Scotland and Ayrshire Divisions of the British Veterinary Association, without whose help the work could not have been accomplished.

ADDENDUM.—Since this paper was written a further 44 cases have been investigated. From humans with suspected animal ringworm the laboratory results were: *T. verrucosum*, 31; *T. mentagrophytes*, 3; *T. rubrum*, 1; *T. sulphureum*, 3; micropositive, 5; negative, 1. The results from specimens obtained from 23 of the animals were: *T. verrucosum*, 11; *T. equinum* and *M. equinum*, 1; micropositive, 4; negative, 7. Horses were given as the source of two human infections which were positive by microscopy only. *T. verrucosum* was isolated from one of the horses and both *M. equinum* and *T. equinum* from the other.

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INFANTILE HYPERCALCAEMIA WITH KERATOPATHY AND SODIUM DEPLETION

BY

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Infantile hypercalcaemia, a condition which presents with failure to thrive, is known to occur in two forms, the benign (Lightwood, 1952) and the severe (Schlesinger, Butler, and Black, 1956). The essential feature in both forms of this condition is the raised serum calcium level, and, although the cause of this remains uncertain, the symptoms of anorexia, vomiting, and failure to thrive are ascribed to this increase in the serum calcium.

Treatment, therefore, is directed towards bringing about a fall in the serum calcium level, and to this end the child's intake of calcium is reduced. A low calcium milk, a proprietary preparation of which is now available ("locasol"), is usually given and no vitamin D supplements are allowed. In the case described below treatment in this way was successful in bringing about a fall in the serum calcium level, but was followed by a marked degree of sodium depletion. The case is also unusual in that during the course of the illness a band keratopathy developed, and, while this has been described as occurring in a number of conditions associated with a raised serum calcium level (Albright and Reifenstein, 1948), it does not appear to have been described hitherto in a case of infantile hypercalcaemia.

Case Report

The patient was first seen at the age of 1 week, when he presented with many extensive areas of subcutaneous fat necrosis of typical distribution—the external surfaces of the limbs, the buttocks, and the posterior part of the chest wall being affected. He seemed otherwise well at that time, but his general progress thereafter was unsatisfactory, so that by the time he was 3 months old he was only 9 oz. (255 g.) above his birth weight. He then developed a bilateral otitis media and, a few days later, persistent vomiting, which necessitated his admission to hospital.

He was pyrexial, temperature 105° F. (40.6° C.), he had bilateral otitis media, and one of the areas of fat necrosis situated in the left deltoid region was now fluctuant and evidently the site of a secondary infection. This subsequently had to be incised, and about 1 oz. (28 ml.) of thick yellow pus was evacuated. Swabs were taken of the aural discharges and a heavy growth of *Staphylococcus aureus*, coagulase-positive, was obtained. This organism was sensitive to penicillin, streptomycin, chloramphenicol, erythromycin, and the tetracyclines.

He was treated with intramuscular penicillin, 100,000 units six-hourly, intramuscular streptomycin, 100 mg. twelve-hourly, and sulphadiazine, 0.25 g. six-hourly. Initially he appeared to respond, but within 48 hours had developed head retraction, and a lumbar puncture showed the cerebrospinal fluid to be opalescent. Analysis of this gave the following results: leucocytes, 300 per c.mm., 70% of these being lymphocytes; protein, 100 mg. per 100 ml.; chlorides, 675 mg. per 100 ml.; sugar, 62 mg. per 100 ml. The culture was sterile.

He remained acutely ill for about two weeks, during which time he had several convulsions, and he was intermittently febrile. Treatment was continued with chloramphenicol and later with oxytetracycline, and the C.S.F. became normal in every respect.

It was thought likely that he had had an intracranial thrombophlebitis, and his vomiting was at first ascribed to the effects of this. But not only did he continue to vomit intermittently, he also became increasingly fretful, anorexic, and constipated. His urine contained a trace of albumin with an occasional granular and hyaline cast, and examination of his blood on August 8, 1956, by which time he was 4½ months old, gave the following results: serum calcium, 20 mg./100 ml.; serum phosphorus, 5.2 mg./100 ml; alkali reserve, 56.7 vols per 100 ml.; serum sodium 330 mg./100 ml.; serum potassium, 20.5 mg./100 ml.; serum chloride, 625 mg./100 ml.; urea, 64 mg./100 ml.; blood cholesterol, 182 mg./100 ml.

A diagnosis of infantile hypercalcaemia was now made and he was started on treatment with locasol. One week later the serum calcium had fallen to 13.5 mg. per 100 ml. and, after a further week, to 11.3 mg. On all subsequent occasions the serum calcium was found to be within normal limits.

In spite of the fact that his serum calcium remained normal, his symptoms showed no improvement. He remained fretful, anorexic, and constipated. It was possible to induce him to take only in the region of 20 oz. (570 ml.) of milk a day, although he was thirsty and would readily take extra drinks of water. He showed no consistent gain, his weight fluctuating between 10 and 11 lb. (4.5 and 5 kg.).

On October 31 the electrolytes were checked once more. The findings were within normal limits, serum sodium being 310 mg. per 100 ml. When these determinations were repeated on January 1, 1957, it was found that there had been a marked fall in the serum sodium level to 285 mg. per 100 ml.

It was decided that, without any other change in his feeding regime, added sodium chloride should be given, and this was done by making up the feeds of locasol with one-fifth normal saline instead of with distilled water. This increased his intake of sodium chloride by approximately 1 g. a day. There was an immediate change, which was apparent first in the child's behaviour. He became a happy, friendly, and responsive infant. He also ceased vomiting, his appetite increased, until he was taking 40 oz. (1,140 ml.) of milk a day. When solids were introduced into the diet, these too were taken well and the sodium chloride supplement was discontinued. His weight increased steadily during the next six weeks, the increase averaging 12 oz. (340 g.) a week. His serum sodium level at the end of a week had

risen to 300 mg. per 100 ml. and, after a further week, to 305 mg. He was discharged home on February 14, and his good progress continued.

Bilateral corneal changes were first noticed on July 8, when he was 3½ months old. These consisted in both eyes of a horizontal band of opacity present in the superficial corneal layers, and having the appearances described by Albright and Reifenstein (1948) as band keratitis. These opacities gradually diminished, and at the time of his discharge both corneae had returned to normal.

Discussion

The most usually adopted form of treatment in infantile hypercalcaemia is the substitution of a low-calcium milk for ordinary milk. Locasol, as reconstituted in the proportions advised by the manufacturers, has a calcium content of not more than 56.8 mg. per pint (100 mg. per litre). The sodium content is also less than that which is found in cow's milk, 1 pint (570 ml.) of reconstituted locasol containing 0.28 g. of sodium chloride, while cow's milk contains 0.78 g. of sodium chloride per pint. According to Butler and Talbot (1944) the average requirements of sodium chloride per day in infancy is about 1 g. Locasol, therefore, while being low in calcium, also provides an intake of sodium which is less than the average normal requirements. Two factors appear to have been responsible for this child's sodium depletion. Firstly, the diet which was offered was of itself deficient in sodium, and, secondly, the intake of milk was severely reduced because of anorexia. That his symptoms of anorexia, vomiting, and failure to thrive, which initially were due to hypercalcaemia, were eventually due to sodium depletion was shown by the immediate improvement in the child's condition when the intake of sodium was increased, and also by the rise in serum sodium level which followed. The insidious way in which the sodium depletion developed is particularly noteworthy and indicates the need for regular determinations of the serum electrolyte levels in similar cases.

According to Albright and Reifenstein (1948) two types of lesion occur in cases of hypercalcaemia, whether due to vitamin D poisoning, hyperparathyroidism, or other causes such as sarcoidosis or the syndrome associated with excessive milk and alkali intake: (1) a band keratopathy as was seen in the present case, and (2) conjunctival crystals, which appear medially and laterally to the limbus. Both these lesions are said often to be visible only with the slit-lamp. In this case changes were present at the age of 3½ months; they were clearly visible to the naked eye and, had their significance been appreciated, the diagnosis of hypercalcaemia would have been made a month earlier than it in fact was.

Summary

A case of infantile hypercalcaemia is described in which corneal opacities developed in the early stages of the disease and sodium depletion was a late complication.

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A gramophone record on how to relax has been made by Miss MARGARET SMITH, M.C.S.P. It is intended for the "tense" patient. On the first side Miss Smith discusses the importance of abolishing tension and describes the technique of relaxation in a general way; on the other side the patient is given detailed directions, in slow time, on how to relax the muscles of the various parts of the body. The record (No. RSL/1243) is obtainable from Recorded Sound Ltd., 127, Bryanston Street, London, W.1, price 21s. (postage 2s.). Royalties on the sale of the record are being given to the Iona community.

THE SEQUELAE OF KERNICTERUS

BY

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It is well recognized that neurological symptoms in later life may follow haemolytic disease of the newborn, and a number of such cases have been reported. Lande (1948) described eight cases and Evans and Polani (1950) reviewed the literature, adding 16 cases of their own. The serological diagnosis of the disease at birth has been satisfactory only since Coombs, Mourant, and Race (1946) introduced the anti-human-globulin test, and all the published reports have been of children or young adults. Little is known at present about the subsequent fate of affected children, and the following case showing neurological sequelae in an adult may be of interest, especially since it was possible to obtain confirmation of the aetiology by laboratory tests.

Case Report

A warehouseman aged 44 was admitted to hospital under the care of Sir Charles Symonds following an epileptiform attack during which he had lost consciousness for a few minutes. He stated that he had suffered for many years from episodes occurring two or three times a year, each lasting a few minutes, during which he felt dissociated from his surroundings and objects appeared to recede into the distance. He had lost consciousness on only one previous occasion five months earlier, when he had an attack in bed. That episode had been preceded by an involuntary cry, and terminated with incontinence of urine. The following history was obtained from the patient and his mother.

The patient was the younger of two siblings. His elder sister had died of tuberculosis, and his father was also dead. The patient had become deeply jaundiced soon after birth and had remained so for a few days. When 5 years old he developed chorea. He recovered from this slowly, and at the age of 10 was well enough to attend school, where his record was satisfactory. His subsequent development was uneventful.

The birth and childhood of his elder sister were normal. His mother had never had a blood transfusion nor an intramuscular injection of blood. She had had no miscarriages. No other member of the family had suffered from epilepsy.

On clinical examination the patient appeared to be of normal intelligence, and no abnormal physical signs were found other than a slight dysarthria.

Investigations.—The C.S.F. was normal. The Wassermann and Kahn tests in blood and C.S.F. were negative. An electroencephalogram was normal. An audiogram revealed bilateral perceptive deafness. An *x*-ray picture of the skull showed extensive calcification in the region of the basal ganglia and dentate nuclei. No similar changes were found in *x*-ray pictures of his mother and of three maternal relatives.

The combination of calcification of the basal ganglia and perceptive deafness, together with his history, suggested that he might have been affected by haemolytic disease of the newborn. His blood group was A Rh-positive, probable genotype CDe/cde. His mother's group was A Rh-negative, genotype cde/cde, but no antibodies to the Rh antigens C or D were detected in her serum, and an indirect anti-human-globulin test was negative. As she was aged 70, and in good health, it was thought justifiable to investigate the effect upon her of a small injection of Rh-positive blood. With her permission, after the nature of the investigation had been explained to her, she was given an intravenous injection of 5 ml. of fresh heparinized group A Rh-positive