their own obsessions. I say this with fear and trembling, expecting the retort that such a remark is merely an interesting demonstration of my own complexes or resistances. But not even the youngest of our therapeutic or diagnostic measures is infallible. Yet it is just as erroneous merely to treat the patient as a test-tube in which certain drug reactions can be made to occur, and the man who does so not only fails to realise his responsibility but misses a most interesting part of his work. The old injunction to examine the whole of a patient must be interpreted to include the consideration of his environment in its widest sense. With this widened conception opening out before us, the practice of medicine becomes an occupation of even more absorbing interest, to which we can devote our lives with pleasure and enthusiasm.

HEAT HYPERPYREXIA.¹

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DURING the summers of 1916, 1917, and 1918 a large number of cases of illness due to exposure to high atmospheric temperature occurred amongst the troops in Mesopotamia, and it is upon a careful study of these cases that this paper is based.

Ætiological Factors.

First among these is *climate*. High temperature, coupled with the flatness of the country and absence of shade and the great humidity in certain parts owing to floods and swamps, make Mesopotamia one of the most dangerous countries in the world as regards risk of heat hyperpyrexia.

A maximum shade temperature of 110° F. appears to be the dangerous limit. When this is reached some cases of "effects of heat" are sure to occur, and each degree of rise above this limit is attended by an increasingly larger number of cases. Temperatures of 120° or over are exceedingly dangerous, and on these days, in spite of all precautions, large numbers of heat-stroke cases occur. A comparison of the temperature records of 1917 and 1918 for Baghdad shows the reason of the far greater number of cases of heat-stroke in the former year. Thus, in 1917, on six days in July the shade temperature was 120° and upwards, and on 15 days it was 115° and over. In 1918 the temperature did not reach 118°, and on only two occasions was it 115° or over.

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

As showing the effect of *stagnation of air*, free currents of air have great protective value by promoting evaporation from the skin and loss of heat. Electric fans and punkahs were largely used in

Mesopotamia in hospitals and dwellings occupied by troops from 1916 onwards. They were of vital necessity to the British troops during the hot months. When the temperature is very high the air from a fan is like a hot blast, and unless the body be covered with a moist sheet, or a moist screen intervenes, the fan is of little value as a cooling agency, and, indeed, may do more harm than good.

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

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

Exertion taken during the heat of the day is a great predisposing cause, especially if heavy kit is carried.

In regard to *predisposing diseases*, any disease causing pyrexia predisposes to heat-stroke in the hot weather. Malaria is one of the commonest of these, and in Indians was almost always the cause of heat hyperpyrexia. During an attack of sand-fly fever in the hot months hyperpyrexia occasionally occurred. Captain H. C. Sinderson has suggested² that sand-fly fever is a common cause of heat hyperpyrexia. I cannot agree with this, because in the great majority of cases of heat-stroke there is no evidence whatever of sand-fly fever, and during the great incidence in July and August, 1917, sand-fly fever was entirely absent. Enteric group disease, if occurring in the hot weather, is sometimes complicated by heat hyperpyrexia, as also is typhus fever both in the acute stage and apyrexial period. I have seen heat-stroke occur during the early stages of small-pox before the appearance of the rash.

Pathology and Morbid Anatomy.

I have no doubt that heat hyperpyrexia is a clinical entity and that it occurs apart from inter-current diseases. The clinical history of cases of pure heat hyperpyrexia and the course of the symptoms after the acute stage, which often exhibit a prolonged pyrexia and profound toxæmia with the absence of any bacteriological infection, strongly indicate that the cause is an auto-intoxication as the result of the effect of heat on the body tissues. My experience leads me to believe that this is the cause of heat hyperpyrexia. The auto-intoxication occurring in heat-stroke cases was found to be associated with the presence of acetone and diacetic acid in the urine in only a small proportion of cases (about 12 per cent.), and in these the reaction was of only moderate intensity. It thus appears that heat hyperpyrexia is not due to an acid intoxication. The presence of indican in excess in the urine in acute cases is of importance and is an evidence of the auto-intoxication present.

Suppression of sweating did not, in my experience, always precede heat hyperpyrexia, and though it is undoubtedly an important predisposing cause it cannot be regarded as the primary one. Thus some cases of heat hyperpyrexia occurred suddenly in exposed men who were in good health without

¹ Abstract of paper read before the Medical Society of London on March 8th, 1920.

² Brit. Med. Jour., July 19th, 1919.

any previous evidence of lack of skin action, and in cases of the gastric type of heat hyperpyrexia no previous suppression of sweating was observed, though they had been under observation in hospital for several days.

Clinical Types of Illness Caused by Exposure to Heat.

(1) Heat exhaustion (mild type); (2) gastric type; (3) choleraic or gastro-intestinal type; (4) heat hyperpyrexia (sunstroke). In types 1, 2, and 3 heat hyperpyrexia may suddenly develop unless great care be taken in the removal of patients from a hot atmosphere. Types 2, 3, and 4 are all dangerous, and in them the prognosis is grave. Of 80 severe cases of effects of heat, of which I made careful notes, 13 (16·2 per cent.) were of the gastric type, 9 (11·2 per cent.) of the choleraic, and 58 (72·5 per cent.) were hyperpyrexial.

Gastric type.—This is one of the most interesting and treacherous forms. The patient may have a flushed face, and be restless and irritable with marked nausea and occasional vomiting, the mouth temperature and pulse normal for several days, but the rectal temperature often rising about 2°. The liver usually shows a fatty enlargement. The knee-jerks were lost in all of nine cases on which it was specially tested. This is a valuable diagnostic sign. The knee-jerk reappears when convalescence is established, the time varying with the severity of the case. Many of these cases, after four to ten days of premonitory symptoms suddenly developed a fatal heat hyperpyrexia.

Gastro-intestinal type.—In this type the onset is sudden, marked collapse occurring. The patient usually has a temperature of 101° to 103° F. Vomiting is marked and diarrhoea occurs. There were cramps in the abdomen and legs in some of my cases. The face is pale, the eyes sunken, and the skin pale and clammy. The knee-jerks are generally lost; in 4 cases specially examined they were absent in 3 and diminished in 1. The general appearance of the patient is similar to that of cholera. This type was of uncommon occurrence, but the mortality was high, death often supervening within three or four days.

Heat hyperpyrexia.—The onset is often quite sudden, the patient being taken ill when on duty with a sudden high rise of temperature and loss of consciousness. In some cases the patient, when off duty, may be found by his comrades unconscious and dying. Heat hyperpyrexia frequently occurred in the very hot weather in hospital patients suffering from another disease. In them the temperature would often suddenly rise to 110°, coma and convulsions supervening.

In many cases, however, the onset is more gradual, malaise, headache, and restlessness occurring and sometimes nausea and vomiting. Frequency of micturition is a characteristic early symptom, and is sometimes associated with urethral pain. The temperature is somewhat raised, 100° to 102° or so, and the skin is hot and dry. These preliminary symptoms usually last for a few hours, sometimes as long as 48, after which mental excitement and delirium supervene and the temperature rapidly rises to about 110°. With the hyperpyrexia coma and stertorous breathing occur, the face is flushed and cyanosed, and the conjunctivæ congested. The pupils are often dilated in the early stage and contracted in the comatose condition. Fibrillary twitchings of muscles and convulsions are very common, and the breathing may be Cheyne-Stokes in character. Incontinence

of urine and faeces accompanies the coma. Unless the temperature is reduced by treatment death rapidly occurs from hyperpyrexia, the mode of death usually being asphyxial in type.

Marked cardiac dilatation, often associated with a systolic murmur, occurs in the severe cases. This may remain for a few weeks after the attack and needs special care. In some cases bronchitis and congestion of the bases of the lungs occur. In one case definite air-hunger was observed, and in another a spasmodic type of breathing like that of uræmic asthma. Pulmonary oedema was a terminal event in the fatal cases.

On examining the urine, indican was found present in excess in all of 6 acute cases in which a special examination was made. Acetone and diacetic acid were found present in small amount in 1 out of 8 acute cases specially tested. Albumin was detected in small amount in 3 out of 8 acute cases. No casts were seen.

Of nervous symptoms, restlessness and delirium occur with the onset of hyperpyrexia, and are quickly followed by stupor and coma, with incontinence of urine and faeces. Muscular twitching and convulsions are very common with the high temperature. The knee-jerk is almost always absent in the pure heat-stroke cases during the acute stage. Out of 32 cases specially tested the knee-jerk was absent in 27, diminished in 2, and normal in 3; in these latter cases the hyperpyrexia was probably malarial in nature. In severe cases the knee-jerk does not return for three or four weeks, in milder cases it returns earlier. The presence of knee-jerks is a valuable prognostic sign, for when they have returned there appears to be much less risk of a relapse, and the patient may then be evacuated with safety. This sign was of great practical help in deciding on the disposal of heat-stroke patients. After the acute stage in the severe cases marked mental symptoms often remain for some weeks or longer; irritability, mental confusion, and delusions are not uncommon. Usually, but not always, these symptoms clear up. Defective articulation (anarthria) occurred as an after-symptom in 4 of the severe cases of my series. Nystagmus occurred in 1 case, and squint with diplopia in 1. Multiple neuritis associated with weakness and marked wasting of the legs, the tibialis anticus muscles being most affected, occurred in 2 cases.

Many of the severe cases after the hyperpyrexia has subsided show a pyrexia, the temperature remaining for several days about 102° or 103°. In 1 case the fever lasted three weeks. Numerous blood cultures were made on these pyrexial cases with negative result. Recurrences of hyperpyrexia are very likely to occur after a heat-stroke attack unless the patient is kept in a cool atmosphere, but this is often impossible.

Treatment.

For the gastric type of case removal to a cool atmosphere, bicarbonate of soda in frequent full doses by the mouth, and free purgation give the best results.

The choleraic type of case is treated on similar lines to cholera.

Heat hyperpyrexia demands treatment with sprays of ice-cold water and fans, quinine bihydrochloride being given intravenously or intramuscularly if there is the slightest suspicion of malaria. Convulsions are treated by venesection, or morphia or chloroform inhalations.

[The prophylactic measures were fully dealt with by the author.]