and others established that heat-stroke is caused by overheating the body. It is not an infectious disease, as Sambon has suggested (supported by Manson's authority), but is more liable to occur in those whose vasomotor mechanism is unstable, in the constipated, the drunken, the fatigued, or infected. Heat-stroke can be produced in normal men by appropriate conditions in the experimental chamber of the physiologist. Many cases occurred in fresh troops sent straight from England and certified free from malaria on their arrival at Basra. It can be prevented by simple treatment based on the physical laws of cooling. 107° to 108° F. seems to be the critical rectal temperature at which grave effects such as unconsciousness and convulsions result. Speedy reduction from such a temperature is demanded to avoid not only a fatal termination but tissue degenerations.

Men vary in their capacity of sweating, and thus, when in Mesopotamia in 1917, the temperature rose in stages, groups of cases occurred at 105°, 110°, 115°, 120° F. Such shade temperatures out of doors meant 130° to 140° F. in the still air of a tent on the hottest days! Thick walls, double roofs, and wide verandahs well ventilated, and fans are required to make such conditions tolerable. There were none of these things to start with. The treatment often recommended in 1917 appears to have been the rubbing of the skin with ice and ice enemata. Venesection and infusion were also tried as a last resource.

While evaporation of water at body temperature carries away 0.59 calorie per gram, the melting of ice only takes away 0.08 calorie per gram; rubbing the skin with ice, moreover, obstructs evaporation. Again, 70 grams of water evaporated from the skin takes away as much heat as 1,000 grams of iced water used as an enema.

The direct cause of heat-stroke in a hot, dry atmosphere such as Mesopotamia seems to be exhaustion of the sweating mechanism, hence diaphoretics are useless; the one treatment to immediately apply is artificial sweating, established by means of a water spray and fan. Such a method of treatment was, as Director-General Goodwin tells me, well known and practised in India before the war, but it had not become generalized and taught in the books. Fans and sprays were wanting to meet the fierce heat of the summer of 1917 in Mesopotamia.

G. K. Hearne found that he could pick out in his wards at Basra all cases in danger of heat-stroke by their dry skin; cessation of sweating, generally coupled with increased micturition, in every case preceded the rise of body temperature. Those who ceased to sweat asked for the roof fans to be turned off, because they, like the dry "kata" thermometer, were heated up by the wind—the air temperature being above body temperature. Those who were sweating, on the other hand, begged for the fans to be kept on because they were cooled by evaporation, just as the wet "kata" thermometer was cooled.

Hearne used the "kata" thermometer at Basra, and so secured a demonstration of these facts. Finding out the "dry" cases by frequent visits to each patient in his wards, he put on the water spray and fan treatment; by these simple means, so he tells me, he was able to stop all further cases of heat-stroke both in the wards and in the troopships. The great thing is to recognize the danger of the dry skin and get treatment in time. People must be warned of the danger of the skin going dry. Hearne found no signs of general exhaustion of the sympathetic system; it is the sweating mechanism in particular which fails. In the final stage of heat-stroke the patient lies cyanosed and comatose, with signs of respiratory embarrassment. The quick shallow breathing excited by heat washes CO₂ out of the blood, and so increases the alkalinity of the tissues. There is no acidosis then and no indication for the intravenous injection of a sodium bicarbonate solution. Gum saline may be tried for cases which come under treatment when collapsed, but the spray and wind treatment must first be set going.

Post mortem the organs of a case of heat-stroke show capillary congestion, as in wound-shock. Cramer lays stress on the signs of hyperaemia, followed by exhaustion of the thyroid and adrenal glands. Either infection or exposure to over-warm atmosphere has these effects. Hyperadrenalism leads to increased change of glycogen into sugar and greater heat production. Injection of a small dose of tetrahydrocannabinol into mice produces pyrexia with signs of increased activity of the sympathetic system. If the mice are kept in a cool room

they survive, but if crowded in a warm place they die of heat-stroke. The drug causes intense congestion of the thyroid, with changes in the colloid and very active secretion of adrenalin. It dilates the pupil, constricts the blood vessels, causes rapid respiration, restlessness, and spasms, changes liver glycogen into sugar, and increases the respiratory metabolism and heat production. Narcotics such as chloral counteract this experimental heat-stroke, and an animal kept narcotized long enough recovers from a fatal dose. It is obvious that, while narcotics might delay, they cannot stop heat-stroke when the air temperature is above body temperature and the patient cannot sweat.

Cramer's experiments show how infection and a hot atmosphere may have an accumulative effect in producing heat-stroke by each over-stimulating and exhausting adrenal action. Observations on lower organisms show that death from excessive heat is due neither to coagulation of proteins nor to want of oxygen, for the change produced by heat is reversible if not carried too far, and is not affected by increased oxygen tension. The ultra-microscope shows an augmentation in the number of granules in the living cell and a growth in their size as the temperature rises; the increasing aggregation, reversible up to a certain point, stops the reactions of life.

NOTE ON THE DIETETIC TREATMENT OF DIABETES MELLITUS.

BY

R. T. WILLIAMSON, M.D., F.R.C.P.,

CONSULTING PHYSICIAN TO THE ROYAL INFIRMARY, MANCHESTER.

In an article in the *BRITISH MEDICAL JOURNAL* in 1915 (March 13th) I recommended a line of treatment for certain cases of diabetes mellitus which I had found of much service in checking the glycosuria, temporarily at least. In cases uncomplicated by "acidosis" and certain other secondary affections, when the ordinary restriction of carbohydrates in the diet failed promptly to check the glycosuria, I advised complete rest, and for seven days a diet composed of a mixture of casein, cream, and water, given every two hours from 8 a.m. to 10 p.m. without any solid food. In many cases this diet promptly checked the glycosuria when other treatment had failed. At the end of the seven days a change was gradually made to a suitable solid diet, the amount of carbohydrates which the patient could tolerate being carefully noted.

During the war period, in place of the casein and cream solution I often used a diet composed chiefly of eggs and milk, given every two hours for seven days,¹ or a diet chiefly of suitable vegetables and jelly.² Both of these are more palatable, and are often useful, but not so frequently successful as the casein and cream solution. At the end of the war period the casein obtainable was often very unsatisfactory in taste and odour, and I have recently employed, with great success in many cases, the following mixture and course of treatment, which can be easily carried out. The mixture is a useful substitute for the casein and cream solution.

If "acidosis" and other serious complications can be excluded, and if an ordinary diabetic diet, with restriction of carbohydrates, fails to check the glycosuria promptly, then the patient is advised to rest on the sofa for seven days (or for a shorter period) and to take a mixture of eggs, cream, and water, prepared as follows: Three eggs are beaten up with 3 oz. of cream and a little water. More water is then added gradually until the mixture measures four pints. Of this mixture the patient takes half a pint (a tumblerful) every two hours from 8 a.m. to 10 p.m. (that is, eight times in the day). In addition he takes coffee or tea at 8 a.m. and 4 p.m., and also beef tea (warm) half a pint at 12 noon, 6 p.m., and 10 p.m. No bread, no meat, and no other foods are taken during this period.

In many cases the glycosuria is promptly checked in from four to seven days. This diet is not continued longer than seven days, and is then slowly changed to a solid diet containing only a small amount of carbohydrate food, the amount of bread and other carbohydrates which can be tolerated being carefully noted.

It is often found that not only has the glycosuria been temporarily arrested, but the power of carbohydrate

destruction in the system has been increased, so that the patient can take for a short or a long period (without glycosuria occurring) an amount of carbohydrate in the diet which would have been attended with glycosuria before the treatment.

If the glycosuria returns only after a long period, the week's treatment with the egg and cream diet may then be repeated. If the glycosuria returns in a few days after the first week's treatment with the egg and cream diet, then a satisfactory course is to advise this diet for one or two days every week-end, and to allow an ordinary solid diabetic diet, containing only a small quantity of bread and carbohydrate, during the other five or six days of the week.

A useful practical course, in these cases, is to advise that the urine should be tested every Friday; if it is found to contain sugar, then the egg and cream diet should be taken on the Sunday or Saturday, or on both days. If the Friday's urine is free from sugar, then the egg and cream diet is unnecessary on the Sunday.

This line of treatment is of great service in many cases, but unfortunately not in all. In some cases it fails, like every other treatment. In other cases, after being successful for a long period, it finally fails. It cannot, of course, be regarded as a cure for diabetes, but it is often a very useful method of checking the glycosuria at least temporarily, and of enabling us to keep it under control for a long period.

The grounds for the satisfactory action are the same as those I have described for the casein and cream diet. The treatment is unsuitable in cases with marked "acidosis," or with certain complications, such as phthisis and marked wasting, or cardiac failure.

Most patients can take the egg and cream diet for seven days quite satisfactorily. Some state that they feel remarkably well on it; others feel rather weak; only rarely do the patients complain of slight sickness, and in such cases the diet should be changed to the egg and milk diet or the vegetable diet previously mentioned.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, February 3rd, 1917. ² Practitioner, January, 1918.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

NEPHRITIS: ABDOMINAL HAEMORRHAGE: DEATH.

I HAVE had lately under my care a case which, though investigated *post mortem*, remains a puzzle.

I was called to attend a spare woman who gave a history of overwork during the war in the absence of her husband on service, with the suggestion that the town in which she had been living had not agreed with her health. She had a slight rise of temperature, and complained of pain and difficulty of movement in her legs and back. There was no paralysis; the temperature was slightly raised and the pulse rather rapid. Examination disclosed no cause for the condition; there were no catarrhal conditions. Provisionally I diagnosed influenza, and for a day or two she seemed to improve. The pains in the limbs disappeared, but the pain in the lumbar region of the back persisted and seemed severe. After the menses, which were present at my first visit, had ceased, I obtained some urine, and found it to contain blood and albumin. She remained some three weeks in this condition, with small rises of temperature—sometimes better and free from backache, at other times much the same. My partner, Dr. Dick, saw her, but could find no cause for the disease. She was sent into the Cottage Hospital, where in the night she had a "collapse," from which she recovered with stimulants. The bowels had acted, but the severe pain was referred to the right hypochondriac and epigastric regions. She remained in a low state for forty-eight hours, but during the night had another attack of pain, with collapse, and died.

My colleague, Dr. Gordon Ward, made a *post-mortem* examination. The peritoneal cavity was filled with blood and blood clots, for which no source could be discovered after careful examination. The heart was hypertrophied, and the walls of the left ventricle much thickened. At the

right edge of the liver was a much tumefied area, rather smaller than the palm of the hand—red, soft and friable, like a blood clot. There was blood behind the peritoneum near the right kidney, and petechial spots on the mesentery. There was also blood tracking up behind the peritoneum and into the thorax. A portion of the affected liver and a portion of kidney were sent to the Clinical Research Association, who reported:

There is a good deal of leucocyte infiltration beneath the peritoneal surface of the liver extending some distance along the fibrous septa. There is also considerable fatty degeneration. There is no pyogenic membrane adjacent to the clot, but there are a good many polynuclear leucocytes and a little pus at one or two points in it. We think there must have been an abscess. The kidney shows some thickening of the capsule, and a slight increase of fibrous tissue in places. There is much swelling and disintegration of the renal epithelium, however, so that in addition to a slight chronic nephritis there are acute degenerative changes.

Granting that this was a case of acute nephritis grafted on a chronic condition, and that such condition may cause a haemorrhagic diathesis by causing or failing to excrete a toxin, it must, I feel sure, be rare to find death due to such extensive haemorrhage with so few symptoms of kidney insufficiency.

Sevenoaks.

JAMES E. BLOMFIELD, M.D.

A CASE OF ACUTE YELLOW ATROPHY OF LIVER.

PRIVATE J. R. was admitted to the Military Hospital, Devonport, on December 26th, 1919. Four days before, without previous symptoms or any acute beginning, he noticed that his skin was becoming yellow, and that his urine was dark. He had had no previous serious illness. He served in France in 1918, but had not been anywhere else abroad.

On admission to hospital he was jaundiced, and had slight nausea, but no vomiting; the tongue was coated with white fur; the bowels were regular, the stools were solid and of a light slate colour. The temperature was normal; the pulse 64, regular, and of good volume. Nothing abnormal was found by palpation, percussion, or auscultation.

On December 30th the jaundice was intense, and he was very drowsy in the afternoon. The pupils were dilated. On the night of the same day he became very excited and violent, shouting, singing, and swearing. The pulse remained slow and of good volume, and the temperature normal. On the morning of December 31st he was semi-comatose; the pulse was rapid and feeble, and the pupils widely dilated; the temperature remained normal. A catheter specimen of urine was examined for leucin and tyrosin, which were not found. In the afternoon the coma increased, the temperature rose rapidly to 105° F., and the pulse was hardly perceptible. Cheyne-Stokes breathing supervened, and death occurred at 2.30 p.m.

Post-mortem Examination.—The liver weighed only 29 oz.; the capsule was wrinkled and loose, the substance of liver was limp and flabby, and bright yellow, with a few red patches. The heart showed numerous haemorrhages beneath the visceral pericardium. There were numerous haemorrhages also in the substance of both lungs. The aorta was bile-stained. The kidneys were soft and swollen, bile-stained, and showed many small haemorrhages. The spleen was apparently normal. The brain was not examined owing to objection of relatives.

E. C. WHITEHEAD, M.B., Major R.A.M.C.,

Officer in Charge, Medical Division, Military
Hospital, Devonport.

NOTE ON A TUBERCULOUS COW.

RECENTLY the inspector called my attention to a cow which had been slaughtered in my district. The animal was a cross-bred one, apparently about 10 years of age, and had reached this particular slaughterhouse, by the present exceptional conditions of grading and sale, whereby the butcher is the only man connected with the transaction who has no "say" in the choice of the animals he has to pay for.

The carcass was that of a third grade animal weighing alive about 10 cwt., and could not be said to be emaciated, and even could be described as decent beef in these times, when all animals are sent to the market in an underfed