5-1-1937

Acute disorders in man caused by heat

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ACUTE DISORDERS IN MAN CAUSED BY HEAT

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SENIOR THESIS

PRESENTED TO THE COLLEGE OF MEDICINE,
UNIVERSITY OF NEBRASKA, OMAHA, 1937.
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INTRODUCTION

This paper deals with the acute effects of excessive heat on the body. In it are considered the morbid effects of heat under conditions of such severity as to go beyond the limits of physiological toleration. These effects are manifested in three rather specific conditions; heat cramps, heat stroke, and heat exhaustion.
PHYSIOLOGICAL ASPECT

Man's ability to endure for a time wide ranges of external temperature is due mainly to the fact that his body temperature is maintained at a fairly constant level in spite of wide variations in the surrounding environmental temperature. Man is a warm blooded animal. By this is meant that he possesses the possibility of physiologically regulating his body temperature so as to maintain it at a fairly constant level. This is necessary for such constant degrees of activity as are required. He cannot suffer the variations of physical ability possessed by the cold blooded animals. With his present thermoregulatory system, man is able to live and work under great extremes in existing environmental conditions. It is interesting to note that as early as 1775 Blagden and Fordyce(3), in an attempt to find how high a temperature man could withstand, found that a man could tolerate, for a period of fifteen minutes, an exposure to dry atmosphere of 250°F. without ill effects; while a beefsteak exposed to the same temperature was cooked in thirteen minutes.

The heat generated by combustion of the various foodstuffs during metabolism in the body causes the body temperature under normal conditions. This amounts to approximately 3000 calories in the average individ-
The temperature is about 1°F. higher in the rectum than in the mouth or axillae. The average mouth temperature is 98.6°F. and the average rectal 99.6°F. Of course, individuals vary and the normal range of temperature is from 97.5°F. to 99°F. There is a slight diurnal change in the temperature as a rule. The temperature is lowered in the morning, gradually rises to a maximum (sometimes 1°F.) between 5:00 p.m. and 7:00 p.m., then falls during the night.

The body temperature can vary over rather wide limits, but the optimum physiological function is carried out within rather limited ranges. If there is much change from the normal temperature of the individual, the physiology of body function is upset and pathological changes occur.

In order to maintain a relatively constant temperature under conditions in which heat production may increase as high as three times over the normal, or the environmental temperature may be 20°F., or more, higher than body temperature; corresponding adjustments are required in heat dissipation. Heat is lost in the following ways: (1) in the excreta, (2) in warming inspired air, (3) in evaporating water from the lungs, and (4) in the evaporation of water from the skin. (69) It is estimated that of the heat lost in these different ways approx-
imately 5% is lost by the warming of food, respired air and excretions, another 15% by the saturation of air in the lungs with water and heat absorbed in eliminating carbon dioxide from the lungs. (73) The remaining 80% is lost by radiation, conduction, and water evaporation from the skin. (73)

Since 80% of the heat loss of the body is accomplished by radiation, conduction, and evaporation from the skin these are the main channels of the elimination of heat from the body, and are most concerned in response to high environmental temperature.

Loss of heat by radiation is governed by several factors. The proportion of body surface to volume is of great importance. The surface of the body increases as the square of its area, while the body volume increases as the cube of the body area. The surface area in proportion to the body volume is much greater in small, or lean people than in large or, obese individuals. The latter do not lose nearly so much heat by radiation as do the former. Radiation from the skin is more efficient the nearer the skin temperature is to that of the interior of the body. Vascular dilatation in the skin will therefore increase radiation of heat, provided that the external environment of the skin is at a lower temperature than the skin. If not, the reverse will be true. Pigmentation
of the skin may have some effect on radiation. It is thought that a dark skin may radiate more heat than a light skin.

Loss of heat by conduction and convection depends upon the difference in temperature existing between the body and the objects or air with which it is in contact. The movement of the air and its moisture content have a great effect on its ability to absorb heat. The addition of moisture to the air greatly increases its power to absorb and radiate heat. The actual amount of moisture in the air is the absolute humidity. The relative humidity is the ratio between the actual amount of moisture present and the amount that could be held at the same temperature if fully saturated.

A substantial amount of heat is required to convert a liquid, especially water, into its corresponding vapor. This amounts to 570 small calories (.57 large calories) per gram of water vaporized. For this reason vaporization of sweat exerts a powerful cooling effect on the body. When the temperature of the air outside the body is above that of the body, it is the only means possessed by the body to maintain the body temperature. The relative humidity is an important factor in the process of evaporation. The rapidity of evaporation at a given temperature is inversely proportional to the relative humidity.
Another important factor is the physiological saturation deficit, that is, the difference between the amounts of water held in the air at the surrounding temperature and when saturated at skin temperature. This difference determines the cooling effect of sweating.

The amount of water vaporized is determined by the content of water in the skin, by the degree of sweating, and by the temperature, humidity, and the movement of air, in accordance with physical principles. When heat dissipation equals the heat eliminated the body temperature remains constant. If it is less the body temperature increases and hyperpyrexia results.

When the body is exposed to an external temperature higher than that of the body, the sweat glands are stimulated. The respiration increases in rate and amplitude and, thus, considerable heat is lost through the saturation of larger quantities of air in the lungs.

The mechanisms which regulate heat dissipation appear to be controlled largely by nerves, and hence finally involve the vasomotor, sweat, respiratory, and cardiac centers of the medulla. The involvement of a higher integrating center is demonstrated by the fact that regulation of heat dissipation is not abolished by progressively higher sections of the brain stem, until the diencephalic region is reached. It is above the level...
of the pons and probably about the level of the superior colliculus. (73)

The heat regulating center or centers is set into action by alterations of the blood temperature or reflexively through the nerves; at least that is the prevailing opinion at the present time. (73) The thermal regulator may be regarded as a slow reserve mechanism preventing too great a lowering of the body temperature; the nervous regulator may be regarded as a rapid one, more commonly called into action to keep the body temperature constant. (73)

Water is lost from the skin by two processes differentiated as "insensible" and "sensible" perspiration; the former referring to evaporation of invisible water on the skin, and the latter to an accumulated film of visible water on the skin. (73) The present consensus of opinion is that insensible perspiration is due to the passage of water through the skin by osmosis. (73) Water lost by this method amounts to approximately 30 grams at rest in the prone position. Of this type of water elimination about 15% is from the hands and feet. (73) It is little affected by the fluid intake but is changed by the blood flow through the skin.

The final physiological mechanism concerned with heat effects on the body is that of sweating. Sweating is
of two types, mental and thermal. Mental sweating appears under mental stress and is ordinarily confined to the palms of the hands, soles of the feet, axillae, and forehead. Thermal sweating involves the entire body. (14)

Normally in cool, comfortable surroundings we are cooled mainly by radiation and convection. The cool moving air carries away the heat from the skin. Loss of heat by evaporation of sweat from the skin is kept at a minimum, while that from the respiratory membrane is kept at a maximum by breathing cool air at a low vapor tension. (33) The vessels of the skin are kept in good tone. Activity and metabolism are kept at a good level by the cool touch of the moving air. (33)

Hard physical labor may be kept up many hours if the external temperature and humidity are low, and especially if there is a good wind movement. Under these conditions the increased rate of heat production by the body is met by an increased rate of heat dissipation (mainly by evaporation of sweat), and the internal temperature remains relatively constant. If the individual is exposed to a warm environment the heat loss will be diminished and the surface temperature will rise. If no vasomotor adjustments were possible the temperature of the body as a whole would also rise. However, warmth leads to a vascular dilatation which increases the heat
conductivity of the subcutaneous tissue and skin, due to an increased blood flow allowing heat to be conducted with more efficiency, and the surface temperature to be raised to the necessary extent without alteration of the internal temperature. (2) If on the other hand, the external temperature is so high that heat dissipation cannot occur the body temperature will rise and a state of physical exhaustion will be brought about promptly. (4)

With increased temperature and sweating it can be seen that the power of the surrounding air to hold moisture and thus cause cooling by evaporation is important. The following table will show the increased evaporative power of air due to ability to hold moisture with increasing temperature. (3)

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Water (grains/cu.ft)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30°F</td>
<td>1.94</td>
</tr>
<tr>
<td>50°F</td>
<td>4.08</td>
</tr>
<tr>
<td>70°F</td>
<td>7.98</td>
</tr>
<tr>
<td>80°F</td>
<td>10.9</td>
</tr>
<tr>
<td>90°F</td>
<td>14.7</td>
</tr>
<tr>
<td>100°F</td>
<td>19.7</td>
</tr>
</tbody>
</table>

Air movement enormously increases the evaporative power of the surrounding air, provided the air is saturated at a point below body temperature, by bringing fresh unsaturated air into contact with the body. As every gram of water evaporated from the skin or respiratory membrane
carries away 0.59 calories of heat, the emergency method of body cooling by sweating is very effective. (33)

The effect of enclosure of still air is very important. When surrounded by stagnant moist air in an enclosure at body temperature, any loss of heat by convection, radiation, or evaporation becomes impossible. The body temperature rises and this in turn accelerates the rate of combustion within the body and a vicious circle is established.

Hunt (35) who lived in Deccan, India, where during the summer months the maximum temperature is rarely under 100°F. and in some parts 110°F. or more for long periods of time and there is a low moisture content of the air, found that health and comfort can be maintained in dry heat for long periods of time where the temperature is above that of the body. However, the water requirements of the body are very high due to the amount of sweating necessary to neutralize the heat of metabolism and also the heat added by radiation and conduction.

The water loss in man under normal conditions of rest is approximately 550 c.c. (61) With sweating this is increased depending upon the amount of sweating done. A healthy laborer may lose as high as seven or eight liters of fluid during the day, with only 7% of this being excreted by the kidneys. (13) Moss (48) found, that
If the work output be constant, an increase in sweating accompanies an increase of temperature. He also found that in colliers there is a difference in the sweating between a man acclimatized to hard manual labor under high temperature conditions and men not acclimatized. Under nearly equal conditions the acclimatized individual loses twice as much weight by sweating and suffers no ill effect. Haldane(26) found that colliers not acclimatized to heat the maximum amount of sweat lost per hour was about 1.4 pounds whereas in colliers accustomed to the work the maximum loss was 5.25 pounds. Due to the large loss of fluid by sweating much more water is drunk to supply the body with fluid. If this is not done there is danger of dehydration. In experimental work done at Boulder Dam the fluid intake was found to be greatly increased and closely related to the change in temperature.(64) The volume of urine remained unchanged but the specific gravity was increased.(64)

The sweat glands secrete a hypotonic solution containing sodium chloride, potassium chloride, and low concentrations of lactic acid, urea, and other substances. (64) The composition of sweat is not constant but varies with internal and external environment. The only proven function of the sweat glands is their part in temperature regulation. There would be no disadvantage if sweat were
free from electrolytes, since the kidneys and lungs are able to maintain the normal acid-base equilibrium.\textsuperscript{(12)} The sweat glands when excreting large amounts of sweat may upset this equilibrium and cause harmful effects to the body.

The amounts of sodium chloride contained by the sweat is not definitely agreed upon. This is probably due to the fact that the concentration of sodium chloride varies with different bodily conditions. Drinker\textsuperscript{(14)} places the sodium chloride concentration in sweat at about 0.3\%. Moss\textsuperscript{(48)} found it varied from 0.325\% to 0.168\% with an average of 0.224\%. Dill and co-workers \textsuperscript{(12)} from work done at Boulder Dam found it to be 0.2\% before acclimatization and 0.15\% afterward.

With excretion of large amounts of sweat, the sodium chloride concentration is reduced while the total amount of fluid lost is increased. This is substantiated by the work of Moss\textsuperscript{(48)}, Haldane\textsuperscript{(26)}, Dill and co-workers\textsuperscript{(12)} and Talbot, Edwards and Dill\textsuperscript{(64)}.

Fishberg and Bierman\textsuperscript{(20)}, in a study of patients submitted to high temperatures with a great amount of sweating, found that the economy of base in sweating was due to the ability of the sweat glands to excrete sweat at a much lower pH than the blood plasma. The excretion of lactic acid and lactates resulted in the sparing of
fixed base because of proportion of lactic acid passing out unionized in the sweat. The presence of lactic acid and lactates acting as a buffer to prevent the lowering of the pH below four with damage to the skin.

Due to amount of sodium chloride present in the sweat it can be seen that a large amount may be lost from the body with excessive sweating. Drinker(14) estimates that a person can lose 25 grams of sodium chloride during a day's work in a hot environment. The average excretion of sodium chloride is about 12 grams per day. From this comparison it can be seen the degree to which work in high temperature can deplete the body chlorides. This depletion is one of the most definite and important effects of high temperature on the body.
HEAT CRAMPS

Definition

Heat cramps is the term applied to painful spasms of the voluntary muscles following muscular activity in a high environmental temperature, caused by a decrease of the sodium and chloride concentrations in the blood.

History

The failure of the human body to cope with a high environmental temperature is generally associated with the clinical entities known as heat stroke and heat exhaustion, which have been known since early medical history. Heat cramps is of more recent origin. Probably the reason for its not being recognized as a clinical entity is due to the various descriptive terms by which it has been known. It has variously been called miner's cramp, stoker's cramp, mill cramps, fireman's cramp, and even "the bends" because of the contortions of the individual during an attack. These terms associate the disease with its occupational occurrence rather than with the causative mechanism.

Talbott(65) in his monograph on heat cramps states that one of the earliest reports on the disease was issued from the gold mines of Virginia City, Nevada, and published in 1878. Coplin(8) in 1892 noticed the muscle cramps occurring in workers laboring under high
temperatures, with high humidity in the air and a slow air movement. He thought it an early symptom of heat stroke. Edsall(16) described two cases of the disease very well in 1904, but did not think it was due to the high external temperature because the patients did not show much elevation of body temperature. He was one of the first to start work on the pathological physiology of the disease. Two of the most significant of his findings were the diminished excretion of chloride, and the increased excretion of nitrogen from patients after the onset of the cramps.(17) In 1909 after further study he separated the condition from heat stroke and heat exhaustion. Elliot(18) in 1908 gave a report of what he termed heat prostration among the stokers and coal passers on board the battleships of the United States Navy, but which, from the description of the symptoms shown by the patients, was evidently heat cramps instead. He noted it occurred among the men working in high temperatures with a low air humidity. Welsh(71) in 1909 gave a description of a typical heat cramp case. Since this time numerous cases have been reported. With the development of industrial methods more and more cases came to note, and the increasing frequency of the condition in the workers in hot industries was a great incentive to the recognition, and research done pertaining to this disease.
Incidence and Distribution

The disease is widespread and may be found in any industry where great heat is present constantly.\(^{(61)}\) It is essentially an occupational disease and the majority of the cases are seen by the industrial surgeons. The incidence has increased co-incident with the rapid growth of the various industries of this country. This has been especially true of the iron and steel mills. Heat cramps are most common in the summer months, particularly in certain endemic centers. These are mainly in the eastern United States, in the iron and steel mills, and other hot industries. The incidence is highest in the "heavy" industries where a high working temperature is customary and in certain other occupations in temperate and tropical climates that require strenuous muscular activity under excessive heat.\(^{(65)}\) The distribution is worldwide and there is no racial immunity, negroes being afflicted as readily as whites.\(^{(18)}\)

It is difficult to make an estimate of the number of cases per year as many cases are not reported. Many of the workers who have an attack do not see a physician. This makes any estimation very faulty. However, Starkov \(^{(61)}\) reports that in the mines located in the Ural mountain district of Russia up to 50% of the workers suffered attacks before prophylactic methods were intro-
duced. Even as late as 1929 there were 164 cases, in 1930 there were 155 cases, in 1931 there were 131 cases and in 1932 there were 131 cases. Dill(13) states that in a steel mill employing 12,000 workers it was not rare to have twenty-two men put to bed in one day before prophylaxis was begun.

The disease is confined almost entirely to adult males. This is apparently due to the fact that only adult males are exposed to the causative factors. Practically all cases are found in individuals between twenty and forty-five years.

Etiology

Heat cramps are caused by dehydration of the body, and a decrease of the blood chlorides brought about by muscular activity in an environment with a high temperature. The causative mechanism is the lowering of sodium and chlorides in the blood serum due to loss of these in the sweat without adequate replacement.(63) Elliot(18) in 1908 was the first to have this conception of the etiology. At this time the work on heat cramps was in an early stage and there was little known about the cause of the condition. His work was not widely publicized and it was not confirmed until later. Moss(48) in 1923 after extensive work among colliers laboring at high temperatures in England thought that heat cramps were
due to a water poisoning of the muscles, caused by the large amounts of water drunk by the laborers. In this he was supported by Haldane(26) who thought it was a form of water poisoning of the muscles brought about by the combination of a great loss of chloride from the body by sweating, excessive drinking of water, and temporary paralysis of renal excretion. He based his idea of the temporary paralysis of renal excretion on his and Priestly's(25) work where they thought they had found evidence that during hard work there is a complete or almost complete cessation of kidney function owing to the blood being diverted to the muscles. The kidneys do not regulate the sodium chloride water ratio so a change in osmotic pressure results. This causes a fall in the osmotic pressure of the blood which causes the cramp symptoms.(27) Allen(1) agrees with this theory. Later experiments do not agree with this idea. Talbott and co-workers(64) from experiments and observations at Boulder Dam conclude that there is no shunting of the blood from the kidneys to the periphery.

In a study of heat cramp cases at Boulder Dam in 1933 Brock and Dill(14) reported that the one striking finding in these cases was the reduction of the serum chloride concentration brought about by excessive sweating and inadequate intake of sodium chloride. In 1933
Talbott and Michaelson (63) advanced the hypothesis that the etiologic factor causing heat cramps was a loss of base, chlorides, and water from the body principally by way of the sweat glands without adequate replacement of the same. In fairness it must be said that Oswald (49) in 1923 and Sir Josiah Court (9) in 1924 suggested that the cause of heat cramps was due to loss of sodium chloride from the body by sweating, but neither substantiated their suggestions with proof. Starkov (61) in 1936 agrees: "the etiologic factor is a water-salt metabolism disturbance due to a decrease of the chlorides of the blood due to a loss of chlorides by sweating". Hospital studies show a very considerable depletion of sodium chloride in the body with the blood plasma concentration of sodium and the chloride below the threshold level of the kidney. (13) McCance (42) reports that in the experiments on normal subjects with a sodium chloride deficiency muscle cramps occurred on exercise.

A high working temperature associated with a high atmospheric temperature is an important predisposing factor. It is noticed that there are more cases when the outside temperature is also high. This is probably due to the fact that the individual cannot cool off after work and continue to sweat. The failure of the body to adapt itself to a rapidly increasing environment-
al temperature is, also, a factor on the production of heat cramp. The condition is seen early in heat waves whereas heat exhaustion and heat stroke are seen later. (65) Of less importance than the temperature is the humidity of the air. It is incorrect that the cramps occur only in an atmosphere with a relatively high humidity. (65) It is true that the most cases occur in an atmosphere of relative high humidity but that is because these cases occur in enclosed places and in locations which favor the presence of high humidity. (24) A low wind velocity favors decrease cooling by less evaporation of sweat, so it also may be considered a contributory factor.

Acclimatization, also, plays a part. The individual, who is not used to the high temperature, and whose sweat glands have not become adapted to it, runs an increased risk because of great sodium chloride loss to the body.

Vomiting favors development of heat cramps because fluid and chloride are lost. It is probably for this reason that alcohol is considered a contributory factor. The man who drinks alcohol to excess may vomit thus losing valuable body constituents which favor the development of heat cramps (65) However, alcohol per se has an injurious effect on the heat regulating mechanisms of the body, and by this action may bring about heat cramps. (73)
Diarrhea causes the same results as vomiting and is important for the same reason. Lack of appetite also helps decrease the fluid and mineral intake and so is a predisposing factor.

Poor general hygiene, irregular habits, and lack of sleep prevent the body from operating at its maximum efficiency, and thus must be considered as accessory factors in the development of heat cramps.

A recent attack of cramps which has been inadequately treated favors the development of another attack which is usually more severe because of the greater decrease in the concentrations of sodium and chloride in the blood.

Pathology

Exposure to a high air temperature with slight air motion and radiant heat places a terrific strain on the heat regulating system of the body. There is a large amount of fluid lost from the body. The amount varying, according to different investigators, from five to ten liters a day. Most of this is lost as sweat. Since sweat is figured to contain on an average of about 0.2% of sodium chloride it can be seen that an enormous amount of sodium chloride is lost. Drinker(14) finds that as much as 22.5 grams of sodium chloride may be lost. The usual intake of sodium chloride is ten to twenty grams per day, so the individual creates a salt deficit, and
must draw on the body sodium chloride unless provision is made for an added intake of same. Patients suffering from heat cramp often show a decrease in body weight. This increases with improvement until it reaches normal. This loss in weight is due to increased sweating.

Talbott (65) states that "the concentration changes in the protein of the serum and hemoglobin of the blood are of a magnitude infrequently seen in clinical medicine". In cases he studied there appeared an increase in the serum protein of from 0.5 to 5.6 grams. Dill and co-workers (12) also report an increase in the serum protein concentration. The total serum protein increase is accounted for by the increased serum globulin. (65) The serum albumin was below 3.4% in seven patients, where a high total globulin was present. The serum globulin showed an increase of 2.5 to 7.4%. The severity of the cramps, however, is not proportional to the increased concentrations of protein. The increase seems to be principally an index of the degree of anhydremia present, as it is decreased when the anhydremia is lessened. (65) Dill, Brock, and Edwards (11) found that with sweating, there is a loss of sodium chloride in the sweat and a decrease in the body weight associated with an increase in the serum protein concentration of the blood when water is drunk whenever wanted. This would seem to indicate that
dehydration occurs with a sodium chloride deficiency even with an adequate water intake. So it is probable that while the changes in the serum protein concentration are very great, these changes are secondary through the dehydration of the blood and are not related primarily to the pathogenesis of the disease. Not only is there a loss of water from the serum but also from the red blood cells varying from 0.1% to 5%.

The hemoglobin is increased and the increased concentration of the hemoglobin corresponds roughly to the change in the serum protein concentration. The maximum increased notice was 5.6 volume per cent. (65) An increase in the hemoglobin has been noted by Starkov (61), also.

The increase in the number of red blood corpuscles corresponds closely to the increase in hemoglobin. The highest count found was 6,480,000, an increase of 34% over that found on recovery. (65) Heilman and Montgomery (31) found an increase of 1,000,000 to 3,000,000 red blood cells per cubic millimeter in their series of heat cramp cases.

Talbott (65) concludes that the blood volume is diminished. This is in agreement with the findings of Starkov (61) and Heilman and Montgomery. (31)

There is a slight increase in the number of white
blood cells with a relative increase in the polymorphonuclear neutrophilic leucocytes. The average increase in the number of white cells was from 4,000 to 6,000 per cu.mm. (65) The significance is unknown and is probably a non-specific reaction caused by the heat.

Both Heilman and Montgomery (31) and Talbott (65) found a decrease in the plasma chlorides. The former found the average range of reduction was 4% to 10% with a maximum of 17% in one case. Talbott (65) found that changes in the concentration of chlorides and sodium are always present. The serum chloride concentration was below 100 milli-equivalents with a minimum of 79.8 milli-equivalents.

The concentration of electrolytes in the serum is altered in mild cases as well as severe one. All the electrolytes show some variation from the normal except the bicarbonate ion. This may be increased, decreased, or normal. (65) The variation of pH was from 7.08 to 7.48 in the cases studied by Talbott. (65) In most cases it was below 7.4. This apparent acidosis was due to a depletion of base rather than an accumulation of acids. (65)

An increase in the concentration of inorganic phosphate in the serum is a constant finding. The reason for this is unknown for there is little evidence of a primary disturbance of phosphate metabolism. (65) The lactic
acid content of the blood shows little variation from normal. Changes in the sodium concentration of the serum are similar to those of the chloride concentration. In thirty-two patients Talbott(65) noted the minimal serum concentration of sodium was 121 milli-equivalents and the maximum 140 milli-equivalents. In all patients there is a return to normal of the sodium and chloride concentrations of the serum with recovery. The serum sodium is the only inorganic base whose concentration is known to be decreased.(65) The serum potassium is normal or increased slightly. The serum calcium is not decreased. The blood sugar remains about normal. The non-protein nitrogen content of the serum is usually elevated. This is probably due to the increased metabolism caused by the high temperature. It is usually only moderately elevated, 10 to 25 milligrams per cent.

In relation to the pathological findings in the blood it is interesting to note that McCance(42) in a study of experimental sodium chloride deficiency in men found that the venous blood was more viscous than normal and of a darker red color. The number of red cells per cubic millimeter was increased. The hemoglobin content of the blood was increased. There was an increase in the concentration of the plasma protein of the blood. There was a decrease in the concentration of the sodium and the chlorides of the blood plasma.
With this there was also a decrease in chloride concentration of the red blood cells. There was an increase in the concentration of the blood urea. In the subjects studied there was a loss of weight.

Flinn(21) working with dogs subjected to high temperatures found that the alkali reserve of the body fell with an increased temperature. The carbon dioxide content of the blood was decreased. There was little change in the blood sugar level. There was an increase in the solids of the blood and the blood concentration was raised.

The results of these two experiments agree with and bear out the pathology found by the investigators working with heat cramp cases and lend additional evidence to their correctness.

Symptoms

In few diseases of the human body is the onset more abrupt or dramatic than in heat cramps. There may be prodromal symptoms or there may not. Often prodromal symptoms are vague and not associated with the onset of the disease. The prodromal periods may vary from hours to days before the attack. During this time there may be a mild vertigo, headache, or a premonitory fibrillary twitching of the muscles. Diarrhea and vomiting may occur as early as a week before an attack.(31) It is questionable whether diarrhea and vomiting are prodrom-
al symptoms or contribory factors to the developments of the attack of heat cramps by loss of fluid and chloride from the body.

The most prominent symptoms are the muscle spasms, or cramps and the patient's complaint of the severe pain that accompanies them. Fibrillary twitching of the muscles may precede the attack and be so active that there appears to be an undulating mass under the skin. Very gentle palpation of the muscles or a sudden pressure on the muscles may produce a violent contraction of the muscles following which they become boardlike and painful. (65) The patient has no control over the contractions and cannot produce voluntary movement in the muscles affected. The severe pain stops immediately with the end of the muscle cramps. The muscles most used are more apt to be involved. This probably accounts for the apparent predilection of certain groups of muscles for the cramps. These are the muscles of the shoulders, of the calf of the leg, and of the flexor group of the forearm. (31) Cramps have been observed in the muscles of the face, neck, upper arm, hands, back, upper leg, and feet also. (65) The muscles of the fingers and abdominal wall are affected to a lesser degree. There is apparently no vasomotor changes in the involved parts. (65)

The severity of the muscle cramps is variable. The
patient may have a mild attack with only the muscles of the fingers or toes involved, or the attack may be so widespread as to resemble a tetanic seizure. The pain is in proportion to the severity of the attack. The time of the day the attacks occur is not characteristic. They may come on at any time while the individual is at work, or after he has gone home, or during the night. (65)

There is evidence that the pain associated with the muscle cramps originates in the muscle itself. Weisenberg (70) reports an interesting case of a cook, suffering from muscle cramps who had, when a child, anterior poliomyelitis. This had left him with a paralysis and atrophy of the left leg with a loss of the reflexes. When the patient suffered with heat cramps the muscle spasms were as marked in this leg as elsewhere in the body, and the pain in this leg was as severe. This is rather reliable evidence that the spasms and pain are produced in the muscle by degenerative changes, and not by involvement of the nervous system. That the nervous system is not involved is further born out by the fact that Trouseau's sign and Chvostek's sign are negative. (70)

There may be vomiting during an attack but it is not a constant finding. Gastric distress and nausea are fairly common findings. Heilman and Montgomery (31) think that the vomiting may be due to spasms of the involvum-
tary muscle of the gastro-intestinal tract. Talbott(65) thinks there is little evidence that involuntary muscle is ever involved.

Physical examination of the patient shows little that is consistent, except the muscle spasm. The rectal temperature is seldom below 98°F. or above 100°F. The pulse is rapid, regular and strong. The blood pressure is usually somewhat lowered. The respirations are usually increased. If the chest muscles are involved and the spasms sufficiently severe there may be some interference with breathing.(31) In which case the face may be flushed with a tendency toward cyanosis. There may be profuse sweating or the sweating may be suppressed.

Laboratory examination will show a decreased chloride content of the urine or an absence of chlorides. There will be an increased red blood cell count and an increased hemoglobin content of the blood. The serum protein of the blood will show an increase. The non-protein nitrogen in the blood may be slightly increased.

Complications

Complications are rare. No reference was made to any complications in the literature I reviewed. This is probably due to the disease being caused by a deficiency of sodium and chloride which are a part of every diet and are replaced in a short time if there is not an ex-
cessive loss. Another factor is that the condition, when it is severe, is in its onset so spectacular and the pain so great that the patient either gets away from the adverse environment under his own power or with the assistance of his companions. Still another factor is that it is primarily an industrial disease, and the patients suffering a severe attack are seen by a physician in a short time and adequate treatment instigated.

Diagnosis

Heat exhaustion, heat stroke, and heat cramps have a common point of origin. They are found in men doing hard work at high temperatures and may be associated with profuse sweating, large fluid intake and diminished urine output. For this reason they may enter into a differential diagnosis. Usually this is not difficult. Muscle cramps are not commonly found with heat exhaustion. In heat exhaustion weakness and signs of shock predominate. There may be convulsions in heat stroke which simulate muscle cramps but the presence of an exceedingly high body temperature, dry, burning skin, coma, incontinence and other signs of central nervous system irritation which are present in heat stroke will differentiate the two conditions.

Nocturnal cramps and cramps following violent exer-
cise may not be easily differentiated from heat cramps. It is further confusing because heat cramps may not develop until several hours after the work period. Because the time of onset may be about the same time nocturnal cramps occur, any workman who develops muscle spasms during sleep after working at a high temperature should probably be treated as a case of heat cramps. (65)

Abdominal colic must be considered, when the muscle cramps are confined to the abdominal muscles. Less frequently encountered diseases that might be confused with heat cramp are: delirium tremens, strychnine poisoning, epilepsy, tetany, trichinosis, uremia, hysteria, and writer's cramp. (65) A good history and careful physical examination should make a diagnosis of any of these conditions. Convulsions, as a symptom of an organic disease, should be kept in mind until a complete study makes the diagnosis.

Prognosis

The prognosis for patients with the disease are excellent. Most of the cases will recover without treatment, but the pain and duration of the disease are much reduced by treatment.

Prophylaxis

The men doing hard strenuous labor under high
temperature conditions should be in a state of good physical health. They should have frequent physical examinations to ascertain if their good physical condition is being maintained. They should have a suitable environment when not working. Adequate rest and sleep are essential. A well balanced diet should be maintained. Contrary to the old idea that during hot weather or in a hot environment the individual should eat little protein; it is not reasonable to expect that men doing hard work can maintain their body needs without an adequate amount of protein in the diet to provide for the repair of the body tissue.

It is now recognized that under such conditions means must be provided to insure a replacement of the sodium and chlorides lost by excessive sweating. It is interesting to note that the coal miners of England have for years drunk salted beer to prevent heat cramps. (48) This may be accomplished by adding sodium chloride to the drinking water of the men. The favored amount is from 0.1% to 0.2% in the water. This is not objectionable to the taste of the water if it is cooled below 50°F. (31) If over 0.2% of sodium chloride is added to the water the taste may be objectionable to the workmen. Another means of providing sodium chloride is by having the laborers take sodium chloride tablets. If this is done it
is preferable that enteric coated tablets should be used because sodium chloride in such concentration may be irritable to the gastric mucosa. (31) Glover (24) recommends that a 16 grain salt tablet be taken with every drink of water. Mechanical dispensers are placed near the drinking water fountains. (24) Investigators who report good results from the use of sodium chloride as a prophylactic measure are: Starkov (61), Dill, Brock, Edwards, and Kennedy (13), Drinker (14), Heilman and Montgomery (31), Johnson (36), Brock and Dill (4), Talbott (65), Schofield (54), Derrich (10), Oswald (49), McCord and Ferenbaugh (43), Court (9), Fantus (19), Eaton (15), Brockbank (5), Moss (48), and Glover (24). The results by this method of prophylaxis are convincing. Dill, Brock, Edwards and Kennedy (13) report, from their experience in the steel mills of Pennsylvania, that in 12,000 workers there were only eleven cases of heat cramps in 1936. Previous to the use of salinized water the incidence had been much higher. Heilman and Montgomery (31) report a drop in cases of heat cramps from 589 cases in 1927 to 326 cases in 1928. Glover (24) states that in a plant employing between 1,000 to 1,800 workers there was not a single case in 1928 when this method of prophylaxis was used. In a steel mill employing 3,000 to 3,500 men forty-eight days were lost in 1927, nine days in 1928, four days in 1929 and none in 1930.
Another factor in reducing the number of heat cramp cases is the acclimatization of new workers to the conditions under which they must labor. This takes several days. This is done because the sweat from individuals not used to a hot environment has a greater sodium chloride concentration and thus the loss of sodium chloride is much greater than when the laborers become used to the heated conditions. (65) (61)

Reduction of heat as much as possible, proper ventilation, and short rest periods in a cooler place during the work shift are also of benefit to the workers.

Treatment

The patient should be removed to a cooler place and placed in a recumbent position. Rest alone may be sufficient in mild cases. The mainstay and most important part of the treatment is the replacing of the sodium and chlorides lost from the body. Sodium chloride is given by mouth, rectum, hyperdermoclysis or intravenously. The therapeutic effect of sodium chloride has been known for some time. Elliot (18) in 1908 used saline enemas with excellent results in severe cases. It is now generally accepted that the most rapidly effective treatment is the administration of physiological salt solution intravenously. This gives relief almost immediately in many cases and in
all cases in a few hours. Heilman and Montgomery(31) believe that the addition of sodium bicarbonate(2%) gives more rapid relief in severe cases. Schofield(54) and Fantus(19) believe that the addition of dextrose(5%) to the physiological saline is beneficial. Since the blood sugar is not reduced the rationality of this is not clear. However, the dextrose will help relieve fatigue and combat any toxemia that may be present. Subcutaneous administration of saline gives slower relief and it may be irritating when given in large amounts. Oral administration of sodium chloride, either in solution or tablet form, will give relief but it takes longer for the therapeutic result to be gained. Milk is a good source of sodium chloride and may be used if saline solution is distasteful to the patient. A nice feature about sodium chloride therapy is that there is little danger to the patient if excess amounts are given.

Morphine has been used to give relief from the cramps but it is not as effective as might be thought.(63) Welsh(71) used apomorphine, 1/12 to 1/20 grain, which gave immediate relaxation of the muscles and relief from pain. However, this usually caused vomiting and there was marked general prostration. This left the patient in about as poor a condition as he was before the drug was administered.

The patient should be placed on an adequate, well
balanced diet with sufficient mineral content and not allowed to return to work until the sodium and chloride content of the blood is known to be normal. This will help prevent the development of subsequent attacks of heat cramps.
HEAT STROKE

Definition

Heat stroke is a disease caused by exposure to a high external temperature in which the heat regulating mechanism seems to be overwhelmed, with high body temperature and general prostration resulting.

History

The injurious effects of excessive heat on the human body have been known since the earliest history of mankind. The pathological entity known as heat stroke is one of the oldest known diseases. Until modern times it was known as sun stroke and this name is still used by many.

At least three cases are mentioned in the Bible. In the fourth chapter of the second Book of Kings there is recorded an instance of the disease affecting a lad who went out to help his father with the harvesting of the grain. In the apocryphal Book of Judith there is given an account of an adult who was attacked by heat stroke, which resulted in his death. (69) This patient, too, was struck during the harvest time. In the Book of Jonah there is mentioned again a case of heat stroke. This time it was Jonah, himself. In Isaiah (49:10) and the Psalms (121:6) mention is made of the sun's injurious effects on the human body.
The ancient Egyptians were acquainted with the disease and they associated the injurious effects of the sun's rays to Sirius, the Dog Star, and dog days. This was probably due to the fact that the star was most iminent then, and their conception of medicine was allied with astrology and religion. (65)

The physicians of Ancient Greece and Rome were familiar with the disease but confused it with other forms of cerebral apoplexy. (69) This misconception was carried through the ages up to the nineteenth century.

In 1789 Dr. Benjamin Rush called attention to a disorder occasioned by the drinking of cold water in warm weather and described the symptoms of heat stroke very accurately. (67) He was the first to give a clear differentiation of heat stroke from other forms of apoplexy. The idea that the drinking of cold water was the cause of heat stroke was so thoroughly accepted, that in the summer of 1814 the pumps in Philadelphia, Pennsylvania, were posted with notices of caution against drinking of cold water while warm, by order of the Humane Society. (67)

Gerhard clearly separated heat stroke from apoplexy by noting that there were no gross changes in the brain with heat stroke. (67)

Condie was the first to describe heat stroke as
occurring elsewhere than in the sunlight. He reported a patient who was attacked while in church. (67). The best illustration, which arose after this observation was made, that it could occur elsewhere is found in the many names the disease has received. Some of these are: ictus calorus, prostratic thermic, coup de chaleur, calpo die calore, hirtzschlag, wormschlag, heat apoplexy, heat asphyxia, heat dyspnea, thermic fever, ardent fever, phrenitis aestive, phrenitis calenture, erethismus tropicus, insolation, sirissia, sun stroke, and heat hyperpyrexia. (67)

Incidence and Distribution

The distribution is widespread in the tropical and temperate climates. It is more frequent in some areas than in others due to prevailing weather conditions such as high temperature, high humidity, and low air movement. (22) Rarely does a heat wave occur in this country without some cases of heat stroke occurring. There are more cases in some years than others due to more severe weather conditions. White people are more subject to develop the disease in a tropical climate than the natives, although the latter are by no means immune. (57) Persons with systemic diseases, especially cardiac trouble, are apt to have heat stroke. It may occur in postoperative surgical patients. Moschovitz (47) reports twelve cases, and Martin (41) reports four
cases occurring in postoperative patients.

Willcox(72) and Hearne(30) found the number of cases of heat stroke was increased by army maneuvers during especially hot weather.

Accurate data as to the number of cases of heat stroke occurring in a year is not possible. Heat exhaustion and heat stroke are classified by many as the same disease so the reports do not list these diseases separately. Heat stroke is more common in cities during hot weather and in other areas of heavy concentration of population such as army camps, etc. In July, 1916, in the city of Chicago there was admitted to Cook County hospital one hundred and fifty-eight patients suffering from heat stroke and heat exhaustion. From the description of the symptoms shown by the patients and the high mortality it would appear that most of these were heat stroke victims.(23) In July, 1743, eleven thousand persons are said to have perished from the effects of heat in Peking, China.(38)

The disease is of course more common in the summer months than during the rest of the year. Persons of either sex may develop heat stroke, but it is more common in males. This is probably due to the fact that men are doing harder work and more exposed to high temperature than females. Below the age of twenty it is of about equal
incidence in both sexes.(57) Infants under one year are more susceptible than those above that age.(55) This is probably caused by the inefficiency of the heat regulating mechanism which is not fully developed. It is most common between the ages of twenty and fifty-nine years. (56) Undoubtedly the reason for this is that these are the periods of greatest activity and exposure in the individual's life.

There is no immunity conferred by an attack. On the contrary an individual who has had an attack of heat stroke is more susceptible to heat and liable to have another attack.

Etiology

Until the past twenty-five years sun stroke was thought to be a separate disease and due to a peculiar action of rays from the sun on the body, especially the brain. For this reason protecting headgear was worn in the tropics. Thick hats or a drape from the back of the hat over the neck was considered very important for protection from these rays. This idea that sun stroke is separate from heat stroke and due solely to rays other than heat rays from the sun has now been generally discarded. Willcox,(72) and Hearne(29) from a wide experience with numerous cases in the World War declare there is no evidence that other rays than heat rays cause heat stroke.
Numerous instances are recorded of cases of heat stroke with no history of exposure to the sun. (46) Marsh (40) in a series of experiments on rabbits to determine the factors principally involved in the production of heat stroke decided that it was clear that heat alone was responsible and that the rays of the sun had no direct action on the brain tissue. He also found that heat stroke occurred independent of the sodium and chloride concentration of the blood. In this he was supported by Drs. Martin, Pembrey, Castellini, and Evans.

It is now generally accepted that sun stroke is not apart from heat stroke and the principle etiologic factor is a high environmental temperature with a thermoregulatory incompetency which allows the body temperature to rise to such a height that it may be incompatible with life. (59) This incompetency may be due to failure or exhaustion of the heat regulating mechanism. (33)

A high relative humidity and absence of, or slight air movement are important contributing factors because these do not favor evaporation of sweat from the body and thus help raise the body temperature. Heat stroke can occur, however, with a low humidity if the temperature is high. (46)

Willcox (72) from experiences in Mesopotamia, where the incidence of heat stroke was high, states that a
maximum shade temperature of 110°F. appears to be dangerous. When this was reached cases of heat stroke began to appear and with each degree rise above this there was an increasing number of cases. Temperatures of 120°F. or above, were extremely dangerous and on these days a large number of cases occurred in spite of all precautions. Morton(46) reports that with a temperature of 110°F., to 120°F., or more, cases of heat stroke will occur when the humidity is low.

The effect of heat is cumulative. One or two days of very hot weather does not cause very many cases but it is a succession of several hot days that causes the greatest number of cases. The cumulative effect was shown according to Willcox(72) by the number of individuals who developed heat stroke after the temperature had fallen considerably.

Other etiologic agents have been advanced at different times. Milner(45), because of noting the large number of patients who had malaria, suggested that heat stroke was a symptom of malignant tertian malaria. Sinderson (58) felt that heat stroke should not be considered a disease, but as a symptom that was apt to occur with sandfly fever.

As a subsidiary factor in the production of heat stroke, aside from the high environmental temperature, Hearne(29 (30) feels that suppression of sweating leads
to the development of heat stroke. This view is not accepted by Willcox(72) who reports cases having heat stroke who have not had suppression of sweating. Milner (45) thinks the patient ceases to sweat because he has heat stroke, not that he gets heat stroke because of suppression of sweating.

Other predisposing factors are alcohol because it interferes with the heat regulatory system of the body.(72)(31423). This is especially true if the alcohol is taken during the day when the individual is exposed to the highest temperature.(72) Exertion is important, particularly, hard labor. Lack of an adequate supply of drinking water is an important contributing factor. A large amount of water is necessary due to the increased amount of sweating. Any disease causing fever, especially malaria, sandfly fever, typhus fever, and typhoid fever, is known to predispose. Constipation is considered a contributory factor by Willcox(72), and Hamilton and co-workers(28). The latter feel that heavy clothing and hot quarters favor the development of the disease.

Pathology

It is thought by Willcox(72) Smith(59), and Morton(46) that there is an intoxication of the body. The clinical history of prolonged hyperpyrexia and profound toxemia are in support of this. Willcox(72) found acetic acid
end diacetic acid in the urine of a number of cases. Hyaline casts and albumin were also found in the urine. This would indicate that there is some damage to the kidney function. There was also an excess amount of indican in the urine during the acute stage.

Postmortem examination shows there is edema of the brain or leptomeninges or both. There is marked generalized passive hyperemia, especially of the brain and lungs. Edema of the lungs is present. The spleen may show a hyperplasia. Clowdy swelling is found in the liver, kidneys, and myocardium. Petechial hemorrhages are found in the mucous membrane, skin, brain, and viscera. Fatty changes may be found in the liver. The cerebral spinal fluid is increased in amount, and is clear and colorless.

McKenzie and LaCount(44) found a foremen magnum furrow in the brain stem in fourteen cases of a total of thirty-seven necropsies performed. In one case small softenings in the lenticular nucleus are noted. The brain in some cases was swollen with flattened convolutions and tightly closed sulci. The arachnoid meshwork was obliterated and the parietal and visceral layers in intimate contact. This condition was always most marked at the vertex and sides of the cerebrum.

Morton(46) reports that the heart is contracted and hard, and the blood very liquid in heat stroke cases.
There is usually a slight leucocytosis with a relative increase in the polymorphonuclear neutrophilic leucocytes. This probably has no specific relationship to heat stroke because it is present with other disorders due to high temperature.

Symptoms

The onset may be abrupt and acute or may develop over a period of three or four days. Headache, faintness, vertigo, malaise, restlessness, and nausea and vomiting may precede the acute onset. Frequency of micturition is a characteristic early symptom. Sweating is often decreased or suppressed before the acute attack. The temperature may be raised to 100°F. to 102°F. with the skin dry and hot for as long as forty-eight hours before the severe symptoms develop.

With the acute onset there may be sudden collapse and coma. Severe headache is often present. The patient may show great mental excitement or delirium. Diarrhea is often present early. With coma there is incontinence of the urine and feces. The patient may complain of tinnitus, photophobia, diplopia, and extreme emotional instability. (31)

The skin is dry and burning with the face moderately livid. Spots of petechial hemorrhage may be found on the surface of the skin. The pupils may be dilated.
in the early stages but are constricted later, especially if the patient is comatose. The pulse is irregular and rapid; 120 to 140 beats per minute is often found. The blood pressure is elevated. The respiration is at first rapid and labored, later it is irregular and stertorous. The body temperature is elevated usually from 3°F. to 10°F. Commonly the temperature is from 106°F to 110°F. It has been noted to be as high as 115°F. The rectal temperature is often 3°F to 4°F higher than the oral or axillary temperature. There is often a fibrillary twitching of the muscles. Convulsions, commonly occurring, may be clonic or tonic. Coma is a frequent finding. The reflexes may be absent. They vary with the depth of the coma. The knee jerks are almost always absent during the acute state of the disease. The presence of knee jerks was considered by Willcox (72) as a valuable prognostic sign, for when they occurred there was less danger of relapse.

The loss or diminishment of the reflexes is in accord with experiments of Heyman (32) who demonstrated that the central nervous system of animals was sensitive to heat. Von Leuwen and Van de Made (39) found that the reflex response may be considerably modified by temperature and that the maximum response occurred when the temperature of the spinal cat was about 38°C. At a tem-
perature above this the reflexes diminished rapidly.

Pulmonary edema and the signs accompanying it develop late in the course of the disease.

Lumbar puncture shows a clear fluid under increased pressure. Albumin and casts (hyaline and granular) are found in the urine. (23)

Complications

The complications following heat stroke are mainly of nervous origin. Marked mental symptoms often remain for weeks. Irritability, mental confusion, and delusions are the most common mental disturbances. They usually clear up in time. Defective articulation occurred as a complication in our cases mentioned by Willcox. (72) Multiple neuritis associated with weakness of the legs, the tibialis anticus muscle being most affected, was a complication in two cases of the same series. (72) Headache, impairment of memory, and incoordination are not uncommon. (31) Weisenberg (70) reports a case of severe heat stroke followed by multiple nervous lesions producing acute cerebellar ataxia, loss of speech, and spastic symptoms. He also states that he has never found a single instance of sensory involvement following heat stroke.

Willcox (72) found that marked cardiac dilatation, often associated with a systolic murmur, occurred in
several severe cases. This remains for several weeks and needs special care. He also found that bronchitis and congestion of the lungs developed in some cases.

Recurrences of heat stroke are apt to occur unless proper precautions are taken because these individuals are more susceptible to the effects of heat.

Diagnosis

The history, sudden onset, high temperature, and signs of central nervous system involvement will usually make the diagnosis. If the patient is elderly cerebral hemorrhage must be considered. Malignant malaria and sandfly fever must be ruled out. Diabetic coma, epilepsy, acute alcoholism, and shock may bear some similarity to heat stroke. These may be differentiated by history and observation.

Prognosis

Heat stroke is a serious condition which requires rapid, adequate treatment. If the patient is seen early and the temperature is reduced promptly the prognosis is favorable. The later the patient is seen the less favorable the prognosis. Sequellae are liable to develop and require attention.
Prophylaxis

Heat stroke can be prevented if proper measures are taken. During hot weather, work should not be done during the hottest part of the day. There should be adequate ventilation of working and living quarters. The new method of air cooling or conditioning residences and places of business will no doubt be of great value in preventing heat stroke.

A large amount of water must be drunk to replace that lost by sweating. Alcoholic beverages should not be taken especially during the day. Hamilton(28) and Wilcox(72) feel that constipation is a contributing factor and relief of this is of some value in preventing the disease. The clothing should be light and loose so as to interfere as little as possible with the movement of air about the body. The diet should be light and consist mainly of carbohydrates. Protein, however, must be taken in sufficient quantities to provide for body repair. Persons who have systemic disease, especially cardiac disease, or are debilitated, or obese, may require special measures such as cool baths or cold applications to the head as a prophylactic measure.
Treatment

The first and most important procedure in treatment is the reduction of the high temperature. There are several ways of doing this. The most effective method is to sprinkle cool water over the patient, or place a wet sheet over the patient, and place fans so that there is good air movement over the wet surface. The skin should be rubbed vigorously at short intervals to keep the blood flowing through it. If not the circulation will be decreased and the cooling effect will be partially lost. This is more effective than rubbing the patient with ice. While evaporation at body temperature carries away 0.59 calories per gram of water evaporated, the melting of ice takes away only 0.08 calories per gram of water melted. (33) Rubbing the skin with ice also obstructs evaporation. Seventy grams of water evaporated takes away as much heat as one thousand grams of ice melted. (33)

More drastic cooling procedures such as packing in ice, ice water baths, ice water enemas, or ice water gavage should not be employed owing to the danger of producing collapse. (19) The temperature should not be reduced below 103°F. or 102°F. because of the circulatory disturbance present. This usually requires from ten to thirty minutes. Fluid should not be given until the tem-
Temperature is below 103°F. or less because pulmonary edema or cardiac failure may occur at any time. The increasing of fluids would contribute to this hazard.

If there are any signs of cardiac failure or pulmonary edema venesection is beneficial. Good results have been obtained by withdrawing 400 c.c. to 500 c.c. of blood. (19) This is also of value in stopping or preventing the occurrence of convulsions because it tends to reduce intracranial pressure. For the same reason withdrawal of 30 c.c. to 40 c.c. of cerebral spinal fluid may give relief. (19) If convulsions are very severe an anesthesia may be necessary to control them. Chloroform or ether may be used. The latter is preferable.

Cardiac stimulation may be necessary if signs of cardiac failure develop. Caffeine sodio-benzoate, digitalis, strychnine, or epinephrine may be administered.

For restlessness and delirium morphine, chloral hydrate, or bromides are useful. Mechanical restraints may be necessary.

Diaphoretics have no effect; apparently because the sweating mechanism is exhausted and fails to respond to stimulation. (33)

Failure of respiration is best treated by artificial respiration or oxygen.

Prolonged bed rest is indicated after the acute
attack has passed. The patient must be watched closely and kept cool or relapse may occur. The patient should be warned that alcoholic indulgence is dangerous during hot weather, and that he will probably be more susceptible to heat effects due to increased resistance against high temperatures.
HEAT EXHAUSTION

Definition

Heat exhaustion is a disease caused by a high environmental temperature and characterized by pronounced weakness, dizziness, pallor, profuse perspiration, diminution of urine, and acceleration of the pulse rate and respiration. It is sometimes called heat prostration.

History

The condition has not been separated from heat stroke until the past two or three decades. For this reason it is difficult to obtain information. The disease has undoubtedly been present as long as man has been exposed to high temperature.

Incidence and History

It is most common during the hot times of the year. It is especially prone to appear in elderly or debilitated individuals. Shattuck and Hillferty(56) found it causes more deaths after the age of sixty years than heat stroke, while heat stroke causes more deaths between the ages of twenty to sixty years. Among the deaths attributed to heat as a secondary factor, heat exhaustion is more common than heat stroke. (56). Starkov(61) reports that it occurs chiefly in new men in the steel mills who have not become acclimatized or who cannot become acclimatized. It is apparently
more common in the rural communities and small cities than in the more densely populated cities.\textsuperscript{(56)} The reason for this is difficult to explain.

**Etiology**

The condition is caused by a high environmental temperature which results in an inadequate heat regulation in the body. Morton\textsuperscript{(46)} believes that it is due to an intoxication caused by a general parboiling of the body. He thinks it may be due to the presence of some substance like histamine which has been produced in the body as a result of faulty metabolism. Sayer\textsuperscript{(53)} concludes that the symptoms of heat exhaustion represent the sustaining, and finally failing efforts upon the part of the body to overcome its adverse environment.

Diseases of the circulatory system and lowered vitality favor the heat exhaustion response to high temperature. Overwork, lack of sleep, constipation, and poor physical condition have a predisposing effect toward the development of the disease.

**Pathology**

The two important features are dehydration of the body and a shock-like condition of the patient. The blood sugar is reduced and may be as low as 64.3 milli-
grams per cent. (31) The plasma chlorides of the blood are reduced and the red blood cell count is slightly increased, but not to such a degree as is found in heat cramps. (30) The urinary output is decreased. (46) This is due to the dehydration of the body.

Symptoms

The onset is usually rapid but in some cases there are prodromal symptoms for several days. The patient complains of weakness, malaise, anorexia, constipation, and diminution of urine. This stage in severe cases is followed by collapse.

The actual onset is rapid with weakness, giddiness, faintness, and a feeling of oppression and prostration. The onset is not necessarily associated with exertion. Morton (46) tells of a case occurring at night. The patient resembles a case of shock. The pulse is weak, rapid, and thready. The blood pressure is low, the respiration rapid and irregular. The patient is pale, bathed in cold sweat and mentally apprehensive. The temperature may be subnormal or slightly elevated (102°-103°F.). Morton (46) stated that although the mouth and axillary temperature may be subnormal or normal, the rectal temperature is always above normal, usually 101°F. The eyeballs are shrunken and the pupils dilated. The skin is cold, clammy, and moist with profuse perspiration.
The skin is usually pale but some cyanosis may be present.

The patient presents a picture of exhaustion and may show anxiety. In severe cases there may be stupor or unconsciousness.(31)

According to Morton(46) in some cases after the immediate effects of the shock wear off persistent vomiting followed, the vomitus becoming bile stained. Some of his patients were in severe discomfort due to cramps in the legs and abdominal muscles.

The above author(46) states that an increase in the amount of urine excreted is always an early favorable sign of recovery. As the patient responds to treatment, the vomiting and cramps cease, the mouth temperature increases and a slight fever appears which lasts for three of four days. In some cases headache persisted for a week or ten days after convalescence is established.

Complications

Complications are not common. The patient may develop heat stroke unless precautions are taken to prevent this. Most cases recover in two or three days, sometimes in six or seven hours.
Diagnosis

The condition may be confused with heat cramps or heat stroke. In heat cramps the painful muscular spasms, lack of subnormal temperature, strong heart action and absence of shock-like symptoms will make the differentiation. In heat stroke there is a highly elevated temperature, dry, burning skin, absence of sweating, and symptoms of central nervous system involvement such as stupor, delirium, and convulsions.

The patient should be examined to determine if there is any injury or condition that might be the cause of the shock.

Prognosis

The prognosis is good. Most cases recover in two or three days, often within six or seven hours. The mortality is slight.

Prophylaxis

The incidence can be much reduced by precautionary measures. Improvement of working conditions by better ventilation, cool rest rooms and proper rest periods during working hours will do much to prevent the disease in industry. The acclimatization of the individual to the heated conditions should always be done. Heilman and Montgomery(31) find the use of jelly drops and sugar do much to reduce the number of cases. During
their experiments in a steel mill by use of these the number of cases dropped from 326 in 1928 to 67 in 1935. The workers also reported that they felt much less exhaustion at the end of the days work. Beverages containing sugar are beneficial. Lemonade is particularly recommended.(31) The clothing of the workmen should be light and loose. The water supply should be adequate to provide for loss of fluid by sweating. Enough sodium chloride should be taken to insure against sodium chloride deficiency, although there is no evidence to show that lack of sodium chloride may cause the disease. Constipation should be avoided. Debilitated individuals should be closely observed if exposed to high temperature.

Treatment

If the temperature is subnormal the patient should be kept warm to increase temperature up to normal. The patient should be put to rest. Stimulation may be necessary to offset the depression. Caffeine sodio-benzoate is very effective for depression. Strychnine may also be used. Fantus(19) uses these two drugs alternately. Caffeine sodio-benzoate 0.25 grams to 0.5 grams being given subcutaneously every four hours, and strychnine sulphate 2 milligrams every four hours. If the patient is in a critical condition aromatic spirits of ammonis,
1 c.c. in water, subcutaneously, orally or by inhalation gives a brief response which is sometimes valuable in promoting the absorption of the previous drugs. (19)

Morton (46) has good results with an intravenous injection of a 2% sodium bicarbonate in normal saline plus 5% dextrose. This supplies fluid base and sugar to increase the decreased blood sugar base and chlorides. Heilman and Montgomery (31) report excellent results from the administration of glucose and sodium bicarbonate intravenously.
CONCLUSIONS

1. There is sufficient evidence to justify the consideration of the acute effects of heat on the human body as being manifested by three clinical entities. These are heat cramps, heat stroke, and heat exhaustion.

2. Heat cramps are caused by a decrease in the sodium and chloride concentration of the blood and a dehydration of the body. This is brought about by the loss of sodium chloride in the sweat, without adequate replacement of the same.

3. The administration of sodium chloride is very effective in the prophylaxis and treatment of heat cramps.

4. Heat stroke is caused by an overwhelming of the heat regulatory mechanism possessed by the body, due to exposure to high external temperature. There is no reliable evidence that other than the heat rays of the sun are the cause of heat stroke.

5. In heat stroke there are no significant changes in the blood.

6. Heat exhaustion is not as severe a condition as heat stroke. It differs sufficiently from heat stroke to merit separate consideration. The clinical picture of fatigue and shock, and the decreased blood sugar concentration are its outstanding characteristics.


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