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Heatstroke : a review of etiology and treatment

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HEATSTROKE

A REVIEW OF ETIOLOGY AND TREATMENT

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TABLE OF CONTENTS

Incidence of Heatstroke

The problem of morbidity caused by excessively high body temperatures is still very real particularly among military personnel in hot climates, the elderly and debilitated during summer heat waves, in surgical patients, and in chronic alcoholics. Stonehill and Keil[1] reported on heat illness at Lacklund AFB and noted that in the summer of 1956 there were thirteen cases of heatstroke and two deaths. In the summer of 1957 there were thirty-seven cases of heatstroke. Nationally, according to the Bureau of Vital Statistics[2] there were one hundred and sixty-eight deaths due to excess heat and insulation in 1960 and one hundred and ninety-one deaths in 1961. These are largely preventable deaths with an adequate knowledge of factors predisposing and relating to heat illness and appropriate treatment of those affected. There may also be many more deaths due to heatstroke than are actually recorded as such on the death certificate because of other conditions present such as known heart disease and this may be recorded as the primary cause of death.

This may occur particularly in the elderly with other illnesses present and who die during summer heat waves in greater numbers than at other times of the year.

Heat Induced Illnesses

Sodeman[3] reviews three conditions in man which are related to excess environmental heat or heat beyond what the body can tolerate. Heat exhaustion is the first condition to be considered and is characterized by a sensation of weakness, possible fainting, and a moist, pale skin. There may be moderate hypotension, and the rectal temperature is either normal or only slightly elevated. This will respond quickly to rest in a quiet place with adequate cooling.

Heat cramps may occur in those undergoing strenuous muscular activity and perspiring freely and thereby losing a great deal of water and sodium chloride. The resulting electrolyte imbalance tends to produce very painful muscle spasms of the voluntary muscles. This may be prevented by supplementary salt tablets along with adequate water intake in those doing active physical labor in a hot climate.

A particular type of dermatosis has been noted in some hot environmental situations associated with headache, dizziness, and warmth. Sweating ceases over the body and extremities but may continue on the face and neck. It is thought that there may be a hyperkeratotic plugging of sweat ducts. A papular skin eruption occurs with desquamation later. Sweating resumes after a time of rest in a cool environment. This phenomenon has been termed 'thermogenic anhidrosis'.

Heatstroke, discussed by Harrison[4], is the most severe condition of the four. The rectal body temperature is over 106 degrees Fahrenheit, the pulse is rapid, and there may be a sudden cessation of sweating just prior to onset of the high body temperature. There may also be visual changes, irritability, nausea, vomiting, hot, dry, red skin, flaccid muscles, and collapse or coma. Once these findings are present the mortality rate ranges between 10% and 80%, as noted by Ferris, et al[5].

Body Temperature Regulation

Because we are dealing with problems resulting

from excessive body temperatures the various methods by which the body relieves itself of heat must be considered. These fall into two broad divisions consisting of physical methods and physiological methods. Maxcy[6] discusses the physical methods among which is conduction which is defined as the transfer of body heat to the air in contact with the skin and clothing. After sufficient heat has passed into this air to warm it and air becomes less dense and rises, being replaced by cooler air. This system functions well when the air temperature is lower than the body temperature.

Heat is also lost from the body by the principle of convection in which the motion of air itself tends to bring cooler air to the body and remove heated air.

By radiation heat is absorbed by the body and the chief source of this radiation is the sun. The wearing of adequate, porous clothing tends to limit the amount absorbed and to facilitate heat loss.

Evaporation of sweat is the primary source of heat loss from the body at high temperatures where other

methods mentioned may be of small effect. However, when the relative humidity reaches 100% evaporation ceases. In environments with relative humidities in the 80% and 90% range problems with poor evaporation also occur.

Guyton[7] refers to physiological methods of heat dissipation which are the result of stimulation of the supraoptic and preoptic areas of the anterior hypothalamus possibly by increased temperature of the blood supply to result in stimulations to the sweat glands, stimulation of the vasodilator nerves, and reciprocal inhibition of the posterior hypothalamus which removes vasoconstrictor tone to the skin vessels resulting in more vasodilatation and also decreasing the metabolic stimulus to body cells. Vasodilation facilitates heat loss in increasing surface blood flow and therefore, skin temperature increases allowing more heat loss by the processes of conduction and convection. Accompanying the shift in blood flow there may be a drop in blood pressure with an increase in heart rate. According to Maxcy, noticeable perspiration usually occurs with external temperatures about 81-88

degrees Fahrenheit at rest and at lower temperatures if active labor. Up to two liters per hour of fluid may be lost with marked sweating but only that which evaporates functions to cool the individual.

Factors Predisposing to Heatstroke

Certain factors predispose to the development of heatstroke and may be grouped under environmental, physical, and iatrogenic factors. Environmental factors consist first of high air temperatures, chiefly those above the body temperature of 98.6 degrees Fahrenheit. When this situation occurs heat loss by the body can take place only by evaporation of sweat. If the second factor of high humidity accompanies high air temperatures heat loss by evaporation is markedly depressed and the body may be unable to cool itself adequately resulting in heatstroke.

Physical factors include acclimatization which is a term referring to adjustment to high temperatures after exposure of about two to four weeks so that fairly normal physical labor can be done without serious results. Hertig et al[8] has noted some criteria

for the presence of acclimatization which include a decrease in pulse rate, less rectal temperature rise, lower skin temperature, increased ability to complete assigned tasks, and more subjective comfort on heat exposure. The body mechanisms leading to acclimatization have been studied and Bass et al[9] indicated that the major adaptations are cardiovascular and urinary with conservation of sodium and chloride to compensate for their losses in sweat with resulting isotonic expansion of plasma and interstitial cell volumns. Conn[10] postulated that increased adrenal cortical activity with reduction of sodium and chloride ions both in sweat and urine was of chief importance and also that a negative nitrogen balance occurred. He gave Desoxycorticosterone acetate experimentally to men in various stages of acclimatization and found that after its abrupt cessation the process of acclimatization seemed to be impeded and acclimatization seemed to be lost temporarily in those previously acclimatized.

It has been noted by a number of authors including Ferris that heat stroke is not uncommon in the elderly

and debilitated. This may be partially explained on the basis of decreased ability to adapt readily to the stress of heat due to compromized cardiovascular system, inadequate living arrangements with no facilities for good temperature control, and poor nutrition. Chronic alcoholism also figures in mortality due to heatstroke. With inadequate hydration or already present electrolyte imbalance there is less ability to respond to salt and water losses via sweat thus making compensation difficult. Knockel et al[11] relate that in the state of acclimatization sodium ions are conserved by the body and potassium ions lost and this partial hypokalemia may contribute to heatstroke occurring.

Harris et al[12] and Chapman and Bean[13] reported on cases of heatstroke felt to be related to iatrogenic causes. A number of cases have occurred following surgical proceedures or during anesthesia. These may be due in part to some dehydration of the patient because of food and drink restrictions with inadequate intravenous feeding, overheated surgery suites combined with a heavily draped patient thereby decreasing the body's ability to dispense excess heat. The premedication with

drugs such as atropine which blocks acetylcholine, the mediator for stimulation of the sweat glands may prevent heat loss by the action of the sweat glands.

Etiology of Heatstroke

The exact etiology of heatstroke has been a subject for for conjecture and remains so at the present time. A number of theories have been proposed under the primary headings of primary cardiovascular injury, sweat gland fatigue, and direct hypothalamic injury. Bazett[14] postulates that the cardiovascular system beats the brunt of injury in high temperatures and as a result of alterations here heatstroke can occur. With high body temperatures there occurs dilatation of peripheral arterioles, veins, and capillaries, in an effort to dispell the heat. This dilatation causes decreased peripheral resistance and greater blood flow peripherally. To compensate for this there is central splanchnic vasoconstriction. On the balance between these depends the level of blood pressure and cardiac output. With more heat stress there is sweating leading to some decrease in blood volume with then inadequate venous

return causing central vessel and arteriolar constriction but eventually resulting in a more inadequate venous return generating inadequate cardiac output which results in some of the pathological changes found throughout the body in cases of heatstroke.

Gold[15] postulates that a high output type of cardiac failure may be responsible for the manifestations of heatstroke. A massive cutaneous circulation is set up and acts like an arterio-venous shunt of large dimension finally resulting in heart failure.

Schwartz[16] has studied the sweat gland in relation to its secretory failure upon prolonged stimulation with cholinergic drugs. He found that once failure to secrete occurred it took about six hours for the glands in the unacclimatized to again secrete. This recovery time was cut in half in acclimatized individuals. He indicates that sweat gland fatigue and the retarded restoration of function are prime factors in the induction and progression of heatstroke. Burch[17] noted decreased sweating in patients with congestive heart failure and high venous pressures and postulated that as the venous pressure increased, the sweating

decreased but as the venous pressure decreased, sweating increased thus linking venous pressure changes with sweat gland function.

Malamud et al[18] postulated a primary irreparable injury to the thermostatic function of the hypothalamus so that the autonomic nervous system was unable to reestablish sweating or maintain an adequate peripheral circulation. There has been no evidence to prove or disprove this theory. At present no one theory is completly acceptable and exact cause remains to be worked out.

Complications of Heatstroke

The pathological complications of heatstroke are found throughout the body. A case report by Knockel et al of heatstroke showed collapse, tonic seizures, and shock followed by oliguria and uremia with death occurring on the forty-third post attack day due to massive gastrointestinal hemorrhage. At autopsy there was found a myocardial infarction with no gross abnormality of the coronary arteries. The bone marrow was hypercellular with erythroid hyperplasia and

almost no megakaryocytes. The liver showed bile stasis, the pancreas had evidence of fat necrosis, and the kidneys were markedly congested with bile staining and heme casts in the tubules. Mucosal ulcerations were found in the esophagus and stomach. The brain was slightly congested.

Another case with eventual recovery reported by Vescia et al[19] had liver damage during the acute phase of his illness and liver biopsy showed focal bile collections and areas of inflammation with occasional mitoses. This patient had neurological involvement with slurred speech, cogwheel rigidity, and frontal headaches. Thrombocytopenia, gastro-intestinal ulceration, and transient nonspecific T wave changes on the electrocardiogram were noted.

Concerning the problem of jaundice in these patients Herman and Sullivan[20] reviewed the literature and found that in two hundred and twenty-four cases of heatstroke jaundice was present in ten cases. They also reported a case of a patient with heatstroke who manifested jaundice, neruological changes, petechiae and purpura of the skin, subconjunctival

hemorrhages of the eyes, and fatty metamorphosis on liver biopsy. Baxter and Teschan[21] reported on three cases with similar findings to those previously mentioned in that petechial hemorrhages, liver necrosis, focal neurological degeneration, and kidney damage were present.

Freedman and Rourke[22] postulated that there may be a selective sensitivity of the Purkinjie cells of the cerebellum in heatstroke with resultant dysarthria ataxia, and dysdiadokinesis.

Gore[23] reviewed seventeen fatal cases of heatstroke and attributed the pathological changes as largely due to tissue anoxia as a result of cardiovascular changes and also direct hyperthermic effects on essential cellular enzymes and enzyme reactions.

Treatment

Over the many years in which heatstroke has been recognized as a specific entity there has been no completely desirable form of treatment after the body temperature becomes 106 degrees Fahrenheit or greater. Attempts were made to cool the body as rapidly as

possible but whatever method was used the mortality still remained high. Treatment often used in heat-stroke includes the following methods. The patient should be placed in a cool room and his clothing removed. Sheets may be wetted in ice water and wrapped loosely about the patient and fans placed to blow air across his body. Ice water sponging may be used or the patient may be rubbed with ice and massaged. The massaging tends to facilitate blood flow into the surface vessels which may have become constricted with exposure to ice or cold water while the internal part of the body was still very warm. This should be maintained until the body temperature becomes almost normal.

A second form of treatment as noted by Ferris has been the immediate immersion of the patient in a tub of ice water until the body temperature is 101 degrees Fahrenheit. Because of the difficulty of having a tub of ice water available in certain instances, the marked shock to the patient with rigidity, violent struggling, and delirium, this method of treatment has not been desirable but has remained in use because

of lack of any more effective methods. Immersion in a tub of ice water may be a fatal shock to an elderly person and the more conservative treatment of sponging and massage would be indicated.

Steroids have been tried to reduce body temperature in heatstroke as a result of their antipyretic effects in pyrexia in pneumonia, typhoid fever, Rocky Mountain Spotted Fever, and generalized peritonitis. Waugh[24] used evaporative cooling and cortisone in the treatment of a case of heatstroke but concluded that the cortisone had no apparent effect. Schillhammer and Massonneau[25] reported on three cases of heatstroke with body temperatures of 107.8 degrees, 108 degrees, and 109 degrees Fahrenheit respectively, rectally, with ice water baths, hydrocortisone intravenously, Thorazine, Levophed, and oxygen with all three patients surviving. They used the steroids because of the hypothesis of there being an acute, severe, adrenal depletion due to stress. They concluded that the steroids seemed to be of some benefit. A difference of opinion on effectiveness of this mode of therapy still remains and more studies must be done.

Treatment tried on a patient with heatstroke with an axillary temperature of over 109 degrees Fahrenheit by Hoaglund et al[26] consisted of wet towels, oxygen, intravenous dextrose in normal saline, Promethazine intravenously, then Dipyrone and Chlorpromazine, and Hydrocortisone. Three days later kidney and liver function tests were normal and he recovered uneventfully. Dipyrone is an antipyretic agent and Chlorpromazine is a muscle relaxant. These act to decrease metabolism and therefore the oxygen demand of the tissues. The Phenothiazines inhibit the thermoregulatory centers to cause vasodilatation facilitating heat loss. They postulated that the most severe lesions in heatstroke are due to anoxia and therefore the proper treatment should decrease the oxygen demand. Immersion in ice water would be directly opposed to this by tending to increase the oxygen demand.

Laborit, cited by Jesati,[27] attributes an increase in thermolysis to be responsible for the hypothermia when thermoregulation is blocked by the central action of Chlorpromazine. The homothermic person is made

poikilothermic and his defenses against hypothermia are impossible. Jesati also successfully treated a hyperthermic patient with intravenous Chlorpromazine and topical ice bags with good results.

Until further studies on the treatment of heatstroke by drugs are done and their effectiveness proven we must continue to use the methods previously discussed of some form of sponging and massaging and ice water immersion if the patient may be expected to tolerate this drastic proceedure. A safe, effective drug would be a much more convenient way of dealing with the problem than present methods.

Prevention

As in almost any disease prevention plays a key role in heatstroke. Many unnecessary deaths in both the young and the old may be prevented by adequate knowledge of the measures which can be taken to prevent heatstroke. With recognition of the entity of heatstroke and knowledge of groups in whom it is apt to occur doctors can more easily recognize it and also council people on preventive measures and early

recognition of the illness.

Measures to prevent heatstroke should be taken in every circumstance in which it could occur. Among people such as outdoor construction workers, farmers, and servicemen an attempt should be made to correlate the amount and difficulty of the work performed with the environmental heat, humidity, and wind velocity. In high temperatures heavy physical work should either be left for cooler periods of the day or frequent rest periods should be interspersed with shorter work periods. Since body salt and water will be depleted through sweating, salt tablets and water or 0.1% Sodium Chloride should be available for drinking.

Correct clothing is protective in hot environments. The cloth should be white or light colored, to reflect the shorter infrared light rays, of light weight material, and thin. For protection against radiant heat outdoors metal lined helmets are effective. In some cases metal treated clothing has been tried and found effective.

The problem of cooling buildings has been partly overcome by the use of air conditioning. This is a desirable method but not always feasible due to the

expense involved. People living in crowded, poor living quarters often cannot afford air conditioning. Many of the nursing homes for the elderly may not be air conditioned and other methods of cooling must be used. Aluminum foil or shades at windows help to keep out some heat. Fans help to circulate the air. Some buildings may be cooled at night by using large exhaust fans in the attic and driving out the heat from the house and drawing in cooler night air. Water sprayed on a roof or flooding of the roof with water is helpful. The circulation of cold water through pipes and radiators may be used.

In very hot, humid weather cool, moist sponging with water or alcohol will help cool an elderly of sick person. Exercise and direct exposure to the heat of the sun should be minimized. Outdoor work whould be done in the cooler parts of the day.

Any person showing symptoms of heat exhaustion, heat cramps, or cessation of sweating should be promptly moved to a cool area and treated for whatever condition he has at the time to prevent further illness and possible heatstroke. With potentially better awareness

and preventive measures this illness should decrease.

Summary

Heat exhaustion, heat cramps, thermogenic anhidrosis, and heatstroke are caused by excessive temperatures. Conduction, convection, radiation, and evaporation are physical methods of temperature regulation. Physiological methods include sweating and peripheral vasodilatation. High air temperatures and high humidity combined make heat loss by the body very difficult.

Lack of acclimatization, old age, chronic disease, alcoholism, dehydration, and electrolyte imbalance predispose to heatstroke.

The exact etiology of heatstroke is postulated to be on three aspects; cardiovascular, sweat gland fatigue, or direct hypothalamic injury with no one theory proven. Complications of the disease are manifold and tend to involve all major body systems.

An adequate and safe form of therapy is still being sought. Drug therapy is a new field and needs more study. Prevention plays a major role in this illness. With better awareness and better preventive measures and treatment mortality should decrease.

Conclusions

1. Heatstroke is a medical emergency.

2. It is a preventable cause of death.

3. The exact etiology is still unknown.

4. Complications are severe and often fatal.

5. Antipyretic drugs need to be further investigated as a method of treatment.

6. Preventive measures are of great importance.

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BIBLIOGRAPHY

1. Stonehill, R.B., Keil, P.G., Successful Preventive Medical Measures against Heat Illness at Lacklund AFB, Am. J. Pub. Health. 51.1:586-590, 1961.

2. U.S. Bureau of the Census, Statistical Abstract of the United States, Washington, D.C. 84th edition, 66, 1963.

3. Bean, William, Physical and Toxic Agents. (In: Sodeman, W.A., ed. and others, Pathologic Physiology, 3rd, ed., Phil. and London, W.B. Saunders Co., 1961. p. 287-317).

4. Talbott, J.H., Disorders due to High Environmental Temperatures, (In: Harrison, T.R., ed. and others, Principles of Internal Medicine, 4th ed., New York, McGraw-Hill Book Co., 1962, p. 833-835).

5. Ferris, E.B., and others, Heatstroke: Clinical and Chemical Observations on 44 Cases, J. Clin. Invest. 17:249-262, 1939.

6. Baetzer A.M., Environmental Medicine. (In: Maxcy, K.F., ed. and others, Rosenau Preventive Medicine and Public Health, 8th ed., New York, Appleton-Century-Crofts, Inc. 1956 p.937.)

7. Guyton, A.C., Textbook of Medical Physiology, Philadelphia and New York, W.B. Saunders Co., 1961, p. 950-965.

8. Hertig, B.A., and others, Artificial Acclimatization of Women to Heat, J. of Applied Phys. 18:383-386, 1936.

9. Bass, D.E., and others, Mechanisms of Acclimatization to Heat in Man, Medicine 34:323-375, 1955.

10. Conn, J.W., Acclimatization to Humid Heat: A Function of Adrenal Cortical Activity, J. Clin. Invest. 25:912-913, 1946.

11. Knockel, J.P., and others, The Renal, Cardiovascular, Hematologic, and Serum Electrolyte Abnormalities of Heatstroke, Am.J. of Med. 30:299-309, 1961.

12. Harris, T.A.B., Hutton, A.M., Heatstroke during and after Anaesthesia, Lancet. 271:1024-1025, 1956.

13. Chapman, John, Bean, W.B., Iatrogenic Heatstroke, J.A.M.A. 161:1375-1377, 1956.

14. Bazett, H.C., The Effect of Heat on the Blood Volume and Circulation, J.A.M.A. 111:1841-1845, 1938.

15. Gold, Joseph, Development of Heat Pyrexia, J.A.M.A. 173:1175-1182, 1960.

16. Schwartz, I.L., Itoh, S., Fatigue of the Sweat Glands in Heatstroke, J.Clin. Invest. 35:733-734, 1956.

17. Burch, G.E., The Influence of Environmental Temperature and Relative Humidity on the Rate of Water Loss through the Skin in Congestive Heart Failure in a Subtropical Climate. Am. J. of Med. Sc. 211:181-188, 1946.

18. Malamud, Nathan, and others, Heat Stroke- A Clinico-Pathologic Study of 125 Fatal Cases, Mil. Surg. 99:397-449, 1946.

19. Vescia, F.G., Peck, O.G., Liver Disease from Heat Stroke, Gastroenterology 43:340-343, 1962.

20. Herman, R.H., Sullivan, B.H., Heatstroke and Jaundice, Am.J. of Med. 27:154-165, 1959.

21. Baxter, C.R., Teschan, P.E., Atypical Heat Stroke with Hypernatremia, Acute Renal Failure, and Fulminating Potassium Intoxication, AMA Arch. Int. Med. 101:1040-1050, 1958.

22. Freedman, D.A., Rourke, J.C., Selective Sensitivity of the Purkinje Cells of the Cerebellum, Am J. of Med. 27:861-865, 1954.

23. Gore, Isa, The Pathology of Hyperpyrexia, Am. J. Path. 25:1029-1042, 1949.

24. Waugh, W.H., Cortisone and the Treatment of Heat Stroke, Ann. Int. Med. 41:841-843, 1954.

25. Schillhammer, W.R., Massonneau, R.L., Heat Stroke: A Review of Three Cases, U.S. Armed Forces Medical Journal, 9:1001-1006, 1958.

26. Hoaglund, R.J., Bishop, R.H., A Physiologic Treatment of Heat Stroke, Am J. Med. Sc. 241: 415-422, 1961.

27. Laborit, H., Therapeutique Neuroplegique et Hibernation Artificielle: Essai d'eclaircissement d'une Equivoque, Presse Med. 62:359-362, 1954. (Cited by:) Jesati, R.M., Management of Severe Hyperthermia with Chlorpromazine and Refrigeration. N.E.J.M. 254:426-428, 1956.