If the morbid state is accompanied by physical symptoms, such as loss of speech, deafness, or paralysis, these must be removed. The patient is first carefully examined so as to be certain that there is no structural cause for his loss of power. Then he is told that his disability is not based on any serious bodily condition, and that he can be cured without difficulty.

The majority of hysterical patients, like children, are unduly suggestible. But, in most instances, it is unnecessary to employ hypnotic suggestion. Provided the examination has been carried out carefully and sympathetically and nothing has been said or done to confirm the patient's belief in the severity of his disease, the physician will have acquired sufficient suggestive power to remove such physical disabilities as paralysis or loss of speech. Sometimes this suffices to produce a permanent cure; but it must not be forgotten that behind these obvious manifestations may lie a state of anxiety. This must be dealt with seriously and systematically, or the patient will relapse on the first occasion that his conflict is reawakened.

The number of lightning cures that were possible during the war was a measure of the ignorance of the bulk of the medical profession as to the nature of these maladies. Paralyses of mental origin were mistaken or neglected, and the patient was not subjected to systematic psychical treatment.

All such loss of function can be easily recognized by its character. It follows a conceptual and not a physiological or anatomical distribution. A patient with hysterical loss of speech can write and read fluently, and one with complete aphonia can cough loudly. When all power of recognizing the position of one upper extremity appears to be lost, the patient has no difficulty in finding the tip of his affected forefinger with that of the normal hand; but he carries out the reverse operation with difficulty, because it seems natural to him to do badly with the "bad" hand and well with the "good" one. But, when the sense of position is disturbed from an organic lesion of the cortex, the condition is usually the exact opposite. The normal forefinger cannot be brought into contact with that of the affected hand because its position is not known, whereas the reverse movement can be carried out without difficulty, because the situation of the normal hand is accurately recognized. It is easy to make fair shooting with a bad rifle if we know the position of the target; but the best rifle in the world is useless if we are ignorant of the direction of our aim.

The following difference between two apparently allied signs is equally significant. When a patient with tabes dorsalis or any disturbance of the functions of the posterior columns is made to stand with his eyes closed, he tends to sway or fall. In many psychopathic states he also becomes unsteady under similar conditions; this has been called by the outrageous name of "pseudo-Romberg." But the true nature of this sign is evident from the following procedure: Let the patient stand on both feet and tell him to close his eyes; he tends to fall. Then tell him you are going to examine his eyes; stand him facing the light, and close first one eye with your hand and then the other; he will remain steady on his feet. By the first method of examination, attention was attracted to his power of standing with his eyes closed, but, on the second occasion, he was told that his eyes were under examination and no question of equilibrium arose in his mind. All these conditions may be classed together as "paralysis by idea."

Most of these defects of function arising during the war have long ago been removed by treatment, and the few patients of this class who remain are of a poor mental type. The large bulk of the functional neuroses which now demand attention are those consisting of states of anxiety and obsessions. This corresponds to the normal experience of civilian life. An ex-soldier is troubled about his work or is anxious about the illness of his wife and the difficulty of obtaining a lodging. Officers returning to their pre-war employment find it uninteresting and badly paid. Moreover, they have not the mental stamina to work long hours, and then return home in an intolerably overfilled train. Old nightmares recur and sleep is troubled; their general efficiency goes down, and they are in real danger of finding themselves without employment.

It is the business of the physician to investigate these conditions with the greatest care. An unfavourable environment must, if possible, be changed. The sleep of one of my patients improved enormously as soon as quarters were found for him in London, so that the railway journey at the beginning and end of the day's work became unnecessary. Abnormal mental experiences must be brought into the main stream of the individual personality, and, if possible, the patient must be induced to regard them from a more favourable point of view. A terrifying object, that can be logically examined, tends to lose its fearful aspect. We dread the unknown; and to drag these half-appreciated horrors into the light may discharge the greater part of their emotional energy. If possible, a sorrow must be sublimed; the loss of some dearly loved person should not be repressed, but be brought up to form an integral part of the sacrifice at the altar.

Obsessional states are the hardest to remove permanently; for any want of physiological or mental fitness is liable to lead to regression. If, however, the patient can be taught to recognize the significance of this reappearance, its explosive force can be greatly lessened.

I have entered a plea for regarding the psycho-neuroses as a disturbance of functions, common both to the nervous system and to the mind. The form they assume depends on the personality of the patient, and the nature of the emotions and ideas with which he has had to deal; it has nothing to do directly with the effect of external physical forces. Such expressions as "shell shock" and "neurasthenia" do not correspond categorically to the manifestations of the functional neuroses, which are in reality the forms assumed by the reaction of the patient to his individual mental experiences.

THE NATURE, PREVENTION, AND TREATMENT OF HEAT HYPERPYREXIA.*

THE CLINICAL ASPECT.

BY

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THE three summers of 1916, 1917, and 1918 spent on active service in Mesopotamia as Consulting Physician to the Mesopotamian Expeditionary Force gave me the opportunity of seeing a very large number of cases of illness due to exposure to high atmospheric temperature, and I made a careful study of the clinical manifestations of the different types of disease due to this cause.

Heat hyperpyrexia was the most striking and most dangerous form of illness met with, and as this was likely at any period to occur as a complication in the milder types of these effects of heat, it was chosen as the title of the paper.

ETIOLOGICAL FACTORS.

Climate.

Mesopotamia and the country round the Persian Gulf have been well known for many years as having an unenviable reputation for danger from "heat-stroke." The sun's rays seem to have a peculiar deadly power in this part of the world, and the risk of heat-stroke appears to be much greater than in countries like India, where the temperature is equally high.

Probably the explanation is to be found in the extreme flatness of the country, and the absence of trees and vegetation except in the small cultivated areas. The clearness of the atmosphere also and the absence of dust and moisture in the upper strata undoubtedly contribute, so that the full force of the sun's rays was encountered, with scarcely any absorption from dust or moisture in the air and unrelieved by shade.

The soil of Mesopotamia is really the dried alluvial mud deposited by the river floods, and this forms a light brown dry barren deposit, which radiates and reflects to a great extent the sun's rays, so that one is exposed not only to the direct rays of the sun but to those reflected and radiated from the surrounding soil.

* Papers read in introducing a discussion on heat hyperpyrexia at a meeting of the Medical Society of London on March 8th.

Temperature.

A maximum shade temperature of 110° F. appeared to be the dangerous limit. When this was reached some cases of "effects of heat" were sure to occur, and each degree rise above this limit was attended by an increasingly larger number of cases. Temperatures of 120° or over were exceedingly dangerous, and on these days, in spite of all precautions, large numbers of heat-stroke cases occurred.

A comparison of the temperature records of 1917 and 1918 for Baghdad (see atmospheric temperature curves) shows the reason of the far greater number of cases of heat-stroke in the former year. In 1917, on six days in July the shade temperature was 120° and upwards, and on fifteen days it was 115° and over. In 1918 the temperature did not reach 118°, and on only two occasions was it 115° or over.

The curves showing the temperature and cases due to effects of heat illustrate the influence of temperature.

Cumulative Effect of Heat.—The effect of heat was undoubtedly cumulative in action; thus one or two very hot days were not necessarily followed by a large number of cases of heat-stroke; it was the succession of several hot days which was dangerous. The case incidence curve followed the temperature curve, with a delay of a few days in the rise of the former. The cumulative effect of heat was shown on the individual; a man might be exposed to heat for one or more days and then develop an attack of heat hyperpyrexia in the night or early morning after the atmospheric temperature had fallen considerably.

Humidity of atmosphere undoubtedly predisposed to heat hyperpyrexia, owing to the diminished heat loss from a lower rate of evaporation from the skin, and also from the greater heat conductivity of a hot, damp atmosphere. The effect of a high relative humidity was shown by the greater case incidence of effects of heat at Basrah than at Baghdad (see tables), the relative humidity being considerably higher at Basrah, as shown by the wet and dry bulb records.

Special Rays as Causes of Heat Hyperpyrexia.—There is no evidence that other rays than the heat rays from the sun cause heat hyperpyrexia. The influence of actinic or ultra-violet rays as possible causes was investigated, with negative result, by Mackenzie and Le Count in America.[1]

Effects of Heat in Mesopotamia (British).

	1917.		1918.	
	Cases.	Deaths.	Cases.	Deaths.
January and February ...	—	—	—	—
March	61	—	2	—
April	612	1	10	—
May	390	—	68	1=1.4%
June	307	7= 2.2%	121	—
July	2,949	425=14.4%	189	10= 5.2%
August	1,086	63= 5.8%	147	17=11.5%
September	810	27= 3.3%	24	3=12.5%
October	18	1= 5.5%	12	—
November	9	—	1	—
December	—	—	—	—
Total	6,242	524=8.4%	574	31=5.4%

Effects of Heat in Mesopotamia (Indian).

	1917.		1918.	
	Cases.	Deaths.	Cases.	Deaths.
January, February, and March	—	—	—	—
April	15	—	5	—
May	6	1=23.3%	11	4=36.3%
June	22	2= 9.1%	38	1= 2.6%
July	565	59= 1.4%	64	4= 6.2%
August	179	12= 6.7%	49	4= 8.1%
September	109	15=13.7%	4	1=25.0%
October	—	—	1	—
November and December	—	—	—	—
Total	896	89=10%	172	14= 8.1%

Effects of Heat (Mesopotamia).

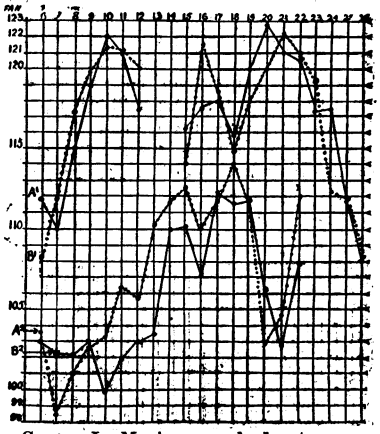
Week ending:	British.		Indian.	
	Cases.	Deaths.	Cases.	Deaths.
July 14th, 1917	770	104=13.5%	181	7= 3.9%
July 21st ,, ...	1,272	197=15.5%	216	38=17.5%
July 28th ,, ...	815	122=14.4%	163	12= 7.4%
August 4th, 1917	239	27=11.6%	34	3= 9.0%
August 11th ,, ...	60	8=13.3%	7	2=28.5%

Mesopotamia: Heat-stroke Figures for Different Areas (1917).

Week ending:	Baghdad.		Basrah.		Amara.	
	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
July 14th ...	94	24=25.5%	249	40=16.5%	60	8=13.3%
July 21st ...	186	68=36.6%	403	69=17.0%	108	11=10.2%
July 28th ...	194	53=27.3%	183	22=12.0%	47	8=17.0%
August 4th ...	57	17=29.8%	69	3= 4.4%	18	6=33.3%
August 11th ...	36	7=19.4%	15	—	9	1=11.1%

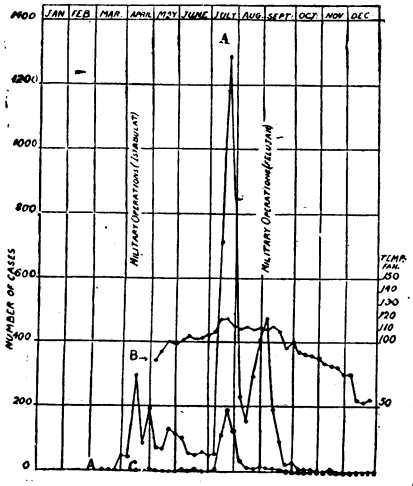
Night Temperature.—In Mesopotamia the month most dangerous as regards heat-stroke was July, since the maximum shade temperature was for a great part of the time over 110°. August was also a dangerous month. Even in these months, however, the night temperature fell considerably; thus, on the hottest day in my experience (July 20th, 1917) the maximum shade temperature was 122.8°, but at night the temperature fell to 81.6°, a drop of 41.2°. The relatively cool nights even in the hottest months render Mesopotamia a possible country for the white man; were it not for this, the incidence of heat hyperpyrexia would undoubtedly be much greater.

Stagnation of Air.—Free currents of air have a great value in protection by promoting evaporation from the skin and loss of heat. Electric fans and punkahs were largely used in Mesopotamia in hospitals and dwellings occupied by troops from 1916 onwards. They were a vital necessity to the British troops during the hot months. In this connexion it is interesting to note that when the temperature was very high the air from a fan would be like a hot blast, and unless the body was covered with a moist sheet, or a moist screen intervened, the fan would be of little value as a cooling agency and indeed might do more harm than good. The Arabs do not appreciate the value of air currents; in the native quarters of the towns the narrow streets and houses with little window space, show that they are designed with the object of shutting out the hot air rather than promoting free ventilation. In July, 1917, on some days the heat was so intense that when a slight breeze arose it was necessary to take shelter



CURVE I.—Maximum shade temperatures for July, 1917 and 1918. A^1=Baghdad. 1917; B^1=Basrah, 1917. A^2=Baghdad, 1918; B^2=Basrah, 1918.

in a covered dwelling to escape the hot wind. On July 20th, 1917, at Kut such an instance occurred when people ran for shelter to escape the scorching blast, and several fatal cases occurred.



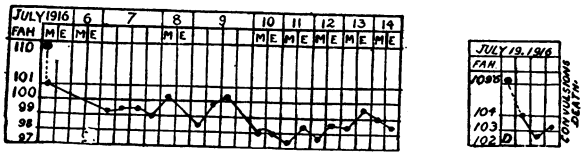
CURVE II.—Effects of heat, 1917 (Mesopotamia—British). A=Cases per week; B=Temperature; C=Deaths.

Fans.—The best use of fans in very hot weather was obtained by keeping the rooms closed as far as possible so as to keep out the hot air, the fans providing the necessary air currents in the cooler air within. Aron has shown by experiments on monkeys that still air is a potent cause of heat-stroke.

The "kata" thermometer, introduced by Dr. Leonard Hill, F.R.S., was used in Mesopotamia in 1918, and was very valuable in estimating the degrees of stagnation of air, and thus the risk from heat-stroke. For example, in a hospital ward by its means the suitability of the beds for cases liable to heat-stroke could be tested.

Dwellings for Protection against Heat.

Dwellings must be constructed of very thick walls of non-conducting material—for example, of stone, bricks, or dried mud—and it is of special importance that the roofs of huts should be thick. A coating of at least six inches of



CHARTS 1 AND 2.—Heat hyperpyrexia.

dried mud on a roof is the minimum protection against the sun's rays. Tents afforded poor protection, and in the double fly E.P. tents the temperature would often reach 135° or 140°. During the hot days it was necessary to wear in them a head protection—for example, a topee.

A further protection to a tent was an additional roof of rush matting, but the difficulty of fixing this rendered it impracticable for general use.

Dug-outs with mud walls around and a tent roof, which were used in some cases for troops, were unsatisfactory, since, though cooler in the daytime they were too hot at night; further, the dug-out earth was a favourable breeding ground for sand-flies, and sand-fly fever was always prevalent under such conditions.

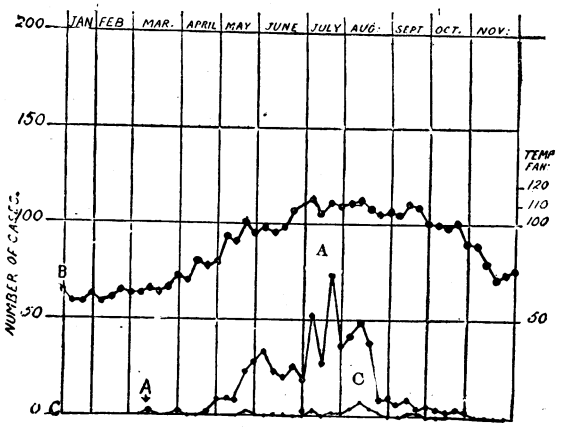
Camps under palm trees were tried, but the stagnation of air caused thereby, and the greater prevalence of mosquitos and other insects, outweighed any advantages derived from the shade of the trees.

It appeared that on the open desert double fly tents, due precautions being taken as regards personal protection during the day, were on the whole best suited for troops if huts or buildings were not available.

Age and Race.

The risk of heat hyperpyrexia depends largely on personal protection, and if care be not taken any age is subject to it. The case mortality was undoubtedly greater in men over 40.

Race is very important. Heat hyperpyrexia due to heat



CURVE III.—Effects of heat, 1918 (Mesopotamia—British). A=Cases per week; B=Temperature; C=Deaths.

alone, and not to the complication of other diseases, was very uncommon amongst Arabs and Indians; indeed, when it occurred in them almost always some complicating disease such as malaria was found as the cause. White races are much more susceptible to the effects of heat, and in British troops a large percentage of the cases were due to heat *per se*.

Alcohol.—If alcohol is taken during the heat of the day it undoubtedly predisposes to heat-stroke. Numerous individual examples of this came to my notice.

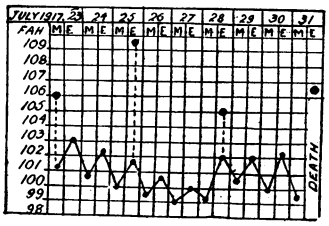


CHART 5.—Recurrent hyperpyrexia.

Exertion.—Exertion during the heat of the day is a great predisposing cause, especially if heavy kit is carried. Numerous examples of this occurred; perhaps the best instance is that shown in the 1917 curve of heat effects, where the two accessory rises in April and September were due to military operations at Istabulat and Felujah respectively. (See Curve II.) A tragic example was the sad death of Sir Victor Horsley, which was undoubtedly due to his having to walk long distances during the heat of the day in the performance of his duties as consulting surgeon.

Water.—Absence of a large supply of drinking water is a cause of heat hyperpyrexia. During the hot weather from two to three gallons a day are necessary for a man exposed to heat. In military operations this was in some cases impossible, and cases of "effects of heat" occurred in consequence.

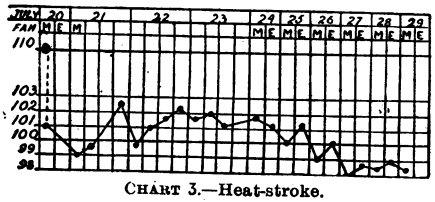


CHART 3.—Heat-stroke.

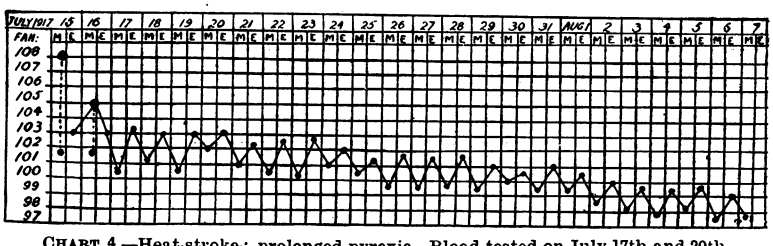


CHART 4.—Heat-stroke; prolonged pyrexia. Blood tested on July 17th and 20th, was negative on each occasion.

Personal Protection.

Clothing.—The use of efficient heat protection from the sun's rays, as by thick pith topees or an efficient service helmet, was very important. The service helmet appeared to give adequate protection, and, as it was capable of withstanding rough usage, it was perhaps better adapted for troops than the pith topee. Spinal pads 9 in. wide were necessary for the protection of the spinal cord from the

sun's rays. Light loose clothing, not too thin, was desirable.

Constipation was undoubtedly a predisposing cause, and Army Orders were issued warning against this. The high percentage of indicanuria in cases of heat-stroke is confirmation of the importance of promoting free elimination from the bowel.

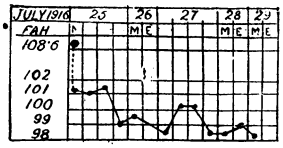


CHART 6.—Malaria and heat-stroke. July 26th, quinine gr. vi intramuscularly.

Dark Glasses.—It has been thought that the action of the reflected glare of the sun's rays on the retina was a predisposing cause of heat-stroke, and the dark green glasses issued to troops undoubtedly afforded comfort and probably protection.

Umbrellas afforded protection to those compelled to walk in the heat of the day, and were used with advantage.

Hand fans were of value in promoting loss of heat and in keeping off flies.

Predisposing Diseases.

Any disease causing pyrexia predisposed to heat-stroke in the hot weather.

Malaria was one of the commonest of these, and in Indians was almost always the cause of heat hyperpyrexia. (See Charts 6 and 7.)

Sand-fly Fever.—During an attack of this fever in the hot months hyperpyrexia sometimes occurred, and I have notes of cases of heat hyperpyrexia occurring in the apyrexial period following sand-fly fever. Captain H. C. Sinderson has suggested[2] that sand-fly fever was a common cause of heat hyperpyrexia. I cannot agree with this, because in the great majority of cases there was no evidence of sand-fly fever, and in the great incidence of heat-stroke in July and August, 1917, sand-fly fever was entirely absent.

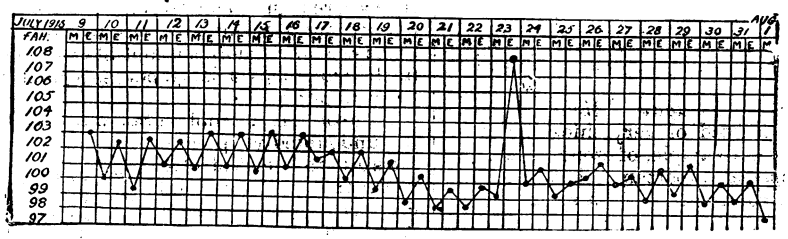


CHART 8.—Paratyphoid A, malaria, and heatstroke. July 24th, quinine gr. x intramuscularly.

The enteric group diseases, if occurring in the hot weather, were not uncommonly complicated by heat hyperpyrexia (see Chart 8).

Typhus fever, both in the acute stage and apyrexial period, might be complicated by heat hyperpyrexia (see Charts 9 and 10).

Small-pox.—I have seen heat-stroke occur during the early stages of small-pox before the appearance of the rash.

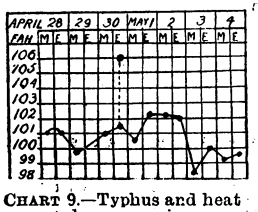


CHART 9.—Typhus and heat hyperpyrexia.

PATHOLOGY AND MORBID ANATOMY.

I have no doubt whatever from my experience that heat hyperpyrexia is a clinical entity and occurs apart from intercurrent diseases. The influence of these has already been considered. The clinical history of cases of pure heat hyperpyrexia, and the course of the symptoms after the acute stage, which often exhibit prolonged pyrexia and profound toxaemia in the absence of any bacteriological infection, strongly indicate that the cause is an autointoxication as the result of the effect of heat on the body tissues. My experience led me to believe that this is the cause of heat hyperpyrexia.

The autointoxication occurring in heat-stroke was found to be associated with the presence of acetone and diacetic acid in the urine in only a small proportion of cases (about 12 per cent.), and in these the reaction was of only moderate intensity. It thus appears that heat hyperpyrexia is not due to an acid intoxication. The presence of indican in excess in the urine in acute cases is of importance and is an evidence of the autointoxication present.

Dr. W. Cramer[3] has shown that beta-tetrahydronaphthylamine will cause hyperpyrexia in animals, and it is likely that similar chemical bodies produced as the result of the effects of heat on the tissues in man may have a like effect.

No evidence of bacterial infection was obtained in a numerous series of blood cultures in cases of pure heat hyperpyrexia.

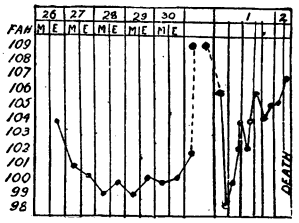


CHART 7.—Malaria and heat-stroke. From the 26th to 30th quinine gr. x t.d.s. by mouth; on the 28th and 30th intramuscular quinine.

Suppression of sweating has been stated by Dr. K. G. Hearne[4] to be the cause of heat-stroke. Undoubtedly suppression of sweating is a most important predisposing cause in many cases, especially those of Group IV in my classification, and in this connexion Dr. R. J. Love[5] has called attention to the risk of giving atropin injections, since he had observed hyperpyrexia follow the administration of atropin before an anaesthetic.

Suppression of sweating did not, in my experience, always precede heat hyperpyrexia, and though it is undoubtedly an important predisposing cause, it cannot be regarded as the primary cause. Thus some cases of heat hyperpyrexia occurred suddenly in exposed men who were in good health, without any previous evidence of lack of skin action, and in cases of the gastric type of heat hyperpyrexia no previous suppression of sweating was observed, though they had been under observation in hospital for several days.

Though entirely agreeing with Dr. Hearne's conclusions as regards treatment, I cannot accept his explanation as the cause of heat hyperpyrexia.

Morbid Anatomy.

In the *post-mortem* examinations made in Mesopotamia oedema and general hyperaemia of the brain and leptomeninges were observed, and cloudy swelling of the liver, kidneys, and myocardium was found; petechiae of the skin and mucous membranes were seen in some cases.

No definite signs beyond these were observed beyond those indicating intercurrent diseases, such as malaria with enlarged spleen, etc.

CLINICAL TYPES OF ILLNESS CAUSED BY EXPOSURE TO HEAT.

1. Heat exhaustion (mild type).
2. Gastric type.
3. Choleraic or gastro-intestinal type.
4. Heat hyperpyrexia (heat-stroke or sunstroke).

In Types 1, 2, and 3 heat hyperpyrexia might suddenly develop unless great care were taken in their removal from a hot atmosphere. Types 2, 3, and 4 were all dangerous and the prognosis grave. Of 80 severe cases of "effects of heat" of which I made careful notes, 13 (16.2 per cent.) were of the gastric type, 9 of the choleraic (11.2 per cent.), and 58 (72.5 per cent.) were hyperpyrexial.

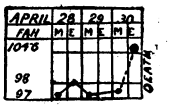


CHART 10.—Typhus and fatal hyperpyrexia.

1. *Heat Exhaustion.*

This would commence suddenly with weakness, giddiness, faintness, and inability to walk; the pulse was rapid and weak. The majority of these cases were associated with a mild pyrexia of 102° or 103°, which lasted two or three days and then the temperature became normal, the patients requiring a few days' rest before being fit for duty. A small proportion of the cases were associated with cardiac failure, the temperature being subnormal.

Returns from two British Stationary Hospitals at Baghdad (July 8th to 19th, 1917).

	Heat Hyperpyrexia (Temp. 105° and over).		Effects of Heat (No Hyperpyrexia).	
	Cases.	Deaths.	Cases.	Deaths.
Stationary Hospital (A) ...	63	15=24.0%	67	4=6.0%
Stationary Hospital (B) ...	37	12=32.4%	162	9=5.6%

Heat-stroke and Effects of Heat (July, 1917).

	Heat Hyperpyrexia.		Effects of Heat.	
	Cases.	Deaths.	Cases.	Deaths.
British Stationary Hospital at Baghdad	40	14=35%	430	31=7.2%

the skin cold and clammy, the face pale, and the pulse very rapid and feeble.

During military operations in hot weather large numbers of cases of heat exhaustion occurred. The mortality from this type of case was very slight.

2. *Gastric Type.*

This was one of the most interesting and treacherous forms; it constituted 16.2 per cent. of my series of severe cases. The patient would have a flushed face and be restless and irritable, with marked nausea and occasional vomiting.

The mouth temperature and pulse would be normal for several days. The rectal temperature showed often a rise of about 2°. Usually there was fatty enlargement of the liver. The knee-jerk was lost in all of nine cases in which it was specially tested. This was a valuable diagnostic sign. The knee-jerk reappeared when convalescence was established, the time varying with the severity of the case. Many of these cases, after four to ten days' premonitory symptoms, suddenly developed fatal heat hyperpyrexia. The following are notes of two typical cases:

CASE I.

H. Onset July 15th, 1917; the patient was morose and irritable, with a feeling of nausea. When admitted to hospital on July 17th the face was flushed, the tongue very furred, the temperature normal; vomiting occurred daily, and the liver extended two inches below the right subcostal border. On July 22nd the mental condition was somewhat muddled, and the temperature was 99.2°; nausea was present, and vomiting occurred once. At 11.45 p.m. the temperature suddenly rose to 108° F., and the patient died on July 23rd, at 1.30 a.m.

CASE II.

K. Onset July 13th, 1917. The patient became faint and collapsed and vomited, some cyanosis being present. When admitted to hospital the temperature was normal, the face flushed, the tongue furred, and nausea and daily vomiting occurred; the liver extended three inches below the right subcostal border. The mental condition showed depression and irritability, with some mental confusion. On July 23rd stupor and coma developed, the temperature suddenly rose to 108° F., and death occurred a few hours afterwards.

3. *Gastro-intestinal Type (Choleraic).*

In this type (which constituted 11.2 per cent. of my series) the onset was sudden, marked collapse occurring. The patient usually had a temperature of 101° to 103°. Vomiting was marked, and there was diarrhoea. Cramps in the abdomen and legs occurred in some cases. The face was pale, the eyes sunken, and the skin pale and clammy. The knee-jerks were generally lost; in four cases specially examined they were absent in three and diminished in one. The general appearance of the patient was similar to that of cholera.

This type was of uncommon occurrence, but the mortality was high, death often occurring within three or four days.

4. *Heat Hyperpyrexia.*

In this type, which accounted for 72.5 per cent. of my series of eighty severe cases, the onset was often quite sudden, the patient being taken ill when on duty, with a sudden high rise of temperature and loss of consciousness.

In some cases the patient when off duty would be found by his comrades unconscious and dying.

CASE III.

In one such case a soldier complained of headache in the morning; he returned to duty and was apparently better in the afternoon, and was last seen playing with a dog, but about 5 p.m. was found unconscious and dying. He died at 5.30 p.m. before medical aid could reach him, and 1½ hours afterwards the rectal temperature of 110° indicated that heat hyperpyrexia was the cause of death.

Heat hyperpyrexia frequently occurred in the very hot weather in hospital patients suffering from another disease. In them the temperature would often suddenly rise to 110°, coma and convulsions supervening. (See Charts 1 and 2.)

In many cases, however, the onset was more gradual, malaise, headache, and restlessness occurring, accompanied sometimes by nausea and vomiting. Frequency of micturition was a characteristic early symptom, and was sometimes associated with urethral pain. The temperature would be somewhat raised—100° to 102° or so—and the skin be hot and dry. These preliminary symptoms usually lasted for a few hours, sometimes as long as forty-eight. Then mental excitement and delirium would supervene and the temperature rapidly rise to 110° or so. With the hyperpyrexia coma and stertorous breathing occurred, and the face was flushed and cyanosed, the conjunctivae being congested. The pupils were often dilated in the early stage and contracted in the comatose condition. Fibrillary twitchings of muscles, and convulsions were very common in this stage, and the breathing might have the Cheyne-Stokes character. Incontinence of urine and faeces occurred with the coma. Unless the temperature was reduced by treatment death rapidly occurred from hyperpyrexia, the mode of death usually being asphyxial in type.

Marked cardiac dilatation, often associated with a systolic murmur, occurred in the severe cases. This remained for a few weeks after the attack and needed special care.

Bronchitis and congestion of the bases of the lungs occurred in some cases.

Air hunger was definitely observed in one case, and in another a spasmodic type of breathing like uraemic asthma.

Pulmonary oedema was a terminal event in the fatal cases.

The Urine.

Indican was present in excess in all of six acute cases in which a special examination was made. *Acetone* and *diacetic acid* were present in small amount in one out of eight acute cases specially tested. *Albumin* was found present in small amount in three out of eight acute cases. No casts were present.

Nervous Symptoms.

Restlessness and delirium occurred with the onset of hyperpyrexia and were quickly followed by stupor and coma with incontinence of urine and faeces. Muscular twitching and convulsions were very common with the high temperature.

The knee-jerk was almost always absent in the pure heat-stroke cases during the acute stage. Out of thirty-two cases specially tested the knee-jerk was absent in twenty-seven, diminished in two, and normal in three; in these latter cases the hyperpyrexia was probably malarial in nature. In the severe cases the knee-jerk did not return for three or four weeks, in the milder cases earlier. The presence of knee-jerks was a valuable prognostic sign, for when they had returned there appeared to be much less risk of a relapse, and the patient might then be evacuated with safety. This sign was of great practical help in deciding on the disposal of heat-stroke cases.

After the acute stage in the severe cases marked mental symptoms often remained for some weeks; irritability, mental confusion, and delusions were not uncommon. Usually these symptoms cleared up.

Defective articulation (anarthria) occurred as an after-symptom in four of the severe cases of my series. *Nystagmus* occurred in one case, and squint with diplopia in one.

Multiple neuritis associated with weakness and marked wasting of the legs, the tibialis anticus muscles being most affected, occurred in two cases.

Lumbar puncture was performed in several cases; the cerebro-spinal fluid was clear and sterile and the pressure was above normal.

Parotitis occurred in three cases of my series.

Petechiae were observed in a few of the cases showing marked toxaemia.

Many of the severe cases showed, after the hyperpyrexia had subsided, a pyrexia, for several days the temperature being about 102° or 103° (see Charts 3 and 4). In one case the fever lasted three weeks. Numerous blood cultures were made in these pyrexial cases, with negative results. Recurrences of hyperpyrexia were very likely to occur after an attack of heat-stroke unless the patient was kept in a cool atmosphere, which was often impossible. There were several instances of two and three recurrences (Chart 5).

After convalescence from hyperpyrexia attacks of headaches were common, and exposure to slight heat would induce them. These patients for a long time were very sensitive to heat, and evacuation to a cool climate was essential. In some of the severe cases showing marked persistent nervous symptoms it was clear that permanent organic changes had resulted from the effect of heat on the brain cells. Dr. R. M. Stewart[6] has described a case with persistent cerebellar symptoms following an attack of heat hyperpyrexia in Egypt.

TREATMENT.

1. *Heat exhaustion* required no special treatment beyond rest, keeping cool, and aperient medicine.

2. *The gastric type* was dangerous, and required great care in treatment. Free purgation, great care in protection from heat, large doses of sodium bicarbonate (30 grains every three hours), and rectal injections of cold solution of the same salt (2 drachms to the pint) gave the best results. The diet in all "heat" cases was chiefly lacto-vegetarian, the fat and protein being reduced as far as possible.

3. *The choleraic type* required treatment on the lines of cholera cases, including normal or hypertonic saline subcutaneously, and cardiac stimulants—for example, digitalin, strychnine, adrenalin, pituitary extract given hypodermically. Protection from heat was most important.

4. *Heat hyperpyrexia* demanded immediate application of measures to reduce the temperature. Thus, the stripping of the patient and constant application of a spray of cold water, or rubbing with blocks of ice, were essential until the rectal temperature fell to 102°, the patient being under a fan during the process. In the hospitals in Mesopotamia special heat-stroke wards were established, with overhead fans and portable electric fans for each acute case. Ice-cold water was supplied to each bed, so that the patient could have a constant spray of this; there was also an ample supply of ice. At my suggestion the acute cases were treated on an ordinary iron bed, a rush mat, with a smooth Japanese reed mat above it, being placed over the iron framework. This allowed a free current of air all round the patient, from below as well as above.

By these means the hyperpyrexia could be controlled. If enlargement of the spleen was observed, or if there was a previous history of malaria, quinine bihydrochloride gr. x was given either intravenously or intramuscularly without delay, and during the carrying out of the hydrotherapeutic measures. An injection of quinine gr. x was given if the temperature was not readily controlled by the above methods. Blood films were made at once and examined for malaria, and if the result was positive, active quinine treatment was continued.

Convulsions, which were of very common occurrence, were treated most satisfactorily by venesection, 10 to 20 oz. of blood being withdrawn. Intravenous saline after venesection was not found beneficial, since it tended to cause recurrence of the convulsions. Rectal injections of ice-cold water or of ice-cold solution of sodium bicarbonate (2 drachms to the pint) were beneficial.

Cardiac failure was treated by digitalin, strychnine, adrenalin, or pituitary extract hypodermically.

Failure of respiration was treated by artificial respiration and oxygen, or oxygen bubbled through alcohol.

Some cases with violent delirium and convulsions were treated by morphine hypodermically, and chloroform inhalations, but these methods were usually inferior to venesection.

PROPHYLAXIS.

Every care was taken by the army authorities in the prevention of heat-stroke. As far as possible no military operations were carried out during the hot weather, and as far as possible also troops were relieved from any duty between 10 a.m. and 4 p.m. Head and spinal protection were ensured by the issue of efficient helmets and topees, and spinal pads. Special light summer clothing was issued to troops in the hot months. A free supply of water was allowed to troops, and every man had a 1 gallon canvas *chagul*, so that by keeping drinking water in it efficient cooling was obtained by evaporation.

Army Orders were issued, on the advice of the D.M.S., warning against the risks of constipation and the taking of alcohol.

Billets and huts for troops were supplied with electric fans. In the case of troops under canvas special care was taken as regards the management of tents so as to ensure maximum heat protection.

Hospitals were equipped with an ample supply of electric fans and ice. In hospitals where patients suffering from other diseases showed a tendency to hyperpyrexia it was found advisable to place them in the coolest part of the wards under a fan and to cover them with a sheet kept wet with cold water.

Heat-stroke stations, with all facilities for treating cases were established in each of the base areas, and each division of troops had six such stations, so that every facility was provided for the treatment of emergency cases.

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THE PHYSIOLOGICAL ASPECT.

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HEAT-STROKE results from the rise of body temperature to a height incompatible with the maintenance of the equilibrium of the physico-chemical reactions in the cell on which life depends. Such rise may result from inability of the heat-regulating mechanism to control the body temperature under the atmospheric conditions, or from exhaustion of this mechanism. Infection, drink, fatigue, by weakening the mechanism, enhance the risk of heat-stroke.

Heat-stroke may occur in either dry or moist hot shade conditions, or from exposure to sun on close, warm days. While sunburn is an inflammation of the unacclimatized and untanned skin resulting from the sun rays, sunstroke is only another form of heat-stroke. There is unreasoning fear of exposure to the sun in this country; so long as the body is exposed to cooling breeze, exposure to the sun cannot produce sunstroke. The heating effect of food, especially of protein-rich food, must be borne in mind in the tropics. Alcohol is contraindicated if taken in place of natural foods. It contains no vitamines or protein-building groups, but is a fuel to the body. Monkeys fed on a light diet of bananas and rice and given plenty of water can be trained to stand exposure to the tropical sun out of doors for many hours a day. The sweating mechanism becomes more efficient by training.

The effect of enclosure and 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 of sweat becomes impossible; as heat production continues, the body temperature inevitably rises. The rise of temperature accelerates the rate of combustion in the cells, and a vicious circle is established. In the Black Hole of Calcutta the air entangled between the bodies and in the clothes of the victims became saturated with water vapour and heated to body temperature; thus heat-stroke, not suffocation, put an end to their sufferings. As the body, weighing some 60 to 70 kg., takes time to heat up several degrees to the critical temperature, people withstand temporary exposure to the heat of a