

### III. BIOLOGIC EFFECT OF EXPOSURE

#### Extent of Exposure

Several field studies have been performed to assess the extent of heat stress to which workers in different occupations in the United States are exposed and to determine the extent of physiological strain which develops as a consequence of this exposure.

The field studies performed by the U.S. Public Health Service<sup>1,2,3,4</sup> investigated conditions in ferrous and non-ferrous metal factories, in glass and chemical industries, as well as in surface coal mining, dam building and other outdoor operations involving mainly heavy equipment operation. Minard et al.<sup>5</sup> recently reported their observations on steel workers.

The pertinent results of these field studies are summarized below.

The workers in hot jobs are a highly select population. Workers who feel that they cannot cope with the prevailing heat stress change their job for a less demanding one. As a result of this natural selection process, the majority of the workers in hot jobs have high levels of physical performance and capacity and are highly adaptable to work in heat.

Heat disorders are more likely to occur at times when the workers are unacclimatized as during the first hot spell in the summer or when physical fitness is diminished as on Mondays after a leisurely weekend or the first day after a vacation, or return to work after an illness.

Because jobs in hot environments may be better paid than other jobs, it often happens that workers try to stay with the hot job even after their health or fitness becomes inadequate for the job. Since there is no obligatory standard for physical fitness for these jobs and since periodic

medical examinations have been haphazardly done in many industries, if done at all, these workers stay on the job and run a high health risk.

Oral temperatures in excess of 99.6°F (corresponding to a deep body temperature of 100.4°F) or first-minute recovery heart rates in excess of 110 have been very seldom observed. They occurred mainly in jobs where the environmental conditions exceeded the upper limit prescriptive zone (ULPZ), (see part V) particularly if the workers worked overtime or worked two shifts in sequence.

There are many work practices in industry which are unofficial and are aimed at ameliorating the workers heat strain on excessively hot days. Such practices are:

1. Only the unavoidable operations are performed. Other less important jobs are postponed.
2. Workers involved in auxiliary jobs are reassigned to help out those who work in the hot areas.
3. The younger and more fit take over some of the work from the older and less fit.

These practices, if not recognized, may give the wrong impression that the old and less fit worker tolerates the work in heat as well as the younger and more fit.

Most workers in hot jobs drink less water than they lose by sweating. According to many laboratory and field studies, this affects physical fitness adversely, particularly if the water loss is more than 1.5% of total body weight.<sup>6</sup> Such dehydration could be prevented by:

1. Making drinking water of good quality easily accessible to the worker.

2. Providing a 0.1 percent salt solution as drinking water, available from drinking fountains which cool the water.

3. Providing salt tablets for salt supplementation to the workers.

4. Advising the worker about the significance of drinking water often in small installments and using much salt on food when he is exposed to hot working conditions.

In many jobs the workers' heat exposure could be substantially reduced by relatively simple measures, such as wearing certain protective clothing, turning on all available fans and opening all windows, distributing the job more evenly during the workday and breaking up the work cycles into shorter work-rest cycles. Unfortunately, either because of ignorance or carelessness, the workers often expose themselves to greater heat stress than would be necessary.

Often with little expense the climatic conditions could be ameliorated or the work load diminished.

#### Early Historical Reports

Nearly 70 years ago concern for the health of the Cornish Tin Miners lead to one of the first studies of the effects of heat on the health of workers and stimulated the search for a method of expressing in simple terms the impact of a hot working environment. Except for the studies of Bedford,<sup>7</sup> on the effects of atmospheric conditions on the industrial worker, little progress was made until shortly before and during World War II. In the late 1930's the interest was directed toward the industrial worker and the health and safety consequences of working in hot industries. The classical research of Bazett,<sup>8</sup> Bedford,<sup>7</sup> Dill,<sup>9</sup> Drinker,<sup>10</sup> Talbott,<sup>11</sup> Yaglou<sup>12</sup> and several others identified the acute and chronic heat disorders and their dependence

on the intensity of the heat stress. Recommendations for engineering controls and medical prevention and treatment were made which are still pertinent to the solution of today's industrial heat stress problems. Military operations in the tropics and the African Campaign of World War II stimulated a major research effort on the physiological consequences of exposure to high temperatures. Much of the vast quantity of basic information on acclimatization, water and salt requirements, heat disorders and permissible exposure levels developed during that period has been presented by Adolph et al.,<sup>13</sup> Newburgh,<sup>14</sup> and the Medical Research Council of England,<sup>15</sup> The Thermal Standards in Industry,<sup>12</sup> published in 1947, recommended limits and procedures which are considered valid and applicable in today's industries.

During the past 25 years much effort has centered around the problem of expressing in relatively simple terms the total impact of the hot working environment upon the worker. Several attempts have been made to formulate a predictive scheme which would translate the heat load into biologically meaningful values. These predictive schemes can be roughly grouped into: (a) those that sought a device which would respond to the major environmental factors in a manner similar to man's, (b) those based upon measured human responses which could be used to evaluate combinations of environmental conditions, and (c) those based on calculations to determine whether it is possible to maintain thermal balance under any combinations of the climatic factors and work intensity and if so, how much physiological strain is involved. Each approach has its logic as well as its failings. A detailed discussion of the more important of the indices for estimating the biologic impact of a hot environment is presented later in the text.

### Epidemiological Studies

Neither prospective nor retrospective epidemiological studies have been made in which the health experiences of workers have been correlated with the length and intensity of heat exposure at the work site during the working life of the individuals. Health data for retrospective studies could probably be found in the health and medical records of some insurance companies and larger industries. Particularly lacking in most of the morbidity and mortality reports, however, are measurements of the level of heat exposure and the time spent on the hot jobs.

Health experience statistics for some hot industries have been reported over the past 50 years.<sup>16,17,18,19,20,21,22</sup> In a study of 23,000 coal miners,<sup>18</sup> lost time due to sickness was 63 percent higher in miners working at temperatures above 80°F. than in those working at temperatures of 70°F. or less. Death rate increase of about 35 percent was reported in 1937<sup>20</sup> for miners working hotter mines. In another study, Britten and Thompson,<sup>21</sup> found organic heart defects were more frequent in foundry workers. Enlarged hearts and arteriosclerosis were found more often among steel and glass workers. The frequency of industrial accidents increases in higher temperatures but the increase is mostly in minor accidents. It is not possible to generalize these reports of heat experience in chronic heat exposure in industry several decades ago to present-day industry. However, personal communications and experiences of medical and scientific personnel suggest that chronic exposure in hot working environments can have serious health and safety consequences.

The acute effects of heat on health and safety have been documented by literally hundreds of carefully controlled laboratory and field studies. The incidence of heat illness in young men in industry and military service who were not acclimatized to heat has been reported in several studies to be between 1.5 and 3.5 per 1000 at risk in the United States, under conditions of environmental heat and work loads which approximate the 1971 ACGIH TLV for Heat Stress. Age, sex, individual tolerance and many other factors will influence the incidence rate.

#### Effects of Heat

Environmental heat (or resistance to removal of metabolic heat) leads to well-documented reactions in human beings: increased cardiovascular and respiratory activity, increased body heat content, sweating, etc. If the heat load is excessive or prolonged, then frank heat disorders result. In this section subpathological effects will be considered which may modify performance, behavior or responses to other simultaneously imposed stresses.

Physical performance is affected by heat. Heat stress involves cardiovascular strain, e. g., demands are made for blood flow to the periphery for thermo-regulatory purposes. Cardiac output, therefore, is not totally available to active muscles. The competition increases with increasing heat load when the combined demands exceed the maximum cardiac output, the upper limits of tolerance are reached, and work output must of necessity be reduced.<sup>23</sup> These conditions may obtain under emergency conditions or in highly motivated individuals. Such motivation can lead to overstrains.<sup>24</sup> That real limits of endurance

exist was demonstrated quantitatively by Wyndham et al.<sup>25</sup> Productivity of mining recruits varied with quality of their supervision up to saturated environments of 28.9°C (93°F). Further increase to a saturated environment of 35.5°C (95.8°F) resulted in almost total cessation of productive work.<sup>26</sup> Other examples of heat-limited work are given in Leithead and Lind.<sup>16</sup> Because of these experiences and those of Minard et al.,<sup>27</sup> upper limits for unrestricted work have been set at environments near the 29.5°C (85°F) effective temperature (ET) level. The Wet Bulb Globe Temperature Index was derived from the ET concept (see Section V); the recent tentative TLV for heat<sup>28</sup> specifies 30°C WBGT as the upper limit for continuous moderate work (approximately 200 Kcal/hr for acclimatized men).

Recently, the tentative TLV has been challenged as being too conservative.<sup>29</sup> Experience shows that often men have worked effectively for years in environments and at metabolic rates exceeding those suggested by the TLV with no apparent detrimental effects. Individual differences in heat tolerance and selection may in part account for this, errors in establishing time weighted average values for the metabolic costs of physical tasks and the environmental heat load may also prove to be partially responsible,<sup>30,31</sup> especially in the heavier jobs.

It should be noted that decrement in performance was not especially noted in the above cited references up to the 30°C ET (or WBGT) levels for tasks involving large muscle groups in gross efforts, e.g., marching, shoveling, heavy work in the hot industries. Lower levels of heat may adversely affect the efficiency with which the heavy tasks are performed<sup>32</sup> or may interfere with accomplishment of more skilled manipulative or psychological tasks.<sup>23</sup> Some of the factors that influence performance in the heat have recently been reviewed.<sup>33</sup>

Psychological performance is also affected by heat. Pepler has reviewed the effects of heat on skilled tasks (tracking, telegraphy) or mental tasks (learning).<sup>34,35</sup> Qualitatively, there is no doubt that heat interferes with these types of activities. It is interesting to note that sensorimotor coordination deteriorates more rapidly in cold than hot environments.<sup>35</sup>

It is common experience that heat exposures accelerate the onset of fatigue; prolonged hot conditions (e.g., summer heat waves) may further contribute to general fatigue by robbing the individual of sound sleep.<sup>35</sup> One effect, whether from heat directly or as an indirect effect of fatigue is that accuracy of response deteriorates.<sup>36</sup> Studies by Duggar<sup>37</sup> of a delicate assembly task indicated that though production of good pieces was maintained (subjects on incentive pay), the quantity of scrap increased somewhat. Thus, the workers were actually having to work harder in the heat to maintain production, a further indication of lowered efficiency.

More recently, Pepler has studied the effects of air conditioned versus non-air conditioned classrooms on the process of learning.<sup>38</sup> There seems to emerge statistically that even relatively slight increases in environmental temperatures affect learning adversely.

Several psychomotor tasks were examined in comfortable and warm environments (up to 80°F, 60 percent RH) by Griffiths and Boyce.<sup>39</sup> Examination of the results revealed an optimum performance at a temperature similar to the optimum comfort temperature. As Hatch points out,<sup>40</sup> the establishment of criteria for upper levels of heat exposure which has as its



primary goal to maintain physiological well-being and health should be determined by medical personnel. Below this level, the major considerations are in the province of management: decisions relative to productivity, employee relations and the like, except perhaps as these less-than-injurious exposures may influence accidents.

Wherever there exists molten metal, hot surfaces, steam, etc., there exists the potential hazards of accidental contact of the worker with the hot object. Burns of varying severity result. Often the accident will be caused by a secondary agent, such as water escaping into molten metal, malfunction of pressure relief valves on water heaters and the like. Aside from the direct burn hazard of heat and hot objects, environmental heat appears to increase the frequency of other kinds of accidents in general.

Mechanically, the heat may tend to promote accidents due to slipperiness of sweaty palms or interfering with vision through fogging of safety glasses. Beyond these obvious effects, accidents have been documented to increase in hot jobs (e.g., Vernon et al.<sup>18</sup>). A striking demonstration of environmental effect on accident rates was compiled from records of a steel mill over a four-year period (Figure 1).<sup>41</sup> There is a definite parallelism between weather and accident frequency. The accident peaks, however, exhibit a downward trend over the years, most likely reflecting the efforts of intensive safety programs. Belding et al. have suggested the weather effect may be due to reduced general tonus of bodily activities and alertness related to high environmental temperatures. Again, increased bodily temperature and discomfort

increase irritation, anger, and other emotional states which may induce workers to commit rash acts or divert attention from hazardous tasks.<sup>42</sup> In extreme heat, emotions may spill over into fights<sup>16</sup> or other manifestations of emotional crises, e.g.<sup>43</sup>

At lesser stresses, more subtle disturbances in emotional state, e.g., depression, may be evident. Extensive folklore has been generated around the deleterious effects of the "Foehn" of Europe, the "Sharav" of Israel, and other warm "ill winds".<sup>44</sup> While the correlation between these climatic changes and illness seems real, the aetiology remains controversial.<sup>45</sup> Intuitively, disturbed emotional states should reduce alertness on the job, setting the stage for accidents.<sup>46</sup>

Effects of stresses in the occupational environment have been the subject of many quantitative studies. From these, threshold limit values (TLV) were derived, values which have served well as guides to reduce occupational exposures.

In general, the TLV's were established from experiments with single stress exposures. Often, however, more than one stress will occur and, in fact, it will be the rare case where only one stress obtains. There has been an increasing awareness of the alteration in physiological response to a stress where other stresses are present. Because of the ubiquitous nature of heat stress, it has received attention as a potentiator or mediator of response to other physical and toxic agents; and it certainly influences the course of diseases.

In combination, heat (85°F, ET) and carbon monoxide (100 ppm) have been shown to have a greater deleterious effect than either stress alone.<sup>47</sup> It is difficult to quantitate the effect; manifestations included

inability to complete the four-hour exposure, irritability, and, occasionally, syncope. The effects were more pronounced in women than in men. The subjects reported persistent headaches, anorexia, irritability, depression, and general malaise. These postexposure symptoms were markedly more severe after exposure to heat and CO than after exposure to either alone. It is interesting to note that these were physiological disturbances and that the more severe occurred in the hours following the exposures. No decrement in performance on a battery of psychomotor tests (e.g., tracking tasks) was seen from either of the stresses alone or in combination during the exposure. It would be interesting to test the subjects on the psychomotor tasks at intervals after the exposures.

According to Baetjer,<sup>48</sup> heat also influences the effect of drugs on experimental animals. Certain substances, e.g., coal tar, cresols, create exceptional photosensitivity of the skin. Even a short exposure in the late afternoon when the sun is low is likely to produce severe sunburn.<sup>49</sup> These problems are primarily associated with ultraviolet radiation and so are limited to outdoor workers.

These data indicate the complexity of the interactions of multiple stresses. They raise fundamental questions as to the validity of those TLV's based on single stress experiments for toxic substances in the presence of heat.

Renshaw<sup>50</sup> investigated the effects of **noise** (41, 80, 90, and 100 dBA) and heat (72, 78, 84, and 90°F ET) on performance on a 5-Choice Serial Reaction Task. The effect of heat on "gaps" was statistically significant. Subjects committed 18 percent more gaps at 90°ET than at 72°ET at the same noise level.

It has been a common observation that mortality increases during prolonged hot spells, e.g., St. Louis, 1966.<sup>51</sup> Similarly, the frequency of illnesses seems to be dependent on the heat load. There are numerous instances cited in the literature where increases in dispensary visits, etc., accompany hot weather, even as do accidents (as noted above). Pepler<sup>34</sup> reviewed a number of experiences reported by the several military services of Great Britain. The illnesses, aside from frank heat illnesses, ran the gamut of nonspecific complaints, general malaise, and even psychoneurotic illnesses.

Bannister<sup>52</sup> observed that injection of a bacterial pyrogen caused a sudden cessation of sweating lasting upwards of an hour. This suggests that concurrent infections may predispose an individual to greater sensitivity to heat stress.

Heat alters the number of free alveolar macrophages in rats.<sup>48</sup> While somewhat afield from the problem under review here, the implications are that there are many subtle little understood physiological adjustments to heat stress whose role in rendering a worker on hot jobs more or less resistant to bacterial invasion is unknown.

#### Correlation of Exposure and Effects

The physiological and medical consequences of exposure to heat are not directly proportional to the intensity throughout the entire range of heat stress. Over a rather large range of temperatures, physiological functions are independent of the temperature. In the environment driven zone (EDZ See Part V) the physiological strain increases exponentially so that at high levels of heat stress a small incremental increase in stress

results in a large increase in strain. The safety factor becomes progressively smaller as the total heat-work stress is increased. Consequently, as the heat stress becomes higher, more care and precaution must be exercised to insure the health and safety of the worker.

Many factors, which can exist in limitless combinations, interact to determine relationships between exposure and effects. The more important of these factors include the Environmental Factors, the Human Factors, and the Task Factors (Table I). The impact of some of these factors on performance and heat tolerance has recently been reviewed.<sup>33</sup> It is emphasized that for any specific environment-worker-job situation the total stress and health and safety consequences can be brought to acceptable and desirable levels by adequate control of one or more of the factors.

One of the most dramatic and successful physiological mechanisms possessed by man is his ability to increase his tolerance to work in heat. The physiological and psychological processes involved in acclimatization to heat have been described in many technical papers and several comprehensive reviews.<sup>16,53,54</sup> Acclimatization to heat is a series of physiological adjustments that occur when one who is accustomed to working in a temperate environment is suddenly placed in a hot environment. These physiological adjustments which occur over a period of one to two weeks reduce the strain experienced on the initial exposure to heat. The physiological changes during acclimatization which are most easily observed are the responses of the body temperature and pulse rate, both of which increase during the first day of heat exposure and then progressively decrease with each

succeeding day of exposure. The sequence is shown in Figure 2. On first day of exposure to heat, ability to perform muscular work is impaired, body temperature and pulse rates are increased and lassitude and discomfort is experienced. When the conditions are extremely severe, acute heat disorders may occur. After the major part of acclimatization has taken place, work in the heat can be performed with little strain and a major reduction in distress. The exposure-effects relationships, therefore, are strongly dependent on the state of acclimatization of the individual.

### Medical Considerations

The three major clinical disorders resulting from excessive heat stress on susceptible workers are: (1) heat stroke, from failure of the thermoregulatory center; (2) heat exhaustion, from depletion of body water and/or salt; (3) heat cramps, from salt loss and dilution of tissue fluid.

Other clinical entities from heat effects are heat syncope, heat rash, anhidrotic heat exhaustion, heat fatigue-transient, and heat fatigue-chronic.<sup>16</sup> (See also Figure 3.)

#### 1. Heat Stroke

a. Diagnostic criteria: Heat stroke (the term sunstroke is obsolete) is the most serious of the heat disorders, constituting a medical emergency of major magnitude.

The three cardinal signs of heat stroke are: (a) hot dry skin: red, mottled, or cyanotic; (b) hyperthermia: a body temperature usually of 106°F or higher and rising; (c) brain disorders: mental confusion, delirium, loss of consciousness, convulsions, and coma.

b. Treatment: Heat stroke is uniformly fatal unless treated promptly and adequately. Treatment consists in rapid cooling of the body preferably by immersion in chilled water accompanied by vigorous massage of the skin or alternatively by wrapping the unclothed body of the patient in wet sheets and fanning vigorously with cool dry air. First aid treatment of the victim should always be initiated immediately and not delayed while waiting for transportation to a medical facility. First aid consists in moving the patient to a cool area and thoroughly soaking the clothing with cold water and fanning to increase convective cooling. Definitive medical treatment required rapid cooling until body temperature is reduced to 100-102°F, then monitoring body temperature to avoid overcooling and to detect recurrent rise, and treating shock if present. Major complications are renal failure, hepatic failure, hemorrhagic disorders, and myocardial impairment. These complications as well as the permanent brain injury which is a frequent sequela are in part consequences of prolonged and uncontrolled hyperthermia and in part the result of tissue hypoxia when shock supervenes. These complications can be avoided by prompt and effective emergency treatment.

In four groupings of cases surviving long enough to be admitted to a hospital for treatment and reported in the medical literature, the mortality rate increased in direct ratio with the increased temperature on admission (Minard and Copman, 1963):<sup>56</sup>

Admission Temperature (°F)	No. of Cases	Mortality Rate (%)
106	188	14.9
106-108	122	20.5
108-110	155	34.2
110 or over	118	61.0

Because thermal injury to vital tissues, particularly the brain, is a rate limited process depending both on degree of temperature elevation and time, injury can occur even at relatively low body temperature; e.g., 105°F if hyperthermia is prolonged. By the same token, survival with complete recovery is possible at extreme hyperthermia; e. g., 108 or above, if cooling is prompt and effective.

Malamud, Haymaker, and Custer<sup>55</sup> who described a wide range of pre-mortem brain disorders in 125 fatal cases of heat stroke occurring in military trainees in World War II, state that "damage to the central nervous system was manifest from the onset and persisted to the end. In cases of longer duration, dementia, aphasia, or hemiplegia indicated that the effect on the central nervous system was probably lasting and irreversible. A direct relationship between the nervous manifestations and the degree and duration of hyperthermia was always evident."

Early recognition and treatment of heat stroke can prevent both death and permanent brain damage. Between 1956 and 1960, twenty-one cases of heat stroke in the Marine Corps recruits were admitted to the dispensary at the Marine Corps Recruit Depot, Paris Island, South Carolina, with



rectal temperatures ranging from 105.5 to 109.6°F (mean 107.1°F, S. D. 1.08°F). All recovered. Eighteen completed both recruit and advanced training. Three were medically discharged. Of these, one who had presented a long history of heat intolerance dating from childhood recovered with no sequelae. In the other two, the clinical course of recovery had been complicated by acute renal failure. Although renal function in both eventually returned to normal, it was the opinion of the physical evaluation board that the risk of recurrent renal disease would be less incivilian life.

c. **Underlying mechanisms:** Hyperthermia in heat stroke results from suppression of sweating, which may be gradual or abrupt in onset. Failure of the principal mechanism for dissipation of body heat under heat stress, i.e., cooling by sweat evaporation, leads to storage of body heat, the rise in body temperature being more rapid in individuals whose heat production is elevated during work. The upward spiral of body temperature is accelerated by the  $Q_{10}$  effect, the rate of metabolic heat produced in tissue cells being increased between 2 and 3X for each 10°C rise in temperature. Why the central thermoregulatory drive for sweating fails is not known; a reduced response, or "fatigue" of the sweat glands, to the central drive may be a contributing factor.

Heat hyperpyrexia is a term sometimes applied to cases of thermoregulatory disorder in which body temperature is elevated to 105 or 106°F, but sweating is still evident and disorders of consciousness are mild or absent. These cases of hyperthermia may represent early stages of heat stroke or a transitional stage between milder heat disorders, such as heat exhaustion, and heat stroke. Treatment by active cooling is indicated unless rest in a cool area leads to immediate and positive signs of recovery.

d. Predisposing factors: In industrial workers and military trainees, the primary underlying factor in heat stroke is lack of acclimatization often associated with poor physical fitness and/or obesity. Precipitating factors are prolonged exertion under heat stress with inadequate time allowances for rest and recovery.<sup>57</sup> Recent alcoholic overindulgence in otherwise seasoned workers has been identified as a probable factor in some cases.<sup>58</sup> In elderly individuals living in poorly ventilated housing, the risk of heat stroke during prolonged heat waves in northern cities is greatest in those with a history of chronic cardiovascular or cardiorespiratory disease.<sup>51</sup> In such cases impaired circulatory capacity to transport heat from body core to the skin is the underlying cause of hyperthermia and thermoregulatory failure rather than elevated metabolic heat production during work.

## 2. Heat Exhaustion

a. Diagnostic criteria: The diagnostic term "heat exhaustion" encompasses disorders which may vary in etiology, but manifest similar clinical signs and symptoms. These are chiefly weakness or extreme fatigue, giddiness, nausea, and headache in persons working in the heat, or often while resting between bouts of work. The skin is clammy and moist, indicating that sweating remains active. The complexion may be pale, muddy, or flushed. Oral temperature may be normal or low, but rectal temperature is usually elevated (99.5 to 101°F). If sitting, the patient may faint on standing with a weak thready pulse and low blood pressure. The underlying disorder in heat exhaustion is depletion of body water due either to restricted water intake, or to deficient salt intake, or more often to both. In the water restriction type, urine is highly concentrated

and small in volume; thirst is a prominent symptom. In the salt deficiency type, circulatory insufficiency is more extreme, urine is more dilute, larger in volume, but chlorides are absent ( $< 1$  gm/liter). Thirst is less evident. Blood electrolytes may be slightly elevated with hemoconcentration in the water restriction type and somewhat below normal in the salt deficiency type. Laboratory facilities are often not available to differentiate the two types. Results of blood analysis, however, are not essential in making a diagnosis of heat exhaustion as this can be determined on the basis of the clinical signs and symptoms noted above.

b. Treatment: Treatment is based on correcting dehydration which is the underlying disorder common to both types of heat exhaustion. Many mild cases recover spontaneously following rest in a cool area and taking water. The severe case of heat exhaustion should be removed to a treatment facility. Dehydration is corrected by administering salted fluids by mouth. If the patient is unconscious or vomiting, normal saline is infused intravenously. He should be kept at rest until the urine volume and salt content indicate that salt and water balances have been restored. Recovery is complete and usually rapid except in cases of extreme salt depletion in which several days of treatment may be required.

c. Underlying mechanism: Physiological control mechanisms involving the hypothalamus, the posterior pituitary gland, the adrenal cortex and volume receptors in the vascular system and the kidney regulate the osmolarity and volume of extracellular fluid. With restricted water intake combined with losses of water and salt in the hypotonic sweat, extracellular fluid tends to become hypertonic. Excess salt is excreted via the kidney with maximum reabsorption of water through mediation of ADH.

Osmolarity is maintained but at the cost of reduced volume of extra and intracellular fluid. In salt deficiency with continued intake of water, extracellular fluids tend to be diluted. Osmolarity is maintained by reduced renal reabsorption of water and retention of salt through release of aldosterone, acting both on the kidney and on the sweat glands. Intracellular fluid volume may increase. These compensatory mechanisms lead to dehydration from negative water balance. The effect is to reduce circulating blood volume.

In both types of dehydration, there is a contraction of circulating blood volume, more marked in the salt deficiency type. Thus, under heat stress, circulatory insufficiency results from the competing demands for blood flow to the skin to dissipate heat and for blood flow to the active muscles, with consequent weakness, hypotension, and syncopal symptoms.

d. Predisposing factors: In unacclimatized men working in the heat, salt concentrations in sweat tend to be high. Dietary intake of salt, particularly if heat strain results in impaired appetite, may be inadequate to balance losses in the sweat. Drinking salted water (0.1 percent) is the best method for supplementing salt intake. Also, lack of acclimatization in men losing up to several liters of sweat per day often leads to voluntary dehydration, a term indicating that the thirst mechanism fails to provide an adequate stimulus to drink water in sufficient quantities to balance the losses in sweat. Workers should be instructed to drink more than necessary to satisfy thirst. Failure of supervisors to provide ready access to water, or to provide breaks at frequent intervals, may lead to

degrees of dehydration which cannot readily be compensated because the volume of water necessary to be ingested causes gastric distention and distress.

### 3. Heat Cramps

a. Diagnostic criteria: Heat cramps is a heat disorder characterized by painful spasms in skeletal muscles of workers who sweat profusely in the heat and drink large volumes of water without replacing salt losses. The muscles involved may be in the arms, legs, or abdomen, those used in performing the job being chiefly affected. Onset may be during or after work hours.

b. Treatment: Salted liquids may be given by mouth or hypertonic saline infused intravenously for more immediate relief.

c. Underlying mechanisms: Water intake with continuing salt loss in sweat leads to dilution of the extracellular fluid. Osmotic transfer of water into active muscle fibers causes spasm. Fatigued muscles are the most vulnerable.

d. Predisposing factors: Water intoxication of this type may be observed in seasoned workers as well as in unacclimatized new employees. Prevention is by instructing workers to use more salt at meal times, or by providing 0.1 percent salt in drinking water during work. Salt tablets as a supplement are less desirable because of individual intolerance to solid salt and possible excessive salt loading. Salt should never be taken during hot work unless ample water is also available.

### 4. Other Clinical Entities

a. Heat syncope: A minor disorder characterized by syncope in unacclimatized workers standing erect and immobile in the heat. Pooling

of blood in dilated vessels of skin and lower part of the body results in inadequate venous return to the heart and cerebral ischemia. Recovery of the patient is prompt when recumbent. Intermittent activity to assist venous return prevents the occurrence.

b. Heat rash: Heat rash, commonly known as prickley heat, results from imbibition of water by keratin and plugging of the orifices of sweat ducts, which leads to inflammation of the glands, and is observed as tiny red raised vesicles in the affected area. It results from unrelieved exposure to humid heat with the skin being continuously wet with unevaporated sweat. It is important because if extensive, or complicated by infection, discomfort from heat rash may not only interfere with restful sleep and impair efficient performance, but can result in temporary total disability. Heat rash is prevented by providing cooled recovery or sleeping quarters to allow the skin to dry between heat exposures.

c. Anhidrotic heat exhaustion: Rarely seen in peacetime, this disorder was observed in military personnel stationed in hot climates in World War II and was characterized by areas of nonsweating skin on the trunk, and limbs which showed a papilliform eruption, like gooseflesh, on heat exposure. This was termed miliaria profunda and represented obstruction of sweat gland ducts deep in the skin. Hyperhidrosis of the face was a characteristic finding. If the nonsweating areas were extensive, impaired evaporative cooling led to heat intolerance with symptoms of heat exhaustion and moderate hyperthermia. There was usually a history of extensive heat rash, with occasional further skin trauma by sunburn.

Associated with the skin disorder and heat intolerance was polyuria, a high chloride concentration in sweat, and a lowered blood chloride. There was no specific treatment but return to cooler climates led to gradual recovery. An extensive review of the etiology of this disorder and miliaria rubra as well as the possible role of endocrine or other systemic factors has been published. 59

d. Heat fatigue - transient: This term applies to the impairment in performance of complex sensorimotor, mental, or vigilance tasks on exposure to heat. The decrement in task performance produced by heat exposure is greater in unacclimatized and unskilled workers. Discomfort and physiological strain rather than physiological failure of regulatory mechanisms are the major underlying factors. Acclimatization and training for work in the heat reduce the degree of impairment.

e. Heat fatigue - chronic: Formerly termed tropical fatigue, this designates a long term impairment in work performance and social behavior in workers and military personnel transferred from temperate home environments for long residence in tropical latitudes. Factors of boredom and isolation from the customary social environment interact with the physiological strain imposed by unremitting climatic heat and humidity to cause psychological strain and behavioral disorders, including lack of motivation for work, lowered standards of social conduct (e.g., alcoholic overindulgence), inability to concentrate, etc. Prevention is based on selection of personnel for such assignment and on their prior orientation to life abroad (customs, climate, living conditions, recreational opportunities).

The objectives of a preventive program are to prevent clinical disorders from heat stress and also to prevent aggravation of existing impairments by heat and to maintain optimum health and work efficiency. The objectives can be accomplished through the following procedures: pre-placement and periodic medical examination, acclimatization of workers to heat, and monitoring of oral temperature and heart rate.