xenon. As a result of this work flash discharge tubes have been made available which will produce intense flashes of light of very short duration suitable, among other things, for the photography of the human eye. These tubes are energized from a condenser charged to a high voltage. They emit in addition to the complex line spectrum of xenon a large amount of continuous radiation similar to that from an incandescent body. A further development has been the production of a continuously burning gas arc in which xenon is electrically excited to a high level. In the xenon lamp of either the flash or the gas arc type the light is white and is produced at a comparatively high efficiency.

Conclusion

Modern light sources fall, therefore, into four principal groups, each of which has distinct characteristics: (1) Incandescent sources in which the light is emitted by virtue of the temperature of a heated body. (2) Metallic-vapour discharge lamps in which the electrical excitation of the metallic vapour by the electric discharge causes the emission of characteristic radiation from the metallic ion. (3) Fluorescent lamps in which the primary radiation is produced from the low-pressure mercury discharge, but in which its radiation is absorbed by inorganic fluorescent compounds which are caused to glow with visible light. (4) Gas discharge lamps in which one or other of the rare gases is excited by the passage of the electric discharge; in this case the discharge may be of very short duration as in the repeating flash tube, or it may be a continuous discharge of high current density as in the gas arc.

None of these different types of light sources seems to have characteristics which will eventually make it supersede the others. All have a particular contribution to make in the production of artificial light for the many varied purposes for which it is required.

HAEMOLYTIC DISEASE OF NEWBORN DUE TO IMMUNE ANTI-A AGGLUTININ

BY

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The importance of ABO blood-group incompatibility as a cause of haemolytic disease of the newborn has given rise to considerable controversy during the past few years; some have even doubted the existence of the hazard. However, new tests for sensitization and refinements in technique have furnished ample evidence that such incompatibility does at times produce a definite clinical condition. There is also no doubt that ABO incompatibility between mother and foetus has a sparing effect on Rh response and reduces the risk of Rh sensitization where Rh incompatibility exists.

Pickles (1949), in reviewing the recorded cases, has emphasized that rigid criteria should be complied with before an alleged case of ABO sensitization is accepted

as such, and has stressed the necessity for stringent tests to exclude all other abnormal antibodies, and for proof, by a positive direct Coombs reaction, of adsorption of the antibody on the affected baby's erythrocytes. Such criteria are now minimum requirements in specialist laboratories. Additional confirmatory tests include parallel titrations in saline and serum or albumin at different temperatures, especially 4° C. and 37° C. (Hubinont, 1949). Boorman *et al.* (1949) have also carried out differential agglutination tests of survival rates and have shown that cells of the type in question are eliminated abnormally rapidly. Mollison and Cutbush (1949) have reported similar results.

In our experience the condition is relatively common in Bantu races, in whom it is probably as important as Rh sensitization, as only 5% of the Bantu are Rh-negative and the incidence of classical Rh sensitization is correspondingly low; moreover, distribution of the Rh subgroup is very different from that in Europeans (Zoutendyk, 1947). Similar results among negroes in the United States have been reported by Wiener, and he has also described two cases treated by exchange transfusion (Wiener *et al.*, 1949).

The following case demonstrates the importance of ABO incompatibility and emphasizes that the obstetric history may, in fact, be worse than in the case of Rh incompatibility, as in the latter state the first pregnancy almost invariably ends successfully provided that the Rh-negative mother has never received Rh-positive blood even in minimal amounts by any route.

Case Report

The patient, aged 24, had been married for 18 months when her first pregnancy ended in an abortion at 14 weeks. Serological investigations were not made because at that time blood-grouping and Rh-testing were not routine procedures. However, complete investigations were carried out on the blood of the patient and her husband in December, 1948, when the second pregnancy had progressed to 14 weeks. The reactions were as follows:

	Group	Anti-C	Anti-D	Anti-E	Anti-c	Anti-M	Anti-N	Blood Type
Patient	O _{A₁}	+	+	-	+	-	+	O CcDe N _{A₁} CcDe N
Husband	A ₁	+	+	-	+	-	+	

It will be seen that, apart from the difference in the ABO grouping, the reactions are identical. No abnormal saline or albumin agglutinins were demonstrable in the wife's serum against a wide range of Group O test cells both of known type and of others taken at random, and the indirect Coombs test was negative in every instance. The anti-A titre, however, as ascertained against the husband's red cells, was 1/10,000 at 37° C. The anti-B titre was 1/50, which is normal.

The abnormally high anti-A titre at 37° C. was considered to be of obstetric significance, and systematic investigations and titrations were tentatively planned for 32, 36, and 38 weeks. Unfortunately the patient went into labour at 31 weeks and, although the foetal heart had been heard 24 hours previously, a stillbirth resulted. Investigations on the cord blood showed that the baby belonged to Group A₁, but in other respects the reactions were the same as those of the mother. The maternal serum agglutinated the baby's cells to a titre of 1/20,000 in saline and 1/50,000 in serum. The anti-B titre remained unchanged at 1/50. The baby's red cells gave a strongly positive direct Coombs reaction. Exhaustive tests of the maternal serum on a representative panel of Group O cells in saline, serum, and albumin and by the indirect Coombs method failed to reveal irregular antibodies.

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.