and wondered whether the inhalation of the dust particles might have caused his illness. Regrettably, no samples of the old paint were available for laboratory analysis.

Physical examination on admission showed no abnormality. In particular, the patient had no pallor, no perianal or gum lead line (he was edentulous), his blood pressure was normal, there were no cardiovascular or objective neurological signs. A straight abdominal radiograph showed gas-filled, dilated loops of bowel with several fluid levels. A barium enema examination showed grossly distended and redundant colon, which accommodated 6 litres of barium with only partial filling (see figure). The haemoglobin concentration was 7.9 g/dl with small (mean cell volume 69.8 fl) hypochromic red cells showing pronounced basophilic stippling. Blood lead concentration (whole blood) on admission was 6.8 $\mu\text{mol/l}$ (140 $\mu\text{g}/100\text{ ml}$), and the erythrocyte protoporphyrin concentration was 1.94 ng/l (normal values: lead <1.5 $\mu\text{mol/l}$ (30 $\mu\text{g}/100\text{ ml}$); erythrocyte protoporphyrin <0.49 ng/l). The blood lead concentration was measured by anodic-stripping voltametry; the erythrocyte protoporphyrin concentration was measured fluorimetrically.² An electrocardiogram showed T-wave inversion in the lateral leads with high amplitude QRS complexes, suggesting left ventricular hypertrophy and strain. Manometric studies showed normal function of the myenteric plexus of the lower colon.³

X-ray film of barium enema, showing grossly distended and redundant colon.

Treatment was a modification of the method outlined by Chisholm,⁴ using a five-day course of a combination of dimercaprol 3 mg/kg four hourly intramuscularly and sodium calciumedetate 12.5 mg/kg four hourly intramuscularly. After five days of chelation treatment was continued with penicillamine 500 mg orally twice daily for six weeks, when the blood lead concentration had fallen to 3.1 $\mu\text{mol/l}$ (65 $\mu\text{g}/100\text{ ml}$), and the urinary lead excretion was 0.77 $\mu\text{mol/l}/24\text{ h}$ (160 $\mu\text{g}/24\text{ h}$). The patient has remained symptom-free, and a barium enema examination performed two months after treatment had been completed showed no abnormality.

Comment

The mechanism of lead colic is still unknown, though the condition was familiar to Hippocrates (370 BC), and lead poisoning in people handling paints was mentioned by Pliny (AD 27-79). In 1678 Vernatti (cited by Hunter⁵) graphically described lead colic: "The accidents to the workmen are, immediate pain in the stomach, with exceeding contortions in the guts, and costiveness that yields not to cathartics, hardly often to repeated clysters . . ."

The common symptoms and signs of individuals who become intoxicated with lead include colic; muscular weakness, which may lead to palsy—for example, wrist-drop; a lead line on the gums and about the anal margin, caused by bacterial action on the blood lead at these sites, precipitating out insoluble lead sulphide; pains in the muscles and joints; and anaemia. Severely affected individuals may develop encephalopathy and optic atrophy. Chisholm gives a complete review.⁴

Our patient had colic, muscular weakness, paraesthesia, joint pains, and anaemia. The profound degree of anaemia together with basophilic stippling and raised erythrocyte protoporphyrin values suggest that

he may well have experienced more chronic exposure than was apparent from the immediate history. The remarkable feature of this case, however, is the surprising degree of colonic dilatation when the patient first presented and the remarkable return to normal, both functionally and radiographically, once chelation treatment was completed. Acute dilatation of the colon may not be uncommon in patients with lead colic and may even play a part in the pathogenesis of the extreme constipation and pain.

We thank Dr Fred Hunter for performing the rectal manometric studies and Dr Allan Rosenberg for valuable advice on the chelation treatment.

- ¹ Schuster MM. Megacolon in adults. In: Sleisenger NH, Fordtran JS, eds. *Gastrointestinal disease*. 2nd ed. Philadelphia: WB Saunders, 1978:1814.
- ² Morell G, Geridhar G. Rapid micro method for blood lead analysis by anodic stripping voltametry. *Clin Chem* 1976;**22**:221-3.
- ³ Meunier P. Accuracy of manometric diagnosis of Hirschsprung's disease. *J Pediatr Surg* 1978;**13**:411-5.
- ⁴ Chisholm JJ. Treatment of lead poisoning. In: Center for Disease Control. *Preventing lead poisoning in young children*. Atlanta: US Department of Health, Education and Welfare, 1978:i:22-40.
- ⁵ Hunter D. *The diseases of occupations*. 6th ed. London: Hodder and Stoughton, 1978:248-305.

(Accepted 1 April 1981)

Department of Medicine, Louisiana State University, New Orleans, Louisiana 70112

JAMES H RICHARDSON, MD, interne in general medicine
PAUL D BUISSERET, MRCP, FACP, assistant professor of medicine

Myopathy induced by rifampicin

Myopathy is a well-recognised complication of treatment with certain drugs, particularly corticosteroids and anti-malarial agents,¹ but has not been reported with any of the conventional antituberculous drugs. We report a case of severe proximal myopathy caused by rifampicin.

Case report

A 44-year-old woman presented in 1974 with typical active pulmonary tuberculosis proved by sputum culture. She was treated for 16 months with isoniazid and rifampicin, supplemented for the first three months by daily injections of streptomycin.

In October 1980 symptoms recurred and chest radiography showed fresh cavitation. Direct smear of sputum was negative for acid-fast bacilli, but Löwenstein-Jensen cultures subsequently yielded colonies of mycobacteria. Treatment was started with streptomycin, rifampicin, isoniazid, and ethambutol. After four weeks she was readmitted, complaining of increasing proximal muscle weakness in the arms and legs and inability to stand unaided. There was no paraesthesia, anaesthesia, or muscle pain. Facial and ocular muscles were not affected, and she did not have dysphagia or abnormal fatigability to suggest myasthenia gravis.

Physical examination showed no abnormality apart from appreciable proximal muscle weakness, which was greater in the legs than the arms and was accompanied by symmetrical proximal muscle wasting. No rash, muscle tenderness, or fasciculation was present. Normal values were obtained for full blood count; urea and electrolyte, serum calcium, and plasmaprotein concentrations; creatine kinase activity; and thyroid function tests. Erythrocyte sedimentation rate was 48 mm in first hour. A Tensilon test showed no response in any muscle group.

All drugs were stopped and within 24 hours muscle power had increased dramatically. An electromyogram performed six days after the drugs were stopped showed no abnormality. Eight days after the drugs were stopped muscle power had returned to normal. Isoniazid was reintroduced at full dosage without any problems. Rifampicin was then added at full dosage and within 24 hours severe muscle weakness recurred. An electromyogram again showed no abnormality, but a muscle biopsy specimen showed predominant type II muscle fibre atrophy associated with sarcolemmal nuclei proliferation and increase in connective and fatty tissues, consistent with a drug effect. Rifampicin was withdrawn and within five days power returned to normal. Ethambutol was subsequently reintroduced without any further problem. Rifampicin antibodies were absent six weeks after the drug was stopped.

The mycobacterium cultured from the original sputum and a specimen one month after treatment was identified as *Mycobacterium xenopi*, highly resistant to all antituberculous drugs except rifampicin and streptomycin. Lobectomy was performed to eradicate the disease.

Comment

Several antibiotics, including streptomycin, interfere with neuromuscular transmission.² Neuromuscular blockade usually develops after introduction of the drug into the peritoneal or pleural cavity during operation or after parenteral administration and leads to respiratory difficulties in the immediate postoperative period.

Myopathy, however, has not previously been recorded with antituberculous drugs. This case shows that this complication may occur with rifampicin. Thrombocytopenia, acute haemolytic anaemia, the "flu" syndrome, and acute renal failure have all been associated with rifampicin. These side effects are usually seen with intermittent treatment³ or after early reintroduction of rifampicin after completion of a course.⁴ Renal failure may occur after a treatment gap of more than two years.⁵ Rifampicin antibodies are more commonly detected in patients who develop side effects, but titres fall at the end of treatment. The rapid onset of myopathy in our patient when rifampicin was reintroduced, compared with the slow development initially, together with a previous uneventful course of treatment suggest a similar mechanism.

The proximal myopathy in this patient was directly related to the rifampicin treatment, and this important adverse effect should

therefore be considered in any patient who complains of undue weakness during antituberculous chemotherapy.

- ¹ Walton JW. *Disorders of voluntary muscle*. Edinburgh: Churchill Livingstone, 1974:466-72.
- ² Argov Z, Mastaglia FL. Drug induced disorders of neuromuscular transmission. *Adverse Drug Reaction Bulletin* 1979;No 74.
- ³ Poole G, Stradling P, Worledge S. Potentially serious side effects of high dose twice weekly rifampicin. *Br Med J* 1971;iii:343-7.
- ⁴ Nessi R, Bonoldi GL, Dedaelli B, Difilippo G. Acute renal failure after rifampicin: a case report and survey of the literature. *Nephron* 1976;16:148-59.
- ⁵ Davison AG, Empey DW. Acute renal failure following reintroduction of rifampicin after a prolonged interval. *Br J Dis Chest* 1981;75:103-4.

(Accepted 23 April 1981)

Department of Thoracic Medicine, Westminster Hospital, London SW1P 2AP

PAUL JENKINS, MB, MRCP, senior registrar

PETER A EMERSON, MD, FRCP, consultant physician

Serum 25-hydroxy vitamin D in surface and underground coalminers

During studies on the effects of photochemotherapy on serum 25-hydroxy vitamin D (25-OHD) in psoriasis^{1,2} we wondered whether, as some of the patients were coalminers, the variation that we found before treatment might in part be associated with the length of time spent underground. Because the nature of their work makes coal-

underground coalworkers and those in other occupations in the Newcastle region suggests that the degree of solar irradiation obtained by underground workers between shifts was adequate to maintain vitamin D synthesis in the skin. The similar concentrations in those who had just started and those who had been working a

Serum 25-OHD concentrations in coal miners and other workers (nmol/l)

	Underground workers							All underground workers	Surface workers	Miners not at work	Non-miners
	Time of shift				Duration of shift						
	0600-1400	0900-1700	1400-2200	2200-0600	1 day	3 days	> 7 days				
Mean	62.8	77.3	65.5	99.5	76.8	64.5	79.8	73.8	82.3	83.5	67.5
SE	11.5	8.8	15.3	43.0	14.0	10.3	10.3	7.3	15.5	27.5	8.8
n	20	60	17	4	22	30	49	101	19	6	32

Conversion: SI to traditional units—25-OHD: 1 nmol/l $\approx$ 0.4 ng/ml.

miners prone to bony injury reduced serum 25-OHD concentrations might be important; we therefore measured serum 25-OHD concentrations in surface and underground workers.

Subjects, methods, and results

A total of 107 coalminers aged between 36 and 61 years from eight collieries in the Newcastle upon Tyne region participated in the study. Four worked a night shift (2200-0600 h) and 103 one of three day shifts (0600-1400, 0900-1700, 1400-2200 h). Duration of work on the shift was noted for each individual. Six participants had not worked for some time because of minor ailments. All blood samples were taken between 0900 and 1100 during May to July 1980. Within the same period blood samples were taken from a control population who were not coalminers, comprising 36 men and women who had been treated for minor skin disease and lived in the Newcastle region. Serum 25-OHD concentration was measured by competitive protein binding.³

The table shows that there were no appreciable differences in serum 25-OHD concentrations between surface and underground workers even when compared with either the control population or the small group of coalminers on sick leave. There were also no substantial differences in serum 25-OHD concentrations between the coalminers working the early, middle, and late day shifts, but there were insufficient numbers to compare the night-shift with the day-shift workers. Likewise there was no difference in serum 25-OHD concentrations between those who had just started day shifts and those who had worked day shifts for longer periods.

shift for some time, as well as in underground workers and the small group of coalminers on leave for various minor ailments, is in keeping with this view. As the study was done in the summer, the period of maximal sunlight, we cannot exclude the possibility that exposure to ultraviolet light might prove inadequate in miners working underground in the short daylight hours of the winter.

We thank the colliery nursing staff for their invaluable help.

- ¹ Rogers S, Marks J, Hillyard CJ, Shuster S. The effect of PUVA therapy on serum 25-OH vitamin D in psoriatics. *Br Med J* 1979;iii:833-4.
- ² Shuster S, Chadwick L, Moss C, Marks J. Serum 25-OH vitamin D after photochemotherapy. *Br J Dermatol* (in press).
- ³ Greenberg PB, Hillyard CJ, Galante LS, Colston KW, Evans IMA, McIntyre I. Affinity of calciferol analogues for 25-hydroxy cholecalciferol receptors. *Clin Sci Mol Med* 1974;46:143-7.

(Accepted 30 March 1981)

University Department of Dermatology, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP

SAM SHUSTER, PHD, FRCP, professor

LAWRENCE CHADWICK, chief technician

National Coal Board (North East), Whitburn, Sunderland

A S AFACAN, MD, MSC, area medical officer

Institute of Occupational Medicine, Edinburgh

MAURA D ROBERTSON, FIMLS, research technician

Comment

The similarity of serum 25-OHD concentrations in surface and