NASA Technical Memorandum 101011

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April 1989

NASA

National Aeronautics and
Space Administration

Ames Research Center
Moffett Field, California 94035

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SUMMARY

This review concerns the responses and mechanisms of both natural and artificial acclimatization to a hot environment in mammals with specific reference to humans. The purpose is to provide basic information for designers of thermal protection systems and countermeasures for astronauts during intra- and extravehicular activity. Areas covered are energy metabolism, thermal balance at rest and during exercise, water and electrolyte balance during exercise and immobilization, and heat diseases.

THERMAL BALANCE AND ITS CONTROL—GENERAL APPROACH

A major evolutionary advance was made when mammals began regulating their internal body temperature essentially independently of the environmental temperature, provided the temperature extremes were not too great. This independent regulation in homeotherms allowed for more freedom of movement and, as a result, greater survival potential. Because of their large potential capacity for dissipation of heat by means of sweat evaporation, humans are unique among the mammals. Since humans have a much greater range of physiological adaptive responses to a hot compared to a cold environment, Macpherson (1958) believed that temperate-zone man was and continues to be, basically, a tropical animal. Except for periods of intense physical activity, the human's large capacity for heat loss through massive cutaneous vasodilation and evaporation of eccrine sweat would be of little use in a cold environment where well-developed mechanisms for heat conservation would be paramount. Macpherson (1958) suggested that "a temperate climate does not represent the neutral condition in which environmental stress is minimum and from which adaptation is possible both to hotter and to colder conditions to an equal degree. On the contrary, man in a temperate climate is approaching the extreme range of adaptation to cooler conditions —that is, maximum adaptation to cold. It follows that it is fruitless to attempt to demonstrate profound physiological changes in temperate man on exposure to severe cold because, the greater part of the possible adaptation in this direction having already been made in a temperate climate, any further changes must necessarily be small. This hypothesis assumes added weight when pronounced by a Londoner.

The body temperature is an abstract concept; it is the instantaneous integration of an infinite number of temperatures within each cell in the body. Claude Bernard's concept of the constancy of the internal environment ("fixité du milieu interne") must be applied to thermoregulation with caution, except when the body is in the basal or resting states in a temperate environment. Bazett has emphasized that skin/blood temperature can vary by 20°C and exercising skeletal muscle temperatures vary by 10°C: "Mammalian physiologists have been so fooled by a fictitious belief in the constancy of the temperature of the whole body that little work has been accomplished on temperature as a variable factor" (Bazett, 1949).

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The most elementary concept of the temperature distribution is to consider the body as a two-phase system (core-skin): a warmer core where heat was produced and the temperature (37°C) remained relatively constant, and a cooler shell (34°C) consisting of skin and ancillary subcutaneous tissue, whose temperature was more variable. About 24% (17 kg) of body tissue lay within 1 cm and 54% (38 kg) is within 2.5 cm of the skin surface (DuBois, 1951). While this two-phase concept can be used for the resting subject, it cannot be used with any degree of accuracy when core temperature changes appreciably; for example, during exercise and/or heat exposure. For these latter situations three-phase (core-fat-skin) and four-phase (viscera and internal muscles – external muscle – subcutaneous fat and tissues – skin) systems have been proposed (Houdas, 1975).

The extreme limits of core temperatures compatible with life appear to fall above 24°C and below 45.6°C (DuBois, 1951). At rest, death usually occurs when core temperature falls below 27°C (during immersion in cold water) and when it exceeds 42°C (Gagge and Herrington, 1947). Unacclimatized subjects at rest in a hot and humid environment (42°C, 90% rh) with a rectal temperature of only 38.5°C are near their limit of tolerance. The average lower limit of core temperature for the onset of heatstroke is 41-42°C (Leithead and Lind, 1964; Shibolet, 1976), but a few cases have been reported with core temperatures between 40-41°C (Leithead and Lind, 1964). At the end of hard endurance exercise, routine rectal temperatures of 40-41°C cause no lasting adverse effects (Pugh, 1967; Robinson, 1963). Thus, the absolute level of core temperature alone cannot be used to determine the physiological or pathological state of an individual.

If the average normal resting core temperature is assumed to be 37.0°C, then the limits of survival occur with a decrease in body temperature of about 10°C, but with an increase in temperature of only 5°C. The presently designated normal resting core temperature is therefore not set midway between the tolerance limits, but closer to the upper limit. Thus, tolerance to overheating would be expected to be much less than tolerance to overcooling. As a result, the body should have and does have a greater array of heat-dissipating mechanisms compared with heat-conservation mechanisms. These observations would argue against that portion of Macpherson's hypothesis which states that temperate-adapted humans are normally near their limit for cold adaptation. We would appear to be normally adapted closer to our limits for heat adaptation since we react to hyperthermia with greater and more fully developed responses for heat dissipation. These enhanced responses would be appropriate for tropical mammals since they would be responding more frequently to overheating than to overcooling. So the more important question is not so much whether humans are basically tropical mammals, but why the normal resting core temperature is apparently set closer to the upper survival limit. The constant metabolic heat production would dictate efficient heat-dissipating mechanisms; but, in turn, it would seem the resting body temperature should have been set about 2°C lower to allow more range for moderate thermoregulation, for instance during exercise or heat exposure, before severe hyperthermia occurs. During prolonged bed rest (14 days) the basal oral temperature (measured properly) averaged about 35.8°C (Greenleaf, 1980) much closer to the theoretical midsurvival level of about 35°C. The resting temperature of skin and muscle, which comprise about 52% of the body weight, is 34.0-36.0°C (table 1). If the normal basal core temperature is considered instead of the normal resting (sitting) temperature, then normal body temperature is "set" approximately midway between the lethal limits.

Heat is transferred to and from the body—according to the laws of thermodynamics—by conduction, convection, radiation, and evaporation of water. The temperature of the body (i.e., the heat content or storage) is the net result of heat production and heat dissipation. Heat can be gained as a "by-product" of cellular metabolism which varies with the level of exercise (muscular contraction), from shivering thermogenesis, from the chemically stimulated nonshivering thermogenesis, by injection or ingestion of hot food or fluids, by breathing hot air or other gases, by diathermy and by conduction, convection, and

radiation from the environment. Heat can be lost in excreta, expired air, by conduction, convection, radiation, and by evaporation of sweat—the latter being the most important and efficient avenue of loss during exercise. Heat is transferred within the body mainly by conduction through the cardiovascular system. Control of vasodilatation, vasoconstriction, and the volume and distribution of blood are very important factors for thermoregulation; these aspects will be discussed later.

Gross heat exchange between the body and its environment can be expressed by the heat-balance equation:

$$M \pm Cv \pm Cd \pm R - E = \pm S$$

where M is the level of metabolic heat production; $\pm Cv \pm Cd \pm R$ is the amount heat gained or lost by convection, conduction, and radiation, respectively; E is the amount of heat lost by evaporation of water, including respiratory evaporative heat loss; and S (storage) is the amount of heat gained or lost by the body tissues.

Similarly, conductive heat exchange (Cd) occurs when a material touches the skin.

$$Cd = \frac{KA(T_2 - T_1)t}{d} \text{ in kcal/m}^2\text{/hr}$$

where K is the thermal conductivity of the material touching the skin; A is the area of contact (cm^2); T_2 and T_1 are the temperatures of the warmer and cooler surfaces, respectively (°C); t is time (sec); and d is the thickness of the conductor (cm).

Metabolic heat production, measured indirectly from expired air composition, ranges from the basal metabolic rate of 60 to 80 kcal/hr to that expended during short-term maximal exercise using large muscle groups of about 1800 kcal/hr in champion athletes. Estimated heat production of body tissues in a 63-kg man at rest is presented in table 1. Body heat is derived from the oxidation of substrates. The efficiency of this process is about 23%, so one quarter of the energy is converted into compounds with high-energy phosphate bonds (adenosine triphosphate guanosine triphosphate (ATP GTP), creatine-phosphate) and about 75% is converted to heat. Heat production can be increased by oxidizing more substrate, as with exercise, by decreasing the amount of energy (ATP) converted to phosphate bonds (uncoupling of respiration), or by increasing the degradation of ATP. The latter can be induced by direct hydrolysis of high-energy phosphate bonds by the action of Na^+/K^+-ATPase, by fatty acid breakdown and resynthesis, and by hydrolysis of the high-energy bond in creatine-phosphate during muscle contraction (Steiner, 1973). Christensen (1953) has categorized working intensities as follows: light work—up to 150 kcal/hr, moderately heavy—150 to 300 kcal/hr, heavy—300 to 450 kcal/hr, very heavy—over 600 kcal/hr.

Heat is exchanged by convection (Cv) depending upon the difference between air and skin temperatures. Skin temperature lower than air temperature results in heat gain, and vice versa. For nude men, the following equation approximates heat exchange (Nelson, 1947):

$$Cv = 0.5\sqrt{V(\overline{T}sk - Tdb)} \text{ in kcal/m}^2\text{/hr}$$

where V is air velocity (ft/min), $\overline{T}$ is mean skin temperature (°C), and Tdb is ambient dry-bulb temperature (°C).

Radiation heat exchange occurs with a temperature difference between solid surroundings or the sky and the skin.

$$R = 5.7(\overline{T}sk - Tg) \text{ in kcal/m}^2\text{/hr}$$

where $\overline{T}sk$ is skin temperature (°C), and Tg is black globe temperature (°C).

Evaporative heat loss (E) from sweating occurs when the vapor pressure of sweat on the skin is higher than the vapor pressure of the surrounding air. Evaporation of 1 liter of sweat removes about 600 kcal of heat from the body. Accurate estimation of evaporative heat loss requires knowledge of percent of skin wetness, a difficult measurement. Sweat that runs off the skin is not evaporated and removes no heat. Fully wetted skin loses heat by evaporation as follows (Clifford, 1959):

$$E = 0.45^{V0.63} \text{ (Psk} - \text{Pa) in kcal/m}^2\text{/hr}$$

where V is air velocity (ft/min), Psk is skin vapor pressure (mmHg), and Pa is air vapor pressure (mmHg).

Body heat storage (S) is greater with an increase in body temperature. When a quantity of heat is "stored" in a tissue, that amount of heat is no longer exchangeable and the associated temperature rise must be represented as a negative number, that is, a negative S. The concept of heat storage is applicable only when there are no rapid or large changes in body temperature. In a cool environment S is best approximated by the following equation (Burton, 1935):

$$S = 0.83W \text{ (0.65 Tre} + 0.35 \overline{T}sk) \text{ in kcal/m}^2\text{/hr}$$

where 0.83 is average specific heat of body tissues, W is body weight (kg), Tre is rectal temperature (°C), and T is mean skin temperature (°C). This equation adequately estimates heat exchange of subjects in a stirred water bath (Burton and Bazett, 1936) as well as in lightly clothed subjects lying on a bed at Tdb = 23.3°C (Burton, 1935). In cooler environments the body core tissue component would be smaller and the peripheral tissue component larger caused by peripheral vasoconstriction; hence, the relatively smaller coefficient for core temperature and the larger coefficient for the skin temperature component. In warmer environments (Tdb = 22 to 35°C) S is best approximated by (Hardy and DuBois, 1938):

$$S = 0.83W \text{ (0.8 Tre} + 0.2 \overline{T}sk) \text{ in kcal/m}^2\text{/hr}$$

where 0.83 is average specific heat of body tissues, W is body weight (kg), Tre is rectal temperature (°C), and $\overline{T}sk$ is mean skin temperature (°C). The larger rectal temperature coefficient here reflects a greater amount of heat from the core in the periphery caused by vasodilation.

Since the body is about 70% water, it would seem that thermoregulation would depend, at least in part, upon the thermodynamic and other physical properties of water. Some of these properties are its dielectric constant and related ionizing potential, high thermal conductivity which facilitates equilibration of temperature between and within cells, high latent heat of evaporation, its power as a solvent, and its high specific heat which facilitates transfer of large quantities of heat with relatively small changes in tissue temperature (Henderson, 1958).

Calloway (1976) has extended Henderson's thesis and suggests that the level of resting body temperature in homeotherms is set between 36 and 40°C as a result of the peculiar thermodynamic properties

of water. The temperature at which the equilibrium constant of a chemical reaction has half the value between the biokinetic range of 0-100°C (the range of least thermodynamic stress) is 40°C. The reaction rate at 40°C (313.2 K) is about half the difference between the value of the equilibrium constant at 0°C (273.2 K) and 100°C (373.2 K). Since these calculations are based upon logarithmic functions, the values obtained are related to e, the base of the natural logarithms. Equations used to calculate thermodynamic responses of aqueous physical-chemical systems (e.g., electrode potentials, equilibrium constants, free energy, ionization constants, neutral membrane-liquid junction potentials, osmosis, surface tension-vapor pressure junctions, and viscosity) all contain the variable of temperature (T) and the basic expression e to the $1/T$ power. In man, the normal resting temperature appears to be a close function of many thermodynamic properties of water as related through e. The difference between the boiling point (373.2 K) and freezing point (273.2 K) divided by e (2.71828) equals 36.8, the normal resting body temperature. Between 0 and 100°C the temperature of the lowest specific heat of water (where the least amount of heat is expended to raise 1 g of water 1°) is 35-36°C, the basal body temperature. Other properties of water which are probably important for optimal enzyme function, namely thermal expansivity and compressibility, have their least thermodynamic stress at 38.5 to 40°C, respectively, the equilibrium body temperature range during moderate to intensive physical exercise. Calloway (1976) concludes, "There is little evidence that temperature is fixed because of the nature of the cell contents other than water and perhaps some of its inorganic solutes." Thus, there is a physical-chemical basis for determining basal body temperature, its true set point, and also why body temperature rises during exercise.

If it seems reasonable to accept the premise that the physical-chemical properties of water contribute substantially to the setting of normal basal body temperature, we should then consider the factors regulating temperature above and below this basal set point. The anatomy of the thermoregulatory system in man has not been defined precisely. It involves integration of responses of the nervous system, the vascular-fluid-electrolyte system, and the endocrine system.

Nervous System

Stimuli are sensed by peripheral and by central thermal receptors sensitive to heat and lack of heat (cold) and transmitted to perhaps more than one controller-integrator in the central nervous system (CNS). Efferent signals are activated to effect appropriate thermoregulatory responses; for example vasodilation, sweating, or shivering. Thermoreceptors have been located within the skin, spinal cord, and the septal, thalamic, and hypothalamic regions of the brain. These CNS thermosensitive structures or areas appear to have specific functions for temperature detection and regulation. The pre-optic anterior hypothalamic neurons respond mainly to body heating and instigate heat loss responses; the posterior hypothalamic neurons respond mainly to body cooling and instigate heat conservation responses. The posterior hypothalamus seems to function as an integrator of effector signals and may also act as a thermocontroller where signals emanating for heat production and heat conservation.

The observation that some thermoregulation is still possible when these hypothalamic areas have been destroyed has led Bligh (1973) to propose a two-level process for thermoregulation: a wide-band (coarse) control which is used only when there is a large deviation in body temperature from the normal range of 35-36°C to 40-41°C, and the normal daily range of 1-2°C, which is the narrow-band (fine) control range. The wide-band control is supposed to be activated only in dire circumstances. The persistence of wide-band control of body temperature after ablation of the anterior hypothalamus has led Bligh to hypothesize that the neurons involved with the coarse control responses may be different from those involved with the fine control responses.

Vascular-Fluid-Electrolyte System

Responses of the peripheral vascular system provide the first line of defense against heat stress and cold stress. Exposure to cold stress involves mainly heat conservation responses including increased heat production by shivering and increased thermal insulation by constriction of cutaneous arterioles and veins, which also limits heat conduction from the core to the periphery. Control of fluid redistribution during heat stress is a smoothly coordinated and complicated process that is not fully understood.

Efficiency of the control is increased by physical exercise training in cool and especially hot environments, and decreased by deconditioning due to inactivity (Greenleaf, 1980). Rowell (1983) has prepared the most recent and comprehensive review of the mechanisms of cardiovascular adjustments to thermal stress. They involve the interaction between cutaneous blood flow, cardiac output, central and total blood volumes, and regional blood flow including that in specific organs. Change in posture to the standing position adds an additional load for the resting control system. Further stress occurs with imposition of environmental heat alone and with environmental heat plus metabolic heat from exercise. The final common pathway which needs to be protected is maintenance of an adequate volume and distribution of blood at sufficient pressure to adequately perfuse the brain to maintain consciousness, and to facilitate metabolism in working muscle and other organs. These basic functions are competing with blood being shunted to the periphery for heat dissipation.

Composition of the plasma appears to influence thermoregulation (Greenleaf, 1979). Hypernatremia is associated with increased body temperature and hypercalcemia with decreased temperature. More dramatic results are obtained when sodium and calcium ions are infused into cerebral ventricles or into the hypothalamic region. Myers and colleagues (1970a, 1970b, 1982) have developed an hypothesis suggesting it is the ratio of Na^+/Ca^{2+} ions in the area of the posterior hypothalamus that determines the set point for thermoregulation. This hypothesis and others relating to the influence of fluid and electrolyte changes on thermoregulation will be discussed more fully later.

Endocrine System

Understanding the interaction of the neural and endocrine systems, if they may be categorized as systems, is a requisite for understanding the mechanism of thermoregulation. Gale (1973) has stated that all hormones except the parathyroid have known effects on thermoregulation. Different types of hormones appear to act with different time-constants similar to the dual time-course of thermoregulatory responses proposed by Carlson (1973): a fast-reacting neural net response measured in seconds, and a slower reacting neurohumoral response measured in hours. Hormones which respond quickly include calcitonin, epinephrine, gastrin, glucagon, insulin, secretin, and some hormones located in the anterior pituitary, posterior pituitary, and hypothalamus; slower acting hormones include the steroids, the thyroid hormones, and growth hormone (Sellers, 1973). Many of these and other hormones that are directly involved with energy utilization and heat production probably affect body temperature to a greater degree than those associated with functioning of specific systems, for example the reproductive system. Most research concerning hormonal involvement with temperature regulation has been directed at responses to cold. The role various endocrine systems play in the mechanism of thermoregulation to heat exposure, especially during exercise, has not been a popular area for study; much additional work needs to be done. These latter aspects will be discussed later. "Hormones can be regarded as component parts of the elaborate communication system which makes it possible for the body to maintain homeostasis. The system includes direct communication (with feedback controls) from a single cell to its environment, from

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cell to cell within tissues, from groups of cells to cells in other tissues, and also includes the nervous system, which is able to rapidly integrate the visceral and somatic functions of the body as a whole. The role of particular molecules transmitting information (hormones) must be regarded as part of the communication system rather than as entities separate from it." (Sellers, 1973).

Heat illnesses have been fully described and characterized by Rothman (1954). The World Health Organization classification of heat illnesses has been modified slightly by Weiner and Horne (1958) and comprises nine categories; heatstroke, heat hyperpyrexia, heat cramps, heat exhaustion, sunburn, prickly heat, anhidrosis, heat neurotic reactions, and heat edema. Leithead and Lind (1964) have condensed these nine categories into three major categories:

1. Disorders resulting from failure of thermoregulation:

   a. <u>Heatstroke</u>. Thermoregulatory failure, usually of sudden onset, following exposure to a hot environment especially during exercise, and is characterized by disturbance of the central nervous system, by generalized cessation of sweating, and by a core temperature above 40.6°C (105°F). It can be fatal. Synonyms: Heat apoplexy, hyperthermia, siriasis, sunstroke, thermoplegia.

   b. <u>Heat hyperpyrexia</u>. The preheatstroke condition where the patient has a core temperature above 40.6C (105°F), but is conscious, rational, and some sweating may be present; not to be confused with exercise hyperthermia.

2. Disorders involved with thermoregulation:

   a. <u>Circulatory instability</u>.

      (i) Heat syncope. Fainting and subjective feeling of giddiness, or acute fatigue resulting from a decrease in blood pressure caused by collapse of vasomotor tone, peripheral venous pooling, and cerebral hypoxia which occurs in the absence of obvious salt or water depletion. Synonyms: Heat collapse, exercise-induced heat exhaustion.

   b. <u>Water-electrolyte imbalance</u>.

      (i) Heat edema. Slight swelling of the extremities particularly the hands, ankles, and feet, usually limited to the first 10-14 days of exposure to a hot-moist environment. Synonyms: Deck ankles, Colombo flop.

      (ii) Water-depletion heat exhaustion. Dehydration caused by inadequate replacement of water losses because of prolonged sweating, characterized by thirst, fatigue, oliguria, hyperpyrexia and, in advanced stages by delirium, syncope, and death. Synonyms: Water-deficiency heat exhaustion, dehydration heat exhaustion.

      (iii) Salt-depletion heat exhaustion. Salt depletion caused by inadequate replacement during prolonged sweating, vomiting, or diarrhea, characterized by fatigue, nausea, vomiting, light-headedness, muscle cramps, and in later stages by circulatory failure. Synonyms: Salt-deficiency heat exhaustion, type-I heat exhaustion.

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(iv) Heat cramps. Characterized by painful spasms of skeletal muscles during or following hard physical work in the heat. Synonyms: Mill cramps, and cane-cutter's, fireman's, miner's, and stoker's cramps.

c. Disorders with skin lesions.

    (i) Prickly heat. Inflammatory skin eruptions and rash accompanied by prickling or tingling occurring during sweating probably caused by blockage of sweat duct orifice. Synonyms: Miliaria rubra, lichen tropicus, heat rash, strophulus.

    (ii) Anhidrotic heat exhaustion. Profound fatigueability, headache, dyspnea, and heat intolerance occurring in people after several months of exposure to hot and usually humid environments, and characterized by the presence of prickly heat or more deep-seated vesicles (miliaria profunda) mainly on the skin of the trunk and proximal parts of the limbs, and by diminished or absence of sweating (anhidrosis) in the area of the miliaria. Synonyms: Tropical anhidrotic asthenia, thermogenic anhidrosis, type-II heat exhaustion, sweat-retention syndrome.

3. Disorders characterized by apathy or fatigue:

    a. Acute heat fatigue. A loss or reduction in skilled performance of many physical and mental tasks. Synonyms: Mild heat asthenia, acute heat neurasthenia.

    b. Chronic heat fatigue. Loss of efficiency of human performance and in meeting external demands with prolonged heat exposure of many months, characterized by increased rate of sickness and skin diseases, continual awareness of the heat, and increased feelings of lassitude and irritability. Synonyms: Tropical neurasthenia, tropical fatigue, heat fatigue.

These categories are not necessarily mutually exclusive since, for example, water- and salt-depletion heat exhaustion often contribute to and are accompanied by heat syncope; and most category 2 disorders contribute to or are forerunners for category 1 disorders.

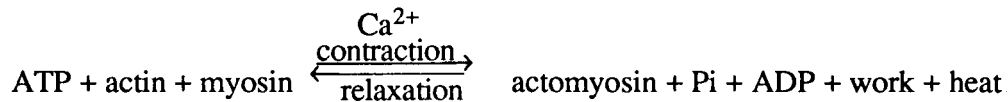## HEAT PRODUCTION AND THERMAL BALANCE DURING EXERCISE

The essence of life in all biological systems involves cellular energy transformation of carbon compounds to form carbon dioxide, water, and the high-energy phosphate compound adenosine triphosphate (ATP). There appear to be three major oxidative enzyme systems for energy transfer that function at progressively longer time intervals of muscular contractions (Brooks, 1984):

Power (0-3) sec): $ATP + H_2O \rightarrow ADP$ (adenosine diphosphate) + Pi (inorganic phosphate)

Speed (4-60 sec): $Creatine - P + ADP \xrightarrow[\text{kinase}]{\text{creatine}} ATP + creatine$

Endurance (>60 sec): $ADP + ADP \xrightarrow[\text{kinase}]{\text{adenylate}} ATP + AMP$

Muscular contraction involves the reaction of contractile proteins (actin and myosin) with ATP:

$$\text{ATP + actin + myosin} \xrightleftharpoons[\text{relaxation}]{\substack{Ca^{2+}\\ \text{contraction}}} \text{actomyosin + Pi + ADP + work + heat}$$

Since the efficiency of human metabolism is about 23%, then 77% of the transformed energy is released in the form of heat. Because the body cannot transform heat into any other form of useful energy, the heat produced must ultimately be dissipated to the environment. The mechanism for this dissipation is a tightly controlled process because of the rather narrow range of body temperature necessary to maintain life. The often quoted reason that a somewhat elevated body temperature results in more efficient work output, because of the doubling of enzymatic reaction rates with each 10°C increase in body temperature (Q_{10} effect), is further strengthened by Calloway's (1976) observations that the most efficient enzyme reactions occur at 38.5°C and 40.0°C. A core temperature of 38.5°C is present at a work intensity of about 55% of the maximal exercise capacity, a level which can be sustained for hours. A core temperature of 40°C is about the upper limit for resting people with a fever before drastic reactions occur; but a temperature of 40°C is normal during prolonged hard exercise (marathon running) when more efficient energy conversion would be appropriate.

When an isolated muscle is shortened and performs work, it produces more heat (energy) than if shortening is prevented. The extra heat produced is approximately equal to the amount of work done. For a given load on the muscle, the extra heat produced increases with increased shortening (the Fenn effect). Heat production merely reflects the speed with which the chemical energy can be activated to do various levels of work.

Under _isometric_ conditions, when a muscle is not allowed to shorten when stimulated, there is an initial liberation of heat before any tension is developed. There is also an additional slower acting heat production beginning when tension develops as the contractile units stretch the series elastic elements in the muscle. With relaxation, the energy stored in the stretched elastic elements is liberated as heat. When a muscle shortens during an _isotonic_ contraction, a constant amount of heat is liberated for each centimeter of shortening. The amount of heat is independent of the load on the muscle, the time and velocity of shortening, muscle temperature, the work done, the hydrostatic pressure, or the pH (Ramsey, 1960). Thus, it is the movement of the muscle (shortening) that produces the larger amount of heat, so body temperature (metabolism) rises more with isotonic than with isometric exercise. Stated another way, the more work done the more heat production; the faster the rate of rise and higher equilibrium levels of core temperature that occur with progressively greater exercise loads reflect this process (Christensen, 1931). Muscle metabolism and heat production are increased when an isolated resting muscle is stretched, but the quantity of heat produced is much greater than the work done on the muscle. When a contracting muscle is reextended during the relaxation phase, the work done in reextension appears as heat which is in addition to that released during contraction. When a muscle is stretched during a contraction phase, the muscle does negative work and total heat production is less than the isometric heat production alone (Ramsey, 1960). There is a negative heat of lengthening (negative work) when compared to the positive heat of shortening (positive work).

The different mechanical forces acting on joints during positive and negative work probably change the number and type of active muscle fibers which, in turn, would vary the quantity of muscle and perhaps tendon that would store and release elastic-strain energy (heat). The tissue that stretches more stores more energy. Active skeletal muscle fibers can be stretched by no more than 3% while tendons can be stretched by 6% before rupture (Flitney, 1978). Most elastic-strain energy will be stored mainly in muscle when the

fibers are longer than the associated tendons, and stored mainly in the tendons when the tendons are longer than the muscle fibers (Alexander, 1977). Thus, heat production from muscle would vary in relation to the degree of muscular stretch which would be influenced by variable tension from forces generated by positive and negative work.

Whole-body temperature responses to negative work, by resisting a calibrated load induced on a cycle ergometer, are not the same as those resulting from positive work at the same oxygen uptake (Nadel, 1972; Stolwijk, 1973). Equilibrium levels of core (esophageal) temperature are about 0.5°C lower during negative work while quadriceps muscle and mean skin temperatures are 1 to 2°C higher, respectively. Blood flow to working muscle is reduced during negative work which causes higher muscle temperature; skin blood flow is increased as a consequence of increased cardiac output (Thomson, 1971), which causes higher skin temperature. Because the rise in core temperature is attenuated during negative work exercise, the greater sweating is related more to the elevated skin temperature (Nielsen, 1969). Stolwijk and Nadel (1973) have concluded that while "the patterns of body temperature differ between positive and negative work exercise, the basic thermal inputs to the regulatory center appear to operate in the same manner independently of the type of exercise."

It is worth reiterating some early observations concerning thermoregulation during exercise as reviewed and observed by Christensen (1931), because most subsequent work has been variations on his themes: a) moderate exercise elevates core temperature to 38.5-38.8°C within ambient temperatures of 7 to 27°C; b) at low to moderate loads the equilibrium level of rectal temperature is reached after at least 1 hr of exercise; c) increased core temperature during exercise is not a pathological response because normal resting temperature is restored within 1 hr of recovery and the subject has no untoward aftereffects; d) within certain limits, the magnitude of the increase in rectal temperature can be determined by varying the intensity and duration of the exercise; e) the increase in core temperature during work is an adaptive process that increases the velocity of chemical reactions in the working organs; f) the greater the work rate, the greater the rate of rise in core temperature; g) at the same rate of metabolism (O_2 uptake), both arm and leg work result in the same increase in core temperature (this implies that equal active muscle masses produce the same rise in temperature); h) core temperature increases in proportion to the increase in oxygen uptake, but the magnitude of the temperature rise differs in different subjects; (i) the increase in metabolism that occurs with fatigue may be partly due to the elevated core temperature; and j) working capacity is decreased considerably at core temperatures above 40.5°C and the danger of heat stroke increases markedly. Most of these observations would support the hypothesis that change in body temperature during exercise is a regulated process. But merely measuring various body temperatures and metabolism (O_2 uptake) during exercise does not define the total regulatory mechanism since the various avenues of heat dissipation were not considered. For the first time, in addition to core temperature, Nielsen (1938) measured skin temperatures and sweat loss during exercise at various intensities at various ambient temperatures in a controlled environment chamber. In addition to observing that equilibrium levels of rectal temperature and regulatory sweating were directly proportional to the absolute exercise load, he also found that the equilibrium levels of rectal temperature were independent of ambient temperatures between 5°C and 29°C when a constant exercise load was employed. This wide range in ambient temperature induced large changes in conditions for heat dissipation and required various combinations of convective, radiation, and evaporative heat exchange to be employed to maintain the constant equilibrium core temperature. These finely tuned and integrated responses indicate thermoregulation rather than thermal failure. A few years later Winslow and Gagge (1941) confirmed Nielson's findings that changes in sweating and evaporative cooling were proportional to changes in heat production during exercise. They also observed the onset of sweating during exercise at a lower ambient temperature in the summer (16°C) than in the winter (20°C), providing the first observations of an increased sweating sensitivity presumably caused by heat acclimatization. Since skin temperature is usually constant or decreases slightly during

exercise at normal ambient temperature while the rate of sweating follows the increase in core temperature, Winslow and Gagge (1941) concluded that core temperature, not skin temperature, must control the sweat-secreting mechanism. We now know that both core and skin temperatures can influence sweating depending upon various environmental parameters, state of hydration, level of fitness, exercise load, degree of heat acclimation, and body position (Greenleaf, 1979; Myers, 1982; Nadel, 1984; Robertshaw, 1983; Rowell, 1983). But as mentioned earlier, there is much more to understanding the mechanism of exercise thermoregulation than just integrating various body temperatures, blood flows (heat conduction), and heat-dissipating responses. Models based upon only these parameters will indicate what happens, but not how it happens.

The major mechanism for thermoregulation during exercise in normal and hot environments is evaporative heat loss that is accomplished in man partially by respiration, but mainly by eccrine sweating. DuBois (1939) stated it succinctly: ". . . there is no one human attribute of more importance than the ability to sweat skillfully." Much has been written concerning the physiology of sweat and sweating during exercise; however, its control and integration into the total mechanism of exercise thermoregulation is not completely understood. Sweat glands are innervated by sympathetic nerve fibers and the glands respond to cholinergic stimuli; sweating is activated by pilocarpine and abolished by atropine (Dale, 1934). But adrenaline will cause sweating when applied directly on the gland (Wada, 1950), and intravenous infusion of isoprenaline and epinephrine increases sweating in the presence of atropine (Allen, 1972). Robertshaw et al. (1973) found that circulating catecholamines enhance existing sweating caused by neural stimulation during exercise in monkeys. A dual control mechanism is common in systems of great importance for survival, in this case for protection against excess hyperthermia and heatstroke.

The sequence of thermal events during exercise begins with increased heat production from muscular contractions which causes an increase in muscle temperature. Cardiac output increases and warmed venous blood is circulated through the cardiovascular system. The blood is redistributed from organs to active muscles by a complex interplay of vasoconstriction and vasodilation of many vascular beds and, as body temperature rises, plasma is shifted to the interstitial (extravascular fluid space in proportion to the exercise load most likely as the result of increased blood pressure. At this time plasma osmolality is essentially unchanged, but there is hemoconcentration. The warmed blood presumably reaches the thermal sensors in the hypothalamic area, spinal cord, and elsewhere. In some unknown manner, nervous and endocrine stimuli instigate heat dissipation responses which include cutaneous vasodilation, to allow heat to reach the skin and sweating to remove it from the body by evaporation. Reflex sweating begins within 3-5 sec after muscular movement (Van Beaumont, 1963) begins and activation of sweat glands (recruitment) during exercise usually proceeds from the trunk to the arm and legs (Nadel, 1971). Sweat gland activity follows a cyclic pattern and sweat starts to run off the skin when sweating has reached about one-third of the maximal evaporative capacity. Only sweat that evaporates transfers heat. Evaporation cools the skin, but skin temperature cannot be uniform because of recruitment, different degrees of skin wettedness, as well as differential rates of inhibition of sweating as exercise progresses. There must be an infinite number of different skin temperatures that must be integrated by the central controller. As exercise of moderate intensity proceeds, an equilibrium is reached whereby the rate of heat dissipation equals the rate of heat production, and core temperature levels off; this takes about one hour. The equilibrium level of core temperature is related to the maximal working capacity (O_2 uptake) (Astrand, 1960; Greenleaf, 1969; Saltin, 1966); that is, to the level of metabolism relative to the subject's maximal oxygen uptake (max $\dot{V}O_2$). A relative exercise load of 50% of the subject's max $\dot{V}O_2$ results in an equilibrium core temperature of about 38°C. A subject with a high $\dot{V}O_2$ max may expend three times the energy as another subject, but both attain the same core temperature. Obviously the rate of heat dissipation in the former subject must be three times greater than that in the other subject; this is achieved by increased sweating and evaporative heat loss (Greenleaf, 1972; Saltin, 1966). In this case the increased sweating is not due to

differences in core or mean skin temperatures, but possibly to increased heat production (O_2 uptake) or other unknown factors proportional to the intensity of the absolute exercise load, that is, the degree of stress (Greenleaf, 1969). Muscle mechanoreceptor function is probably not involved (Ekblom, 1971), but reduction in tissue heat conductance accompanies the increase in sweating (Greenleaf, 1969) suggesting an altered response of the peripheral vascular system and its complicated control mechanism (Rowell, 1983).

Because of the important functions of provision of an adequate blood volume for heat conduction to the periphery and sufficient plasma and interstitial fluid for the formation of sweat, the following section will discuss the influence and major significance of water and electrolyte balance on exercise thermoregulation.

WATER AND ELECTROLYTE BALANCE DURING EXERCISE

Total body water is divided arbitrarily into two major compartments: that contained within cells (cellular) and the extracellular water. The latter is further divided into the vascular fluid (plasma) and the extravascular (interstitial) fluid which is the intermediate fluid compartment between the plasma and the nonred blood cells.

Total body water comprises 50% to 70% of the body weight in normal healthy people, the average being about 61% (Oser, 1965). The higher the percentage of body fat the lower the percentage of water because fat contains less water than muscle (table 2). The volume of body water is regulated daily to ±0.22% (±150 g) of the body weight (Wolf, 1958) and the plasma volume to ±0.7% (±25 g) (Greenleaf, 1979).

With the exception of insensible water losses through the skin and from the lungs, water lost by the body is always accompanied by solutes, mainly sodium and chloride. Sodium is the major cation and chloride the major anion in extracellular fluid (table 3). Changes in extracellular fluid volume are reflected inversely by changes in plasma sodium concentration. Potassium is the major cellular cation, and phosphates and protein the major anions. Despite the significant difference between the cellular cation and anion osmolality (175 and 135 = 310 mosmol/kg), the total cellular osmolality is equal to the extracellular osmolality (155 and 155 = 310 mosmol/kg). Thus, essentially equal osmotic concentrations are maintained between two fluid compartments of widely different solute content. The fluid composition of the body, as well as cellular integrity, is maintained according to the factors controlling osmotic pressure: a) nine-tenths of the osmotic pressure of the body is maintained by the electrolytes and the remainder by proteins and other macromolecules; b) body water distribution is determined mainly by extracellular sodium and protein, and cellular potassium distribution; c) osmotic equilibrium between plasma and erythrocytes is achieved mainly by transfer of water; and d) marked changes in body water distribution can occur with no changes in total volume.

A mentioned previously, the fluid-electrolyte shifts and balances that occur during exercise are affected by changes in both body and environmental temperatures, as well as by such factors as age, posture, degree of adaptation to physical exercise training and to heat (heat acclimatization), preexercise hydration status and the exercise hydration regimen. A detailed and extensive review of these various factors that influence fluid and electrolyte shifts during exercise and heat stress has been prepared by Harrison (1985). An accurate description of fluid and electrolyte responses depends upon a precise description of all these factors. The following discussion will pertain to a young, hydrated, moderately

trained but unacclimatized male exercising in the sitting position on a cycle ergometer at normal (24°C) room temperature with minimal air flow.

Before exercise commences, the subject should be allowed to rest for 40-60 min in the exercise body position for fluid status to reach equilibrium (Hagan, 1978; Diaz, 1979). Any body motion (exercise) or change of position will induce fluid shifts between the plasma and interstitial spaces. The more vertical the body position the greater the initial plasma to interstitum shift, and vice versa (Diaz, 1979). When exercise commences there is an initial shift (loss) of plasma to the interstitial space that tends to be restored (increase) toward the plasma control level as exercise proceeds (Greenleaf, 1977). The rate and equilibrium level of plasma shift is directly proportional to the exercise intensity (fig. 1) and can reach 16-18% with peak exercise (Greenleaf, 1977; Van Beaumont, 1973). Reflex sweating starts within 3-5 sec after limb movement begins (Van Beaumont, 1963) and thermoregulatory sweating begins after 5-10 min in response to the increased core temperature. Sweat is a derivative of interstitial fluid. During rest in the heat, sweating depletes the cellular and extracellular fluid compartment about equally. Sweating during prolonged (3 hr) exercise in a cool environment depletes the cellular space exclusively, while sweating during exercise in a hot environment depletes both the cellular and extracellular spaces; the latter by 50% more than the former (Kozlowski, 1964). At the same time, water is shifted from the interstitial space into active muscle cells (Sjogaard, 1982) and metabolism of carbohydrate releases bound water within the cells. Oxidation of 300 g of muscle glycogen liberates about 810 ml of water which could be used for maintenance of the extracellular volume and sweat formation (Greenleaf, 1969; Olsson, 1970). In spite of earlier movement of water into muscle cells, plus the increased oxidative free water content, the exclusive loss of water from the total cellular space after prolonged exercise suggests a greatly reduced capacity of cells to hold water. Apparently there is a net flux of water from the cellular to the extracellular space to provide for sweating and increased peripheral blood flow. The increased plasma potassium concentration and content during exercise (Greenleaf, 1977; Van Beaumont, 1973) probably accompanies and is a result of this cellular to extracellular fluid shift.

When exercise ceases there is an immediate shift of fluid (3-4% units) back into the vascular compartment, which is followed by a slower rate of restoration of 3-4% units per hour (Greenleaf, 1977). Voluntary ad libitum fluid intake does not appear to accelerate the rate of recovery (Greenleaf, 1965), but forced drinking to some extent does (Costill, 1973).

Continued sweating at rest without adequate fluid replacement results in dehydration with more severe signs and symptoms as the level of dehydration increases (fig. 2). For each 1% loss of total body water (700-800 ml), there is approximately a 2% (70-80 ml) loss or shift of plasma volume. Assuming body position is stabilized, performance of exercise will add an additional plasma to interstitial space fluid shift (fig. 1). The mechanism of this exercise-induced fluid shift is not entirely clear. The major forces that can effect fluid transfer across microvascular membranes are hydrostatic pressure, changes in systemic blood pressure, colloid osmotic pressure (proteins) and crystalloid osmotic pressure (ions).

At the beginning of exercise there is an abrupt loss (shift) plasma that can reach 16-20% after 3 min of peak intensity (Greenleaf, 1977, 1979a). The composition of the extruded fluid is essentially a protein-free plasma filtrate; that is, it has the same composition as plasma minus most of the proteins. A small fraction (1-2%) of the plasma proteins is shifted with the filtrate (Greenleaf, 1977, 1979a; Wilkerson, 1982). Thus plasma osmolality remains within the control range (below 290 mosmol/kg) during short-term exercise of less than 30 min. Plasma osmolality rises with moderately heavy exercise greater than 50% of peak capacity (Convertino, 1981) and lasting longer than 30 min with no fluid replacement, but plasma volume remains depleted (Greenleaf, 1977). In spite of the increased plasma osmolality and protein concentration, there is only a slight restoration (1-2 percentage units) of the depleted plasma volume as

exercise continues (Greenleaf, 1977; Hagan, 1980). The characteristic increase in plasma potassium content and concentration during exercise suggests cellular to extracellular fluid shift reflecting presumably a contribution of water released, in part, from glycogenolysis. It is possible that increased osmolality of the interstitial compartment could "pull" filtrate from the vascular system, but any such action would probably take place after exercise is well under way to allow sufficient time for the increased cellular osmols to move to the interstitial space. The shift of 16-20% of plasma filtrate within 2-3 min during heavy exercise suggests that increased capillary (transmural) pressure is the major driving force (Greenleaf, 1977, 1979a; Van Beaumont, 1973). The partial restoration of plasma volume as exercise proceeds is probably caused by the increasing plasma colloid and especially crystalloid osmotic pressures. Within 30 sec after heavy exercise ceases, plasma osmolality increases by 5-10 mosmol/kg (Greenleaf, 1979a). This hyperosmolality, coupled with the hyperproteinemia and reduction in systemic blood pressure, would favor restoration of plasma volume. The hyperosmolality gradually decreases as plasma volume increases toward normal levels during recovery (Greenleaf, 1979a).

To maintain body water content during exercise, water lost in sweat, and through the respiratory tract especially in cold environments, can be replaced most efficiently by drinking. In men, unfortunately, the rate of voluntary fluid intake is about one-half the rate of water loss so that exercising men are always in negative water balance unless there is forced drinking. This condition has been termed voluntary dehydration (Greenleaf, 1965; Rothstein, 1947), but more appropriate terms are involuntary hypohydration (Greenleaf, 1967) or involuntary dehydration (Greenleaf, 1983), because people do not dehydrate voluntarily. Involuntary dehydration can be expressed in terms of fluid balance (H_2O intake – H_2O loss) or in terms of fluid deficit (H_2O loss – H_2O intake)/(H_2O loss) $\times$ 100. The terms hyperhydration, euhydration, and hypohydration refer to the equilibrium states of an excess, normal ($\pm$1-2% of body H_2O), or deficit volume, respectively, of body water. The terms overhydration and rehydration refer to changes in body water from eu- to hyperhydration and from hypo- to euhydration, respectively; dehydration is reserved for the process of fluid depletion when going from hyper- to euhydration and from eu- to hypohydration (Greenleaf, 1979).

The greatest stress on the fluid balance system is when a dehydrated person exercises in a hot environment. When those three factors are separated, it was found that heat exposure had the least inhibitory effect, prior dehydration an intermediate effect, and exercise per se had the greater inhibitory effect on voluntary rehydration after fluid loss (Greenleaf, 1965). Regardless of the method used to reduce body water and the level of hypohydration attained, the rate of normal rehydration was the same when flood and fluids were available ad libitum. The greater the level of hypohydration (the more stressful the conditions), the longer it takes by drinking to replace the lost fluid (fig. 3). Thus, drinking should begin before or immediately upon exposure to a stressful, fluid-depleting situation before feelings of thirst have been perceived. Significant levels of dehydration will occur before thirst appears. In previously hydrated subjects, the threshold for involuntary dehydration occurs with a sweat (water) loss of 75 g/hr; in the heat the threshold is about 275 g/hr (Greenleaf, 1965; Rothstein, 1947), illustrating the moderate inhibitory effect of heat on voluntary drinking.

Adaptive responses during consecutive exercise training exposures in a hot environment (heat acclimation) result in greater voluntary water intake such that a progressively greater portion of the progressively increasing sweat loss is replaced (Greenleaf, 1983). The level of involuntary dehydration decreases from 52% before to 37% after acclimation (fig. 4), indicating that about one-half the loss was replaced before and about two-thirds was replaced after acclimation. During the consecutive acclimation exposures, there is a progressively shortened time to the first drink, a greater number of drinks per exposure, and a larger volume per drink. After the acclimation exposures started, it took 2 days before fluid intake was significantly large to overcompensate for the sweat losses. This delay suggests a hormonal

mechanism. The mechanism of increased drinking during exercise-induced dehydration in the heat appears to be related more to the reduction in body fluid volume and responses of the renin-angiotensin system than to predominate responses of the sodium-osmotic-vasopressin system (Greenleaf, 1983).

Much has been written extolling the virtues of various rehydration drinks for use before, during, and after exercise and heat exposure (Murray, 1987). They usually contain one or more carbohydrates to provide for energy requirements, and numerous minerals to ostensibly restore rather small losses in sweat.

The composition of an ideal rehydration drink would be such that it would stimulate drinking, under all conceivable exercise and environmental conditions, at a rate equal to the rate of uptake of the ingested fluid from the gut into the extracellular space; i.e., an optimal rate of intake. To meet this difficult requirement the drink must be palatable at any temperature, and it must not allay thirst for any length of time since involuntary dehydration is almost always present. In fact, it should contain substances that stimulate thirst, perhaps caffeine, that would aid in attainment of the nebulous optimal rate of drinking.

There are two major problems with the many available rehydration drinks that contain carbohydrates and minerals. First, some data indicate that too great a carbohydrate concentration (2.5 to 10%) delays gastric emptying (Costill, 1974; Coyle, 1978). But other researchers have found no effect of heavy exercise for 1 hr on the rate of gastric emptying of a 13% glucose and 0.3% NaCl solution or on the absorption from the ileum or jejunum (Fordtran, 1967). Kasabach (1931) found no effect of mild exercise on gastric emptying time of a 50% dextrose solution. In fact, mild exercise has been reported to increase the rate of gastric emptying (Nielsen, 1921). The second problem involves the efficacy of ingesting minerals, mainly sodium, chloride, and potassium, to replace those lost in sweat. As mentioned previously, with moderate to heavy exercise over 30-min duration there is a progressive increase in plasma osmolality caused mainly by increases in sodium and chloride and, to a lesser extent, potassium. Ingestion of these ions would tend to further increase the existing hyperosmolality. Loss of 4.1 liters of sweat in conjunction with a body weight loss of 5.8% resulted in losses of only 5-7% of the estimated total extracellular sodium and chloride contents, and only 1% of cellular potassium and magnesium contents (Costill, 1976).

Under some circumstances small amounts of sodium chloride in ingested fluid appear to facilitate gastric emptying (Shay, 1934). If hypotonic saline in the gut requires the transfer of sodium from the blood to increase the tonicity of gut water to provide more optimal (isotonic) conditions for absorption (Hunt, 1968), the exercise-induced hyperosmolality should provide sufficient sodium, but there may be some delay in the uptake of the water while the sodium is transferred to the gut fluid. In this case, the uptake delay must be weighed against the consequences of exacerbation of plasma osmolality via the ingested ions. Also, if it were important to "replace" ions lost in sweat, the hyperosmotemia could provide a ready supply; additional ingested ions, especially potassium, would not be needed and, under stressful circumstances, could be harmful. Except in severe conditions of prolonged work for hours in very hot climates without normal meals, there is no physiological justification for consuming rehydration drinks containing significant amounts of sodium, chloride, potassium, or any other ions. If regular meals are not available, then the rehydration drink would have to contain calories and this then becomes an additional problem of providing nutrients in addition to water.

ACCLIMATIZATION TO HOT ENVIRONMENTS

The adaptive potential of an organism influences survival, and the capacity to respond appropriately to thermal stress is no exception. The thermal stress could arise internally from increased metabolism or from inhibition of the normal heat dissipation responses, externally from increased ambient heat flux, or from both sources such as exercising in a hot environment.

Terminology definition has been confusing. The following definitions (Greenleaf, 1970; Sciaraffa, 1980), which have been collated from those of Fry (1958), Hart (1957), Eagan (1963), and Prosser (1964), will be used:

1. Adaptation. Phylogenetic changes that have been fixed in the heritage of any particular species.

2. Acclimatization (natural). Long-term responses throughout the life of an individual to the complex of natural environmental variables. The term heat acclimatization should not be used if reference is only to the effects of heat on the organism because other variables would be acting simultaneously.

3. Acclimation (artificial acclimatization). The day-to-day organismic changes caused by the effect of a single environmental variable, such as heat, as would be investigated in controlled laboratory conditions.

4. Habituation. A change in physiological response resulting from a decreased response of the central nervous system to constant stimuli.

Most research has been directed toward understanding the process of acclimation and short-term acclimatization as it modifies (enhances) performance of moderate to hard physical work in a hot environment (Greenleaf, 1970; Sciaraffa, 1980); for example, for military requirements, athletic endeavors, and work in hot industrial settings. As a result, the research protocols have focused mainly on describing the magnitude and time courses of the progressive "clinical" responses—such as heart rate, blood pressure, body temperatures, and sweat rates—during consecutive intermittent heat exposures while resting and exercising. Often the ultimate purpose was to devise remedial procedures for improving work performance. Very little work has been directed toward understanding the long-term (years) acclimatization process in humans (Christensen, 1947; Greenleaf, 1970; Höfler, 1969; Sciaraffa, 1980).

The first practical response to undesirable heat exposure is to increase the rate of heat transfer between the organism and environment—by exposing a larger area of skin, by removing clothing, or by seeking cooler surroundings—or to reduce energy-induced heat production. Most aspects of this so-called behavioral thermoregulation have been covered admirably by Bligh (1973) and will not be discussed further here.

It is clear that the major physiological mechanism for maintaining thermal homeostasis in humans exposed to heat is to increase heat dissipation by accelerating the delivery of heat to the skin, by means of cardiovascular adjustments (Rowell, 1983), and by increasing sweating to facilitate evaporative heat loss (Eichna, 1950; Robinson, 1954; Höfler, 1969). Changes in total body resting and exercise metabolism during and following heat acclimation and acclimatization are variable and appear to be a less important part of the adaptive response.

In this section we will present a description of the physiological changes, including energy metabolism, resulting from heat acclimation. This will be followed by a discussion of the cardiovascular adjustments, sweating and evaporative heat loss responses, and the role of fluid and electrolyte balance in the heat acclimation or acclimatization processes.

Acclimated subjects work in the heat with lower heart rates, lower skin and core temperatures, more stable blood pressures, greater sweat rates, and with much less discomfort than they did before acclimation (Greenleaf, 1970; Sciaraffa, 1980). Acclimation develops most rapidly when the environment is hot and is retained longest if the subjects remain in a warm environment. Some findings from heat acclimation research are:

1. Resting in the heat induces some acclimation, but a greater adaptive state can be induced by intermittent exposure to exercise in the heat.

2. Strenuous exercise during the first few heat exposures is not well tolerated and will lead to collapse if the work is continued.

3. Subjects should not work beyond moderate comfort levels during the first 3-4 acclimation exposures.

4. Subjects trained for endurance exercise acclimate more rapidly than subjects of lesser fitness because some of the adaptive responses from exercise training are similar to those induced by heat exposure alone.

5. A degree of heat acclimation can be induced by just living and working in a hot climate; continuous heat exposure is not a prerequisite. Heat exposure for 2-3 hr/day, or every other day, is sufficient.

6. An optimal acclimation state is achieved most efficiently by a single daily exposure of 100 min while walking at a moderate pace of 5.6 km/hr at an ambient temperature of 49°C.

7. A few subjects are not able to "adapt" to the heat acclimation procedure; the reasons are unknown.

8. Replacement of water lost in sweat during acclimation exposures is essential since dehydration retards the acclimation process. Thirst is a poor indicator of the need for fluid.

9. The best performance of subjects exercising in the heat is attained by replacing water lost in sweat hour by hour, and salt lost in sweat meal by meal.

10. The degree of environmental stress in a hot, dry desert is two to three times greater than that in the humid tropics. As a result there is a large degree of cross-acclimatization such that acclimatization to a hot, dry environment results in nearly total acclimatization to a hot, humid environment.

11. The rate of sweating in humid heat, which is independent of fluid intake, is much greater than thermoregulatory demands require and wastes salt and water.

12. Sweat sodium chloride concentration decreases progressively as acclimation proceeds.

13. Artificial or seasonally acquired cold acclimation appears to be unaffected by heat acclimation. The physiological changes induced by both cold and heat acclimation can exist simultaneously in the same person and respond independently. The gain or loss of one acclimation state is not influenced by the other.

14. The acute phase of the heat acclimation process is essentially complete in 8-10 days, but it may continue more slowly for up to one month.

15. Heat acclimatization is attained more rapidly and lost more slowly in subjects living in a warm climate (summer) than in those living in a cold climate (winter).

16. There seems to be no significant difference between the rate of attainment or the final level of acclimation between men and women with due consideration for age, body size, lean body mass, living habits, and level of endurance fitness. The menstrual cycle has no effect on heat tolerance at rest or during exercise. Prepubertal girls, like prepubertal boys, have a lower heat tolerance than adults.

17. Maintenance of heat acclimation requires periodic reexposure to work in the heat at intervals of 1-2 weeks. Subjects who retain high levels of endurance fitness also retain their acclimation status longer and better. The level of retention of acclimation can be assessed satisfactorily from the results of a single exposure to the acclimation exercise—heat stress. Subjects who have been deacclimatizing for one week should be reacclimatized by one 100-min exercise/heat session before returning to work in a hot environment.

Loss of acclimation begins when subjects move to a cooler environment. Some rates of losses of acclimation parameters after 1, 2, and 3 weeks are: exercise heart rate and sweat rate—50%, 80%, and 100%, respectively; and exercise rectal temperature—25%, 40%, and 50%, respectively. A substantial part of the acclimation can be lost after 7 days and the decay is virtually complete after 23-28 days. Some subjects are able to retain an appreciable degree of acclimation for up to 2 months, especially if exercise training is continued after heat exposure ceases (Greenleaf, 1970; Sciaraffa, 1980).

METABOLISM

There appears to be no clear trend in responses of energy metabolism (oxygen uptake) to heat acclimation at rest or during exercise. One reason is that measurements of oxygen uptake taken before, during, and after acclimation have not been compared with similar points of reference. For example the metabolism of heat acclimated subjects, who remain in a hot environment, may be different than if the subjects returned to the cooler preacclimation environment. Another reason is the difficulty in determining the point during the acclimation process when the strain had reached an equilibrium and when or if it had returned to preacclimation levels. Energy cost should increase with increased stress and decrease with decreased stress. It could be hypothesized that one basic response following heat acclimation should be a reduction in basal, resting, and exercise metabolism. That is, it should cost less to run the system if the heat acclimated subject continues to live in the heat. Conversely, while the system is in the process of acclimating to a more stressful situation (exercise and heat), the energy cost should increase over that needed during similar exercise in a cool environment. A further complication is the difficulty in determining the level of exercise training and the concomitant degree of heat acclimation responses that were induced by the training before the formal heat acclimation procedure began. When assessing the degree of acclimation, there is also the problem of whether to use the same absolute exercise loads for all test

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subjects, or to use similar relative loads. The earlier studies for the most part used similar absolute loads, thereby further complicating interpretation of the data.

Basal Metabolism

Basal metabolism is unchanged with acute heat (33°C) exposure (Spealman, 1948), and also appears to be unchanged during 1 month of chronic, intermittent heat (41.7°C) exposure (Stein, 1949). These observations would suggest that thyroid function in resting subjects was unchanged.

Submaximal Exercise Metabolism

It is difficult to draw firm conclusions regarding the oxygen uptake responses to submaximal exercise during acute heat exposure or during the acclimation process because of differences in subjects' hereditary working capacities (peak oxygen uptakes), their levels of training, and the differences in utilization of the various testing equipment (e.g., ergometer, treadmill, and bench stepping) for the exercise.

Acute heat exposure– Compared with exercise response in a cool environment, oxygen uptake increases by 2-3% during comparable exercise in the heat in previously untrained and unacclimated male subjects (Rowell, 1968, treadmill; Sawka, 1983, treadmill). In untrained and unacclimated females, submaximal oxygen uptake has been reported to increase significantly by about 10% during the first exercise-in-heat exposure (Wells, 1974, treadmill). In exercise-trained or exercise-trained/heat-acclimated men, submaximal oxygen uptake is either unchanged (Pandolf, 1974, treadmill) or reduced slightly (by 0.2 liter/min) during the first heat exposure (Williams, 1962, ergometer). Neither cardiac output nor arteriovenous oxygen difference was altered during exercise in heat compared with exercise responses in cool conditions. In the heat, the major change in cardiovascular dynamics was a decrease in stroke volume coupled with a compensatory increase in heart rate. Increased (excess) blood lactate concentrations occurred during work in heat suggesting that the working muscles were relatively more "hypoxic" in heat, and that this hypoxia may have accounted for the lower oxygen uptakes (Williams, 1962). It thus appears that exercise training and/or heat acclimation reduces the strain sufficiently to eliminate or reverse the increased oxygen uptake during submaximal exercise with acute heat exposure.

During and after acclimation– In exercise-trained subjects working at the same absolute loads under laboratory conditions, submaximal oxygen uptake may increase slightly during the first few acclimation exposures (as mentioned under acute heat exposure), but it then returns to or slightly below a level proportional to the exercise load which would have occurred in a cool environment during the remainder of the acclimation exposures (Wyndham, 1976, ergometer). Other investigators have reported no change in submaximal oxygen uptake at the end of exercise-heat acclimation exposures in previously trained (Cleland, 1969, treadmill; Piwonka, 1967, treadmill) and in previously untrained men and women (Rowell, 1967, treadmill; Weinman, 1967, treadmill). On the other hand, compared with submaximal exercise responses in a cool environment, there are numerous reports of significant reductions in oxygen uptake (by 2%-17%) after heat acclimation in physically trained men during submaximal exercise in hot environments (Eichna, 1950, treadmill; Gisolfi, 1973, treadmill; Robinson, 1945, treadmill; Senay and Kok, 1977, bench stepping; Shvartz, 1977, bench stepping; Strydom, 1966, bench stepping), and during submaximal exercise in cool environments (Eichna, 1950, treadmill; Senay and Kok, 1977, bench stepping; Shvartz, 1977, bench stepping). Only Consolazio et al. (1973) have reported significant increases in submaximal oxygen uptake after acclimation in essentially untrained subjects. Thus, the bulk of the

evidence indicates unchanged or lower submaximal exercise metabolism after heat acclimation. It is probably the result of less strain on the system, but the mechanism is still unknown.

Peak Exercise Metabolism and Training

In exercise-trained subjects peak (maximal) oxygen uptake is essentially unchanged during exercise in heat (Williams, 1962; Wyndham, 1970). Peak oxygen uptake has been reported to increase from 2.39 to 2.82 liter/min (18%) in untrained mine workers after a period of intense acclimation (Wyndham, 1972), but Kupprat et al. (1980) found the increase to be only 4.7% during treadmill exercise after exercise/heat acclimation in previously untrained women.

Results from an earlier study, where highly trained men failed to show the characteristic cardiovascular and thermoregulatory responses during exercise-heat acclimation (Greenleaf, 1964), suggested that these subjects were essentially acclimated to heat. It is now well-established that having a high peak oxygen uptake helps attain, retain, and restore the acclimated state (Eichna, 1945; Pandolf, 1977; Shvartz, 1977). Men with higher peak oxygen uptakes have greater heat tolerances than men with lower (<2.5 liter/min) uptakes when the same absolute exercise load was performed by all subjects during the heat acclimation exposures (Kok, 1973; Pandolf, 1977). But tolerance to a heat-ergometer stress was essentially the same during early acclimation exposures in men with low (42 ml/min/kg) and high (64 ml/min/kg) peak oxygen uptakes working at 47°C ambient temperature and at 28 ±1% of their peak oxygen uptakes. Thus, there seems to be no significant relationship between peak oxygen uptake and tolerance time during early acclimation exposures (heat stress) and, under these conditions, the high-capacity subjects had no appreciable advantage in tolerance over the lower capacity subjects during early exposure to heat when they all worked at the same relative loads. Thus, the advantage trained subjects have over untrained subjects when exposed to exercise/heat stress is the lower strain when both are subjected to the same absolute exercise load.

Since peak oxygen uptake is determined mainly by genetic factors, there is still the question of the proportion of a person's heat adaptive capacity that can be induced by the normal acclimation process. The general hypothesis is that many adaptive physiological responses induced by exercise training in a cool environment are similar to those induced by the submaximal exercise/heat acclimation process; for example, lower exercise heart rates and core temperatures, and enhanced sweating. The finding that physical training in cool environments induces some degree of heat acclimation, but training in a hot environment is necessary for full heat acclimation (Edholm, 1969; Gisolfi, 1969; Piwonka, 1965; Roberts, 1977), suggests that it may be the degree and duration of the elevation of core temperature that is the major stimulus for the acclimation response. If the attainment of heat acclimation is defined only as a significant increase in sweating in subjects resting in heat (Fox, 1963), then heat acclimation can be induced by merely raising body temperature without a significant increase in metabolism (exercise). If heat acclimation is defined as an enhanced capacity to work in heat, then the appropriate method to raise core temperature is by repeated exposures to exercise in the heat. Clearly, the increase in metabolism and the associated stimuli to the cardiovascular system during exercise, in addition to the hyperthermia, adds other adaptive dimensions for the acclimation process.

Body Temperature and Fluid-Electrolyte Balance

The major physiological response during exercise/heat acclimation is the lessening of strain by means of greater and perhaps more efficient heat dissipation responses. This involves greater heat

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conduction by the blood from working muscles to skin, and subsequent dissipation by evaporation of sweat. The cardiovascular adjustments to thermal stress have been extensively reviewed recently by Rowell (1983). Solid data on changes in skin blood flow during acclimation are sparse because of the difficulty in making valid measurements. It seems that the absolute flow decreases as acclimation proceeds. But since the rate of decrease of flow is less than the rate of decrease of the equilibrium level of core temperature, there appears to be adequate blood flow available to transfer the "excess" heat to the periphery (Rowell, 1983).

Sweating– The other important part of the heat dissipation-thermoregulatory mechanism, which may not function totally independently of blood flow, is sweating and evaporative heat loss. Care must be taken when inferences are drawn concerning rate of sweating and subsequent rates of heat dissipation. Increased sweating does not always result in a proportional increase in evaporative heat loss because, particularly in humid environments, some sweat drips and some is absorbed in clothing which would not be available for evaporation. It is the percent of wetted skin area open to the environment for unimpeded evaporation that accounts for the effective evaporative heat loss. In general, sweat rate and evaporative heat loss increase progressively during the first few acclimation exposures, particularly in dry heat where dripping is minimal. It appears that control of sweat solute composition is a regulated process and the magnitudes of sweat sodium, potassium, calcium, and chloride concentrations are independent of flow rates (Verde, 1982). The increased evaporative heat loss contributes significantly to the progressive lowering of core temperature as the heat-exercise acclimation exposures continue.

Nadel et al. (1974) have concluded that increased sweating resulting from exercise training in a cool environment is achieved by "peripheral mechanisms" (greater increase in sweat output per degree rise in core temperature), and by "central mechanisms" during exercise-heat acclimation (a lowering of core temperature per unit output of sweat). Thus, increased sweating from the classical exercise/heat stimuli point of view may be controlled by or is the net result of two or more somewhat distinct mechanisms. Indeed, Du Bois (1939) said it best: ". . . there is no one human attribute of more importance than the ability to sweat skillfully."

There is no unanimity regarding the magnitude of the increase in sweating during successive exercise-heat exposures. Both Cleland et al. (1969) and Greenleaf et al. (1985) failed to observe increases in whole body sweating in women who had undergone 8-9 days of acclimation to moderate exercise at 41°C-42°C ambient temperature. Since the maximal rate of sweating is directly related to the level of physical fitness (peak oxygen uptake), fitness status must be evaluated carefully before conclusions are drawn regarding the attainment of acclimation based upon changes in sweat rate. Since heat production is directly proportional to metabolic rate, people with high peak oxygen uptakes can produce more heat than people of lower capacity both on an absolute basis and also when both groups are working at the same relative exercise load (Greenleaf, 1972). When high capacity (peak $\dot{V}O_2$ = 5.1-6.0 liter/min) and lower capacity (peak $\dot{V}O_2$ = 3.9-4.4 liter/min) subjects were exercised at a relative oxygen uptake of 28% at 41°C ambient temperature, the high-capacity subjects' sweat rate was 800-1,000 $g \cdot hr^{-1} \cdot m^2$, while the lower capacity men sweated only 300-400 $g \cdot hr^{-1} \cdot m^2$. These differences in sweating were directly related to exercise load and metabolic rate but were not related to the intensity of relative neuromuscular stimuli or to the absolute levels or changes in rectal or mean skin temperatures (Greenleaf, 1972). These results suggest that nonthermal factors related to exercise load and/or metabolic rate also exert some control over sweating.

The control of sweating and sweat composition during exercise/heat acclimation has been studied intensively from the descriptive point of view (Robinson, 1954), but relatively little work has been devoted to elucidating the mechanism of sweat volume and electrolyte changes via the neuro-endocrine

control system (Sato, 1977). Again, an understanding of this mechanism is complicated by the action of the combined stresses of exercise metabolism (internal heating) as well as environmental heating. There would be little or no uncertainty if it could be assumed that the primary stimulus for the acclimation process was only the increase in core temperature whether induced by increased metabolism, environmental heating, decreased heat dissipation, or any combination of these factors. Since sweating can be activated reflexly within a few seconds of the onset of exercise before any body temperature change occurs (Van Beaumont, 1963, 1966), and increased sweating can be induced by local heating of the skin without increase in core temperature (Hardy, 1961; Collins, 1966), it is probably that increased metabolism and body heating from exercise would activate somewhat different stimuli for sweating than if sweating were induced only by local heating. Also, it is probably that exercise exerts a unique stimulus of its own.

Analogous with kidney function, control of sweating can be separated, perhaps artificially, into fluid formation/excretion and solute excretion. It has been well documented that, during the first few heat/exercise acclimation exposures, total body sweat rate increases by 30%-50% (Greenleaf, 1983; Roberts, 1977), there is faster onset of sweating; i.e., an increased sensitivity of the sweating mechanism (Roberts, 1977), better distribution of sweat over the body surface, and a reduction in sweat sodium chloride concentration (Fox, 1965). Collins and Weiner (1968) and Greenleaf (1979) have reviewed some aspects of hormonal control of sweating, but relatively little definitive work has been performed on this question due partly to a lack of sufficiently accurate assays; a situation which has now been overcome. The extensive information on the endocrine responses to exercise (Terjung, 1979) will not be repeated here, but many endocrine reactions will be present during exercise/heat acclimation exposures. The comment by Gale (1973) that all hormones except the parathyroid appear to participate in thermoregulation is worthy of note.

Fluid and solute formation/excretion– The increase in plasma and interstitial fluid volumes with exercise/heat acclimation serves to enhance cardiovascular function by increasing stroke volume and decreasing heart rate during exercise. The relative reduction in peripheral blood flow resulting from increased evaporative heat loss allows more blood to be shunted to working muscles. It is tempting to speculate that the hypervolemia also provides additional fluid for increased sweat formation. There is probably sufficient interstitial fluid reserve, which can be replenished from the much larger intracellular fluid volume, to provide for an additional 300-500 ml of sweat per hour for a couple of hours before the progressive decline of sweating (hidromeiosis) occurs.

Attempts have been made to alter sweat secretion with various vasopressive agents (Pitressin, Pituitrin) administered subcutaneously or intramuscularly in men sweating under various thermal stimulation. The results have been equivocal with reports of an increase (Ladell, 1948), a decrease (Fasciolo, 1969; Ladell, 1948), or no change (Gibinski, 1961; Ladell, 1960; Pearcy, 1956; Ratner, 1964) in sweating. These variable responses may have been influenced by the vasoconstrictive action of this hormone and by the pharmacologic doses administered. Equilibrium levels of plasma vasopressin (PVP) in blood were unknown. One would assume that PVP would act to conserve water (reduce sweating) and increase sweat sodium concentration as observed by Fasciolo et al. (1969). Thus, the increased sweating and reduced sweat sodium concentration which occurs with consecutive heat acclimation exposures would be the response to an inhibition of vasopressin. Progressive changes in plasma PVP during exercise-acclimation exposures have not been measured but, during exercise above 50% of the peak oxygen uptake, plasma vasopressin concentration is reduced by half after 8 days of exercise training (2 hr/day) at 25°C ambient temperature (Convertino, 1983). Plasma PVP is essentially unchanged at exercise loads below 50% of the peak oxygen uptake after training. Since most exercise intensities during acclimation exposures are less than 50% of peak $\dot{V}O_2$, inhibition of PVP may not be of primary importance for the increase in sweating unless the ambient heating plus exercise raises plasma PVP sufficiently. On the other hand, there was a

tendency for resting plasma PVP concentration to decrease during 12 days of exercise-heat acclimation exposures in men (3.3 ±0.9 to 1.1 ±0.4 pg/ml) and women (1.0 ±0.2 to 0.7 ±0.2 pg/ml) (Greenleaf, 1985). Candas et al. (1984) observed significant sweating depression but no significant change in PVP concentration in subjects resting in humid heat (43°C, 32 Torr) for 2.75 hr/day for 5 consecutive days.

The effect of PVP on sweating during exercise-heat acclimation is still unresolved, but the data point to either no change or to a reduction in PVP at rest and no change during exercise below 50% relative oxygen uptake loads during consecutive exercise/acclimation exposures (Convertino, 1981).

The mechanism of sweat solute formation and excretion is not independent from sweat fluid excretion. Collins and Weiner (1968) and Greenleaf (1979) have reviewed some aspects of hormonal control of sweat solute and fluid excretion, but relatively few hypotheses have been tested to account for the decreased solute excretion after acclimation. The adrenocortical hormones desoxycorticosterone, corticosterone, and especially cortisol have been implicated as factors influencing changes in sweat composition during acclimation (Collins, 1968, 1979; Finberg, 1977; Francesconi, 1984; Harrison, 1975). The general conclusion is that these glucocorticoids respond to the stresses of dehydration, exercise, and heat exposure, but they have essentially no direct significant influence on sweating or sweat composition during the heat acclimation process. But because of the plasma protein stimulating effect of cortisol, the latter may function indirectly to enhance sweating by contributing to the increase in plasma and interstitial fluid volumes via increase in osmotic pressure.

Because of its specific and powerful sodium and fluid retaining action, the renin-angiotensin-aldosterone (RAA) system has been assumed to be a major mechanism in the heat acclimation process because of its influence on sweat rate and sweat solute composition (Collins, 1968). It is clear that exercise and heat exposure administered separately cause large responses in the RAA system, which effectively controls salt and some aspects of water metabolism, peripheral capillary filtration, and tone of the capacitance (venous) blood vessels (Collins, 1979; Geyssant, 1981; Kirsch, 1975; Melin, 1980). In contrast to men resting in the heat, with exercise in the heat there is increased conservation of urinary sodium (Francesconi, 1977). The increases in plasma renin activity (PRA) and aldosterone concentration with heat and exercise appear to be the result of the fluid and electrolyte shifts induced by these two stresses (Convertino, 1980, 1981; Finberg, 1974, 1976). The PRA and aldosterone responses to exercise and hypohydration are attenuated exercise/heat-acclimated subjects (Francesconi, 1983). The absence of significant sodium deficits, even in the presence of copious sweating, has led Finberg et al. (1976) to hypothesize that aldosterone must be mediating some of the physiological adjustments observed with acclimation. But the action of aldosterone is not limited only to preservation of body sodium content; it also facilitates an increase in extracellular volume via sodium retention. Davies et al. (1981) have concluded that while PRA and aldosterone concentration are increased significantly by the acute stress of heat and exercise, their responses were unaffected by acclimation per se. But during intense exercise, exercise-trained subjects exhibit greater sweating but less reduction in hematocrit (plasma volume) than untrained subjects (Melin, 1980), suggesting that sweat formation in the trained subjects was derived more efficiently and preferentially from the enhanced interstitial fluid than from the plasma volume. Increased sensitivity to methacholine-induced sweating and sweat gland hypertrophy are two important features of sweat glands from physically trained and acclimated subjects (Sato, 1983). Recently, it has been reported that the hypervolemia of exercise training and heat acclimation was associated directly with thermal effects and also with nonthermal effects such as the increased plasma levels of PRA and vasopressin, and the increased osmotic and albumin contents (Convertino, 1980a, 1980b).

If the main function of acclimation is to facilitate heat dissipation via evaporation of sweat, then greater and more effective sweating would be of prime importance for survival of the organism. Thus,

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during acclimation, the main function of the salt-retaining hormones (RAA system) may be to conserve sodium via the kidney and sweat glands and, in conjunction with the protein stimulating action of cortisol, to facilitate an increase in the extracellular fluid volume. The latter could then meet the competing demands from increased cardiovascular requirements from exercise and the increased peripheral blood flow needed to transport heat to the skin for evaporation.

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TABLE 1.– TEMPERATURES AND HEAT PRODUCTION OF BODY TISSUES IN A 63 kg, 1.8 m^2 MAN AT REST

Tissue	Temperature, °C	Heat production, %	Body weight, %	Heat production, kcal/hr
Brain	37.2[a]	18	2	13
Heart	37.0[a]	11	1	8
Kidney	37.0[a]	7	1	5
Hepatic-Portal	37.2[a]	20	4	15
Muscle	36.0	20	44	15
Skin	34.0	5	8	4
Other		19	40	14
		100	100	74

From Carlson and Hsieh (1965); Oser (1965); Bazett (1949), Saltin et al. (1968); and Afonso et al. (1962).

[a]venous blood temperature.

TABLE 2.– WEIGHT AND WATER CONTENT OF BODY TISSUE FROM A 70.6-kg MAN[a]

Tissue	Percent body weight	Percent water content
Striated muscle	31.6	79.5
Skeleton	14.8	31.8
Adipose tissue	13.6	50.1
Skin	7.8	64.7
Lungs	4.2	83.7
Liver	3.4	71.5
Brain and spinal cord	2.5	73.3
Alimentary tract	2.1	79.1
Alimentary tract contents	.8	---
Heart	.7	73.7
Kidneys	.5	79.5
Spleen	.2	78.7
Pancreas	.2	73.1
Bile	.2	---
Teeth	.1	5.0
Hair	.1	---
Remaining tissues		
Liquid	3.7	93.3
Solid	13.5	70.4
Total body	100.0	67.2

[a]Modified from Oser (1965).

TABLE 3.– NORMAL COMPOSITION OF FLUID SPACES IN MAN

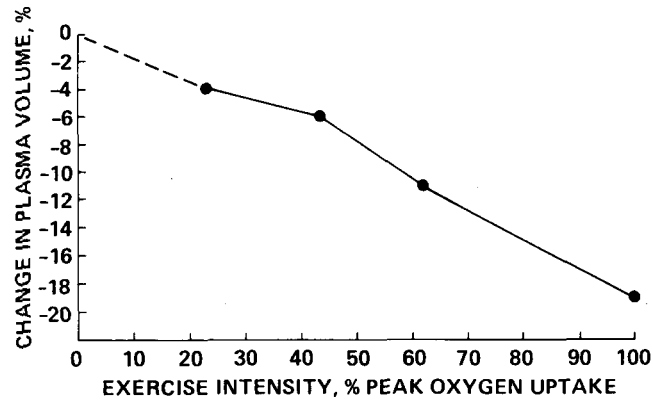| Fluid space	Cations				Anions			
			Other$^+$				Other$^-$	
	Na$^+$ mEq/l	K$^+$ mEq/l	Ca^{2+} + Mg^{2+} mEq/l	Osmols$^+$ mOsm/kg	Cl$^-$ mEq/l	HCO^{3-} mEq/l	PO$_4$$^{3-}$ + PRO mEq/l	Osmols$^-$ mOsm/kg
Extracellular	142	5	8	155	103	27	25	155
Cellular	10	145	20	175	2	8	190	135
Total	152	150	28	330	105	35	215	290

From Greenleaf (1982).

Figure 1.– Plasma volume loss (shift) with increasing intensity of exercise. From Greenleaf (1982) with permission.
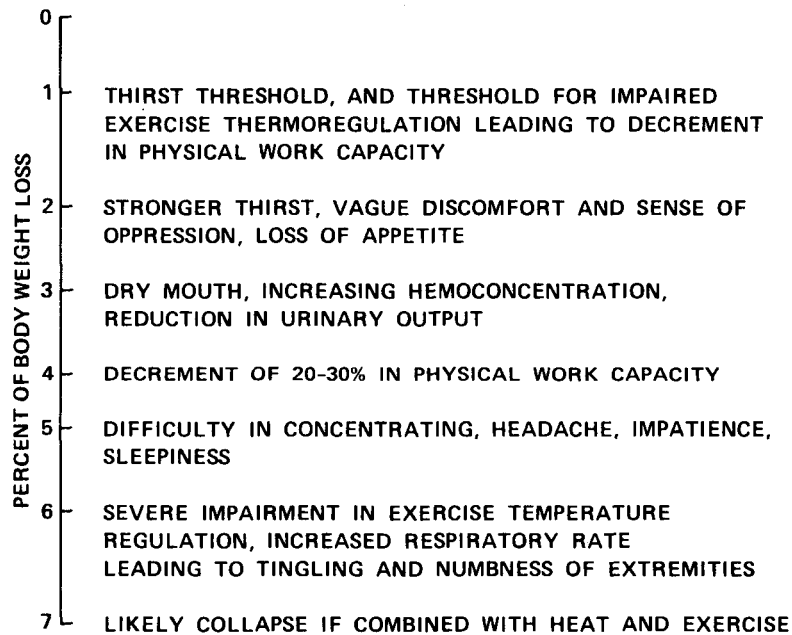

Figure 2.– Adverse effects of dehydration. From Greenleaf (1982) with permission.
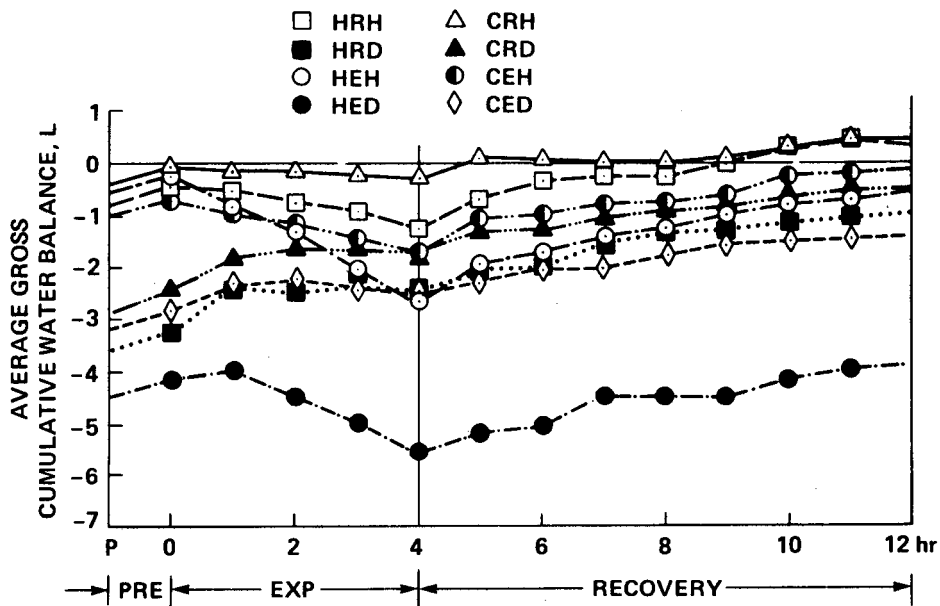
Figure 3.–Gross cumulative water balance during experimental (stress) and recovery periods for 8 conditions: H = heat, C = cool, R = rest, E = exercise, H = hydration, D = dehydration. From Greenleaf et al. (1965) with permission.
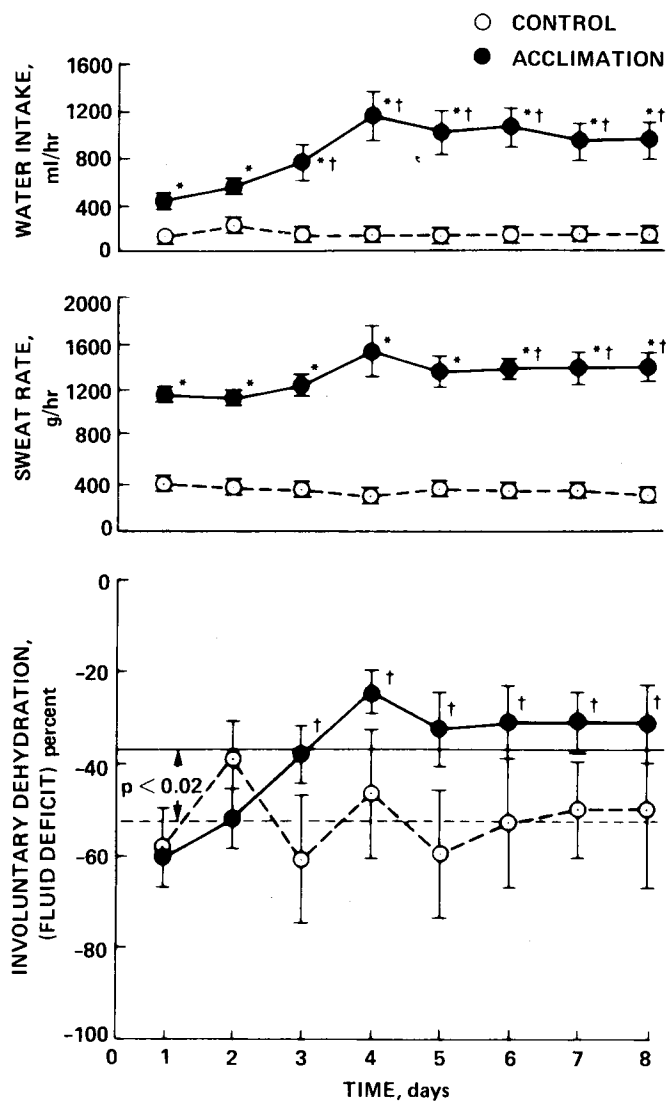
Figure 4.– Mean (±SE) water intake, sweat rate, and levels of involuntary dehydration (fluid deficit) during exercise in cool (control) and exercise in hot (acclimation) environments. Dashed line is mean of control data (52%); solid line is mean of acclimation data (37%). *p < 0.5 from corresponding control data; †p < 0.05 from day 1 data. From Greenleaf et al. (1983) with permission.

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Report Documentation Page

1. Report No. NASA TM-101011	2. Government Accession No.	3. Recipient's Catalog No.

4. Title and Subtitle	5. Report Date
Acclimatization to Heat in Humans	April 1989
	6. Performing Organization Code

7. Author(s)	8. Performing Organization Report No.
John E. Greenleaf and Hanna Kaciuba-Uscilko*	A-88222
	10. Work Unit No.
9. Performing Organization Name and Address	199-21-12-07
	11. Contract or Grant No.
Ames Research Center Moffett Field, CA 94035	
	13. Type of Report and Period Covered
12. Sponsoring Agency Name and Address	Technical Memorandum
National Aeronautics and Space Administration Washington, DC 20546-0001	14. Sponsoring Agency Code

15. Supplementary Notes

Point of Contact: John E. Greenleaf, Ames Research Center, MS 239-17, Moffett Field, CA 94035
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16. Abstract

This review concerns the responses and mechanisms of both natural and artificial acclimatization to a hot environment in mammals with specific reference to humans. The purpose is to provide basic information for designers of thermal protection systems and countermeasures for astronauts during intra- and extravehicular activity. Areas covered are energy metabolism, thermal balance at rest and during exercise, water and electrolyte balance during exercise and immobilization, and heat diseases.

17. Key Words (Suggested by Author(s))	18. Distribution Statement
Human physiology Thermal regulation Exercise physiology	Unlimited – Unclassified Subject category: 51

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of pages	22. Price
Unclassified	Unclassified	44	A03

NASA FORM 1626 OCT 86   For sale by the National Technical Information Service, Springfield, Virginia 22161