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 bush-buck is important, it would be interesting to repeat the experiment in areas where *G. morsitans* and *G. swynnertonii* 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.

REFERENCE

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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

faeces had been obtained oxytetracycline, 50 mg. six-hourly, was begun. After 24 hours his condition had much improved and there was no dehydration. Subsequent progress was uneventful, and oxytetracycline was discontinued on December 11 after a five-day course. At that time he appeared very well, the stools were normal, and the feeds had been increased to full-cream National dried milk.

On December 5 and 6 a pure culture of a smooth type of *E. coli* was isolated from the faeces. It was, however, an anaerogenic strain and was not typable with our sera (O26, O55, O86, O111, O114, O119, O125, O126, O127, and

the bilirubin content was 250 mg./100 g., and the stercobilinogen content 10 mg./100 g. of moist stool. Assays of the bilirubin and stercobilinogen contents of the stools from two infants of the same age who had just recovered from infantile gastro-enteritis showed 50 mg. and 110 mg. of bilirubin and 15 mg. of stercobilinogen per 100 g. of moist stool. The levels found in the patient were obviously greatly in excess of these, indicating that the anaemia was haemolytic in origin.

Red-cell fragility on two occasions was normal. During the period of acute anaemia there was no recurrence of diarrhoea. The stools were of an orange colour, and this was clearly due to the large amount of unaltered bilirubin present. The reduction of bilirubin to stercobilinogen may be imperfect in infants, and the removal of the normal flora by the action of oxytetracycline undoubtedly slowed down the process. Repeated stool examinations did not show any pathogenic organisms. Apart from the small blood transfusion mentioned, no other treatment was given except for several injections of vitamin B₁₂ on the basis that a great deal of blood regeneration was necessary to compete with the haemolysis that was occurring. Between December 11 and 17 there was pyrexia of 99 to 101° F. (37.2 to 38.3° C.), but from the 17th onwards he remained afebrile.

From December 22, when the haemoglobin rose steadily, there were no setbacks, apart from a cross-infection with *E. coli* type O127 which led to a mild gastro-enteritis on January 3, 1957. The blood count was unaffected, but on this occasion the patient was treated with neomycin. By January 7 the haemoglobin had risen to 12.3 g./100 ml. (83% Haldane) and the red cells appeared normal. Follow-up on February 12 showed exactly the same figures.

COMMENT

From the history and clinical examination the case appeared to be one of infantile gastro-enteritis. A smooth *E. coli* was isolated from the stool which was not a recognized pathogenic type, but which might nevertheless have been a pathogenic strain. This has occurred not infrequently, and some strains have been identified by Dr. Joan Taylor, at Colindale, as identical with those causing gastro-enteritis from other areas.

A severe haemolytic anaemia developed about 10 days after the onset of the illness. Haemolysis continued, and in spite of a very active marrow no rise in haemoglobin was recorded for about 10 days. The red-cell fragility was normal and no circulating antibody was demonstrated by the Coombs test. The nature of the haemolysis remains unknown. At the time of onset of the haemolytic anaemia the child appeared to have recovered from gastro-enteritis and there was nothing to suggest a source for the circulating toxin.

The acute haemolysis occurred after a course of oxytetracycline. Thrombocytopenic purpura following this antibiotic has been described by Welch *et al.* (1954) and Beckett *et al.* (1955). The former mention the occurrence of other blood dyscrasias, but make no direct reference to haemolytic anaemia. A search of the literature has not revealed a previous case; nevertheless, oxytetracycline remains a possible cause.

Cross-infection later with another known pathogenic type of *E. coli* (O127) led to a mild gastro-enteritis on January 3, but the blood count was unaffected. This infection was treated successfully with neomycin. Subsequent follow-up has not shown any abnormality in the blood picture.

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Chart of Case.

O128 sera are used routinely). On December 12 only rough *E. coli* were isolated. The haemoglobin on December 7 was 12.6 g./100 ml. (85% Haldane); the Chart shows the further levels.

On December 12 the child was noted to be rather pale; his temperature was 99° F. (37.2° C.). Next day the pallor was marked and there was a subconjunctival haemorrhage in the right eye. The sclerotics showed a suspicion of icterus. He became progressively more ill and on the 15th there was marked air hunger. A small transfusion of about 200 ml. of compatible group A Rh-negative blood was given. The haemoglobin rose slightly on the 16th, but fell again next day. From December 22 there was a steady rise in the haemoglobin value. From December 12 to 22 the bone marrow was very active, but was only able to maintain the haemoglobin at a constant level. The circulating red cells showed an astonishing degree of anisocytosis. Many macrocytic cells were present together with about 5% normoblasts and a maximum reticulocytosis of 23%. The white-cell count increased, reaching a peak of 24,100/c.mm. with many myelocytes and metamyelocytes.

On December 14 the serum bilirubin was 1.3 mg./100 ml. and the liver-function tests were normal. Next day the direct Coombs test was negative on two specimens of blood. On the 19th the test for occult blood in the faeces was negative. The faecal stercobilinogen content was 130 mg./100 g. of moist stool; this figure was thought to be too low for a patient suffering from haemolytic anaemia until it was realized that in an infant's stool most of the pigment was likely to be in the form of bilirubin. There was insufficient material for a quantitative assay of bilirubin in the stool, but qualitative tests showed a very large quantity to be present. On December 21 the faecal stercobilinogen content was less than 20 mg./100 g. of moist stool; bilirubin amounted to 400 mg./100 g. of moist stool. A week later