REFRESHER COURSE FOR GENERAL PRACTITIONERS

CLINICAL EFFECTS OF EXPOSURE TO HEAT AND SUNLIGHT

I. HEAT HYPERPYREXIA AND THERMOGENIC ANHIDROSIS

BY

B. G. MAEGRAITH, D.Phil., M.B., M.R.C.P.

Professor of Tropical Medicine, School of Tropical Medicine, Liverpool

Three conditions of clinical importance may result from exposure to heat—namely, heat pyrexia and hyperpyrexia, thermogenic anhidrosis, and heat exhaustion. Exposure to sunlight may give rise to acute sunburn, to hyperpigmentation, or to a variety of chronic intractable skin conditions.

This article deals with heat hyperpyrexia and thermogenic anhidrosis, both conditions in which the basic pathogenic factor is inhibition of sweating. Part II, which will appear next week, will be concerned with heat exhaustion, in which disturbances of both the cardiovascular system and the body electrolyte balance are prominent, and with the clinical effects of sunlight, particularly sunburn.

HEAT PYREXIA AND HYPERPYREXIA

This syndrome covers a wide variety of clinical conditions, ranging from mild fever to hyperpyrexia, and is usually defined as a body temperature of 107° F. (41.7° C.) or higher. The fully developed syndrome of hyperpyrexia is described here; the milder forms of pyrexia call for no special comment. There is a good deal of confusion about this syndrome, owing mainly to the failure to appreciate that it may occur with or without the complicating factors of water and salt deficiency and vascular collapse.

Exposure to heat must continue for some time before the syndrome can develop. Its appearance is not dependent on exposure to direct sunlight. Short bursts of exposure, as, for instance, in stokers, do not usually give rise to serious symptoms. Hyperpyrexia may appear in either sex and at any age; it is commoner in the elderly and unacclimatized. It may occur in indigenous individuals normally exposed to high temperatures.

Predisposing Factors

The evaporation of sweat and the consequent escape of heat may be reduced by excessive or ill-designed clothing, by absence of air movement, or by enclosure in a confined space. The production of sweat may sometimes fall below that required for evaporation, as in extreme dehydration or febrile illnesses, particularly malaria. Overproduction of heat may occasionally be important. Excessive protein intake or overactivity of certain endocrine glands, especially the thyroid, may stimulate metabolism unduly, but the commonest factor involved in excessive heat production is muscular exercise, sometimes initiated or sustained by excitement or overindulgence in alcohol.

A very common predisposing factor is absence or loss of acclimatization to heat. Individuals unaccustomed to hot surroundings are likely to be affected more easily than those acclimatized, but when the latter have lost their acclimatization they become highly susceptible, as do miners when they return to work after a vacation (sometimes after only a few weeks). A history of previous heat intolerance is present in only a small percentage of cases.

Clinical Picture

The onset of acute symptoms may be very rapid, but usually well-defined prodromal symptoms first appear several days before the full syndrome. There is frequently a history of progressive dizziness, sometimes associated with staggering gait, headache, drowsiness, restlessness, and mental confusion. Speaking and swallowing may be difficult and severe and increasing thirst often occurs. When the latter

is present and ample fluids are available there may be associated polyuria and frequency. Anorexia, nausea, and vomiting are common in the few days before the onset of fever.

On admission the patient may be in delirium or deep coma. If conscious, he is likely to be restless, anxious, and confused. The oral temperature is very high. In some cases it may be over 110° F. (43.3° C.). The rectal temperature is about 1° F. (0.55° C.) higher. *The skin is dry and flushed.* There may be some cyanosis. The hot dry skin is characteristic of the syndrome and indicates its pathogenesis—namely, complete inhibition of sweating. The breathing is rapid (30 or more respirations a minute) but not panting (the normal reaction to exposure to heat). In coma it may be stertorous or intermittent, or of the Cheyne-Stokes type. The pulse is thin; the rate is fast, often over 130 beats a minute. Occasionally soft systolic murmurs are present, and transient myocardial changes may be recorded in the electrocardiogram. Notable changes in the blood pressure do not occur unless the syndrome is complicated by shock.

The central nervous symptoms are outstanding. In rapidly fatal cases delirium or coma, if present at the outset, is likely to persist to the end. When the illness lasts for some days before a fatal issue early coma may temporarily regress, returning terminally. In most treated cases coma and delirium disappear quickly as the temperature is brought down. Convulsions and muscular twitchings, especially in the limbs, are common. Neurological signs are irregular and vary considerably from patient to patient. They are most severe in patients in whom the effects of the high fever predominate over those of vascular collapse. Their severity is roughly proportional to the temperature.

There are no characteristic changes in either the urinary volume or constituents. If dehydration is present at the time of onset the volume will be reduced and the concentration increased; if salt is deficient it may be absent from the urine. If the patient has suffered from severe thirst and has been drinking freely there may be some polyuria, which may persist for some time after recovery from hyperpyrexia; in these cases the urinary volume is high and the urine dilute, although the total output of electrolytes may be within normal limits. Granular or hyaline casts may also be present in small numbers and there is often some albumin. The blood urea nitrogen is usually a little high—that is, 40–50 mg. per 100 ml. Occasionally, in patients with very high fever, the total blood non-protein nitrogen may rise without a corresponding rise in the urea nitrogen. Total blood and plasma chloride concentrations depend on the prevailing water/electrolyte balance. The concentrations may be within normal limits or may be low when salt deficiency is also present. There is usually a gross reduction in the number of blood platelets and a corresponding increase in clotting and bleeding times.

Without treatment the severe case will progress rapidly to a fatal end. The morbid changes found in the body after death depend largely upon whether the dominant feature was hyperpyrexia or vascular failure. The latter (shock) is a very serious complication. The first effects of shock are reversible, but with the passage of time irreversible and fatal changes occur in the organs, particularly the brain, liver, and kidneys. The development of shock is heralded by remarkable changes in the patient's appearance. The temperature falls rapidly, sometimes to below normal. The flushed dry skin becomes pale, sometimes cyanosed, cold, and moist with sweat. The breathing quickens and becomes

shallower. The systolic blood pressure falls rapidly; the diastolic pressure falls more slowly and may ultimately become unmeasurable. The effective circulating blood volume is greatly reduced and haemoconcentration becomes evident. The patient collapses. If not already comatose he may pass into coma or stupor, or become very restless, irritable, and anxious. Watery diarrhoea is common and there may be incontinence. The urinary volume is reduced and the oliguria may progress to anuria, associated with acute uraemia.

Sometimes evidence of damage to hepatic function is found. The liver edge becomes palpable and tender; epigastric discomfort, nausea, vomiting, and sometimes hiccups develop; some degree of jaundice appears; and bile may be found in the vomit, faeces, and urine. The vascular damage which occurs in shock may become clinically evident. Petechial haemorrhages may appear on the skin and mucous membranes, and the vomit and diarrhoea may contain blood. Pulmonary oedema may dominate the final stages.

In patients in whom there was some disturbance of water/salt balance at the time of the onset of hyperpyrexia, there may be visible signs of dehydration—pinched facies, flabby inelastic skin and tissues, and reduction of chloride in the blood and urine. If vomiting is severe and persistent there may be muscle cramps and sometimes tetany.

Course and Prognosis

If effective treatment is applied early the mortality is low. Death occurs within the first 24 hours in about 70% of fatal cases. It may be delayed for up to 12 days after the onset. Early death results largely from the acute effects of high fever, and later death usually from the effects of complications. The prognosis in an individual case depends to some extent on the length of illness and the height and duration of the fever. The best criterion is, however, the development or otherwise of shock. Shocked patients have less chance of survival. In general, cases with a very acute onset associated with rapidly developing coma are the most likely to die. In practice the overall mortality should be less than 5%.

Diagnosis

There must always be a history of exposure to high environmental temperature for a considerable time. There is usually some history of predisposing causes, particularly ill-advised clothing and heavy exercise. The patient will often volunteer the history of noticeable reduction of sweating, thirst, and polyuria. Diagnosis can be made from the high fever, the flushed dry skin, and the mental condition. *It is essential to exclude other causes of fever, especially falciparum malaria, one of the complications of which is hyperpyrexia clinically indistinguishable from that resulting from exposure to heat.*

Treatment

The high temperature reached in hyperpyrexia is due to failure of sweating. The aim of treatment is to reduce the temperature and promote sweating.

Reduction of temperature is best achieved by evaporation of water from the body surface. The patient should be stripped, covered lightly with a moist sheet, and fanned vigorously, water being applied to the sheet so as to keep up continuous evaporation. The temperature can be reduced by this means much more efficiently and rapidly than by other methods such as sponging with cold water or the application of ice baths. The temperature must be recorded every few minutes. It will begin to fall rapidly after the start of treatment. In the first instance it should not be brought down below 102° F. (38.9° C.), otherwise shock and fatal collapse may ensue. When the temperature has fallen to about this figure active treatment should be temporarily stopped. The temperature usually continues to fall to normal or below after cessation of treatment. Secondary rises are to be expected. If the secondary rise is high, further treatment on the above lines should be instituted.

In most cases natural sweating is restored after the first treatment; under these circumstances prognosis is favourable. In very severe cases the return of sweating is delayed and treatment may have to be repeated several times.

The patient should be nursed in as cool a room as can be obtained. Air conditioning is the ideal. He must be kept quiet until well into convalescence.

Unless the case is complicated by water/salt deficiency or vascular failure, no other treatment is necessary. Complications are treated as they arise (see under heat exhaustion in Part II). Sequelae may be resistant to treatment. Mental symptoms require expert handling. Continuous headache may respond to nicotinic acid.

THERMOGENIC ANHIDROSIS

This condition was first clearly defined in the second world war. The syndrome is characterized by some inhibition of sweating, especially on the trunk and limbs, which is, however, never complete, so that high fever does not develop. It is not known whether the inhibition of sweating is central or peripheral. On the whole most authors believe that it is essentially central in origin. It has been pointed out, however, that the high sweat chloride content may be an indication of exhaustion of the sweat glands. There are characteristic skin changes and sometimes enlargement of the axillary and inguinal lymph nodes. The affected skin shows an occluding hyperkeratosis associated with retention of sweat, and ultimately rupture of the ducts of the sweat glands, leading to disorganization of the epithelium and the production of intradermal vesicles. It has been suggested that inhibition of sweating results, at any rate in part, from pathological changes in the local sweat glands. In any given section of the skin, however, relatively few of the glands are affected. Occasionally the skin changes may be observed without the general signs or symptoms. The syndrome may be complicated by water/salt imbalance and cardiovascular disturbances.

Clinical Picture

Thermogenic anhidrosis develops most commonly in individuals who have been exposed to high temperatures for some time. The signs and symptoms are at their worst during the hottest part of the day. Dyspnoea and palpitation are severe after exercise. Increasing periods of rest are required between attempts to work, until finally work becomes impossible. Most patients complain of giddiness, frontal headache, insomnia, anorexia, and epigastric discomfort. There are often subjective feelings of warmth and discomfort, and frequently a tight feeling in the affected skin. The symptoms pass off with rest in cool conditions.

There is nearly always a history of extensive and recurrent prickly heat, which improves and may disappear a week or two before the anhidrosis develops. The patient often notices that in the two or three days preceding the development of the syndrome sweating from the body, but not the face, had progressively diminished. A few days of excessive sweating on the trunk may precede this inhibition.

On examination the patient is restless and apprehensive. His oral temperature ranges from 99 to 102° F. (37.2 to 38.9° C.). The pulse rate is fast, varying from 100 to 130 beats a minute. The respiration rate is increased; rates of over 40 are common. In the uncomplicated syndrome there are no characteristic changes in the cardiovascular system. On the limbs and most of the trunk the skin is warm and dry, except for occasional small islands of sweating. The head and neck, on the other hand, sweat freely, and the palms, soles, axillae, and groin are moist.

The skin changes are characteristic. There are myriads of tightly packed tiny greyish papules about 1 mm. in diameter, many surmounted with vesicles containing clear fluid. Papules occur in patches, especially on the trunk and the upper dorsal aspects of the arms and legs. They commonly develop first on the arms, then on the trunk, and finally on the legs. In some cases the arms, legs, and trunk are

involved simultaneously. The papules do not appear on any moist areas which may be present and are never found on the head and neck. Superficially the skin condition resembles goose flesh, but it is not related to the skin hairs. It may be mistaken for prickly heat, but there is no erythematous ring of the papules. The skin between the papules is dry and usually warm. Hard oedema of the fingers and arms, associated with enlarged discrete axillary and groin lymph nodes, has been described in some cases.

There are no characteristic changes in the blood or urine except in cases complicated by water/salt imbalance or shock. In some cases in which thirst is a striking feature polyuria may occur, the urine being dilute, with a low chloride concentration; the output of salt in 24 hours is within normal limits. The salt concentration of the sweat is usually high and frequently exceeds 0.5%.

Treatment

Treatment consists chiefly in rest in cool surroundings. Disturbances of water/chloride balance should be controlled by adequate administration of water and salt; parenteral administration is not required in uncomplicated cases.

Treatment brings about a rapid response of temperature, respiration, and pulse. The skin changes disappear more slowly, however, and are often succeeded by branny desquamation which is particularly noticeable on the limbs. Recovery is indicated by increasing ability to exercise without return of symptoms and by the reappearance of sweating, usually first on the trunk. In the early stages of treatment, exertion, exposure to heat, or injection of pilocarpine give rise to a return of symptoms and the peculiar distribution of sweating already described. After recovery, exposure to heat and pilocarpine produce a normal sweating reaction. The enlarged lymph nodes subside after some weeks.

In the uncomplicated case full recovery may be expected in 10 to 14 days. The condition tends to relapse, especially if rest in cool conditions is continued for too short a time. It may be necessary in cases which relapse to remove the patient from the hot environment.

[Part II will be published in our next issue.]

APPENDICITIS IN THE WEST INDIES*

BY

AUBREY LEACOCK, M.Chir., F.R.C.S.

Late Surgeon Specialist, Barbados General Hospital

In 1946 I was appointed to the Public Hospital at Georgetown, British Guiana, and in 18 months' work there I was interested to observe that appendicitis was far less common than it is in England. In British Guiana, in a hospital of 600 beds, we removed fewer than 100 appendices each year. However, we noticed from its *Annual Report* that in the Barbados General Hospital of 300 beds about 400 appendicectomies were carried out annually, and we congratulated ourselves on our diagnostic acumen in avoiding unnecessary operations.

On my transfer to Barbados in January, 1948, I was therefore expecting to find appendicectomy being used as a cure-all for a host of conditions. Very soon, however, I found that my expectations were wrong, and indeed since 1948 the number of appendicectomies in Barbados has continued to increase.

In the West Indian Colonies it is possible to discover with reasonable accuracy the incidence of a non-notifiable surgical disease, since almost all operations are done in Government hospitals and the size of the population at risk is known. I have therefore obtained

the figures for appendicectomy in most colonies for the 11-year period 1938-48, from their annual medical reports or from the Directors of Medical Services.

In the first Table, the figures relate to the number done each year. In general, few cases of definite appendicitis would be refused operation in these territories, and

TABLE I.—Appendicitis Rates, 1938-48

	Average Population	Average Annual Incidence per 100,000	Crude Death Rate per Million Population	Average Case Mortality (%)
Jamaica	1,260,000	200	28	1.4
Barbados	200,000	171	20	1.2
Antigua	40,000	115	21	1.8
Dominica	52,000	83	11	1.3
B. Honduras ..	61,000	80	38	4.8
Trinidad	554,000	70 (4 yr.)	19 (4 yr.)	2.7 (4 yr.)
St. Vincent ..	62,000	36	7	2.0
B. Guiana	366,000	30	14	4.6
Grenada	85,000	25 (7 yr.)	23 (5 yr.)	9.2 (5 yr.)
St. Lucia	74,000	21	7	3.3
Oxfordshire ..		203*		
England and Wales			71.5†	
Italy			39.0†	
Switzerland ..			102.7†	

* Moloney *et al.* (1950). † Young and Russell (1939).

inaccuracies from this cause are not likely to be appreciable. Neither is there likely to be sufficient inaccuracy in the collection of statistics by medical departments to explain the differences. Small differences in the crude death rate should be discounted, but the large ones, as between 28 for Jamaica and 14 for British Guiana, are apparently significant, bearing in mind that the survey covers 11 years.

In most territories the annual incidence shows no definite trend, the figures being roughly constant from year to year in the period of the survey. The figures for Antigua, however, show a marked rise in the last two years of the survey, which has continued in 1949; and those for Barbados show a steady increase in the last five years of the survey, which has continued in 1949 and 1950.

Aetiology

In his monograph on appendicitis, Aschoff (1932) stated that "the frequency of the disease is almost uniform among all peoples of the earth who are under adequate medical and surgical observation." If this is so, can we explain these statistics?

I cannot believe that the professional ability of doctors in these territories varies so tremendously as to account for the observed variations in incidence. The possibility that many appendicectomies in Barbados are unnecessary has been examined by a follow-up (see later).

Different incidences might, however, be due to differences in the standards of hospital service and the availability of medical advice. I have obtained from a well-informed and unbiased source, which must remain anonymous, a classification of the medical services of these colonies into four categories, of excellence or the reverse (Table II).

TABLE II.—Classification of Medical Services

Category	Colony	Appendicitis Incidence
A	None	—
B	Barbados	171
	British Guiana	30
	Jamaica	200
	Trinidad	70
C	Antigua	115
	British Honduras	80
	Grenada	25
D	Dominica	83
	St. Lucia	21
	St. Vincent	36

*Read at the Conference of Caribbean Branches of the B.M.A., in Trinidad, January, 1951.