5.—HEAT DISORDERS: A TABULAR PRESENTATION

D. Minard

TABLE 5–1.—Classification of heat disorders

I.C.D. Code	Clinical designation	Etiologic category
992.0	Heat stroke	Thermoregulatory failure.
—	Heat hyperpyrexia	
992.1	Heat syncope	Orthostatic hypotension.
992.2	Heat cramps	Salt and water imbalance.
992.3	Heat exhaustion, water depletion	
992.4	Heat exhaustion, salt depletion	
992.5	Heat exhaustion, unspecified	
992.6	Heat fatigue, transient	Behavioral disorder.
—	Heat fatigue, chronic	
705.1	Heat rash	Skin disorders and sweat gland injury.
—	Anhidrotic heat exhaustion	

TABLE 5–2.—Heat stroke (HS) and heat hyperpyrexia (HH)

Predisposing factors

Lack of acclimatization
Lack of physical fitness
} + exposure to environmental heat.

Sustained exertion under environmental heat (essential factor in HH).

Individual susceptibility, manifested by: abnormal tolerance to hyperthermia.

Dehydration — salt deficiency (or excess intake leading to K^+ depletion).

Pre-existing acute diarrhea or febrile disease.

Chronic degenerative disease in the elderly.

Therapeutic drugs, e.g. anticholinergics, diuretics, etc.

Drug abuse, e.g. alcoholism, amphetamines.

TABLE 5–3.—Signs and symptoms HS and HH

Heat stroke	Heat hyperpyrexia
Skin	
Dry, hot (absence of sweating).	Moist, hot (sweating persists).
Body temperature	
Hyperthermia, usually over 105 F and rising.	103 to 108 F*.
Cerebral dysfunction	
Bizarre behavior, delerium, coma, convulsions.	Same.

*Shibolet, S., R. Coll, T. Gilat, and E. Sohar. Heatstroke: Its clinical picture and mechanism in 36 cases. *Quart. J. Med., New Series* 36: 525–548, 1967.

TABLE 5–4.—Underlying thermoregulatory Disorder HS and HH

Heat Stroke: (Basic Physiologic Disorder of Thermoregulatory Center remains unknown) Excessive heat load leads to failure of central drive for sweating. Loss of capacity for evaporative cooling leads to self sustaining and accelerating rise in body temperature during continued exposure to environmental heat.

Heat Hyperpyrexia: Rate of blood flow to skin during sustained exercise at elevated ambient temperature is insufficient to meet demand for transfer of metabolic heat to the body surface. Heat storage and hyperthermia result.

In both HS and HH, victims are individuals with abnormal tolerance for hyperthermia.

TABLE 5–5.—Heat stroke and heat hyperpyrexia treatment

TREATMENT:

Cooling, *immediate* and *active* by any available means. *Delay leads to permanent thermal injury of the brain and complications in other systems.*

COMPLICATIONS:

A.) Shock with resulting tissue anoxia and metabolic acidosis.

B.) Disseminated Intravascular Coagulation (DIC) with resulting hemorrhagic diathesis.

A and B add to thermal injury of kidney, liver, heart as well as the brain.

SEQUELLAE:

Immediate and effective cooling leads to complete recovery. Delayed or inadequate cooling results in permanent thermal injury to brain (Cerebellar syndrome most frequent) and to added injury to kidney, liver, heart and other tissues resulting from complications.

TABLE 5–6.—Heat exhaustion (salt and/or water depletion)

Predisposing factors
Lack of acclimatization and physical fitness.
Lack of indoctrination in hot weather hygiene.
Failure to replace salt and/or water lost in sweat.
Lack of work pauses of sufficient frequency and/or duration resulting in accumulative heat strain.
Improper clothing, interfering with body heat loss or failing to protect against radiant heat.
Pre-existing diarrhea, gastroenteritis and vomiting, or acute febrile illness (e.g. from immunizing innoculations).

TABLE 5–7.—Heat exhaustion symptoms

Symptoms
Weakness, lassitude, dizziness, visual disturbances.
Feelings of intense heat and thirst in water depletion syndrome. Thirst seldom prominent in salt depletion syndrome.
Headache.
Nausea, vomiting, diarrhea, muscle cramps (abdomen and/or extremities).
Breathlessness and palpitations.
Tingling and numbness of extremities (often associated with hyperventilation).

TABLE 5–8.—Heat exhaustion signs

Signs
Skin: Pale and clammy or warm, flushed, and moist. (Active sweating is present) Loss of skin turgor in moderate to extreme dehydration.
Body Temperature: Oral T is normal or low. Rectal T is normal or slightly elevated (100-101 F).
Circulatory System: Pulse is rapid and thready. Blood pressure usually normal if patient recumbent. Pulse pressure reduced on standing which may precipitate orthostatic hypotension and syncope.
Respiration: Hyperventilation if present may be associated with hypocapneic alkalosis and tetany.

TABLE 5–9.—Heat exhaustion underlying physiological disorder

Disorder
Water-Depletion Type: Failure to replace body water lost in sweating leads to dehydration from *hypertonic* contraction of the Extra Cellular Fluid compartment and reduction in plasma volume. *Anti-diuretic hormone* (ADH) is released as a compensatory response to conserve body water.
Salt-Depletion Type: Failure to replace body salt lost in sweating leads to dehydration from *hypotonic* contraction of the ECF compartment and reduction in plasma volume. Aldosterone is released as a compensatory hormonal response to conserve body salt.

TABLE 5–10.—Heat exhaustion, laboratory findings and treatment

Test	Water depletion type	Salt depletion type
Urine	Highly concentrated chlorides present	Less concentrated chlorides absent (<1 gm./1)
Serum Electrolytes	Na and Cl normal or above average. Hemoconcentration absent or slightly increased	Na and Cl usually below average. Degree of hemoconcentration greater than in water depletion type.
	Treatment	
Treatment	Remove to cool area. Rest with administration of saline and salted liquids by mouth. I-V infusion of normal saline if vomiting or unconscious.	

TABLE 5–11.—Heat cramps

Predisposing Factors: Heavy sweating and ingestion of water without replacing salt lost in sweat. May occur in seasoned workers.
Underlying Physiological Disorder: Absorption of ingested water dilutes interstitial fluid lowering its osmolarity. Water diffuses into active muscle cells, causing hyperirritability and spasm.
Signs and Symptoms: Acutely painful and often incapacitating cramps in muscles used during day's work. Other signs and symptoms of salt depletion syndrome largely absent.
Treatment: Ingestion or I-V infusion of normal saline. More rapid relief by I-V infusion of hypertonic saline.

TABLE 5–12.—Prevention of heat disorders

1. Selection and periodic health evaluation.
2. Acclimatization: Graduated increase in physical activity in the heat during first 4 to 10 days on the job.
3. Properly spaced intervals for rest in cooler recovery area. Add additional workers if pace is set by job.
4. Ensure ample and easily available supply of drinking water. Up to 2 gallons per 8 hour shift.
5. Judicious use of supplemental salt during acclimatization.
6. Avoid indiscriminate intake of NaCl and resulting K^+ depletion.
7. Adequate NaCl and K^+ intake at meal time.
8. Restful sleep in quarters cooled by natural ventilation or cooled mechanically.
9. Proper clothing, including protective clothing if needed.
10. Engineering control of heat stress at hot work locations.
11. Indoctrinate supervisors and workers in hot weather hygiene.

STANDARDS FOR OCCUPATIONAL EXPOSURES

TO HOT ENVIRONMENTS

PROCEEDINGS OF SYMPOSIUM

February 27–28, 1973

Pittsburgh, Pennsylvania

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Sponsored by
Industrial Health Foundation
and
Graduate School of Public Health
University of Pittsburgh

Supported by
NIOSH Grant No. 1 R 13 OH 00382–01 SOH

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
Division of Biomedical and Behavioral Science

Cincinnati, Ohio 45202

January 1976

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402

HEW Publication Number (NIOSH) — 76-100