Two pints (1 litre) of dextran 6% in saline was given when matched blood and plasma were temporarily unavailable. In retrospect it is clear that this was inadvisable, as a number of reports have incriminated dextran as a cause of a haemorrhagic tendency (Carbone *et al.*, 1954; Scott, 1955; McKenzie and Langlands, 1956). The mechanism of the process is not clear, and suggestions made include interaction of dextran with fibrinogen, haemodilution, production of thrombocytopenia, and prolongation of the bleeding-time. The patient, of course, had developed abnormal bleeding before the dextran was given, but it is possible that the dextran aggravated the haemorrhagic tendency.

Friesen *et al.* (1952) described similar cases in which post-operative bleeding occurred as a result of a haemolytic transfusion reaction, when over 250 ml. of incompatible blood had been given. They felt that fibrinolysis was an important factor, but considered that a "heparinoid disturbance" was present. Toluidine-blue dye, given intravenously to two patients in a dosage of 15 mg. per kg. of body weight, apparently corrected the bleeding tendency. Such treatment might be worthy of a trial in any refractory case.

Treatment with corticotrophin (200 units intramuscularly) or cortisone (400 mg. intravenously) has been recommended, in addition to fibrinogen, by Stefanini and Dameshek (1955).

If the possibility of a bleeding state after incompatible transfusion had been known, the second laparotomy would probably not have been carried out, and appropriate treatment might have been started earlier.

Summary and Conclusions

A case is described in which there was fatal post-operative bleeding, associated with afibrinogenaemia, following a haemolytic transfusion reaction.

Treatment is discussed, and the following points may be of value in controlling such a condition: (1) early recognition of the fact that a haemorrhagic state has developed; (2) administration of large amounts of the fibrinogen fraction—for example, 2–6 g., repeated if necessary—or the use of quadruple-strength reconstituted raw dried plasma if fibrinogen is not available; (3) fresh frozen plasma, or fresh blood, if there is a poor response to adequate fibrinogen; (4) avoidance of dextran; (5) corticotrophin, cortisone, or toluidine-blue dye intravenously, if not responding.

I am grateful to Dr. John Wallace, the director of the West of Scotland Regional Transfusion Centre, and Dr. A. S. Douglas for their assistance and advice. I should like to thank Mr. K. I. Macrossan for permission to publish details of this case.

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THE ISOLATION OF TRYPANOSOMA RHODESIENSE FROM A BUSHBUCK

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It has long been believed that an animal reservoir of Rhodesian sleeping sickness exists, but only one serious attempt has been made to isolate polymorphic trypanosomes from game animals and to test their pathogenicity in man. This was done recently by Ashcroft (1958), who killed 74 animals of various species in two endemic areas in Tanganyika and inoculated blood from them into rats. One polymorphic strain was isolated from a Coke's hartebeest (*Alcelaphus cokei*) near Babati in the Mbulu District and inoculated into man with negative results; this strain was presumably *Trypanosoma brucei*. It must be stressed that the only sure way of differentiating *T. rhodesiense* from *T. brucei* is by the inoculation of man. *T. rhodesiense* infects man while *T. brucei* does not: both trypanosomes are morphologically identical.

The present experiment was carried out on Utonga Ridge, a small peninsula projecting into Lake Victoria about 40 miles west of Kisumu in the Nyanza Province of Kenya. This area was chosen because not only were a few natives suffering from Rhodesian sleeping sickness found near by but because *T. rhodesiense* had been isolated from *Glossina pallidipes*, which is now the predominant tsetse fly on the ridge. Between April and August, 1958, 24 animals, which included 13 duikers, 10 bushbucks, and one serval cat were shot and their blood inoculated into rats. The serval cat was negative, but a strain of polymorphic trypanosomes and *T. congolense* were isolated from the duiker, and two polymorphic strains and two strains of *T. congolense* from the bushbuck. The polymorphic strains were inoculated into human volunteers and one of those from the bushbuck (*Tragelaphus scriptus* Fall.) was pathogenic to man (see below). This strain, which is highly pathogenic to rats and produced numerous posteronuclear forms, is presumably *T. rhodesiense*. This is the first time that *T. rhodesiense* has been isolated from animals and establishes finally that Rhodesian sleeping sickness is a zoonosis.

The bushbuck strain was inoculated into the forearm of Mr. C. A. W. Guggisberg, mammalogist on our staff. The inoculum was heavily infected and consisted of 25 ml. of blood from a subinoculated rat with numerous trypanosomes in its blood (over 100 per oil-immersion field). On the seventh day there was a hardish lump at the site of the inoculation and trypanosomes were seen in material aspirated from the swelling. On the eighth day scanty trypanosomes appeared in the blood and the temperature rose to 103° F. (39.4° C.). On the ninth day the arm was very swollen, with a hard inflamed area about 3 in. (7.5 cm.) in diameter on which was a blister about the size of a half-crown. Fluid from the blister contained about three trypanosomes per oil-immersion field. At 11 p.m. the temperature rose to 106.9° F. (41.6° C.) and the patient felt very ill. At 11.45 a.m. he was given his first injection of antrypol. Response to treatment was dramatic and recovery uneventful. Rats inoculated on the ninth day with exudate from the blister and blood taken from an earlobe became infected.

The technique employed is considered of great importance. It is essential to inoculate enough blood (30 ml. at

least) from the animal killed into enough rats (six at least), otherwise light infections can easily be missed. In our opinion Ashcroft inoculated too little blood into too few rats; this may be the reason why he isolated so few strains from such a comparatively large sample of animals. Experiments of this kind must be designed with a broad sweep on a large scale and may have to be continued for a considerable time.

Although the isolation of *T. rhodesiense* from a bushbuck is important, it would be interesting to repeat the experiment in areas where *G. morsitans* and *G. swynnertoni* are vectors of sleeping sickness to discover whether the same or other species of animals are acting as reservoirs. This note gives only a brief account of our work; detailed results will be published elsewhere.

We are much indebted to Messrs. Harvey, Harmer, and Highton for their enthusiastic work in the laboratory and the field.

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SECOND BACTERIOLOGICAL STUDY FROM A MATERNITY HOSPITAL

BY

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The measures used in a maternity hospital to check the growth and build-up of pathogenic micro-organisms in lying-in wards and nurseries have already been described (Marsh and Rodway, 1954). That paper covered a period of some 38 months, and the methods used were described in some detail. The present paper extends the study for a further four years—to nearly eight years in all.

Floors were treated with "marinol D." After the discharge of a patient, disinfection of a ward was effected by spraying chlorhexidine 1:5,000 from a "microsol" disperser. Blankets were washed in "lissapol N" (1:200) and disinfected with "fixanol C" (1:200). For a while "cirrasol O D" was used, but fixanol C was preferred. Blankets of the mothers were treated every two to three weeks; blankets of the babies every 10 days. Mattress sterilization was by Sparkhall, each time a new occupant used the bed. The rubber or sorbo mattresses of the infants were treated by wiping with a dilute solution of "dettol," "roccal," or chlorhexidine. The pillows were at first disinfected with formalin, but later this treatment was abandoned and plastic covers were used for the pillows. Since May, 1955, hand cream has been supplied for the nursing staff and also for the mothers, after they had washed their hands. Chlorhexidine and roccal were incorporated in the hand cream, 50% of each; the two compounds seemed to be equally effective.

Results

Records were kept of 211 blood-agar plates exposed in the wards during periods of activity. No colonies of *Staphylococcus aureus* were observed. Numerous colonies of *Staphylococcus albus* were identified. On two occasions a few colonies of *Streptococcus haemolyticus* (Lancefield group A) were found on a plate. A total of 477 swabs (nasal 243, eyes 165, and umbilical 69) from infants were examined. *Staph. aureus* was isolated from 58.8% of nasal, 48.4% of eye, and 55% of umbilical swabs taken between the eighth and the tenth days of life. (Penicillin-resistant staphylococci were found in 75 to 90% of the earlier (pre-1954) series, and in 65 to 80% of the present series (1955–7). Infection among 4,919 infants (1955–7) was 2.5% (respiratory infections 0.2%, minor skin lesions 1.3%, eye infections 0.18%).)

A total of 309 swabs from mothers were examined (110 nasal, 124 throat, and 75 nipple). *Staph. aureus* was isolated from 23.6% of nasal, 6.5% of throat, and 47.9% of nipple swabs. (*Staph. aureus* was insensitive to penicillin in 75% positive swabs in 1955, and in 65% positive swabs in 1957.) From January, 1950, to December, 1953, among 4,830 patients (including 64 admitted after delivery) there were 5.3% puerperal pyrexia cases. In 1953 three cases of breast abscess occurred; there were none in 1955–7. (A few post-natal women—a week or two after discharge—developed a breast abscess.) A total of 310 swabs from nursing staff were examined (150 nasal, 111 throat, and 49 hand). *Staph. aureus* was found in 34% of nasal, 4.5% of throat swabs, and in one hand swab. Of these strains 90% were resistant to penicillin.

During the second four-year period, 22 midwives and nurses and 202 pupil midwives were employed. Infections occurred in 75 individuals (33.6%). Four-fifths were minor respiratory cases, mainly tonsillitis and influenza. Other cases included minor skin lesions, boils, and furuncle of the ear (3.5%), eye infections (styes) 1.8%, gastro-enteritis 0.27%, and one case of glandular fever. *Staph. aureus* was not isolated from exposed plates—that is, from air-borne settles—but the micrococcus was isolated from the nasal, eye, and umbilical swabs. Of 255 neonatal deaths, respiratory infection was found in 2.3%.

The methods evolved for the prevention of cross-infection show some success; but *Staph. aureus* implanted in the noses of infants did not seem to be unfavourably affected.

It is thought that *Staph. aureus* confined to the noses of infants may not be harmful unless some means of dissemination occurs.

I thank Miss Helen Rodway, consultant obstetrician, Thorpe Coombe Maternity Hospital, London, for collecting the data and for enlightened supervision of the fight against cross-infection; and Dr. H. Caplin, director of pathology, Forest Hospitals Group, for undertaking many of the swab examinations.

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Medical Memorandum

Acute Haemolytic Anaemia in a Case of Infantile Gastro-enteritis Treated with Oxytetracycline

Anaemia of mild or moderate degree following infantile gastro-enteritis is not uncommon, and is usually thought to be due to a toxic depression of blood formation, often in association with a nutritional deficiency.* It is not known whether haemolysis plays any significant part. By infantile gastro-enteritis we mean a specific disease due to a number of serologically distinct types of *Escherichia coli*: an acute severe haemolytic anaemia following this condition has not been seen before in this unit. The following case is reported because other workers may have had similar experience and may be able to shed further light on its aetiology.

CASE REPORT

A male infant aged 6 months was admitted to hospital on December 5, 1956, with a five-day history of diarrhoea with some vomiting. His temperature was 98° F. (36.7° C.), pulse 134, and respirations 32. He was an ill child, with moderate dehydration and sore buttocks, but otherwise clinical examination was negative. He had not had any previous illness and there was nothing relevant in the family history. Glucose saline (2.5% glucose in 0.3% sodium chloride) was given by mouth, and after two specimens of