H E A T S T R O K E

A THESIS FOR THE M.D. EDINBURGH

__submitted by__

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h. J. 1913.

H E A T S T R O K E.

(Heat-exhaustion, Insolation, Thermic fever, Sun stroke, Siriasis, Coup de Soleil).

Recently the subject of heat stroke has awakened considerable interest. Although one of the oldest of recognised diseases and a fairly well defined clinical entity, the causation of the condition has not been definitely settled, and recently has given rise to much speculation and several theories as to the mode of production of "Sunstroke" and heat stroke, have been suggested.

In 1898, Sambon (B.M.J. March, 19, 1898) first suggested that the condition was an infectious disease. Although this theory has received some sort of support, it cannot be said that a convincing case has been made out for its general acceptance and most writers have repudiated the theory.

Another suggestion, and one which seems to be to some extent supported by practical experience, is that the condition is due, not so much to the ordinary heat rays of the sun, as to the actinic

or chemical rays.

Colonel Maude first suggested this as far back as 1885 and he also pointed out that liability to heat stroke could be diminished if a red or yellow lining was worn under the head covering and coat, the reason being that the actenic rays are intercepted by the red and yellow lining.

The causation is one worthy of careful study and in the present thesis I have endeavoured to give an outline of the recent developments on the subject, together with some critical remarks on them, as well as my personal observations on the condition.

I desire, at the outset, to describe what is meant by the term sunstroke or Heat stroke.

Fayrer's Classification:

Fayrer in his book on Tropical Diseases divides the condition into three classes:

Syncopal type:

1. Simple Syncope from exhaustion caused by heat. Failure of heart is the essential feature of this type - Syncopal type.

Asphyxial type:

2. A condition analogous to shock, due to the action of the direct rays of a powerful sun on the brain and cord, the nerve-centres, especially the respiratory, are affected;

respiration and circulation rapidly fail and death may result, recovery infrequent though not perfect always - asphyxial type.

(N.B. in my opinion the failure of the vasomotor centre seems to be primary and more important than the failure of the respiratory centre which is usually, if not always, secondary. I have taken the help of sphygmomanonuter (Riva Rocei) in all my cases and have come to the above conclusion. Moreover we, at the present day, know, from the experimental studies of Crile, how very important vasomotor centre is in relation to the production of shock).

Hyperpyrexial type:

3. Overheating of the whole body, blood and nerve centres, either from direct exposure to the sun's rays or more frequently to a high temperature out of them, causing intense pyrexia, and then respiration and circulation fail and asphyxia follows. Recovery may occur, but is incomplete owing to structural changes in the centres giving origin to a variety of symptoms indicative of lesions of a grave character. - Hyperpyrexial type.

Roger's Views:

Rogers (Journal of the R.A.M.C., Jan. 7th. 1908), believes that under the terms Heat Exhaustion, Sunstroke, Heat stroke and Siriasis

two broadly different conditions have been included.

Heat Collapse: First: Syncopal attacks due to exposure to the direct rays of the sun or to hard labour during -great heat, e.g. in the stokeholds of Red Sea or Persian Gulf streams. In these cases there may be no marked elevation of body temperature, and if properly treated recovery is the rule with or without some permanent mental injury.

True Heat Stroke: Second: True heat stroke with hyperpyrexia and acute pulmonary congestion coming on very suddenly, with or without any actual exposure to the sun's rays. Such cases only occur under very trying atmospheric conditions, either excessive dry heat or lesser degrees of moist heat. This is true heat stroke.

Duncan's Classification:

Duncan A. (Journal of Tropical Medicine, P.101, Vol. V.) describes the varieties of Heat Stroke as follows:

A. Heat Collapse.

B. Heat Stroke.

(a) Direct Heat Stroke or Sunstroke proper.

(b) Indirect Heat Stroke.

Heat Collapse A. Heat Collapse - patient suddenly turns giddy and falls; skin moist and cool. Breathing

hurried but never stertorous, pulse small and soft, pupils dilated, temperature at or below normal, no loss of consciousness and recovery the rule.

Heat Stroke : B. Heat Stroke.

(a) Direct (a) Direct heat stroke or sunstroke proper. Onset may be sudden or gradual, fatigue may be complained of. As a rule there is violent headache and oppression, great intolerance of light ensues, there is excessive sweating, skin moist, with cyanosis, shallow breathing, injected eyes, swollen veins, and thenpartial collapse, unconsciousness or convulsions set in. There may be actual coma lasting for 24-48 hours, in which case death is almost certain. If death does not occur, the intense pain in the head may last from six to eight weeks unrelieved by any drugs, but there may be slight evening remissions. It then gradually abates. Great intolerance of light may last for a long time. Such attacks not infrequently occur in persons unaccustomed to marching and they are especially attacked when the air is moist.

(b) Indirect: (b) Indirect heat stroke. This is the syncopal form occurring not in the open but in the hot-house or bungalow.

Duncan finds it the most frequent type. At the onset the skin becomes pale, there is nausea, colic and incontinence of urine. Convulsions now follow to be succeeded by cyanosis, dyspnoea and insensibility.

The breathing is stertorous, the pupils contracted and the body temperature may reach 108°F to 110°F, remaining high post mortem.

Really two types:
(a) Heat exhaustion and (b) Heat-stroke:

From the careful study of the writing of the above authors, two essential forms can be recognised. Most books at the present day seem to recognise two types: Heat-Exhaustion and Heat Stroke, and there seems to be general agreement that the conditions are essentially different clinically (though perhaps not aetiologically).

In Heat Exhaustion the skin is moist, pale and cool, the breathing is easy though hurried, the pulse is small and soft, the senses remain

entire.

Whereas, in heat stroke there is usually hyperpyrexia and unconsciousness.

Definition: True heat stroke or siriasis may be defined as an acute disease characterised by hyperpyrexia, profound coma and intense pulmonary congestion. Heat stroke is universally considered as the effect of exposure to excessive heat, though opinion varies greatly as to the way in which elevated temperature acts in producing the peculiar symptoms of the disease.

Theories as to how heat produces these symptoms: Some authors attribute the condition to a gradual heating of the blood to a degree incompatible with the maintenance of the nervous function.

Others believe that heat paralyses the centre or centres which are supposed to regulate the disposition of bodily heat and thus causes hyperpyrexia and other symptoms of the disease. This paralysis, according to some, is supposed to cause greater heat production, according to others it causes retention of heat.

Little's Suggestion:

Little considered heat stroke to be the result of pressure exerted upon the cerebro-spinal matter by the hea expanded cerebro-spinal fluid. (This of course is untenable, since post-mortem, we do not find any suggestion of flattening of convolutions or dilatation of ventricles; during life we do not find symptoms of increasing pressure inside the skull).

Baxter and Zübz thought that the great rise in the bodily temperature, depended upon interference with the heat loss as a result of suppression of cutaneous perspiration. (Evidently it is much more than interference with the heat loss. As a matter of fact, in heat-exhaustion, the skin is moist and temperature usually normal)

Lavean's Suggestion:

Lavean suggested that the cause of the disease might be paralysis of the ganglia of the heart. (Laveán Bulletin de l'acad de Med de Paris.1894)

This, however, does not explain cerebral and other symptoms.

Vallin's Theory:

Vallin's theory was that the condition was due to a sudden rigidity of the left ventricle and diaphragm owing to the coagulation of myosin. Post mortem examination cannot support this view.

Senfleben's Suggestion:-

Senfleben (Berliner Klinischer Wochenschri June 24, 1907, page 775) ascribes the malady to disorganisation of the blood and accumulation of urea, (But it has not been proved experimentally that more than normal quantity of urea or its products are present in the blood)

Bauer's Suggestion:

Bauer thought that the main factor was an increased liberation of carbonic acid gas in the blood already saturated with it(cp Caisson's Disease). An ingenious suggestion but lacks experimental evidence.

Smart's Suggestion:

Smart attributed the symptoms to a deficiency in the serum of blood from long-continued

profuse sweating. (This is not true in all cases, as there are cases in which there is little or no sweating).

These explanations however conflicting are all based on the idea that heat is the sole cause of the disease.

Reasons for thinking that heat is the sole efficient cause.

The idea that Siriasis is due to excessive heat is of course derived from the fact that it occurs during the hottest season of the year. There is no doubt that this is one of the most important factors. But this does not seem to be the only efficient cause. In one locality the condition may be extremely prevalent, in another it is totally absent, though the region may be quite adjacent and quite as hot. Again it ravages the same locality, but in different years varies immensely and quite irrespective of temperature.

The belief that heat alone could not account for heat stroke has often been expressed by those who were able to observe the disease in its habitat.

Sambon's Microbic Theory:

Sambon, the principal advocate of the microbic theory, which is also supported by Sir

Patrick Manson and others, bases his principal arguments on the following points. -

Reasons:

1. Heat Stroke prevails only in the low-lying or Sea Coast districts and valleys of certain rivers and is never found above 600 ft. elevation.

2. Its distribution is capricious, and irrespective of the atmospherical temperature curve, and

3. Attacks occur mostly at night when the air-temperature is not at its highest point, for which reasons it is asserted that the condition cannot be caused by heat alone.

Roger's observation on the relation of meteorological condition to Heat-Stroke:

Major Leonard Rogers (Journal of the R.A.M.C, Jan. 1908) examined the records of cases of heat stroke in the British Army for three consecutive years and his results disproved the above statements. It was found that an exact relationship existed between the meteorological conditions and incidence from stroke.

An important point brought out in these ob-

servations is the relationship of atmospheric humidity to sunstroke.

It was found that heat stroke occurred at a much lower mean temperature when a high degree of moisture was present.

The alleged capriciousness in the occurrence of heat stroke cases entirely disappears if the degree of moisture in the air as well as the actual temperature is taken into consideration.

If a high degree of moisture is present interfering with free evaporation of perspiration then the hyperpyrexia of heat stroke may ensue with a maximum air temperature of just about the blood-heat.

With a drier atmosphere the body cooling mechanism may fail when the maximum air-temperature exceeds that of the body.

A knowledge of these facts, according to Rogers, will allow of the occurrence of this dangerous affection being anticipated and measures taken for the early detection and prompt treatment which alone will save the lives of the vast majority of sufferers.

Jardin's observation points to the same:

Another interesting report on the relationship of humidity of the atmosphere to sun-stroke is that by Jardin. (Clin. Modern.N.22-24 An X11) He discusses his experience during 1905 in regard to cases of sunstroke at Florence.

During that year which was marked by access of great heat with high aqueous vapour tension and marked electric state thereof of the atmosphere the number of cases of sunstroke was considerably above the average.

The greatest mortality occurred at the beginning of the heat wave before people had had time to get acclimatised or to take proper precautions.

The above observation does not seem to be borne out in every case.

This relationship of the atmospheric moisture to sunstroke is, however, not borne out by my own experience in every case as will be seen from the instance appended.

Example:

The rainfall in Dera Ismail Khan averages only six inches per annum and the climate is intensely dry. Yet it was found that out of a regiment of Moplah Sepoys, who came to Dera

Ismail Khan from Malabar (which was a humid climate), twenty four were attacked with heat stroke on their first day's march. The curious feature was these Moplahs coming from hot humid climate suffered so severely in an extremely dry one. The station they had come from had at this time of the year (September) a higher temperature than Dera Ismail Khan. In these cases it may also be that other factors come into play and acclimatization seems to be an important one of them.

Acclimatization is an important factor.

Fresh arrivals in the tropics are more prone to suffer than those who have become accustomed to the climate. It is well known that a native can bear an amount of sun on his bare head and naked body with indifference, that would rapidly prostrate a European.

Colin and De Sauti's view:

Colin and de Sauti have considered heat stroke as a form of malaria. Long before them Dr. Cheves and other Anglo-Indian surgeons who have given us the best descriptions of the condition had remarked on the probability of sudden attacks of the disease being raised

by "malaria in a concentrated form".

The idea that malaria was a cause of sunstroke probably sprang from the fact that those suffering from malaria are most liable to the disease, or again from the fact that pernicious malaria may closely simulate it, but it is intolerable because in many regions where malaria is most prevalent, heat-stroke is unknown, and on the other hand it is greatly prevalent in places which have no malaria. Moreover in heat stroke, spleen is not enlarged and the blood does not show the typical parasite in any stage.

Simpson's criticsm of actenic theory:

With reference to the actenic theory of sunstroke first suggested by Colonel Maude, it cannot be said to have been definitely established and has been subjected to severe criticism in a paper by Lieutenant Col.Simpson in the Journal of the Royal Army Medical Corps, 1908, page 441.

Duncan's support of the Actenic theory:

A. Duncan, however, in the Journal of Tropical Medicine 1907, page 83, says:- "There can be no doubt that this actenic theory of sunstroke

is the correct one. All soldiers' helmets should be given out to them lined with orange-red covers." Those who have adopted this head-gear have certainly found it effective in warding off heat-stroke.

Author's experiment on two young cats

Two young cats about four months old formed the subjects of the experiment. Two glass boxes of convenient size and of same capacity (not at all unlike the ordinary incubator) were taken. They were provided with two ventilating holes each; but one box had the covering with red glass and the other with the white. Cats were shaven very closely and were kept in those boxes, one occupying each box. Both were fed on milk and fish. During the month of June and the earlier part of July they were daily and regularly exposed to the sun on the roof of my bungalow for five hours from 11.30 a.m. till 4.30 p.m., and their temperatures were daily taken at least thrice.

Before experiment both showed the temperature of 101°F. Nothing special happened for the first few days, except slight rise of tem-

perature during the time of exposure, temperature rising up to 103°F or 102.4°F. Then I gradually began to dininish the amount of milk. Both the cats began to go off their food. One that was kept inside the white glass began to show more weakness and get more and more wasted. Elevated temperature seemed to tell on it more powerfully. Its breathing began to get hurried and shallow. Day by day the disparity of temperature began to be more and more marked. On the 27th day, the cat that was kept inside the white box showed a temperature of 112°F, its respiration became shallow and jerky, particularly during inspiration. The period of pause also lengthened; and in a few hours I lost the cat. The one that was kept inside the red box also began to lose weight. It also got more and more exhausted, but never showed a temperature higher than 103°F. I took it out of the glass box, after the death of the other one. The one kept inside the red box lived a long time.

From the above experiment, the following conclusion may be drawn. Heat rays may do harm to the living protoplasm. But actenic rays of the sun seem to exert some deleterious effect on the

system, far more potent than the heat rays. Excessive pigmentation of the skin in the coloured races, may be assumed to be a natural protection against this powerful, penetrating and perhaps chemically active actenic rays of the sun. My view is that these rays first of all exert their deleterious effects on the cutaneous structures and the sudoriferous glands, fix them, as it were, by means of fixing reagents and render them inert and incapable of carrying on the function of the skin. Heat is thus retained. These cells, may, in addition, produce some poisonous materials which are carried to the general circulation, giving rise to the symptoms of heat-stroke. The above experiment may also throw light into the different actinological natures of heat-stroke and heat-collapse, the former being the result of the actenic rays of the sun alone, the latter the result of heat mainly (other factors playing a minor part).

Cause of hyperpyrexia is the retention of heat, in the first place, and then auto-intoxication, disturbing the heat-regulating mechanism.

Other secondary conclusions may also be

drawn from the above experiment.

Body or tissue fluid is important in preventing or delaying the precipitation of heat stroke or heat-apploxy.

Starvation and previous exhaustion predispose to heat-stroke.

For in the above experiment, reduction of the amount of milk told quickly on the cat in the white box, nothing special happening ere this.

Pathology and morbid Histology

The pathology of the condition has been made the subject of a special study by Lambert and Van Giesson (Medical Record, July 4, 1897). After a study of over eight hundred cases of insolation, these observers found that heat alone is not sufficient to explain all the clinical and pathological observations.

The ~~predominal~~ prodromal symptoms of sun-stroke are those of acute functional disturbance while the later symptoms much more serious point to grave changes in the blood and all the nerve-centres, especially those of the latter which control the thermic mechanism of the body.

Van Giesson examined the brain and cord of several of Lambert's fatal cases and found universal presence of acute degeneration of the neurous of the whole neural axis.

In the cerebral cortex and cerebellum, the cells showed the same degenerative changes; the cells of the spinal cord were not so extensively involved. A toxic agency of the symptoms of insolation seemed to be shown by changes found in the ganglion cells. The experiments by Vallin would tend to show that coagulation of albuminoid bodies occurs. The toxemia would thus occur as a result of arrested matabolism. The blood is dark though fluid, and the corpuscles are cremated. In the hyperpyrexial form leucocytosis and degeneration of red corpuscles may also be noted. Extravasations in the peripheral tissues are often found and the body undergoes rapid putrefaction. Acute pulmonary congestion is also a notable feature.

Cagicol & Lapierre's observation.

Cagicol and Lapierre (Montreal Clinique 1898) found a micro-organism in the blood of patients suffering from heat apoplexy and regarded it as the specific cause of the disease. It

is linear incurved and slightly constricted in the middle. Viewed in the blood it is from 2 to 2.5 lb. long and 0.5 lb. thick, in cultures it is somewhat larger. It presents filaments, is slightly mobile but possesses no cilia, stains easily with aniline colours, but not by Gram's method. There are free spores in the cultures as well as in the rods. It is aerobic, does not cause fermentation in sugars and does not give rise to indol.

These findings have not, however, been confirmed by later workers and so cannot be accepted yet.

The following cases are of interest as they present some features outside the usual experiences of heat stroke. They also illustrate the course and lines of treatment,

Case 1: - sent for me on June 3rd, complaining of persistent dull headache, sleeplessness and depression. T. 101°, Pulse 90. He was a thick set, short necked man of 45, a life-long teetotaler and of regular habits. He has been more or less

ill for a week, his chief trouble being sleeplessness. He said he has slept only at intervals of an hour or two in a sitting posture, as any attempt to lie down caused his head to throb violently and unendurably. He complained very much of heat. He never had any similar trouble.

Treatment: Saline purgative, milk diet, quinine sulphate gr. X daily. He was also given chloral to induce sleep and he was ordered to be removed to cooler climate; but could not arrange to start at once. The next day the morning temperature was 100.6°F. and evening temperature 100°F. General condition as before. Next day I saw him. He had spent a very bad night, walking up and down on the flat roof of his house. He was cyanosed and very restless. T. 102.8. Pulse 100. He was very nervous and had a feeling of impending death - so much so that he made a hasty will. Icebag was applied to his head and he was given a dose of calomel gr.5 and had abundance of cold water poured over him from the "Chisti's Masak" or leather water bag and gave him a hypodermic injection of strychnine. He was perspiring profusely. By midday his condition was much improved. He slept four hours and woke

up much refreshed. T. 100. Pulse 92, full and regular. But towards the evening he had great difficulty in breathing. Icebag was applied on his forehead and ice water enema was given. He felt a little better. Next day he was again seized with similar symptoms and died quite suddenly before any assistance could be given. The body temperature four hours after death was 106° and the face was congested.

Peculiarities:

1. Long premonitory symptoms,
2. Teetotaler always,
3. Never had any similar or serious illness before.

Case 11

July 5. - aged 26 has arrived here after a long journey. He has been feeling unwell for three days past, running a temperature of 100° to 101°F. He had no appetite and perspired freely, his bowels were not constipated. He consulted me at 2 p.m. I noticed his speech was rather incoherent and he was much flushed. He complained of occipital headache, great depression, dizziness and flashes of light before his

eyes. Temperature at the time was 104°, pulse 110, full and bounding. I put him on a grass bed-stead in my house and had a large quantity of cold water poured on him at once. Icebag was applied on to his head, he became semiconscious almost immediately and his temperature rose to 106°F. He was in a critical condition. Water was constantly poured over him and a hypodermic strychnine was injected. In about 15 minutes his temperature dropped to 103°F; he gradually recovered consciousness, but remained in a dazed condition. That evening his temperature was 101°F. He recovered very slowly from the cerebral condition. In about a week he was physically right. He was advised to go to a cooler climate. However, he suffered from weakness of memory, loss of power of concentration and from intense emotional excitement from trivial cause.

Peculiarities:

1. Comparatively young man.
2. Incoherent speech at the start.
3. Mental enfeeblement afterwards.

Case 111. June 29th.

Twenty sepoys of various casts started to

relieve an outpost at 9 p.m. They formed a guard of an ammunition-waggon which broke down at midnight. This necessitated off-loading involving tremendous exertions, the night temperature being 99°F. Nine of these men (4 Sikhs, 3 Pathans and 2 Doegras) collapsed during this process and became unconscious. Medical aid was not at hand. But their companions carried them to an adjacent house and laid them round the well. Cold water was poured in large quantities over them. In the early morning, four of them were so far recovered as to march back seven miles to the station. The others were all well in a day or two.

These are really cases of heat exhaustion and not heat stroke proper. Natives seem to stand heat well.

Case IV. -

A Singh-Sepoy, aged 22, collapsed on parade at 7 a.m. There had been no previous symptoms. He was carried to a Hospital in a perfectly conscious condition. Temperature 107 and a running pulse of 120°. Respiration 40 per minute, quick

and shallow. He was placed on an ice-pack, but the temperature was only lowered to 104° and quickly rose again in spite of cold water enemata. Every effort was made to induce perspiration as his skin was very dry. He suffered much from intense headache and had violent cramps in both legs. Hyperpyrexia (T. 107°F) continued even when he was placed in a bath, and the temperature lowered by ice. His temperature rose to 110°F. and he died an hour after.

Interesting points:

1. Persistent dryness of the skin.
2. Patient had never touched alcohol.
3. The patient never completely lost consciousness, in spite of pyrexia.

No autopsy was possible owing to caste prejudice.

Case 5:

October - - a thin anaemic subject, had suffered one year previously from a touch of the sun in Madras. He had three months of intense dry heat in Dera Ismail Khan and stood it well. He remained well until October when he suddenly collapsed from the heat during a mid-day march

at the Pindi-manoeuvres. His skin was pale and cold, pulse feeble and rapid, temperature 101°F, respiration quick and shallow.

Strychnine, brandy, saline purge were given, and his consciousness returned. He gradually got better, but he stood heat badly later on.

1. One attack predisposes to another.
2. Heat exhaustion or heat stroke not common in October.

Should the Roza (the Mahomedan Fast) occur during the hot weather there is always a large increase of cases of heat collapse. This tends to show that under-feeding and lowered vitality tend to share the chief predisposing causes. Alcoholism is a pre-disposing cause only in so far as it lowers the vitality.

It is generally recognised that over-exertion tends towards heat apoplexy and troops are moved as little as possible during the hot weather, marches are usually accomplished at night.

Silebrar, Assam has an average rainfall of 160 inches per annum, and is adjacent to Cherapon-jee with a rainfall of 424 inches, by far the wettest district in the world. The temperature

in Silchar averages 94°F at the hottest time of the year. Here natives do not wear pagris and many coolies and Europeans work all day in the tea gardens. Heat stroke, however, is not uncommon, but cases are absolutely different from those met with in Upper India. Definite symptoms characterised by sudden onset and initial high temperature are always present and the malady runs a course much less severe, but the after-effects are often long continued and serious and permanent mental derangement often may follow the mildest cases of heat stroke in this part of India.

Case 6: -

A Deputy Inspector of School, aged 55. He had suffered from sciatica for about three months before this attack and was rather weak. He derived much pleasure from all kinds of intellectual pursuit. One evening at a meeting of a school committee he gradually began to slide down from the chair he occupied, with his eyes half-closed. The members of the Committee at once helped him to a couch and I was sent for. On arriving there, I found him almost unconscious

he was breathing feebly; his pulse was very fast and hardly perceptible, and of low tension. There was no temperature. On the other hand I found his skin moist. I at once poured some cold water on his body & ordered an ice bag and gave a hypodermic injection of strychnine. His pulse and breathing improved a little, but he lay still unconscious. Calomel, gr. 5 and an ice-water enema was given, and the injection of strychnine with m 5 of adrenalin chloride was ordered. He lay unconscious for nearly 12 hours. Next day when I saw him he was found to be much better. His pulse was full and bounding, his consciousness returned and his breathing, though slightly hurried, was almost normal. He, however, could not account for why he was there where he was. He was almost all right in three days and was advised to go to a cooler climate and he followed my instruction. But he wrote to me later saying that he suffered from the want of power of concentration and from weak memory.

Interesting feature of the case was the inability to fix the time and occurrence of the heat apoplexy.

Case 7. July 1910.

- - Stoker in one of the Austrian-Lloyd steamers, in the Persian Gulf. A man of about 30, well built and strong, suddenly collapsed. His skin was moist and pale. His pulse was quick and feeble. Temperature low. Breathing shallow and weak. The ship-surgeon treated him as follows. The doctor made an emulsion of egg and milk and put in plenty of mustard . And the patient was helped to drink it. The back of his throat was tickled by means of a feather. That made him vomit. This procedure was repeated twice. Then he was given a hypodermic injection of strychnine and digitalis. The patient's pulse improved. He, however, lay semi-conscious and prostrate. He was given some brandy repeatedly. Next day his temperature rose up to 103°F and he was given a cold water enema. In three or four days he became almost all right except for a little weakness.

The ship-surgeon told me that the rationale of his treatment by inducing vomitting was to eliminate the autogenous poison from the stomach and also to stimulate respiration.

S U M M A R Y.

Two types of Heat Stroke or Sun-stroke are observed clinically. One is Heat-exhaustion or heat-collapse, and the other is true Heat-stroke or Heat apoplexy.

From my experiments on two young kittens, I am led to the conclusions:-

That true Sun-stroke is the result of actenic or chemical rays of the sun, as distinct from the heat rays of the sun.

Heat collapse or Heat-exhaustion is the result of heat rays on the body. Long march, fatigue, excessive humidity of the atmosphere, existence of debilitating diseases and want of acclimatization markedly predispose to heat-exhaustion or heat-stroke.

Actemic rays act as a kind of fixative to the cells of the skin and sudoriferous glands. This prevents evaporation and causes retention of heat - then the retained heat may cause coagulation of albuminoids of the tissues and give rise to auto-intoxication. This throws the heat-

regulating mechanism out of gear, giving rise to hyperpyrexia.

In heat, exhaustion and also auto-intoxication seem to play a prominent part. Fatigue we know is due to accumulation of poisonous material (sarcolactic acid among other things) in the muscle substance. Here exhaustion or fatigue seem to be primarily important. The toxic substance, the result of fatigue, is carried to the general circulation. Hence the untoward symptoms. But here, cutaneous evaporation, as a rule, can go on. The skin is not affected at the very start Hence the body temperature is not particularly high.

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