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SUNSTROKE, OR PARALYSIS OF THE HEART BY HEAT.

BY R. CRESSON STILES, M.D., PROF. OF PHYSIOLOGY IN THE UNIVERSITY OF VERMONT.

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THE number of cases of sunstroke which occurred in the Second Corps of the Army of the Potomac on the marches of the last summer's campaign, and the diversity of opinions among our medical officers respecting its nature and treatment, decided me to seek the earliest opportunity of bringing the currently received pathology of the disease to the test of experiment. As the result of a large number of observations on animals subjected to an elevation of temperature in various modes, and which presented a series of symptoms very closely allied or identical with those of sunstroke as I had seen it, and as I found it described by careful observers, I have reached the conclusion that the disease results from the direct action of heated blood upon muscular tissue, and that its fatal termination is owing to a paralysis of the heart from the same cause.

By publishing my experiments I hope to secure greater interest in accurate observation of the disease and greater unanimity respecting its rational treatment. They may be divided into five classes:—

1st. Those in which warm-blooded animals were subjected to an artificial elevation of temperature, the symptoms they manifested observed, and *post-mortem* appearances noted.

2d. Those in which artificial respiration was practised as soon as the animal became comatose or the pupil became contracted.

3d. Those in which blood heated to 120° Fahr. was injected into the veins of the animal from which it had been drawn, or in which the blood of an animal perishing from the disease produced by heat was injected into the veins of an animal of the same species.

4th. Those in which it was proved that the muscular system of an animal could be paralyzed by heat while its nervous system was left intact.

5th. Experiments on muscular contractility as affected by heat.

Class 1st.—The following may be taken as a type of this class.

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A rabbit is placed in a glass jar, or bell glass, of sufficient capacity for free respiration during the experiment. A thermometer is suspended from the cover of the jar, and the jar immersed in hot water. The temperature of the interior of the jar is raised above 115° , but not allowed to rise above 120° . Soon after this heat has been reached, after a period varying with the size of the animal, the animal becomes restless and respire more rapidly; the bloodvessels of the ears enlarge, and their pulsations (which are almost imperceptible on a healthy rabbit) are distinctly felt. The heart beats more rapidly. At length the animal utters a few cries, and becomes comatose. The pupils are dilated, but in a few minutes become contracted. When contraction of the pupil has ensued, the animal is removed from the heated medium. He lies motionless, but the eyelids close on touching the conjunctiva. The ears continue hot. Their bloodvessels are still full, their pulsations forcible and rapid. In a variable period (from a quarter to a half hour after contraction of the pupils) the bloodvessels cease to be distended; the ears and mucous membranes, where visible, take a bluish tint; respirations diminish in frequency. Occasionally life is terminated by an attack of convulsions, but oftenest ceases gradually. When the thorax is opened, the ventricles of the heart are found rigidly contracted, or become so in five or ten minutes, both auricles distended with blood and manifesting fibrillar movements. Neither brain nor lungs are congested. The venæ cavæ are filled with dark blood, which is alkaline and coagulates rapidly. The cut surface of the ventricles is acid to litmus paper. Under the microscope the blood is normal, but the muscular fibres of the heart have lost their transverse striations. Rigor mortis comes on between one and two hours after death. The rigid heart becomes relaxed and quite flaccid in a few hours. The odor of putrefaction is very soon manifest, although the temperature of the laboratory is not above 60° .

Various animals were immersed in water at 115° , held by the back of the neck, the head above the surface. The larger animals, if allowed to respire the free air, panted violently, but resisted the fatal influence of the heated medium. Small animals, as mice and rats, soon perished; the mucous membrane of the mouth, at first redder than natural, became livid; the respirations gradually ceased, with gasping. In a rat that had struggled violently against efforts to secure him and was immersed in water at 120° , the mucous membranes became pale in five minutes, and in five minutes more all the muscles of the body became rigidly and permanently contracted. In these experiments, a thermometer introduced immediately after death into the thoracic and abdominal cavities did not rise above 115° ; it often gave one or two degrees below that temperature. Distension of the stomach by a full meal, and loss of blood, hastened the fatal termination. A litter of kittens served for these comparative experiments.

Class 2d.—In these experiments the trachea was opened, a tube inserted and artificial respiration kept up as soon as the respiratory movements began to fail. Life was not prolonged by this proceeding.

Class 3d.—In a cat that had died in heated air with all the symptoms of sunstroke, the thorax was opened and blood drawn from the vena cava ascendens with a hypodermic syringe. This was immediately injected into the jugular vein of another cat previously etherized. Not the slightest toxic symptom was manifested.

Class 4th.—The reflex actions manifested by animals apparently moribund from the effects of heat presented a decided contrast to those of narcotism or exhaustion of the nervous system. But the effects of heat on cold-blooded animals served to show more plainly its comparative integrity. On account of the sluggishness of their circulation, the heart is the last organ to feel the influence of a heated liquid. The circulation in the web of a frog's foot is temporarily arrested by a temperature of 115° . When a frog is thrown into water at that temperature all the muscles of the body become rigid in one or two minutes. If a limb does not share in the immersion it will remain unaffected, or only that portion of the limb which is immersed becomes rigid. In frogs immersed to the thighs in water at 120° even, and kept exposed to its influence some time after the anterior extremities and the muscles of the trunk have become rigid, irritation of the spinal cord in the cervical region will cause the muscles of the hind legs to be convulsed. The thigh of a frog, with the sciatic nerve projecting, immersed in water at 120° , will become rigid, but the foot will be convulsed on irritation of the projecting nerve. These experiments show plainly that the nervous system is less sensitive than the muscular to the influence of a high temperature.

Class 5th.—The more violent the activity of a muscle the lower the temperature at which it will pass into a state of rigidity. This was proved in several ways. A frog with one sciatic nerve divided was thrown into convulsions with strychnia. The hind legs, separated from the trunk, were immersed simultaneously in heated water. When the limb which had been convulsed had become rigid, the other was removed from the water quite flaccid. The hearts of reptiles, which beat long after removal from the body, were immersed in heated water at a temperature just sufficient to arrest their pulsation. If in this condition they were irritated and caused to beat, they immediately became rigidly contracted.

The cut surface of a muscle in an animal killed by section of the medulla oblongata is not sensibly acid; that of a muscle made rigid by heat is decidedly acid. The muscles of a frog tetanized by strychnia are hardly more acid than in the natural state; those made rigid by heat are decidedly acid. The bloodvessels of the ear of a rabbit rendered comatose by heat could not be made to contract by

galvanism or immersion in ice-cold water. The non-striated muscular fibre of the arterioles seemed to have lost its power of contraction, or it was at least too debilitated to resist the pressure of the blood communicated from the dilated arteries below; but the peristaltic action of the intestines is well marked in an animal dying from the influence of heat. The pupils of a frog, which contract after death, are dilated widely when the eye, removed from the orbit, is immersed in a liquid at 115°.

Effects of Elevation of Temperature on the Human System.—Under the names of sunstroke, coup de soleil, ictus solis, heat apoplexy, heat asphyxia, sun fever, erethismus tropicus, thermohamia, thermic fever, a well-marked affection has been described, not to be confounded with cerebral congestion, apoplexy, meningitis, pulmonary apoplexy, epilepsy, or simple exhaustion and prostration occurring during the prevalence of excessively hot weather and favored by a high temperature, which have often been mistaken for or have complicated the disease in question.

Causes.—The greatest number of cases are the result of muscular exercise in the sun in excessively warm weather; but the direct action of the sun is merely a favorable condition, it is by no means a necessary cause. Mr. T. E. Dempster, in giving a description of the disease as it occurred in the English Army in Bengal in 1849, says:—"During the first few marches a number of men fell victims to that fearful disease [sunstroke]; the fatal seizures usually occurred about 3 o'clock in the morning, and long before the sun was above the horizon." Mr. H. S. Swift, in an article on sunstroke published in the *New York Journal of Medicine* in 1854, describing the disease as seen in the New York Hospital, gives the following:—"Eleven cases were attacked one morning in the laundry of one of our principal hotels; several were brought to us from a sugar refinery," "and we had the opportunity of comparing their symptoms and lesions with those who became exhausted after laboring in the sun, but were unable to satisfy ourselves of any distinction." The direct heat of the sun, which made a gun-barrel unpleasantly hot to the touch, was the most frequent condition of the disease as it occurred in the Army of the Potomac last summer. I had occasion to notice its unusual prevalence during marches under a cloudless sun, before a thunder storm. Such was the morning on which the Second Corps broke camp near Morrisville and marched to the Rapahannock in the advance to Culpepper Court House and the Rapidan.

Foreigners from the north of Europe, and particularly those of recent arrival in the country, are peculiarly liable to the disease. Their skins are too thick and sluggish for our high and variable temperature. Diseases of the abdominal viscera, as diarrhœa, and a febrile state particularly, predispose to the affection.

Symptoms.—The disease, when fatal, presents, usually, two well-marked stages. It is ushered in by sudden pain in the head, weak-

ness, præcordial distress, vertigo and insensibility. This last is so sudden that the term "stroke" is properly applied to the onset. The insensibility deepens into coma, with dilatation of the pupils. The pulse, however, is both full and frequent. Respiration is hurried. The mucous membranes are red and dry, the skin dry and hot; both of a deeper color than natural. The first stage occupies the principal portion of the time. The average duration of the fatal cases is in all only four hours. This is succeeded by a stage of collapse. The pupils become contracted, or may remain dilated; respiration becomes stertorous, with moaning; the pulse feeble, remaining frequent; the mucous membranes become livid, the skin cool. Life disappears gradually, with occasional gasping respirations and tracheal râles; or death may take place suddenly, with an attack of convulsions.

The disease may present, at the onset, the symptoms of the second stage, the first having been wanting, or imperfectly marked, and the approach of death is then speedy. Contracted pupils and moaning respiration are signs of a fatal termination.

The milder cases, which recover, present the symptoms of the first stage only. When the momentary or short stupor passes by, the panting and manifestations of præcordial distress are often pitiable. Thirst is excessive, but deglutition and articulation difficult. One half of the cases, as seen in the hospitals of New York and Philadelphia, are fatal. In the army, the proportion of fatal cases among those falling out of the ranks with the symptoms of sunstroke, is certainly much less.

Pathological Anatomy.—I have not witnessed a *post-mortem* examination in a case of sunstroke. The speedy decomposition of the body renders the opportunity of making valuable *post-mortem* observations transient. Prof. Pepper, of the University of Pennsylvania, found the heart dilated and flaccid. Dr. H. S. Swift states that "the *post-mortem* appearances, though of a negative character, are precisely opposite to those found in congestion of the brain or apoplexy produced by insolation." Dr. Horatio C. Wood, in the *Am. Journal of the Med. Sciences* for October, 1863, reports a number of *post-mortem* examinations made from one to two hours after death. He notes that the heart was rigidly contracted, but as rigor mortis of that organ usually ensues within that period, there could have been nothing remarkable about the contraction, except its rigidity and completeness. He notes the absence of cerebral and pulmonary congestion, and the liquidity of the blood.

Physiology.—I can find only three modes in which endeavors have been made to explain the physiology of sunstroke:—through the nervous system, through the lungs, and through the blood. The theories of cerebral apoplexy may be neglected. Jas. Ranald Martin, F.R.S., in the London *Lancet* for 1859, thus resumes an argument respecting the nature of the disease:—"Extreme venalization

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of the blood, acute congestion at first, proceeding to passive congestion and narcotism of the lungs, heart and brain." The following is a quotation from the article referred to by Dr. Swift. His theory is in close accordance with the facts of the disease. "There are many points of resemblance in the appearance of those who have died from the effects of heat, and cases reported of death from lightning. Does the heat produce death by destroying the 'vital principle,' as Hunter supposed was the effect of lightning? Does it produce some chemical change in the blood itself, so that it can no longer subserve the purposes of innervation, or does it produce its effects primarily on the nervous system? This is the most plausible theory. The vital powers, already enfeebled by fatigue and the heat of the atmosphere, are unduly stimulated. The natural balance of the circulation is destroyed, and the heart contracts with a 'morbid activity.' The lungs are engorged with blood and the heart labors to overcome the increased obstacle, until at length it is exhausted by this 'morbid activity,' and passive congestion takes place in the capillaries throughout the body."

Dr. H. C. Wood attributes the disease to a poisonous property acquired by the blood, and suggests the name of thermic fever or thermo-hæmia. I commenced my experiments with a prejudice in favor of this theory, but was soon compelled to abandon it, or to regard the only poisonous property of the blood to be its elevated temperature. The remarkable contraction and rigidity of the heart directed my attention immediately to the muscular system. They have proved that a temperature of the blood below 115° Fahr. is sufficient to destroy life in vertebrate animals of all classes; that a temperature between 115° and 120° is sufficient to cause rigid contraction and paralysis of muscles, whether connected with or separated from the body of the animal; that the more violently a muscle is made to contract, the lower the temperature at which it will become paralyzed; and that a disease is produced by heat in the lower animals presenting symptoms almost identical with those of sunstroke, in which the heart is found rigid immediately after death.

The cases in which Prof. Pepper found the heart flaccid were those in which rigidity had disappeared from the rapidity of *post-mortem* changes; those in which Dr. Wood found the heart contracted are explicable from the time of the examination. That the blood rises in sunstroke to a temperature sufficient to produce muscular paralysis is proved by the fact that Dr. Dowler found a temperature of 112° in the axilla. The dilatation of the capillaries is explicable by the direct effect of the heated blood upon the muscular fibres of the arteries and arterioles. The cerebral symptoms, and the full and forcible pulse may also be due to this dilatation. Bernard found that section of the sympathetic nerve in the neck of a horse raised the column of mercury in a cardiometer placed on one of the small arteries of the neck sixty millimetres (about 2½ inches) above the

level indicating the pressure in the artery before the section. The dilatation of the arteries must have been the only cause of this increase of pressure.

The effect of heated blood upon the heart is termed *paralysis* rather than exhaustion, because heat has been shown to have the power of directly paralyzing its muscular tissue, and because in sunstroke the first stage, or that of frequent and forcible contraction of the heart, is not an essential part of the disease. But whatever may be the value of my theory, the facts of experiment are such as I have recorded them.

A CASE OF FRACTURE OF THE THIGH IN AN INFANT SEVEN MONTHS OLD.

BY DAVID W. CHEEVER, M.D., ONE OF THE SURGEONS AT THE CENTRAL OFFICE OF THE BOSTON DISPENSARY.

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CHAS. HENRY MYERS, between six and seven months of age, a puny child, but one neither scrofulous nor rickety, fell from his sister's arms to the floor, April 23d, 1864, and sustained a fracture of the right thigh. This was on Friday noon. On the following Monday he was brought to the Surgical Department of the Dispensary for treatment. It was now three days after the receipt of the injury, during most of which time he had cried and worried constantly. The right thigh was tilted up, forming a prominent angle at its middle, upwards and outwards. The femur was broken in the middle of its shaft. The limb was swollen to double the size of the other. The groin and perinæum were excoriated, on the injured side. Crepitation and tenderness on pressure were found over the prominent centre of the femur; and, as an additional sign, the greater trochanter did not describe its usual arc of a circle on rotating the femur from the knee. Following the position of flexion and abduction natural in the young infant, the thigh was more tilted up and outwards than is usual in the same injury in the adult; and the tendency to displacement arose mainly from the spasmodic action of the psoas and iliacus muscles. In this respect it resembled the adult fracture of the upper third of the femur, where the two intra-pelvic muscles just mentioned raise the upper fragment, and also rotate it outwards, from their insertion into the trochanter minor, which lies behind rather than inside the femur.

The indications for mechanical treatment were plain enough, but the difficulty of carrying them out was very great, on account of the infant's tender age. Treatment by a weight, by the double-inclined plane, or by counter-extension from the perinæum, were all obviously impracticable. In the applications which were made the first few days we had to pay regard to the swelling, the tenderness