Case 1. She was the tenth-born in her family and weighed 2.75 kg. (6 lb.) at birth. She also was fed on Ostermilk No. 2, reconstituted as follows: $2\frac{1}{2}$ measures Ostermilk No. 2 + $3\frac{1}{2}$ measures water + 1 teaspoonful of sugar, four-hourly. Her initial sodium level was 194 mEq/l., potassium 7.1 mEq/l., and chloride 156 mEq/l. The carbon dioxide combining power was 17 mEq/l., and the blood urea nitrogen was 198 mg./100 ml.

Treatment was carried out as in Case 1, and within 48 hours her electrolyte values had returned to normal. Convulsions occurred repeatedly throughout that period despite anticonvulsant therapy and the maintenance of normal blood sugar and calcium levels. She died suddenly at 51 hours, however, and necropsy permission was refused.

COMMENT

In both cases hypernatraemia was caused by excessive administration of solute in relation to fluid intake owing to improper reconstitution of dried-milk formulae. In Case 1 the mother had prepared feeds, using double the powder content recommended, but she believed that one tablespoon contained one fluid ounce of water. In Case 2 the initial feeding history obtained seemed quite normal. It only became clear on specific questioning that the mother used the dried-milk scoop for measuring fluid in the belief that each scoop contained a fluid ounce of water. The feeding instructions issued by the manufacturers of three commonly used dried-milk preparations—namely, Ostermilk No. 2, full-cream Cow and Gate, and full-cream National Dried Milk—would be quite clear to most mothers (see Table), but none of those instructions make it absolutely clear that the measure supplied is for dried milk

and not fluid. The National Dried Milk instructions do indicate that one fluid ounce is approximately two tablespoons and for that reason are the most explicit. It is probably quite fortuitous that in both cases Ostermilk No. 2 was the dried milk powder used, but further errors could probably be avoided if feeding instructions were even more simple than at present. Thus it should be made clear that the measure supplied is for dried milk only and not fluid, and that one fluid ounce is approximately equal to two tablespoonfuls.

Comparison of Feeding-tables for Normal Babies of Normal Weight at 1 Month (Extract from Manufacturers' Instructions)

Preparation	Dried-milk Content	Fluid Content
Ostermilk No. 2	Level measures, $3\frac{1}{2}$	Hot water, fluid oz. 4
Cow and Gate (Full-cream)	" " $3\frac{1}{2}$	Make up with hot (boiled) water to 4 oz.
National dried milk (Full-cream)	Amount of milk powder for each feed (in level measures), $2\frac{1}{2}$ or 3	Boiled water in ounces (1 oz. = 2 tablespoons approx.), $3\frac{1}{2}$ or 4

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Anaemia Due to Blood Loss from the Telangiectases of Scleroderma

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Telangiectases affecting the face, neck, oral mucous membranes, and extremities are not uncommon in scleroderma. In appearance they resemble closely those found in Osler-Weber-Rendu disease, though they are not hereditary (Verel, 1956; Winterbauer, 1964). However, it has been said that in contrast with Osler-Weber-Rendu disease the telangiectases of scleroderma rarely bleed and do not cause significant loss of blood (Winterbauer, 1964; Carr *et al.*, 1965). This report concerns two elderly women with scleroderma and telangiectases who have been gravely debilitated by severe hypochromic anaemia, for which the most likely explanation was gastrointestinal blood loss from telangiectases.

CASE 1

A 74-year-old widow was first seen elsewhere in 1958, when she complained of the symptoms of Raynaud's phenomenon for many years and the development of red blotches on the face. A diagnosis of scleroderma with telangiectases was made and as there was no evidence of systemic involvement a good prognosis was given. The blood was not examined, though two years previously her haemoglobin had been only 75%. Occasionally her nose had bled, but this had never been serious. None of her family had been troubled by anaemia or telangiectasia.

In October 1965 she was admitted to hospital because of increasingly severe symptoms of anaemia. She was pale, with striking telangiectasia affecting the face, lips, buccal mucosa, tongue, hands, and feet (see Fig.). The telangiectases varied in size up to a maximum of 5 mm. in diameter; they did not blanch on pressure. The skin of the fingers and toes was thickened and tense, with shortening of

the terminal phalanges of some fingers. Elsewhere the skin was normal, but both normal and sclerotic skin was affected by telangiectasia. There were the signs of congestive heart failure. Blood pressure was 180/80 mm. Hg. Sigmoidoscopic examination was normal and mucosal telangiectases were not seen.

Investigations.—Hb 4.8 g./100 ml.; M.C.H.C. 24%; reticulocytes 5%; blood film—marked hypochromia; W.B.C. 13,200/cu. mm.; E.S.R. 14 mm./hour. Blood urea 65-59 mg./100 ml., plasma calcium 4.8 mEq/l., plasma proteins 6.6 g./100 ml., serum electrophoretic pattern normal. Antinuclear factor, gastric parietal cell, and thyroid antibodies negative. Urine normal. Faecal fat excretion 7.2 g./24 hours. E.C.G. normal. Radiological examinations: chest—marked cardiac enlargement, small pleural effusion on left side; hands—pronounced soft-tissue calcification affecting several digits; barium meal—dilated atonic oesophagus with impaired emptying, otherwise normal; barium enema—number of large sacculi in sigmoid colon.

Case 1. Telangiectases affecting face and lips.

The principal cause of the congestive cardiac failure was judged to be the severe anaemia. She was treated with diuretics and carefully transfused. Two weeks later the haemoglobin was 13.8 g./100 ml.; she was feeling better and was no longer in heart failure. However, despite iron by mouth her haemoglobin fell during the next six weeks to 10.5 g./100 ml., but after a further six months her haemoglobin was 12.4 g./100 ml. (M.C.H.C. 32%, reticulocytes 3.2%), though she had had further bleeding from the nose necessitating cautery of small telangiectases in both nares.

CASE 2

A 73-year-old widow, first seen in May 1957, complained of pain and tenderness in the tips of the fingers of both hands which had developed suddenly one year previously. The skin was thin and atrophic and the hands were blue and cold. Radiological examination showed small erosions of the tips of the terminal phalanges. The haemoglobin was 13.3 g./100 ml. and the peripheral blood film normal.

A diagnosis of scleroderma was made. She was seen at frequent intervals with recurrent ulceration of the fingers and Raynaud's phenomenon. One year later telangiectases on the face, lips, and hands were noticed. None of her relatives had had similar lesions. A radiograph of the hands in 1960 showed calcification of the tip of one index finger. Barium study of the oesophagus with the patient in the prone position showed no peristalsis.

In January 1965 she was admitted to hospital because of shortness of breath on effort for six weeks and melaena for four days. She was pale and now had more numerous telangiectases on the face, lips, and arms; the buccal mucosa was spared. The hands showed marked sclerodactyly, the skin being thickened and atrophic with scarring of the fingers and wasting of the pulp. The skin elsewhere, notably on the face, was of normal texture. There were signs of mild congestive cardiac failure. Blood pressure was 120/60 mm. Hg. Sigmoidoscopic examination was normal.

Investigations.—Hb 7.5 g./100 ml.; reticulocytes 11.2%; blood film—hypochromia; M.C.H.C. 31%; W.B.C. 9,600/cu. mm.; E.S.R. 34 mm./hour; serum iron 6.8 µg./100 ml. Blood urea 39 mg./100 ml.; plasma calcium 5 mEq/l.; plasma phosphate 3.7 mg./100 ml. Plasma proteins 5.5 g./100 ml.; serum electrophoretic pattern normal. No L.E. cells detected; antinuclear factor, gastric parietal cell, and thyroid antibodies negative. Faecal fat excretion 2.2 g./24 hours. E.C.G. normal. Urine normal. Radiological examinations: chest—normal; hands—no soft tissue calcification; barium meal—normal stomach and small intestine; barium enema—numerous small diverticula throughout colon.

After blood transfusion and administration of iron the haemoglobin rose to 12.6 g./100 ml. Digitalis and diuretics were given in addition, and the signs of cardiac failure disappeared. Over the course of the next 14 months, despite oral and parenteral iron, the patient required blood transfusion on four occasions. Occult blood was detected in the stools repeatedly. Later the gastrointestinal blood loss during a 15-week period of study using ^{59}Fe and a whole-body counter (Callender *et al.*, 1966; Holt *et al.*, 1967) was estimated to be 1.5 litres per 100 days. Absorption of an orally administered dose of 5 µc. $^{59}\text{FeCl}$ was 90%, normal for a patient with iron deficiency. Frank bleeding from telangiectases on the inner aspect of the lips has since occurred.

DISCUSSION

Telangiectases occur most frequently in the variety of scleroderma that consists of sclerodactyly, Raynaud's phenom-

enon, and subcutaneous calcinosis in the absence of marked systemic involvement. It characteristically has a good prognosis (Tuffanelli and Winkelmann, 1961; Winterbauer, 1964; Carr *et al.*, 1965). Both the above patients had this variety of scleroderma, which would have caused them little trouble had they not been disabled by anaemia.

The most likely explanation for their anaemia was continuous bleeding from telangiectases in the gastrointestinal tract. The blood loss is just compensated for by oral iron in Case 1, but the second patient required periodic blood transfusion. Both patients had diverticulosis of the colon, but the nature of the blood loss made it unlikely that this was the underlying cause. It is of interest that in Case 1 the radiological features of the diverticula were those of scleroderma (Heinz *et al.*, 1963). Scleroderma may be associated with intestinal malabsorption (Sonneveldt *et al.*, 1962), and the faecal fat excretion in Case 1 was marginally raised. However, poor absorption of iron was unlikely to be a factor in the pathogenesis of anaemia in either patient, since iron given therapeutically in the first case was sufficiently absorbed to correct the anaemia, and in the second case absorption of ^{59}Fe was normal.

The telangiectases of scleroderma cannot be distinguished clinically from those occurring in the Osler-Weber-Rendu disease (Verel, 1956; Winterbauer, 1964). It is therefore surprising that stress has been laid on the apparent absence of a haemorrhagic tendency (Winterbauer, 1964; Carr *et al.*, 1965). There is no doubt that internal telangiectases occur in scleroderma as in Osler-Weber-Rendu disease, since two patients discussed by Winterbauer who suffered gastrointestinal haemorrhage were shown to have mucosal telangiectases in the stomach, and in one case in the large bowel also.

We believe it probable that the telangiectases of scleroderma can cause clinically significant blood loss. They occur mainly in a variety of scleroderma that is otherwise benign, and, as this usually affects elderly patients, iron-deficiency anaemia should be searched for frequently, since any symptoms of anaemia may be wrongly attributed to age and ignored.

Dr. H. R. Vickers kindly referred the second patient to us.

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