In Case 8 the improvement has so far continued for six months after decompression. In Case 9 the paraplegia recurred four years later but there are now multiple metastases.

TABLE III

Case No.	Outcome	Length of Follow-up
1	Complete recovery	22 months
2	Death	
3	"	
4	Improvement following laminectomy for 2 years, when further metastases caused return of paraplegia, which was terminal	
5	Death probably due to radiation myelitis and not metastases. Temporary and partial improvement	
6	Complete recovery	3½ years
7	Death due to terminal quadriplegia	
8	Complete recovery	6 months
9	Complete recovery, but later on signs of further cervical cord compression were developing	4 years

The prognosis of thyroid cancer, particularly when well differentiated, may be surprisingly good, especially in the younger age groups (Halnan, 1966). Thus the presence of distant metastases may not be a terminal event even when paraplegia occurs, unless the latter itself is allowed to lead to death. Furthermore, bone metastases characteristically occur from follicular cancer with good uptake of radioiodine, as in all our cases that recovered. An optimistic and energetic approach seems justified in this type of case, quite different from the management of a patient with rapidly growing multiple metastases from undifferentiated carcinoma, whether originating in the thyroid or elsewhere.

Conclusions and Summary

Thyroid carcinoma is rare, but commonly metastasizes to bone, the well-differentiated variety sometimes spreading exclusively to bone.

Cases of paraplegia due to thyroid metastases are rare and few have been reported in the literature.

Metastasis to the spine causing cord compression should be treated as an emergency procedure and attempts to find the primary tumour should not delay this, particularly since in some cases no primary tumour may be found.

A thyroid tumour should always be considered as a possible origin of spinal metastases, and a history of thyroidectomy (even though apparently for benign disease) should add to rather than remove suspicion.

Though the results of surgical decompression for spinal metastases are often poor this should not rule out operation, since, even though life may not be prolonged, relief of the paraplegia is of considerable benefit. After thyroid metastases the extent and duration of recovery are significantly better than after most other tumours.

The duration of the paraplegia should not contraindicate operation. With thyroid metastases full recovery may occur even when two months have elapsed.

The first local treatment should be surgical, but this must be followed by treatment to both the primary and the secondary tumours; thyroidectomy is sometimes appropriate, but either or both radioiodine and *x*-ray therapy are almost always needed.

We are grateful to all colleagues at the Christie Hospital and elsewhere who have assisted in the care of these patients.

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Medical Memoranda

Need for Clarity in Infant Feeding Instructions

Brit. med. J., 1967, **3**, 536–537

The danger of hypernatraemia occurring as a result of accidental salt-poisoning or excessive administration of solute is well recognized in infancy (Finberg and Harrison, 1955). The present report describes two cases admitted to the Royal Hospital for Sick Children, Edinburgh, within the past year with hypernatraemic dehydration resulting from parental failure to interpret correctly the manufacturers' instructions for reconstituting dried-milk formulae.

CASE 1

A 4-week-old male infant was admitted to hospital in January 1966 with a two-day history of irritability, refusal of feeds, and a high-pitched cry. He had been born in hospital at full term. This was the mother's first pregnancy and was uncomplicated. His birth weight was 4 kg. (8.8 lb.). There were no immediate neonatal problems, but on discharge from hospital on the fifth day of life his feeding was changed from half-cream milk to Ostermilk No. 2, reconstituted as follows: 6 measures dried-milk powder+3 tablespoonfuls water+1 teaspoonful of sugar, four-hourly. Despite the paste-like consistence of this mixture he had accepted feeds until two days before admission to hospital. When admitted he was dehydrated, with early peripheral circulatory failure. His cry was high-pitched and feeble. Neurologically he was generally hypertonic, with neck retraction, twitching of the eyelids, vertical nystagmus, and tremor of the limbs. His abdominal wall was doughy in consistency. Examination showed nothing else of note. Initial electrolyte values were—sodium 207 mEq/l., potassium 7.5 mEq/l., chloride 165 mEq/l., and calcium 7.2 mEq/l. His *p*H was 7.08, P_{CO_2} 71 mm. Hg, and standard bicarbonate 15.8 mEq/l. The blood urea nitrogen was 250 mg./100 ml.

He was treated as recommended by Harrison and Finberg (1964), being gradually rehydrated with a solution containing 20 mEq sodium per litre. Over the next three days he had repeated convulsive episodes, but gradually the serum electrolytes and blood urea nitrogen levels returned to normal. His subsequent progress has not been satisfactory and he is now severely handicapped mentally.

CASE 2

This 4-week-old female infant was admitted to hospital in October 1966, with symptoms and clinical features identical to those in

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: 2½ measures Ostermilk No. 2+3½ *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, 3½	Hot water, fluid oz. 4
Cow and Gate (Full-cream)	" " 3½	Make up with hot (boiled) water to 4 oz.
National dried milk (Full-cream)	Amount of milk powder for each feed (in level measures), 2½ or 3	Boiled water in ounces (1 oz. = 2 tablespoons approx.), 3½ or 4

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Anaemia Due to Blood Loss from the Telangiectases of Scleroderma

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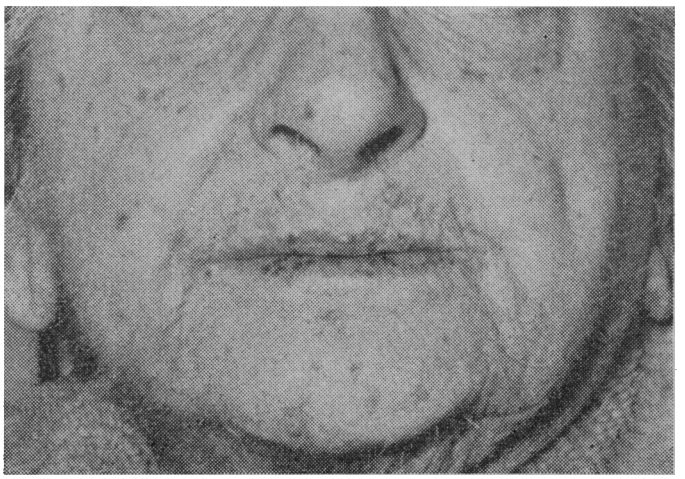
Telangiectases affecting the face, neck, oral mucous membranes, and extremities are not uncommon in scleroderma. In appearance they resemble closely those found in Osler–Weber–Rendu disease, though they are not hereditary (Verel, 1956; Winterbauer, 1964). However, it has been said that in contrast with 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 saccules in sigmoid colon.



Case 1. Telangiectases affecting face and lips.