 days and 2 nights) should be placed on operations involving continuous exposure to water. If this is not possible, casualties from fungus infection may be disabled for 2 or more days after a five-day operation.
   b. Proper care of the feet.
      (1) One of the most important measures is to insist that troops wear boots and socks only when necessary while in base camps. Shower clogs or thongs are recommended as substitutes.
      (2) During operations, commanders should have a few men at a time remove their footgear and allow their feet to dry as the tactical situation permits.
      (3) Dry socks should be included in resupply missions in the field whenever possible. Mesh socks are preferred.
   c. Exposure of the skin to the sun.
      (1) Where possible, exposure of as much of the body as possible to the sun for 30 minutes every day is recommended. To avoid sunburn, new arrivals should be gradually exposed for short periods of time until a protective tan develops.
      (2) In base camps, during daylight hours when mosquitoes are not a problem, troops should be allowed to wear abbreviated clothing such as shorts. This should be limited to those troops whose operations mission predisposes them to skin disease.
   d. Cleansing of the skin.
      (1) As soon as troops return from an operation, they should remove dirty clothing and shower immediately. It appears that showering in potable water will reduce the incidence of skin diseases. However, showers using nonpotable water are preferable to no showers.
      (2) The use of antibacterial (germicidal) soaps should be encouraged. Any of the nationally advertised brands are acceptable.
   e. Laundering field clothing. Field clothing should be washed in potable water. Quartermaster or modern contract laundries are preferred. Starching of field clothing reduces ventilation, and is not recommended for troops in active combat operations or other strenuous physical activities.
   f. Underclothing. Troops should be discouraged, but not prohibited, from wearing underclothes while on operations in the field. Underclothes reduce ventilation of the skin.

(AVHSU-PM)

FOR THE COMMANDER:

ROBERT C. TABER
Brigadier General, US Army
Chief of Staff

WILLIAM H. JAMES
Colonel, AGC
Adjutant General

This Regulation supersedes USARV Reg 40-20, 25 Jan 66

EXHIBIT 4-2

MEMORANDUM ON PREVENTION OF SKIN DISEASE IN NINTH INFANTRY DIVISION

DEPARTMENT OF THE ARMY
HEADQUARTERS 9th INFANTRY DIVISION
APO SAN FRANCISCO 96370

AVDE-MD 28 October 1968
SUBJECT: Prevention of Skin Disease SEE DISTRIBUTION

1. The maintenance of the health of a unit is a command responsibility. Tropical skin diseases are the most common cause of non-effectiveness within the 9th Infantry Division Area. Commanders have adequate medical personnel, effective medications and proven techniques to reduce this very serious problem
2. Diseases of the foot and boot area developed rapidly after 48 hours of continuous exposure to the rice paddies and swamps, and may affect 35% to 50% of the combat strength of an infantry unit after 72 hours. With each succeeding exposure, skin infections are more severe and require over three weeks of intensive treatments.
3. Consequently, commanders will limit operations to 48 hours in the paddy followed by a minimum of 24 hours utilization in a dry area. This limitation will be exceeded only in situations which override the inevitable loss of combat strength from skin disease.
4. Additionally, to reduce the non-effectiveness rate caused by skin disease, commanders will conduct foot inspections and require their men to put on dry socks daily. Men should remove their boots and socks whenever possible (up to four hours daily), to let their feet dry out. After an operation all personnel will be examined by medical personnel.

JULIAN J. EWELL
Major General, USA
Commanding

DISTRIBUTION:
A

EXHIBIT 4-3

MEMORANDUM ON PROPER FOOT CARE FOR SOLDIERS AT FORT GORDON, GEORGIA

DEPARTMENT OF THE ARMY
HEADQUARTERS U.S. ARMY SIGNAL CENTER AND FORT GORDON
FORT GORDON, GEORGIA 30805-5000

AZTH-CG 20 November 1990
MEMORANDUM FOR Commander, U.S. Army Training and Doctrine Command
ATTN: ATCD-SE (COL Charles Ball), Fort Monroe, Virginia 23651-5000
SUBJECT: Proper Foot Care for Soldiers at Fort Gordon

1. Every summer an unnecessarily large number of soldiers at Fort Gordon require treatment in the Dermatology Clinic for severe eczema of the feet, usually with secondary infection which results from excessive heat and humidity. It is aggravated by the wearing of wool winter socks and combat boots during periods of high heat and humidity. Soldiers suffering from this problem lose many hours of training and are restricted from physical training until the skin of the feet can heal. After a severe episode of foot eczema, the skin is easily broken down for many weeks following recovery and relapses are common.
2. Standard treatments for this condition include topical and internal medications. An integral part of treatment, however, is evaporation of perspiration through the wearing of adequately ventilated foot wear and cotton or cotton blend socks.
3. Therefore, recommend that OD cotton socks be added as an additional issue item primarily to those soldiers participating in basic and advanced individual training in the summer months where excessive heat and humid climate exists. With the addition of cotton socks, daily rotation of boots and normal foot care during the summer months, many cases of foot eczema can be prevented and excessive lost training time and physical training restrictions can be minimized.
4. Point of contact at Fort Gordon is Ms. Ree Hill, Chief, Supply Brand, Installation Supply and Services Division, Directorate of Installation Support, AUTOVON 780-5186/4507.

PETER A. KIND
Major General, USA
Commanding

JAMES E. HASTINGS
Brigadier General, MC
Director, Health Services

TABLE 4-1

COMPARISON OF IMMERSION FOOT SYNDROMES

Syndrome	Site	Symptoms	Signs	Systemic Involvement	Healing Time	Pathological Changes
Trench foot	Foot	Prehyperemic: Early: numbness, pain, paresthesia Late: anesthesia, "walking on blocks of wood" Hyperemic: tingling, progressing to throbbing, burning pain, and hyperesthesia; distal anesthesia may persist Posthyperemic: deep ache joint pain, prolonged anesthetic changes	Prehyperemic: pale, swollen, vesiculobullous lesions, distal cyanosis Hyperemic: increased edema, warm, dry, erythematous, bounding pulses, vesicles, bullae, ecchymosis Posthyperemic: Early: cool, moist, patchily or entirely cyanotic, normal to decreased pulses Late: skin and muscular atrophy, osteoporosis, deformity	None	Visible changes in 4 wk–6 mo; neurological and structural changes in months (may be permanent)	Prehyperemic: thrombosis, edema, vasoconstriction Hyperemic: thrombosis, capillary rupture, hemorrhage, vasodilation, edema, subepidermal vesiculation Posthyperemic: fibrin deposition in vessels and muscles, edema of nerve axons, variable lymphatic damage
Immersion foot	Usually foot, occasionally knee, thigh, or buttocks	Same as trench foot	Same as trench foot	None	Same as trench foot	Same as trench foot
Tropical immersion foot	Dorsum of foot, ankles	Early: burning pain (aggravated by pressure from footwear, walking, or both) and itching Late: paresthesia, hyperesthesia, anesthesia	Early: erythema, edema, macu-lopapular rash, vesicles or bullae, and tenderness, delineatedby top of boot Recovery: decreased fever, adenopathy, tenderness, and pain by 72 h; erythema and edema subside in 5–7 d followed by fine branny desquamation of all affected areas	Fever (38°C–39°C), femoral adenopathy	3–7 d	Parakeratosis, acanthosis, lymphocytic perivascular infiltrate, edema, telangiectasia, extravasation of erythrocytes
Warm water immersion foot	Plantar surfaces of feet	Pain on weight-bearing, tingling, "walking on rope" sensation	Early: swelling, wrinkling, and pallor of plantar surfaces Recovery: resolution of changes in 24 h; shedding of stratum corneum starts in 4–6 d, lasts 7–14 d; feet remain tender until new callus develops	None	1–3 d (symptoms); 7–14 d (fully functional)	Thickening of stratum corneum

Pathogenesis	Water Exposure	Water Temp.	Relation to Water Temp.	Treatment	Prophylaxis	Susceptibility Factors
Direct vascular injury by cold	2–14 d continuously wet (but not necessarily immersed)	15°C	Lower temperature hastens injury	Removal from wet environment, avoidance of weight-bearing, rewarming of body, elevation and cooling of feet, nutritious diet, asepsis, tetanus prophylaxis, prophylactic antibiotics, conservative surgical approach, avoidance of smoking	Individual education in first aid and recognition; frequent rotation out of wet, cold areas; maintenance of nutritional status; informed command elements	Dependency, immobility, trauma, anoxia, poor nutrition, improper warming
Same as trench foot	1 d or more of continuous immersion	15°C	Same as trench foot	Same as trench foot	Enclosed survival craft, individual protective suits	Same as trench foot
Passage of water through epidermis, with secondary subacute dermatitis	3–10 d of continuous immersion	22°C–32°C	None	Allowing feet to dry until asymptomatic	24 h of drying for each 48 h of exposure	Previous episodes may increase susceptibility to repeated episodes
Hyperhydration of stratum corneum	1–5 d of intermittent immersion	22°C–32°C	Increased temperature hastens injury	Allowing feet to dry until asymptomatic	Daily drying (overnight), silicone barrier greases	Thicker stratum corneum predisposes to injury

Wars.[7,8] Yet these lessons seem to have been lost on modern armies. In Europe during World War II, American forces sustained 11,000 cases of trench foot in November 1944 with more than 6,000 in the Third U.S. Army alone.[9]

Trench foot is nearly identical to gradual-onset frostbite, but the maximum temperature at which trench foot can occur has not been established. Ice crystals will not form in tissue above 0°C, but from 0°C to 10°C to 15°C clinical trench foot will develop if exposure lasts 48 hours or longer.[8] Other contributing factors include nutritional deficiency; trauma (rubbing or walking on affected feet); wind; improper clothing type and integrity; circulatory stagnation and tissue anoxia from dependency, inactivity, hemorrhage, or shock; and improper technique used to rewarm an injured limb.[7]

Clinically, trench foot is insidious in onset, the soldier first noting a cold sensation giving way to numbness. Paresthesia and pain may be noted with weight bearing.[8,10] With continued exposure, complete anesthesia to touch, pain, and temperature occurs: a feeling described as "walking on blocks of wood."[9]

The feet appear pale and swollen and may exhibit vesiculobullous lesions.[6–8] The degree of edema during this ischemic or prehyperemic stage depends on whether the feet are intermittently rewarmed during the course of exposure (which results in less edema).[9] The feet may appear mottled or purple, suggesting impending gangrene, yet such permanent damage is usually minimal with proper care (Figure 4-1).[9–10]

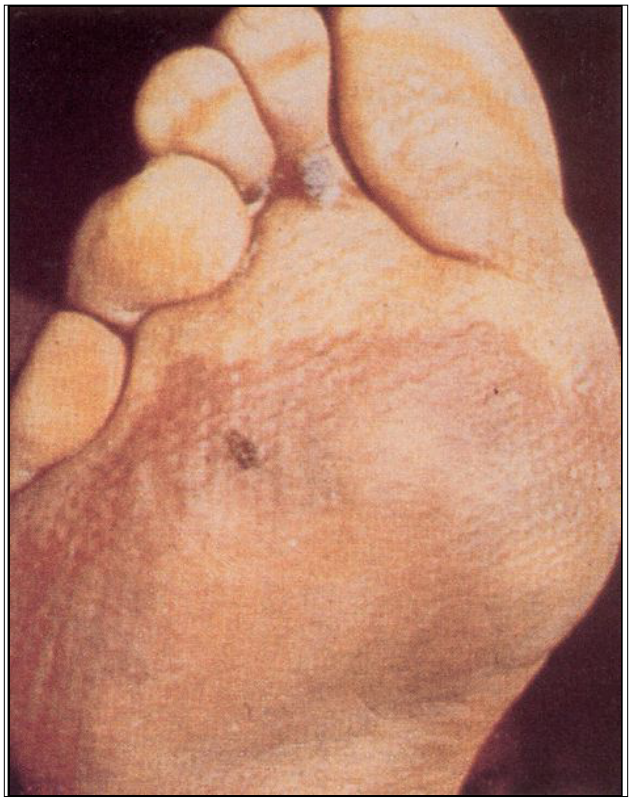

Fig. 4-1. Mild edema and a mottled appearance of the plantar aspect of the feet are characteristic of the early hyperemic stage of trench foot. Prolonged exposure to cold, damp conditions leads to prolonged incapacity. Photograph: Courtesy of David Corbett, CDR, Dermatology Branch, National Naval Medical Center, Bethesda, Md.

The hyperemic[6] or inflammatory[9] stage occurs several hours after removal of footgear and rewarming of the extremity. Sensation returns proximally to distally, first as a tingling sensation that rapidly progresses to an extreme burning, throbbing pain.[8–10] Warmth cannot be tolerated and soldiers become more comfortable with cooling of the extremity.[9] Hypesthesia replaces anesthesia except for the most distal areas, which may remain anesthetic for weeks or months. The feet rapidly swell and become warm, dry, and erythematous, with bounding pulses.[9,10]

In milder cases, this stage peaks at 24 hours. Severe cases, however, may progress for 48 to 96 hours and produce areas of blistering and circulatory compromise that are more likely to become gangrenous. Hemorrhage and ecchymosis may be present.[8,11]

Milder cases of trench foot subside slowly over 1 to 4 weeks and are frequently accompanied by a variably scarring exfoliation[6,9] of the affected areas. More severe cases progress to the posthyperemic stage. Although trench foot patients are susceptible to sepsis, the uncomplicated course of this injury has no systemic manifestations.

The posthyperemic or postinflammatory stage is prolonged. The previously hot, dry foot becomes cool, moist, mottled or entirely cyanotic, with pulses difficult to find. The extreme pain of the hyperemic stage subsides to a deep ache, noted especially distally and often associated with the smaller joints.[8,9] Hyperesthesia and paresthesia disappear rapidly, whereas anesthesia tends to remain for months or years.[6,9] Late changes in more severely affected soldiers may include atrophy of the skin,[6] osteoporosis,[9] and muscular atrophy and deformity (especially of the clawfoot type).[9,11]

Histologically, trench foot is a manifestation of injury to the microvasculature.[8,10] Peterson and Hugar[12] state that prolonged exposure to cold causes increased blood viscosity and sludging of red cells within the vessels. Combined with sympathetic vasoconstriction and loss of serum proteins through damaged endothelium, the result is thrombosis,

ischemia, and cell injury.

Thrombosed vessels of the dermis and subcutaneous tissue, reflex vascular dilation, capillary rupture, and increased vascular permeability all contribute to the edema, vesiculation, and ecchymoses of the hyperemic stage.[8–10] The work of Smith et al,[8] in which trench foot was duplicated in rabbits, also showed fibrin deposition in vascular walls and muscle bundles, edema and neutrophilic infiltration of dermal collagen and muscles, edema of nerve axons, and vacuolization of muscular fibers of vascular endothelium. Smith and coworkers observed variable damage to lymphatic tissue.

Biopsies of tissue in the posthyperemic stage have demonstrated atrophy and thinning of the dermis, fibrosis and collagen deposition around nerve endings and blood vessels, and replacement of muscle bundles and fibrils by scar tissue.[6]

Immersion Foot

Immersion foot can be considered the sailor's counterpart of the soldier's trench foot. The term "immersion foot" was first used during World War II to describe a syndrome of clinical conditions occurring in extremities exposed to prolonged, continued immersion in water of temperature ranging from above freezing to 15°C. Seen most dramatically during World War II, immersion foot typically develops in shipwrecked persons who are adrift either in water or in lifeboats partially filled with water.[6,7,9,10] It also was reported in Vietnam, the result of prolonged immersion in rice paddies.[11]

Clinically, soldiers with immersion foot show the same prehyperemic, hyperemic, and posthyperemic stages as do those affected with classic trench foot.[6,9–11] In immersion foot, however, the injury may extend more proximally to include the knees, thighs, and buttocks, depending on the depth of immersion.[6] Also, because of the continuous exposure, immersion foot may begin the first day, whereas trench foot usually begins after several days of lesser and, perhaps, intermittent exposure. The histopathological findings seen in immersion foot are similar to those of trench foot.

Management

Treatment of nonfreezing injuries such as trench foot and immersion foot is based on reversing the ischemia while not aggravating the edema, red cell extravasation, or inflammation of the hyperemic stage. With rewarming, damaged tissue cells have a greatly increased need for effective blood flow to remove products of necrosis. As this reflex vasodilation occurs, previous thrombosis and direct injury to endothelial cells by cold and anoxia cause a massive transudation of plasma and red blood cells, which leads to variable degrees of edema, vesiculation, and hemorrhage.[6]

To reduce metabolic demand and reflex vasodilation, the healthcare provider must raise the core temperature of the body while keeping the affected extremities cool.[6,9,10] Elevating the patient's uncovered feet in a stream of cool air from a fan while keeping the remainder of the body warm and well nourished usually achieves this goal. Patients notice a decrease in pain, and edema, hyperemia, and vesiculation subside.[6] Cooling of the extremities continues until the hyperemic stage has subsided and circulation is reestablished. The practice of rubbing the affected extremity with snow or ice further injures already compromised tissue and has no place in modern therapy.[6,8–10]

Other general measures include avoidance of weight bearing and direct trauma, aseptic precautions, prophylactic antibiotics, avoidance of smoking, tetanus prophylaxis, analgesics, a nutritious high-protein diet, and possible plasma transfusion as indicated. Surgical treatment should be delayed as long as possible to allow natural demarcation of tissue loss, and amputation should be correspondingly conservative.[6,9,10]

Other forms of therapy suggested for frostbite have not been specifically investigated for nonfreezing injuries and are *not* recommended. These treatments include rapid rewarming, low molecular weight dextran, sympathetic blockade, ultrasound, continuous epidural anesthesia, anticoagulation, and regional sympathectomy.[12–15]

Treatment of the posthyperemic stage is mostly symptomatic, utilizing physiotherapy, exercise, and surgical correction of deformity.[9] Early sympathectomy in more severe cases may prevent late sequelae such as fibrosis, contractures, and scarring,[6] but such intervention awaits further study.

Prevention of trench foot and immersion injury is difficult, especially in wartime circumstances. Of primary importance is the proper choice, use, and care of protective footgear. Individual education in first aid and recognition of impending injury, attention to personal hygiene, frequent rotation out of wet and cold areas, maintenance of nutritional status and morale, and informed command personnel are all necessary to prevent trench foot. Immersion foot may be prevented by the use of enclosed survival craft and by the availability of cold water protective suits for individuals on ships at sea.

INJURIES IN WARMER CLIMATES

As with trench foot and immersion foot, pain and disability characterize the following two preventable warm water syndromes. While the healing time is shorter for the warm water syndromes—several days to 2 weeks as compared with several weeks to months in cooler climates—the impact on fighting strength is obviously dramatic. Prevention by responsible policies and adherence to them by the commander are of paramount importance to the accomplishment of the unit's mission.

Tropical Immersion Foot

Investigators first referred to what they felt was a variant of classic immersion foot occurring in a considerably warmer environment in troops fighting in the Philippines during World War II.[9] A similar hot, wet environment experienced by ground forces in Vietnam was recognized as the cause of many foot casualties. Such casualties frequently resulted in greater loss of combat unit strength than did all other medical causes combined and often were instrumental in limiting the duration of field operations.[16]

Tropical immersion foot, commonly known as "paddy foot," occurs after continuous or near-continuous immersion of the foot in water or mud of temperatures above 22°C for periods ranging from 2 to 10 days.[9,16–18] The first symptoms include burning[16] and itching[18] sensations on the dorsum of the foot. With continued exposure, walking becomes progressively more difficult.[9,18]

When footgear is removed, the foot is edematous (Figures 4-2 and 4-3). Usually, the shoes cannot be replaced.[9,16] The feet may initially appear pale,[9] but they rapidly become intensely erythematous in a distribution sharply demarcated at shoe- or boot-top level (Figures 4-4 and 4-5). This erythema affects the dorsum of the foot but not the plantar surfaces—an important differentiating point from warm water immersion foot.[9,16–19] Papules, vesicles, or both may appear, sometimes with a hemorrhagic component.[18–20]

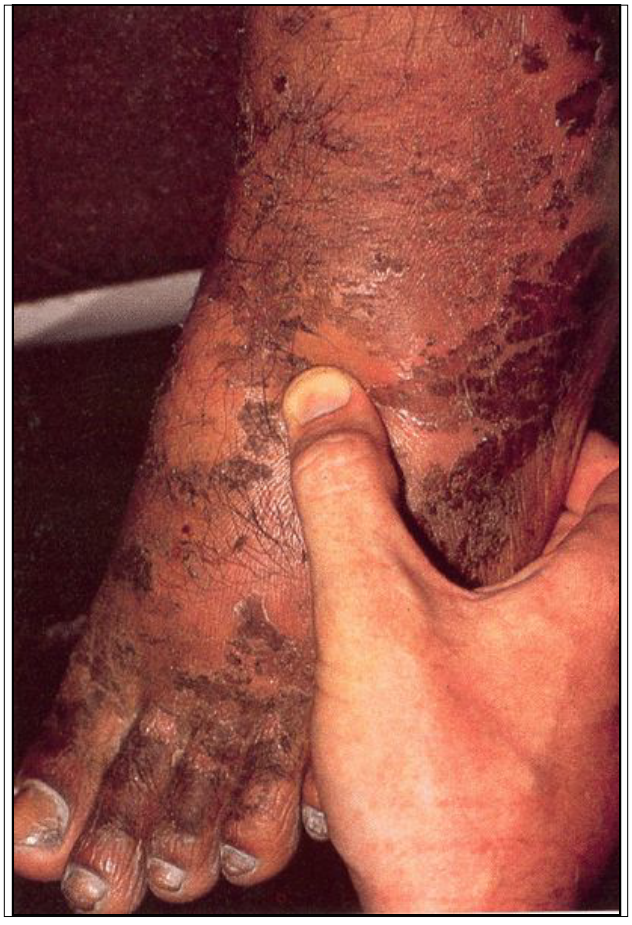

Fig. 4-2. Physical examination of this soldier whose feet had been continuously immersed in the warm water of a rice paddy for several days reveals tropical immersion foot with striking edema. Photograph: Courtesy of David Taplin, PhD, Dermatology Department, University of Miami School of Medicine, Miami, Fla.

Fig. 4-3. The erythema of tropical immersion foot (shown here in an early stage) affects the dorsal surface of the foot. Warm water immersion foot, which results from intermittent rather than continuous exposure to warm, wet conditions, affects only the soles. Photograph: Courtesy of David Taplin, PhD, Dermatology Department, University of Miami School of Medicine, Miami, Fla.

Fig. 4-4. On close examination of a patient with early tropical immersion foot, erythema, peeling, and fissures are present. Skin changes such as these are often accompanied by adenopathy and fever. Photograph: Courtesy of David Taplin, PhD, Dermatology Department, University of Miami School of Medicine, Miami, Fla.

Fig. 4-5. In tropical immersion foot, the erythema extends up the leg to a point of sharp demarcation at boot-top level. Photograph: Courtesy of David Taplin, PhD, Dermatology Department, University of Miami School of Medicine, Miami, Fla.

Although tenderness and pain (especially on weight bearing) are often prominent in tropical immersion foot, hyperesthesia, paresthesia, and anesthesia are more common. Most notable are the systemic reactions. Severely affected soldiers have tender unilateral or bilateral femoral adenopathy and a fever of 38°C to 39°C.[9,16–18]

No definite predisposing factors are known, but physicians and commanders estimate that severe tropical immersion foot develops within 4 days in 3% to 5% of exposed individuals. These individuals seem predisposed to repeat injury.[16] In about 80% of those exposed, some degree of the disorder develops after 10 to 12 days.[18]

Histologically, tropical immersion foot shows epidermal parakeratosis and acanthosis,[20] dermal edema and telangiectasia, and a lymphocytic perivascular infiltrate with associated extravasation of red blood cells.[16,18–20] Willis,[21] in an experiment exposing the backs and arms of volunteers to controlled continuous water contact, achieved similar histological changes. He postulated that such changes are caused by loss of barrier function of the swollen stratum corneum, with secondary irritation or damage to the underlying tissues.

Management of tropical immersion foot consists of bed rest, elevating and drying the feet, analgesics if necessary, and antibiotics if indicated. Usually, fever and adenopathy resolve within 72 hours, and the erythema and edema subside in 5 to 7 days, followed by a fine branny desquamation resulting in normal-appearing feet.[16,18] Even the most severe cases usually resolve within 2 weeks without sequelae such as gangrene, persistent sensory changes, or orthopedic disability.

Prevention is easily accomplished if a 24-hour

drying-out period is alternated with each 48 hours of water exposure.[16,18] In a military setting, the commander's attention to this matter is critical. Although rapid-drying boots and socks may delay the onset of tropical immersion foot,[16] silicone greases used as a barrier ointment have not proved effective.[20] Since persons affected with the disorder seem predisposed to reinjury,[16,19] special attention to preventive measures is indicated for these individuals.

Warm Water Immersion Foot

Although warm water immersion foot may seem the most innocuous of the immersion foot syndromes, it can incapacitate an individual for 3 to 14 days. This condition occurred in many service members in Vietnam when they were subjected to variable periods of intermittent exposure to wet, warm conditions. More recently, warm water immersion foot has been noted in persons wearing insulated boots, without water exposure, presumably from the buildup of perspiration—the so-called "moon-boot syndrome."[22,23]

After 1 to 3 days of exposure, affected individuals begin to note pain on weight bearing, tingling, and a sensation described as "walking on rope."[16] When footwear is removed, the soles of the feet are thickened, severely wrinkled, and macerated (Figure 4-6).[16,17,24,25] Although these changes may extend to the sides of the foot, they do not affect the dorsum.

Warm water immersion foot appears to develop faster at higher water temperatures.[13] Persons with thicker, callused soles tend to experience more severe (although not earlier) symptoms.[16,17,24,26] Microscopically, hyperhydration of the stratum corneum is the only finding.[27]

Treatment consists of bed rest and drying the feet.[16–18] The wrinkles and maceration resolve within 24 hours, but tenderness may last 2 to 3 days. The patient is asymptomatic by the third day. Shortly thereafter, however, thick portions of the sole begin to fissure and peel, shedding completely within 1 to 2 weeks. During this peeling, the stratum corneum may be more susceptible to infection via the fissures,[17] and patients experience tenderness on walking as new calluses develop.[18]

Prophylaxis consists of drying the feet for 6 to 8 hours (overnight) of every 24 hours.[16,17,28] Silicone grease applied to the entire foot[24,25,28] or to the soles alone[15] retards the development of warm water immersion foot. Footgear with adequate drainage and composed of rapidly drying materials may also slow the development of this condition.

a

b

Fig. 4-6. (**a**) Warm water immersion foot is the mildest of the immersion foot syndromes; however, it can incapacitate soldiers for 3 to 14 days. The clinical appearance results from hyperhydration of the stratum corneum. (**b**) Closer view of characteristic thickened, wrinkled skin. Photographs: Courtesy of David Taplin, PhD, Dermatology Department, University of Miami School of Medicine, Miami, Fla.

SUMMARY

As is the case for most cutaneous diseases seen in soldiers, the counterpart of immersion foot exists in the civilian community. Immersion foot problems in homeless individuals have recently been reported following continuous exposure to a damp environment over a period of days to weeks.[29] Ski instructors have also been reported to develop this syndrome.[11]

Military medical history continues to teach recurring critical lessons; the kinds and amounts of skin disease occurring in soldiers can be predicted on the basis of knowledge of such factors as climate, terrain, and environmental conditions. In order to significantly decrease the impact of skin disease on combat effectiveness, military dermatologists should be integrated at the division level, so that command policies and tactical considerations can incorporate these medical matters and ensure logistical support for the successful outcome of military operations.

In wartime, a soldier who becomes a "foot casualty" is as useless to his commander as one who sustains a bullet wound. It is up to the soldier-physician to advise commanders appropriately on the prevention of these environmental injuries. A familiarity with the clinical and pathophysiological aspects of immersion foot syndromes also enables the physician to render appropriate care, which in turn may prevent or ameliorate long-term disability.

(The sections "Injuries in Cool or Cold Climates" and "Injuries in Warmer Climates" and Table 4-1 are reprinted from: Adnot J, Lewis CW. Immersion Foot Syndromes. J Assoc Mil Derm. *1985;11(1):87–92.)*

REFERENCES

1. Gillett MC. *The Army Medical Department, 1775–1818*. Washington, DC: Medical Department, US Army, Office of The Surgeon General, and Center of Military History; 1981: 192.

2. Brown HE. *The Department of the United States Army from 1775 to 1873*. Washington, DC: The Surgeon General's Office. 1873: 104.

3. Risch E. *The Quartermaster Corps: Organization, Supply, and Services*. Vol 1. Washington, DC: Office of the Chief of Military History, Department of the Army; 1953: 102–103, 108, 110.

4. Allen AM. *Skin Diseases in Vietnam, 1965–72*. In: Ognibene AJ, ed. *Internal Medicine in Vietnam*. Vol 1. Washington, DC: Medical Department, US Army, Office of The Surgeon General, and Center of Military History; 1977: 7, 81.

5. Pillsbury DM, Livingood CS. Dermatology. In: *Infectious Diseases and General Medicine*. Vol 3. In: Havens WP, Anderson RS, eds. *Internal Medicine in World War II*. Washington, DC: Medical Department, US Army, Office of The Surgeon General; 1968.

6. White JC, Scoville WB. Trench foot and immersion foot. *N Engl J Med.* 1945;232:415–422.

7. Green, R. Frostbite and kindred ills. *Lancet*. 1941;2:689–693.

8. Smith JL, Ritchie J, Dawson J. On the pathology of trench frostbite. *Lancet*. 1915;2:595–598.

9. Whayne TF, DeBakey ME. *Cold Injury, Ground Type, in World War II*. Washington, DC: Office of The Surgeon General, Department of the Army, 1958.

10. Webster DR, Woolhouse FM, Johnston JL. Immersion foot. *J Bone Joint Surg Br.* 1943;24:785–794.

11. Gold RH. A review of immersion foot. *J Am Podiatry Assoc.* 1966;56:414–415.

12. Peterson G, Hugar DW. Frostbite: Its diagnosis and treatment. *J Foot Surg.* 1979;18:32–35.

13. Bouwman DL, Morrison S, Lucas CE, et al. Early sympathetic blockade for frostbite—Is it of value? *J Trauma.* 1980;20:744–749.

14. Weatherly-White RCA, Sjostrom B, Paton BC. Experimental studies in cold injury. Part 2: The pathogenesis of frostbite. *J Surg Res.* 1964;4:17–22.

15. Weatherly-White RCA, Paton BC, Sjostrom B. Experimental studies in cold injury. Part 3: Observation on the treatment of frostbite. *Plast Reconstr Surg.* 1965;36:10–18.

16. Allen AM, Taplin D. Tropical immersion foot. *Lancet.* 1973;2:1185–1189.

17. Samitz, M.H. Immersion injuries. In: *Cutaneous Disorders of the Lower Extremities*. 2nd ed. Philadelphia: JB Lippincott; 1981: 179–183.

18. Akers WA. Paddy foot: A warm water immersion foot syndrome variant. *Milit Med.* 1974;139:605–618.

19. Allen AM. Taplin D, Lowy JA, et al. Skin infections in Vietnam. *Milit Med.* 1972;137:295–301.

20. Douglas JS, Eby CS. Silicone for immersion foot prophylaxis: Where and how much to use. *Milit Med.* 1973;137:386–387.

21. Willis I. The effects of prolonged water exposure on human skin. *J Invest Dermatol.* 1973;60:166–171.

22. Blogg H. Moon-boot foot syndrome. *Br Med J.* 1982;285:1774–1775.

23. Philipp R. Moon-boot foot syndrome. *Br Med J*. 1983;286:562.

24. Taplin D, Zaias N. Tropical immersion foot syndrome. *Milit Med.* 1966;131:814–818.

25. Rietschel RL, Allen AM. Immersion foot: A method for studying the effects of protracted water exposure on human skin. *Milit Med.* 1976;141:778–780.

26. Taplin D, Zaias N, Blank H. The role of temperature in tropical immersion foot syndrome. *JAMA.* 1967;202:546–549.

27. Gill KA. *Naval Medical Field Research Laboratory Report: Field study on silicone ointments MDX-4-4056 and MDX-4-4078*. Camp Lejeune, NC: Department of the Navy; 1967: 1–7.

28. Buckles LJ, Gill KA, Gustave TA. Prophylaxis of warm-water immersion foot. *JAMA.* 1967;200:681–683.

29. Wrenn K. Immersion foot: A problem of the homeless in the 1990s. *Arch Intern Med.* 1991;151:785–788.