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*Introduction*



# Hyperpyrexial Heatstroke

## Introductory remarks

while present with the British forces in Mesopotamia from the beginning of August 1916 to the end of October 1918, a large number of cases of heatstroke came under my care.

During part of this period, I was (from May 1917 onwards) attached to No 3 British General Hospital, Basra, and thus had a very favourable opportunity of studying the disease in its clinical form, especially throughout the exceptionally hot summer of 1917.

Although present for nearly 3 summers in this country, I saw no cases of heatstroke in 1916, having arrived shortly after the commencement of the "Shamal", (north wind) which usually terminates the "heatstroke weather" and makes the general living conditions much more comfortable. It is, therefore, chiefly with the summer of 1917, (and to a much less extent that of 1918) that this paper deals.

Owing to the present confused state of the nomenclature of heat diseases, it would seem advisable to mention that throughout this article, the word "heatstroke" (unless otherwise stated) means the hyperpyrexial form only, that being by far the commoner type which came under my notice.

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Some idea of the climatic conditions that prevail during the Mesopotamian summer may be gathered from an inspection of the 1917 meteorological chart (see page 62) and the tables of other meteorological statistics on pages 58-60.

From the chart it will be seen that in July a very definite heatwave of about three weeks duration occurred, when the maximum daily temperature reached the region of  $122^{\circ}$  F. on 3 separate occasions.

During the latter part of July and the beginning of August these excessive heat conditions abated somewhat; but towards the end of August another less definite heatwave commenced, and continued well into the month of September.

The maximum temperatures here recorded agree very closely with those of Baghdad, though the humidity readings differ somewhat; so that it may be said, in general, that the chart represents approximately the climatic state in existence at the time throughout the whole of the occupied territory.

Under these trying circumstances it is not surprising that a large number of the troops were affected, and when it is remembered that the temperature inside tents was, on an average,  $10^{\circ}$  F. higher than the official shade temperatures recorded, one can form an estimate of the suffering

and danger to which the troops were exposed.

In This practically barren Country, where a few scattered towns provided the only permanent building accomodation in existence, the problem of sheltering the troops was an extremely difficult one.

Mesopotamia possesses, naturally, no building materials suitable for the rapid erection of substantial huts, and everything of this class had to be imported from India. Delays, of course, were unavoidable; but the material, as it arrived, was soon put to good use by the engineers, who did much to improve the Conditions by the construction of wooden huts with mud-protected roofs.

It was chiefly at the bases however that these huts were built; so that in the matter of accomodation, and in many other ways, those stationed in these regions fared better than their less fortunate companions, who were situated in parts of the country where canvas, or improvised shelters, formed the only possible refuge from the fierce blazing sun.

Even in outlying parts, the permanent camps, such as stations on the Basra to Kairiyah railway etc. were hatted to a greater or lesser extent, but all temporary camps, intended to last a year or so, had to remain satisfied with tents.

At the bases, too, a state of comparative luxury reigned; for here were established

large power houses for the generation of electricity. Ice and Soda-water factories were also erected, and pumping stations distributed filtered and chlorinated water through pipe systems to the various hospitals and Camps.

The hospitals were liberally supplied with electric punkhas and fans, and, as far as possible, the main official buildings, and principal parts of the neighbouring Camps were similarly treated.

Despite these favourable conditions heatstrokes occurred on a large scale during this particular summer; the total number that took place in Basra alone being 541. Of these, no less than 226 were treated at N<sup>o</sup> 3 British General Hospital.

The scenes at the Basra hospitals in the evenings of the hottest days were very disquieting. Every few minutes a motor ambulance would arrive with an unconscious patient, perhaps in convulsions, or struggling with the attendants who were rendering first aid treatment; while the hospital nursing staff, already overworked with previous admissions, had the greatest difficulty in fulfilling the extreme demand that all cases of heatstroke make upon their services. These duties, much to the credit of the nurses and orderlies, who, at times, were taxed to the utmost limit of their powers, were carried out admirably.

Although every unit in Mesopotamia made the best provision for rendering first aid to heatstroke cases that the local conditions would allow, it was, of course, possible at the bases to make arrangements for this purpose on a more elaborate scale.

At the busiest centres, and on the main thoroughfares, "heatstroke stations" were established. Each consisted of a double-roofed hut, containing a stretcher, a well stocked ice chest, and an ample supply of cool drinking water, stored in porous "chaltees", from which individuals could obtain a cool drink on application to the attendant.

Much useful work was done by them, and, as they were all in easy telephone communication with the motor-ambulance units, there was little time lost in transferring heatstroke cases to hospital after first aid had been rendered.

In all parts of the country, during the heat of the day, outside work was entirely suspended except in cases where absolute necessity demanded its performance.

The troops were dressed in the lightest possible clothing, consisting of shirts (open at the neck) khaki-drill shorts, helmets, neck shades, spine pads, light puttees etc; while tinted goggles were worn during exposure to the sun. (see photograph on page 74.)

Bathing in the river after sunset was freely allowed, and probably helped considerably to keep the men fit during the trying summer months.

It must not, however, be supposed that the majority of cases of heatstroke occurred among troops who were engaged in laborious duties, or even those that were exposed to outside conditions. On the other hand it was chiefly among men who were resting in their huts or tents during the heat of the afternoon that the attacks took place, while occurrences among hospital patients were exceedingly common, most of the cases treated at No 3 B.G.H. having been attacked while lying in bed in the huts of the hospital itself.

The prevalence in hospitals was largely accounted for - in Basra at least - by a severe epidemic of sandfly fever, which was in progress at the time, and which no doubt was a very important complicating factor.

On page 14 will be found a rough plan of No 3 British General Hospital (generally known as No 3 B.G.H.) and, as occasional reference to it will be made throughout this paper, it would seem advisable to include a brief descriptive note here.

The "Palace" (see photograph, page 12)

a large thick-walled brick building, the property of The Shaikh of Meahammerah, formed the central figure of the whole hospital. In it were contained the hospital offices, X ray room, dispensary, pathological laboratory, and heatstroke room, together with a large amount of accommodation for patients who were more or less seriously ill.

Almost immediately in front of the palace was a T shaped pier, which provided a berth for large ocean-going hospital ships, and from it were embarked all the patients who were being invalided from Mesopotamia to India.

Extending along the bank of the Shatt-el-Arab river, on both sides of the palace, was a double row of hospital huts of different types, and varying in size from 20 to 60 beds. (see photographs on page 13)

Each hut had a shade verandah about 8 to 10 feet wide extending along the 2 sides, and from the projecting edges of this, "Chics" were hung so as to screen the rays of the morning and evening sun.

The upper portions of the side walls could be completely thrown open by hooking up the hinged screens (wood + "cha tai" matting) which constituted them, and, although it was found preferable to keep the huts closed during the hot hours of the day, the benefit of raising the screens after sunset was very

noticeable.

The roofs were of Corrugated iron supporting a layer of mud and Cement about 4 inches thick, and along the uppermost ridge ran a covered ventilating space.

The floors of the huts were of Cement.

Although these huts were much cooler than tents they were, nevertheless, very hot, the temperatures registered inside them approximating fairly closely to the curve of maximum temperatures shown in the statistical chart on page 62.

Various devices were tried for the purpose of cooling them, and, of these, perhaps the most successful were the playing of the fire hoses on the roofs during the hottest afternoons, and the hanging of wet "Ker-Ker tatties" over the open doors. The "tatties" however required constant attention to keep them wet, but, provided it was possible to obtain the labour for this purpose, a decided improvement resulted.

Inside the huts the atmosphere was kept more or less in motion by the electric roof punkhas (approximately one over every 3<sup>rd</sup> bed) which revolved day and night throughout the hot season.

The full accommodation of this hospital was 1,040 beds, and, during the 1917 summer, not only was this fully occupied, but the large dining hall was also pressed into service as a ward for some time, while at one period a considerable portion of the adjacent Indian General Hospital

had to be taken over as an extension of No 3 for the purpose of receiving British patients, the available accommodation in the Indian hospital being explained by the fact that Indians do not suffer from effects of heat to the same extent that Europeans do.

My duties at the hospital during the 1917 summer included the charge of Hut 3; (p. 13) all of its 60 beds being occupied throughout this period with cases of acute sandfly fever, hence these patients were particularly liable to attacks of heatstroke, and constant supervision was necessary to prevent occurrences.

Heatstroke on Ships

About the middle of August 1917 large numbers of British troops began to arrive in Mesopotamia direct from England, and, as the transports in which they made the voyage were unable to cross the Shatt-el-arab bar, and thus complete the journey to Basra, it was the custom for each ship to anchor near the lightship at the head of the Persian Gulf, and there transfer its troops to a liner of smaller draught which took them up river to Basra, a distance of about 80 miles from the anchorage.

I was given instructions to proceed from Basra to the bar to meet these ships as they arrived, and to be responsible for the prevention and treatment of

heatstrokes among the troops, both at the bar and during the journey up river.

The region of the bar and lower reaches of the river was noted for the particularly hot and trying atmosphere which usually prevailed there, and this, together with the necessarily crowded conditions that existed on the smaller transports during the seven hours journey up river, greatly predisposed to the occurrence of attacks.

During these trips I had a favourable opportunity of observing the effects of climate on unseasoned troops, and of testing the efficacy of arrangements designed to counteract its ill effects.

Heatstroke in 1918

Guided by the experience of the heatwaves of 1917, special arrangements were made early in the summer of 1918 at No 3 B.G.H. for the prevention and immediate treatment of heatstroke cases.

During this season, however, many factors contributed toward bringing about an immense reduction in the number of occurrences. Chief of these were the more favourable atmospheric conditions with an almost entire absence of the south wind, which appeared to play a great part in causation during the previous summer.

I have not charted the temperatures and occurrences for this year, but the chief

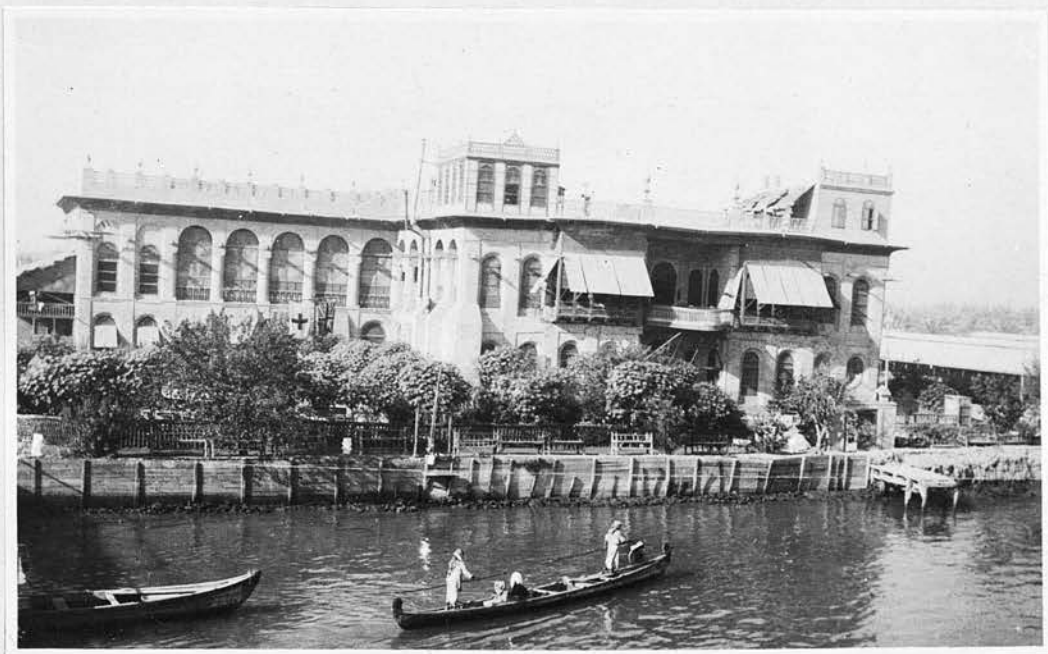
Meteorological figures are given in tabulated form on page 58, the days on which heatstrokes occurred at N° 3 B.G.H. being marked with an asterisk. It will be noticed that the highest temperature was 116° on July 2<sup>nd</sup>.

Other important factors were the greatly reduced number of British troops in the country at the time, vastly improved hut accommodation, and the absence of a severe epidemic of febrile diseases, such as sandfly fever, which prevailed at the corresponding period of 1917.

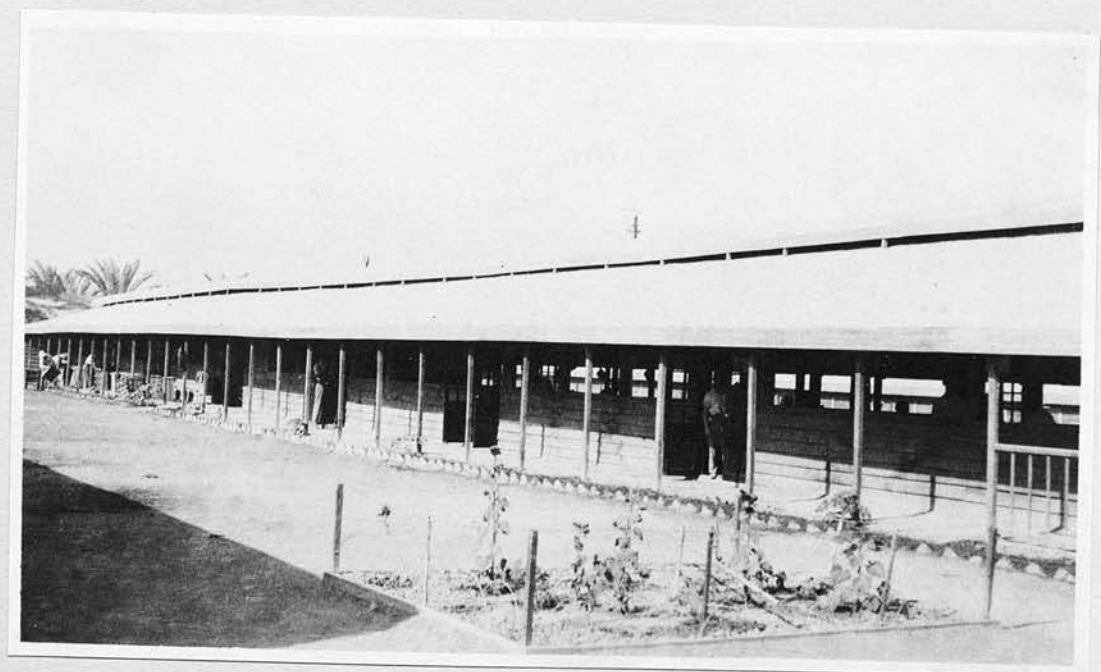
During the greater part of this summer N° 3 B.G.H. was only about half full. In fact the conditions of accommodation were entirely reversed, the neighbouring Indian general hospital having to extend into N° 3 British, owing to a severe epidemic of influenza which broke out among the Indians at this time, while the British were comparatively unaffected.

As regards heatstroke occurrences for the whole base (Basra) during this year, I have, unfortunately, no reliable figures, but the total number of deaths from this condition at the base was 14.

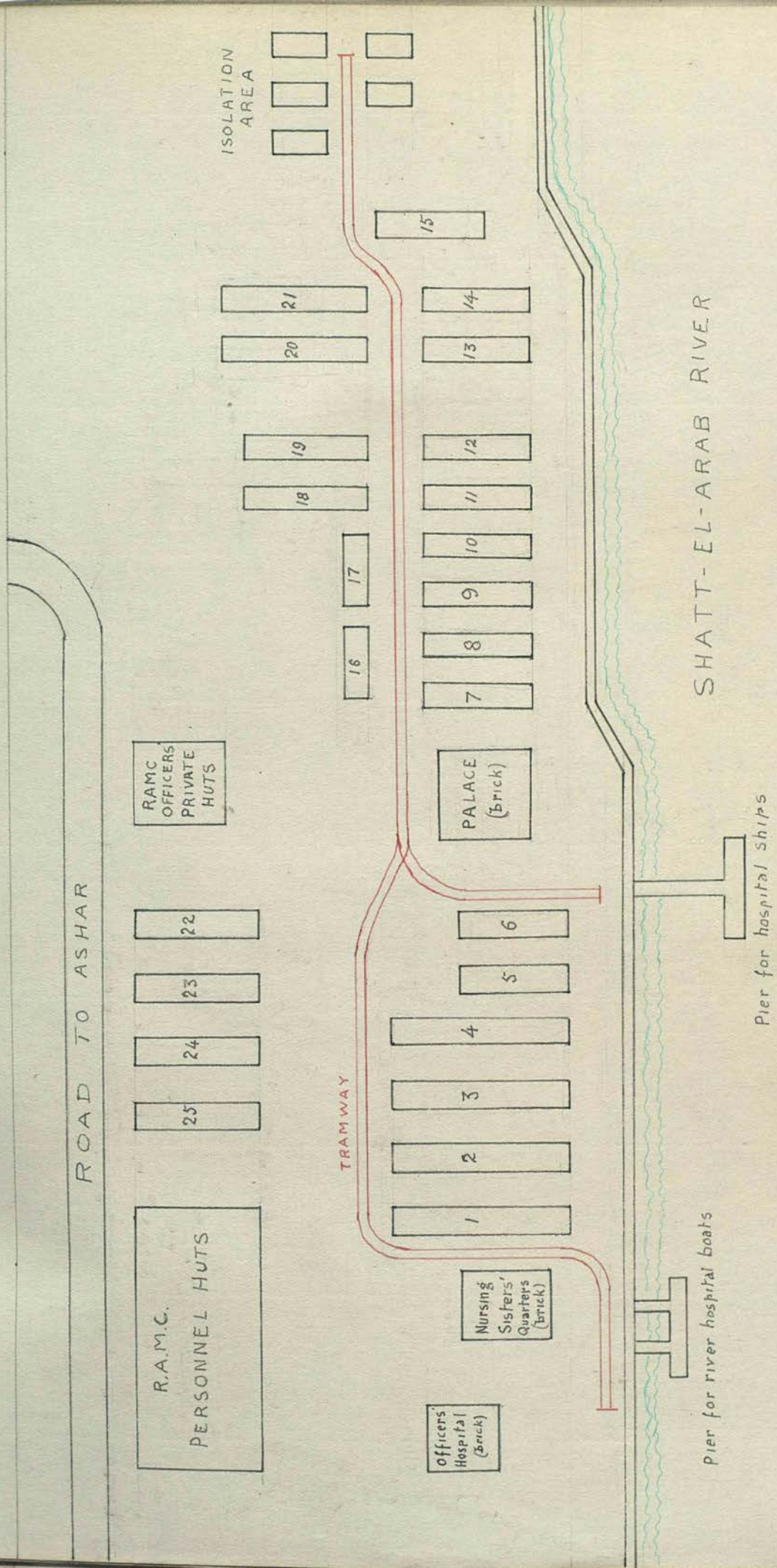
At N° 3 B.G.H. the number of cases that occurred in the hospital huts was only 4, and the deaths (from this condition) nil.



N°3. B.G.H. "The Palace"



HUT 3 N° 3 B.G.H.



Nº3 B.G.H. BASRA - PLAN OF GROUNDS & HUT ACCOMMODATION.

*Etiology*

# Hyperpyrexial Heatstroke

## Definition

Heatstroke is a condition of hyperpyrexia with loss of consciousness, during more or less prolonged exposure to excessive heat, and without necessarily subjection to the influence of the direct rays of the sun.

## Etiology and Pathology

On studying the tropical text-book literature of heatstroke one is struck with the variety of opinions that are expressed by different authors regarding the etiology + pathology of the disease, and the general position may be said to be more or less in a state of chaos.

There is general agreement that its occurrence is "intimately connected with the hot season", and, although Roper<sup>1</sup> and many others believe that atmospheric heat, or heat plus humidity can alone account for it, this opinion is not held by Manson<sup>2</sup> & others<sup>3,4</sup> who support Sumbour's theory in which the condition is considered to be "not a heat fever but a specific infectious disease", which is called by "its oldest name - Siroiasis"

E.R. Pitt<sup>5</sup> expresses his view as follows -

" There is undoubtedly much in favor of the views of those who regard heatstroke and heat prostration as due to an auto-intoxication from the accumulation of toxic substances, resulting from increased metabolic activity due to excessive heat retention, and having a selective action on the nerve cells.

Skitt (continued)

Others think, that as the result of more active metabolism, there is a retention of Carbonic & lactic Acid, with a demand on the alkali content of the blood, resulting in an acidosis.

It would seem advisable to take the ground that heat retention, resulting from lack of heat radiation and insufficient skin evaporation, causes various manifestations of discomfort or bodily injury "

Many other opinions have been expressed from time to time, but the above mentioned are the chief ones that prevail at the present day in leading text books on tropical diseases, several of these books bearing the dates of 1917 and 1918.

In a recent British Medical Journal <sup>6.</sup> it has still further been suggested -

"that heatstroke, as a clinical entity, is non-existent, and is nothing more than a symptom, on a hot day, of malignant tertian malaria."

While such disagreement exists among writers on the subject, and the pathology of the disease remains so imperfectly understood, it is hardly to be expected that a practitioner newly arrived in the tropics, or one with little or no practical experience of the malady would have complete confidence in any particular line of treatment.

This is obviously an undesirable state of affairs, which is very apt to lead to the adoption of therapeutic measures, largely of an experimental, and possibly harmful nature;

or, perhaps (if the practitioner happens to be enamoured with one or other of the toxic theories) even to the universal practice of venesection and intravenous injections of various solutions at an early stage, and in every case, for, if the toxic etiology is accepted, something must obviously be done, in addition to the urgent treatment for hyperpyrexia, to combat the toxins.

The chief object of this Chapter, then, is to endeavour to clarify the situation by showing reasons why some of these theories should be no longer entertained, in the hope that, by their exclusion, a more rational form of treatment may result.

To do this I propose to briefly review the methods by which an insight into the nature of heatstroke may be obtained, offering comments on some of the arguments that have led up to the adoption of the diverse views expressed above, and to produce evidence, derived from a clinical study of the disease in all its various stages, under exceptionally favourable conditions, and from the response of cases to early treatment, together with the results of preventive treatment, that heatstroke is capable of a very simple explanation.

We will first consider etiological evidence based on argument.

Writers who strive to prove that heat cannot cause heatstroke, and that some other cause must therefore be sought, will freely quote numerous instances of human beings exposing themselves to, and doing strenuous

work in very high temperatures without developing the Condition.

Such instances<sup>2, 4</sup>, to mention just a few, are -  
Stokers on large steam vessels, even in the tropics, work for 4 hours at a time in temperatures varying from 150° to 180° F; and many of our workmen such as metal casters, glass blowers, and furnace men, are exposed for hours together to far greater heat than ever emanated from a tropical sun, yet they never suffer from Siriasis.

In the United Consols mine at Gwennap, Cornwall, the temperature is 125° F. Siriasis has never occurred among its miners.

Before regarding these arguments as strong evidence on the subject we require to ascertain if there is any artificial provision by which these workers can, even temporarily, escape the severity of their surroundings?

In the case of the stoker, for instance, anyone who has frequently visited stokeholds in the tropics knows that the stoker spends a portion of his time (sometimes a considerable portion) under the wind chutes or artificial draughts provided, and that his body, bathed in perspiration, can lose an enormous amount of heat in a few seconds by this means.

It is probable, also, that in all similar examples some such modifying factors exist.

In any case, stokers (and probably most workers in hot atmospheres) are on duty for 4 hours at a time, after which a rest for a number of hours in a more or less cool atmosphere follows, and such conditions of comparatively short exposure, with recuperative opportunities in between, can in no way be compared with a severe and prolonged heatwave.

There is still the probability, too, that although these workers may not actually develop heatstroke, a systematic examination of them at the end of work would show them to be temporarily affected with a rise of bodily temperature, as were the subjects in Haldane's experiments<sup>7</sup>, which will be referred to later (see page 27.)

Still arguing in favor of Siriasis - an epidemic microbial infection - Sambon and Manson have stated -

1. Its endemic areas are strictly limited.
2. It is unknown in Europe.
3. It is never found in high lands, nor above a relatively low altitude - 600 feet.
4. The interior of Continents is exempt.
5. It is unknown in places with a temperature of  $120^{\circ}$  to  $130^{\circ}$ , while it appears in other places after several days of temperature  $96^{\circ}$ .
6. Attributing the disease to solar influence we should expect it to occur in the hottest hours of the day, instead of which it happens mostly by night.
7. Its relapses are a strong proof of the infectious nature of the disease.

O'Grady<sup>8</sup> has cited and described what is undoubtedly a case of hypopyrexial heatstroke, which occurred in a stoker on board a warship in Plymouth Harbour (England) just after the ship had finished her trials, and thus produces evidence against the microbic theory and in favour of the thermic one.

Rogers, too, has collected and carefully analysed statistics from the British Army in India for the years 1904, 5 + 6. From this material, together with the cases that he has personally studied in Calcutta, and various parts of India, he exhibits strong evidence against the microbic theory.

The following summary is given in his own words -

" To sum up - with regard to the distribution of heatstroke in India, the principal statements on which Sambon & Manson rely in support of their theory that the disease is not due to heat, but to a hypothetical microbe are either untrue or can be quite well explained by the simple heat theory of the causation of the disease "

The full discussion (which is necessarily long) can be read in Rogers " Fevers in the tropics ". It is unnecessary to repeat these arguments here, and impossible to give even a summary of them in reasonably concise form; they are cited because they form the only possible answer to Sambon's contentions regarding geographical distribution, and a

Comprehensive argument against such statements must necessarily be statistical in nature.

Two of the points, however, I am able to comment upon from my own experience; they are -

(a) Time of occurrence of the attack.-

Although heatstroke may occur at night this is very rare. I have known attacks at 2 am. and even at 5.30 am. but they seldom occur after 9 pm.

The usual time is from 2 to 6 pm, and especially between 3 pm. & 5 pm. i.e. shortly after the hottest part of the day.

(b) The relapses.-

The suppression of sweating, which is well known to accompany an attack of heatstroke, frequently persists for several days after the attack, and such a patient will tend to relapse into the hyperpyrexial state unless his temperature is regulated for him; this tendency is determined very largely, however, by the atmospheric conditions surrounding him, and is no evidence of the presence of toxins. (The question of suppressed sweating will be fully discussed later)

Experiments on Animals

R. C. Stiles\* is credited with having first proved the possibility of producing in animals a sunstroke with symptoms & pathological changes similar to those which occur in man. He concluded that the symptoms are the results of the direct action of heat.

\* Boston Med & Surg Journal June 1864, P349. (abstract from Peppers medicine)

The experiments of H.C. Wood<sup>9</sup>, made in 1870 and 1871, and first published in 1872, throw a great deal of light on the subject of etiology, and from his summary the following points are extracted.

Stroke may be produced in animals as readily as in man either by natural or artificial heat, the symptoms being similar to those seen in man.

The heating of the brain of a mammal produces sudden insensibility, with or without convulsions, at a temperature of 108° F. and death when a temperature of 113° F is reached.

The effect of the local application of heat is the result of the direct action of heat on the cerebrum.

No poisons are developed in the blood, but ~~the~~<sup>a</sup> deterioration of the vital fluid takes place, due to the rapid tissue changes induced by the fever, and the more or less complete arrest of excretion.

This deterioration is secondary to the nervous symptoms, and if the heat be withdrawn before it has produced permanent injury to the nervous system, blood, or other tissues, the convulsions & unconsciousness are immediately relieved, and the animal recovers.

"As a postulate" he continues "from these facts & deductions, I think it follows that the nature of stroke is that of a fever, or, in other words, that Coup de Soleil is a fever, not dependent on blood poisoning, but on heat. It is of course possible that the external heat causes the fever simply by preventing the body from throwing off calories which it is constantly forming."

Aron<sup>10.</sup> made a large number of experiments on animals in the Phillipines; those performed on monkeys were the most instructive. He found that -

a monkey continually exposed to sunshine for 70 to 80 minutes dies, but, if protected from the sun, can be kept in the same place for any length of time without suffering injury.

If a monkey is shaved and exposed to the sun, his rise of temperature, and death, take place more rapidly than if unshaved.

That the animals die as the direct result of hyperpyrexia is shown by the fact that monkeys exposed to the sun as before, but with electric fans playing on them, withstand several hours of exposure with only slight rise of temperature, whereas a control animal exposed a few yards away, but with no fan, died of hyperpyrexia in 58 minutes.

S. Koizumi<sup>11.</sup> conducted "experiments bearing on the pathogenesis of thermic fever", and investigated the effects on rabbits in cages where the temperature and humidity were accurately controlled.

He observed that -

At a temperature of 30° to 38° C. for 5 to 8 hours, daily, for 10 days, no symptoms beyond a rise of temperature, dyspnoea, faintness and diarrhoea were produced.

Increasing the humidity up to 85 to 90%

## Koizumi (Continued)

failed to produce the Symptom Complex of Sunstroke, but increase in density, and decrease in alkalinity of the blood were noticed.

At a temperature of  $41^{\circ}\text{C}$ . prolonged exposure of the animal produced slight injury to the red blood cells.

When vigorously exercised under heat conditions for 2 to 5 hours the animals suddenly developed Cyanosis, dyspnoea, and Convulsions, and died in a few minutes; in these cases the postmortem signs were similar to those occurring in man, viz - delayed Coagulation of the blood, and decreased alkalinity or Acidosis. The blood was also of a dark colour, & haemolysis had occurred.

Under heat & humidity for 3 hours at  $35^{\circ}\text{C}$ . the oxygen combining power of rabbits blood was 41.9%, whereas after exercise it was 29.3%.

The normal colour & combining power was restored in the drawn blood by a short exposure to the air, and injection of the blood into other rabbits before, and after exposure to the air, produced very different results.

The author believes that these experiments prove that the blood is unable to dispose efficiently of the products of metabolism, which are produced in abnormal amounts under the severe conditions of exercise in high temperature and humidity.

The above experiments and conclusions would

seem to imply that muscular exertion is a necessary factor in the production of heatstroke, an opinion which is also definitely expressed by Jules Amar<sup>12</sup>, whereas it is well known that heatstroke in human subjects can occur though the person may have been lying in bed for days previously, muscular fatigue being entirely out of the question.

The majority of cases treated at N° 3 B.G.H. occurred under these conditions.

### Experiments on the human subject

This method of studying the production of heatstroke would seem more likely to give reliable results, for here we are dealing directly with the genus concerned, and, although the investigations cannot of course be carried beyond a certain limit, they at least provide us with information which forms a sound basis from which the end result may be inferred, and when considered along with the opinions derived from animal experimentation do much to pave the way toward a definite conclusion.

A selected few of these experiments will now be stated.

In January 1774, + April 1775, Dr George Fordyce, Dr Blayden<sup>13</sup>, Mr Banks + others exposed themselves to dry + moist heat in specially prepared rooms, + observed the results. They found

that they could remain in a dry temperature at  $260^{\circ}$  without apparent

discomfort or rise of bodily temperature for 13 minutes while a beef steak was well cooked, (assisted however by blowing the hot air on it with a bellows) and eggs were roasted hard.

A jar of water reached a temperature of  $140^{\circ}$  after  $1\frac{1}{2}$  hours exposure, and oil, poured on the surface to stop evaporation, caused the water to boil briskly "after a time".

Exposure to a moist atmosphere at  $130^{\circ}$  for  $\frac{1}{4}$  hour caused a rise in mouth + urine temperature to  $100^{\circ}$ .

The conclusions were that if the air be moist, as well as hot, evaporation of perspiration cannot proceed rapidly enough to prevent the bodily temperature from rising.

Castellani & Chalmer's<sup>29</sup> record similar results to the above from experiments carried out in the moist still atmosphere of engine rooms of liners in the Red Sea, & tropical regions; but here 2 different effects were produced.

1. A person in good health first sweats violently without rise of temperature, but, later, his temperature and pulse rate increase till temperatures of  $102^{\circ}$  + over are reached, when the person breaks off the experiment because he feels uncomfortable, after which his temperature slowly returns to normal.
2. On one occasion (a person who was not in very good health) the temperature, after reaching  $101^{\circ}\text{F}$ . ceased to rise. The skin became cooler, the tension of the pulse altered remarkably, & the patient began to look ill. Stimulants were administered, & the patient did not feel quite right till some hours afterwards.

The observers concluded that high atmospheric temperatures can act in 2 ways. In the first class of case the temperature, if continued long enough, would probably have resulted in hyperpyrexia. In the second case the heart became embarrassed, and, if the experiment had been continued, the person would probably have passed into a condition of syncope.

J.S. Haldane<sup>7</sup>, in commenting on experiments such as those described above, mentions that there appears to be great lack of knowledge of the exact limits of air temperature and humidity which can be borne for considerable periods without serious physiological disturbance, and points out that this is remarkable considering its importance.

With the object of determining these danger limits he carried out experiments on himself and assistants, partly in the hot Dolcoath mine, partly in the warm incubating room at the Lister Institute, and partly in a warmed room at the Physiological Library Oxford, and in a Turkish bath.

The results of these experiments were published in 1905, and from them he concluded that

"In still & warm air what matters to the persons present is neither the temperature of the air, nor its relative saturation, nor the absolute percentage of aqueous vapor present, but the temperature shown by the wet bulb thermometer"

If this exceeds a certain point (about 78°F.) continuous hard work becomes impracticable.

If the person is stripped to the waist, and is doing no work, 88°F. is the limit.

In air moving at the rate of 2 miles per hour 93° F. is about the critical wet-bulb temperature.

Steady rise of temperature, profuse sweating, increase of pulse rate, dyspnoea, and exhaustion result from excess of these limits.

The observations of Zuntz + Teudlau<sup>14</sup> on a man whose skin possessed no sweat glands are of considerable interest. They found that the heat of the sun in summer, or the performance of muscular work for a little while, caused a rise of the subjects body temperature to 102° F.

The man himself counteracted the effects of this natural disability by soaking his shirt in water frequently during hot weather.

Information from the clinical study of the disease in its early stages.

Cases of fully developed heatstroke, when admitted to hospital in the Comatoze or Convulsive state, do not, as a rule, exhibit much evidence as to their causation.

On the other hand, when attacks occur in rapid succession among patients who have been in hospital for 2 or 3 days previously, and under the direct observation of the medical officer, a great deal can be learned from carefully studied cases.

During the exceptionally severe summer of 1917, the hospital huts, though their

design could not have been improved upon unless they had been built of brick or stone - an absolute impossibility under the conditions - were, nevertheless, nothing short of incubators in which unavoidable opportunities to study the disease in the human subject occurred under natural conditions, and exceptional facilities for observation were provided just as they would have been during a series of pre-arranged experiments.

For the purpose of trying to devise some means of preventing the appalling number of occurrences that was taking place at the time, I remained in the hut constantly, during the hours of the day when heatstrokes were most likely to occur, and observed a great deal which led me to the following conclusions -

- (a) That suppression of perspiration precedes the attack of heatstroke for a more or less considerable time, and is the definite cause of it.
- (b) That diaphoretics, in strong doses, are powerless to produce a natural perspiration when suppression has set in.
- (c) That by the recognition of suppressed sweating and rise of temperature as definite signs of onset\*, heatstroke becomes an entirely preventable disease, prevention being attained by an artificial imitation of the physical ~~process~~ <sup>effect</sup> of sweating, such as by covering the naked patient with a wet sheet,

\* On a very hot day all patients in the hut were inspected every hour to detect those requiring preventive treatment.

and assisting the evaporative procedure, if necessary, with the current of air from an electric fan.

The preventive measures were subjected to a severe test during practically 2 months of continuous heatstroke weather, both at the hospital, and on troop ships during the journey from the bar to Basra; the results were invariably successful, and this fact is mentioned here owing to its bearing on etiology. The details will be referred to later.

Having related the principal methods of studying heatstroke, and briefly commented on the chief arguments that have been used in support of theories which do not recognize heat as the cause, we may now consider the microbic theory<sup>4</sup> in general (the salient features of which, cited in the original wording, are fairly completely summarized in the following paragraphs) and judge as to how far it is supported by a Mesopotamian experience of the disease.

"The symptoms of the disease, its relapses, its morbid anatomy, its peculiar geographical distribution, its epidemic outbreaks, the conditions of climate and soil under which it prevails, the relative immunity to attacks afforded by acclimatization, all point to the specific nature of the disease."

"Siriasis is characteristically a land infection. At sea it is extremely frequent

in the Red Sea,----- the disease having been contracted while Coaling or stopping along its Coast;----- very few cases have been recorded as having taken place on the open sea, and, although the necessary period of incubation might account for them, they were mostly only cases of "Syncope"

Then under the heading "mode of infection" appears,

"The specific organism of shistosiasis is probably spread in the superficial layers of the soil like other pathogenic micro organisms, and may be conveyed to man with dust blown by the wind, or thrown up under the tread of a marching Column; These are inhaled into the lungs, or ingested into the alimentary Canal, where it produces the deadly toxin, which, probably as in Cholera, becomes absorbed, and sets up the symptoms of the disease"

In answer to the above paragraphs the following incidents may be related.

On one of the occasions that I met freshly-arrived British troops at the Shatt-el-Arab bar (as explained on page 9 q.v.) a large Castle liner anchored at 8.30 am. on 14/8/17. The ship on which I travelled from Basra arrived alongside at 11.30 am. And, on

Crossing to the larger transport, I found the military medical staff already engaged in treating a case of heatstroke, which died 10 minutes after my arrival. About 3 pm. on the same afternoon one of the Ships Crew died, and again at 9 pm. another death took place among the ships crew\*

Preventive measures were commenced immediately on my arrival<sup>†</sup> and continued all through the next day (15/8/17) during which time the 2 ships lay together, transferring guns & war material in the cooler hours of the day. No cases of heatstroke occurred though quite a number were under preventive treatment on the poop deck.

On the following morning (16/8/17) the troops were transferred to the smaller ship, which started for Basra.

The net result of the journey up river was

5 cases of heatstroke<sup>‡</sup> (4 of them discovered lying unconscious in various parts of the ship)

43 under preventive treatment (most of them being transfers from the observation area)

80 (roughly) in the observation area<sup>‡</sup>

\* Medical arrangements for the military & for the ship were entirely separate, & I did not know of these cases at the time.

† A special ships order was read to the troops, & notices posted in conspicuous places explaining the danger of heatstroke, & its chief signs of onset. The men were warned to report sick on deck. Suspicious cases were detained for observation in the "observation area", while preventive treatment (the naked patient covered with a wet sheet in the breeze on deck) was carried out in the "treatment area", both being the coolest spots obtainable on the deck.

‡ One of these, although he had reported sick over 3 hours previously, developed heatstroke while in the "observation area", but, owing to pressure of work, a regular hourly inspection was impossible, hence he was not discovered in time to apply preventive treatment.

None of the patients lying in wet sheets were attacked, and none were placed under this treatment until they had developed suppression of sweating with a temperature of about  $103^{\circ}$ ; hence, out of the whole community, they were the selected few who were most liable to attack; the maximum temperature at Basra (never more than 80 miles away) was  $109.8$  on this day.

On enquiring into the movements of the Castle liner during her voyage from England, I find that she travelled to Mesopotamia via the Cape, left Durban on 30/7/17, and did not coal or stop at any port until her arrival at the Shath-el-Arab lightship on the morning of 14/8/17.

We might assume that the troops were not infected with the organism of Siriasis while at Durban, for this part of Africa is not included in the "peculiar geographical distribution" or "limited endemic regions" to which this disease is said to be confined, nor is it within reasonable distance of such areas.

There remain, then, only 2 possibilities.

1. That the infection took place through the medium of dust blown from the shore while the ship was passing through the comparatively narrow Straits of Ormuz\* on 12/8/17, & that the incubation period was 2 days.
2. That there is comparatively no incubation period at all, & that dust blown from the Mesopotamian shore (the nearest land is about 12 miles from the anchorage) brought the organisms, which commenced their deadly work almost directly the ship anchored.

Are either of these suggestions at all likely to explain the outbreak?

\* The straits are about 40 miles wide but ships may pass within 2 or 3 miles of the western shore at this part.

Here then, clearly, is a case of this land disease having (for all practical purposes) developed on the high seas, which Sambon<sup>4</sup> emphasises it cannot do; or, if it be granted that the microbes were lying latent in the ship, and that the disease can, "in rare instances", appear at sea, how could the mere placing of wet sheets over patients in the breeze on deck prevent them from "inhaling or ingesting" the organisms? for from midday on 14/8/17, when preventive treatment was commenced, the epidemic suddenly stopped among those who availed themselves of treatment, yet it continued among those who did not report sick in time, and who thus gave me no opportunity of considering whether they were likely to develop the condition or not.\*

Prevention acted just as effectively in the cases of the remaining ships that were met, but the above-mentioned (being the best example) is alone described because it embraces all of the points necessary in an argument which is already quite long enough.

Experiences at the hospital on land, too, were precisely similar.

During the whole of the 1917 summer, I was, (with occasional absences for duty at the bar) in charge of Hut 3, as already explained on page 9

\* My conviction is that of the 43 cases that required preventive treatment while going up river, at least 30 would have developed heatstroke before disembarkation at Basra had they not been artificially protected. This estimate is based on previous experience, and the result of delay is well illustrated by the man who developed the condition while in the observation area; he had been selected as a likely case, & he proved to be. The troops were crowded on a ship which was never built for tropical climates, and on which (according to my information) a large number of heatstrokes had occurred about a week previously.

In the first few days of the July heatwave this hut produced 9 cases of hyperpyrexial heatstroke, of which 4 died, but, from the day that preventive measures were commenced only 2 cases occurred in it during the remainder of the season, though they continued to occur daily, often in large numbers, elsewhere. (See statistical chart page 62)

Neither of the 2 patients referred to had been placed under preventive treatment, and considering the great pressure of work, and the shortage of nursing staff at the time, it is not surprising that an occasional case should be missed.

What do these experiences point to?

Do they not form strong evidence that heatstroke cannot be a microbial infection? and do they not indicate that the onset of the condition is extremely simply explained by the fact that the patient has stopped sweating, and has therefore lost his natural means of disposing of surplus bodily heat by the evaporation of perspiration?

### Frequency of Micturition & Suppression of perspiration

One of the most outstanding features in connection with observations on heatstroke cases in Mesopotamia was the fact that, where I had not the opportunity of observing these signs for myself, the patient, on recovery, would almost invariably give a history of suppression of sweating & frequency of micturition having preceded the attack.

In most writings on the subject frequency of micturition seems to be generally recognized as a valuable premonitory sign\*, and, although a

\* Longmore appears to have been the first to point this out.

"dry, and burning hot" skin is almost constantly mentioned as an accompaniment of the fully developed attack, I have never seen it stated as a definite premonitory symptom; nor am I aware of any records of this important point having been thoroughly enquired into, either by direct previous observation of the patient, or by obtaining the patients history of it in a large number of cases\*.

This seems very remarkable since frequency of micturition is, as a rule, extremely uncommon during hot weather, and its occurrence under these conditions would naturally suggest that it was purely the result of the suppression of perspiration.

When making enquiries from patients on these points after their recovery, great care was exercised not to ask "leading questions", and, in dealing with intelligent patients, I would often adopt an attitude calculated to make them qualify their statements, by which their value might be more accurately judged.

On one of the occasions that this method was tried the patient had claimed suppression of sweating at a time which worked out at 49 hours previous to the attack, and, although he noticed no frequency of micturition, stated that

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\* There is no intention to infer that stoppage of sweating has not previously been suggested as a possible cause. Baxter & Zuber thought that "the great rise of body temperature depended on interference with heat loss as a result of suppression of cutaneous perspiration." 4

Rogers; in producing evidence against the Microbic theory, states "In one of my Calcutta cases" (out of a series of 14) "there was a definite history of the hyperpyrexia & unconsciousness shortly following the cessation of free perspiration. This points to a cessation of the functions of the skin as an essential factor in the production of the hyperpyrexia, which, in turn, will, by itself fully explain the other symptoms met with without the assumption of any Microbe-produced toxins." (p. 293 <sup>7 cases in</sup> the Tropics)

he passed water in a particularly large quantity at a time.

I suggested that, during his stay in the heatstroke ward he may possibly have heard that these signs usually precede heatstroke, and asked if his statements were not modified by this belief? He answered that he had not heard this, but, thinking that the symptoms had something to do with the climate, he had noted them in his diary at the time. This he showed me, entered on the date of occurrence, which was 12 days previous to the date on which I was taking his notes.\*

In another instance an R.A.M.C. orderly, attached to my hospital, was attacked with heatstroke at the extremely unusual hour of 5.30 A.M.

On the day after his recovery when taking his notes I obtained a history that on the afternoon previous to attack his sweating had entirely stopped; he passed water 4 times between 1 pm & 6 pm, and required to get up 4 times during the night for a similar purpose, while the suppression of sweating still continued.

I drew his attention to the fact that, as an orderly of this hospital, he should have known the importance of these signs during hot weather, and reported sick.

To this he replied that he knew it quite well, and reported sick at 6 pm, but, not wishing to go into hospital, he only complained of slight headache (his temp. was only 100.6) and asked for Aspirin. He then had a warm bath, & went to

\* patient had just arrived from India. Suppression had occurred when the ship had reached the Shatt-el Arab bar, & heatstroke developed while in hospital on the 2nd afternoon after arrival at Basra. This was not one of my ship cases, the patient being sent into hospital by the M.O. of the transport, undiagnosed (Fever N.Y.D. Temp 101.4). The case was then, no doubt, in the early stage of heatstroke.

bed, believing that sweating would start and he would be all right through the night, but he intended to report sick again the next morning if the signs persisted.

at reveille (5:30 a.m.) he was found fighting with his mosquito net, and in a fully developed attack of heatstroke.

There can be absolutely no doubt as to the preexistence of suppressed sweating in this disease; the point has been so frequently observed, and so exhaustively enquired into.

### What Causes the Suppression of Sweating?

The suppression, I would suggest is due to exhaustion of the sweating mechanism.

There is considerable reason to believe that the sweat glands themselves are the particular part of the system involved, and it would seem possible that the exhaustion is brought about as the result of a number of days of profuse sweating immediately preceding the attack.

The chief evidence on this subject is obtained from the effect of subcutaneously injected Pilocarpine.

In 10 cases, an hour or so after reduction of hyperpyrexia, I have injected  $\frac{1}{5}$  grain of pilocarpine with the object of producing sweating, and the only result was profuse salivation, but no perspiration.

The Sympathetic System in general would appear to be intact, as shown both by the efficient response of the Salivary Glands, and the fact that the onset of suppressed sweating is not accompanied by any remarkable

Changes in the character of the pulse, beyond an increase in its rate.

These facts may possibly admit of the inference that the salivary glands act because they have had no undue strain thrown upon them, while the sweat glands fail owing to exhaustion of their secretory powers.

Wherever the situation of the derangement may be it is most probably of the nature of an exhaustion, and not due to a toxæmia of any other febrile disease (however slight) because many individuals show no clinical indications whatever until after suppression has set in.

The skin is usually flushed during the stage preceding the attack, and indicates an ample blood supply to the sweat glands.

New arrivals in the Country seemed distinctly liable to attack, possibly because their sweating mechanism had not been trained to meet the severe test suddenly imposed upon it, while the fact that some of the "seasoned" individuals who were attacked in Mesopotamia in August, though they had passed successfully through the extremely severe July heatwave (and under practically the same conditions of daily occupation) might be accounted for by the prolonged strain during 2 months of intense heat. (chart page 62)

Heatstroke patients have often told me, on recovery, that, as a rule, they sweat profusely in hot weather, and, possibly, such sensitization mechanisms may be more liable to early exhaustion.

Is hyperpyrexial heatstroke always preceded by suppression of perspiration?

One needs only to study Haldane's experiments<sup>7</sup> to be convinced that, had they been carried far enough, heatstroke would have resulted; and this opinion is borne out by the experiments of Blagden + Fordyce<sup>13</sup>, and of Castellani + Chalmers.<sup>29</sup>

Probably the only clinical difference between cases produced in this way, and those which I studied in Mesopotamia would be the absence of the dry skin, for in none of these experiments is even the tendency to suppressed sweating hinted at.

In reading clinical descriptions of cases that have occurred under natural conditions in various parts of the world, however, it is not always easy to eliminate the possibility that where "wet" cases are referred to they are the syncopal forms.

Manson<sup>2</sup>, in quoting H.C. Wood's<sup>9</sup> account, for instance, says "skin always intensely hot, and generally, but not always, dry; when not dry it was bathed in profuse perspiration"

The fact that the skin was always intensely hot seems sufficient ground to exclude the syncopal form here, and the conclusion therefore is that Wood has met some cases of true hyperpyrexial heatstroke that were not accompanied by suppressed perspiration, and therefore extremely unlikely to be preceded by it.

In Gauss + Meyers<sup>15</sup> cases (a description of 158 cases of sunstroke + heat exhaustion) they state that the skin was "commonly hot, dry + cyanotic"

but, as the cases were admittedly a mixed lot, the description does not help to clear the point under consideration.

At one time I thought that the feature of suppressed perspiration was possibly a characteristic of comparative dry climates such as that of Mesopotamia, while hyperpyrexial heatstroke, with a moist skin, may be common in humid climates such as that of Calcutta, and perhaps Chicago\*, but on referring to Ross's "Fever in the Tropics" this is evidently not so, for the following remark appears on page 293.

"On examination, during the fully developed attack, the most noteworthy feature is the intense heat of the skin and its dryness, with no trace of perspiration, even in the sweat-producing damp temperature of Calcutta heatstroke weather" †

He then passes on to refer to one of these cases that gave a history of preceding suppression, but apparently had no evidence from the others on this point. (See footnote page 36 para. 2.)

Quite apart from the occurrence of heatstroke in mines, engine rooms, or under other artificial conditions, it would seem quite possible that in the natural open atmosphere, the wet bulb temperature, or the humidity, may,

\* Gauss & Meyers Cases in Chicago occurred under the following meteorological conditions - A monthly mean temperature (July 1916) of 78.4 - The highest on record - the hottest day being 101 F. registered on July 30th. From July 26th to 31st The minimum never fell below 82°F, and 100% of sunshine was recorded. The average relative humidity was 58.

† For Calcutta humidities, see Table page 61.

at times, rise sufficiently high to cause heatstroke in a resting subject who was profusely sweating simply through insufficient evaporation, but such natural atmospheric conditions would probably rarely occur.

If, however, the subject were thickly clothed, and doing physical work in addition, the likelihood of heatstroke would of course be much greater, and could occur with less severe atmospheric conditions\*.

J.W. Gregory<sup>16</sup>, in citing a number of high wet bulb readings from various parts of the world mentions that "At the station of Beaufort, in North Borneo - - - readings of close on  $90^{\circ}$  are by no means uncommon".

An almost similar state of affairs is seen in the meteorological chart, page 62. Yet, so far as I am aware, Mesopotamian cases were of the dry skin type, & consequently not due to insufficient evaporation, but to lack of it.

Since the dry & burning-hot skin is so constantly mentioned in clinical descriptions of the fully developed attack, & since all of the 226 cases treated in 1917 at No 3 B.G.H. were of this variety, and since the pre-existence of this suppressed sweating has been so thoroughly established in the majority of these latter cases, I think it fair & logical to conclude that "dry" cases have all been preceded by suppressed sweating. The "dry" is obviously by far the commoner variety, so that the conclusion is that "heatstroke is practically always preceded by suppressed sweating."

\* Muscular exercise causes a rise of temperature in the rectum and urine of healthy men. The temperature may rise to  $102^{\circ}$  without causing distress, or pathological effects, & is an advantage to the performance of muscular work. Experiments on troops conducted at Aldershot, after a 7 mile march, showed rectal temperatures varying from  $98.8^{\circ}$  to  $102.4^{\circ}$  F. (Pembrey)

Heatstroke and Malaria

Malarial parasites are occasionally found in the blood of heatstroke patients, and this occurs with sufficient frequency to justify the necessity of taking a blood film in every case as a routine practice.

There is, however, in my experience, no evidence in support of the suggestion by Capt. Milner R.A.M.C.<sup>6</sup> that

"heatstroke as a clinical entity is non-existent, and is nothing more than a symptom, on a hot day, of malignant tertian malaria!"

On the other hand there is very clear evidence in opposition to this belief.

Even a number of negative results of blood examinations, although forming a valuable indication, constitute no absolute proof as to the absence of malaria. For strong testimony on this point, however, it would not appear necessary to go further than review the incidents already related on pages 31-33, from which it is quite clear that a sudden and severe outbreak of heatstroke took place on these 2 ships, and the state of affairs would have been far more serious if prevention had not been applied.

Careful enquiry showed that, among these troops, the few who had previously travelled to malarial countries was entirely negligible.

I have the full movements of this Castle liner, with accurate dates, from the time she left England till her arrival at the Shatt-el-Arab lightship, and these show that, although Capetown and Durban were called at, there was no

Stoppage at any port, such as Freetown (Sierra Leone) where the troops might reasonably be said to have developed malaria, and, furthermore, on referring to the official voyage report of the Military Medical Officer to the ship, it is there stated that no cases of malaria occurred during the whole voyage.

This outbreak, then, was quite obviously not malaria.

I am not citing experiences on other ships in evidence against a malarial cause, as, in these instances, the troops travelled via Taranto, and the possibility of their infection there would require to be considered.

The views of several others might be here quoted.

Capt Hugh Scott M.S.<sup>17</sup> states that, out of 33 heatstroke admissions to the Hospital ship "Madras" (Mesopotamia) in June 1915\* 13 were diagnosed, either microscopically, or clinically, as suffering from concomitant malaria.

Rogers†, in referring to the "after fever" which sometimes follows heatstroke, says -

"This, I at one time thought might be evidence of malaria on which hyperpyrexia had ensued, but in 8 of the Calcutta series" (14 in all) "I examined the blood for malarial parasites with a negative result in all. Moreover, as they did not occur in the malarial season, that disease can be excluded as a common factor in their production"

\* The H.S. "Madras" carried patients from Mesopotamia to India. The British troops, at this time, were practically all men who had lived in India for some considerable period, and were, no doubt, very largely malarial subjects.

† "Fever in the tropics," page 296.

In Pembrey's analysis<sup>27</sup> of 50 cases, occurring in the British Army in India, between June 1909 and August 1910, the following is recorded.

In 21 cases the blood was examined for malaria. It was negative in 19, and positive in 2.

There can, of course, be no doubt that any febrile condition, e.g. malaria, sandfly fever, enteric fever, relapsing fever, etc. will have a powerful additional effect in raising the temperature of patients who have stopped sweating, and such people are, in consequence, more liable to heatstroke, but, in my opinion, heatstroke is a distinctly separate clinical entity, and not necessarily associated with malaria or any other febrile disease.

### The results of immediate treatment as evidence in the pathology of heatstroke.

If one is fortunate enough to have, ready at hand, the means of treating an attack from the moment of its development, a great deal can be learned from the case, and it is only then that the phenomena, which appear to illustrate the true pathology of the disease, stand out clearly.

This experience has happened to me on 7 or 8 occasions, and the response of these patients to immediate, and energetic treatment has been practically similar in all instances.

What was perhaps the best of these examples occurred whilst on heatstroke duty on a ship.

A man walked along to the "treatment area"

(where materials for treatment were of course kept ready for immediate use) and asked to be examined, as he was not feeling well.

He had the typical hot dry skin of oncoming heatstroke, and, as he was conscious, I put a thermometer in his mouth, laid him on the deck, and proceeded to cut his clothing away.

Whilst his temperature was being taken he showed a great tendency to grind his teeth on the thermometer, and I had barely taken this from him when he went completely unconscious, and commenced to shout and struggle vigorously.

The mouth temperature showed  $108.6^{\circ}$ .

Cooling measures (cold water, ice and an electric fan) were immediately applied, and pressed energetically. The next temperature (rectal) taken a few minutes afterwards was  $109.2^{\circ}$ , and from this time the temperature steadily receded, and the chief signs gradually subsided until in about 15 minutes, when the temperature had fallen to  $104.8^{\circ}$ , the patients struggling had entirely ceased, & he was nearly conscious. Shortly afterwards he was answering questions, apparently sensibly.

Treatment was stopped, and when I visited him about an hour afterwards he was perfectly conscious, and able to discuss the proceedings up to the time he was laid on the deck, but did not remember my asking him questions at the end of treatment.

Beyond the usual slight rise of temperature, during which the patient regained a little

nursing attention while his suppression of sweating lasted, he made a quite uneventful recovery.

The point about this case, then, is that the attack developed with the rise of temperature, and recovery took place with its fall.

The simple inference is that the physical effect of heated blood, having been allowed to act for the minimum of time, produced the minimum amount of damage, which was, in consequence, quickly recovered from, although the symptoms were startling enough at the time, and, from what I have seen of such early treated cases, I believe it represents what would happen in practically all instances if we could only get the patients under energetic and careful treatment from the moment of onset.

Had the patient, however, been attacked in the hot cookhouse (from which he came) and lain there insensible until his discovery, perhaps half an hour or so later, then his clinical state on arrival, and subsequent response to treatment, would have been very different. After reduction of the hyperpyrexia he would, perhaps, have lain for hours in a clinical condition, strongly suggesting the action of a toxæmia, and, possibly, death may have resulted.

Do toxins or acidosis play a part in  
Heatstroke?

From the foregoing remarks it is quite clear that, in early-treated cases, at least, there is no reason to suspect the action of toxins or acidosis at all. The evidence points to overheated blood as the cause, and the patient becomes normal again on removal of this cause, provided it has not been allowed to act sufficiently long to produce a more or less lasting effect.

It is possible, however, that prolonged action of heat may bring about metabolic changes resulting in products, which have an additional damaging effect on the highly specialized tissues.

As regards acidosis, we meet occasional assurances throughout the literature that "some believe an acidosis to be present."

Koizumi<sup>11</sup> appears to have produced some definite evidence on this subject by his experiments on animals (see page 23)

Here, however, he states that the rabbits required to be vigorously exercised for 2 to 5 hours before he could produce heatstroke at all, and that the acidosis was the result of it; but exercise is by no means necessary to the production of heatstroke in the human subject.

It was not, of course, possible at our military hospital to enter into fine points of research in all directions<sup>†</sup>, and we were not able to carry out investigations that would enable us to prove, or disprove the presence of acidosis\* but the simple urinary tests gave no encouragement, only the slightest trace of acetone being found occasionally.

\* Early in 1918 private arrangements were made for obtaining apparatus from England, but this did not arrive.

† The laboratory, in general, however, was very well equipped.

It is a significant fact, however, that Sellards<sup>18</sup> recently published book on acidosis does not mention heatstroke, or any of its synonyms, throughout its pages, and since there is no clinical appearance suggestive of acidosis, which could not be equally well explained by the physical action of bodily heat, it seems quite unnecessary to search for a complicated solution of the problem when a simple one will amply suffice.

My experience, however, has been largely confined to cases that have developed the condition while resting. Possibly, in a large outbreak of heatstroke among troops on the march, the factors of toxæmia & acidosis may play a part, even, perhaps, a considerable part.

To sum up the remarks under this heading, I would suggest that, in a resting case to which treatment has been immediately applied, the question of toxic and acidosis complications may be entirely disregarded. Where treatment has been delayed for some time (say 20 minutes or more) the possibility of their presence may be considered; while, in cases definitely associated with prolonged muscular fatigue, these complications may be even probable.

As a general observation, however, I am distinctly of opinion that measures to counteract these elements (venesection and intravenous injections) have been much overdone, and that the assumption of their presence in every case is a great mistake.

The part played by Actinic rays

Much discussion has taken place regarding this subject, and many arguments have been advanced, both for, and against, the probability of actinic rays playing an important part.

After nearly 3 summers in Mesopotamia (with almost cloudless skies) and a moderate experience of other hot climates in different parts of the world, I have been unable to collect any evidence, with recording, that would tend to prove a damaging effect from the sun by other than its heat rays.

The experiments conducted in the Philippines<sup>24</sup>, among troops of the U.S.A. Army, with orange red underclothing, together with other experiments, showed no evidence that the sun's rays could, or did, produce the ill effects of climate in that country, and, even if the actinic rays had any influence whatever on the system, it was believed that they were sufficiently excluded by the khaki uniform and campaign hat.

Strong evidence against the actinic theory is also supplied by Arous<sup>10</sup> experiment, in which he placed a monkey in a double walled box, with an air space between the walls; the monkey's head only being exposed outside to the full force of the sun's rays. The animal showed no rise of temperature, or other ill effects

though exposed in this way from morning to night, and, after a total exposure of 54 hours, lasting over a period of 12 days, no signs of ill health appeared.

In the same series of experiments other monkeys died after less than  $1\frac{1}{2}$  hours exposure of the full body, and under conditions that strongly suggest the effects were due to heat alone. (see experiments, page 23.)

From personal experience, I am inclined to believe that the discomfort and nauseating effect caused by the exposure of the head & back to a hot sun are exactly comparable to the effects produced by a similar exposure to a strong fire, or by placing a hot water bottle against the spine, in which cases actinic rays are quite out of the question.

The helmet is undoubtedly the best form of head dress in hot sunny countries, and the spine-pad, since it protects the parts that are most liable to suffer, while allowing other parts of the body to be thinly clad, is also invaluable.

Major C. F. Wankill, <sup>25</sup> R.A.M.C. after conducting experiments on the subject, considers that "attention should be paid rather to the means of effective ventilation than to the colour of the lining of helmets"

personally, when choosing a spine-pad, I would select one for its thickness rather than for its actinic-ray-proof colour.

It would seem that we require an exhibition of clinical evidence, greater in amount, and more definite in type, before the profuse charges hurled against the actinic rays are justified. It is difficult to believe, too, that the bony casings of the brain & spinal cord do not, in themselves, offer sufficient protection from such rays.\*

Duncan<sup>26</sup> strongly supports the actinic theory, and advises a red lining to the helmet and coat. He quotes from a personal experience of several attacks of sunstroke, and speaks highly of the comfort that follows upon the adoption of these protective measures.

He cites the instance of Col Maude, R.E. from whose helmet a brother officer had surreptitiously removed the red lining, with the result that Col. Maude developed an attack shortly afterwards.

Much, of course, depends on the thickness of the cloth, or other material forming the lining, and on the fact that a layer of air intervenes between the lining & the helmet. Both are poor conductors of heat, & the wearing of the red lining is not necessarily a proof that the resulting comfort is due to setting up a barrier against actinic rays.

Painting the inside of the helmet red would appear to be a more conclusive test.

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\* Puntone is said to have "shown by an ingenious experiment that the human cranium is diathermal to the yellow-red, & ultra violet rays", and recommends green clothing, covered with white material, to protect important parts, more especially the head, neck, & spine.  
(Manson<sup>2</sup>)

## Post-mortem examinations

Very little, as a rule, is to be found on P.M. examination of these cases. The chief points worth noting are the following -

### Brain + Spinal Cord.

Exudation of fluid under meninges. Cerebral sinuses are engorged; no evident haemorrhages in either brain tissue or meninges.

The organs, in general, are in a state of congestion, the lungs especially so, and almost black in appearance. Fluid blood exudes from the cut surface. There is no consolidation, and pieces cut from the congested areas float in water.

The mucous membrane of the stomach and intestine shows dark patches of congestion.

### Microscopical

In 2 of the postmortem cases, portions of tissue from 5 parts of the brain, and from the lung, kidney, liver, spleen + heart were preserved in formalin.

The sets were taken in duplicate - a set of both cases being sent to the Central laboratory, Basra, from which I was favoured with a report in due course; and the other 2 sets I brought with me from Mesopotamia to Edinburgh, where Professor Lorain Smith kindly allowed me to cut, and examine, sections at the University.

The chief points that have a bearing on heatstroke, and are worth mentioning, are the following -

### Liver (Boland)

marked degree of congestion, especially in the

Central zone, where the cells are somewhat atrophied. Hepatic cells at peripheral zone are swollen & granular.

Section treated with Ferrocyanide and Hydrochloric acid shows the liver cells to contain a large amount of haemosiderin pigment, giving the prussian blue reaction. This pigment is situated in cells, more especially at the periphery of the lobule. There is also a large amount of pigment in the cells of the central zone, which has given a negative iron reaction, and is, in all probability, haemofuscin, or leptochrome pigment.

There is also a moderate degree of coarse, black-brown granules situated in a few of the endothelial cells of the sinuses, also in the connective tissue & lymphatics in the portal tracts. This pigment is probably malarial.

No parasites are seen in the circulation, but the evidence of haemosiderin suggests haemolysis, and haemozoin pigment confirms protozoal infection.

Major Stevenson, I.M.S. Central laboratory, Barrack, in commenting on the duplicate section of liver from this same case, says -

"Passive venous congestion, note that the pigment is of 2 varieties, one of blood derivation in liver cells - the other in endothelial cells, blacker, & more scarce. This, in the opinion of Major Christophers" (in charge central laboratory) "is undoubtedly malarial in origin, but he states that

death was not due to malaria, as, otherwise, the pigment would be much more abundant, and the endothelium would be swollen."

### Rolandic Area of brain (Brewster)

#### Haematin + Eosin

The vessels are in a state of acute hyperaemia, a few of the larger arteries showing evidence of haemorrhage into perivascular space, but no haemorrhages seen in brain tissue.

No characteristic pathological changes can be made out, either in nerve cells, or neuroglial tissue.

#### Toluidin blue

Shows marked absence of Nissl granules in nerve cells, several have an atrophied appearance, and a few show evidence of vacuolation. Many are hyaline, and stain a light, uniform tint, while others show a very fine basic granulation throughout their protoplasm. There are, however, extremely few which show the characteristic chromatin staining granules of Nissl.

Sections from the pons, under the different stains, show similar changes to the above.

In both sections there is a fair amount of chromatin-staining fine granules accumulating in the perivascular lymph spaces. The nature of these granules is doubtful, but many give the same blue staining reaction as seen in the protoplasm of ganglion nerve cells.

## Summary of Etiology and pathology

To sum up the foregoing remarks - The whole trend of my experience in Mesopotamia tends to show that great atmospheric heat causes a suppression of sweating, probably by exhaustion of the sweating mechanism.

This exhaustion is probably confined to the secreting powers of the sweat glands themselves, and may, possibly, be due to overwork of these during a prolonged period.

With sweating suppressed the body has lost its chief natural means of parting with heat, and tends, in accordance with physical laws, to adjust itself to the temperature of the atmosphere, and even to rise higher, for a rise of bodily temperature causes increased respiratory & metabolic changes (as shown by Sutton<sup>21</sup> & by Graham & Poulton<sup>22</sup>) with consequent production of further internal heat.

When the body temperature rises to about the region of 108° or more, unconsciousness takes place, due to the physical effect of heated blood on the highly specialised cells of the brain, a similar action on the motor cells causing the spasms & convulsions by irritation of them.

If the case be treated early, these effects will disappear as the temperature is reduced, but if the heat be allowed to act for a time, more or less lasting damage is produced, causing death in extreme cases, or, perhaps, a permanent disability (e.g. paralysis of various

kinds, or mental deterioration\*) if the extreme has been just avoided.

The conditions under which these observations were made gave a rare opportunity of studying the nature of the disease, throughout the whole of its course, in the human subject, and the success of the preventive measures confirms many of the points mentioned.

There can be no doubt that Haldane's experiments would have ended in heatstroke too, if carried far enough; so that it is possible, also, for heatstroke to be caused by insufficient evaporation from the skin, (in hot moist atmospheres) as well as by lack of it.

\* Dr R.M. Stewart gives a good instance of this. See Annotations Lancet 26/10/18. Page 561.

# 1918 METEOROLOGICAL FIGURES BASRA 1918

	JUNE			JULY			AUGUST			SEPTEMBER		
	MAX.	MIN.	Max.W.B.	MAX.	MIN.	Max.W.B.	MAX.	MIN.	Max.W.B.	MAX.	MIN.	Max.W.B.
1	95.3	78.4	73.6	112.6	81	85.6	108.1	77.4	85.8	104.4	72.6	76.8
2	98.1	76.6	77	116*	82.4	85.4	109.1	80.8	84.2	102.1	69.6	76.4
3	91.3	77.7	72.5	111.6	84.5	87	108.4	80.1	89	104.1	71.6	74.8
4	90.8	75.8	68.9	104.1	81.6	79.8	112.1	85	85.6	105.7	73	76.4
5	91.9	68.8	71.4	106.1	80.1	79.6	107.2	79.6	86	103.1	73.4	75.4
6	95.9	76	73	104.1	78.6	77.4	108.1	77.4	83.8	103.1	73.5	77.4
7	102.1	72.8	77.8	102.1	75	76.4	109.2	77.6	85.8	103	71.6	78.8
8	103	74.6	78.2	102.1	75	77.8	111	79.6	86.2	105.5	71.6	77.8
9	98.8	78.6	76.6	103.1	75.6	78	108.1*	76.6	82	106.5	72.6	75.8
10	99.1	72.4	76.3	99.9	74	76	108.6	76.6	78.8	103.2	72.6	79.8
11	93	78.4	71.8	101.9	79.4	73.3	105.9	75.6	82.6	105.7	68.6	78.8
12	93.6	77	72.6	103	72.6	79	113.6*	76.4	84.4	108.7	73.4	79.6
13	94.2	74.6	72.2	103.5	74.9	78.8	109	76.2	82.3	110.1	74	81.8
14	100	69.7	75.3	110.5	74.2	81	110.1	79.6	81.6	108.1	75.6	79.8
15	99.9	70.3	79	110.1	78.4	82	107.6	84.2	78.4	109.6	74.6	81
16	105.3	71.8	82.2	107.1	78	80.8	99.3	80.4	73	109.7	77.6	82
17	102.3	77.6	79	112.1	76.4	83.4	103.1	76.4	80.8	110	74.4	83.3
18	100.8	72.6	77.6	111.5	74.6	85	106.1	76.6	82.4	106.5	72.6	78.8
19	104	74.1	79.4	111.7	80.6	88	108.7	81.6	79.8	104.3	72.6	81
20	110	75.6	79.1	106.3	87.6	80.3	104.1	76	77.6	108.1	73	81.3
21	110	79.6	79.7	102.7	74.6	77.1	105.1	79.6	74	108.9	71.6	78.2
22	109.9	74.8	82.8	107.9	82.6	83.2	103.5	76.4	77.8	105.5	72.6	79.8
23	107.6	73.5	82.6	109.1	83.1	82.8	92.9	72.6	70.8	103.2	70.4	79.8
24	106.1	82.8	80.4	109.1	75.6	81.8	98.1	68.1	73.2	108.1	75	79
25	97.9	82.2	76.3	109.7	77.6	83.2	99.1	72.6	70.8	104.2*	76.6	81.8
26	103.2	74.7	80	109.4	79.6	88.4	102.9	68.6	77	100.7	75	73.8
27	103	73.7	82.3	105.3	77	82	103.3	70.8	77	102.1	75.6	76.8
28	107.6	77.4	82.4	106.5	77.6	85.8	103	70.8	74.6	102.9	70.6	77.8
29	107.5	77.8	79.6	108.1	79	83.8	104.9	72.6	75.8	104.9	70.6	79.8
30	108.9	77.4	86.8	107.3	76.6	85.4	103.7	71	76.8	102	71.6	78
31				109.6	76	85	101.1	72.2	77			

## MONTHLY SUMMARY 1918

	JUNE	JULY	AUGUST	SEPTEMBER
AVERAGE MAXIMUM TEMPT <sup>RE</sup>	100.6	107.2	105.9	105.4
HIGHEST MAXIMUM TEMP.	110	116	113.6	110.1
LOWEST MAXIMUM TEMP.	90.8	99.9	92.9	100.7
AVERAGE MINIMUM TEMP.	75.6	78.1	76.4	72.9
HIGHEST MINIMUM TEMP.	82.8	87.6	85	77.6
LOWEST MINIMUM TEMP.	68.8	72.6	68.1	68.6
AVERAGE DAILY WIND MOVEMENT	272.6 MILES	200.6 MILES	245 MILES	155 MILES
MAXIMUM DAILY WIND MOVEMENT	467 MILES	413 MILES	497 MILES	296 MILES
TOTAL RAINFALL	—	—	—	—
GREATEST DAILY RAINFALL AMOUNT	—	—	—	—

\* ON EACH OF THESE DAYS ONE HEATSTROKE OCCURRED AT N°3 B.G.H.

# MESOPOTAMIA

Condensed from annexure to General Routine Order N°305 dated 5/4/17

WEATHER AVERAGES FOR PAST 13 YEARS BASRA					
	MEAN DAILY MAXIMUM SHADE TEMP. DURING MONTH	MEAN DAILY MINIMUM SHADE TEMP. DURING MONTH	HIGHEST INDIVIDUAL MAXIMUM SHADE TEMP. REGISTERED IN 13 YEARS	LOWEST INDIVIDUAL MINIMUM SHADE TEMP. REGISTERED IN 13 YEARS	MEAN RELATIVE HUMIDITY AT 8 AM IN PAST 12 YEARS
JUNE	100.6	80.3	116.0	70.3	58
JULY	103.1	81.4	114.4	65.7	59
AUGUST	104.4	80.2	120.5	68.7	59
SEPTEMBER	100.2	74.9	115.0	58.0	62
WEATHER AVERAGES FOR PAST 27 YEARS BAGHDAD					
JUNE	104.4	75.9	119.2	62.8	38
JULY	109.2	79.7	120.2	69.6	39
AUGUST	109.9	79.0	121.0	68.9	42
SEPTEMBER	103.3	72.4	117.2	56.0	44

Babylon.—2. *Temperature.*

	Extremes recorded.		Normal monthly extremes.	
	High.	Low.	Max.	Min.
January ... ..	a. 297	a. 267	a. 294	a. 270
February ... ..	301	270	298	272
March ... ..	308	274	304	277
April ... ..	314	278	311	281
May ... ..	319	287	316	288
June ... ..	322	289	319	291
July ... ..	322	289	320	294
August ... ..	323	290	320	293
September ... ..	320	287	319	289
October ... ..	313	281	312	283
November ... ..	306	270	303	275
December ... ..	300	266	296	270
The Year ... ..	323	266	320	270
Period ... ..	5½ years, June, 1907–December, 1912.			

Babylon. 4.—*Humidity.*

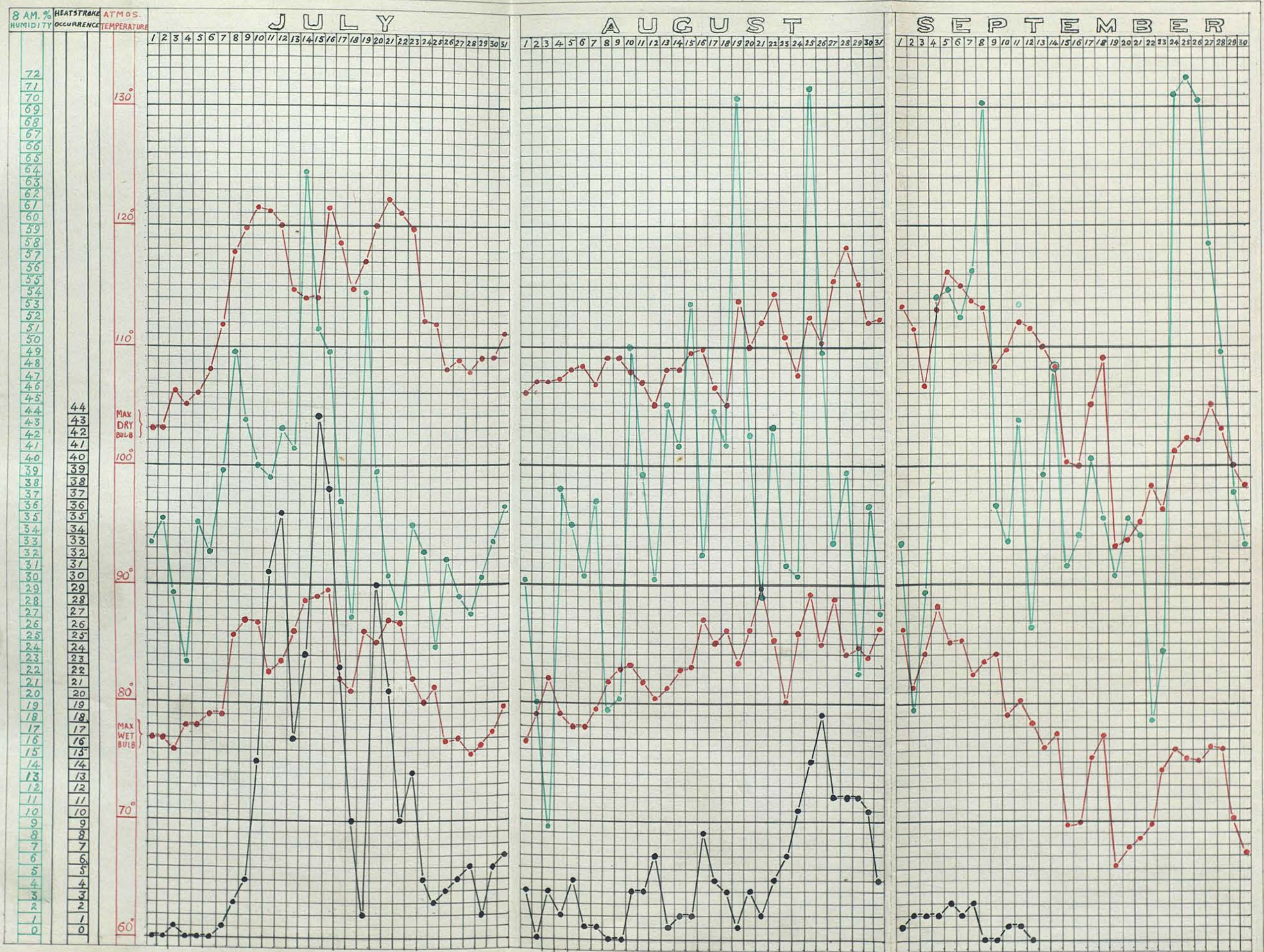
	Normal Vapour-Pressure.			Normal Relative Humidity.		
	At 7h.	At 14h.	At 20·5h.	At 7h.	At 14h.	At 20·5h.
January ...	mb 6·6	mb 7·4	mb 7·4	% 84	% 49	% 69
February ...	7·1	7·2	7·3	73	40	53
March ...	9·9	8·0	8·2	66	30	44
April ...	11·5	9·6	10·3	58	27	40
May ...	13·9	12·2	12·5	47	22	34
June ...	12·9	10·9	12·0	36	17	26
July ...	13·0	10·8	12·8	33	13	25
August ...	11·8	10·5	11·2	32	13	22
September ...	10·7	9·5	10·0	38	13	23
October ...	9·9	9·9	9·8	49	21	34
November ...	8·6	9·4	9·6	68	34	50
December ...	7·6	8·7	8·5	84	48	68
The Year ...	10·3	9·5	10·0	56	27	41
Period ...	5½ years June 1907–December 1912.					

# INDIA

## MONTHLY AVERAGES

MONTH	MULTAN			PESHAWAR			JHANSI			CALCUTTA			BOMBAY		
	MAX	MIN	% HUMIDITY	MAX	MIN	% HUMIDITY	MAX	MIN	% HUMIDITY	MAX	MIN	% HUMIDITY	MAX	MIN	% HUMIDITY
JANUARY	69.7	43.3	71	63	39.6	72	75.9	51.5	65	77.3	55.5	85	82.5	67.9	72
FEBRUARY	73.6	47.3	65	65.7	42.5	72	80.7	55.6	56	82	60	81	82.8	68.4	71
MARCH	85.8	58.2	58	75	51.7	71	92.8	66.3	43	90.9	69.3	80	85.7	73.2	74
APRIL	97.9	68.4	47	85.5	60.3	61	103.3	76.9	35	95.6	75.7	78	88.6	77.5	75
MAY	106.9	78.1	42	98.1	69.9	43	108.4	84.4	38	94.5	77.5	79	90.8	80.7	74
JUNE	108.3	84.2	50	105.9	77.3	41	103.8	85	56	91.5	78.8	85	88	79.7	82
JULY	104.3	84.3	63	102.7	79.2	61	91.9	79.3	79	88.4	78.6	88	84.6	77.7	86
AUGUST	101.2	82.6	68	99.2	78	70	89	77.4	83	87.6	78.4	89	84.2	77.1	87
SEPTEMBER	100.5	77.5	65	95.6	70.5	64	91.5	76.1	77	88	78	87	84.7	76.7	87
OCTOBER	95.9	65.1	56	88.2	57.9	59	93.1	69.8	56	87.2	74.3	84	87.8	76.7	82
NOVEMBER	84.8	53.5	60	77.3	46.1	66	85.8	59.4	52	82	64.3	82	87.2	73.5	73
DECEMBER	73.5	45.0	69	67	39.2	70	78.3	52.4	59	77	56	81	84.7	70	72

# 1917. BASRA - HEATSTROKES & METEOROLOGICAL



Prevention

## Premontory Subjective Symptoms.

In some cases the patient does not regard any uncomfortable feelings that he may have previous to attack as being of any special importance; he merely attributes his discomfort to the exceptional weather, which he believes should be borne by himself with the same resignation as by his fellow creatures around him. He will, consequently, go about his duties, perhaps in a hot cookhouse, without considering it at all necessary to seek medical advice, till he suddenly falls to the ground unconscious and perhaps in convulsions.

Others, again, having felt distinctly "seedy" for an hour or so owing to headache, nausea, disinclination for exertion, drowsiness, etc, will heed these warnings and report sick. The more observant of them will perhaps mention that for some variable time past they have been passing water frequently, and have also stopped sweating\*. Intense thirst is sometimes complained of.

Still others may be so overcome by a sense of drowsiness that they feel inclined to do nothing but lie down and rest, believing, no doubt, that a couple of hours thus spent will improve their condition. Sound sleep almost immediately overtakes them, and, after a short interval, during which the bodily temperature has been rapidly rising, they

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\* Curiously enough this symptom rarely forms one of the patient's own reasons for reporting sick.

pass suddenly into the condition of heatstroke, and, unless spasms, or the rhythmical groaning which is so typical of the attack draws attention to their condition, they are apt to lie undiscovered until they reach the moribund state, or perhaps may be found dead.

With the onset of the attack, however, they may suddenly leap up and run about excitedly for a few minutes, (usually colliding with obstacles in their path) and draw attention to themselves in this manner.

Intolerance of light and chromatopsia have been mentioned as premonitory symptoms, but, in my experience, (mainly cases produced while under shelter) intolerance of light is rare, and chromatopsia, I have never met with at all.

Particular attention should be drawn to the fact that the patients own sensations form by no means an infallible guide as to the immediate danger of his condition.

Gauss and Meyer<sup>15</sup>, in their report of 158 cases at Chicago, cite several instances of this, chief of which, perhaps, is that of a teamster, who left his wagon to get a drink of water; he felt perfectly well when he left his seat, but, as he stooped to drink from the fountain he felt himself slipping, and remembered nothing further until he awoke in the hospital; his temperature on admission was 110°.

Others, however, had distinct premonitory symptoms,

and fell in the street while on their way to seek help.

In Pembrey's<sup>27</sup> analysis of 50 cases, reports show that in 20 of them the person was unwell before the attack.

Among cases of heatstroke that occurred during my experience on ships, despite the clearly worded warnings that were published among the troops, I would always ask the patients, on recovery, to explain why they did not report sick in time; various answers were given, the commonest being a statement to the effect that, although they had noticed the skin dry they felt all right at the time, and, consequently, thought there was no need for great hurry.

The diagnosis of oncoming heatstroke.

The most reliable information is supplied by the state of the skin; this is usually flushed, absolutely dry, and typically harsh to the touch; it is more or less hot, according to the degree of temperature present, and in a far advanced case, burning hot.

There is generally a history of frequency of micturition, but in cases of sudden suppression of perspiration this may not have time to develop before the heat of an intensely hot day brings on the fully developed attack of heatstroke.

The presence of these signs, together with a considerably raised body temperature, on a day when heatstroke is likely, form the indications for preventive treatment.

Prevention of Heatstroke

The outline of this simple method of prevention has already been referred to on pages <sup>29 & 30 and</sup> <sub>footnote P.32,</sub> This has been necessary since the results of prevention had to be called in evidence against other etiological theories when dealing with that subject in the preceding section (see pages 34 & 35)

Though involving some repetition, it might here be stated that heatstroke gives ample warning of its onset (anything from 1 to 48 hours) the chief signs being absolute suppression of sweating, rise of temperature and (usually) frequency of micturition, and by the timely application of some simple means of artificially imitating sweating the attack can be entirely prevented.

Under ideal conditions, such as in hospital, this can be done by covering the naked patient with a wet sheet as he lies in bed, and the effect of an electric fan directing a current of air over the wet sheet may be added where necessary.

Some of the incidents which led to the adoption of these preventive measures will now be stated, and further points which have a bearing on prevention will then be discussed.

On entering my hut one morning, about the first day of the July heatwave in 1917,

My attention was drawn to the report of the night sister, who mentioned that a certain patient had been getting up about every hour during the night to pass water. This fact did not strike me at the time as being of any very urgent importance, so I commenced the "round" in the usual way, and, on eventually coming to this patient's bed, he told me about the frequency of micturition, and talked in quite a cheery fashion for about a minute or so, when he suddenly sat up in bed, and passed into a state of wild delirium, shouting and fighting like a maniac.

The typical attack of heatstroke was immediately treated with ice, and, on his recovery to full consciousness, he mentioned that since 5 o'clock the previous evening he had been unable to sweat.

Having no personal experience of the disease at this time, and being obsessed with ideas of accumulated and retained toxins, I gave diaphoretics to all patients with suppressed sweating, and, as these were useless, I increased the doses, but still no action from the diaphoretics took place, and heatstrokes continued, events reaching a climax by the occurrence of 3 different cases within 2 or 3 minutes of one another on one particularly hot afternoon, about the 4<sup>th</sup> day of the heatwave.

To make matters worse there were 6 or 8 other patients whose suppression of perspiration

and steadily rising temperature indicated that they were liable to develop the condition at almost any moment.

Some convalescent patients willingly assisted, and at my instructions, splashed water over those who were threatened with attack as they lay in bed. This temporary measure was continued till I was able to procure from a neighbouring electric power station a considerable length of flexible wire, and, in about an hour from the outbreak of the 3 heatstrokes, 2 portable electric fans (one lent by the padre and the other from my own private quarters) were connected up in some sort of fashion to the electric supply of the hut. These were applied to the worst cases, and through this means further outbreaks were prevented on that afternoon.

On the next day the plan of hourly inspections and application of wet sheets, as described on page 29, was put into operation, the fans being made adaptable to any of the electric light fittings so that they could be quickly and conveniently moved to any bed where required.

A little experience, however, showed that the fans were not often required, wet sheets and the overhead punkhas being sufficient.

As already stated, the adoption of this method of prevention kept the whole trouble completely in hand.

The inspection was done hourly, of course, only on the hottest days, and after the system was thoroughly established the times of inspection were left entirely at the discretion of the nursing staff. Their watchfulness, however, was excellent, and, considering the pressure of work with which they had to deal at the time, the results reflect great credit on the manner in which their duties were performed.

In my hut, where, on some hot days, as many as 10 to 15 patients would require preventive measures, I used, for keeping the sheets wet, a Holder-Harriman pneumatic disinfecting spray (See photograph page 74) This, of course, was filled with Cold water, and by its use an enormous amount of time and trouble was saved; the sheet was sprayed in position without the necessity of uncovering the patient, and only the required amount of water was used, so that the mattress of the bed did not become soaked.

A convenient apparatus of this kind is of considerable importance, for it often means the difference between efficient and absolutely useless treatment. Where many patients are concerned the objections to pulling a sheet off a naked patient, (in a public ward) dipping it in a bucket of water, wringing it out, and reapplying it, are quite obvious, and the bed is

eventually, as a rule, soaked. Rubber sheets to protect the bed are not permissible because they retain an enormous amount of heat, and, worse still, a pool of water, which is decidedly warmer, if not hot, collects under the patient.

The pneumatic spray is by far the best idea; its advantages are many & obvious; one of these would probably be obtainable for this purpose in most hospitals in India, and a "bhisti" could, no doubt, be spared for the special duty of going from ward to ward spraying these patients during a severe heatwave.

So long as the atmospheric temperature remains excessively high, a patient with suppressed sweating will require some such means of protection as already described, but in mild cases, a convenient modification of the method; and one which is more comfortable for the patient, is to raise the wet sheet on 2 bed cradles, thus forming a kind of Cool Chamber in which the patient lies naked; the temperature inside (usually between 80° and 90° F) varies, of course, with the atmospheric temperature, but by playing the air current from an electric fan on the outside surface its effect can be intensified if necessary. (see photograph, page 74.)

In the hot hospital huts it was a common occurrence for a patient to ask for the roof punkha above him to be switched off, his argument being that hot currents of air were driven down from immediately below the iron roof.

On examining the patient, however, it was found that he had, either completely, or nearly, stopped sweating, and, later on, a request of this nature came to be regarded as a sign that the patient required watching. Those who were sweating freely strongly objected to the punkhas being stopped.

The question was investigated, in the first place, with an ordinary atmospheric thermometer, and, although slight but erratic variations were produced occasionally, there was no evidence that the punkha caused any appreciable increase in the temperature of the patients surroundings.

The point, however, was studied later by the aid of the Ratathermometer - an instrument devised by Professor Leonard Hill for the purpose of measuring the cooling power (or in very hot atmospheres the warming power) of the atmosphere, on a surface (1) dry, (2) wet at body temperature, in mille Calories, per Sq. Centimetre, per second. (mille Calorie =  $\frac{1}{1000}$  small Calorie)

The results brought to light some very interesting facts, and several of these experiments are here quoted.

		Dry bulb	Wet bulb	Wet Kathethermometer	Dry Kathethermometer
				Heat loss	Heat gain
Experiment 19	Punkhas turned off	109.2	83.1	6.37	2.17
Experiment 20	— — on	108	82.4	15.16	5.1
Experiment 27	Punkhas turned off	109	80.6	10.58	3.03
Experiment 28	— — on	109	78.9	20.03	5.96

These experiments showed that, in a patient who was sweating, the current of air from the punkha considerably increased his rate of heat loss, but in a patient with suppressed sweating the opposite effect took place, a corresponding gain of heat by convection being registered.

The punkha is, therefore, a safeguard against heatstroke in the one case, and a dangerous means of hastening its onset in the other.

A patient's temperature, after suppression has set in, can mount up with remarkable rapidity, an occasional case giving no cause for anxiety at one inspection, yet being perilously near to heatstroke at the next; this was my reason for making the inspections hourly on the hottest days. It was not, however, till 1918 that I obtained the instruments which enabled these important effects of the punkha to be actually measured, and it will be readily seen that the gain of heat by convection can account for the rapid changes in bodily temperature observed in the patient.

The obvious remedy, of course, is not to switch the punkha off, but to put a wet sheet over the patient.

The inspections may at first sight appear cumbersome, but, as a matter of fact they occupy very little time, and in a 60 bed hut can be easily carried out in less than 10 minutes. All that is necessary is to estimate roughly (by touching the skin) as to whether a patient has stopped sweating, and has any pyrexia; doubtful cases are noted for an accurate thermometer reading to be taken.

So far, prevention in hospitals only has been dealt with, but the plan can be easily carried out under almost any conditions, even among troops on the march, while soldiers sleeping in barracks or in tented camps during hot afternoons can be periodically inspected by an intelligent orderly or staff of orderlies.

On specially hot days clearly-worded orders issued by the medical officer and stating the chief signs of onset will enable most cases to be detected by the troops voluntarily reporting sick, but, where the attack threatens during sleep, the inspections are of course necessary.

Even under the most unfavourable conditions that are likely to be met with, the essentials for preventive treatment (water + something that will act as a fan) are practically always obtainable, and, by the early application of these measures to a threatening case, much can be done to tide the man over till it is possible to remove him to hospital.



*Wet Sheet over Cradles.*



*Holder-Harrinan Spray*



*Summer Clothing of Troops.*

Treatment

## Treatment of the fully developed attack

The treatment will be described as carried out in a well appointed heatstroke room under ideal conditions such as are to be found in a big general hospital.

### The heatstroke room

On page 89 will be found a rough plan of the heatstroke room and after-treatment ward of N° 3 B.G.H. Basra, where the reduction of hyperpyrexia was carried out by means of cold-water douches and electric fans.

Three iron bedsteads, of special pattern\*, stood on a waterproof cement floor; at the head of each bedstead, and slightly to one side, was a powerful electric fan, arranged in the most suitable position for directing a strong current of air on the patient while undergoing treatment.

From a tank, situated in another room at a higher level, a pipe conveying cold water at about 50° F. was led into the heatstroke room; this pipe was insulated from the heat of the surrounding atmosphere by a thick layer of non-conducting material, and distributed the water to the 3 bedsteads by means of hoses, each ending in a finely perforated "rose"; and controlled by a tap.

The other furnishings of the room consisted

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\* In these bedsteads, the part on which the patient lay consisted of crossed & interlaced steel lathes forming a rigid meshwork with about 1 inch meshes; thus no heated water collected under the patient, and free access of air was allowed - conditions which greatly assist the rapid escape of heat from the body.

of a table with all the necessary equipment used in treatment all ready for immediate use (e.g. Chloroform apparatus, surgical instruments for venesection, lumbar puncture, & infusion of various solutions, hypodermic syringe, slides for blood films etc) a writing table, an ice chest, and a Cylinder of Compressed oxygen.

The room was well lighted so that the thermometers could be easily & accurately read, and, in addition to the fans mentioned, 2 electric punkas hung from the ceiling.

The 200 gallon Cooling tank was surrounded with non-conducting material; water was supplied direct from the main by a pipe controlled by an automatic ball-valve, and ice was added to the water as required.

#### The after-treatment ward

This ward, adjacent to the heatstroke room was the coolest ward in the building, and contained 22 beds. The walls were fitted with plugs, arranged at convenient distances, for connecting up electric fans to any bed where the patient required treatment for threatened relapse of hyperpyrexia.

Electric punkas, hung from the ceiling, kept the atmosphere of the ward more or less in motion.

#### Reduction of hyperpyrexia

The chief point to keep in mind is that the longer a heatstroke patient remains in a state of hyperpyrexia the poorer become his chances of recovery; there is no time to lose,

and every minute is valuable.

The patient on arrival in the heatstroke room is placed on one of the treatment beds; his clothes, if not already removed by those who have rendered first-aid, are rapidly cut away.

The douche and fan are immediately turned on the patient, while, from the earliest possible moment, an orderly, specially detailed for the purpose, takes and records the rectal temperature, continuing to do so as often as possible (allowing time for accurate readings) throughout the reduction of hyperpyrexia. Meanwhile the patient is alternately turned on his face and back so that the whole surface of his body, from head to foot, comes under the action of the douche & fan, and the excessive heat is extracted as rapidly as possible, until the temperature is reported to be 104°, massage of the limbs being a useful additional measure.

At this stage I generally switch off the fan and continue with the douche for a little longer until a temperature of 103° is reached, but very seldom lower, and in no case is the patient allowed to go below 102°.

The finishing point is very important, as it generally decides whether the patient will afterwards collapse or relapse into the hyperpyrexial state; if the pulse is good it is usually safe to let him go a little below 103°.

When the patient, on admission, is in spasms

or convulsions, just sufficient Chloroform is administered to keep him quiet, but the congested state of the lungs should be borne in mind.

When reduction of hyperpyrexia is finished the patient is quickly wrapped in a dry sheet, and put to bed in the after-treatment ward, covered only by a dry sheet\*.

The practice of applying hot bottles and blankets, and giving hot stimulatory drinks, as an immediate routine measure, with the object of promoting sweating, is liable to cause relapse requiring perhaps a second visit to the heatstroke room, after which the prognosis will not be so favourable.

The patient requires careful observation and nursing for the first hour or two, and everything should be in readiness to meet, at the earliest moment, any extremes to which his condition may tend.

If treatment has been applied soon after the commencement of the attack, and the reduction of hyperpyrexia carefully carried out there is usually very little trouble, the commonest occurrence being a fall of the temperature to about normal within the first 15 minutes of after-treatment; this is generally succeeded by a rise, within the next hour or so, to about  $101^{\circ}$  or  $102^{\circ}$ , and, unless the atmospheric temperature of the ward is

---

\* Should the after-treatment ward be an artificially cooled room in a city hospital, due allowance must, of course, be made for this, and one or two blankets may be required.

very high, the patients temperature, in such early treated cases, does not frequently tend to rise to a dangerous degree.

The 2 points, however, that one must carefully watch for at this stage (especially in cases where treatment has been delayed) are, 1. tendency to collapse. 2. tendency to recurrence of hyperpyrexia.

For the first hour or so of after-treatment the patients rectal temperature should be taken at intervals of no longer than 10 minutes, and his pulse and general condition frequently observed.

Tendency to Collapse

This must be met on general medical lines. The foot of the bed is raised: The patient, lying between blankets, has hot bottles applied, and is given a subcutaneous injection of digitalin ( $\frac{1}{100}$  grain). Strychnine is said to be contra indicated on account of the liability to induce convulsions; brandy, however, is permissible in the collapse stage.

In severe cases, intravenous injection may be called for, and, on account of the pronounced congestion and oedema of the lungs, brain & other organs, seen at autopsy, of heatstroke cases, it would seem advisable to use the gum arabic solution recommended by Bayliss<sup>20</sup>, and, although I have had no occasion to use it in cases of heatstroke, this solution would appear to be distinctly indicated in selected instances.

## Tendency to recurrence of hyperpyrexia.

Should a relapse of hyperpyrexia tend to occur it is best to raise the sheet on two bed cradles, spray it with cold water, and direct the current from an electric fan on its outer surface, as described under preventive treatment page 70 (See also photograph page 74)

It must be remembered that the patient is not sweating\*, and requires to be protected from the temperature of the ward, which may be  $106^{\circ}$  at the time. In any case, the patient's heat regulating mechanism is disorganised, and any excessive internal heat production cannot be balanced by the natural sweating process.

It seldom happens that this measure is insufficient to check the rising temperature, but its effect can be intensified, if necessary, by removing the cradles and allowing the sheet to come in contact with the patient; this, however, should not be continued for long, but is decidedly preferable to allowing a recurrence of severe hyperpyrexia.

## Spasms and Convulsions

During reduction of hyperpyrexia these are controlled, as already stated, with chloroform. Should they return, however, in the after-treatment ward when the effect of the chloroform has vanished, I generally give a subcutaneous injection

\* It has been stated (following on instructions to put the patient to bed with hot bottles & blankets immediately after reduction of hyperpyrexia) that "very likely, perspiration, a very favourable sign, will then set in". This in my experience is rare. On the other hand, cases sometimes go a fortnight after attack without sweating.

of morphine ( $\frac{1}{4}$  grain) + atropin ( $\frac{1}{100}$  grain) provided, of course, that the state of the pulse shows no contra indication.

This usually quiets the spasms and ensures for the patient a restful sleep which greatly assists his recovery.

Malaria:

At the earliest convenient opportunity after admission, one or two blood films should be made in every case, and sent to the laboratory with a request for immediate examination as to the presence of malarial parasites, or spirillum of relapsing fever.

There are obvious objections to the practice of giving routine intramuscular injections of quinine as recommended by some, and I always preferred to wait for the laboratory report, which was usually to hand about 20 minutes after the despatch of the slides.

In cases of benign tertian, an intramuscular injection of 9 grains quinine was given, while if the malignant form were reported, the same quantity of quinine was administered intravenously in dilute solution, with  $\frac{1}{100}$  grain digitalin added, the fluid being introduced by slow gravitation.

Cases of Cerebral malaria <sup>considerable difficulty</sup> may present <sup>as</sup> this condition may exist either alone, or as a complication of heatstroke. A careful enquiry into the previous medical history of the patient (from friends or official medical records) and the presence of numerous parasites in the blood film, together with palpation of the spleen,

will indicate the correct line of treatment

Cerebro - Spinal meningitis.

The clinical behaviour of some cases of heatstroke bears a strong resemblance to this condition, the spasms sometimes closely simulating head retraction. The possibility of this as a complication should always be eliminated by an enquiry into the history of onset and an examination for Kernig sign, strabismus etc. The evidence from lumbar puncture is, of course, the most valuable, and, as this is sometimes employed as a therapeutic measure in cases of pure heatstroke, there need be no hesitation in carrying it out where further light on the diagnosis is desired.

Threatened failure of respiration.

Prolonged artificial respiration may be required, and this can be assisted by the inhalation of oxygen. Serious respiratory embarrassment is, however, seldom met with in cases that have received early treatment.

Adjuvant methods of treatment.

Emmata of ice water, in accordance with the original suggestion by Parkes, have been frequently used to reduce hyperpyrexia. There is, however, a very strong objection to them on the ground that they destroy the only means of obtaining the reliable body temperature, which, in my opinion, is the most important guide to the successful treatment of the attack, and without this information it is difficult to see how one can proceed on rational lines.

I only use enemata after considerable trial of external treatment, and as a last resource in cases such as that of a very heavy man, with a thick non-conducting layer of fat on his body, and whose sluggish skin circulation indicates the improbability of heat being quickly lost by convection to the surface in the blood stream.

With external treatment by cold water and fans in a dry climate coldwater enemata should seldom be called for.

Foulds<sup>19</sup> speaks highly of the results of ice water enemata, but in his cases the chief difficulty appeared to be the shortage of ice for external treatment; cold water, therefore, had to be used in the most economical way.

With the object of bringing the application of cold into closer relation with the heated blood contained in the large venous trunks, and also in the hope of avoiding the disadvantage that attends the ice water enema, I once washed out a patient's stomach with cold water. This would appear to be a very powerful measure, and should be used with great caution, if at all; it is certainly not recommended.

### Venesection.

Under the assumption that the condition is purely a toxic one, venesection and intravenous injections commonly figure in routine treatment. It has been stated<sup>30</sup> that "These measures are often resorted to too late. As the state is undoubtedly one of toxæmia it requires prompt and energetic measures to

Combat it."

This subject has already been considered in the discussion on etiology, page 48, and my use of this method of treatment is practically confined to plethoric cases where cyanosis and congestion are present, the object being to mechanically relieve the engorged venous system.

To adopt it as a routine practice, is, in my opinion, decidedly wrong, and many cases would be distinctly harmed by it.

Intravenous injections

These are useful in cases of extreme collapse, and firm saline solution should be chosen owing to its more lasting effect.

Normal saline, given with a view to diluting toxins, is not indicated, and, owing to its permeability, would probably do harm to the already oedematous lung and other organs.

Sodium bicarbonate might be included in the firm saline and may possibly benefit a case where acid products of muscular fatigue are suspected of playing a complicating part.

Lumbar puncture.

This, as a therapeutic measure, may be employed in cases where long continued persistence of the Comatose state, signs of cerebral irritation, or violent headache exist.

where this procedure has been carried out the fluid was always found to be under moderate pressure, & its removal was followed by considerable relief of the symptoms. It is however seldom required.

Cold water + fans compared with ice for reduction of hyperpyrexia.

The former method, as used in the heatstroke room at No 3 B.G.H. has several important advantages over the latter. By it the reduction of hyperpyrexia is brought about much more speedily, owing to the fact that the douche + fan are acting on practically all parts of the exposed surface at once, whereas ice, which acts chiefly by conduction, affects only the part with which it is in contact.

The superiority of the evaporation method, as regards speed of action, is well illustrated by experiments with the Katalthermometer.

Direct ice massage, too, seems to have a severe local effect on the skin, constricting the capillaries and limiting the rate at which blood can reach the surface for cooling. In addition, this method involves a considerable and prolonged physical strain on the staff of assistants - a very important consideration during a severe outbreak - and which is entirely avoided by the evaporation procedure.

Provided the douches are finely perforated so as to avoid unnecessary wastage, the cold water system is much more economical. At No 3 B.G.H. the douches were 3 inches in diameter, and the fine needle-point perforations allowed a passage of water at the rate of 3 pints per minute; this is

ample when it is remembered that the object is to cool, and not to bathe the patient.

Before the introduction of the water system, when ice massage alone was used at No 3 B.G.H. the expenditure of ice worked out at 80 lbs per patient; The amount of ice required for cooling the water is far less than this.

Reduction of hyperpyrexia in small hospitals mobile hospitals Camps etc.

Although large general hospitals, in regions where heatstrokes are common, should all be provided with a heatstroke room fitted up somewhat on the lines of that described, this is obviously quite unnecessary in places where perhaps only 3 or 4 cases per season are dealt with.

On the other hand, unless everything is ready, for immediate treatment really good results cannot be expected, and the rarity of cases in a district does not, of course, justify lack of preparation to meet them.

In Camps (and possibly small hospitals) the chief obstacle would appear to be the difficulty in obtaining cold water through lack, or insufficiency of ice.

To obviate this, I would suggest the issue of 15 gallon canvas water bags, of standard type, and designed to present a large evaporative surface to the atmosphere. These should be made of specially selected

Canvas, whose texture allows the escape of just sufficient water to keep the surface damp, but not soaking wet, as, under these circumstances, the maximum cooling effect is produced on the contained water.

Each bag might be provided with a tap, with a canvas hose, ending in a finely perforated douche, attached.

The weight of such a bag, when full, would only be slightly over 150 lbs, and it could be suspended at a convenient height so as to be always ready for instant use.

a finely perforated douche, allowing 3 pints of water per minute to pass, would give a steady flow for 40 minutes, which would be ample for the treatment of one case, some form of fanning being of course employed as an additional aid in treatment.

Such an apparatus is suitable for transport in mobile units, and can be readily set up when heatstroke weather appears, and just as readily dismantled when it has passed.

By such an arrangement a supply of cool water is kept constantly in readiness for instant, convenient, and economical use, thus ensuring prompt and efficient treatment for the patient.

Sir Leonard Rogers states<sup>23</sup> ~~31~~ -

"In Calcutta, invaluable time has often been lost in bringing patients to hospital, instead of first applying cold on the spot

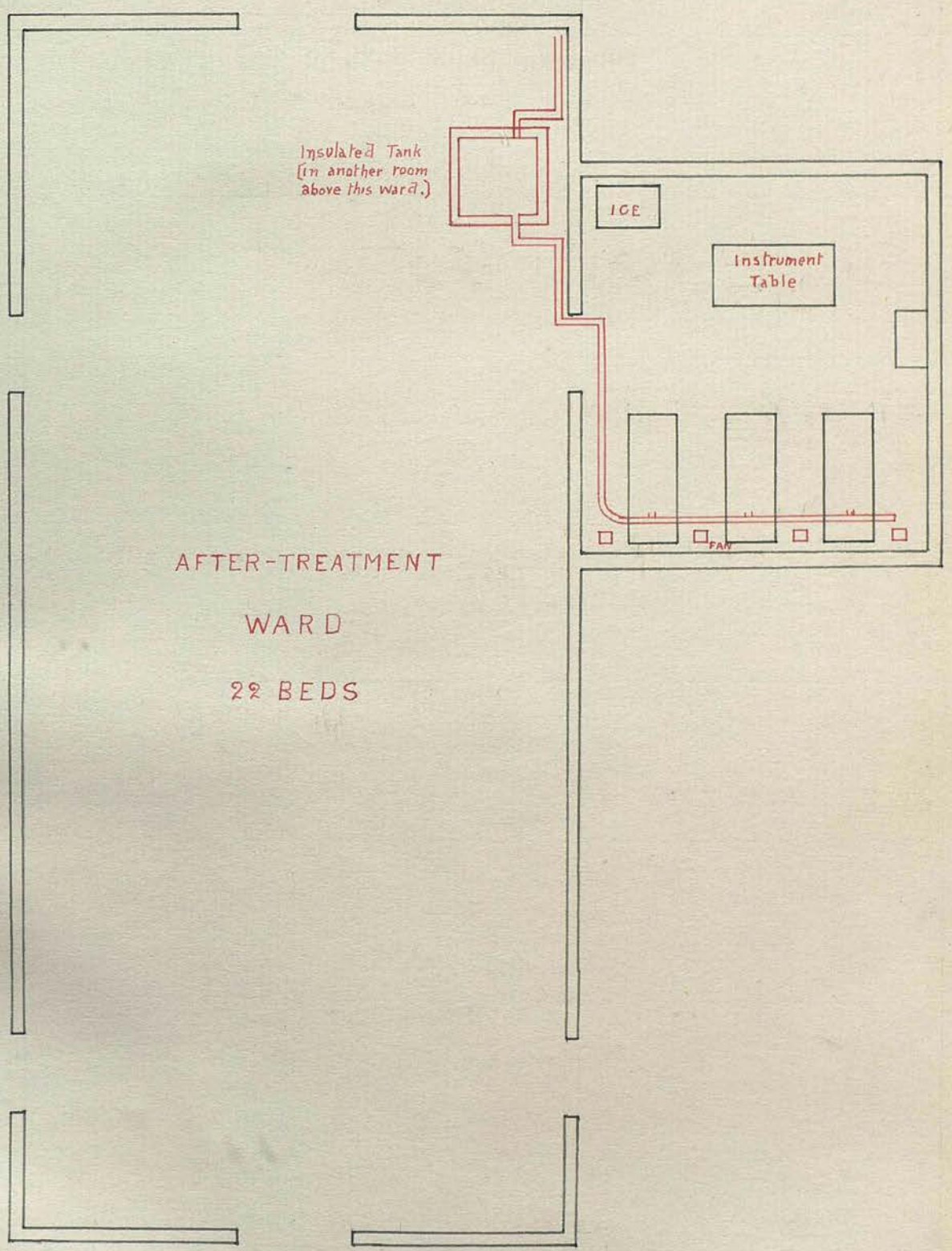
to reduce the temperature, with the result that they only too often come under treatment when in a hopeless condition.----- It is only by detecting, and treating cases early, that the death-rate from heatstroke can be greatly reduced."

### Evaporative treatment in humid climates

Although the more humid atmospheres in several parts of India may tend to reduce the efficiency of evaporative methods of treatment, I see no reason why this should occur to any great extent. They showed no tendency to fail in Mesopotamia, even on the most humid days.

Meteorological figures from selected parts of India, which have a bearing on this point, are to be found on page 61. Figures from these regions have been chosen, either ~~either~~ because heatstrokes are common, or humidities are great there.

Nº3 B.G.H. HEATSTROKE ROOM & AFTER-TREATMENT WARD



Insulated Tank  
(in another room  
above this ward.)

ICE

Instrument  
Table

AFTER-TREATMENT  
WARD  
22 BEDS

FAV

## Conclusions

The chief Conclusions arrived at are here very briefly stated.

Etiology - Heatstroke is practically always preceded by suppression of sweating, itself, probably, a result of exhaustion of the sweating mechanism.

As a result of suppression, bodily heat accumulates, the temperature rising higher & higher, till, by its action on the specialised brain cells, sudden unconsciousness, and convulsions are produced, which characterise the fully developed attack.

Prevention - Heatstroke gives ample warning of its onset, and is entirely preventable by some simple method of artificially imitating the physical effect of sweating - a power which the patient has temporarily lost, and may not regain for several days.

Treatment  
(of actual attacks) all cases should be treated at the earliest possible moment, and apparatus for this purpose must be always kept ready for instant use during heatstroke weather.

charts and a brief note on an interesting and severe case of heatstroke complicated with malaria are appended.

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\* See also

Baylis Proceedings Royal Society (London) B  
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"Methods of raising a low arterial  
pressure"

The following 2 Charts refer to a case of heatstroke, Combined with malaria M.T., and is selected on account of the number of interesting points which it shows.

The patient had only been in Mesopotamia 4 days when attacked with heatstroke in one of the hospital huts.

He left England on 4/7/18, travelling via Cherbourg, Taranto, Alexandria, + Bombay, (where he stayed at Colaba from noon 29/7/18 till 6.30 Am on 1/8/18, + probably caught his malarial infection there) arriving at Barra on 9/8/18.

The chart states most of the interesting details, but an explanatory note regarding the paralysis on 15/8/18 is necessary.

This, I believe to have been due to Cerebral malaria. There was complete left hemiplegia, but without facial paralysis. On recovery of consciousness speech was entirely lost, but he could understand perfectly, + wrote answers to questions with his right-hand. Increased reflexes + Babinski were present.

At 6 am. next morning, 16/8/18, I visited him, + found paralysis completely disappeared, + speech much improved, though not distinct.

The patient was not left-handed, nor even ambidextrous, as far as I could ascertain.

At noon, on 16/8/18, I gave him another heavy dose of intravenous quinine (quinine by the mouth + intramuscular seemed to have no effect) and this considerably upset him for several hours, + he lay in a more or less dazed condition until evening.

His attack of heatstroke was preceded by suppression of sweating. There was no rigor. Patient was a very debilitated type, + had been in hospital on the ship, + during most of the journey from England.



# CLINICAL CHART.

A. F. B181.  
Gratis.  
15-2-10.

(To be attached to the case sheet.)

Corps \_\_\_\_\_ No. \_\_\_\_\_ Rank and Name \_\_\_\_\_ Age \_\_\_\_\_ Service \_\_\_\_\_ Hospital Station \_\_\_\_\_

Disease \_\_\_\_\_ Date of admission \_\_\_\_\_ Date of Discharge \_\_\_\_\_ Result \_\_\_\_\_

Dates of Observation.	19 <sup>th</sup>			20 <sup>th</sup>			21 <sup>st</sup>			22 <sup>nd</sup>			23 <sup>rd</sup>			24 <sup>th</sup>			25 <sup>th</sup>			26	27	28	29	30	31	1	2	3
	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	2	6	10	Time.	Time.	Time.	Time.	Time.	Time.	Time.	Time.	
Day of Disease.	A.M.		P.M.		A.M.		P.M.		A.M.		P.M.		A.M.		P.M.		A.M.		P.M.		A.M.		P.M.		A.M.		P.M.			
Temperature, Fahrenheit.	Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.		Time.			
111	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	
110	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	
109	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	
108	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	
107	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
106	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
105	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
104	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
103	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
102	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	
101	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	6	
100	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	
99	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	
98	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
97	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
PULSE PER MINUTE	92	90	90	92	88	88	86	88	88	88	84	88	80	80	88	72	76	78	80	80	88	84	76	68	72	78	78	76	64	
RESPIRATIONS PER MINUTE	96	96	90	96	90	88	84	88	88	84	76	88	72	88	80	76	76	80	80	88	68	60	72	78	80	62				
Motions.		1	1								1				1						2		1		1					

WET CRADLES + FAN  
Applied continuously for about 36 hrs

Allowed up sitting

Walking about Ward.

Quinine Grs X twice daily

Involved to India per A.T. Sicilia