We considered the evidence conclusive enough to ascribe the foetal death to an immune anti-A agglutinin of abnormally high titre. As the prognosis depended on whether the husband was homozygous or heterozygous for A, family studies were carried out in the hope that information might be obtained on this point, but they failed to be of assistance, as both his parents belonged to Group A.

Follow-up investigations were carried out six months later, when it was found that the anti-A titre had fallen to 1/1,000. In November, 1949, the patient became pregnant for the third time. Systematic investigations and titrations were carried out throughout the pregnancy. The anti-A titre fell progressively to 1/500 and subsequently remained unchanged at this level. The anti-B titre was constant at 1/50. As the pregnancy progressed and no change occurred in the titre we felt increasingly confident that a Group O foetus was present, and we decided to allow the pregnancy to proceed to term. On September 7, 1950, the patient went into labour and was duly delivered of a normal female infant. Immediate investigations on the cord blood showed that the baby did indeed belong to Group O, and the direct Coombs test was negative. The haemoglobin was 19 g.%, nucleated red cells were not observed, and the blood picture was normal in all respects. The maternal serum had no abnormal action on the baby's cells in saline or albumin, and the indirect Coombs test was negative.

The husband was thus proved to be heterozygous AO, and there will be an even chance of a Group O and consequently unaffected baby resulting in any further pregnancy. Should a rising anti-A titre again indicate the presence of a Group A foetus one would recommend premature induction of labour followed by immediate blood investigations and, if indicated, immediate transfusion of the baby with Group O blood, selected before delivery as not being acted upon abnormally by the mother's serum.

Summary

A case history is given in which the importance of an immune anti-A agglutinin of abnormally high titre is illustrated. The sequence was an abortion possibly due to this agglutinin, a stillbirth certainly caused by it, and finally the birth of a normal baby belonging to Group O.

It is emphasized that ABO incompatibility is a relatively important cause of haemolytic disease, and every case with a suspicious obstetric history should be fully investigated from this point of view.

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CHANGES IN SWEATING AFTER PRICKLY HEAT

BY

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In 1944 Ladell, Waterlow, and Hudson described a type of heat exhaustion characterized by diminished sweating, poor heat tolerance, inability to retain water and salt, and a general lassitude, increasing as the day got hotter; the anhidrosis was often associated with healed prickly heat. There was also, especially in cases with a history of severe prickly heat, an unusually high concentration of chloride in the sweat of these patients when tested during convalescence. A similar syndrome of anhidrosis and asthenia was described by Wolkin, Goodman, and Kelley (1944), who did not record any association with prickly heat; but O'Brien (1947), working in South-east Asia, confirmed clinically the anhidrotic effect of prickly heat and the subsequent development of tropical asthenia.

It has not previously been possible to confirm experimentally the effect of prickly heat on sweating and on the composition of sweat, as the lesion cannot be produced in the laboratory, and it is usually endemic in localities where facilities for detailed investigations are non-existent. Even when facilities do exist it is not practicable to test all new arrivals in the country in the hope that some of them will develop severe prickly heat later and present themselves for reinvestigation. Observations on a single case are therefore presented as a clinical record with details that are unlikely to be obtained often.

Case Report

A man arrived in the Tropics for the first time to join the staff of this unit in September, 1950. In October, 1950, he was given a routine acclimatization test, consisting of five 20-minute cycles of work and rest, mean metabolic cost 100 kg.cal./m.²/h. (= 2 met) in a climate of 100° F. (37.8° C.) dry bulb, 93.5° F. (34.2° C.) wet bulb, air movement 50 ft. (17 m.) a minute. Pulse rates, rectal temperatures, and body weights were recorded every 10 minutes throughout the 110-minute exposure; serial samples of sweat were obtained by an arm bag and analysed for chloride; water drunk and urine passed were measured. After the test the subject was an active observer in a series of climatic investigations, in which effective temperatures up to 95° F. (35° C.) were used, three days a week.

Within a few weeks, and shortly after being severely sun-burnt when playing cricket, he developed prickly heat. The skin over the back, forearms, wrists, and belly was most affected; the irritation kept him awake at night and he began to complain of lassitude and debility. The condition was exacerbated by exposure to climates in which, although he sweated freely, evaporation was rapid enough to leave the skin sticky rather than wet; profuse sweating, with perspiration running off the skin, tended to give relief. He continued his work in the climatic chamber while treating his prickly heat vigorously. He gave up wearing tight-fitting cotton singlets, which became sweat-sodden and irritated the skin, and he cut down the use of soap; he applied a lotion of salicylic acid and perchloride of mercury in spirit (30%) to the affected parts every few hours, on the basis of E. C. Smith's (1927) demonstration that prickly heat is a monilia infection, and he anointed himself with red palm oil after bathing, in accordance with O'Brien's observation that the disorder is aggravated by, if not the result of, a deficiency of

The Ministry of Health announces that clean-food drives throughout the country will be aided by a new display set on the dangers of food-poisoning. Photographs and coloured sketches show how easily food becomes infected through dirty and careless handling. Unwashed hands are highlighted as the principal source of infection, and there are warnings against germs spread by unguarded coughs and sneezes, by unprotected cuts and sores, and by flies, rats, and mice. Other panels record the case history of a victim of food-poisoning and give commonsense preventive measures, including hints on cooking and washing-up. This latest 12-panel display set is the fifth in the comprehensive series on hygiene being produced by the Ministry with the help of the Central Office of Information for the use of hospitals, factories, local authorities, and voluntary organizations.

fat on the skin. One month later the skin was clear except for a few small patches on the wrists and back; he was sleeping well again and no longer complained of lassitude.

Reactions to Heat After Recovery

In March, after he had been going into the climatic chamber three or more times a week for 20 weeks, he was again tested to determine to what extent he had become acclimatized. Changes the reverse of acclimatization were, however, found. In Fig. 1 are plotted the mean sweat rates and mean rectal temperatures for successive ten-minute

FIG. 1.—Sweat-rate/rectal-temperature curves before and after prickly heat. The mean sweat rates and rectal temperatures in the successive 10-minute periods are shown as solid circles, with the number of the period. Continuous line=before prickly heat; broken line=after prickly heat.

FIG. 2.—Salt losses in the sweat before and after prickly heat. Cumulative salt losses have been plotted against cumulative water losses at 20-minute intervals throughout each exposure. The small numbers beside the points correspond with the numbers outside the circles in Fig. 1. Continuous line=before prickly heat; broken line=after prickly heat.

periods in each of the two tests. For the first 40 minutes of the second test he showed evidence of acclimatization (MacArdle, 1944); he began sweating at a lower rectal temperature and sweated more at a given rectal temperature. But as the exposure continued and his rectal temperature went higher his sweat rate failed to increase further. The final rectal temperature was 0.5° F. higher in the second exposure, but the sweat rate never surpassed that achieved during his first exposure, when he could not have been acclimatized. The overall sweat production in the first exposure was 1,950 g. for a rectal temperature rise of 2.7° F. (1.5° C.); the corresponding figures for the second exposure were 1,765 g. and 3.9° F. (2.2° C.). The ratio (sweat-output/rectal-temperature rise) over a standard 80-minute period suggested as an index of acclimatization (Ladell, 1951), fell from 668 on the first to 497 on the second exposure. A second, fully acclimatized, subject tested as a control in the chamber on the same two occasions had a ratio of 781 on the first and 776 on the second test.

Fig. 2 shows the cumulative salt losses in the sweat for each of the two exposures, plotted against the cumulative sweat water loss, at the end of each 20 minutes. Salt losses were markedly higher in the second exposure: for a sweat loss of 1,800 g. the subject lost 6.85 g. of sodium chloride on his first exposure, but for the same sweat loss on the second exposure he lost 8.85 g.; the mean concentration of chloride in the sweat had increased from 0.38% to 0.49%, expressed as sodium chloride.

Discussion

With acclimatization the sweat rate increases, the body temperature does not rise so high, and (Conn, Johnston and Louis, 1946; Robinson, 1949) the chloride content of the sweat falls. The subject, however, after nearly five months' work in a tropical climate, which would have tended to acclimatize him but during which he had had severe prickly heat, showed the reverse of these changes. To what extent could this reversal be regarded as the effect of the prickly heat, on the hypothesis of Ladell, Waterlow, and Hudson (1944)? Other factors might have contributed. Thus Thomson (1951) has shown that ultra-violet irradiation will diminish the sweat rate. The subject here was, however, first tested soon after being badly sunburned in the cricket field, and, though he continued to play cricket, the amount of body surface exposed while playing was not extensive.

The small vesicles, the sudamina, described by Thomson, were exhibited by the subject; but these are usual in prickly heat (O'Brien, 1947); the vesicles are the mechanical result of blockage of the mouths of the sweat glands. The treatment with mercury perchloride may itself have been injurious to the sweat glands and so have been a contributory cause to their altered activity. Possibly all three factors were concerned—prickly heat, sunburn, and the mercury.

In spite of the opportunity for acclimatization, the subject lost more instead of less salt in his sweat; this could have been the result of an increased salt intake (Ladell, 1945a), though he did not confess to one. Such an increase, according to Horne and Mole (1949) would itself have precipitated prickly heat; on the other hand, clinical experience in West Africa (Egerton, 1950) suggests that increasing the salt intake relieves rather than exacerbates this skin lesion. It is probably more reasonable to suppose the change in the sweat to be secondary either to the prickly heat itself or to some coincident systemic disorder. The actual validity of the estimation of salt loss in the sweat by arm samples has been questioned by Van Heyningen and Weiner (1950), but the method has been fully investigated and found accurate under the conditions of these tests (Ladell, 1948a).

The increased rise in the rectal temperature could not be attributed to the diminished sweating, as there was always more sweat produced than could be evaporated, and in Thomson's experiments considerably greater reduction of sweat, after ultra-violet irradiation, did not affect heat tolerance. A systemic disorder must therefore be postulated, which would also account for the lassitude and debility. The subject was, in fact, a mild but typical case of Ladell, Waterlow, and Hudson's Type II heat exhaustion. To adopt Selye's (1950) terminology, Type II exhaustion might be called, as a working hypothesis, a "disease of adaptation." To some extent this concept is supported by accumulating evidence that the adrenal cortex may be involved in the control of sweating (Ladell, 1945b; Conn and Louis, 1950); on this theory, prickly heat would be the stress which, added to the several already imposed by a tropical climate, would rapidly lead to what Selye calls the "exhaustion stage," so that even after apparent recovery the heat tolerance remains markedly diminished. A somewhat similar hypothesis has been advanced elsewhere (Ladell, 1948b), but positive evidence for it has not yet been obtained.

Summary

An otherwise healthy subject on recovery from prickly heat showed a diminished tolerance to heat and an increased rate of chloride loss in the sweat, as compared with his figures obtained before prickly heat developed. The implications of these findings are discussed in relation to a certain type of heat exhaustion.

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One of the main aims of treatment at the Cassel Hospital for Functional Nervous Disorders is to prevent desocialization and accustom the patients to community life and responsibilities (*Medical Director's Report, 1948-50*). The team of workers mainly concerned with this aspect of treatment is called a social therapy unit, and is composed of nurses, two psychiatric social workers, and a psychologist. They are responsible for everything except the actual psychoanalysis. A great deal of emphasis is placed on the patients' social life while in hospital, and patients' committees are made responsible for their welfare, entertainment, and other activities. Subcommittees deal with messing, and regularly meet the hospital staff concerned; the housing committee allocates bedrooms, and this method greatly avoids displays of neurotic behaviour. Patients who are fit to do so clean and look after their own rooms, and some are attached to the housekeeper to help with her duties, or assist the administrative, clerical, and maintenance staff.

ACTINOMYCOSIS IN THE NORTH-EAST OF SCOTLAND

BY

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Although clinical accounts of actinomycosis in man had been given by Ponfick in 1882 and by Israel in 1886, it was not until some years later that the causative organism of the disease, *Actinomyces israeli*, was isolated by Wolff and Israel (1891). Wright (1905) obtained a similar organism from 13 cases of actinomycosis in man and two in cattle. Since then these findings have been repeatedly confirmed by other workers.

Within recent years it has been recognized that actinomycosis, although not a common disease, cannot be considered rare. In a period of six weeks from February to March, 1949, three specimens of pus containing sulphur granules and showing on microscopical examination the branched filaments of *A. israeli* were examined in the University Department of Bacteriology, Aberdeen. That three cases of actinomycosis had occurred in such a short space of time was considered unusual, although it is generally held that the infection is not uncommon in this area of Scotland. Examination of the literature revealed few reports of the incidence of actinomycosis in Britain and none for Scotland. Accordingly, it was decided that an investigation of actinomycosis, with special reference to its incidence in the north-east of Scotland, would be of interest.

Scope of Investigation

The survey was restricted to actinomycosis which had occurred in residents in the counties of Aberdeen, Banff, Kincardine, Moray, and Nairn from 1936 to 1948. To ensure that the list was as complete as possible the records of admission to the hospitals in the area and the bacteriological and pathological records of the Aberdeen Royal Infirmary and the City Hospital were examined. The records of cases of actinomycosis which had been treated in the X-ray Therapy Department, Aberdeen Royal Infirmary, were also examined. The period 1936-1948 was chosen, as it was considered desirable to use a period of time in the fairly recent past in order to ensure accurate diagnosis and follow-up of cases.

The north-east area of Scotland, on account of its geographical position, contains a population which is relatively static. At the census of 1931 the total population of the whole area was about 444,000, of which 167,000 people lived in the city of Aberdeen. The chief occupations in the city are fishing, shipbuilding, and the granite and distributive trades. The provincial towns in the area are market towns dependent on the surrounding agricultural districts. The chief occupations of the rural population are dairy-farming and stock-raising.

It was decided that no case should be included as one of actinomycosis unless there was confirmatory bacteriological or pathological evidence of the infection. As a result, at least 10 cases diagnosed clinically as actinomycosis have been excluded. The minimum requirements for bacteriological confirmation were the finding of sulphur granules and the demonstration microscopically of the branched filamentous organism. The finding of