Rosen *et al.* 1969, Reefe & Sierra 1970). Relapsing polychondritis appears to be no more specific a syndrome than any of the other collagen disorders.

It is a sufficiently rare condition to make diagnosis difficult. Indeed some cases have defied diagnosis even at the time of publication and have been labelled as unclassified variants of systemic sclerosis (Middleton 1962), or as idiopathic medial aortopathy (Marquis *et al.* 1968). This is unfortunate because aortic regurgitation or collapse of the cartilaginous upper airways may supervene and be rapidly fatal, while the administration of corticosteroids usually suppresses the disease and saves life.

REFERENCES
Alexander C S, Derr R F & Sako Y (1970) *Amer. J. Cardiol.* **25**, 81
Baum K G (1962) *Z. Laryng. Rhinol.* **41**, 766
Dolan D L, Lemmon G B & Teitelbaum S L (1966) *Amer. J. Med.* **41**, 285
Goldwater C (1963) *Acta rheum. scand.* **9**, 245
Hainer J W & Hamilton G W (1969) *New Engl. J. Med.* **280**, 1166
Marquis Y, Richardson J B, Ritchie A C & Wigle E D (1968) *Amer. J. Med.* **44**, 939
Middleton W S (1962) *Ann. intern. Med.* **57**, 183
Pearson C M, Kroening R J & Verity M A (1969) *Excerpta med. (Amst.) int. Congr. Ser.* No. 165, p 291
Reefe W E & Sierra R (1970) *Med. Ann. D. C.* **39**, 27
Rosen S W, Mackenzie M R, Cohen P J, Friedlander S, Medart W & Reardan J B (1969) *Gastroenterology* **56**, 323

Dr M H Lessof said that polychondritis might not be as rare as the literature suggested, for it was easily missed. At first the history of a 'blind boil in the nose' in this case had been accepted without comment; it was only when an inflammatory lesion of the left ear had been recognized as being due to chondritis that the diagnosis came to be made.

In reply to a question he said that the endocarditis had not been thought to be rickettsial, but rickettsial antibodies had been sought and not found.

Anorexia Nervosa in a Prepubertal Male

S H Roussounis MB DCH
(for T S Savage MRCP DCH)
(*Princess Alexandra Hospital, Harlow, Essex*)

Boy aged 11 years 10 months

History: Three months' refusal of food resulted in weight loss of 22 lb (10 kg), representing 25% of the premorbid weight. He obstinately refused medication, and successfully resisted parental compulsion. He frequently hid food in his clothes and disposed of it later. No vomiting.

Past history: Full-term normal delivery. Birthweight 8 lb (3·63 kg). No problems in infancy. School phobia since starting school at age 5. Onset of anorexia three months before he was due to start at a new 'adolescent' school.

Family history: Parents healthy; father postman. One other sibling aged 20.

On examination (Fig 1): Extremely emaciated. Weight 43 lb (19·5 kg), well below 3rd percentile. Height 55 in. (140 cm), just above 25th percentile. Cheerful. No secondary sexual characteristics.

Investigations: Full blood count normal. ESR 5 mm in 1 hour (Wintrobe). Electrolytes normal. Blood urea 60, fasting blood sugar 102 mg/100 ml. Serum folate 1·9 ng/ml (low result). Serum vitamin B_{12} 390 pg/ml. Plasma cortisol 34 µg/100 ml (fasting morning). X-rays: skull, chest, barium meal and follow-through normal. Stools normal. Tine test negative. Xylol normal.

Discussion

The diagnosis was based on food refusal and severe loss of weight. There was no evidence of schizophrenia, depression or organic disease. In addition he demonstrated a compliant, uncomplaining attitude which was quite striking in view of his extreme emaciation but is often seen in anorexia nervosa.

Anorexia nervosa can occur in males but is ten to twenty times commoner in females and is

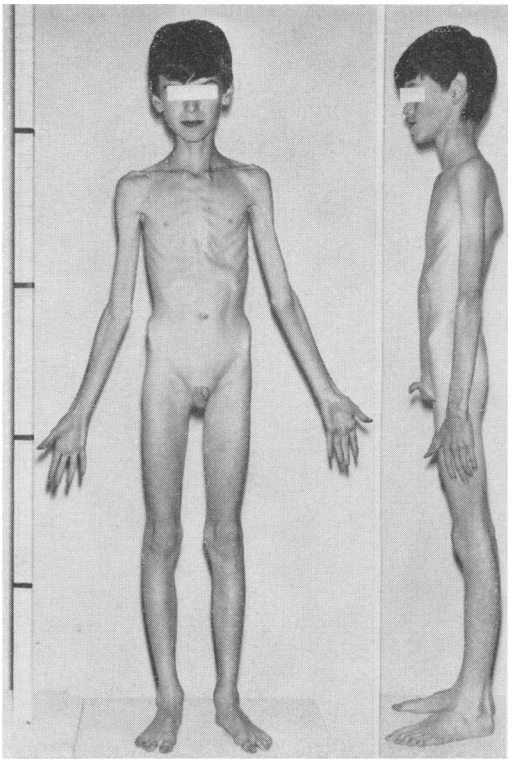

Fig 1 *Severe emaciation observed on admission*

usually confined to adolescents. In this case the essential psychopathology of anorexia nervosa operates, i.e. a refusal to grow up and accept normal adolescent weight; it is seen as an extension of the umbilical cord syndrome as manifested by long-standing school phobia and intensified by the prospect of going to the new school for children over 11.

He was treated with chlorpromazine (Largactil) and gradually increasing calorific intake as an inpatient, with someone watching him at meal times. His premorbid weight was regained in 90 days and has since been maintained. IQ found to be normal. The long-term prognosis is guarded. Long and close follow up may be necessary, psychotherapy having a definite role after normal weight is regained; with such treatment two-thirds of patients recover fully (A H Crisp, 1967, *Hosp. Med.* **1**, 713).

The following cases were also shown:

Carcinoma of the Male Breast
Dr C J Smart (for Mr D F Ellison Nash)

(1) **Dystonia Treated with L-Dopa (Two Cases)**
(2) **Syphilis and Motor Neurone Disease**
Dr E Small (for Dr F B Gibberd)

Meeting November 13 1970

Cases

Closed Trauma to the Popliteal Vessels
R Baker MB FRCS and D G A Eadie MS FRCS
(*The London Hospital, London E1*)

Mr D B, aged 19
Admitted following crush injury to right thigh caused by a fork-lift truck.
On examination: Shocked and pale, pulse 120/min, blood pressure 150/190. Entire right leg was swollen; right foot mottled, cold, pale and without sensation or movement. No pulses palpable below right femoral artery in groin. The skin was unbroken.

X-rays of the leg showed no fracture. A right femoral angiogram showed extravasation of contrast medium in region of adductor hiatus (Fig 1).
Treatment and progress: After resuscitation, the right thigh was explored under general anæsthesia (D G A E). There was a complete transection of the popliteal artery and vein, with a gap of 6 in. (15 cm) between the severed ends, a massive hæmatoma of the thigh and marked swelling of the thigh and leg. The defects in the popliteal artery and vein were bridged by parallel saphenous vein grafts. The incision was extended to decompress the calf and a fasciotomy of the anterior compartment was done. It was not possible to close the wound, but tension sutures were inserted to prevent further skin retraction (Fig 2). Massive blood transfusions totalling 18 pints (8·5 litres) were given during the treatment period.

After operation, the colour and temperature of the right foot began to improve almost immediately and pedal pulses returned. He became acidotic (blood pH 7·1) in the immediate postoperative phase and required intravenous sodium bicarbonate (2·7%) to correct this.

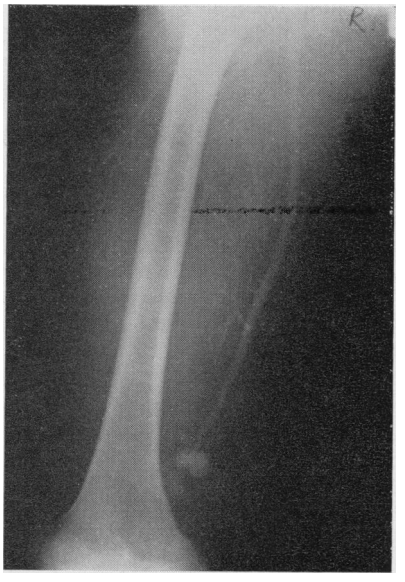

Fig 1 *Right femoral angiogram, showing extravasation of contrast medium in region of adductor hiatus*