example, reflux oesophagitis with or without hiatus hernia, Meckel's diverticulum, or enteritis due to sensitivity to cows' milk. Recurrent iron deficiency must not be ascribed to poor diet alone without excluding other causes even in underprivileged children. Assessment of iron status may require measuring iron stores (serum ferritin) and also iron transport by means such as serum concentrations of iron and iron binding proteins (total iron binding capacity or transferrin) and their degree of saturation. The serum iron and transferrin saturation increase gradually throughout childhood, while serum transferrin is higher and ferritin lower in normal children than in adults, the change taking place in late childhood.[20]

What needs to be done? Firstly, the size of the problem should be assessed by community based surveys of children from various racial, cultural, and social backgrounds. Little such information seems to be available about children in Britain in recent years. A nutritional survey of children of Bangladeshi parents in London showed dietary deficiencies of vitamin D, energy, and iron.[21] Although the amount of iron present in human milk is small compared with that in fortified baby milks, its bioavailability is high, and breast feeding does not cause iron deficiency in the first six months of life.[22] The early introduction of fresh cows' milk into the infant diet may lead to iron deficiency, possibly because of loss of red cells from the intestines.[23] Whether a nationwide campaign of extra iron supplementation similar to the recent British "Stop Rickets Campaign" is desirable or feasible is a matter for debate, but in my opinion the information which would justify such action in Britain is not yet available, although an American programme of dietary aid to needy families does seem to have reduced the prevalence of iron deficiency.[24]

An alternative approach—more in keeping with present knowledge—would be to test infants aged 9 to 12 months especially in socially underprivileged areas and to give a course of iron treatment to those found to be anaemic. Giving iron in this way causes few problems[25] and is less expensive and more efficient than investigating all anaemic infants.[19] Doctors in hospital might also give more attention to detecting and treating mild iron deficiency in children, and all health professionals working with young children should give appropriate dietary advice at the time of weaning. Mild iron deficiency in children can no longer be ignored. In some children, it seems, happiness may indeed be iron.

D P ADDY

Consultant Paediatrician,
Dudley Road Hospital,
Birmingham B18 7QH

1 Oski FA. Iron deficiency—facts and fallacies. *Pediatr Clin North Am* 1985;**32**:493-7.
2 Ehrhardt P. Iron deficiency in young Bradford children from different ethnic groups. *Br Med J* 1986;**292**:90-3.
3 Jacobs A. The non-haematological effects of iron deficiency. *Clinical Science and Molecular Medicine* 1977;**53**:105-9.
4 Oski FA. The non-hematologic manifestations of iron deficiency. *Am J Dis Child* 1979;**133**: 315-22.
5 Finch CA, Miller LR, Inamdar AR, Person R, Seiler K, Mackler B. Iron deficiency in the rat: physiological and biochemical studies of muscle dysfunction. *J Clin Invest* 1976;**58**:447-53.
6 Mackler B, Person R, Ochs H, Finch CA. Iron deficiency in the rat: effects on neutrophil activation and metabolism. *Pediatr Res* 1984;**18**:549-51.
7 Srikantia SG, Prasad JS, Bhaskram C, Krishnamachari KAVR. Anaemia and immune response. *Lancet* 1976;i:1307-9.
8 Reeves JD, Yip R, Kiley VA, Dallman PR. Iron deficiency in infants: the influence of mild antecedent infection. *J Pediatr* 1984;**105**:874-9.
9 Mackler B, Person R, Miller LR, Inamdar AR, Finch CA. Iron deficiency in the rat: biochemical studies of brain metabolism. *Pediatr Res* 1978;**12**:217-20.
10 Massaro TF, Widmayer P. The effect of iron deficiency on cognitive performance in the rat. *Am J Clin Nutr* 1981;**34**:864-70.
11 Coltman CA. Pagophagia and iron lack. *JAMA* 1969;**207**:513-6.
12 Oski FA, Honig AS. The effects of therapy on the developmental scores of iron deficient infants. *J Pediatr* 1978;**92**:21-5.
13 Oski FA, Honig AS, Helu BM, Howanitz P. Effect of iron therapy on behavior and performance of non-anemic iron deficient infants. *Pediatrics* 1983;**71**:877-80.
14 Walter T, Kovalskys J, Steel A. Effect of mild iron deficiency on infant mental development scores. *J Pediatr* 1983;**102**:519-22.
15 Pollitt E, Soemantri AG, Yunis F, Scrimshaw NS. Cognitive effects of iron deficiency anaemia. *Lancet* 1985;i:158.
16 Lozoff B, Brittenham GM, Viteri FE, Wolf AW, Urratia JJ. The effects of short term oral iron therapy on developmental deficits in iron deficient anemic infants. *J Pediatr* 1982;**100**:351-7.
17 Dallmann PR, Siimes MA. Percentile curves for hemoglobin and red cell volume in infancy and childhood. *J Pediatr* 1979;**94**:26-31.
18 England JM, Fraser PM. Differentiation of iron deficiency from thalassaemia trait by routine blood count. *Lancet* 1973;i:449-52.
19 Dallman PR, Reeves JD, Driggers DA, Lo EYT. Diagnosis of iron deficiency: the limitations of laboratory tests in predicting response to iron treatment in 1 year old infants. *J Pediatr* 1981;**99**:376-81.
20 Milman N, Cohn J. Serum iron, serum transferrin and transferrin saturation in healthy children without iron deficiency. *Eur J Pediatr* 1984;**143**:96-8.
21 Harris RJ, Armstrong D, Ali R, Loynes A. Nutritional survey of Bangladeshi children aged under 5 years in the London Borough of Tower Hamlets. *Arch Dis Child* 1983;**58**:428-32.
22 Saarinen UM, Siimes MD, Dallman PR. Iron absorption in infants: high bioavailability of breast milk as indicated by extrinsic tag method of iron absorption and by the concentration of serum ferritin. *J Pediatr* 1977;**91**:36-9.
23 Sadowitz PD, Oski FA. Iron status and infant feeding practice in an urban ambulatory center. *Pediatrics* 1983;**72**:33-6.
24 Miller V, Swaney S, Deinard A. Impact of the WIC program on the iron status of infants. *Pediatrics* 1985;**75**:100-5.
25 Reeves JD, Yip R. Lack of adverse side-effects of oral ferrous sulphate therapy in 1-year-old infants. *Pediatrics* 1985;**75**:352-5.

Claudication

Population studies in countries as diverse as Finland and the United States have elicited symptoms of claudication in around 2·2% of men and 1·8% of women.[1,2] In the United States the Framingham study showed that the incidence increased sharply with age up to 75,[3] and a study in Basle reported a prevalence of 10% over the age of 65.[4] Claudication is the most frequent manifestation of arterial disease for which patients attend a vascular clinic, yet paradoxically it is the condition for which they receive the least practical help. Whether active intervention should be considered depends on the degree of disability, the social circumstances, and the level of arterial occlusion.

The most frequent site of occlusion is the femoral artery beyond the origin of the profunda femoris. For as long as the profunda, together with its inflow and outflow vessels, remains free of substantial obstruction the patient may merely have to moderate his or her walking pace or to pause for two or three minutes' rest at 300 metres. Limitations that are intolerable for a postman or a shepherd may scarcely be noticed by the average person in modern motorised society. Furthermore, patients with mild or moderate claudication due to occlusion of the femoral artery frequently have relatively stable symptoms for many years; and if they can modify their lifestyles and social habits they may experience spontaneous improvement as collaterals develop.

There are additional reasons why reconstructive surgery is seldom recommended for the patient with claudication. Many patients with claudication have a history of myocardial infarction and angina, which increases the risk of anaesthesia. A history of cerebral ischaemia is less common, but ausculta-

tion will often elicit a bruit over the carotid arteries. With or without operation such patients have a five year probability of death from myocardial infarction or stroke of around 20%.[2 3 5 6] Whether or not carotid endarterectomy should be recommended for a patient with an asymptomatic bruit is a separate issue, but there is a certain logic to the practice adopted in some centres in the United States of routinely carrying out "whole body" angiography and of reconstructing stenosed coronary and carotid arteries before dealing with the peripheral vessels.

The surgery itself is not entirely trouble free. Femoropopliteal bypass carries a mortality of 0·5-3·0%, and the five year graft occlusion rate is 15-50% depending on the availability of vein, surgical experience, and selection of patients.[5-7] At 10 years fewer than half of all vein grafts are likely to be patent. Indeed, if femoropopliteal reconstruction is being considered for claudication (and especially a graft to the popliteal artery above the knee) synthetic material may be the wiser choice so that the patient still has veins suitable for later use for coronary bypass or salvage of a limb.

A more active approach may be taken for the minority of patients with claudication who have their main lesions in the aorta or the iliac arteries. This is the level at which dilatation of stenoses by a percutaneous balloon catheter has its best chance of success.[8 9] (This method should not be described as "angioplasty," since it achieves its effect by bursting the inner layers of the vessel.) Occlusions may be treated by endarterectomy or more commonly bypassed; the long term patency rates of these procedures are much better than those of smaller calibre bypasses. Indeed, in patients with aortoiliac occlusion the decision to recommend surgery is determined less by the degree of disability and more by the patient's fitness for major abdominal surgery.

Decisions may be difficult when there are stenoses or occlusions in both the aortoiliac and femorodistal segments. Symptoms are likely to be severe, so that surgery seems more necessary. But the patient with such widespread disease is likely to be older, to have a greater probability of being diabetic, and to be at greater operative risk, particularly from coronary artery disease.[10 11] The difficulty commonly lies in deciding which lesion is the most in need of correction. Reconstruction at one level may be doomed to failure if there is a haemodynamically significant lesion at the other. Simultaneous aortoiliac and femorodistal reconstruction is a big, time consuming operation. The pros and cons of "two tier" reconstruction are matters of lively debate among vascular surgeons, and fine judgment is required on behalf of each individual patient.

What can we do to help most patients—who either are not sufficiently disabled for surgery or are unfit for it? Vasodilator drugs offer no benefits in theory or in practice.[12 13] Drugs with more complex actions may offer marginal benefits. Naftidrofuryl, a vasodilator which also enhances tissue oxygen potential, has not been shown to have any haemodynamic effect[14] but has been reported to give symptomatic improvement in patients over 60.[15] Oxpentifylline—a drug which improves erythrocyte deformability, enhances fibrinolysis, and inhibits platelet adhesiveness—has been shown to increase treadmill walking distances.[16] Treatment with one of these agents may, therefore, merit a limited trial of, say, two to three months in selected patients with severe symptoms.

Neither surgery nor drug treatment should be contemplated in the patient with claudication until the important aetiological factors have been corrected—so far as possible. Diabetes, cigarette smoking, and hypertension have been shown in the Framingham study to be powerful risk factors, while raised serum cholesterol concentrations and increased packed cell volume were weak risk factors.[3] Smoking is the biggest single risk factor which influences amputation rates.[17]

The benefits of stopping smoking are strikingly obvious to those who run vascular clinics. Patients with claudication report considerable improvement, sometimes surprisingly quickly. Since smoking jeopardises graft patency[5 18] many surgeons offer patients an operation on condition that they give up smoking—and commonly they find that the ensuing improvement has made surgery unnecessary. Measurements of ankle pressures with Doppler ultrasound and treadmill exercise tolerance have been observed to improve in patients who give up smoking.[19]

Should we also advise our patients to take extra exercise? The answer appears to be emphatically yes. Studies on the effects of daily muscular exercise have shown that average walking distances are doubled in the first three months.[20-24] That the benefit may be sustained some time after a formal programme of supervised exercise is suggested by the work of Clifford *et al*, who documented an 80% improvement in walking distance in 18 out of 20 patients assessed six months after completion of their programme.[25] These symptomatic improvements occur without corresponding improvement in haemodynamic measurements such as calf muscle blood flow[22] or ankle pressures measured by Doppler ultrasound.[25]

Perhaps the most important thing that the patient with claudication needs from his doctor is reassurance. Many fear that they are heading for an amputation. For most—and particularly those who stop smoking—this is not true.

C VAUGHAN RUCKLEY

Consultant Surgeon,
Royal Infirmary,
Edinburgh EH3 9YW

1 Reunanen A, Takkunen H, Aromaa A. Prevalence of intermittent claudication and its effect on mortality. *Acta Med Scand* 1982;**211**:249-56.
2 Criqui MH, Fronek A, Barrett-Connor E, *et al*. The prevalence of peripheral arterial disease in a defined population. *Circulation* 1985;**71**:510-5.
3 Kannel WB, McGhee DL. Update on some epidemiological features of intermittent claudication. *J Am Geriatr Soc* 1984;**33**:13-8.
4 Widmer LK, Grensher A, Kanner WB. Occlusion of peripheral arteries. A study of 6400 working subjects. *Circulation* 1980;**30**:836-42.
5 King RB, Myers KA, Scott DF, *et al*. Femoro-popliteal vein grafts for intermittent claudication. *Br J Surg* 1980;**67**:489-92.
6 Sonnenfeld T. Reconstructive vascular surgery for intermittent claudication. *Acta Med Scand* 1982;**212**:145-9.
7 De Weese JA, Rob CG. Autogenous vein graft ten years later. *Surgery* 1977;**82**:775-84.
8 Van Andel GJ. Transluminal iliac angioplasty. Long term results. *Radiology* 1980;**137**:607-11.
9 Katzen BT. Percutaneous transluminal angioplasty for arterial disease of the lower extremities. *AJR* 1984;**142**:23-5.
10 Royster TS, Lynn R, Mulcare RJ. Combined aorto-iliac and femoropopliteal occlusive disease. *Surg Gynecol Obstet* 1976;**143**:949-52.
11 Samson RH, Scher LA, Veith FJ. Combined segment arterial disease. *Surgery* 1985;**97**:385-96.
12 Verstraete M. Current therapy for intermittent claudication. *Drugs* 1982;**24**:240-7.
13 Boobis LH, Bell PFR. Can drugs help patients with lower limb ischaemia? *Br J Surg* 1982;**69** (suppl):17-23.
14 Ruckley CV, Callam MJ, Ferrington CM, *et al*. Naftidrofuryl for intermittent claudication: a double blind controlled trial. *Br Med J* 1978;i:622-3.
15 Clyne CAC, Galland RB, Fox MJ, Gustave R, Jantet GH, Jamieson CW. A controlled trial of naftidrofuryl (Praxilene) in the treatment of intermittent claudication. *Br J Surg* 1980;**67**:347-8.
16 Reich T, Cutler BC, Lee BY, *et al*. Pentoxifylline in the treatment of intermittent claudication of the lower limbs. *Angiology* 1984;**35**:389-95.
17 Juergens JL, Barker NW, Hines EA. Arteriosclerosis obliterans: review of 520 cases with special reference to pathogenic and prognostic factors. *Circulation* 1960;**21**:188-95.
18 Myers KA, King RB, Scott N. The effect of smoking on the late patency of arterial reconstruction on the legs. *Br J Surg* 1978;**65**:267-71.
19 Quick CRG, Cotton LT. The measured effect of stopping smoking on intermittent claudication. *Br J Surg* 1982;**69** (suppl):24-6.
20 Larsen DA, Lassen NA. Effect of daily muscular exercise in patients with intermittent claudication. *Lancet* 1966;ii:1093-5.
21 Skinner JS, Strandness DE. Exercise and intermittent claudication I. Effect of repetition and intensity of exercise. *Circulation* 1967;**36**:15-21.
22 Dallhof AG, Bjorntorp P, Holm J, Schersten T. Metabolic activity of skeletal muscle in patients with peripheral arterial insufficiency. Effect of physical training. *Eur J Clin Invest* 1974;**4**:9-15.
23 Ekroth R, Dahllof AG, Gundevall B, *et al*. Physical training of patients with intermittent claudication: indications, methods and results. *Surgery* 1978;**84**:640-4.
24 Jonason T, Jonzon B, Ringquist I, Oman-Rydbug A. Effect of physical training in different categories of patients with intermittent claudication. *Acta Med Scand* 1979;**206**:253-8.
25 Clifford PC, Davies PW, Hayne JA, Baird RN. Intermittent claudication. Is a supervised exercise class worth while? *Br Med J* 1980;**280**:1503-5.