an acute lymphoblastic leukaemia, being, of course, fully aware of the difficulty of classifying very primitive and atypical cells.

The series of related events in our case might be expressed as :



Summary

A case of ankylosing spondylitis complicated by amyloid disease and renal vein thrombosis is described. Treated by irradiation, the patient subsequently developed acute leukaemia, probably lymphatic, and succumbed. The interrelation of the various conditions is discussed.

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REFERENCES

Abbatt, J. D., and Lea. A. J. (1956). Lancet, 2, 1317. Court Brown, W. M., and Abbatt, J. D. (1955). Ibid., 1, 1283. Harrison, C. V., Milne, M. D., and Steiner, R. E. (1956). Quart. J. Med., 25, 285.

25, 285. Luxton, R. W. (1953). Ibid., 22, 215. O'Beirn, S. (1953). Irish J. med. Sci., p. 181. Smith, W. G., and Williams A. Wynn (1955). Lancet, 2, 175.

ENTEROPATHOGENIC ESCHERICHIA **COLI SEROTYPES: INFECTION OF NEWBORN THROUGH MOTHER***

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The epidemiology of gastro-enteritis caused by the enteropathogenic Escherichia coli serotypes has hitherto been studied, with exceptions, in paediatric clinics and nurseries (Taylor et al., 1949; Rogers and Koegler, 1951; Rogers, 1951; Taylor, 1951; Taylor and Charter, 1952; Ocklitz and Schmidt, 1954; Adam, 1956; Rogers and Cracknell, 1956). Outside these establishments it has been difficult to follow the chain of infection (Braun, 1956).

In the children's hospital or ward the primary infection usually originates in the sick babies, but occasionally adults in the nursing staff are carriers of the germ (Ocklitz and Schmidt, 1955), although not ill. The spread of the infection in hospitals may also be caused through contamination of food of babies during preparation (Ocklitz and Schmidt, 1954).

Contact between infants outside hospital is possible too; in Germany the babies are often assembled in the waiting-rooms of the child welfare institution, and they may get infected in the consultation-room. There may also be cross-infection from infant to infant in the family

itself. But all these factors fail to explain the relatively high percentage of healthy carriers among the infants in the general population.

During earlier investigations we found pathogenic E. coli in 5.8% of the infants who had not had contacts with infected babies (Ocklitz and Schmidt, 1954). These results correspond with those of other authors (Taylor, In explaining such results, it is 1953 ; Braun, 1956). again necessary to consider the part played by the contamination of food. This leads us to the person who cares and cooks for the baby every day-namely, the mother-and her epidemiological role in E. coli diarrhoea.

We have tried to examine the factors which link the mother and her baby, especially the newborn baby, in this infection. In our examination we identified pathogenic strains of the well-known E. coli O groups 111, 55, 26, 86, and 44, and the type O25:L11:H6, which we isolated in a ward of our hospital during a severe epidemic of coli-enteritis (Ocklitz and Schmidt, 1952). We can only briefly deal with serological methods, referring to other more detailed literature (Kauffmann, 1954; Kauffmann and Ørskov, 1956).

Material and Method

Cultures were made from the faeces and lochia of women, lochial specimens being collected on throat swabs. From newborn infants rectal swabs were taken, using a sterile Drigalski spatula, care being taken to obtain traces of faeces. Media were inoculated at once at the patient's bedside. To avoid a confluent growth we inoculated several culture dishes, using Endo, Drigalski, and Leifson media (Schmidt, At the same time the antibiotic resistance was 1956. tested.

After 18 hours' incubation, and after the exclusion of the Salmonella and Shigella groups of bacteria, the E. coli colonies were tested with diagnostic sera by way of slide agglutination. We examined at least 10 single colonies or a sample of the bacterial growth, using two polyvalent sera of the following E. coli types:

Serum I: O111:B4:H-; O55:B5:H-; O26:B6:H-.

Serum II: O25:L11:H6; O86:B7:H34; O44:L74:H18.

These sera were so composed as to avoid overlapping reactions of their antigens. In this way a preliminary diagnosis was made which was confirmed in further tests. Having taken pure cultures, we started with the tube agglutination. In this agglutination we tested separately the O and B antigens. At the same time the strain was examined in a U-tube for its motility in semi-stiff medium (Kauffmann, 1954). If the strain was motile we identified the H antigen by means of the immobilization test, using H sera (Kauffmann and Ørskov, 1956).

Results

Mothers in the maternity wards of the gynaecological clinic of the University of Rostock were examined. In 22 (8.8%) of the 250 specimens, pathogenic E. coli serotypes were isolated from the stools or lochia before or after the delivery (Ocklitz and Schmidt, 1955) (see Table I). The fact

TABLE I.-Results of Examination of Women in the Gynaecological Clinic

Specimens	Cases	Negative Proof (No Growth on Endo Medium)	Serotypes of E. coli				
			0111: B4	O55: B5	O26: B6	O25: L11	Total
Last stool before delivery	100	— <u> </u>			1	6	7
delivery Lochial smear	100 50	15	3 1			11	14 1

^{*}Paper, in extract, read by one of us (H. W. O.) at the eighth International Congress of Paediatrics in Copenhagen, Denmark, July, 1956.

that the percentage of positive results was higher after delivery than before indicates the role of the hospital environment as an infecting agent.

The nurses change the napkins and feed the babies. In our clinic we found *E. coli* on the hands of the nurses, in spite of washing and disinfecting; we also found *E. coli* on the rubber teats after they were put on the bottles by the nurses. We found, too, that the nurses were carriers of pathogenic *E. coli* in their own stools. In the gynaecological clinic we found that, at times, almost one nurse out of four working in the labour and other maternity wards for mothers and their newborn babies had enteropathogenic *E. coli* serotypes in her stools (see Table II). As the

 TABLE II.—Results of Examination of Nurses in the Maternity Clinic

Nurses	No.	Enteropathogenic Serotypes of E. coli						
		O111:B4	O55: B5	O26: B6	O25:L11	Total		
In labour room In maternity wards	17 16			2	4 3	6 3		
born babies	10			'	2	2		

nurses in the gynaecological clinic have to care for both babies and mothers, the possibility of the nurse infecting them both is evident. In the spread of pathogenic coli infection, direct contact plays the dominant role compared with both infection of feeds and aerial spread. While infants are touched by doctors only during examination, and not at all by cleaners, they are in the closest contact with the hands of the nurse.

Of approximately 2,000 newborn infants that have passed through our clinic during the last five years, 68 were found to be excreting pathogenic *E. coli* serotypes. All these infants were admitted during the first 12 days of life. The first rectal swab was taken within the first hour after their admission, the second swab some days later. From 16 of these 68 babies positive results were obtained from second swabs only, the first swabs having proved to be negative. In other words, these results were from babies who had, with reasonable certainty, been infected in our hospital, and, in view of this, we excluded these babies from any further consideration (see Fig. 1).



FIG. 1.—Number of newborn infants found with enteropathogenic *E. coli* in the first swab (white columns) and second swab (shaded columns).

We were therefore left with 52 newborn babies in whom the first rectal swab immediately after admission gave a positive result (see Fig. 1). *E. coli*. O111:B4:(H2), isolated from 35 cases, was the most frequent type, being twice as common as all other types—O26:B6:H11, nine cases; O55: B5:(H6), five cases; and O44:L74:(H18), three cases.

How could the newborn infant have become infected? Let us consider first the steady increase in positive findings from the third day onwards (Fig. 1). This cannot be due entirely to infection of infants by attending nurses, as about a third of the deliveries were at home, and therefore the mother could have been the infecting source, although we were unable to investigate this. But that such a postnatal infection of the infant by the mother is possible can be seen from our findings in another context-the isolation of pathogenic coli from stools and from breast milk collected from milk donors. In order to investigate this possibility we examined the donors of human milk, and found in the milk and in the faeces the same type of enteropathogenic coli bacteria (Ocklitz and Schmidt, 1955). Those mothers who gave positive results were delivered in the gynaecological clinic of Rostock University. In this earlier investigation the infection of infants by mechanically collected raw breast milk was demonstrated. Frequently an infant may be fed with the milk pumped from its own mother at the end of the preceding feed; in this instance, too, infection of the infant by its mother is possible, while an infant fed exclusively at the breast may escape infection.

From Fig. 1 it is obvious that a steep decline in the number of positive results occurs during the first two days; this precedes the gradual ascent. This change during the first two days is explicable only in connexion with the delivery. It would not be reasonable to regard the nurse as the only source of infection, as a high percentage of women are delivered without nurses in their own homes. As proof that the baby was infected by its mother during delivery, the same *E. coli* serotype would have to be found in the mother's stool before delivery and in that of the baby immediately after birth.

We made such an investigation. From women coming for consultation to the antenatal clinics we collected stools and investigated them bacteriologically. After delivery we examined the newborn babies in the same way with rectal swabs. This investigation has not yet been completed. The preliminary results obtained from the examination of 111 mothers and their babies are as follows: in five cases pathogenic E. coli strains were found in the stools of the mothers alone, and in three cases in the infant only. The coincidence of positive results in mother and baby was less often seen. In one case we found the same enteropathogenic E. coli serotype in the mother's stool on the last day before her admission to the clinic and, after delivery, in the rectal swab of the newborn. This we may regard as proof of infection during birth. Furthermore, the fact emerges that the possibility of such infection is greater than is often realized.

One may logically include the possibility of an *intrauterine* infection too, as, for example, in a case of premature rupture of the membranes. It is very difficult to obtain evidence of such an infection—much more so than an infection during delivery. In the following case we succeeded.

A newborn baby was delivered by caesarean section because of a premature rupture of membranes, cessation of labour, and inertia uteri after a delayed birth of four weeks. The baby was admitted to the children's clinic within half an hour of birth. On immediate examination we were struck by the penetrating smell pervading it—an odour like that of semen and typical of the enteropathogenic *E. coli* O111. The diarrhoeic stool passed during the examination had the same odour, and was unlike meconium. We immediately took a rectal swab; bacteriological examination of this revealed, apart from *Pseudomonas pyocyanea*, the enteropathogenic *E. coli* O111:B4:H2. The maternity hospital informed us that the amniotic fluid had been a faded muddy-green, and also had a penetrating seminal odour. A bacteriological examination of the fluid was not made. Mother and child were treated with chlortetracycline and made a satisfactory recovery, without complications.

It was not possible to pin-point the actual time of infection. In all probability it occurred during rupture of the membranes. The fact remains that the infection was intrauterine.

It was noticed that the earlier in life these babies were infected the milder was the infection (see Fig. 2).



Conclusion and Summary

It can be assumed that in the maternity clinics, where child-bearing mothers are assembled, the possibility of infection with pathogenic E. coli types exists in just the same way as between infants in the infant wards. The transmission of these pathogenic germs may be from, infant to infant through the nurses and other medical staff. In the same way, the mother may be infected by the nursing or medical staff. The latter possibility, if perhaps not frequent, is still of epidemiological importance; for the mother, who will in the future nurse the baby, leaves the hospital carrying the infection with her. Furthermore, we may assume the possible transmission of pathogenic E. coli serotypes from mother to child in all phases of delivery and after.

The relatively high percentage of positive isolations of E. coli serotypes obtained from babies born at home suggests that not all their mothers could have been infected in hospital. This does not detract from the importance of the clinical infective chain suggested in this communication.

The results of these investigations show the urgent need of an increased stress on prophylaxis. This will mean intensive instruction of the nurses and medical staff, and bacteriological control of all their work in hospital (Ocklitz, 1956). At home such prophylaxis is more difficult to attain. It is evident that our prophylactic efforts in and outside the hospital may be useful not only in controlling infections such as infantile diarrhoea, but also in combating all other infections, especially those caused by strains of staphylococcus.

REFERENCES

Adam, A. (1956). Säuglings-Enteritis. Thieme, Stuttgart. Braun, O. H. (1956). In A. Adam's Säuglings-Enteritis, p. 83. Kauffmann, F. (1954). Enterobacteriaceae, 2nd ed. Munksgaard, Copen-

- hagen.
- Ocklitz
- Rogers, K

- Schmidt
- Addamis, J. (1953). Embeddetchatetat, 2nd Cd. Multisgatt, Copen-hagen.
 and Orskov, F. (1956). In A. Adam's Säuglings-Enteritis, p. 1.
 Ocklitz, H. W. (1956). In A. Adam's Säuglings-Enteritis, p. 491.
 and Schmidt, E. F. (1952). Disch. Gesundh-Wes., 7, 777, 809.
 (1954). Arch. Kinderheilk., Beiheft 28.
 (1955). Helv. Paediat. Acta, 10, 450.
 Rogers, K. B. (1951). Proc. roy. Soc. Med., 44, 519.
 and Koegler S. J. (1951). J. Hyg. (Camb.), 49, 152.
 Schmidt, E. F. (1955). In A. Adam's Säuglings-Enteritis, p. 43.
 Taylor, J. (1951). Proc. roy. Soc. Med., 44, 516.
 (1953). VI Congresso internazionale di microbiologia Roma, September, 1953. 4, 251.
 and Charter, R. E. (1952). J. Path. Bact., 64, 715.
 Powell, B. W., and Wright, J. (1949). Brit. med. J., 2, 117.

Medical Memorandum

Mongolism in a Twin

Mongolism as a clinical entity has attracted a great deal of attention in the literature of oligophrenia, and its occurrence in one of twins has been considered to be so rare as to justify case reports. In a summary of such case reports it was suggested by Morris and MacGillivray (1953) that "mongolism in one of twins is not so rare in British literature as has been suggested," and two such cases were reported, bringing the total to 14 since 1876. A further case is here put on record.

CASE HISTORY

The father, aged 66, is a tradesman, and has been separated from the mother for 16 years; his history indicates that he may have been a psychopath. The mother, aged 65 years, is of normal intelligence. There were four other children, born in 1914, 1915, 1922, and 1924. All are married and there is no sign of abnormality in their offspring. There was no history of miscarriages. The paternal grandparents were normal, having four male and two female children. Of this family, one girl had twin daughters and one brother twin sons. The maternal grandparents were normal and had 14 children without history of twinning.

The twins were born on June 11, 1927, after a normal pregnancy, the boy first of normal weight and the patient, a girl, one hour later; she was only 4 lb. (1.8 kg.) in weight, was heavily jaundiced, and was an obvious mongol. The boy was normal and healthy up to his death in a road accident at 13 years of age. The girl was cared for by her mother until her excitable and destructive behaviour and her mother's ill-health necessitated admission to this hospital. She developed major epilepsy at the age of 14 and has had seizures at intervals since.

The patient is a typical mongol. She is 4 ft. 2 in. (1.27 m.) tall and weighs 9 st. 1 lb. (57.6 kg.). She has a brachycephalic skull, fissured tongue, and low-set ears with rudimentary pinnae. Her eyes are blue ; her hair scanty, straight, and light brown. The arms are short, the fingers stubby, the little fingers incurved, and the thumbs low-set and short; all joints are hyperextensile. The lower limbs are short; there is some bowing of the tibiae, and marked separation between the great and adjoining toes. She averages one major fit a month; menstruation, established at 15 years of age, is normal and regular; her W.R. and Kahn reactions are negative.

She has a mental age of $2\frac{1}{2}$ years on the Terman-Merrill Revision of the Binet scale, Form L, and a social age of 2 years on the Vineland scale. Her speech is mainly an incoherent babble, and her feeding and toilet habits are at the 18 months level. She frequently shows violent temper tantrums, spitting and scratching at those near her. These outbursts are accompanied by head-banging and pulling at her teeth. She has been observed to show aggressive homosexual behaviour.

COMMENT

In this case few aetiological factors emerge, beyond a history of twinning on the paternal side, and, as Benda (1947) concluded that only 63 cases of mongolism in one of twins reported in world literature could reliably be used in discussion, it was considered advisable to report this case.

My thanks are due to Miss C. M. Mathieson, our Senior Clinical Psychologist, for psychometric assessments, and to Mr. A. C. Adams, Group Psychiatric Social Worker, for detailed family investigations.

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REFERENCES

Benda, C. E. (1947). Mongolism and Cretinism. Heinemann, London. Morris, J. V., and MacGillivray, R. C. (1953). J. ment. Sci., 99, 557.