HEAT EFFECTS

A Clinical Survey

by

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with Honours 1930

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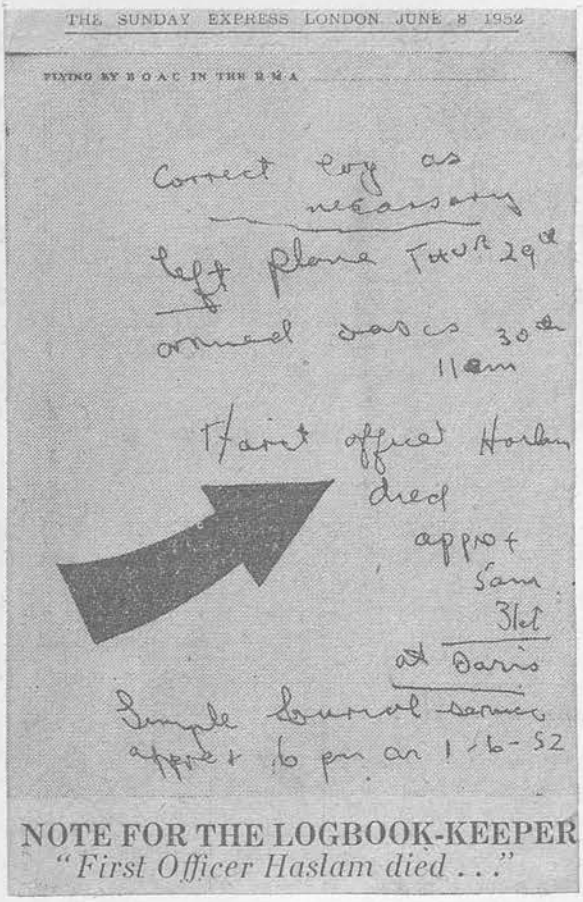
INTRODUCTION AND SCOPE

The following paper represents a clinical recording of all cases of heat effects occurring during one hot season in Cawnpore, together with a brief historical survey extracted from the bibliography on the subject.

The details are necessarily clinical and no attempt was made to obtain biochemical information such as that furnished by Ladell, Waterlow and Hudson (Lancet 14.10.44), but if the appraisal of cases and the estimation of treatment appear somewhat rough and ready, it can yet be claimed that every patient recovered, and that our observations were necessarily conducted at that somewhat neglected spot, the bed-side.

Heat Stroke lacks the profound national consequences of the great mediaeval epidemics or the widespread and all but universal tropical diseases, such as malaria or ankylostomiasis. It is a disease of the white man in the tropics, with a short history but a grim one, and though conditions have greatly changed since Barrack Room Ballads and Soldiers Three were written, yet the danger is eternally present and is avoided only by constant vigilance.



THE SUNDAY EXPRESS LONDON JUNE 8 1952

FLYING BY B O A C IN THE R M A

Correct log as necessary

left plane Tokyo 29th

arrived Karachi 30th 11am

F/Lieut officer Haslam died appx 5am 31st at Karachi

Simple Coronal Sclerosis appx 6 pm on 1-6-52

NOTE FOR THE LOGBOOK-KEEPER
"First Officer Haslam died . . ."

Paradoxically enough the first scientific approach to the problem was made not in the tropics, but in this country by Haldane, whose pioneer work in the Cornish Tin Mines paved the way for a first real understanding of the whole subject of heat regulation.

Of the cases seen, detailed notes of those suffering from hyperpyrexia are alone given, as their progress, almost minute by minute, is of interest and indeed of vital importance. The cases of heat exhaustion and collapse have been considered

statistically rather than individually as they conformed very much to pattern.

Methods of treatment are described and the necessity of advance preparation, speed, and the intelligent co-operation of ward and laboratory staffs are stressed.

NORMAL RESPONSE TO INCREASED TEMPERATURE

(Lovatt Evans)

The normal physiological responses to heat are given by Lovatt Evans as follows:-

Increased respiration.

Rise in blood Ph.

Increased alkalinity of urine.

Rapid pulse.

Fall in blood pressure.

Dilatation of skin vessels.

In extreme cases, a rise in the basal metabolic rate.

Heat Production (Samson Wright, Applied Physiology, 1952, p. 431) takes place chiefly in the skeletal muscles, and to a less extent in large glands, such as liver and kidney.

Samson Wright (Applied Physiology, 1952, p.327) investigated the vascular response to heat and found that on perfusing an organ with saline at 42°C. the capillaries first dilated, followed by the arterioles. Exposure of a healthy subject to 40°C. for one hour (Samson Wright, Applied Physiology, 1929, p.277) increased the pulse rate to 90 or 100 per minute, and the circulation rate by 20 to 50 per cent.

The metabolic rate in such conditions is not increased.

The blood leaving the skin veins is highly saturated with oxygen, showing that the increased skin blood supply is called for not by oxygen requirements, but for temperature regulation.

MECHANISM OF HEAT EFFECTS

It has long been recognised that the factor producing the series of syndromes now known as "heat effects" is, in fact, heat, and not a result of some mysterious constituent of the sun's rays, though Heiss, writing in 1942 (Med.Klin.)refers to "sunstroke" being caused by the effect of the rays of the sun on the meninges, especially in the occipital and cervical regions, resulting in serous meningitis, neck rigidity, Kernig's sign and general symptoms of meningeal irritation - an illuminating example of Teutonic obstinacy, even after their experience of desert warfare.

Experimental work by Marsh* went a long way towards resolving the heat versus sun-ray controversy. Rabbits were enclosed in cooled boxes, from which their heads alone emerged. The animals' heads were shaved and exposed to the direct rays of the sun, at temperatures approximating to desert conditions, and no ill-effects were noticed.

Animals killed by excessive heat showed oedema of the brain, with a large volume of straw coloured fluid in the ventricles. He supposed that the cerebro-spinal pressure was raised and this is also noted in the human subject by Carr (B.M.J. 20.6.1942).

* Lancet, 25.10.1930.

who reports a case of heat hyperpyrexia on board ship with a very high pressure on lumbar puncture.

C.J. Martin is quoted by F. Marsh (Heat Stroke and Heat Exhaustion, British Encyclopaedia of Medical Practice, 1937, volume 6, p.398) as saying "If the bare head of a man whose body is already in a state of pyrexia is exposed to the bright midday tropical sun, local heating to a depth of one or two centimetres of the skull surface and subjacent structures, including some brain tissue, may occur with serious consequences."

It may be taken, however, in view of Marsh's own experiments (Lancet 25.10.1930) that this factor is negligible and that the effects are due to a general incidence of heat on the body and not to its action on any localised area. This is confirmed by Rogers and Megaw (Tropical Medicine, 1944, p.465).

When this was realised, the neck shields and spine pads, apparently so essential to life in the tropics a generation ago, died a natural death, and more importance was given to general measures which allowed the heat centre to function efficiently.

LOCALIZATION OF HEAT REGULATING CENTRE

It was obvious that a delicate centre for regulating the heat production and loss of the body was present somewhere in the central nervous system, and experimental evidence steadily accumulated that this area was in the hypothalamus.

Ott and Carter in 1887 (Therapeutic Gazette, vol. 3, p.597) and Ott himself (Journal of Nervous and Mental Disease, 1888, p.85) found that transection of the spinal cord in the cervical region caused an immediate drop in skin temperature, the decerebrate animal taking the temperature of its surroundings in a manner analagous to the so-called "cold-blooded animals". Further experiment localized the chief centre in the hypothalamus, and in 1912 this work was confirmed by Isenschmid and Krehl (Archives of Experimental Pathology & Pharmacology) and again in 1939 by Ranson (Association of Research in Nervous and Mental Disorders, 1939).

There is also apparently some higher control involved. Pinkston, Bard and Rioch (American Journal of Physiology, 109 of 1934) removed one frontal pole in dogs, and found that the skin of the opposite side of the body was warmer and redder than that of the unaffected side.

ACTION OF HEAT REGULATING CENTRE

It is probable that the heat regulating centres react to the temperature of the blood reaching them, but the rapid response seems to argue a peripheral reaction carried to the centre by special sensory nerve fibres.

Duthie and Mackay (Brain 1940) occluded the venous return from a limb, which was then placed in a hot bath. This effectively prevented heated blood reaching the central nervous system, but a rapid reflex vasodilatation was noticed in the opposite limb. Samson Wright (Applied Physiology 1952, P. 473) supports the dual response - nervous and circulatory. For instance, he finds that warming the blood in the internal carotid artery causes the usual reflex actions ensuring heat loss - peripheral vasodilatation and sweating.

ASSESSMENT OF BODY TEMPERATURE

The temperature of the body varies widely from place to place and in most areas varies with the external temperature. For accurate work one would wish to know the "mean body temperature", but in practice it is found that the rectal temperature not only approximates very closely to this, but is remarkably stable (in health) through wide environmental changes.

Pembrey and Nicol [illegible] (Journal of Physiology, vol.23, p.388 of 1898) found that the average rectal temperature was 99.4°F. with a variation of about 2° in 24 hours. After work, in tropical conditions, the rectal temperature in healthy adults might rise to 101°F.

Pembrey, M.S. (Journal of Royal Army Medical Corps, August 1913) quotes figures from Aldershot Command showing a rise in rectal temperatures among troops after marching.

Oral temperature variations, on the other hand, were much greater, especially after exercise.

Although this does not affect the present discussion, it is noted that marked variation was also found under very cold conditions, but a difference of 4.5°F. was sometimes found between oral and

rectal temperatures.

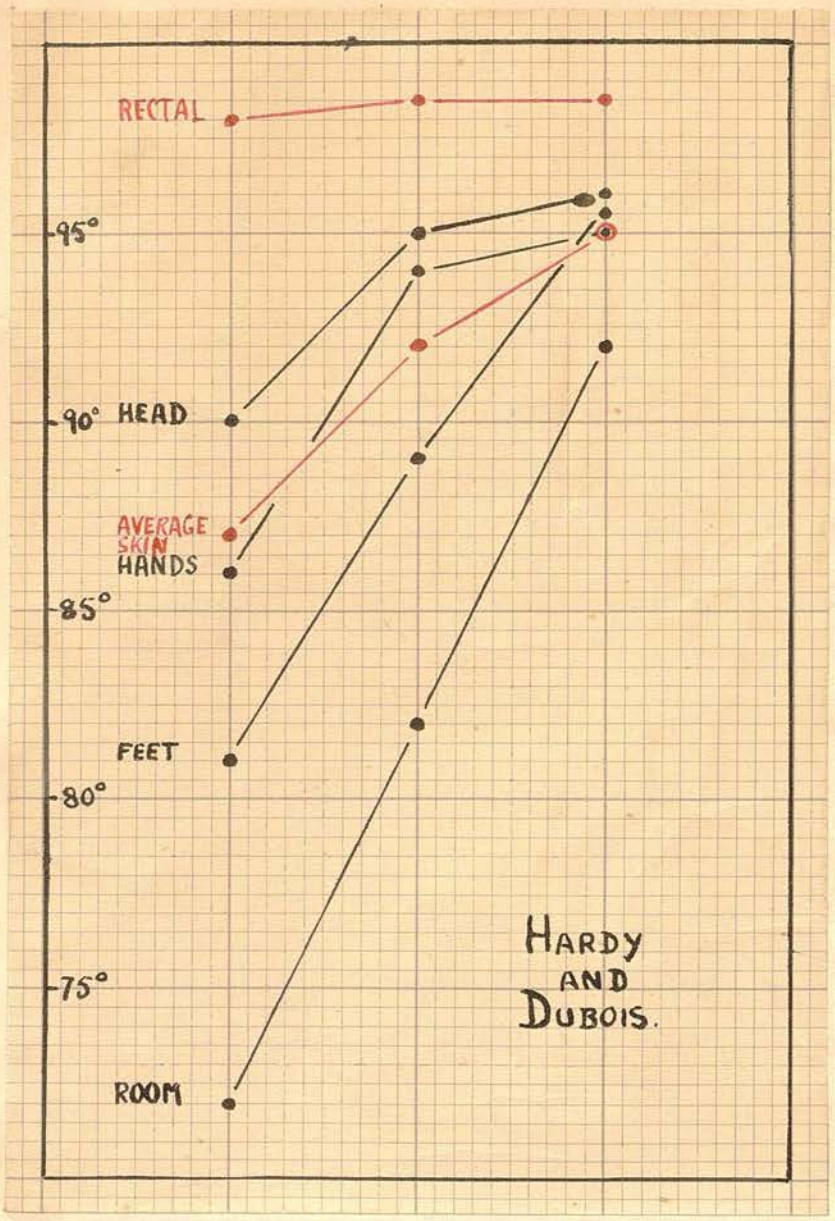
The following table (Hardy and Dubois 1941) gives more exact information, and shows clearly the value of rectal temperatures as contrasted with any other method.

Skin temperatures were taken by Kata Thermometer and the experiments were done over what in the tropics might be considered a very modest range. The angle which the various graphs make with the horizontal gives the variation in temperature at different room temperatures.

Percentage variations are:-

Rectal	0.5%
Skin of head	6.0%
Skin of hands	9.5%
Skin of feet	15.0%
Average skin	8.0%

In view of these results all temperatures mentioned in this thesis are rectal.


RECTAL
95°
90°
HEAD
AVERAGE SKIN
HANDS
85°
FEET
80°
75°
ROOM
HARDY AND DUBOIS.

MECHANISM REGULATING LOSS OF BODY HEAT

Loss of body heat, if such trivial sources as loss by passage of faeces and urine are ignored, is in a major degree from the skin and in a minor from respiration.

The essential factor in the regulation of a heat balance is the skin. Heat is lost by the usual three methods of transfer, convection, conduction and radiation, and by the evaporation of sweat from the surface. At 90°F. up to 12% of the circulating blood may be sent to the skin (Lovatt Evans, Physiology, 1952, p. 1053).

The relative value of loss of heat from convection, conduction and radiation, which can be considered together, and from evaporation of sweat, changes greatly with the state of the individual, and with the surrounding temperature.

Du Bois (Mechanism of Heat Loss & Temperature Regulation, Stanford University, California, 1937) found that at normal temperatures the heat loss was 100 calories per hour in an individual at rest - of this 81% was lost by convection, conduction and radiation, and 19% by vaporization of sweat. After strenuous exercise (2 games of squash rackets) the hourly

loss became 500 calories, 25% by conduction etc., and 75% by evaporation: thus active exercise caused a rise of from 81 to 125 calories by conduction, and from 19 to 375 by evaporation.

At high external temperatures another factor comes into play (Samson Wright, Applied Physiology 1952, p.476). Heat flows from hotter bodies to cooler, and the rate of flow is proportional to the difference in temperature. When the air temperature passes that of the body, the effects of convection, conduction and radiation are reversed, so that evaporation has to cool not only the skin, but also its garment of air at a still higher temperature.

If the surrounding air is in motion, the convection effect is found to vary as the square root of the speed (Hardy and Du Bois), the ultimate effect still depending on whether the air temperature is higher or lower than that of the skin.

Lovatt Evans (Physiology, 1952, p.1054) states that the critical air temperature for sweating in the nude subject is 31°C. (88°F.) and in the lightly clad 29°C. (84°F.).

Naturally, at external temperatures above 98.6° all loss of body heat is by evaporation.

There is a most comprehensive and scientific account by Blagdon (Philosophical Transactions,

1775) of experiments carried out by Dr. Fordyce, by Blagdon himself and by other volunteers, which may be said to have covered the tests carried out 170 years later at Baltimore (page 18). Dr. Fordyce had fitted up a suite of rooms enabling him to test bodily reactions at temperatures of 120° to 130°F. with 100% saturation. In dry heat also Dr. Solander stayed in a room at an original temperature of 210°F. for ten minutes, and Mr.Banks at a temperature of 211° for seven minutes without experiencing any undue distress. Both noted that their presence in the room reduced the temperature by some 15°F. Truly there were giants in these days.

Fordyce states (page 116) that the evaporation from the body occurring when the air is dry "assists its living powers in producing cold," and it is suggested that living tissue, animal or vegetable, can produce as circumstances require either a cooling or a warming effect on its immediate environment. Samson Wright (Applied Physiology 1929, p. 403) makes the statement without references that in a dry room at 240° to 260°F. no rise of body temperature occurred.

The rate of evaporation of sweat varies directly as the difference between the vapour

pressure at the skin surface (i.e. during heavy sweating, 100% saturated) and that of the surrounding air, and obviously under favourable conditions is increased by air movement which replaces the saturated air by dry. Each gramme of water evaporated - the latent heat of evaporation - absorbs 0.58 calories.

The importance of evaporation as a means of regulating body temperature was apparent even in mediaeval times. Boys and youths with gilded skins took part in Florentine and Roman carnivals - for instance Oscar Wilde (The Picture of Dorian Gray) relates how Pietro Riario, Cardinal Archbishop of Florence, "gilded a boy that he might serve at the feast as Ganymede or Hylas." There are reports of deaths among these unfortunates, detailed enough to show that although the cause was unknown, it was at any rate recognised as associated with the gilding. (Lovatt Evans, Physiology 1952, page 1054).

Haldane's article (Journal of Hygiene, vol. 5 No. 4, 1905) is remarkable in covering so minutely and completely ground upon which practically no previous work had been done, and one is struck by his grasp of the subject, and notes how little his facts have dated with the passage of years.

His experiments, which called for considerable hardship on the part of himself and his assistants,

were carried out in the Dolcoath tin mines in Cornwall. He found that in the still, moist air of the mines it was impossible to maintain a heat balance at a wet bulb temperature of 89°F. in fully saturated conditions - i.e. a steadily rising body temperature was observed.

In moving air bearable temperatures were appreciably higher. For instance, with an air speed of 170 feet per second in the same conditions of 100% saturation, 93°F. could be tolerated.

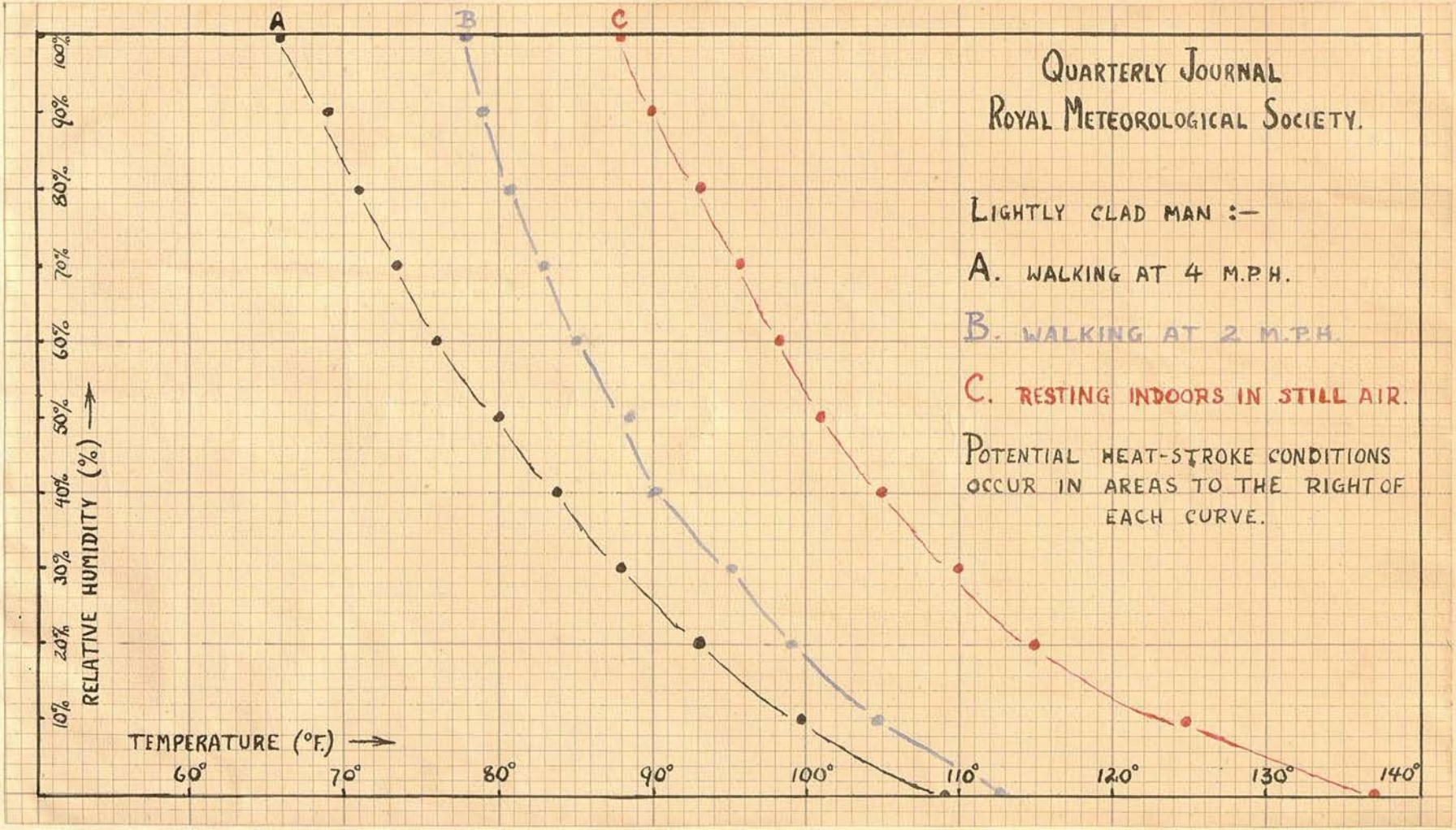
The limit for leisurely work in still air was 73°F. and symptoms of intolerance were ushered in by throbbing in the head and an increase in pulse rate. The latter was in the nature of a rise of 20 per minute for each 1° rise in rectal temperature. On leaving the humid conditions of the mine the pulse dropped at once to normal, while the rectal temperature fell slowly.

The following Chart, originally published in The Quarterly Journal of the Royal Meteorological Society, shows clearly the influence of humidity.

Air temperatures are plotted along the horizontal axis and humidity along the vertical, and the area to the right of each curve represents insupportable conditions in which the temperature regulation centre will fail to keep the balance, and

hyperpyrexia may be expected to occur.

The Lancet (Leading Article 21.10.1944) states that "full saturation of the atmosphere at a temperature of 89°F. makes sweating useless." It will be noticed that this agrees with Haldane's Chart of forty years earlier, where any further rise in temperature at the 100% humidity level would result in the plot being placed in the heat stroke area on the right of the curve.


A
B
C
QUARTERLY JOURNAL
ROYAL METEOROLOGICAL SOCIETY.
LIGHTLY CLAD MAN :—
A. WALKING AT 4 M.P.H.
B. WALKING AT 2 M.P.H.
C. RESTING INDOORS IN STILL AIR.
POTENTIAL HEAT-STROKE CONDITIONS OCCUR IN AREAS TO THE RIGHT OF EACH CURVE.
RELATIVE HUMIDITY (%) →
10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
TEMPERATURE (°F.) →
60° 70° 80° 90° 100° 110° 120° 130° 140°

EXPERIMENTAL WORK ON HEAT EFFECTS

Bean and Eichna (Federation Proceedings, Baltimore, Sept. 1943) in America subjected fifty-six healthy young men of 20 - 28 years of age to an artificial tropical environment. The temperature was kept at 120° from 8 a.m. to 5 p.m. and at 90° for the rest of the day with the exception of two hours, when they enjoyed normal conditions. Humidity was kept at 20% and active work was carried out.

Among these subjects the chief symptoms were high pulse rate, raised temperature, unstable blood pressure, dizziness and mental dulness. On continuing the experiment, however, it was found that a considerable degree of acclimatization occurred after four or five days, all abnormal physical signs disappeared and normal mental alertness returned.

Further experiments were carried out by Eichna Bean Ashe and Nelson (Bulletin of Johns Hopkins Hospital, Jan. 1945) reproducing humid rather than desert conditions. By day the dry bulb thermometer was kept at 90°, with 95% humidity (N.B. this plot is in the heat-stroke area of Chart No. 1 even when resting) and at night the dry bulb was at 85° with humidity 75% to 85%. Similar symptoms to those

noted above were obtained, but it was found that acclimatization began to be evident as early as the second day, and was complete by the tenth.

Bean Eichna and Ashe.

(Journal of Laboratory and Clinical Medicine, 1945: 357)

The above authors summarized their findings as follows:-

The wet bulb temperature is more important than the dry.

Below 91° W.B. men work easily.

Between 91° and 94° W.B. there is loss of vigour and alertness.

Above 94° W.B. less than an hour's work can be done without prolonged rest intervals.

Danger symptoms:

Heart rates of 150 or over.
Rectal T. of 102 - 103.5°F.
Headache.
Nausea and vomiting.
Weakness and cramps.
Dyspnoea.
Collapse or syncope.

The effects of dehydration alone without salt deprivation were investigated in the Journal of Physiology (1944, vol.102, p.406).* The first signs

* Black, McCance and Young.

were change in behaviour - in the words of the article, "Serious natured people became positively sombre while the normally cheerful showed a hollow vivacity." Mental concentration was impaired. By the third day mouths and throats were dry and voices husky, and on the third or fourth day there was usually some cyanosis.

Chemical findings:

Blood urea raised.

Plasma sodium rose above 10%.

Plasma chloride also rose.

Serum potassium fell, and there was increased output of potassium in the urine.

The total osmotic pressure of the body rose since osmotically active salts were being retained in increased concentration.

Elkington and Taffel (Journal Clinical Investigation (Yale) 21. 787) confirmed the rise in osmotic pressure in experimental work in dogs.

From the above it would appear that after a certain level of concentration of the plasma is reached, cell fluid is sacrificed, its presence being shown by the increase in potassium salts in the urine.

Rowntree (Physiological Reviews, vol.2, 1922, page 158) quoted by Bard): In patients with impaired renal function excessive water intake may produce headache, dizziness, vomiting and cramps.

He noted that at the same time there was a rise in body weight due to the retention of water.

Larson, Weir and Rowntree (quoted in Physiological Reviews, vol.2, 1922, page 158) produced water intoxication experimentally in dogs by giving excessive water with injections of posterior pituitary extract to inhibit excretion. The dogs showed symptoms of asthenia and restlessness, followed by diarrhoea, nausea and vomiting, excessive salivation, tremors and twitching, progressing to ataxia, convulsions and terminal coma.

Rogers and Megaw (Tropical Medicine 1944, p.466) list post mortem findings in heat hyperpyrexia:

Body temperature remains high and may even rise after death.

Congestion and petechial haemorrhages in various parts of the body, including the medulla and meninges.

Heart arrested in systole with cloudy swelling of the muscle.

Early rigor mortis.

Blood remains fluid and is darker than usual.

HYPERTHERM TREATMENT

Very exact information on the pathological and biochemical effects of heat can be obtained from a study of patients subjected to hypertherm treatment, with the proviso that these subjects may be expected to show more severe symptoms than young healthy men. A summary of the morbidity of hypertherm treatment over a long series of cases is given in The Lancet (7.10.1944, Wallace and Bushby).

The hypertherm conditions were:

Temperature 110° - 130°F.

Humidity 85% - 100%

The patient's temperature (presumably oral, though this is not stated) was maintained at 106.6°F. for eight hours under strict medical supervision, and with adequate chloride, glucose and oxygen if required.

The following table shows the pathological reactions:

Vomiting	31%
Circulatory collapse	17.7%
Jaundice	14.8%
Incontinence	5.2%
Death	1.2%

Vomiting persisted on an average for forty-eight hours.

Jaundice was of short duration, usually commencing one to three days after the treatment, and lasting two or three days only. It was suggested that the jaundice was due to chloride deficiency, but pushing chlorides did not lessen the incidence. This is in conformity with my own findings, for I had no cases of jaundice in spite of profound chloride deficiency (judged from urinary chloride output) being present in many patients.

The two deaths in the series were due to circulatory collapse preceded by rapid respiration, low blood and pulse pressure, and incontinence. The circulatory collapse (17.7%) was entirely abolished in a second series of seventy cases in which prodromal symptoms were at once treated by the administration of oxygen and carbon dioxide, which supports the suggestion that the cause is failure of the respiratory centre.

Previous theories had postulated a reduction in blood volume due to dehydration, and myocardial failure. The former was disproved by dye methods, and the latter shown to be at any rate unlikely by a series of electrocardiograms, showing normal curve pattern and amplitude.

In the whole series plasma proteins fell and there was a rise in blood urea (c.f. Rowntree,

Review of Physiology 1922, quoted above).

The clinical features, apart from those noted above, and common to practically all cases were:-

Mental dulness (probably, it is stated, due to cerebral anoxia, though my personal opinion is that the severe conditions and bodily hyperpyrexia were enough to account for this).

Cyanosis.

Brown et al. (J.C.I. 22.1943, p.471): These workers also produced a series of hypertherm cases. They found that cases of circulatory collapse (which resulted in 1% mortality in the series) were greatly reduced in number after adequate chloride and water were provided.

Collapse was associated with absence of sweating, anuria, pallor and chill of the extremities. The rectal temperatures rose to 41.5°C. (106.7°F.) or higher, with a fall in blood pressure. The fatal cases progressed to a maniacal state and a terminal coma.

They found oxygen and carbon dioxide of temporary benefit only.

A rise of serum proteins to over 7000 mgm.% was dangerous, as was any appreciable change in the serum specific gravity (normal 1.0255 to 1.0290).

Wilson (Journal of American Medical As-

sociation, Feb. 17, 1940, vol.114, 7) describes four fatal cases of heat stroke, three after hypertherm treatment and one "natural". The post mortem findings in common to all four were:-

Congestion and general extravasation of the whole central nervous system.

Cerebral oedema.

Petechiae in brain.

Extensive haemorrhages in the ventricular septum near the bundle of His.

(c.f. Lancet report, page 23)

Indications for abandoning hypertherm treatment are summarised below, since they are obviously also of value in estimating the severity of heat cases:-

1. Disorientation.
2. Fall in systolic blood pressure below 100 mm.
3. Pulse rate over 160 p.m.
4. Respirations over 50 p.m.
5. Temperature 107° for fifteen minutes.
6. Restlessness and violence.
7. Coma.
8. Pallor following previous cyanosis.
9. Persistent vomiting.

CLINICAL SUBDIVISIONS OF HEAT EFFECTS

Air Vice Marshal Morton (personal communication, and Transactions of the Royal Society of Tropical Medicine and Hygiene, May 1944) divides heat effects into 3 types:-

Heat Syncope
Heat Exhaustion
Heat Hyperpyrexia

This is the generally acknowledged classification, though heat syncope may be considered an early or prodromal form of heat exhaustion.

Heat Syncope

This, and heat exhaustion, is commoner in asthenic subjects, usually young, and particularly affects men recently arrived in the tropics.

The essential features are dizziness and fainting. There is no upset of the chloride balance, urinary chlorides are normal, and the usual signs of syncope - pallor and low blood pressure - are present. Recovery is rapid and complete on transference to a lower temperature.

The condition is common in this country, during sudden heat waves or in crowds, especially if the air is still.

Heat Exhaustion

Heat exhaustion may occur ab initio or as a sequel to inadequately treated heat syncope, and is the commonest type of case to reach hospital - one hundred of my series could be classed as heat exhaustion as against eleven heat hyperpyrexia.

Prodromal Symptoms (B.G. Maegraith, B.M.J. 28.6.52):

A short prodromal period of several days may be present, in which the symptoms chiefly noticed are headache, anorexia, nausea and slight vomiting, with fleeting cramps.

The clinical signs are:-

Collapse and pallor.
Restlessness.
Anxiety.
Rapid shallow respiration.
Low blood pressure.
Rapid, running pulse.
Profuse perspiration.
Skin cold and clammy.
Facies pinched and dehydrated.
Mouth temperature normal, while the rectal temperature may be raised.
Reduction in urinary volume even to complete anuria.
Diminution in blood and urinary chlorides.
Vomiting.
Cramps.
Death due to circulatory failure in a state rather resembling cholera sicca.

In severe cases (Maegraith supra) there is considerable haemoconcentration up to 150% increase in red cell counts and corresponding increase in blood viscosity.

The urine may contain albumen and hyaline and granular casts. The blood urea is raised moderately except in those cases of anuria progressing to uraemia when the values are very high.

The concentration of chlorides in the sweat is raised - large amounts of chloride are therefore lost in the profuse sweating which occurs, and the vomiting, which may be severe and intractable, still further reduces the body chloride.

Heat Hyperpyrexia.

This is a much more serious condition stated to occur most frequently in heavily built, middle-aged subjects. Alcohol is a predisposing factor (Rogers and Megaw, Tropical Medicine 1944, p.465), and Morton states that it frequently attacks men who have spent several years in the tropics. (c.f. Heat exhaustion and syncope, supra). Haldane states that the temperature and condition of the layer of air between the skin and the clothes is the determining factor in the production of hyperpyrexia. He quotes as predisposing causes:-

Fatigue
Alcohol
Over-crowding
Tight clothing
Bad ventilation
Insufficient fluid intake

and states that young people are less susceptible than older as the cardiovascular system is more elastic and flexible in youth.

Cases of heat hyperpyrexia are defined as having a rectal temperature of 107°F. or over, (Ladell, Waterlow and Hudson, Lancet 14.10.44), but I myself have taken 106° as the critical temperature. Maegraith and Morton both confirm that a prolonged and continuous exposure to heat is an essential factor; for instance, Maegraith notes that it is uncommon in stokers working in temperate climates (B.M.J. 21.6.52). His list of predisposing factors is the same as Haldane's (supra) and in addition includes hyperthyroidism and febrile illnesses.

Prodromal Symptoms:

Dizziness.

Headache (almost invariable, according to Morton: personal communication).

Drowsiness and mental confusion or forgetfulness.

Anorexia is occasional, but not so frequent as in heat collapse, and vomiting, I have found, is uncommon.

The clinical signs of a fully developed attack are very constant:-

Hot, dry skin.
Oliguria or anuria.
Dry tongue.
High and rising rectal temperature.
Mental confusion.
Convulsions and coma.
Incontinence.

The sequelae of hyperpyrexia (Willcox) include headaches, lassitude, irritability, insomnia, lack of concentration and defective memory. Occasionally there is blindness and minor paralysis.

Martin adds to this list epilepsy and dementia, and mentions the occasional difficulty in diagnosis between heat hyperpyrexia and pontine haemorrhage.

Various other classifications and nomenclatures have been suggested, but my personal opinion is that Morton's description given in lectures preparatory to tropical postings is accurate, adequate, and above all easy to recognise, so that treatment can be started without delay.

Lee (1940) describes super-dehydration due to excessive loss of fluid by the skin, giving symptoms of thirst, oliguria and dyspnoea with haemo-concentration, nervous disturbances, cyanosis and

a dry, shrunken skin, progressing to uraemia and hyperpyrexia. This is merely a description of untreated heat exhaustion terminating in the state resembling cholera sicca mentioned above. Wilcocks (Practitioner 1941, p.463) describes the blood in such cases as being very dark and fluid at post mortem.

Wolkin, Goodman and Kelley (Journal of American Medical Association, 19.2.1944) use the term Thermogenic Anhidrosis to describe all terms of heat effects, and divide them much as other authors into heat exhaustion with and without muscular cramps, and heat-stroke, i.e. heat hyperpyrexia.

They note that cases of heat exhaustion with a dry, cold skin frequently give a history of profuse sweating over the previous few days (c.f. Lee, quoted supra).

They also state that the dryness is over the trunk and limbs and not the head. The skin was painted lightly with tincture of iodine, allowed to dry, and then dusted with starch - areas of moisture could be seen at a glance by the development of the typical blue colour of the starch-iodine absorption compound.

They describe eight cases, none of which

appears to have been seriously affected.

Maegraith (B.M.J. 21.6.1952) adds Thermogenic Anhidrosis to Morton's other three terms, restricting it to cases of dry skin (again noted as being over the trunk and limbs) without high fever. He considers it may be due to local exhaustion of sweat glands, and describes the skin changes as an occluding hyperkeratosis followed by the production of intra-dermal vesicles. The condition is practically always associated with severe prickly heat, and typically attacks troops who have been exposed to high temperatures for some considerable time.

Rogers and Megaw (Tropical Medicine, J. & A. Churchill, 1944) subdivide cases into heat-fever, heat exhaustion or heat shock and heat cramp.

Jewell and Kauntze have made an impressive collection of synonyms, which again might well be dispensed with for a universal nomenclature:

Heat hyperpyrexia
Insolation
Siriasis
Thermic fever
Diathermasia
Sun fever
Sunstroke
Heat stroke
Heat exhaustion
Sun traumatism
Heat prostration

Willcox describes three types of heat hyperpyrexia, (Price's Textbook of Medicine).

1. The gastric type with a raised rectal but low axillary temperature and frequent vomiting.

2. The gastro-intestinal type in which there is watery diarrhoea as well as vomiting and the axillary temperature is high (c.f. Bard's experimental work quoted supra).

3. The nervous type in which there are cerebral symptoms.

He stresses the early loss of knee jerks, which I found a useful guide in differentiating from cerebral malaria.

The whole subject of heat effects has been exhaustively investigated by Ladell, Waterlow and Hudson (Lancet 14.10.1944) in Shaiba, which has a typically desert dlimate - high day temperatures with low humidity and negligible rainfall, and a sharp drop after sunset.

The urinary output of healthy men was about 500 ccs. daily and the authors found that a daily intake of 7½ litres was required to maintain health at midsummer (117°).

No biochemical abnormalities were observed in a large number of healthy controls.

Twelve cases of hyperpyrexia (criterion - rectal temperature 107° or over) are described. All recovered.

Nine cases (75%) were over the age of 30, as contrasted with 40% in the total series of all cases of heat effects. No case gave a history of excessive exposure - one, of alcohol. Abdominal and tendon reflexes were absent in all but three of the cases.

The authors have subdivided heat exhaustion into two types, one occurring in the early part of the hot season and the other near the end. No other author, so far as I have been able to ascertain, has made this differentiation, though the following

table marks the contrast strongly. Maegraith, for instance, makes no mention of it in his recent article (B.M.J. 28.6.1952). In India the picture is complicated by the varying conditions - the dry heat in the early part of the summer and the rising humidity on the approach of the monsoon associated with a drop in temperature. My cases, as I shall show, had a sudden onset in late May, were numerous in June and fell off rapidly in July and August. They appear to conform to Type I of Ladell's series, as would be expected, and the incidence follows closely the mortality figures given by L. Rogers (India) quoted by Rogers and Megaw in their text book, (Rogers and Megaw, Tropical Medicine, 1944, p. 464).

SUBDIVISION OF HEAT EXHAUSTION

Signs and Symptoms	Type I (early) 45 cases	Type II (late) 55 cases
1. Vomiting	73%	4%
2. Cramps	70%	4%
3. Defective sweating prior to attack	13%	87%
4. Frequency	Nil	82%
5. Dry skin	5%	40%
6. Prickly heat	7%	80%
7. Dehydration	50%	2%
8. Cases with pulse pressure less than 30 m.m. mercury	46%	2%
9. Colour of skin	Pale	Red
10. Average pulse rate	90	76
11. Increase of pulse on standing	40	20
12. Blood pressure	Low	Average
13. Average rectal temperature	100.6°	100.9°
14. Average mouth temperature	98.2°	99.5°
15. Urinary chlorides	0.98%	3.47%
16. Blood urea mgm.%	103	26

ACCLIMATIZATION

The subject of acclimatization is obscure and its mechanism is really not yet known.

Presumably the sweat glands become attuned to the extra work expected of them and cooling by evaporation becomes more effective.

It has been suggested that the fall in body weight common in tropical conditions is also a factor - the cooling area remains virtually unchanged, while the mass to be cooled is reduced.

Eichna, Bean, Ashe and Nelson (Bulletin of Johns Hopkins Hospital, Jan. 1945) have stated that acclimatization begins within a day or two of entry into tropical conditions, and is complete by the tenth day. They point out, however, that s second group of subjects, who entered the tropical environment at the same time but who rested until the tenth day and then commenced manual work, showed little or no signs of acclimatization. After the tenth day, when work commenced, acclimatization also commenced and followed the same pattern as in the first group.

P.A.I. Force reports (1944) state that three-quarters of the cases of heat effects dealt with in 1942 occurred before, during and immediately after disembarkation.

The sharply marked contrast in the two types

of heat exhaustion shown by Ladell et al. (Lancet, 14.10.44) supports the theory of increased sweat gland response to increased demands. The cases of Type I can be said to be in a state of incomplete or inadequate acclimatization, occurring as they did in the early part of the hot season. The late cases, on the other hand, suggest sweat gland damage due to the high incidence of prickly heat and skin lesions, and consequent fatigue of the glands. It is noted that 40% of the sufferers had dry skins, as against 5% in the first type.

Type II appears in some respects to be similar to Thermogenic Anhidrosis, described by Maegraith (B.M.J. 21.6.52, p.1346).

Maegraith also mentions a state of hypersensitivity (p. 1345) caused by loss of acclimatization (which may be quite rapid) on removal from tropical conditions and sudden re-introduction to high temperature.

I was not aware of this theory at the time when material was being collected, so that I am unable to say if many of my cases had recently returned from hill leave, but in any case the period of leave would be too short for this to be of practical importance.

SECTION II

LOCAL CONDITIONS AND CLINICAL MATERIAL

The following series of cases of heat effects, one hundred and eleven in all, occurred in Cawnpore in the summer of 1945, when the author was medical specialist at a Royal Air Force Hospital.

All the cases were British servicemen, and were drawn from a large Royal Air Force maintenance unit and aerodrome some five miles outside the city, from a battalion of the King's Own Royal Rifles stationed at the Wheeler Barracks, and from several smaller Royal Air Force units and Army Ordinance units.

The Maintenance Unit and Aerodrome were well equipped with sick quarters and had provision for treatment of minor cases of heat effects. We found therefore that from them we drew two chief types of case - extremely severe, requiring skilled nursing and all hospital facilities, and other less severe cases who were convalescent but required hospitalization before being sent on sick leave.

The latter cases have not been included in the series since their stay in hospital provided no features of interest and their condition caused no anxiety.

Mild cases of heat syncope, of course, never came into hospital at all, but were dealt with at

their units.

The Wheeler Barracks and the small local units had Medical Inspection room facilities only, so that all cases, however mild, found their way to hospital. Most of the Ordinance personnel had spent some years in the tropics and their conditions of work were satisfactory. The Wheeler Barracks, on the other hand, was manned by a mixed battalion, partly of troops who had seen long service in Burma, and partly of men newly arrived from the United Kingdom and undergoing field training. The incidence of heat effects among the latter was high.

In addition, Cawnpore is an important road and rail junction, and several cases were sent to us from the railway station and from the local transport camp in the cantonment. Most dramatic of all, an officer arrived in the last stages of collapse driving a "jeep", having fought a steadily rising temperature over some fifty miles of blistering road, and fully aware that his one chance was to reach us.

The town is in the United Provinces, latitude 26° 24' N. and has a desert type of climate, temperatures rising during May from 100°F. to 110°F. and to 120°F. during June. In early July the temperature drops steeply, since heavy rain-clouds form, but the humidity rapidly rises and the monsoon

usually breaks about 15th July. There were only two heavy showers between 1st January and 17th July, when the rains came in 1945. During the very hot weather there is a rapid drop in temperature at sunset, the night temperature being at least 30°F. lower.

HOSPITAL CONDITIONS

The main part of the hospital dates from the nineteenth century, replacing the pre-Mutiny building, whose foundations are still preserved nearby. It is well built for its purpose, with thick walls, double balconies, and high roofs, giving very adequate air space.

Ceiling fans are plentiful, and at intervals down the Ward are so-called "Cawnpore coolers" - large fans which suck air through layers of a thick grass called Kuss-Kuss, which is of the consistency of dried heather. The kuss-kuss is kept moist by running water. These we found most effective up to the end of June, but with the rapidly rising humidity of July they lost their effectiveness.

We found one room which could be converted into a heat treatment centre for cases of hyperpyrexia. There were no outside windows. We obtained two small air conditioning sets, and some small table fans, and fitted a false roof to reduce the volume with which the air conditioners would have to deal.

A charpoy - an Indian bed of interlaced tapes on a frame - was installed to allow free air access to all parts of the body.

MEDICAL PREPARATIONS

In the expectation of numerous cases of heat effects, I made all preparations before the really hot weather started. Supplies of ice were promised, and were in fact invariably forthcoming. Supplies of saline and glucose saline were laid in, and I also prepared a solution containing sodium chloride gr.40, calcium chloride gr. 4, in 10 ccs. This was sterilised locally and kept in rubber capped bottles of the type used for blood culture media. When required, 10 ccs. were withdrawn through the cap with sterile precautions and introduced into a pint container of normal saline. This gave us a hypertonic solution which was used in the initial stages of intravenous therapy.

Lemon flavoured saline drinks were provided for all cases in the hospital and fluid intake and output charts were maintained in all febrile cases, especially the heat cases under review.

Drugs kept on the trolleys along with the above solutions, giving sets, etc., included ampoules of quinine, sodium niketamide, calcium gluconate.

TRAINING OF STAFF

Among the entire medical staff only our Commanding Officer, Wing Commander Lewis, had seen previous tropical service, and we got many valuable hints on treatment from him. Otherwise our knowledge was entirely theoretical.

All severe cases were sent at once to the Ward without the usual hospital formalities accompanying admission. Officers, sisters and orderlies were warned that such cases were to be treated as most urgent medical emergencies.

During the time at which cases usually came in - the early afternoon - a medical officer was permanently present in the Ward.

The laboratory staff were instructed that blood slides from hyperpyrexial patients took precedence over all other work.

TREATMENT

1. Heat Exhaustion

These cases varied in severity from minor degrees, some of which recovered in the cooler air of the Ward on saline by mouth, up to the most severe type of algid collapse, with pallor or cyanosis and an all but impalpable pulse.

All algid cases had immediate blood slides (thick and thin) examined for malarial parasites. The urine was examined for chloride and intravenous therapy at once commenced. The saline was given at Ward temperature.

Algid cases received their first pint very rapidly and we found that it was best to use normal glucose saline. After about ½ pint had been given 2 ccs. of sodium niketamide were injected intravenously.

Subsequent administration of glucose saline (hypertonic) was continued until the urinary chlorides appeared adequate, after which normal glucose saline was substituted until the pulse volume was satisfactory and the colour good. Well sweetened coffee was found to be a good restorative in the later stages of treatment.

All these cases had complete bed-rest for at least a week or ten days, and were sent on sick

leave to the hills.

Less severe cases required a varying amount of intravenous fluid, but we usually started with one pint of hypertonic glucose saline, given rapidly, followed by normal saline or glucose saline at a slower rate.

Mild cases were given large quantities of flavoured saline by mouth, and rapidly recovered, causing no anxiety.

After the active treatment the patients were kept warm, encouraged to drink, and carefully watched for signs of relapse, indicated by a return of pallor and dyspnoea, and a drop in blood pressure. It is to be noted that no case, once adequately treated, showed signs of relapse.

The more severe cases suffered from acute fear in their collapsed condition, and their subjective improvement during treatment was most dramatic.

2. Heat Hyperpyrexia

These cases were at once admitted to the treatment room (temperature 70° - 80°F.).

Bloodslides (thick and thin) were taken immediately and examined for malarial parasites.

No covering was used below the patient, who lay

on the webbing of the charpoy. One table fan was placed vertically under the charpoy, giving a constant upward draught. Finely crushed ice was applied to the thorax, abdomen and legs, the patient covered with a single sheet wrung out of iced water, and fans were directed at the saturated material.

All temperatures were taken rectally on admission and at ten minute intervals during treatment, care being taken to avoid a too rapid drop, so frequently followed by pneumonia or cardiovascular collapse. Iced enemata, recommended in some textbooks, were not employed, as the other measures appeared to be sufficient to control the fall in temperature, and no check could then have been kept on progress by rectal temperature readings.

Favourable signs included a steady fall in temperature, a reduction of the delirium in which several patients were admitted, or signs of returning consciousness from the coma which affected others, return of knee jerks and a resumption of sweating. I had not then heard of the starch-iodide method of observing re-establishment of sweating, but we endeavoured to keep the forehead dry in the prevailing confusion of ice, water and damp sheets, to note the first signs of secretion.

The majority of patients were very restless, and

one required repeated minimal doses of pentothal sodium during treatment.

The question of intravenous quinine was considered but as slides could be examined with considerable rapidity it was decided not to employ it, since the drug itself when given intravenously is not without danger.

One case admitted as heat hyperpyrexia was found to be malarial and responded to specific treatment. It is not included in the series.

When the temperature had dropped to the region of 104°F. treatment was temporarily checked and an effort made to reduce the temperature more slowly. By this time the majority of patients were lucid, or at any rate quiescent, and at least two pints of isotonic glucose saline were given fairly slowly.

The urine was not tested in the early stages, as this would have required catheterization, which would have wasted vital time, but a specimen passed after consciousness was regained gave a guide to the amount of saline required.

After-treatment required great care. The temperature was slowly lowered, and here the two chief dangers were relapse and circulatory collapse. Sodium niketamide intravenously was used at once as soon as the slightest suggestion of impending col-

lapse made itself evident.

Relapses were treated by a repetition of the original treatment, but in only one case did the condition cause anxiety.

The patients spent the whole day in the cool room if possible, and during the first night on transfer to the Ward they did not have a mosquito net, so that their condition could be carefully watched.

Rectal temperatures until the evening were taken every half hour, copious drinks supplied, and absence or presence of sweating noted.

CONVALESCENT TREATMENT

Cases of heat syncope were allowed up the day following.

Cases of heat exhaustion spent at least a week in hospital. Blood pressure was measured daily and urinary chlorides estimated night and morning.

A careful cardiological examination was carried out before discharge, and the more severe cases were sent on varying periods of hill leave, and then returned to light duties at their stations until acclimatization was re-established.

Cases of heat hyperpyrexia were confined to bed for at least a week after the temperature had reached normal, and then allowed up cautiously. The condition of heart and lungs was watched and before discharge the central nervous system was systematically examined.

All hyperpyrexial cases were sent on hill leave and two were repatriated (Cases II and III).

The question of repatriation of all cases was considered, but, after consultation with a civil surgeon of many years' tropical experience, it was decided to treat each case on its merits. A close follow-up of the subsequent history of these cases was not possible, since climatic conditions had changed by the time many of them returned from sick

leave; others came from distant units and were admitted to us in transit.

We had, however, no case either of heat exhaustion or hyperpyrexia readmitted for the same complaint during the whole summer.

SUMMARY OF CASES OF HEAT EXHAUSTION

One hundred cases of heat exhaustion and collapse were treated in all, seventy-two from the Army and twenty-eight from the Royal Air Force. As has been mentioned before (page 39), the Royal Air Force units in the neighbourhood had facilities for dealing with mild cases at Station Sick Quarters. The Army depended entirely on the hospital for all treatment, so that a larger proportion of minor cases were seen.

Detailed notes are given below of cases which were classed as severe, nine in all, while moderate and mild cases have been dealt with statistically. The troops have been arbitrarily divided into men with over one year's tropical service, and those who had not previously experienced a hot season, and an attempt has been made to compare the two groups, in relation to special stress, severity of attack, etc. The Army figures are more reliable, for the reason stated above, viz. that minor Air Force cases were not sent to hospital.

ARMY (ALL UNITS)

	Tropical Service			
	Under 1 year		Over 1 year	
Total Cases treated	50		22	
Degree of Severity:				
Severe	3		2	
Moderate	16		8	
Slight	31		12	
Special Stress:				
Indoor work with inadequate air movement	4	8%	5	22%
Route marches	5	10%	0	
Battle exercises	6	12%	0	
Extra fatigues	2	4%	3	13.5%
Guard duty	4	8%	0	
Train journeys	2	4%	2	9%
Motor transport journeys	1	2%	2	9%
Firing range	5	10%	2	9%
Games	0		1	4.5%
No Special Stress:	21	42%	6	27%

INTERCURRENT DISEASE

Of the fifty cases in the first group, twelve (24%) gave a history of some intercurrent disease. Eight of these twelve had succumbed without any special stress, i.e. 66.6% as against the 42% in the total table.

In the second group eight (36%) gave a similar history, including four with no special stress - 50% against 27%.

The intercurrent diseases were of various types, including dysentery, tonsillitis and one severe dental abscess, but quite a high proportion was accounted for by respiratory infection of one kind or another.

PREVIOUS HISTORY OF HEAT EFFECTS

Six cases in the first group, and one in the second gave histories of recent recovery from previous attacks. It was noted that these cases tended to be more severe.

Severe	2
Moderate	4
Slight	1

CONSTIPATION

Eleven of the first group (22%) and two of the second (9%) gave a history of constipation for some days previous to their illness.

SPECIAL SYMPTOMS

Cramps were present in 14 (28%) of the first group and in three (15%) of the second. Only cases with fairly severe cramps, in which the patient himself volunteered the information on taking the history, are included. Many men when directly questioned admitted to vague cramp-like pains of short duration.

In several cases (e.g. VI and IX among the severe cases detailed later) true tetanic convulsions with accoucheur's hand were a feature. Calcium gluconate was administered intravenously, one case requiring three injections of 10 cubic centimetres.

No variation of frequency in cases early and late in the series (see Table page 36) was found.

CONDITION OF SKIN

	Degree			Total
	Severe	Moderate	Mild	
Hot and dry	0	10	18	28
Cold and dry	2	1	1	4
Hot and moist	0	4	14	18
Cold and moist	3	9	10	22

With reference to the theory of exhaustion of sweat glands shown by Ladell's table (page 36), the condition of the skin has been compared in early cases (May and June) and late cases (July and August). As the hot season is relatively short, striking results are not to be anticipated; nevertheless it is worth noting that in August, with the monsoon established, relative humidity 100% and everybody feeling unpleasantly damp, all six cases admitted had dry skin.

	Skin	
	Dry	Moist
May and June	15	35
July	11	5
August	6	0

SKIN LESIONS AND PRICKLY HEAT

Prickly heat was uncommon while the humidity was low, and became almost universal in late July. Skin abscesses and furunculosis also showed a rapid increase. No comparison is therefore possible in the cases under review.

URINARY CHLORIDES

The concentration of urinary chlorides appeared to vary with the severity of the attack, though this was by no means universal. Certainly no differentiation between early and late cases (see table page 36) showed itself.

ROYAL AIR FORCE (ALL UNITS)

	Tropical Service	
	Under 1 year	Over 1 year
Total Cases treated	22	6
Degree of Severity:		
Severe	2	2
Moderate	12	3
Slight	8	1
Special Stress	5	3
No Special Stress	17	3

PREVIOUS HISTORY OF HEAT EFFECTS

Two cases in the first group and none in the second had a history of previous heat effects; no special stress present in either case. Both cases were mild, but it was noted that the previous attacks had been so mild that neither was sent on sick leave.

INTERCURRENT DISEASE

There were two cases in the first group, none in the second. No special stress was present in either case.

PREVIOUS HISTORY OF CONSTIPATION

There were seven cases (30%) in the first group and one in the second.

SPECIAL SYMPTOMS

Cramps	Two only - one severe.
Epistaxis	One.
Tetany	Two - one very severe.

CONDITION OF SKIN

	Degree			Total
	Severe	Moderate	Mild	
Hot and dry	1	5	3	9
Cold and dry	0	2	0	2
Hot and moist	0	2	6	8
Cold and moist	3	6	0	9

As only four R.A.F. cases were admitted in July, no comparison between early and late cases is possible. Three of these, however, had dry skin.

DETAILS OF NINE SEVERE CASES

of

HEAT EXHAUSTION

Army	5
Royal Air Force	4

SEVERE CASES

Army - 5

CASE I

Pte. B.

Age: 19.

Tropical service: 6 months.

Admitted: 5.6.1945.

Stress: On guard during the night.
Parade 9 a.m.
History of constipation.

On admission: Cyanosed.
Algid.
Pulse 150. T. (oral) 95°.
B.P. 90/?
Anuria.

Treatment: Three pints hypertonic saline.
One pint normal glucose saline.
Hospital 8 days.
Sick leave 14 days.

CASE II

Cpl. P.

Age: 28.

Tropical service: 3 years.

Admitted: 5.6.1945.

Stress: Driving truck in convoy.

On admission: Very collapsed.

Pulse 120.

T. 97°.

B.P. ?

Skin cold with profuse sweating.

Severe cramps.

Periodic vomiting.

Almost complete anuria.

Chlorides less than 1 Gm./litre.

Treatment: Three pints hypertonic saline.

Three pints normal glucose saline.

Hospital 9 days.

Sick leave 14 days.

CASE III

V.

Age: 20.

Tropical service: Five months.

Admitted 26.5.1945.

Stress: Train journey.

Had had heat exhaustion in Jhansi 10 days ago.

A thin, asthenic type of man.

On admission: Pallid and collapsed - loss of consciousness.

Pulse 110. T. 96.4°.

B.P. unobtainable - later rose to 115/70.

Haemoglobin 140%.

Urinary chlorides less than 1 GM./litre.

Treatment: Three pints hypertonic saline.

Two pints normal saline.

Hospital 18 days.

Left to rejoin his unit with a recommendation for sick leave and ? repatriation.

Remarks:

It was impossible to get into a vein in this case as the vessels were so collapsed, so we cut down and inserted a cannula without requiring any local anaesthetic.

CASE IV

H.

Age: 29.

Tropical service: 11 years (Indian Army Ordinance Corps)

Admitted: 26.5.1945.

Stress: Nil obtainable.

Previous history of heat exhaustion in 1938 and again one week ago.

On admission: Skin hot and dry.

General condition very dehydrated.

Pulse 102.

T. 103.2°.

B.P. 108/80.

Urinary chlorides 2 Gm./litre.

History of constipation.

Treatment: Two pints hypertonic saline.

One pint normal saline.

Hospital 7 days.

Sick leave 21 days.

Remarks:

A severe case who responded rapidly.

CASE V

R.

Age: 25.

Tropical service: 1 month.

Admitted: 4.6.1945.

Stress: Nil ascertainable.

Sweated profusely the previous night. Profuse diarrhoea (ten motions in 12 hours) with no pathogenic organism present. Vomited. Severe cramps.

On admission: Weak and collapsed.

Skin clammy, very wet and cold.

P. 100.

T. 99°.

B.P. 105/70.

Treatment: Three pints of hypertonic saline.

Two pints normal glucose saline.

Remarks: This patient developed phlebitis and was in hospital for seven weeks before being sent on sick leave.

SEVERE CASES

Royal Air Force - 4

CASE VI

AC C.

Age: 25.

Tropical service: 2 years.

Admitted: 7.6.1945.

Stress: Nil ascertainable.

Had been "light-headed" for two days and had a sudden attack of severe cramp.

History of constipation.

On admission: Conscious.

Severe cramps, with typical accoucheur's hand and some laryngeal stridor.

Pulse 140. T. 101.4°.

B.P. 90/?

Very collapsed - face pinched and anxious.

Treatment: Two pints hypertonic saline.

Two pints normal glucose saline.

Oxygen.

Sodium niketamide and calcium gluconate intravenously.

Hospital 8 days.

Sick leave 21 days.

Remarks: Apart from the severity, the feature of this case was the tetanic condition.

CASE VII

AC C.

Age: 24.

Tropical service: 6 weeks.

Admitted 30.5.1945.

Stress: None ascertainable.

Previous history of rheumatic fever, and occasional fibrositis subsequently. There was also a doubtful history of hepatitis.

Five days' complaint of "thumping in head", giddiness and headache.

On admission: P. 88. T. 102°.

B.P. 120/80, dropping later to 80/?40.

Skin hot and dry with considerable prickly heat.

Urinary chlorides 2 Gm./litre.

Treatment: Two pints of hypertonic saline given slowly.

Sodium niketamide intravenously.

Remarks: This case was admitted in May. On account of the severe cardiac collapse, the previous medical history, and the early date at which symptoms had developed, it was considered that he was unfit for tropical conditions, and he was repatriated.

Hospital 27 days.

CASE VIII

Cpl. B.

Age: 23.

Tropical Service: 8 months.

Admitted: 25.6.1945.

History of this case is lacking.

On admission: Pale, cold, skin very wet.
P. 116.
T. 99.6°.
B.P. 90/?
Slight cramps.
Urinary chlorides 4 Gm./litre.

Treatment: Two pints of hypertonic saline.
One pint normal glucose saline.
Hospital 20 days.
Sick leave 14 days.

Remarks: Classed among the severe cases, as his condition on admission appeared to justify this. The pulse was almost imperceptible, pupils contracted, slight low delirium. Recovery was rapid under treatment.

CASE IX

P.

Age: 22.

Tropical service: 1½ years.

Admitted 21.6.1945.

Stress: None ascertainable.

Anorexia, headache, cramps, numbness of fingers and tinnitus.

History of severe constipation.

On admission: Skin very cold, clammy and pouring with sweat - patient conscious and apprehensive.

T. 100.2°. P. 100.

B.P. obviously very low but was not taken until later when the value was 135/90. The pulse had greatly improved by this time.

Severe cramps and typical accoucheur's hand.

Treatment: Two pints hypertonic saline.

Two pints normal saline.

Three injections of 10 ccs. calcium gluconate.

Hospital 9 days.

Sick leave 14 days.

Remarks: A second case showing well marked tetanic symptoms. Recovery rapid after the early severe phase.

DETAILS OF ALL ELEVEN CASES

of

HEAT HYPERPYREXIA

(with Charts for each)

HEAT HYPERPYREXIA CASES

CASE I

Sgt. R.

Age: 31.

Tropical service: 8 months.

Admitted: 5.6.45.

Temperature: 106.4°

History

Twenty-four hours' premonitory symptoms of headache and minor attacks of giddiness. He had been on clerical duties in a very hot room, with no fan. He noticed on 4th June that he had stopped sweating, but did not report sick.

5th June: Complained of giddiness, severe and increasing headache and became confused in speech.

On admission at 09.00 hours he gave the impression not only of inco-ordination of speech but of movement also. He rapidly became incoherent.

Salt intake had been adequate.

There was a history of constipation.

On examination the usual signs of hyperpyrexia were found.

Prickly heat absent.

Pupils dilated but reacted to light.

Nystagmoid movements of eyes.

T. 106.4°

P. 104 and feeble.

B.P. 90/60.

Muscular tremors.

Spleen not palpable.

Blood slides negative.

No incontinence.

Urine chlorides 1 Gm. per litre.

Subsequent Course

As this was our first case, treatment was, I consider, too drastic, and the temperature fell abruptly. Signs of collapse became obvious and he was removed from the cold room and treated for shock.

One pint of hypertonic and two pints of isotonic glucose saline were given, and 2 ccs. of sodium niketamide were administered intravenously twice.

By evening the temperature was rising but sweating was re-established, and the patient was coherent.

6th June: Urinary chlorides 3 Gm. per litre.
Temperature slowly rising.
B.P. 150/80.

7th June: Temperature reached 103°.
Sweating adequate.
Patient moved to bed beside Cawnpore cooler and the temperature fell steadily.

Stay in hospital 12 days.

Sick leave 16 days.

Returned to unit for light duties.

DISEASE.

HEAT

HYPERPYREXIA.

Notes of Case

Name SGT. R.

Age 31.

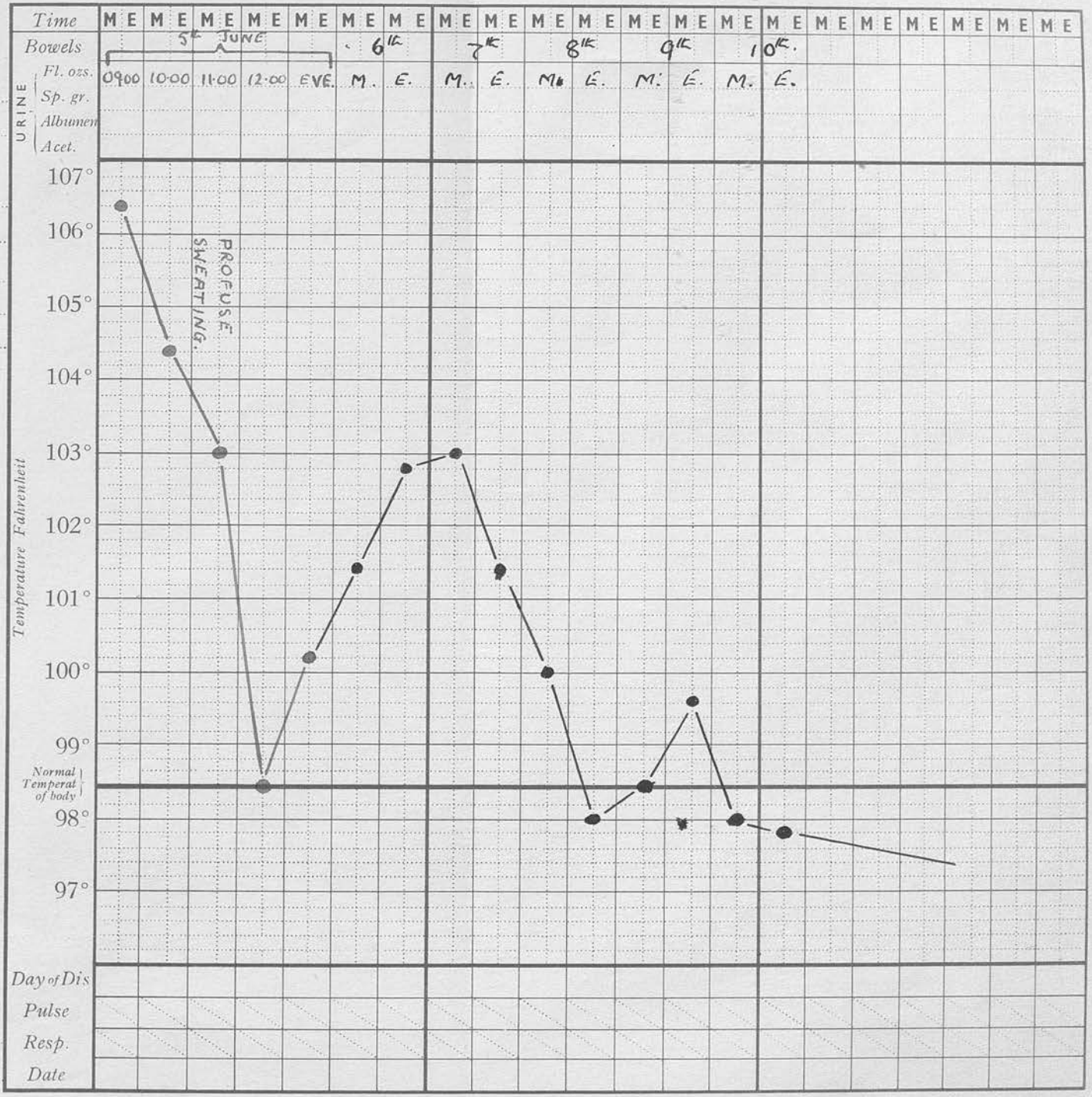
Diet

Case Book No. I

Date of admission

5-6-1945

Result CURE.



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CASE II

Sgt. C. R.A.O.C.

Age: 27.

Tropical service: 2 months.

Admitted: 6.6.45.

Temperature: 106°.

History:

This man had already had two slight attacks of heat exhaustion in his brief tropical service.

He had been working in a hot room with a hand-operated punkah only.

He complained of headache and dizziness and had noted absence of sweating for two hours. There was a history of constipation for two days.

He was coordinated but drowsy.

On Examination the skin was dry but the mouth and tongue were moist.

Prickly heat slight.

Knee jerks absent but no other abnormal C.N.S. signs.

T. 106°.

P. 120 - rather poor volume.

Resp. 24.

B.P. 100/70.

Spleen not palpable.

Blood slides negative.

No incontinence.

Urine: chlorides less than 1 Gm. per litre.

Subsequent Course

This was the mildest case in our series and responded easily to treatment.

Two pints of hypertonic saline were given, and the pulse improved. Sweating was re-established in less than an hour, and the subsequent progress was uneventful.

In view of his obvious failure to stand up to tropical conditions, as shown by the history, it was suggested to the Army authorities that he should be repatriated, and I understand that this was done.

Stay in hospital 9 days.

Sick leave 21 days.

DISEASE.

HEAT

HYPERPYREXIA.

Notes of Case

Name SGT C.

Age 27

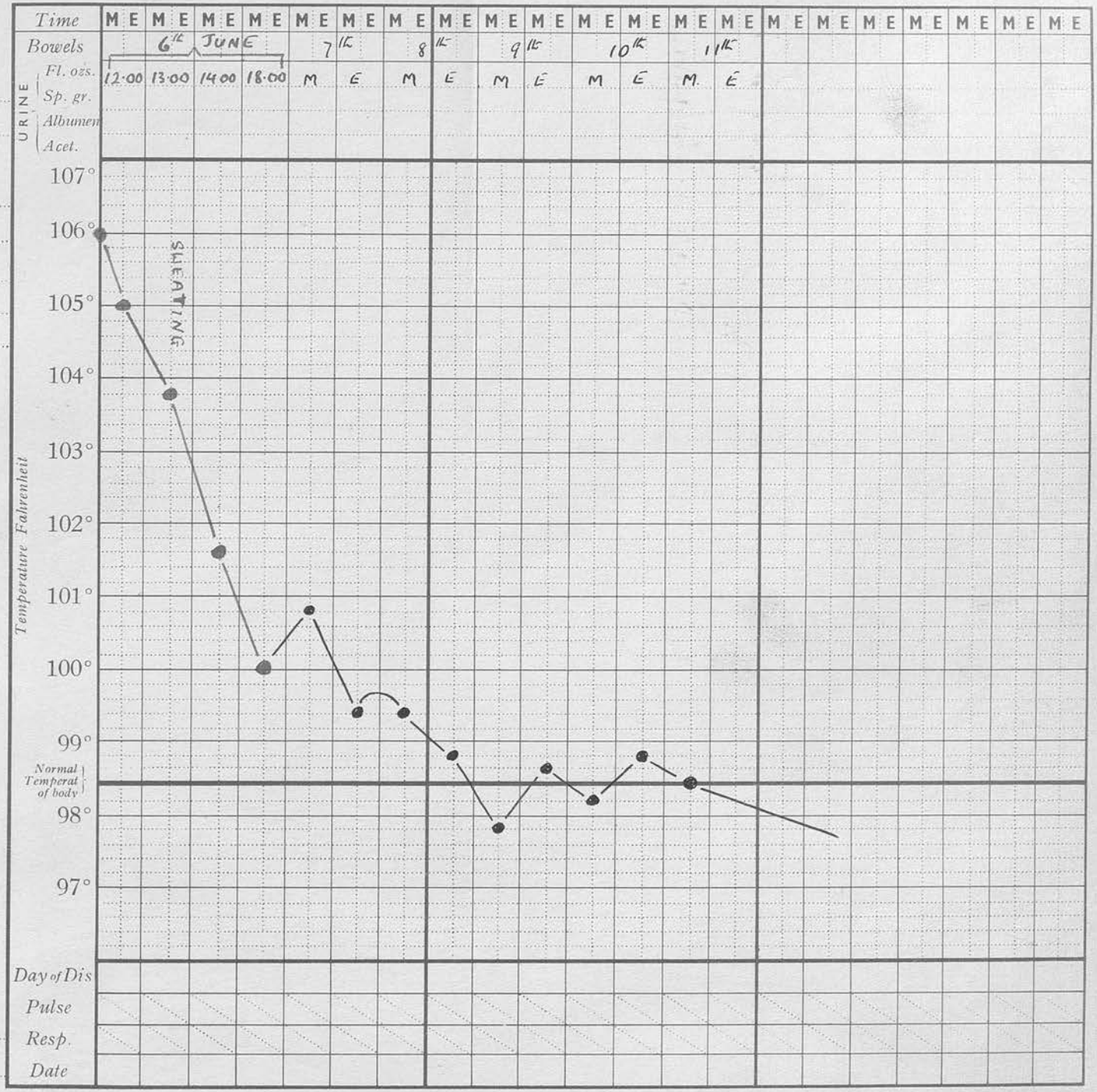
Diet

Case Book No. 2

Date of admission

6–6–1945

Result CURE



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CASE III

AC_2 C.

Age: 20.

Tropical service: 6 months.

Admitted: 6.6.45.

Temperature: 110.6°.

History

Went to bed in the afternoon and lapsed into unconsciousness.

On examination the skin was parched and very hot, cracking in the folds. The mouth and tongue were dry and the tongue swollen.

The patient was deeply unconscious, pupil reflexes were absent.

T. 110.6°.

P. 156 rapid and thready.

Spleen not palpable.

Blood slides negative.

Prickly heat present.

Subsequent Course

This was a most severe and resistant case. Shortly after admission he vomited, and there was double incontinence.

The coma gave place to convulsions, at first

spastic, and then clonic, during which the pupils were fixed and dilated. Following this the spastic state recurred, with head and eyes turned to the right and jaw tightly clenched. Bilateral plantar extensor response.

Vomiting recurred and there was now retention of urine, with bladder dilatation.

The temperature was lowered slowly, watching for signs of collapse, and, in view of the marked cerebral signs, it was decided to give six grains of quinine intravenously in spite of the negative slides. Sodium niketamide (2 cc.) was given just before, and the quinine administered as slowly as was possible in the difficult circumstances.

The niketamide was repeated twice.

In an hour the patient was quiet enough for a drip to be fixed and three pints of hypertonic glucose saline were given, the first two fairly rapidly. The temperature was then 103°, but apart from slight reduction in the spasticity the patient showed no signs of returning consciousness. A catheter specimen of urine showed less than 1 Gm. of chlorides per litre.

The temperature now fell rapidly to 99° and there were signs of collapse, so the patient was moved to the Ward and shock treatment instituted.

In two hours the temperature had risen to 101° and there were signs of return of sweating.

Further and more severe clonic spasms raised the temperature to 102° (pulse 132, but now of better volume).

From this time there was a gradual improvement, but consciousness was not regained for 36 hours after admission. Frequent blood slides were returned negative, and no further quinine was given.

Stay in hospital - 21 days.

Sick leave - 21 days.

Recommended for transfer to U.K.

CASE IV

Major G.
Age: 39.
Tropical service: 3½ years.
Admitted: 7.6.1945.

This officer was driving alone in a Jeep. No history was ascertainable on arrival as he collapsed on reaching the hospital gate, but subsequently it was found that his attack had begun acutely with headache and rapid cessation of sweating.

Having spent some years in the tropics he recognised the symptoms and, opening the windscreen, he drove at top speed to Cawnpore. His memory of the last few miles was extremely hazy and his condition on admission made us wonder how he had driven at all.

T. 105.5° rising to 106.4°.
Skin dry and hot.
Tongue dry and rather swollen.
Mental condition somewhat disorientated.

As no medical history was available, he was given i.v. quinine on admission, but no parasites were found on several examinations.

Urinary chloride normal.
Knee jerks weak but just present.

DISEASE.

HEAT HYPERPYREXIA

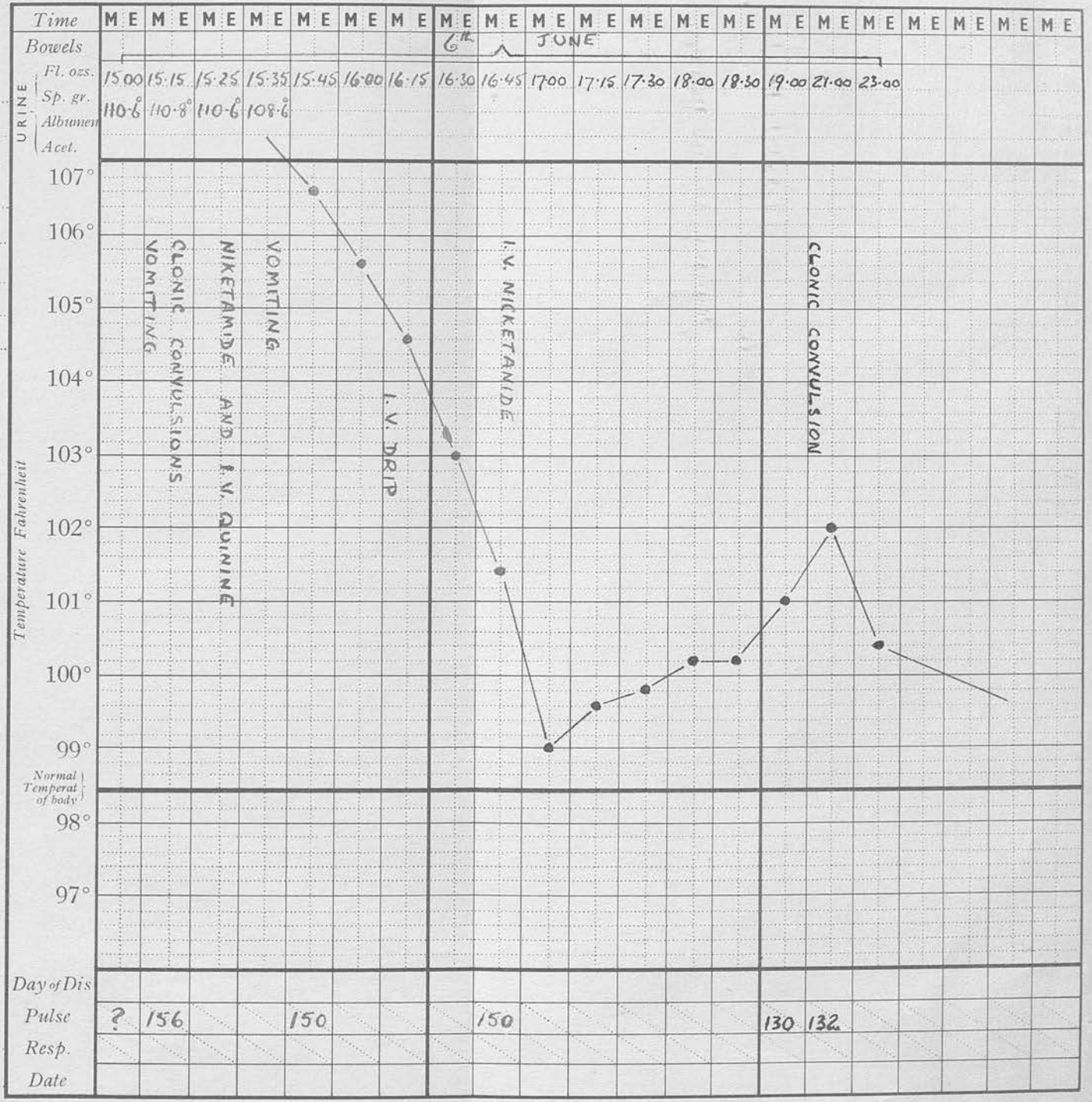
Notes of Case

Name A.C.2 C.

Age 20

Diet

Case Book No. 3



Date of admission 6-6-1945

Result CURE

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Subsequent Progress:

Very satisfactory response to treatment.

No intravenous saline was required, and he was discharged from hospital in ten days to rejoin his unit.

We considered this a case which was well acclimatized to a tropical environment, in which hyperpyrexia had developed owing to very severe conditions. This would explain the rapid recovery.

DISEASE.

HEAT

HYPERPYREXIA.

Notes of Case

Name MAJOR G.

Age 39

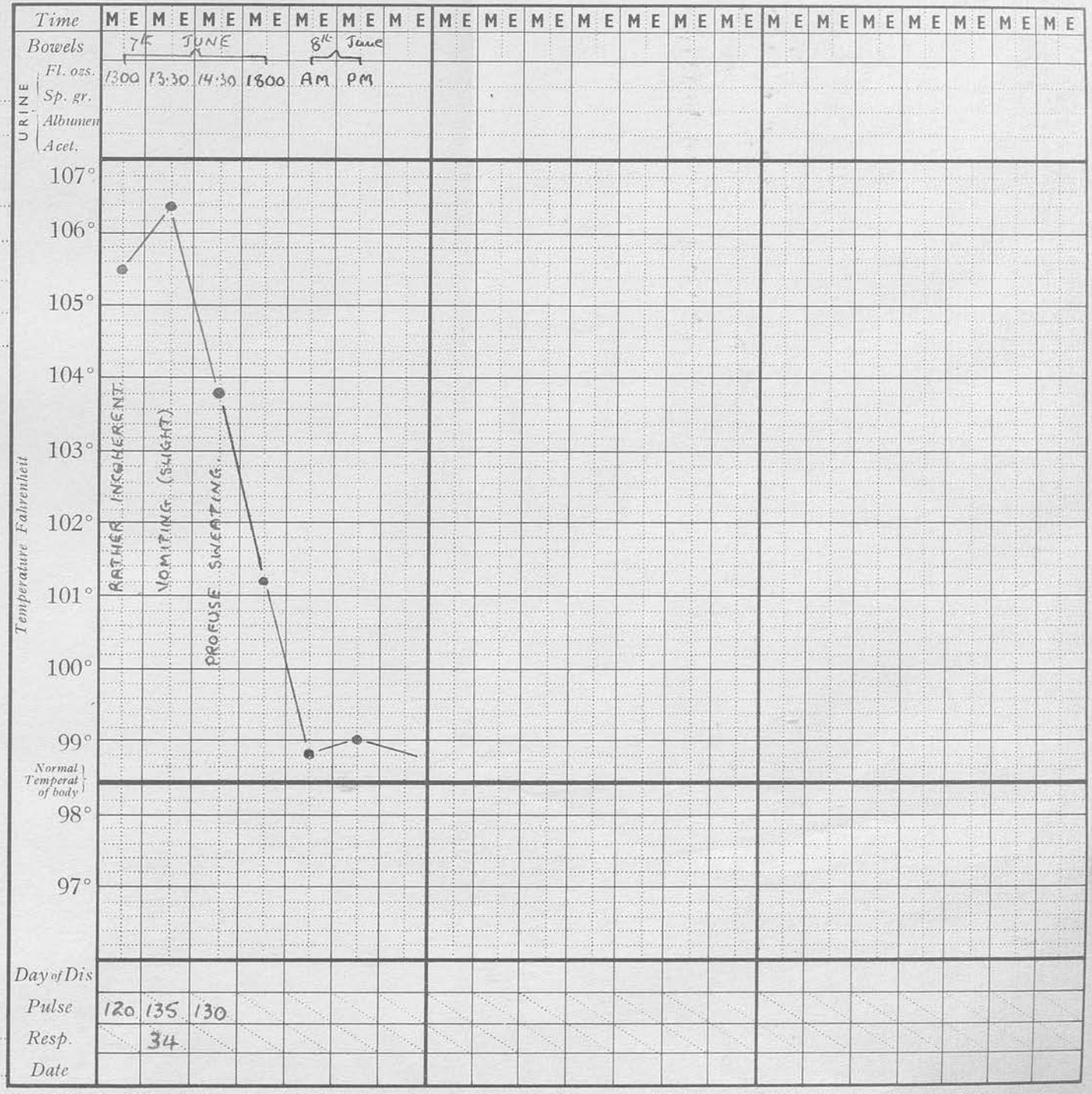
Diet

Case Book No. 4

Date of admission

7/6/1945.

Result CURE



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CASE V

Pte. D. K.O.R.R.

Age: 23.

Overseas service: 3 years.

Admitted: 10.6.45.

History

Was found comatose in his barracks in the late afternoon and admitted at 6 p.m.

Subsequent interrogation established that he had been feeling sick and giddy since the morning, with fairly severe headache. He had not noticed cessation of sweating. There was no history of special exposure.

During his previous tropical service in Burma he had had one slight febrile attack, but no typical malaria.

On admission: Skin hot and dry.

No prickly heat.

Tongue beefy, red and dry.

Conjunctivae very congested.

Mental condition - semi-conscious but not violent. Kept up a low moaning, with slight convulsive movements, chiefly in the legs, but a facial twitch was noticed from time to time.

Incontinent.

Knee jerks absent, and legs spastic.

T. 106.5°.

P. 120 - full and bounding.

Blood slides negative.

No history of constipation.

Treatment

Rapid response to treatment, sweating re-established in two hours. Two pints of hypertonic saline.

Subsequent Progress

A very satisfactory case, which responded quickly. The temperature dropped steadily and convalescence was rapid.

Hospital 9 days.

Hill leave 16 days.

DISEASE.

HEAT HYPERPYREXIA

Notes of Case

Name PTE D.

Age 26

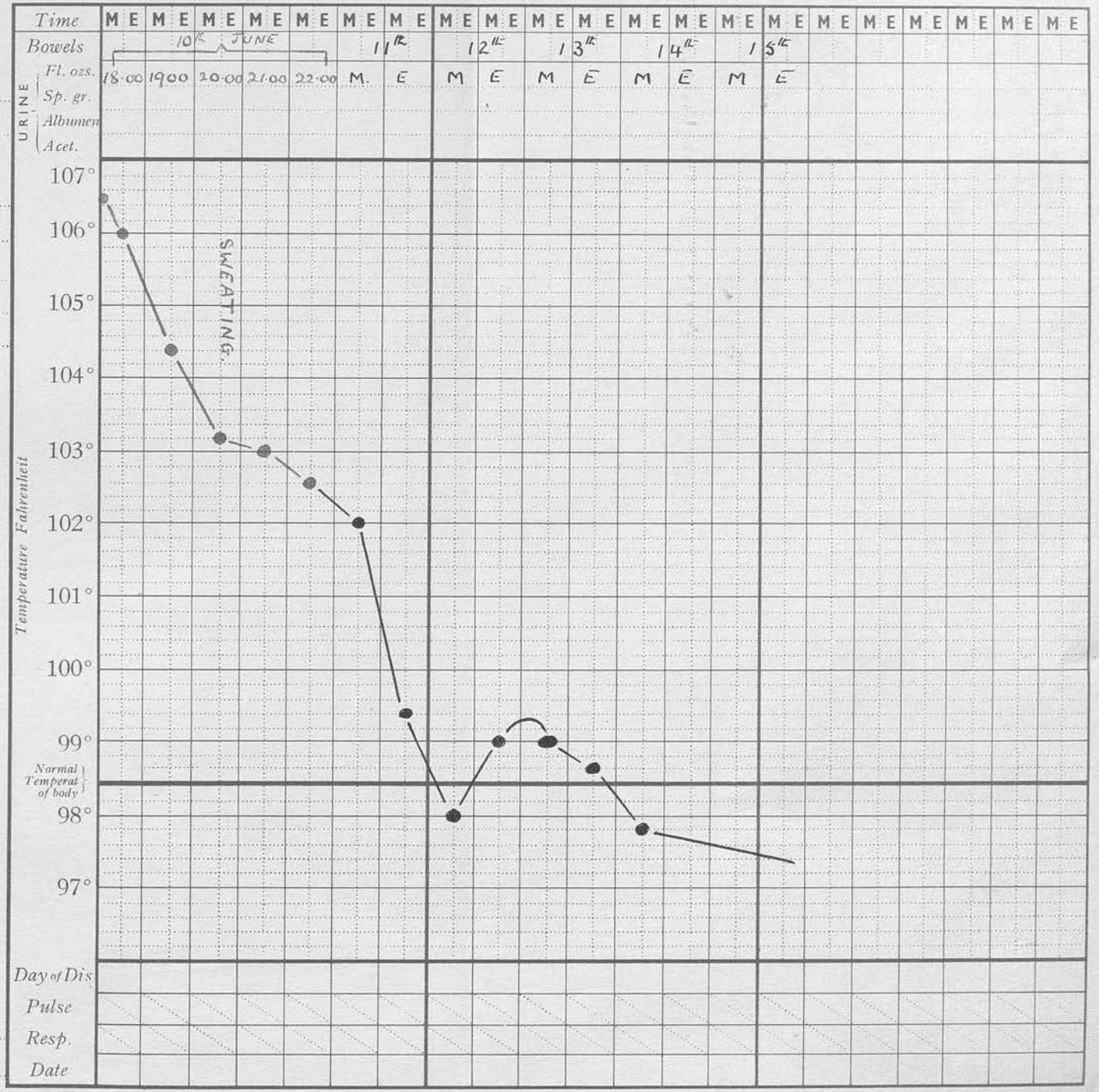
Diet

Case Book No. 5

Date of admission

10-6-1945

Result CURE.



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CASE VI

Cpl. P.
Age: 29.
Overseas service: 6 weeks.
Admitted: 13.6.45.

History

This patient presents a rather confused picture, but was nevertheless a true heat hyperpyrexia. He was admitted by the Railway Medical Officer at Cawnpore as an acute appendix. At this time the midday shade temperature was 116° and in the theatre at the time of operation (8 p.m.) was 105°. Since the sterilizing room communicated directly with the theatre (no intervening door), the relative humidity must have been very high.

Our surgeon was on sick leave and the nearest military surgeon was at Lucknow, fifty miles away. With my Commanding Officer's sanction, I performed the operation. The appendix was perforated, retro-caecal running up towards the right kidney, and tied down with multiple adhesions, and in my unaccustomed hands the operation took two hours. On return to the Ward the axillary temperature was 108°, the pulse 160 and very feeble, respirations 32. Rectal temperature 108.6°.

The patient was removed to the treatment room

and three pints of hypertonic glucose saline were given, with intravenous niketamide. He was of course put on penicillin.

A tepid sponge was given fifteen minutes later and the temperature dropped to 107°, the pulse remaining feeble. There was no sweating.

An hour later without further cooling treatment sweating recommenced and the temperature dropped to 101.8°, pulse 140, respirations 60, and complete collapse seemed imminent. The patient was removed from the treatment room. The temperature rose gradually and the circulation improved, though the pulse was still about 140.

In twelve hours the temperature was 101°, pulse 120, respirations 48, and there was considerable pulmonary oedema.

Convalescence proceeded slowly with many setbacks. The patient's speech was incoherent for ten days and the clinical picture was reminiscent of recovery from encephalitis.

The wound healed well and he was ultimately repatriated.

Hospital 62 days.

DISEASE.

ACUTE APPENDICITIS

HEAT HYPERPYREXIA

Notes of Case

Name CPL. P.

Age 29

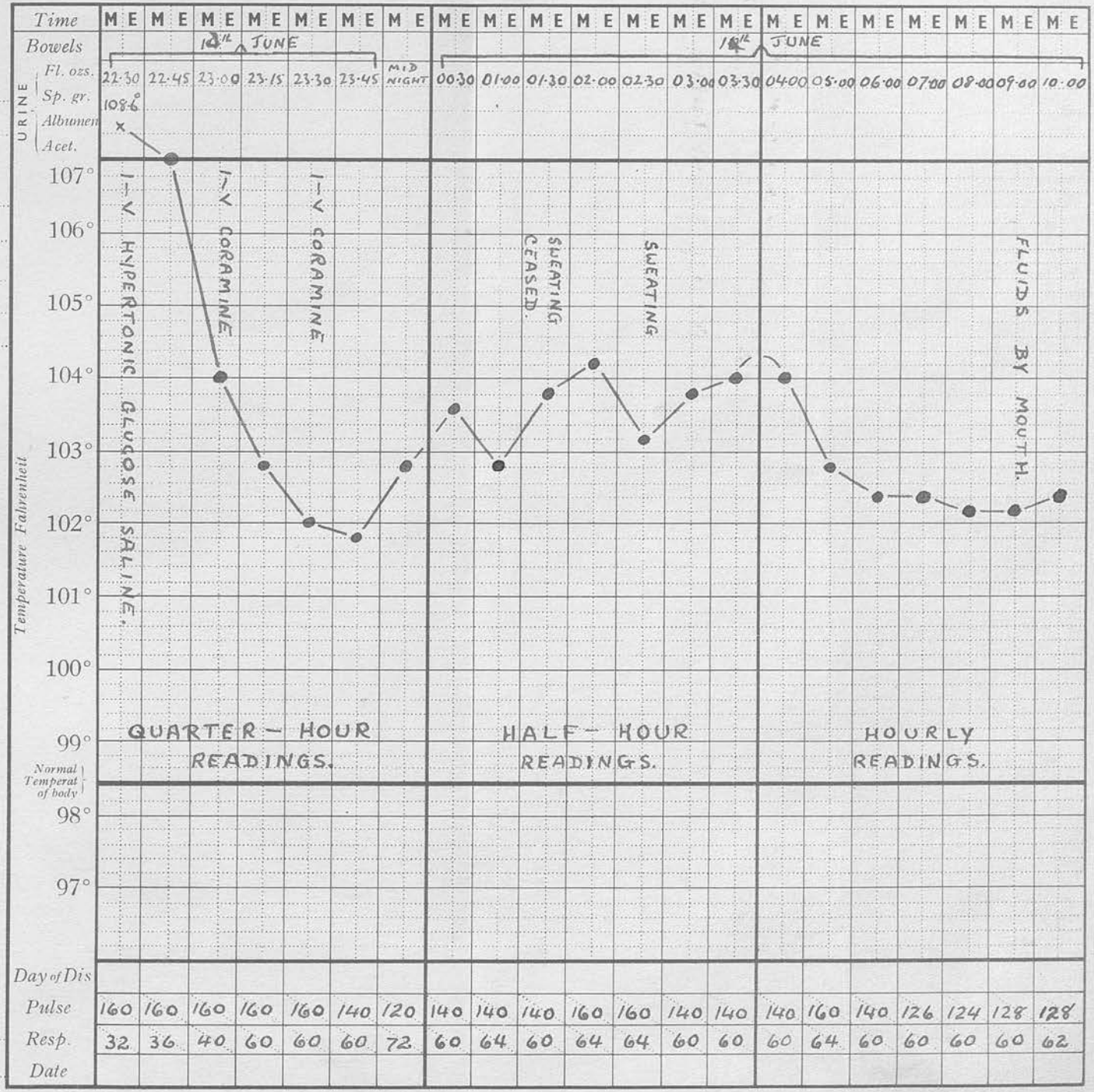
Diet

Case Book No. 6

Date of admission

18-6-1945

Result CURE.



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CASE VII

Pte. M. K.O.R.R.

Age: 23.

Overseas service: 2 years.

History

This man had seen considerable service in Burma. There was a history of fever in the jungle, but no malarial parasites were ever found.

He took ill during physical training - the onset was fairly sudden, with headache, malaise, vomiting and giddiness.

He noticed early on that sweating had ceased, and reported sick. The temperature before admission was 106.2°.

Slightly constipated.

On admission: Skin dry and hot.

Mouth parched.

No prickly heat.

Pupils dilated but reacted to light and accommodation. Conjunctivae injected.

Mental state - semi-comatose, but could be roused sufficiently to answer questions.

Knee jerks absent.

No incontinence.

Spleen not palpable.

Blood slides negative.

Urinary chlorides less than 1 Gm. per litre.

Treatment: Two pints of intravenous saline (hypertonic)

Subsequent Course

The temperature responded well to treatment and fell rather rapidly to 100°.

Vomiting continued at intervals. The vomitus consisted chiefly of a dark, highly acid fluid with little or no food particles. In the later stages it also contained flecks of fresh blood.

At midnight, seven hours after admission, the skin once more dried and the temperature rose to 105°. Treatment recommenced, and the temperature was brought down to 102°. A further rise associated with cessation of sweating occurred three hours later but was checked at 103°, and on this occasion an uninterrupted drop to normal values occurred.

During this time vomiting persisted at intervals and a further two pints of hypertonic saline were given. Urinary chlorides returned to normal, and convalescence was uneventful.

Hospital 9 days.

Hill leave 21 days.

This case, although at first sight milder than many others, showed a very slow response, and the temperature did not reach normal until sixty hours

after admission. Sweating was well established twice and later ceased, and the frequent vomiting was a most distressing feature. It was very fortunate, from this point of view, that consciousness was preserved throughout, so that there was little danger of vomitus entering the lungs.

I.V. quinine was given when the first relapse occurred, but repeated slides failed to demonstrate the parasites, and the course of the disease was not typical of malaria.

DISEASE.

HEAT
HYPERPYREXIA.

Notes of Case

Name PTE HALTON.

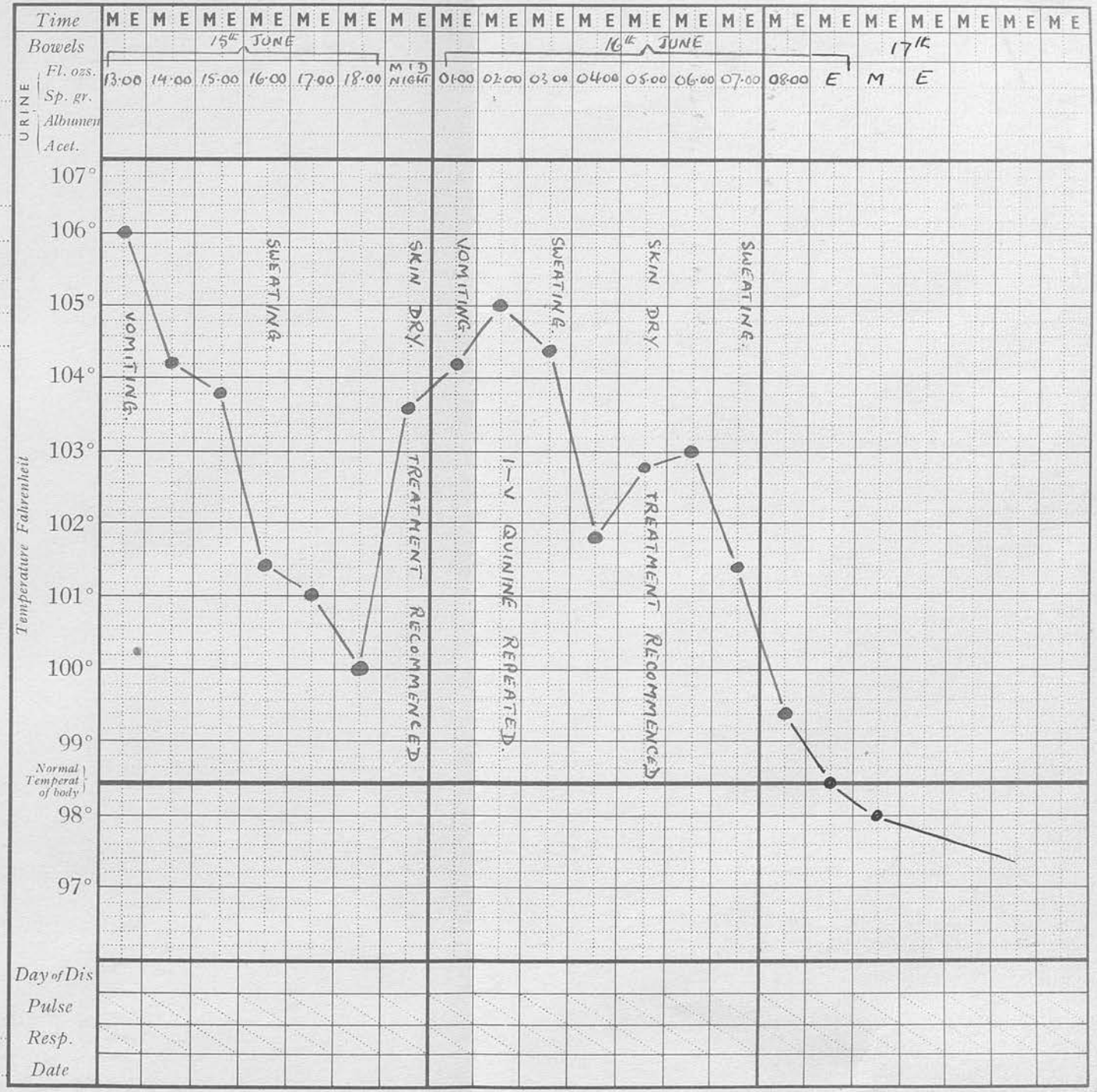
Age 23.

Diet

Case Book No. 7.

Date of admission
15-6-1945

Result CURE.



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CASE VIII

L.A.C. G.

Age: 23.

Tropical service: 1 year.

Admitted: 21.6.1945.

T. 107.5°.

This man had complained of weakness and lassitude for two days and had also reported the passage of blood and mucus P.R.

This was followed by intense headache and photophobia.

There was no history of special exposure.

On arrival at the hospital it was reported that two orderlies had had to hold the patient down for most of the journey, and when I entered the ambulance I found him in clonic spasm. The spasms had been so severe that his nose was actually bleeding owing to a violent blow from his knee. As it was impossible to move him except by force, I gave a small dose of pentothal under which he was hastily removed to the treatment room.

There were very obvious signs of profound fluid deficiency and as the effect of the pentothal wore off the spasm recommenced violently, while the pulse showed signs of weakness. There was incontinence of

faeces and it was noticed that the motions contained a fair amount of blood.

The pentothal was cautiously repeated, the patient immobilised and two pints of hypertonic saline given fairly rapidly, followed by one pint of normal saline.

Intravenous quinine was given in this case and sodium niketamide. Consciousness was slow in returning. Periodic clonic spasms recurred and there were fits of shouting, with incoherent muttering in the intervals. I was, however, reluctant to continue using pentothal on account of the rapid and feeble pulse, and finally the spasms and restlessness disappeared and the patient fell into a profound sleep. Sweating became re-established shortly before this, and, as the pulse pressure was still low, a further pint of hypertonic saline was administered.

Subsequent Progress

Convalescence was slow, and was complicated by the bacillary dysentery which had possibly been the determining cause of the attack.

Stay in hospital 18 days.

Sick leave 21 days.

DISEASE.

HEAT HYPERPYREXIA
COMPLICATING
BACILLARY DYSENTERY

Notes of Case

Name LAC. G.

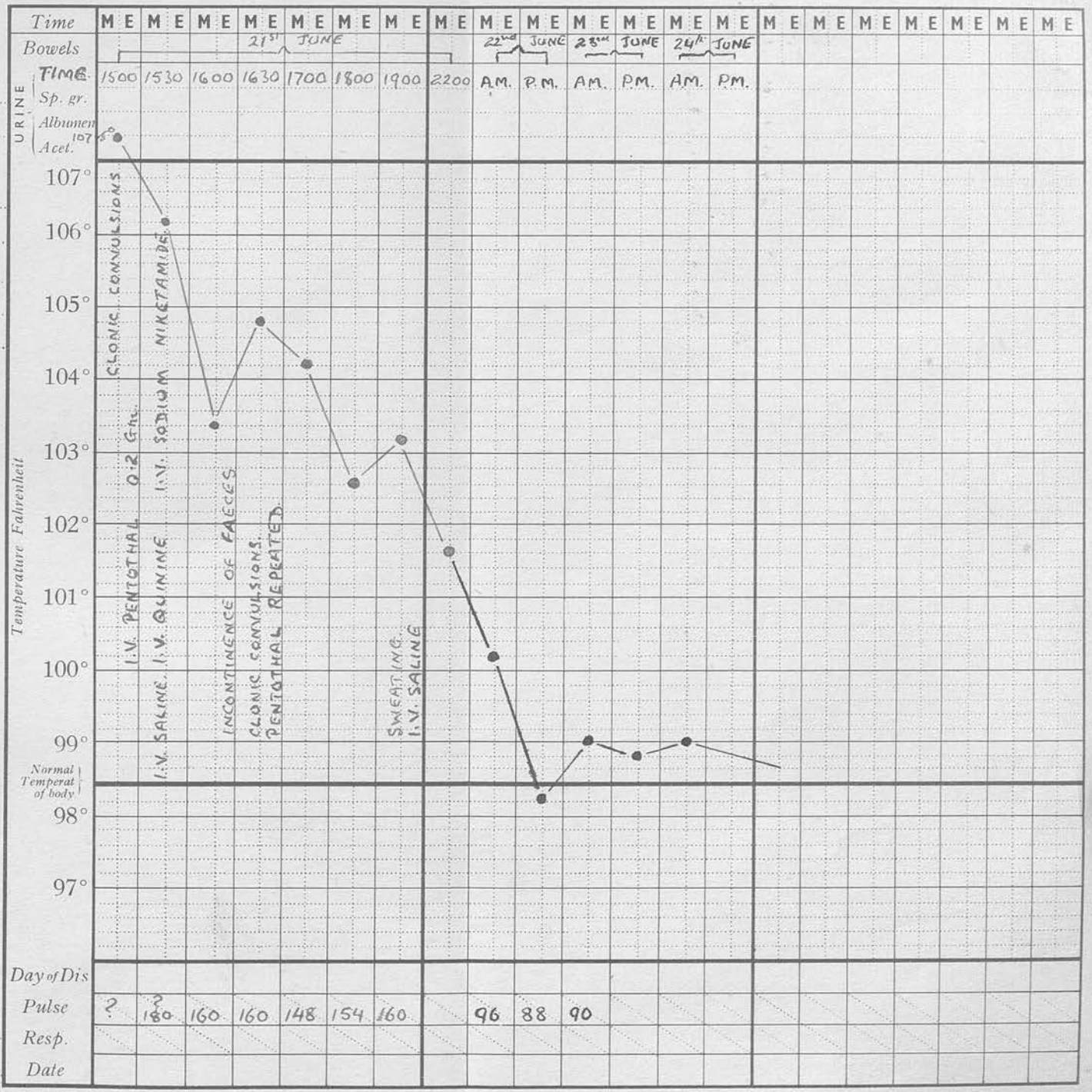
Age 23

Diet

Case Book No. 8

Date of admission
21-6-1946.

Result CURE.



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CASE IX

Lieut. L.

Age: 23.

Overseas service: 3½ years.

Admitted: 23.6.45.

History

Headache, weakness and sudden cessation of sweating.

This patient was in a small unit in Cawnpore to which the hospital acted as a Medical Inspection room. He stated that he had probably got malaria again, and gave a history of two attacks of B.T. and two of M.T. malaria.

On examination: Skin dry and hot.

Tongue and mouth dry.

Conjunctivae suffused.

Mental condition normal, apart from severe headache.

Spleen enlarged and tender.

T. 107.5°.

The peripheral blood was examined frequently in view of the history and the splenic enlargement, but was uniformly negative.

Subsequent Progress

In spite of the less severe initial symptoms, he presented some difficulties in treatment. In his

case intravenous quinine was delayed while several blood slides, both thick and thin, were taken. No true rigors were seen but it is significant that when the temperature rose for the third time, regular quinine treatment with an initial does of gr. vi. intravenously was instituted and no further relapse occurred.

In all, three courses of ice treatment had to be given. In each case the temperature dropped fairly quickly.

On discharge the spleen was no longer palpable.

Hospital 12 days.

Hill leave 21 days.

Remarks

In spite of the repeated negative blood slides it is possible that this was a case of malaria. In view of the severity of the symptoms, however, I feel that at some time or other malarial parasites would have been found.

DISEASE.

HEAT HYPERPYREXIA

? COMPLICATING MALARIA.

Notes of Case

Name LIEUT. L.

Age 23.

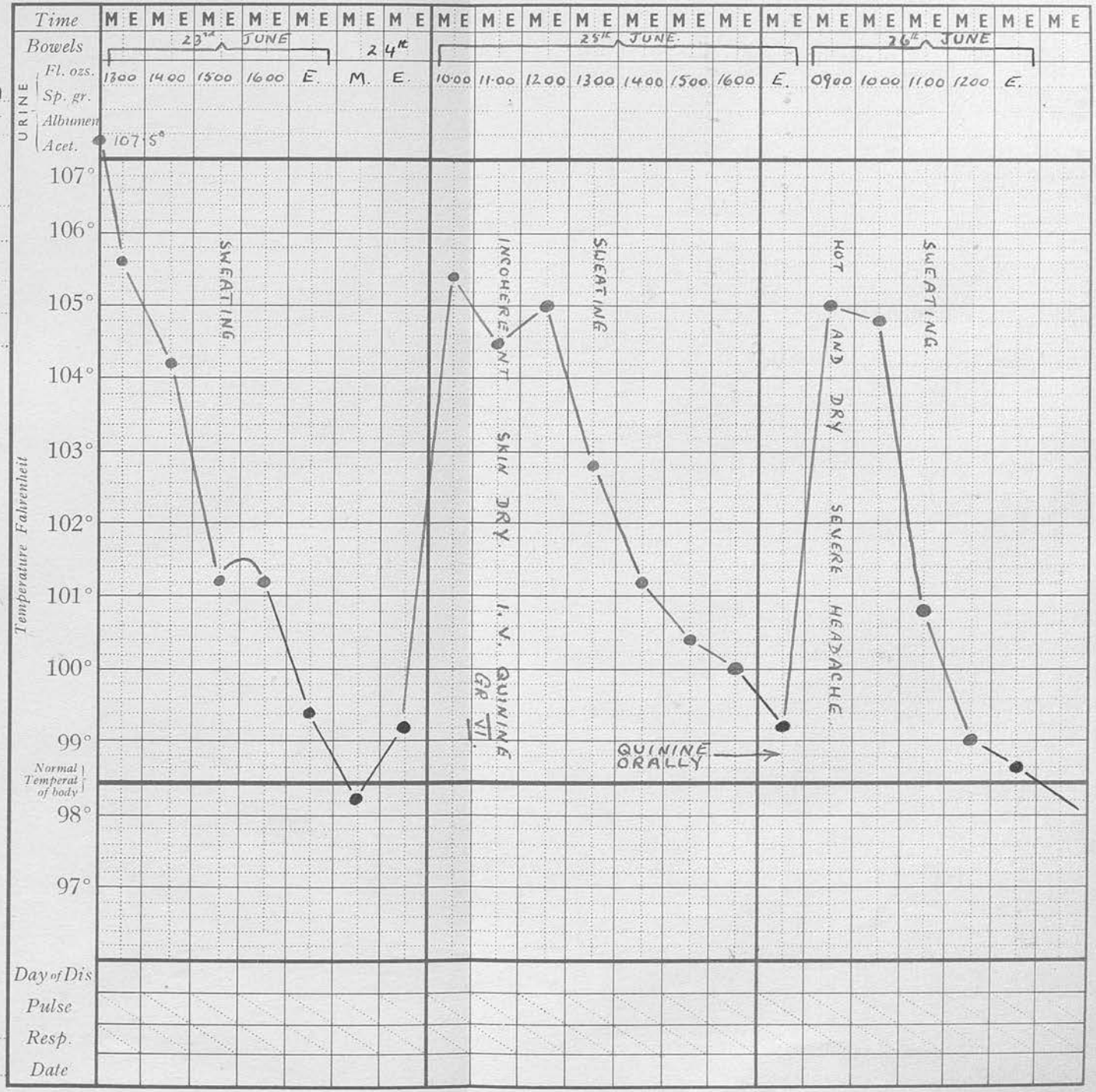
Diet

Case Book No. 9

Date of admission

23 — 6 — 1945

Result CURE.



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CASE X

Private H.

Age: 29.

Overseas service: 3½ years (chiefly in Aden).

Admitted: 23.6.45.

History

This man had been driving a lorry in convoy since 7 a.m. on one of the hottest days of the year - midday shade temperature 118°. He had felt ill the day before but did not report sick in spite of headache, listlessness and sudden cessation of sweating. In any case there were no medical facilities at the previous night's halt.

After driving some miles he was relieved, and sent on to the hospital by the O.C. Convoy on arrival at Cawnpore at midday. His exposure was therefore rather severe.

On examination: Skin dry and hot.

No prickly heat.

Tongue very swollen.

Conjunctivae suffused - pupils dilated but reacted to light. Intense photophobia.

Mental condition - confused but could be roused.

Able to swallow.

T. 106.6°.

P. 140 and bounding.

Blood slides negative (malaria is practically unknown in Aden)

Urine chlorides 3 Gms./litre.

Treatment: Cool room for two hours only.

Subsequent Progress

This patient, in spite of considerable exposure, made a very rapid recovery. No intravenous therapy was necessary as he could drink copiously.

Hospital 8 days.

Hill leave 14 days.

DISEASE.

HEAT
HYPERPYREXIA

Notes of Case

Name PTE HADFIELD

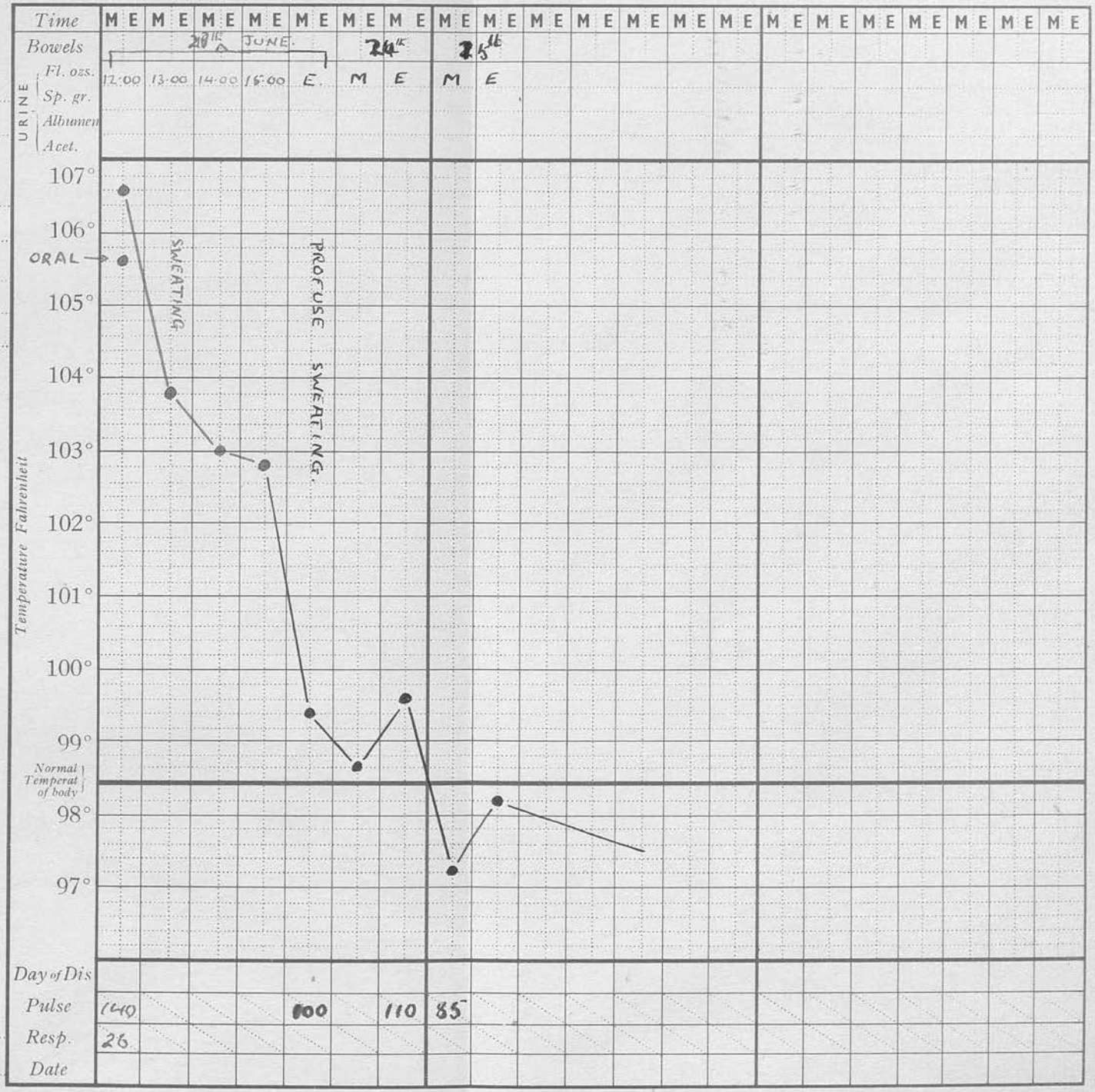
Age 29

Diet

Case Book No. 10

Date of admission
29-6-1945

Result CURE.



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CASE XI

L.A.C. B.

Age: 19.

Tropical service: 5 months.

Admitted: 25.6.1945.

T. 106.8°.

History

Had complained of headache and lassitude for two days, and noticed that sweating had ceased.

No nausea or vomiting.

No special exposure.

Very constipated.

Considerable frequency of micturition.

Urinary chlorides normal.

On 24.6.45 he was admitted to his station sick quarters with a rectal temperature of 105.4°, lowered by treatment to 102°, but rising again at 01.30 hours to 105°. He was admitted to hospital in the early morning, and the chart in this case shows the figures prior to admission as well as subsequently.

On examination: The patient was comatose but could be roused - the general condition was ppor and the pulse rapid and thready.

Skin dry.

Some cyanosis present.

Pupils dilated but reacted

slugglishly.

Subsequent progress

After admission there was a rigor with rise of temperature to 106.8°, increase of depth of the coma and signs of cerebral irritation.

Repeated blood slides were negative, but the patient was given an injection of quinine intravenously.

Lumbar puncture was performed, but the fluid was clear and pressure normal (60 mm. water).

Under energetic treatment the temperature slowly dropped, but convalescence was slow, and he was ultimately sent to the hills on sick leave for 21 days.

Stay in hospital	16 days.
Sick leave	21 days.

DISEASE.

HEAT HYPERPYREXIA

Notes of Case

Name L.A.C. B.

Age 19

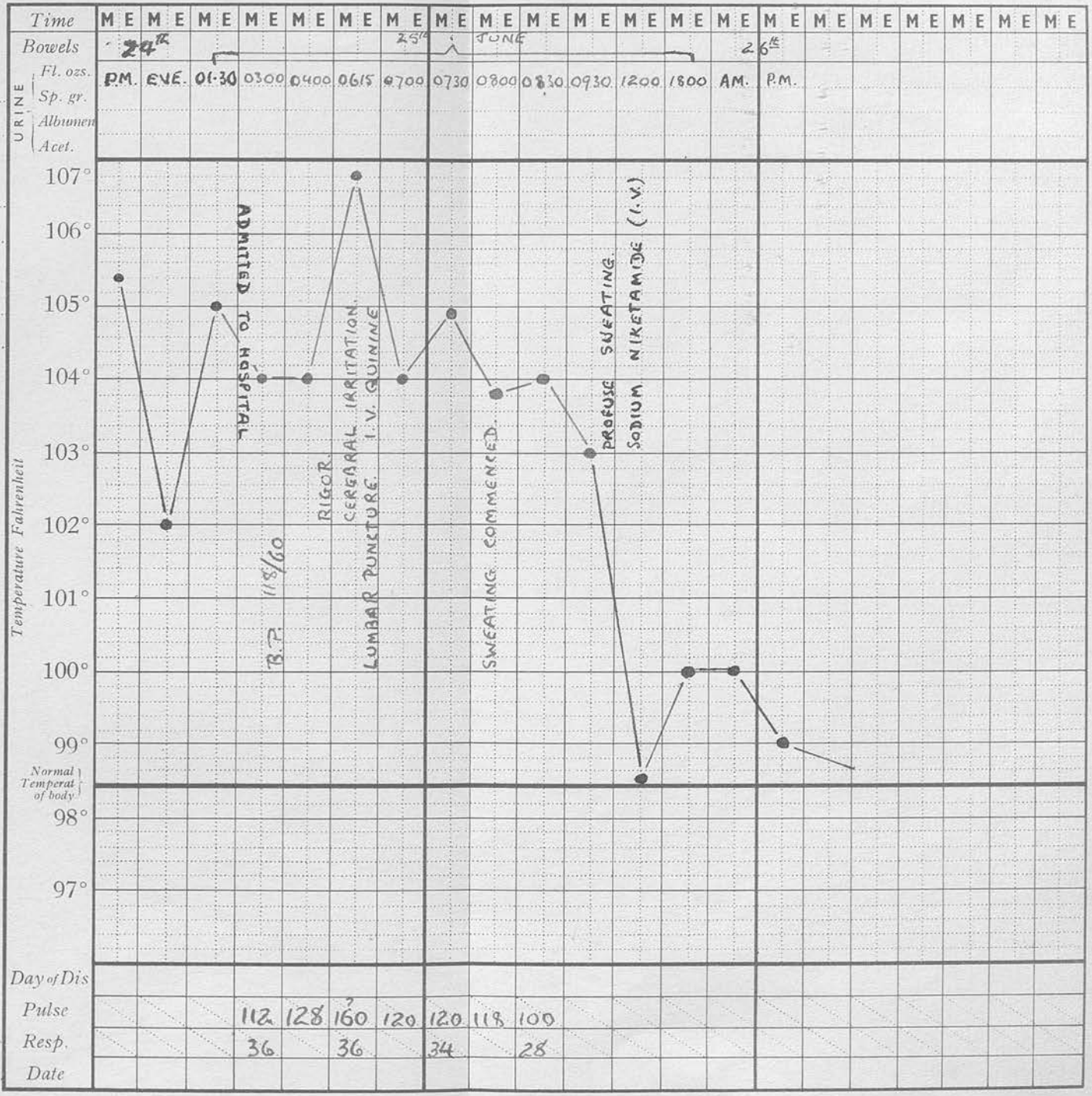
Diet

Case Book No. 11.

Date of admission

25-6-45

Result CURE



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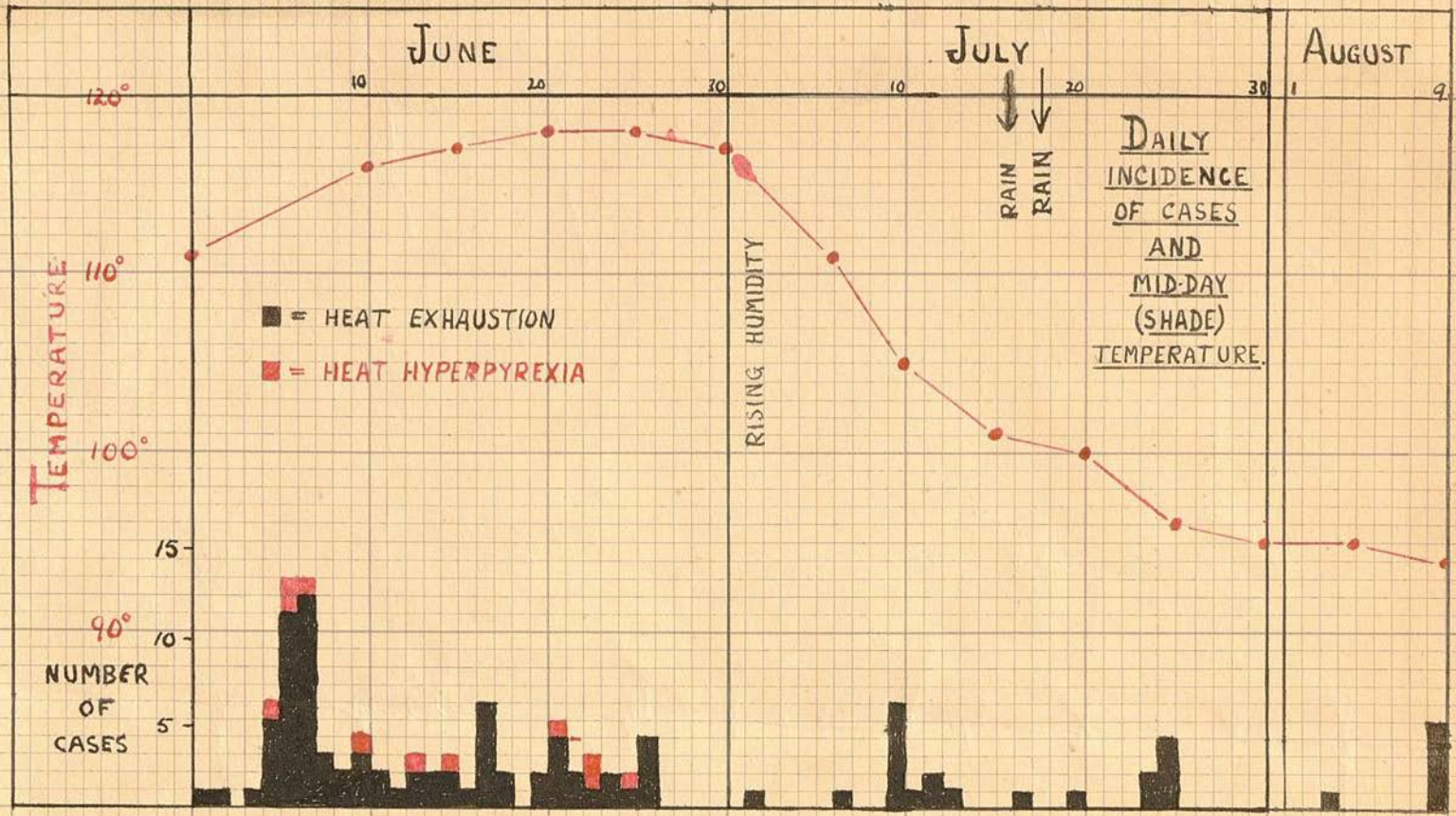
JUNE
JULY
AUGUST
10
20
30
10
20
30
1
9
120°
110°
100°
90°
TEMPERATURE
15
10
5
NUMBER OF CASES
■ = HEAT EXHAUSTION
■ = HEAT HYPERPYREXIA
RISING HUMIDITY
RAIN
RAIN
DAILY INCIDENCE OF CASES AND MID-DAY (SHADE) TEMPERATURE.

SECTION III

CONCLUSIONS

In the treatment of heat effects the first essential is an enthusiastic and well trained staff, and the second a well equipped hospital. I was extremely fortunate in having both these conditions realised, and I cannot speak too highly of the keenness and hard work of all my colleagues, medical officers, nursing sisters and nursing orderlies, and, I would add, convalescent patients, who most cheerfully volunteered to watch recovered cases during our more hectic days.

Emphasis has been laid on the clinical, rather than the biochemical side of the picture, for two reasons. Firstly because I consider the patient's condition as seen at the bed side much the most important factor, and secondly because the facilities for research such as that carried out by Ladell (Lancet, 1944, p. 100) were simply not available, nor was there time to perform the tests. Laboratory work was confined to examining blood slides for malaria, and roughly estimating the urinary chlorides. Early blood pressures of collapsed patients were estimated rather than taken, and everything was subordinated to immediate treatment.

While this may have lessened the theoretical

interest of this paper, I consider the 100% recovery rate fully justifies the methods employed.

I understand, though I have not been able to verify this, that since there has been a garrison in Cawnpore, no year had previously passed without deaths from heat effects. When the tremendous increase of European troops due to the opening of the R.A.F. maintenance units in the neighbourhood is considered, this fact becomes even more impressive.

I consider the most important single cause for the relatively small morbidity to be the education of the men in the necessity for avoiding heat effects. Both by lectures and in pamphlets issued to the troops the dangers of tropical conditions were stressed, and the means of reducing the risk of occurrence and of the recognition of early symptoms were clearly stated.

Commanding Officers have realised the need for a period of acclimatization, and the highest praise is due to the unit medical officers for the speed with which potentially serious cases were seen and sent for admission.

In hospital all preparations were made several weeks before the first cases began to arrive. They were treated as most acute medical emergencies and other work was subordinated to their care. The

trolley with all necessary equipment was at hand. The wardmasters at the admission gate were warned that their job was to get the patient to the Ward, and to obtain the relevant information of number, unit, etc., as and when possible.

One medical officer at least was always on duty in the Ward, prepared to take over at once, and the laboratory staff had instructions that blood slides from hyperpyrexial patients, and urinary chlorides of those in severe collapse had first priority.

Ice appeared to be available in unlimited quantities whenever required.

Given these facilities, speed of treatment and, in the hyperpyrexial cases, energetic continuation appeared to be the criteria of success.

I have come to the conclusion that pentothal sodium has possibilities in the treatment of heat hyperpyrexia. It is obviously impossible to judge from the one case in which it was perforce used; nevertheless the spasmodic clonic movements which occur in many cases of hyperpyrexia must generate heat, besides making treatment more difficult, (Samson Wright, see page 4).

I have myself very considerable anaesthetic experience, and can find no contra-indication to small repeated doses, and it is noted by Langton

Hewer (Recent Advances in Anaesthesia, 1943, p. 91) that ether convulsions occurring in humid tropical conditions may safely be treated by this method. The author also states on page 90 "On the other hand anaesthetised patients tend to lose rather than to gain heat, and the high temperatures seen after an attack are almost certainly due to the intense muscular activity."

Langton Hewer (ibid. p. 262, quoting F.P. Haugen, Military Surgery, 1941, July, p. 72) states that "it has been estimated there is an increase of about 7.5% in Basal Metabolic Rate for every degree of temperature rise above normal." This is confirmed by Guedel (Inhalation Anaesthesia, 1942, p.63) who states (ibid. p. 66) that the barbituric acid derivatives reduce metabolism directly when given in large doses.

Were I to treat hyperpyrexial cases again, I should certainly incorporate in the intravenous drip a side tube allowing pentothal to be given as required. This would have four advantages:-

1. It would abolish the restlessness, allowing intravenous fluid to be given at the earliest stage.

2. It would reduce, by eliminating muscular activity, the actual production of heat in the body.

3. It would reduce the B.M.R.

4. It is a well established fact that anaesthetised patients are over-susceptible to differences in external temperature - hence the precautions governing the temperature of operating theatres. Since in hyperpyrexial cases the heat centre has temporarily ceased to function, no damage can be caused by depressing it still further, in the early stages at least, as all control is being effected by artificial and external means. It would appear that cases might show a more rapid response if anaesthetised.

It may be argued that treatment would involve the presence of an experienced anaesthetist, but in fact the dosage used would be small, just sufficient to abolish muscular activity in the limbs; coramine or niketamide is always available as an antidote and, assuming that a free air way is maintained, the risks are minimal. There appears to be no contra-indication to the use of pentothal in hyperpyrexial cases - in fact, judging from Guedel, it might appear that such cases are less than normally sensitive to its action.

SUMMARY

One hundred and eleven cases of heat effects are described, divided as follows:-

Heat exhaustion and collapse		
Mild	52	
Moderate	39	
Severe	9	100
Heat Hyperpyrexia		11

Mortality - Nil.

Morbidity:

Average stay in hospital	9.5 days.
Average sick leave in the hills	10 days.
(This figure is not really accurate as it fails to take into account that several cases were due for routine hill leave and were sent direct from hospital with their unit parties.)	
Cases repatriated	3

Clinical details of all cases of hyperpyrexia and of the severe cases of heat exhaustion are given. Other cases have been treated statistically.

Laboratory tests were subordinated to clinical findings and only essential investigations (examination for malarial parasites, etc.) were carried out.

Cases were considered in two groups, those with considerable tropical experience, who had spent

at least one hot season in India, and those with under a year's tropical service.

A description of the Hospital and the facilities available is given, and of the advance preparations made to deal with the expected occurrence of cases as the hot season reached its climax.

Details of the general lines of treatment of different types of case have been given in full.

The medical literature on the subject has been discussed, from Haldane's pioneer research to the scientific analysis of Ladell and his colleagues in 1944 and Professor Maegraith's summary in this year's British Medical Journal.

A suggestion is made of the possible value of pentothal sodium as an adjuvant to the treatment of hyperpyrexia.

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