change having disappeared. There was absence of discomfort, tenderness, and pain.

Sensation to pin-prick wellnigh complete, being present over the whole median area with the exception of the tip of the index finger and the two terminal phalanges of the middle finger. Epicritic sensation present over the whole median area with the exception of the terminal phalanges of the thumb, the whole of the index finger, and the middle and distal phalanges of the middle finger. Thenar muscular power largely recovered, opposition of the thumb being present to considerable degree. Slight faradic response of abductor pollicis observable. Tip of thumb appposable to that of index and middle, not quite to that of ring and little fingers.

The hand at this time was restored to considerable practical use, and the man had resumed his occupation, which was that of a skilled electrician.

• HYPERPYREXIAL HEATSTROKE:

A BRIEF NOTE ON ITS ETIOLOGY AND PREVENTION.

BY

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In a recently written thesis on heatstroke—a disease which I studied in Mesopotamia during the exceptionally hot summer of 1917—the etiology, prevention, and treatment of the condition has been discussed more or less fully. It is impossible here to do more than touch lightly upon a few of the chief points which have a practical bearing on the causation and prevention of the disease. The object of this note, therefore, is to suggest the adoption, in heatstroke countries generally, of the following principles of prevention, which have proved completely successful in Mesopotamia after a prolonged and very thorough test under varying conditions. The remarks on etiology are based on a close study of some 240 cases, many of which occurred in the huts of a large base hospital among patients who were already in hospital with some other complaint, and in consequence an excellent opportunity of observing the disease in all stages of its development was afforded.

Etiology.

As regards etiology, and notwithstanding the diverse theories that are put forth in present-day textbooks on the subject, my observations in Mesopotamia point most conclusively to the belief that heatstroke is entirely due to suppression of sweating, which may be present from one to forty-eight hours before the attack.

With sweating suppressed, the bodily temperature tends to adjust itself, in accordance with physical laws, to the temperature of the atmosphere, which may be 115° to 120° at the time, and even to rise higher, for, as Sutton¹ and Graham and Poulton² have pointed out, a rise in bodily temperature produces increased respiratory and nitrogenous changes, resulting in a further increase of internal bodily heat. The processes continue until, when a temperature of about 108° or more is reached, sudden unconsciousness, delirium, and convulsions appear—the result of the physical action of heated blood on the specialized brain cells. The clinical picture of the fully developed attack of heatstroke is thus formed.

I have myself repeatedly observed the pre-existence of suppression—in one case for as long as eighteen hours before the attack—while in numerous other instances I have obtained absolutely reliable histories of it from patients on their recovery.

The cessation of perspiration is probably due to exhaustion of the sweating mechanism during several previous days of intensely severe weather, and there is strong reason to believe that the defect is localized in the sweat glands themselves. Furthermore, large doses of diaphoretics are powerless to produce sweating when once suppression has set in.

Prevention.

By the early recognition of suppressed sweating the prevention of heatstroke becomes an extremely simple matter. The practice which I adopted among hospital patients under my charge in Mesopotamia was to inspect them all periodically—every hour on extremely hot days—and to detect in this way those who required preventive treatment; any who were discovered to have a temperature

of 103°, with complete suppression, were stripped and covered with a wet sheet. This measure simply amounts to an artificial means of producing the same physical effects as sweating. In cases in which the temperature had already risen dangerously high before discovery a portable electric fan directing a current of air over the surface of the wet sheet was placed beside the bed; the evaporative process was greatly assisted by it.

In mild cases, or where the treatment requires to be maintained for several days until the natural sweating power returns, a convenient modification of the method, and one more comfortable for the patient, is to raise the wet sheet on a couple of bed cradles, thus forming a kind of cool chamber in which the naked patient lies protected from the surrounding hot atmosphere of the hut. With this device the additional aid of a fan playing a current of air over the outside surface is usually necessary in order to produce an efficiently cool atmosphere below the cradles on very hot days.

Needless to say, the sheet requires to be kept wet, and much time and labour is saved by using for this purpose some form of spray, such as the Holder-Harrinan pneumatic disinfecting spray, filled with cold water.

If it were possible to remove the patient to a ward which was artificially cooled to a suitable temperature the individual cooling devices would, of course, be unnecessary. The success of these preventive measures can be judged from the fact that in my hut (sixty beds constantly occupied) at the hospital, nine cases of heatstroke with four deaths occurred within the first few days of the 1917 heat wave, but, directly the preventive system was commenced, the outbreak stopped; only two more cases occurred during the remainder of the heatstroke weather, which lasted continuously for two months. The average number of cases occurring in this district (Basra) during the whole period was nine a day. Neither of the two patients who developed the condition in my hut after prevention had been commenced had been placed under preventive treatment.

It will thus be seen that hyperpyrexial heatstroke is an absolutely preventable disease, the leading signs of onset being a hot dry skin, which is typically harsh to touch, and a raised bodily temperature. There is usually also frequency of micturition. These signs are present from one to forty-eight hours before the attack, consequently ample warning is given. The position is simply this: that the patient, having lost his natural means of heat dissipation, requires his bodily temperature to be artificially regulated for him during extremely hot weather until he again begins to sweat, or until the heat wave abates.

If Europeans in heatstroke countries were made acquainted with the premonitory signs and principles of prevention, I see no reason why heatstroke, in a dry climate at least, should occur. I have had no experience of prevention in extremely humid climates such as Calcutta, where the atmospheric moisture may, possibly, hamper the evaporative process, but have no doubt that such a difficulty, if present, could be easily met by employing some other method of cooling the patient.

Even among troops on the march the principle of prevention is applicable. Water, and something that will act as a fan, are practically always obtainable, thus permitting an efficient form of first aid being applied to any man who exhibits the warning signs.

Among sleeping communities—for example, patients in hospital, or soldiers sleeping in barracks on very hot afternoons during heatstroke weather—periodic inspections are necessary in order to detect developing cases. These inspections can easily be carried out by an intelligent orderly, or staff of orderlies. The patient's general condition can be estimated roughly by touching the skin, and only occasionally is it necessary to take a thermometer reading, while those who are freely sweating can, practically speaking, be entirely disregarded.

In the after-treatment following upon an actual attack of heatstroke the tendency to relapses of hyperpyrexia are treated on exactly the same lines as for prevention. The patient frequently does not regain his powers of sweating for days after the attack.

REFERENCES.

- ¹ Sutton: *Journ. of Path. and Bact.*, 1909, vol. xiii, p. 62. ² Graham and Poulton. *Quart. Journ. Med.*, 1912, vol. vi, p. 82.