infant makes great effort to avoid the covering hand so that he can maintain vision on the object of interest. When the eye with poor vision is covered no such avoiding movements are made since the infant is in no way inconvenienced by the test.

Although this screening examination may be carried out by doctors it is much more readily and accurately performed by ophthalmists, whose training and abilities with young children are specifically designed to this end. Dr Valman's statement that if a squint is seen or suspected at any time after 6 months of age the child should be examined by an ophthalmologist is correct; but in the present state of undermanning in the ophthalmological services of the NHS there is an excellent case to be made out for such initial screening to be carried out by orthoptists, and only those infants who are suspected of a squint or poor vision in one eye by the orthoptist should be referred on to the ophthalmologist.

PETER FELS

Moorfields Eye Hospital,
London EC1V 2PD

Vitamin D₃ in osteoporosis

SIR,—Professor B E C Nordin and his colleagues have reported (16 February, p 451) that 1 α -hydroxycholecalciferol (1 α -OHD₃) is of no use when given alone for the treatment of spinal osteoporosis in postmenopausal women. We have studied the effects of 1-hydroxylated vitamin D₃ (1-OHD₃) preparations in a group of patients with osteoporosis.

Five men and twenty women with back pain and no biochemical evidence of osteomalacia took part in the study. Each was allocated randomly either to a group receiving from 0.5 to 1.0 mg of 1-OHD₃ daily for six months (seven patients received 1, 25(OH)₂D₃ and eight received 1 α -OHD₃) or to a group (16 patients) receiving 0.5 ml daily of a placebo. Eleven patients entering the 1-OHD₃ group and nine entering the placebo group had two or more vertebral compression fractures and the average ages ($\pm$ SD) of the patients in the groups were 69.9 ($\pm$ 9.8) years and 73.0 ($\pm$ 8.6) years respectively. An iliac crest biopsy was taken from every patient assigned to the 1-OHD₃ group; none of the biopsies showed evidence of osteomalacia.

All patients were followed up for at least six months. Results from the study showed that intestinal calcium absorption, as assessed by a double isotope technique, rose significantly (paired *t* test *p* < 0.005) in the 1-OHD₃ group, but so did urinary calcium excretion (paired *t* test *p* < 0.05). Neither changed significantly in the placebo group. The incidence of new vertebral compression fractures was the same (20%) in both the 1-OHD₃ and the placebo groups, and the values for bone mineral mass per unit length estimated by photon absorptiometry¹ of the right femur and right radius and ulna did not change significantly (paired *t* test) in either group over the six months. Back pain and mobility were assessed from the patients' spontaneous comments and by inquiries into analgesic consumption. Eleven of those receiving 1-OHD₃ and one of those receiving placebo considered that their back pain was improved; seven receiving 1-OHD₃ but none receiving placebo considered mobility

improved (see accompanying table). Comparing the responses in the two groups in terms of improved versus not improved for severity of back pain and mobility the Fisher exact test gives probabilities of 0.004 and 0.02.

The study provided no objective evidence of improved bone strength and its single-blind design makes the interpretation of symptomatic improvement uncertain. The findings do, however, raise the possibility that the 1-hydroxylated vitamin D₃ preparations produced improvement in the symptoms of some of the patients in the 1-OHD₃ group.

I WANDLESS
S JARVIS
J GRIMLEY EVANS
EDWIN G A AIRD
J STEVENS

Newcastle General Hospital and
University of Newcastle upon Tyne,
Newcastle upon Tyne NE4 6BE

¹ Aird JGA, Pierides AM. *Br J Radiol* 1977;50:350-6.

Cholesterol and mortality rates

SIR,—In New Zealand Maoris, Dr Robert Beaglehole and others (2 February, p 285) noted an inverse relation between serum cholesterol level and overall mortality, and mortality from cancer and "other" causes of death. Hence the authors warned against introducing measures to reduce cholesterol level to ameliorate cardiovascular disease, which in these people has an incidence as high as or even higher than that in New Zealand whites. Conceivably, such measures might worsen Maoris' total mortality rate, since "the risk ratios of total mortality at a serum cholesterol concentration of 4.14 mmol/l (160 mg/100 ml), as compared with that at a concentration of 6.73 mmol/l (260 mg/100 ml), when adjusted . . . were 1.7 for men and 1.4 for women." The authors commendably urged caution in applying their findings on Maoris to other populations.

In the Maori population the lower and upper serum cholesterol values chosen (4.14 versus 6.73 mmol/l) are very wide apart. The point we wish to make is that serum cholesterol and mortality associations, when significant, will vary from population to population, in part because the mean cholesterol level of myocardial infarction patients varies considerably—from 7.2 mmol/l (278 mg/100 ml) in Australia¹ to as low as 4.4 mmol/l (170 mg/100 ml) in India.² Furthermore, the mean level in myocardial infarction patients varies widely even within a given country, as demonstrated in Italy³ and Norway.⁴ Such variation certainly will add to difficulties in investigations but does not detract from the authors' emphasis on "the importance both of studying risk factors on a population-specific basis and of using total mortality as an end point for community-based intervention studies."

A further complicating factor concerns differences in reactivity displayed by different emerging populations having somewhat similar biological variables. Maoris have slightly lower cholesterol levels (about 1 mmol/l (38.6 mg/100 ml) less) than New Zealand whites, the same systolic pressures, but much greater frequencies of obesity in both men and women. They are, as mentioned, very

prone to cardiovascular disease. In Johannesburg, urban blacks at middle age and later have lower cholesterol levels (about 1.0-1.5 mmol/l (38.6-57.9 mg/100 ml) less) than whites but much higher diastolic and systolic pressures, and a greater frequency of obesity, principally in women.⁵ Yet, in remarkably striking contrast to Maoris' experience, the age-specific mortality rate of blacks from ischaemic heart disease is very low, only 5-10% or so of that of whites.⁶ Interestingly, this diversity of ischaemic heart disease experience of South African blacks and whites resembles that noted in immigrant Caribbeans and whites in East London.⁷

Dr Beaglehole and his colleagues, in stressing the use of total mortality as an end point for community-based intervention, have in a sense underlined the view of Rose and Barker⁸ that "the outcome of screening must be judged in terms of its effect on mortality or illness. . . ." This scrutiny of the worthwhileness of intervention has long been urged by Cochrane.⁹ When carried to its logical conclusion this scrutiny, if its results are implemented, has enormous implications regarding many well-known practices, some especially relevant to Third World populations. The authors are to be congratulated on their thought-stimulating contribution.

A R P WALKER

South African Institute for Medical Research,
Johannesburg 2001, South Africa

- ¹ Palmer AJ, Blacket RB, Leelarthapin B. *Med J Aust* 1973;2:19-23.
- ² Chaudhuri S, Das S, Das NC. *Ind Heart J* 1966;18:391-402.
- ³ Keys A, Fidanza F. *Circulation* 1960;22:1091-106.
- ⁴ Jervell A, Meyer K, Westlund K. *Acta Med Scand* 1965;177:13-23.
- ⁵ Seftel HC. *S Afr Med J* 1978;54:99-104.
- ⁶ Walker ARP. *S Afr Med J* (in press).
- ⁷ Pedoe HT, Clayton D, Morris JN, Bridgen W, McDonald L. *Lancet* 1975;ii:833-8.
- ⁸ Rose G, Barker DJF. *Br Med J* 1978;iii:1417-8.
- ⁹ Cochrane AL. *Effectiveness and efficiency. Random reflections on health services*. London: Nuffield Provincial Hospitals Trust, 1972.

Massive bleeding from the large bowel

SIR,—Your leading article (16 February, p 425) in discussing the differential diagnosis of this perplexing problem rightly stresses that known diverticular disease cannot be assumed as the source of haemorrhage.¹ Your review, neglects, however, to discuss the important role played by colonoscopy. This investigation may make a significant contribution to both diagnosis and management, as has been shown by a number of studies,^{2,3} especially when barium enema or the single most useful procedure, selective mesenteric angiography, is not available.⁴

You rightly draw attention to the greater awareness of vascular ectasias as a cause of bleeding, particularly in the elderly. Despite suggestions in the literature⁵⁻⁷ that colonoscopy is not of value in this condition, it seems logical that a mucosal defect will be present if significant blood loss has occurred and that this will be visible in the well-prepared bowel on careful scrutiny by the endoscopist. Such angiodysplastic lesions have been seen in nine patients in the last 500 colonoscopies in this hospital. The mean age of the patients was 70 and the mean haemoglobin concentration at presentation 6.9 g/dl. Two patients have been successfully operated on for multiple lesions and the other seven have had diathermy coagulation applied at colonoscopy⁸ without any recurrence. The diagnosis was confirmed histologically in one of the operative specimens and in six of the seven patients at diathermy coagulation biopsy.

Clinical response in patients with osteoporosis taking placebo and 1-OHD₃

	Severity of back pain			Mobility		
	Decreased	Unchanged	Increased	Decreased	Unchanged	Increased
No taking placebo	1	6	3	1	9	0
No taking 1-OHD ₃	11 (73%)	4	0	0	8	7 (47%)

The paper by Giacchino and his colleagues,⁷ on which your recommendations for management are so clearly based, was criticised in the subsequent discussion for its conclusions that colonoscopy should not be performed and it is surprising that you should perpetuate this erroneous conclusion.

There is clearly much evidence to support a co-ordinated approach to the problem of massive bleeding from the large bowel, involving the gastroenterologist, endoscopist, radiologist, and surgeon in a joint and flexible approach to investigation and management rather than adherence to the more rigid flow chart suggested.

RICHARD H HUNT

Department of Gastroenterology,
Royal Naval Hospital,
Gosport, Hants PO12 2AA

¹ Hunt RH. *Clin Gastroenterol* 1978;7:719-40.

² Deyhle P, Blum AL, Neusch HJ, Jenny S. *Endoscopy* 1974;6:229-32.

³ Rossini FP, Ferreri A. *Acta Endoscop* 1976;6:165-8.

⁴ Forde KA. In: *Proceedings of Second International Congress on Colonoscopy and Disease of the Large Bowel*. Miami, USA, 1980.

⁵ Boley SJ, Sammartano R, Brandt LJ, Sprayregen S. *Surg Gynecol Obstet* 1979;149:353-9.

⁶ Tarin D, Allison DJ, Modlin IM, Neale G. *Br Med J* 1978;iii:751-4.

⁷ Giacchino JL, Geis WP, Pickleman JR, Dado DV, Hadcock WE, Freeask RJ. *Surgery* 1979;86:368-76.

⁸ Rogers BH, Adler F. *Gastroenterology* 1976;71:1079-82.

Diet and ulcerative colitis

SIR,—We read with interest the communication of Dr J R Thornton and others (2 February, p 293) on diet and ulcerative colitis. Since we had observed that patients with Crohn's disease eat more refined carbohydrates and sugar than their matched controls (177 g/day versus 74 g/day),¹ we were interested to know whether patients with ulcerative colitis have the same dietary intake.

The nutritional habits of 114 patients with ulcerative colitis were investigated by means of questionnaire and compared with a control group of 114 persons matched by age, sex, and social status. In contrast to the patients with Crohn's disease patients with ulcerative colitis did not have an increased consumption of refined carbohydrates—94 g/day versus 78 g/day, which comes very close to Dr Thornton's results. However, they consumed significantly more cereal products and potatoes and significantly less milk products, coffee, tea, fruit juices, vegetables, fruit, and alcohol than the control group.²

G A MARTINI
A STENNER
W J BRANDES

Medizinische Universitätsklinik,
D-3550 Marburg,
Federal Republic of Germany

¹ Martini GA, Brandes JW. *Klin Wschr* 1976;54:367-71.

² Brandes JW, Stenner A, Martini GA. *Z Gastroenterol* 1979;17:834-42.

Recurrent abdominal pain in childhood

SIR,—I was interested in reading your leading article about "Recurrent abdominal pain in childhood" (26 April, p 1096).

While I agree with its contents, as a GP I was surprised that you had not mentioned mesenteric adenitis. In my experience this is extremely common in children, it is recurring, and often it is not accompanied by any other abnormal physical signs. There are two other conditions I thought you might have mentioned because they are so common:

constipation, and pain in girls nearing menstruation—sometimes a year before it.

These are very common conditions taking up a great deal of our time, but it is very reassuring to know that the huge majority of abdominal pains in children have no serious underlying pathology.

P C BRUNNER

Pinner, Middx HA5 2BS

Distinguishing direct and indirect inguinal hernias

SIR,—Mr D N L Ralphs and others (12 April, p 1039) have shown clearly how unreliable are the features of distinction between direct and indirect inguinal hernia listed in surgical textbooks, apart from the agreed fact that a scrotal hernia is indirect. The application of certain simple observations, however, can make an accurate distinction in 90% of cases and has the advantage that the patient is not subjected to the discomfort of digital exploration of the external ring.

Firstly, the common type of direct hernia due to muscular weakness is usually bilateral, although the patient may be aware of only one hernia. Secondly, simultaneous bilateral indirect hernias are uncommon, but the development of a hernia in a patient who has had a previous operation for hernia on the opposite side usually indicates that the hernia is indirect. Thirdly, an indirect hernia may on occasion be difficult to demonstrate without some exertion on the part of the patient and some manipulation is often necessary to reduce it. Lastly, a direct hernia cannot be masked and inevitably appears when the patient stands and rarely requires any manipulation to reduce it when he lies down.

Admittedly the unilateral direct funicular hernia and the "pantaloon" sac of the combined direct and indirect hernia are sources of error, but they comprise only a small percentage of inguinal hernias. Many direct hernias are symptomless and it is surprising how many middle-aged and elderly patients will opt for conservative treatment of direct hernia when assured of its safety by a confident surgeon.

W M BEATTIE

Liverpool L18 3EB

Prophylaxis of surgical wound sepsis: cardiac pacemakers

SIR,—Your recent leading article (19 April, p 1063) draws attention to points of importance and relevance in the implantation of permanent cardiac pacemakers, where infection may prove hazardous. Most pacemakers are now implanted by the transvenous or endocardial route,^{1,2} without the need for a thoracotomy. The incidence of infection is of the order of that quoted for other "clean" procedures and varies in reported series from zero to 12.6%.²⁻⁵ Not only does infection cause considerable suffering and expense but it is also occasionally the cause of death.

The organisms mentioned in your article are responsible for the bulk of pacemaker infections and in those cases infected with *Staphylococcus aureus* it is considered necessary to explant all foreign material to eradicate the infection.⁶ The patients are often dependent on their pacemaker for an effective ventricular rate and must necessarily be protected by

having a second pacemaker implanted before removal of the infected material. This may be a temporary pacemaker or a second permanent system provided there is a clean, safe site for its insertion.⁴ The potential risk of infective endocarditis means that both pulse generator and electrode should be removed.⁷ Sometimes the endocardial lead is firmly adherent to the right ventricle because of fibrosis and can be removed only by right atriotomy. This has proved necessary in persisting or severe infections.⁷

Most authorities employ prophylactic antibiotics for new primary implantations, although there are many different recommended regimens.⁸ Opinion is still divided over the use of surgical drains for the pulse generator pocket, and over the use of prophylactic antibiotics for pulse generator replacement operations.^{5,6,8,9}

N PAUL SILVERTON

University Department of
Cardiovascular Studies,
Leeds LS2 9JT

¹ Fishman NH, Hutchinson JC, Edmunds LH, et al. *Am J Surg* 1970;120:187-91.

² Kohn O. *Am Heart J* 1974;88:656-63.

³ Firor WB, Lopez JE, Nanson EM, Mori M. *Ann Thorac Surg* 1968;6:431-6.

⁴ Kenelly BM, Piller LW. *Br Heart J* 1974;36:1133-40.

⁵ Edhag O, Lagergren H. *Ann N Y Acad Sci* 1969;167:761.

⁶ Jora FM, Toledo-Pereyra L, Lewis JW, et al. *J Thorac Cardiovasc Surg* 1979;77:298-300.

⁷ Lemire GG, Morin JE, Dobell ARC. *Can J Surg* 1975;18:181-4.

⁸ Hartstein AL, Jackson J, Gilbert DN. *J Thorac Cardiovasc Surg* 1978;75:219-22.

⁹ Richardson PL, Pate JW. *JAMA* 1973;225:1388.

Unrecognised dehydration during parenteral nutrition?

SIR,—We agree with Dr C C Wise's (19 April, p 1086) correction of the osmolality of Glucoplex 1600 quoted in Drs R W G Prescott and J C Stoddart's letter (22 March, p 847) and have found the osmolality (by freezing point depression) of clinically useful amino-acid and glucose mixtures in a bag delivery system using Aminoplex 12 and the Glucoplex solutions (1000 and 1600) range from 1400 to 1900 mmol (mOsmol)/kg. His letter and that of Dr R G Wilkes (p 1087) indicate that the water derived from the breakdown of glucose to carbon dioxide and the water released in protein synthesis is such that "effective" water content of such intravenous feeding mixtures approaches 100% if the glucose is "burnt." Indeed, if the carbon chains of amino-acids infused in intravenous feeding preparations are catabolised to carbon dioxide there may even be a net gain of water.

While glucose is the energy source of choice in the severely ill patient it is important to give only those glucose calories which can be metabolised. The blanket figure of 0.8 MJ (200 kcal) per gram of nitrogen should be abandoned. Long *et al* showed an improvement in nitrogen balance with increasing carbohydrate intake until the calorie value is equivalent to the patient's metabolic rate, although further enhancement of positive nitrogen balance was achieved by adding exogenous insulin.¹ It should be remembered that the metabolic rate is low in the "ebb" phase of injury and hence such patients may require a calorie: nitrogen ratio of less than 100:1 whereas those in the "flow" phase may utilise in excess of 200:1. The appropriate rate of infusion of calories may be judged by "titrating" the reponse of nitrogen balance against calorie input while maintaining blood glucose