Chapter 3

SKIN DISEASES ASSOCIATED WITH EXCESSIVE HEAT, HUMIDITY, AND SUNLIGHT

LEONARD SPERLING, M.D.*

Lieutenant Colonel, Medical Corps, U.S. Army; Dermatology Service, Walter Reed Army Medical Center, Washington, D.C. 20307-5001

INTRODUCTION

Whenever the armed forces of the United States have first entered extremely hot climates, a large number of heat-related casualties have been encountered.[1] In all 20th-century wars, however, soldiers have quickly adjusted and were able to function successfully. Operations Desert Shield and Desert Storm (1990–1991) were no exceptions. In the Saudi Arabian desert, troops relearned the lessons of North Africa and the Pacific islands of World War II, where the casualties included numerous heat-related deaths. The same lessons were relearned during the summer months in Korea and in the jungles of Vietnam.

Heat injury is generally defined as a fairly acute physiological stress manifested by such conditions as heat cramps, heat exhaustion, and heatstroke.[2] Heat also has many deleterious effects on the skin, and several disorders are caused by, or are severely exacerbated by, high ambient temperatures. This is particularly important because the skin is largely responsible for thermoregulation in humans. Skin acts as a radiator and, in concert with fluids and electrolytes, can allow soldiers to withstand remarkably hot climates for indefinite periods. Certain skin diseases impair this critical thermoregulatory function, predisposing the affected soldier to the various types of acute heat injury.

The various skin diseases related to heat have another important impact: their disabling effects on troops. Skin diseases accounted for over 12% of total outpatient visits during the Vietnam conflict.[3] The three most common ailments in this group were miliaria, pyoderma (bacterial infections of the skin), and tinea (fungal infections of the skin), all of which are intimately related to heat and humidity. Over one half the patients who were evacuated from Vietnam for dermatological ailments suffered from tropical acne, eczematous dermatitis, dyshidrosis, and fungal infections.[3] The tropical environment of Southeast Asia clearly played an important role in the pathogenesis of all of these conditions.

Warm climates present another danger to troops: sun exposure. A variety of dermatoses are caused or severely exacerbated by solar radiation. Tropical climates are notorious for their long hours of intense sunshine. Most desert climates share this abundance of radiation, with an additional ingredient—lack of shelter from the sun.

Although problems due to heat will be discussed separately from those due to sunlight (visible and ultraviolet [UV]), in many cases these factors are related. Perspiring soldiers are apt to remove as much clothing as possible, thus predisposing themselves to burns from UV light. The injury of sunburn can impair the sweating mechanism, and by so doing impair the thermoregulatory mechanisms that allow soldiers to function in the heat.

CLASSIC FORMS OF HEAT INJURY

A discussion of heat-related dermatoses would not be complete without a review of heat injury in general. The subject is covered in detail in Technical Bulletin MED 507, *Prevention, Treatment and Control of Heat Injury*.[2] The resting human eliminates about one fourth of the basal metabolic heat production via the lungs and skin.[2] If body temperature starts to increase above its normal set point, the first physiological responses are vasodilation of the skin and an increase in the heart rate. These allow the skin to function more efficiently as a radiator of excess heat. When the body no longer can lose sufficient heat by simple radiation, and the skin temperature reaches or exceeds a critical level, the activity of the sweat glands increases. *The heat loss that can be achieved by sweating becomes the most important, and sometimes the only, means of maintaining a normal core body temperature*.[2] As environmental humidity increases, the ability of the air to evaporate the sweat decreases, thus reducing cooling. Inadequate cooling results in continued sweating, which results in a loss of body water and salt. Initially, the lost water is drawn from the circulating blood volume. If this fluid is not replaced, the resulting dehydration adds strain to the circulatory system, which has already resorted to an increased heart rate in its efforts to radiate heat from the skin. Hyperventilation (panting) will increase heat loss from the lungs, but creates a respiratory alkalosis that contributes to the signs and symptoms of heat-exhaustion syndromes.

Air motion (convection), in the forms of natural breezes and artificial wind produced by fans, is an important factor in cooling. It replaces the layer of

air next to the skin with cooler and dryer air, improving heat loss by conduction and accelerating the evaporation of sweat. The efficacy of air motion diminishes progressively as humidity and air temperature increase. If air temperature becomes higher than skin temperature, heat transfer by conduction will proceed from the air into the skin.

Other factors in the production of heat injury include the physical work being performed and the physical condition of the individual. Intense work obviously increases body heat and the burden on homeostatic mechanisms such as sweating and heart rate. Lack of acclimatization, obesity, dehydration, excessive intake of alcohol, lack of sleep, increasing age, poor general health, and fatigue of the homeostatic mechanisms over time all decrease a person's ability to withstand high temperatures. The following discussions of heat cramps, heat exhaustion, and heatstroke summarize the various forms of heat injury.[2]

Heat Cramps

Heat cramps result primarily from the excessive loss of salt relative to water from the body. The muscles of the extremities and the abdominal wall are subject to these painful cramps. Body temperature remains normal unless heat exhaustion has set in. Treatment is simply the intravenous administration of normal (0.9%) saline solution in adequate quantities.

Heat Exhaustion

Heat exhaustion results from peripheral vascular collapse due to excessive salt depletion and dehydration. It is characterized by profuse sweating, headache, tingling in the extremities, pallor, shortness of breath, palpitations, and gastrointestinal symptoms. Neuromuscular disturbance (incoordination) and cerebral dysfunction (clouded sensorium) may be present in varying degrees. Rectal temperature is often elevated. The signs and symptoms are basically those of shock; normal saline must be administered, and the soldier should rest. Any physical factor that promotes the return of blood to the heart is indicated, as is true for other forms of shock.

Heatstroke

Heatstroke is a medical emergency with a high mortality rate. It results when the normal thermoregulatory mechanisms become nonfunctional, and the main source of heat loss (sweating) is blocked. Heat exhaustion can act as a prodrome, but the onset of heat shock is abrupt, with loss of consciousness, seizures, or delirium. Core body temperature rises rapidly to 106°F to 108°F or higher. Signs and symptoms of obvious shock are evident, and if elevated body temperatures persist, organ failure (eg, kidney, brain, or heart) occurs. The first goals of treatment are to lower the body temperature and simultaneously to replace fluids and electrolytes.

These forms of heat injury become less likely with proper acclimatization. If troops are exposed to a hot climate and gradual incremental physical activity over a 2-week period, substantial tolerance to the ill effects of heat can be acquired. This process includes improvement in cardiovascular performance, sweating, and probably other factors as well. Even though acclimatization to a hot, dry, desert climate is an effective way to prepare for hot, humid, tropical climates, adjustment to tropical conditions still requires several weeks of increasing exercise at the actual site to be fully effective.

DERMATOSES CAUSED BY EXCESSIVE ENVIRONMENTAL HEAT

Overexposure to environmental heat can cause a number of skin disorders including miliaria, hypohidrosis syndrome, tropical acne, and cholinergic urticaria. The pathogenesis and treatment of these disorders are not militarily unique but are among the most prevalent among soldiers newly introduced to hot climates.

Miliaria

Miliaria is probably the simplest and purest example of a skin disease attributable almost entirely to the effects of heat and humidity.[4] Miliaria crystallina (sudamina), miliaria rubra, miliaria profunda, and miliaria pustulosa are clinical subtypes.[5] This disorder of sweating was one of the most common diseases affecting soldiers stationed in tropical climates during World War II.[6] Not only was miliaria a very common problem in areas such as the Southwest Pacific and the China-Burma-India theater, it was also common within warmer regions of the United States. Sixty-six percent of the naval personnel studied on Guam in 1945 exhibited miliaria rubra at some time during their first 7

months of duty.[7] In Vietnam, miliaria was very common but was almost never a direct cause of disability in troops. However, some physicians thought that it predisposed soldiers to fatigue and heat exhaustion. Miliaria was a major cause of outpatient visits and referrals to dermatologists.[3]

In Vietnam, where the ambient temperature had marked seasonal changes, the incidence of clinically significant miliaria in soldiers rose sharply after the temperature exceeded 38°C. Cooks were especially vulnerable to the disorder because they worked in small, poorly ventilated rooms where both temperature and humidity were especially high. Some soldiers in Vietnam were clearly more susceptible to the condition than others, an observation that was never explained; the condition occurred among those who were heat-acclimatized as well as among those who were not.[3]

Miliaria Crystallina

Miliaria crystallina is characterized by clear, superficial vesicles without any evidence of inflammation (Figure 3-1). It is typically asymptomatic and short-lived. No treatment is necessary, and the condition usually resolves spontaneously in a few hours to a few days.

Miliaria Rubra

The most common form of miliaria is miliaria rubra, more commonly known as prickly heat.

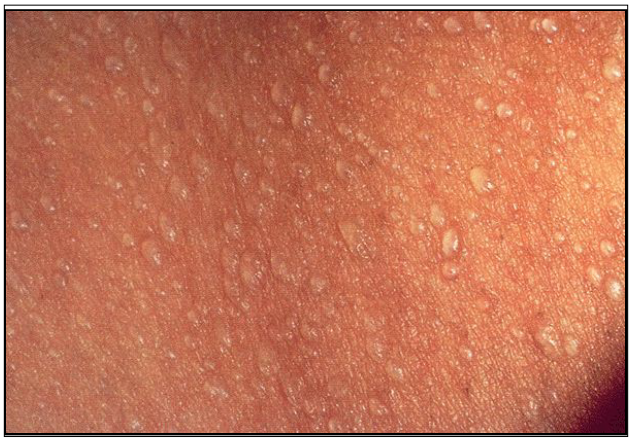

Fig. 3-1. Miliaria crystallina exhibits clear, superficial vesicles with no signs of inflammation. It is usually asymptomatic and short-lived, and no treatment is needed.

Miliaria rubra usually begins in the flexural folds and intertriginous parts of the body. It appears as a deeply erythematous, follicular, papulovesicular eruption. Typically it flares during the day and subsides to some extent at night. Untreated, the rash often becomes increasingly widespread and severe, and eventually persists even during the night. Its character also changes with time, becoming more inflammatory and fixed. If perspiration is excessive, the erythematous papules become capped by small, firm vesicles.[6] Miliaria rubra causes considerable discomfort but is seldom disabling. The condition does not seem to be influenced by race, obesity, complexion, place of former habitat, gender, or tanning.[7]

Miliaria Profunda

A more serious form of miliaria—miliaria profunda—was seen much less frequently than miliaria rubra during the Vietnam conflict. It typically develops from severe and prolonged miliaria rubra. Patients with miliaria profunda present with noninflamed nodules on the trunk and extremities; in most cases, these nodules are nonpruritic and not readily apparent on examination. The disease can be truly debilitating and is the cause of hypohidrosis syndrome (discussed below), also called tropical anhidrotic asthenia, a potentially fatal disorder.

Miliaria Pustulosa

Miliaria pustulosa is always preceded by some other dermatitis that has produced injury, destruction, or blocking of the sweat ducts.[5] Examples of predisposing dermatoses include contact and atopic dermatitis and lichen simplex chronicus. The pustules are discrete, superficial, sterile, and are not associated with the hair follicle; lesions can thus be distinguished from a bacterial folliculitis (Figure 3-2). These pruritic lesions occur most frequently on the intertriginous areas, on the flexure surfaces of the extremities, and on the scrotum.

Pathogenesis and Treatment

The pathogenesis and treatment of miliaria, and the anhidrosis that is its sequela, have been subjects of considerable investigation and debate.[4,8–10] One researcher found that miliaria could be reliably reproduced by applying occlusive plastic film to the skin.[9] After 48 to 72 hours of such occlusion, clinically and histologically typical miliaria was produced. When more than 30% of the total skin

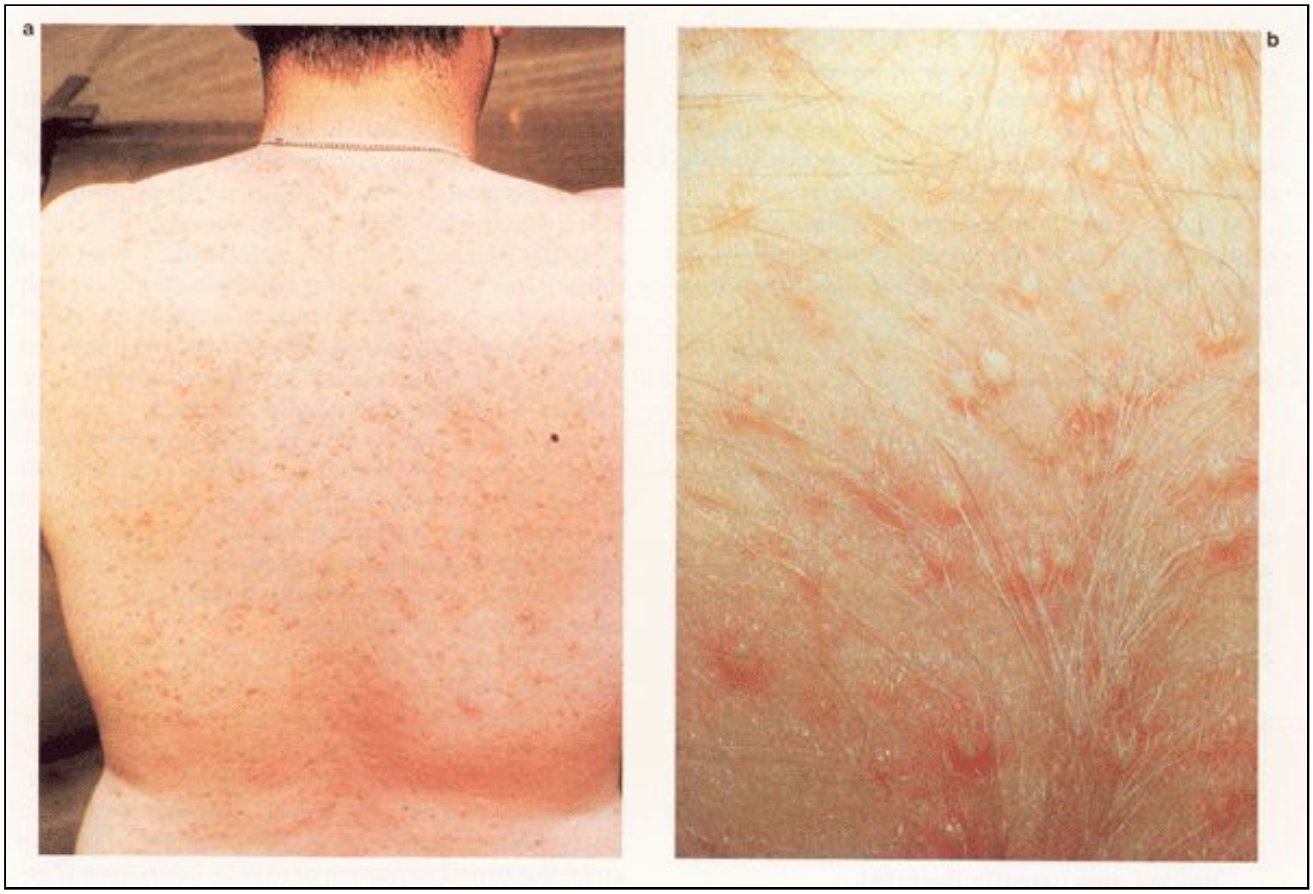

Fig. 3-2. (**a**) These discrete, superficial pustules are typical of miliaria pustulosa. Unlike bacterial folliculitis, the lesions are sterile and are not associated with a hair follicle. (**b**) Miliaria pustulosa—magnified view of sterile, pustular lesions.

surface was wrapped and occluded, the ensuing widespread hypohidrosis produced an increased susceptibility to heat-retention disease.[4] This susceptibility persisted even when the skin appeared normal on casual inspection. Substantial hypohidrosis or even anhidrosis was a typical sequela of experimentally induced miliaria, just as it is in the naturally occurring disease. Experimental hypohidrosis lasted as long as 3 weeks in some volunteers, usually those most severely affected.[10] These volunteers, whose work performance was tested 1 and 2 weeks after suffering from miliaria, were found to have markedly impaired tolerance for working in the heat. After approximately 60% of the body was wrapped, the postmiliarial hypohidrosis caused incapacitating and potentially dangerous heat exhaustion, even in heat-acclimatized volunteers who worked in a hot environment.[10]

The pathogeneses of both miliaria and its consequent hypohidrosis are associated with alterations in the orifices or ducts of the sweat glands. These changes are related to the suppression of normal delivery of sweat to the skin's surface.[4] However, it remains unclear whether any of the observable anatomical alterations cause hypohidrosis or merely result from sweat retention. Inflammation is almost certainly a secondary event because it does not occur until 24 hours after sweat blockade has ensued. One group of researchers[8] found that they could relieve experimental anhidrosis with stratum corneum stripping, which implies a sweat blockage in or just below the horny layer.

Other researchers[5] studied the role of resident bacteria in the etiology of miliaria. They found that the degree of miliaria and hypohidrosis after experimentally induced disease was directly proportional to the increase in the density of resident aerobic bacteria, notably cocci. No anhidrosis resulted when antibacterial substances were used to prevent the proliferation of the microflora. The investigators postulated that cocci secrete a toxin that injures sweat-duct luminal cells and precipitates a cast within the lumen. Infiltration by inflam-

matory cells might create an impaction that completely obstructs the passage of sweat for several weeks. This hypothesis has never been established with certainty; the antecedent to the histological changes of sweat-duct leakage or rupture and the clinical picture of miliaria remain elusive.[4]

Miliaria crystallina requires no treatment. The other, deeper forms of miliaria are best treated by removing the soldier from the hot, humid environment.

Hypohidrosis Syndrome

Hypohidrosis syndrome (also known as tropical anhidrotic asthenia) is a curious and unusual disorder that was first described in American and British troops during World War II.[1] Some of these soldiers were stationed in the desert in the southwestern United States at the time, and others were in southern Iraq. Cases that occurred in Louisiana and the Southwest Pacific were also described.

The syndrome, which can either appear suddenly or have a gradual onset, is characterized by

- absence of sweating over most or all of the cutaneous surface;
- ability to sweat in profuse amounts in a very limited area (usually the face);
- relatively mild symptoms of overheating, weakness, dizziness and headache;
- diuresis; and
- cutaneous changes ranging from prickly heat and transient papules to xerotic skin with a fine scale.[1]

Most patients had already been in a hot climate for some time and had had no previous difficulty sweating. Often the hottest season was drawing to a close when the symptoms emerged.[1]

Most patients suffering from hypohidrosis were not severely ill and did not suffer from hyperpyrexia. Many had been mildly uncomfortable for several days before seeking medical attention. Blood electrolyte studies and skin biopsies, when performed, were normal. Treatment consisted of rest in a relatively cool place, and subjective recovery was usually rapid. However, several weeks to as long as 4 months elapsed until sweating returned to normal. No satisfactory explanation for the cessation of sweating was ever found. Some speculated that a functional fatigue or exhaustion of the sweat mechanism was responsible.[1] Miliaria profunda may be the underlying mechanism in some cases of hypohidrosis.[3]

Tropical Acne

Tropical acne is a severe, disabling condition that is peculiar to hot and humid environments. Although many of the victims have a history of mild acne during adolescence, the skin is often quiescent when the problem erupts. Most young people with a history of acne *do not* develop tropical acne, and there is no way to predict which individuals will develop the condition.[11] Tropical acne has an explosive onset that begins, on the average, 3 to 6 months after the patient is exposed to tropical climatic conditions. Patients are usually about 25 years old when affected, but susceptibility ranges from teenagers through 30-year-olds. Those who are deprived of proper bathing facilities and are subjected to extreme heat and poor hygienic conditions for great lengths of time are most susceptible. Garrison troops with access to adequate facilities are not affected nearly so severely.[6]

The lesions in tropical acne are typical of those found in conglobate acne: pustules, papules, nodules, and draining sinuses.[11] Each lesion tends to be highly inflamed and juicy, with a purulent or sanguinous discharge. In soldiers, the first manifestation is of cystic lesions on the back, making it impossible to carry a pack.[6] Confluence of lesions covering broad expanses of skin is often seen (Figure 3-3). The face tends to be spared, but elsewhere the lesions are more extensive even than those seen in conglobate acne. The neck, arms (even forearms), and entire torso are often involved. Characteristically, the buttocks and upper thighs are affected.

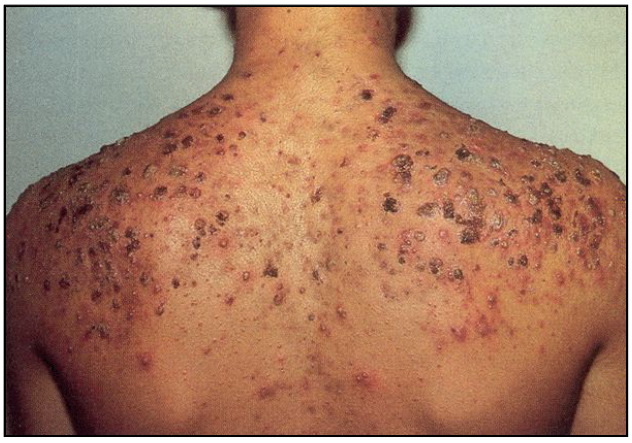

Fig. 3-3. Tropical acne first manifests itself as cystic lesions on the back, making it impossible for a soldier to carry a pack. Therapy is futile unless the soldier is moved out of the hot, humid climate.

As is typical of other highly inflammatory diseases, the sedimentation rate is often elevated and a leukocytosis is present. The patient with tropical acne often feels ill and can no longer function successfully as a soldier. The bacterial organisms recovered from cultures usually show normal skin flora or occasional colonies of *Staphylococcus aureus* and Gram-negative rods. However, bacterial infection is not believed to play an important role, and antimicrobials are ineffective as a form of therapy.[11]

Tropical acne persists undiminished in intensity until the patient is moved to a cooler, dryer climate. The patient is usually treated for weeks or months before the medical officer makes the inevitable decision to evacuate the patient to a more favorable locale.[6] Once the patient is removed from the tropical environment, the condition promptly begins to clear. Before the advent of systemic retinoids, no treatment, including hospitalization, was effective. Few data are available concerning the efficacy of isotretinoin in treating tropical acne, but even this drug would not be practical. Under the best of conditions, the drug takes several months to reverse the changes of severe, inflammatory acne. Frequent monitoring of laboratory parameters would be required during this period, and success is by no means guaranteed. The simplest, most effective, and essential therapy for this devastating disease is removal of the patient from the tropics.

Cholinergic Urticaria

Cholinergic urticaria is a fairly common disorder, and is seen most often in young adults.[12] The condition can be triggered by a variety of factors, including heat, exercise, and emotional stress. The patient first notes a sensation of warmth, which is then followed by an eruption of 1- to 3-mm wheals, which are surrounded by an erythematous flare (Figure 3-4). Usually the torso is affected, but in severe cases the rash is generalized. Wheezing and systemic symptoms (nausea, headache, and abdominal pain) are unusual but have been reported.[13] Angioedema has also been reported.[14]

Avoiding the predisposing factors is the most important aspect of treatment. This is understandably difficult in a hot environment, especially when coupled with the rigorous exercise and stressful training expected of a soldier. Regular exercise can induce tolerance, but could be dangerous in a patient who develops systemic symptoms.[12] H_1-blocking antihistamines such as hydroxyzine (10–25 mg four times daily)[15] can be helpful. Aspirin should be avoided because it often will worsen the eruption.[16] These patients should never exercise alone when their disease is active.[12]

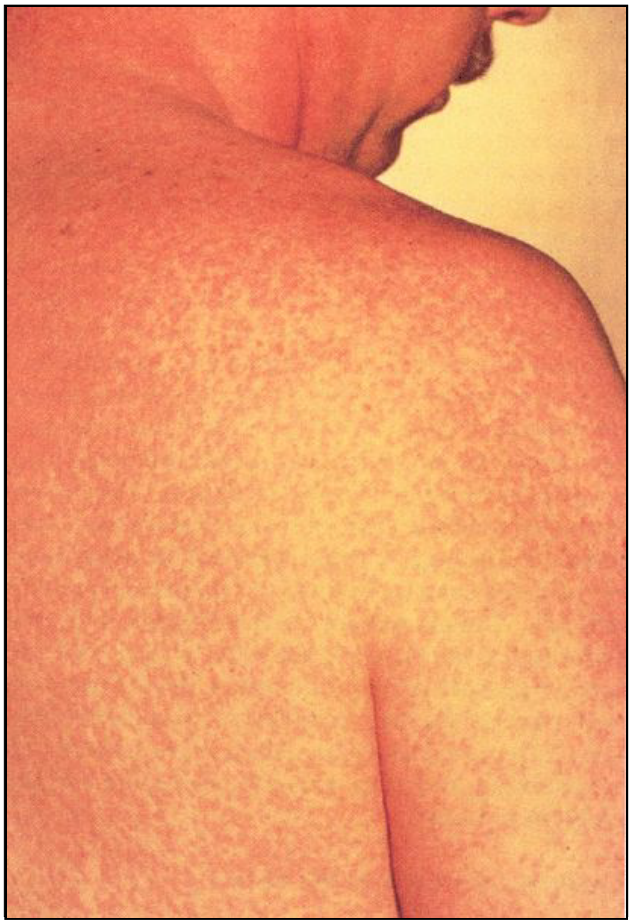

Fig. 3-4. In cholinergic urticaria, multiple small (1- to 3-mm) wheals are surrounded by an erythematous flare. Lesions are induced by exercise and resolve completely in a few hours.

DERMATOSES EXACERBATED BY HEAT AND HUMIDITY

Hot, humid conditions can worsen a variety of dermatoses, among which are dyshidrotic eczema, bacterial and fungal infections, friction blisters, and erythermalgia. Frequently, wet clothing and boots are also contributing factors.

Dyshidrotic Eczema

Dyshidrotic eczema (also known as dyshidrosis or pompholyx) is a vesicular eruption of the palms and soles. Lesions are spongiotic intraepidermal

vesicles that often burn or itch. The characteristic vesicles are usually bilateral and roughly symmetrical in distribution. Sometimes the lesions are arranged in groups and can become confluent to form bullae (Figures 3-5 and 3-6). The vesicle fluid is at first clear and colorless but may eventually become straw colored or purulent. Hyperhidrosis is often present in affected individuals.

The exact etiology of dyshidrotic eczema is unknown. Some investigators believe that hyperhidrosis is not one of the causative factors but that emotional stress is a more important prerequisite.[5] However, combat troops in tropical environments are subjected to both considerable stress and profuse sweating; regardless of the exact etiology, they are prime candidates for the condition. The combination of extreme heat and nervous tension was believed to be largely responsible for the large number of cases seen in Vietnam.[3] Treatment is often difficult. Potent topical corticosteroids and cool soaks (eg, with Burow's solution or just cool tap water) may suffice for milder cases. Severe involvement can require oral or intramuscular corticosteroids. Extensive lesions on palms, soles, or both can be disabling and may require evacuation for the problem to remit.

Bacterial Infections of the Skin

Heat and humidity predispose soldiers to a variety of bacterial infections. The combination of heat, humidity, wet clothing, poor hygiene, and minor skin trauma was believed to be the most important factor contributing to the high rates of cutaneous infections seen in Vietnam.[3] These infections were especially common in combat troops (as opposed to support personnel). Adverse climatic conditions, when coupled with the poor personal hygiene often found in combat troops, can lead to frequent and severe infections. Staphylococcal skin infections (superficial pustular impetigo, furunculosis, pyogenic paronychia, and staphylococcal impetigo), streptococcal infections (impetigo, ecthyma, erysipelas, and cellulitis), and a host of less common infections are found among soldiers in tropical climates. A detailed account of bacterial infections and their treatment may be found in Chapter 13, Bacterial Skin Diseases.

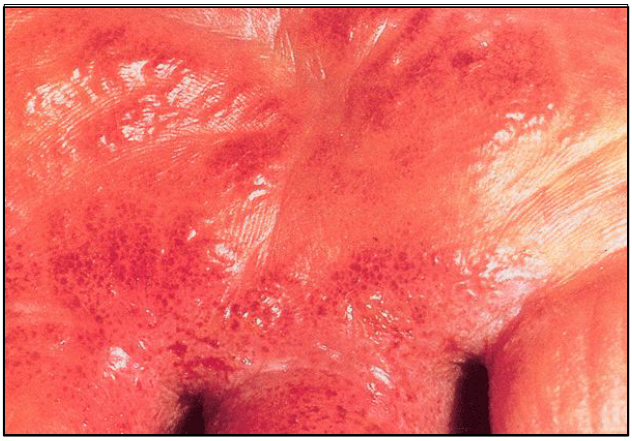

Fig. 3-5. Vesicles characteristic of dyshidrotic eczema are seen here on the palm. The lesions can be intensely pruritic.

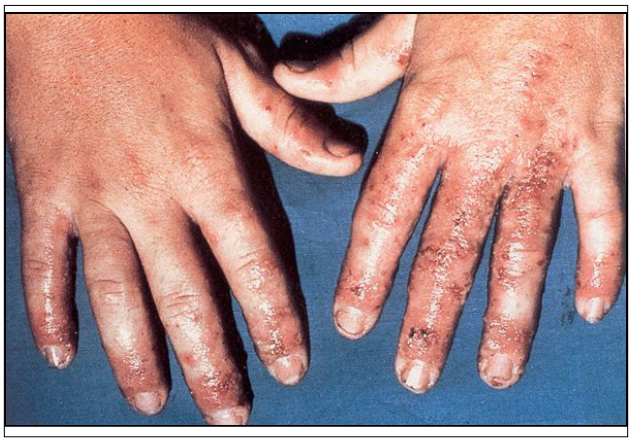

Fig. 3-6. Lesions on the sides of the fingers are common in dyshidrotic eczema. Other frequently affected sites include the palms and the soles.

Fungal Infections of the Skin

The various forms of tinea (ringworm) are more frequent and more severe among soldiers who are stationed in hot and humid environments. Superficial fungal infections were the most common and troublesome of all the dermatologic conditions that occurred among United States forces in Vietnam.[3] Just as in bacterial infections, the combination of heat, humidity, wet clothing and boots, and poor hygiene contributed significantly to the severity and frequency of fungal infections (Figure 3-7). A detailed discussion of these superficial fungal infections may be found in Chapter 17, Superficial Fungal Skin Diseases.

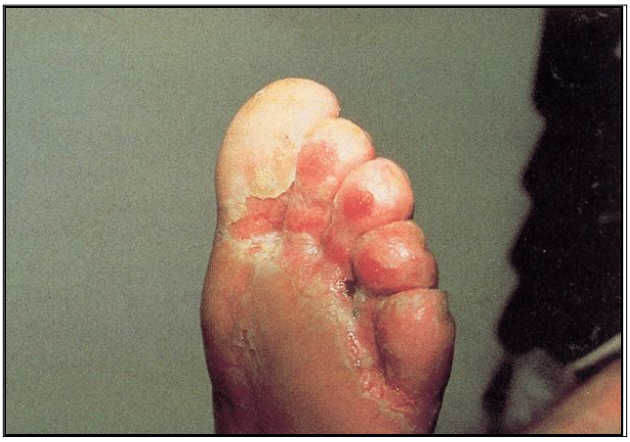

Fig. 3-7. Superinfected tinea pedis. Gram-negative organisms are often found in these mixed infections.

Friction Blisters

Friction blisters are common in all climates but can be significantly more frequent and severe in hot, humid environments. For a detailed discussion of friction blisters, see Chapter 7, Cutaneous Trauma and Its Treatment.

Erythermalgia

Erythermalgia is a condition in which the hands, legs, or feet develop intense erythema and pain resulting from environmental heat. A particularly warm climate is not necessary to precipitate attacks. Simply an increase in local skin temperature may be sufficient to precipitate a prolonged and painful attack. Symptoms usually involve the lower extremities and are almost always bilateral.[17] Men are more often affected than women. Patients try to relieve the discomfort by cooling their affected limbs using fans, cold water soaks, ice packs, and so forth.

Cases can either be primary (with no underlying disorder) or secondary, in which case an underlying condition such as polycythemia vera exists. Other associations seen with secondary erythermalgia include venous insufficiency, diabetes mellitus, hypertension, systemic lupus erythematosus, and rheumatoid arthritis.[12] The pathogenesis is unknown and no treatment is universally effective. Aspirin (650 mg every 4 hours) is the most dependable therapy.[12] In cases of secondary erythermalgia, treatment of the underlying or associated disorder may relieve this painful condition.

DERMATOSES CAUSED BY EXCESSIVE SUN EXPOSURE OR ALLERGY TO SUNLIGHT

Several skin disorders can arise either directly from exposure to the sun's rays or from sunlight's interaction with drugs. With all of these conditions, preventive measures are critical.

Sunburn

Sunburn is a cutaneous photosensitivity reaction that can occur in all humans, although its severity diminishes as melanin pigmentation increases.[18] It seldom occurs in black-skinned individuals. A single instance of severe sunburn can be disabling, and repeated episodes put the person at much greater risk for the development of nonmelanoma skin cancer (usually basal cell and squamous cell carcinomas). Additionally, there is evidence to suggest that several episodes of sunburn or even one severe sunburn substantially increases the risk of developing malignant melanoma.[19] For all these reasons, prevention of sunburn is important to improving not only fighting capability but also the long-term general health of the soldier.

UV-B radiation, with the shortest wavelengths of light (290–320 nm) to reach the earth's surface, is responsible for most if not all of the short-term effects of sunburn. The common name for UV-B radiation is therefore "sunburn rays." UV-A radiation (320–400 nm) and visible light (400–800 nm) have longer wavelengths, and are not associated with sunburn.

UV-B erythema (sunburn) becomes visible within 2 to 6 hours of sun exposure and peaks at 24 to 36 hours.[18] The erythema fades in 3 to 5 days, and in all but the fairest-skinned individuals, is followed by an increase in skin pigmentation (tanning). Severe sunburn is as injurious to the skin as a thermal burn, and severe cases can actually require hospitalization in a burn unit. Because UV-B radiation does not penetrate beneath the epidermis to any significant degree, the actual injury is primarily epidermal. This injury is manifested histologically by dyskeratotic "sunburn cells" and clinically by erythema, edema, and in more severe cases, blistering. Various mediators of inflammation are

activated by the sunburn phenomenon, and the dermis also becomes secondarily involved. These consequences are evident from the vasodilation, dermal edema, and leukocyte infiltration that occur in sunburned dermis. The exact mechanism by which UV radiation causes skin inflammation remains poorly understood.[18] For practical purposes, it is enough to remember that sunburn acts like a true burn, although third-degree injury (dermal necrosis) does not occur.

Once the erythema of a sunburn is well established, its treatment is similar to a thermal burn of equivalent severity. Severe sunburn with extensive blistering may require hospitalization and the kind of management that is utilized in a thermal burn unit. Oral or parenteral administration of high doses of corticosteroids can be of symptomatic benefit, but they must be tapered rapidly to reduce the chance of secondary infection.[18] Most sunburn cases are much milder and can be managed with cool-water soaks, topically applied corticosteroid creams, and oral aspirin (650 mg every 4 hours, as needed) or indomethacin (25 mg three times a day, as needed). If an anticipated case of sunburn is treated early, before erythema has appeared or peaked, topical or oral indomethacin may delay the onset and diminish the intensity of UV-B erythema.[20,21] Although indomethacin might afford some symptomatic relief, there is no evidence to suggest that these drugs prevent the actual epidermal injury that accompanies sunburn. The delayed, post–24-hour erythema appears to be uninhibited by such treatment.[21]

Clearly the most helpful tactic is to avoid sunburn in the first place. The largest amounts of UV-B radiation penetrate the atmosphere at midday, when the sun casts no shadow. Avoiding outdoor work during the hours of 1000 to 1400 will reduce the chance of overexposure. Another rule of thumb is for soldiers to avoid outdoor work when their shadows are shorter than their height, which always corresponds to the time of day when UV-B radiation is most intense.

EXHIBIT 3-1

PHOTOTOXIC AND PHOTOALLERGIC CHEMICALS

Phototoxic chemicals
- Dyes
  - Eosin
- Coal-tar derivatives
  - Acridine
  - Anthracene
- Furocoumarins
  - Psoralen
  - 8-methoxypsoralen
  - 4,5,8-trimethylpsoralen
- Drugs
  - Thiazides
  - Chlorothiazides
  - Doxycycline and other tetracycline-like derivatives
  - Furosemide
  - Nalidixic acid
  - Naproxen
  - Benoxaprofen
  - Phenothiazines such as chlorpromazine
  - Piroxicam
  - Quinidine
  - Amiodarone
  - Sulfonamides

Photoallergic chemicals
- Halogenated salicylanilides
  - Tetrachlorosalicylanilide
- Antifungal drugs
  - Multifungin
  - Fentichlor
  - Jadit
- Phenothiazines
  - Chlorpromazine
  - Promethazine
- Sulfanilamides
- Sunscreens
  - PABA esters
  - Digalloyltrioleate
- Whiteners
  - Stilbene
- Fragrances
  - Musk ambrette
  - 6-methylcoumarin

Chemicals that are both phototoxic and photoallergic
- Phenothiazines
- Sulfonamides

Source: Harber LC, Bickers DR, eds. *Photosensitivity Diseases: Principles of Diagnosis and Treatment.* Philadelphia, Pa: BC Decker, Inc; 1989.

Protective clothing should be worn whenever possible. A broad-brimmed hat is appropriate for any climate, and will reduce UV radiation exposure to the head and neck. Long-sleeved shirts and full-length trousers should be worn whenever possible. A significant amount of light can penetrate loosely woven or soaking-wet clothing, and a tightly woven, heavier fabric is always preferable. Gloves will protect the hands.

When protective clothing is impractical, such as in an extremely hot and humid climate, a chemical sunscreen should be applied. Numerous types are available, but for field conditions a sunscreen with a sun protection factor (SPF) of at least 15 should be used. It is especially important to treat the face, ears, neck, and hands, because it is often impossible to cover these areas with protective clothing (a hat is not protective from reflected light off sand, snow, water, and so forth). Most chemical sunscreens contain *p*-aminobenzoic acid (PABA) or its derivatives. Soldiers who are sensitive to PABA can use equally effective sunscreens based on salicylates, cinnamates, benzophenones, and dibenzoylmethanes.

Individuals with deep tans or naturally dark skin have considerable protection from UV-B radiation but are still susceptible to UV radiation–induced photodamage.[21] However, tanning as a protective measure is both impractical and injurious. Some individuals can never achieve a truly protective tan; rapid deployment to sunny climates eliminates the possibility of achieving a tan without the morbidity of burning. Tanning itself is an indication that UV radiation–induced epidermal injury has occurred, which predisposes an individual to all the long-term adverse sequelae such as skin cancer and premature aging.

Phototoxicity and Photoallergy

Both phototoxicity and photoallergy are due to the combined effects of drugs (or other chemicals) and sunlight. Neither drug nor light is alone sufficient to cause the reaction. In theory, phototoxic drugs are capable of causing an adverse reaction in *all* individuals if administered in high enough doses. Photoallergic drugs cause idiosyncratic responses (only certain predisposed patients will develop the allergy). Some photoallergic and phototoxic reactions occur when the responsible drug or chemical is taken internally and carried to the skin via the bloodstream. Other chemicals are capable of causing photo*contact* allergy (the reaction between light and drug occurs when the offending chemical is applied to the surface of the skin and then irradiated) (Exhibit 3-1).

In general, phototoxic reactions are confined to areas of the skin exposed to light, such as the face, the pinnae of the ears, the "V" of the neck, the nuchal area, and the extensor and dorsal surfaces of the arms (Figure 3-8). Phototoxicity from many systemically administered drugs, such as the tetracycline derivatives, can appear as mild erythema resembling severe sunburn. However, the full spectrum of sunburnlike reactions may occur, including extensive blistering. Reactions to some agents such as the psoralens can cause eventual hyperpigmentation of the involved areas. Certain systemically administered phototoxic drugs such as demeclo-

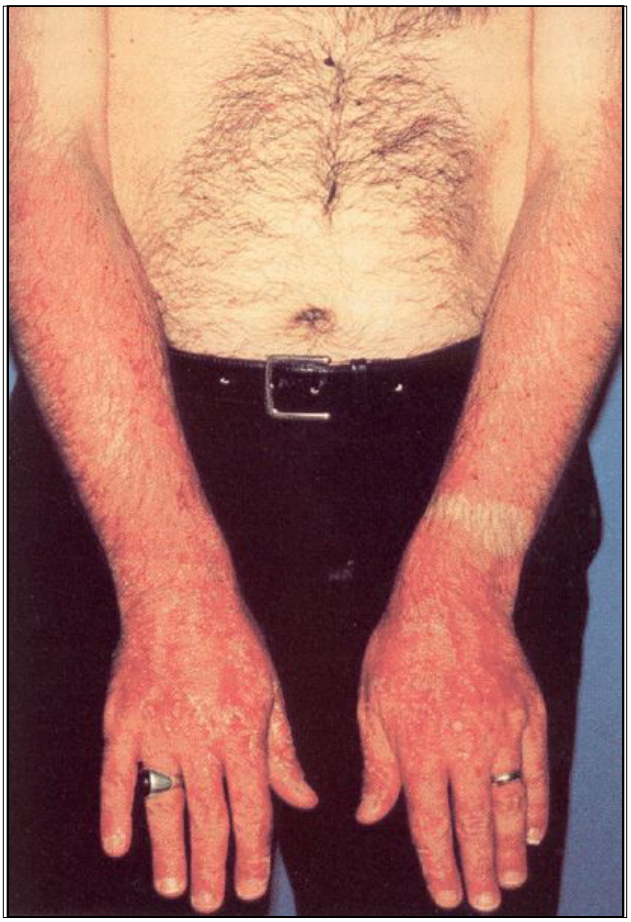

Fig. 3-8. Phototoxic reactions such as this one caused by Tegretol (carbamazepine, manufactured by Basel Pharmaceuticals, Summit, N.J.) are generally confined to regions of the skin exposed to light. A photoallergic eruption might assume a similar appearance.

cycline can also cause photoonycholysis, in which separation of the nail from the nail bed occurs.

Photoallergy is much less common than phototoxicity.[18] It involves an immunological response of the skin to the combination of a specific chemical and light. The general mechanism of action is believed to be the conversion of the offending chemical by light into a photoproduct. The photoproduct then binds to a soluble or membrane-bound protein to form the antigen that precipitates a delayed–hypersensitivity immune response. Once the patient has been sensitized to the chemical, subsequent reexposures will elicit a cutaneous reaction.[22]

The most common groups of chemicals causing photoallergy are topically applied fragrances and antibiotics. Typically, the reactions caused by these agents resemble acute contact dermatitis such as that seen with poison ivy. The eruption is papulovesicular or vesicular and frankly eczematous. Light-exposed areas of the body are the sites of involvement, just as is the case with phototoxic reactions. Postinflammatory hyperpigmentation, however, is less often encountered in photoallergy.

Establishing the diagnosis of photoallergy or phototoxicity requires phototesting (and photopatch testing in cases of photoallergic contact dermatitis). The technique of phototesting, which may require specialized equipment, is beyond the scope of this chapter. Treatment involves avoiding the contributing factors—certain wavelengths of light and the responsible chemical. In theory, avoiding the chemical or drug alone should be sufficient. However, certain drugs such as hematoporphyrin bind avidly to dermal proteins and are only slowly eliminated from the skin, which can take months. Phototoxicity from this drug can therefore be prolonged.

Polymorphous Light Eruption

Polymorphous light eruption (PMLE), a condition of unknown etiology, is one of the most common disorders of photosensitivity.[18] Several other names have been applied to the condition, some of which are either synonymous with or variants of PMLE. These names include solar eczema, summer prurigo, solar dermatitis, and perhaps hydroa aestivale. A photosensitivity disease called actinic prurigo is often included as a clinical subset of PMLE, although some authorities consider it to be a separate condition.[23] The entire group of conditions designated as PMLE may represent a clinical spectrum of severity for a single disease or, alternatively, may actually represent a diverse group of photosensitivity disorders with different pathogeneses. For purposes of this chapter, the entire group will be referred to as PMLE.

PMLE occurs in all ethnic groups, but Native Americans appear to be genetically predisposed to the subset called actinic prurigo. All age groups can be affected, and there does not seem to be a clear-cut difference in incidence between males and females.

The lesions of PMLE can assume a variety of appearances (hence the name polymorphous) including erythema, eczema, vesicles, papules, nodules, plaques, and areas that are excoriated, lichenified, or both (Figures 3-9 and 3-10). The lesions are most common on the face, but relatively sun-protected areas such as the submental region are spared. The eruption is usually confined to sun-exposed areas, but because the lesions of PMLE are usually patchy and irregular, a sharp border is seldom as evident as that seen in phototoxic or photoallergic reactions.

Typically, PMLE begins or recurs in the spring as the amount of UV light and opportunities for exposure increase. Lesions usually erupt several hours after sun exposure has occurred. They begin as papules or plaques in light-exposed areas, and are moderately to severely pruritic. The pruritus is often described as "stinging."[18] Subsequent exposures to sunlight will cause new lesions, again after a lag of several hours. However, as spring and summer progress, many patients experience a lessening of disease severity, as if "hardening" is occurring. Lesions persist for several days. With repeated outbreaks at close intervals, large eczematous or lichenified plaques may form.

The histopathological findings are often helpful

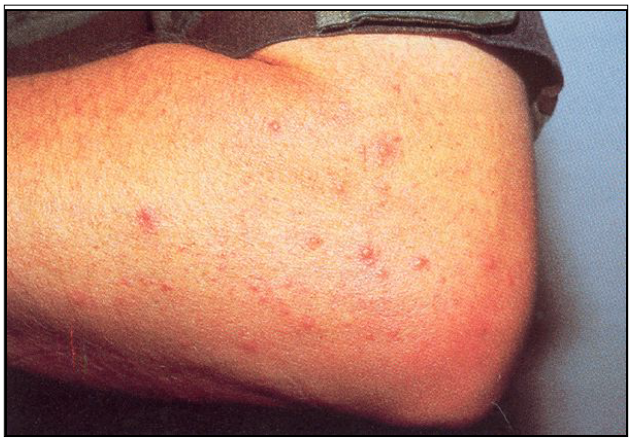

Fig. 3-9. Polymorphous light eruption: papular lesions.

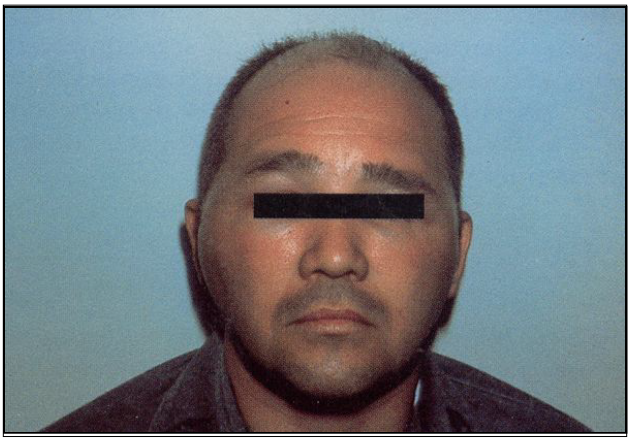

Fig. 3-10. Polymorphous light eruption: diffuse erythema and swelling.

in making a diagnosis. Characteristically, dense aggregates of mononuclear cells are seen in a perivascular distribution in the mid- and upper dermis. Epidermal changes are nonspecific and include edema, spongiosis, parakeratosis, acanthosis, and mild vacuolar degeneration of the basal cell layer.[18]

PMLE can be caused by UV-B or UV-A radiation, visible light, or a combination of these. Different patients seem sensitive to different wavelengths of light. This phenomenon may be a true difference in reactivity between individuals, or may simply indicate that the term PMLE represents a variety of diseases with differing etiologies. Phototesting may pinpoint the offending wavelengths of light, but such an evaluation is relatively complex and is often unrewarding.

The best treatments for PMLE are avoiding sunlight and wearing protective clothing. However, avoidance is often impractical, especially in soldiers whose duties require outdoor work. Sunscreens are usually somewhat helpful, although they seldom achieve complete control. Because many cases of PMLE are caused by UV-B radiation, suncreens containing PABA or other UV-B blockers can be useful. Suncreens that are protective in the UV-A range (eg, Photoplex, consisting of avobenzone and padimate O, manufactured by Allergan Herbert, Irvine, Calif.) are less effective because they screen relatively little UV-A radiation (compared to the excellent blockade achieved by many UV-B sunscreens). Opaque sunscreens such as titanium dioxide are effective against all damaging wavelengths but are also messy and cosmetically unacceptable.

Antimalarial drugs taken systemically are the mainstay of therapy for incapacitating disease that is not adequately controlled by the above methods. Plaquenil (Hydroxychloroquine, manufactured by Sanofi Winthrop Pharmaceuticals, New York, N.Y.) is used most often, and can be effective in low doses (250–500 mg daily). It may only be required during the spring and early summer, when disease activity is at its peak. The major drawback to the antimalarial drugs is the retinal toxicity that they can cause. Patients need to be monitored every 3 months to assure that irreversible ocular changes are not in progress. These drugs also may produce some less serious side effects such as headaches, gastrointestinal upset, blurred vision, and pruritus. Clearly the antimalarial drugs are far from ideal as a treatment for a relatively benign, although potentially incapacitating, disease.

For severe flare-ups of PMLE, systemic corticosteroids are effective. Serious long-term side effects make this class of drugs undesirable for chronic therapy.

Solar Urticaria

Solar urticaria manifests itself by the onset of erythema and pruritus a few minutes after sun exposure; these areas then become edematous or frankly urticarial. The eruption is limited to sun-exposed skin, and in the absence of reexposure, the lesions disappear after several hours. This stage is followed by a period of 12 to 24 hours in which the skin is refractory to new lesions, even if sun exposure occurs. Like typical urticaria, an extensive outbreak can be associated with an anaphylaxis-like reaction.[18]

Solar urticaria can occur at any age, but most reported cases have been in the 30- to 40-year-old age group. Women are more often affected than men. The condition may spontaneously remit within a few months (which occurs in about one fourth of patients), but usually persists for years.

All wavelengths of sunlight have been implicated in causing one or more of the various subsets of solar urticaria. Eruptions caused by visible light seem to have the best prognosis.[18] Phototesting may reveal the culprit wavelengths, which is of obvious practical value in preventing outbreaks. However, because such testing requires special equipment that is usually only available in medical centers, it has little use in field medicine.

No specific therapy exists for solar urticaria. Avoiding sun exposure is the most successful pre-

ventive measure, but this is usually impractical. Antihistamines are often not effective until sedating doses are used. Antimalarials have been found useful in a few cases. Desensitization has proven effective in several cases.[18] This method involves frequent exposure of a small area of the skin to the offending wavelengths of light until no reaction occurs. Then the size of the treated area is gradually increased until the entire body can be exposed without a reaction. Daily or every-other-day exposures of 10 to 20 minutes often keep the patient lesion-free despite normal sunlight exposure.

DERMATOSES EXACERBATED BY SUNLIGHT

Several skin diseases can be markedly worsened by exposure to sunlight, making it extremely difficult for the soldier to function effectively. Fortunately, most of these disorders are uncommon.

Porphyria Cutanea Tarda

Porphyria cutanea tarda (PCT) actually is an example of phototoxicity, but in affected patients the phototoxic drugs are endogenous porphyrins. PCT patients present with blisters and erosions in sun-exposed areas. The disease is caused by a metabolic defect in the production or metabolism of porphyrins. Visible light in the 400 to 410 nm range is the part of the spectrum most responsible for the reaction. Therapy consists of phlebotomy (to reduce hepatic iron stores), antimalarial drugs, or both.

Erythropoietic Protoporphyria

Patients with erythropoietic protoporphyria, a much less common defect in porphyrin metabolism, present acutely with areas of sun-exposed skin that become painful shortly after sun exposure. Erythema and edema then develops in those sites where stinging pain has occurred. Pain is usually more prominent than the visible skin lesions, but small, pitted scars can develop over time. Oral beta carotene has been found to be useful in reducing the severity of the disease. Erythropoietic protoporphyria is an autosomal dominant inherited condition that usually manifests itself in childhood, and is therefore very unlikely to be encountered in active-duty military personnel.

Others

The skin lesions of both discoid lupus erythematosus and systemic lupus erythematosus can be induced or significantly worsened by sunlight. Recurrent herpes simplex viral outbreaks can be precipitated by sun exposure.

The rash of pellagra is clearly a photosensitive disorder. Except in maltreated prisoners of war and soldiers who are taking the drug isoniazid, this deficiency of tryptophan and niacin is very unlikely to occur.

SUMMARY

Soldiers entering hot or sunny climates are at risk for heat-related skin injury or disease, which can often be debilitating. Even the classic forms of heat injury such as heatstroke are influenced by the skin because sweating is so important for temperature homeostasis.

Excessive environmental heat is directly responsible for miliaria, the best known form of which is prickly heat. Tropical acne, cholinergic urticaria, and the hypohidrosis syndrome can also be problems in hot climates.

Some skin conditions may be exacerbated by heat and humidity, turning a mild dermatosis into a disabling disease. Bacterial and fungal infections of the skin are always problems in hot and humid settings, and friction blisters and dyshidrotic eczema are far more commonly seen in those conditions.

The intense UV light found in sunny climates is responsible for sunburn, which can be just as incapacitating as a thermal burn. A higher incidence of phototoxic and photoallergic reactions to drugs is expected with increased sun exposure. Allergy to certain wavelengths of sunlight (polymorphous light eruption) can be especially problematic.

Certain skin conditions such as the porphyrias and cutaneous lupus erythematosus can be significantly worsened by exposure to sunlight. It is unlikely that a soldier with either condition could function effectively in a sunny environment.

REFERENCES

1. Eichna LM. Heat casualty. 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: 195–330.

2. US Departments of the Army, Navy, and Air Force. *Prevention, Treatment and Control of Heat Injury*. Washington, DC: 1980. Technical Bulletin MED 507.

3. 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: 29–52.

4. Sulzberger MB, Harris DR. Miliaria and anhidrosis. *Arch Dermatol*. 1972;105:845–850.

5. Arnold HL, Odom RB, James WD. *Andrews' Diseases of the Skin*. Philadelphia: WB Saunders Co; 1990: 4.

6. 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: 543–674.

7. Sulzberger MB, Emik LO. Miliaria: Clinical and statistical findings. *J Invest Dermatol*. 1946;7:53–59.

8. Papa CM, Kligman AM. Mechanisms of eccrine anhidrosis. Part 1: High level blockade. *J Invest Dermatol.* 1966;47:1–9.

9. Stoughton, RB. Suppression of miliaria rubra (prickly heat) by a topical anticholinergic agent. *J Invest Dermatol*. 1964;42:228.

10. Sulzberger MB, Griffin TB. Induced miliaria, post-miliarial hypohidrosis and some potential sequelae. *Arch Dermatol*. 1969;99:145–151.

11. Plewig G, Kligman AM. *Acne: Morphogenesis and Treatment.* New York: Springer-Verlag; 1975; 194–196.

12. Page EH, Shear NH. Temperature-dependent skin disorders. *J Amer Acad Dermatol.* 1988;18:1003–1019.

13. Kounis NG, MacMahon RG. Cholinergic urticaria with systemic manifestations. *Ann Allergy.* 1975;35:243–245.

14. Lawrence CM, Jorizzo JL, Kobza-Black A, Coutts A, Greaves MW. Cholinergic urticaria with associated angioedema. *Br J Dermatol*. 1981;105:543–550.

15. Lookingbill DP, Marks JG Jr. *Principles of Dermatology*. Philadelphia: WB Saunders Co; 1986: 198.

16. Douglas HMG. Reactions to aspirin and food additives in patients with chronic urticaria, including the physical urticaria. *Br J Dermatol.* 1975;93:135–144.

17. Babb RR, Alarcon-Segovia D, Fairbairn JF II. Erythermalgia, review of 51 cases. *Circulation.* 1964;29:136–141.

18. Harber LC and Bickers DR, eds. *Photosensitivity Diseases: Principles of Diagnosis and Treatment.* Philadelphia: BC Decker, Inc; 1989.

19. Lew RA, Sober AJ, Cook N, Marvell R, Fitzpatrick TB. Sun exposure habits in patients with cutaneous melanoma. *J Dermatol Surg Oncol*. 1983;9:981.

20. Eaglstein WH, Marsico AR. Dichotomy in response to indomethacin in UV-C and UV-B induced ultraviolet light inflammation. *J Invest Dermatol.* 1975;65:238–240.

21. Consensus Development Panel. National Institutes of Health Summary of the Consensus Development Conference on sunlight, ultraviolet radiation, and the skin. *J Am Acad Dermatol.* 1991;24:608–612.

22. Epstein JH. Phototoxicity and photoallergy in man. *J Am Acad Dermatol.* 1983;8:141–147.

23. Lane PR, Sheridan DP, Hogan DJ, Moreland A. HLA typing in polymorphous light eruption. *J Amer Acad Dermatol.* 1991;24:570–573.