Case 2—A 56-year-old man suffered severe unstable angina three months after an inferior myocardial infarction. Treatment with propranolol 160 mg/day and isocardide dinitrate 60 mg/day was ineffective, and nifedipine 40 mg/day was added to the regimen. Three days later severe muscle cramps occurred in both legs and hands. The pain occurred only at night, the patient being asymptomatic during the day. The drug was discontinued and the cramps resolved. After a week the drug was readministered and the pain reappeared within 24 hours. The drug was again discontinued and the patient referred for surgery.

Case 3—A 73-year-old man with severe ischaemic heart disease and old myocardial infarction was referred to the outpatient clinic with severe stable angina. Treatment with isocardide dinitrate 80 mg/day and propranolol 240 mg/day was ineffective and nifedipine 30 mg/day was begun. He felt better for two weeks but then began to complain of severe muscle cramps in both legs during the day and night, which necessitated discontinuation of the drug, with dramatic relief from pain. He died a month later after a myocardial infarction.

Comment

Serious underlying side effects due to nifedipine rarely occur.¹ They are more common at the start of treatment, usually transient, and in most cases mild. The most common side effects are headache, facial flush, a sensation of heat, dizziness, nausea, and fatigue. In a therapeutic evaluation of 5000 patients none complained of muscle spasm.¹ In two other patients treated with nifedipine muscle cramps resolved when the drug was stopped.²

In the three patients reported here the drug was stopped; re-challenge in two cases provoked the symptoms.

¹ Hashimoko K, Kimvra E, Kotayashi T, eds. *First international Adalat symposium: new therapy of ischaemic heart disease*. Tokyo: University of Tokyo Press, 1975.

² Antman E, Muller J, Goldberg S. Nifedipine therapy for coronary spasm. *N Engl J Med* 1980;302:1269-73.

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Postexertional hypotension: a valuable physical sign

Measurement of blood pressure on standing up from the supine position to detect postural hypotension is a well-recognised manoeuvre to assess the adequacy of the circulating blood volume and cardiovascular reflexes. Even though exercise in the upright position is a more demanding test of the integrity of the cardiovascular homeostatic mechanisms, measurement of blood pressure during or immediately after exercise is not generally advocated in clinical practice. I believe that detection of postexertional hypotension may have important diagnostic and therapeutic implications.

Case reports

Blood pressure and pulse rate were measured after patients had rested supine for three minutes, after they had been standing erect for one minute, and when they were standing erect immediately after exercise. A hand-held anaeroid sphygmomanometer calibrated regularly against a standard mercury instrument was used with a Velcro fixed cuff for speed of application. Depending on the patient and the facilities available the exercise consisted of two-step exercise, walking up and down a flight of stairs, or running on the spot. The exercise was judged by the patient's response and stopped at the onset of tiredness or breathlessness. Care was taken to ensure that the patient's arm was hanging relaxed or well supported at heart level to avoid an isometric effect.

Case 1—A woman treated with psychotropic drugs and a diuretic for a personality disorder and hypertension was found to be severely hyponatraemic. Although she was not apparently dehydrated, her blood pressure was 120/80 mm Hg supine, 110/64 mm Hg erect, and 64/40 mm Hg after exercise. This indicated salt and water depletion and she was treated appropriately, having been subjected to water restriction for presumed inappropriate secretion of antidiuretic hormone.

Case 2—A 69-year-old man with dizziness on playing tennis was found to have a blood pressure of 158/80 mm Hg supine, 136/84 mm Hg erect, and 100/66 mm Hg after exercise. Idiopathic "postural" hypotension was eventually diagnosed; his symptoms and hypotension were certainly not postural.

Other cases—Several patients with congestive cardiac failure due to hypertensive heart disease treated with methyldopa and diuretics complained of weakness and breathlessness on exertion associated with postexertional hypotension. These symptoms improved and the hypotension disappeared when the dose of the drugs was reduced.

Comment

On dynamic exercise in healthy people systolic blood pressure reaches 200 mm Hg with a smaller rise or no rise in diastolic pressure.¹ When exercise stops the blood pressure dips transiently but then rises again and slowly falls to normal. Any fall in systolic blood pressure below the resting value half a minute after exercise is probably abnormal.²

The sympathetic nervous system mediates reflexes that maintain blood pressure in the presence of skeletal muscle vasodilatation and are called into play at the onset of exercise.³ The importance of these early reflex responses is highlighted by the pronounced fall in blood pressure regularly observed after only trivial exercise in patients taking adrenergic-neurone-blocking drugs. Impairment of cardiovascular homeostatic mechanisms thus seems to be demonstrable with short spells of uncontrolled mild exercise as described above. Assessment is feasible in all but the most disabled patients even in domiciliary practice.

The sympathetic nervous system plays an especially important part in hypovolaemic states,⁴ and in these circumstances exercise testing has been of particular value. Heart responses have not proved helpful in assessing the relative contribution of neural and blood-volume abnormalities to postexertional falls in blood pressure.

The symptoms associated with postexertional hypotension are often not the classical ones of presyncope or frank syncope⁵ but may, as noted above, consist of tiredness, heavy legs, and breathlessness. Careful measurement of the erect systolic blood pressure before and after dynamic exercise may help in assessing patients with suspected hypovolaemia, neurodepressant drug effects, or otherwise unexplained symptoms on exertion.

¹ Holmgren A. Circulatory changes during muscular work in man. *Scand J Clin Lab Invest* 1956;8,suppl 24:1-97.

² Korner PI. The normal human blood pressure during and after exercise, with some related observations on changes in the heart rate and the blood flow in the limbs. *Aust J Exp Biol Med Sci* 1952;30:375-84.

³ Buskirk ER. Cardiovascular adaptation to physical effort in healthy men. In: Naughton JP, Hellerstein HK, eds. *Exercise testing and exercise training in coronary heart disease*. New York: Academic Press, 1973: 23-31.

⁴ Anderson RJ, Linas SL. Sodium depletion states. In: Brenner BM, Stein JH, eds. *Sodium and water homeostasis*. New York: Churchill Livingstone, 1978:154-77.

⁵ Judson WE. Hypotension; physiologic mechanisms and treatment. *Med Clin North Am* 1953;37:1313-39.

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Nutritional rickets in Rastafarian children

Nutritional rickets in Asian children living in the United Kingdom has been the subject of much discussion during the past 20 years and increasingly in the past decade. We diagnosed nutritional rickets in four black Rastafarian children and believe we have identified another population at risk.

Case reports

Case 1—An 11-month-old girl presented with a painful arm. X-ray examination of the wrist showed classic changes of rickets. Serum calcium

concentration was 2.35 mmol/l (9.4 mg/100 ml), phosphate concentration 0.82 mmol/l (2.5 mg/100 ml), and alkaline phosphatase activity 115 KA units. Liver and renal function were normal and there was mild generalised aminoaciduria.

Case 2—A 13-month-old girl underwent a routine x-ray examination of the hips as part of follow-up for suspected congenital dislocation of the hip detected in the neonatal period. Typical changes of rickets were seen and were also present at the wrists and knees. Serum calcium concentration was 2.24 mmol/l (9.0 mg/100 ml) phosphate concentration 0.73 mmol/l (2.3 mg/100 ml), and alkaline phosphatase activity 148 KA units.

Case 3—A 13-month-old girl was screened for rickets after the disease had been diagnosed in the first two children. Florid radiological changes were present at the wrists, knees, and ribs. Serum calcium concentration was 2.24 mmol/l (9.0 mg/100 ml), phosphate concentration 0.66 mmol/l (2.0 mg/100 ml), and alkaline phosphatase >40 KA units.

Case 4—The 20-month-old stepsister of case 1 was brought for screening by her step-mother. Radiological changes were present at knees and wrists. Serum calcium was 2.14 mmol/l (8.6 mg/100 ml), phosphate concentration 1.07 mmol/l (3.3 mg/100 ml), and alkaline phosphatase 145 KA units.

Comment

All four children were born in the United Kingdom of black Rastafarian parents of West Indian origin. They were breast fed until the second half of the first year of life, when they were weaned onto an essentially vegetarian diet known as I-tal. None of the children received vitamin supplements during infancy and none completed a full course of immunisations.

The Rastafarians do not constitute a homogeneous group and details of the diet vary. They eat very little, if any, meat, milk, fats, or oils, most of the food being vegetable in origin. For Rastafarians in Jamaica fish is an important part of the diet, providing they have scales and are less than 12 in (30 cm) long.¹ In our patients, however, fish seemed to be forbidden. Full dietary assessments were performed in cases 1 and 3. The mean daily intakes of vitamin D were estimated to be 2.2 µg in case 1 and 2.78 µg in case 3 compared with a daily recommended intake of 10 µg.² Daily calcium intakes were 660 mg in case 1 and 696 mg in case 3, recommended daily intake 600 mg.² The diets were also low in iron; indeed, all our patients had low serum iron concentrations, though their haemoglobin concentrations were within normal ranges.

Risk factors for rickets in our patients include prolonged breast feeding without vitamin supplementation and a vegetarian diet low in vitamin D. The families live in a depressed inner city area where opportunities for outdoor play are few and exposure to sunlight is likely to be limited.

This series is similar to that reported by Edidin *et al*,³ many of whom were black and had had prolonged breast feeding, no vitamin supplementation, and few, if any, immunisations. None of their patients were described as Rastafarian.

Infantile rickets has also been reported in Britain in children of West Indian, Cypriot, and other immigrant groups who did not receive vitamin D supplements or welfare foods in London in the early 1960s.⁴ Children of Rastafarian families living in the United Kingdom appear to be at risk of nutritional disorders, particularly rickets, and further studies are indicated to assess the magnitude of the problem.

We thank Dr J H Baumer for detecting case 2 and Drs M G Mott and P M Dunn for permission to describe their patients.

¹ Barrett LE. *The Rastafarians*. London: Heinemann Educational Books, 1977.

² World Health Organisation. *Handbook on human nutritional requirements*. WHO Monograph Series, No 61. Geneva: WHO, 1974.

³ Edidin VD, Levitsky LL, Schey W, Dumbovic N, Campos A. Resurgence of nutritional rickets associated with breast feeding and special dietary practices. *Pediatrics* 1980;65:232-5.

⁴ Benson PF, Stroud CE, Mitchell NJ, Nicolaides A. Rickets in immigrant children in London. *Br Med J* 1963;i:1054-6.

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Cholestatic jaundice in fascioliasis treated with niclofolan

Fascioliasis, which occurs widely throughout the world, can produce the symptoms of urticaria, jaundice, tender hepatomegaly, and eosinophilia and abnormal results of liver function tests. The usual treatment is with emetine hydrochloride, bithionol, and now praziquantel.¹ Niclofolan has been extensively used in the treatment of fascioliasis in sheep and cattle and recently in man.² We describe a patient with fascioliasis treated with niclofolan who developed cholestasis that was probably related to the drug.

Case report

A 37-year-old Yemen woman was investigated in Sanaa, Yemen, for occasional loose stools and dull epigastric discomfort. Ova of *Fasciola hepatica* were found in the stool, but no treatment was given and the diarrhoea subsided. One year later she was found to have a few eggs of *F hepatica* still present in the stool.

White blood count showed an eosinophilia of 10%. Her body weight was 58 kg, and she was treated with niclofolan 0.5 mg/kg twice daily for three days. She had considerable vomiting and abdominal pain during treatment but these stopped when the treatment was stopped. A week later she complained of generalised itching, and four days later she was jaundiced and passing dark urine. She did not feel tired or anorexic. She was not taking any other drug or a contraceptive pill. On examination she was jaundiced with an erythematous rash and scratch marks on her arms and legs. The liver was not enlarged or tender. Examination was otherwise normal. Investigations showed a mild rise in aspartate transaminase and alkaline phosphatase activities with a considerable increase in bilirubin concentration. Severe eosinophilia was noted (table).

Clinical findings*

	Normal range	18 June	28 June	6 July	23 July	29 July
Aspartate transaminase (IU/l)	5-40	37	63	88	25	23
Alkaline phosphatase (IU/l)	30-110	113	154	165	81	72
Total bilirubin (µmol/l)	5-17	184	152	127	28	14
Albumin (g/l)		41	40	39	39	41
White cell count (× 10 ⁹ /l)		7.4			5.1	6.5
% Eosinophils		28			13	11

*Testing for hepatitis B surface antigen yielded negative results.

Conversion: SI to traditional units—Bilirubin: 1 µmol/l ≈ 58.5 µg/100 ml.

Three concentrated stool specimens were analysed, but ova of *F hepatica* were not found. Ultrasonography of the biliary tract and gall bladder showed no abnormality. Liver biopsy showed a normal architecture with pronounced centrilobular canalicular cholestasis; there was mild inflammatory infiltrate in the portal spaces, in which eosinophils were prominent. No eggs were seen. Percutaneous transhepatic cholangiography showed normal intrahepatic and extrahepatic bile ducts.

After three weeks of persistent itching and jaundice the symptoms subsided gradually and results of liver function tests became normal; mild eosinophilia persisted.

Comment

This patient suffered from *F hepatica* infestation presenting as diarrhoea and confirmed by the finding of *F hepatica* in the stools. She never suffered from jaundice or pruritus and had been asymptomatic for one year. One week after starting niclofolan she developed cholestasis, which lasted for five weeks. Percutaneous cholangiography showed patent intrahepatic and extrahepatic bile ducts. The histological picture showed normal liver architecture with centrilobular canalicular cholestasis, mild inflammatory infiltrate, and eosinophils compatible with drug-related liver injury.

Niclofolan has been used to treat fascioliasis in sheep and cattle and paragonimiasis, clonorchiasis, and fascioliasis in man.²⁻⁵ Side effects include transitory muscle and abdominal pain, nausea, sweating, and transient mild rash. Slight increases in serum aspartate transaminase and alkaline phosphatase activities have been noted,⁵ but jaundice has not been described. Severe infections with *F hepatica* and other trematodes such as opisthorchiasis and clonorchiasis cause cholangitis and subsequent cholestasis. Niclofolan can also cause cholestasis, and this may lead to diagnostic confusion. Establishing the temporal relation of the jaundice to administration of the drug