- 1 Evans KL. Diagnosis and management of sinusitis. *BMJ* 1994; 309:1415-22. (26 November.)
- 2 Havas TE, Motbey JA, Gullane PL. Prevalence of incidental abnormalities on computed tomographic scans of the paranasal sinuses. *Arch Otolaryngol Head Neck Surg* 1988;114:856-9.
- 3 Bolger WE, Butzin CA, Parsons DS. Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic surgery. *Laryngoscope* 1991;101:56-64.

Author's reply

EDITOR,—I believe that the points raised by Alexander C MacLennan and Gerald W McGarry were addressed in my review. In fact, we both reference the same paper for the same reason.¹ When an overview of a broad subject is presented to the medical profession as a whole it is possible to imply an inappropriate emphasis on one investigation. I did not, however, imply that computed tomography should be relied on for diagnosing chronic sinusitis; indeed, I stated that it should be requested only "after failure of maximal medical treatment, if a complication arises, or if malignancy is suspected."

I agree that rigid nasal endoscopy is important in diagnosing chronic sinusitis, but it cannot stand alone as a diagnostic investigation as it also has limitations as already stated—for example, the inability to identify concha bullosa or Haller's cells. Not all patients are able to tolerate the middle meatus being inspected with a rigid endoscope, and if they are the maxillary sinus ostia are rarely visualised in the depths of the ethmoid infundibulum, and any view of the frontal sinus ostium will depend on the superior insertion of the uncinate process.

KATHRYN EVANS
Consultant, ear, nose, and throat surgeon

Gloucester Royal Hospital,
Gloucester GL1 3NN

- 1 Havas TE, Motbey JA, Gullane PJ. Prevalence of incidental abnormalities on computed tomographic scans of the paranasal sinuses. *Arch Otolaryngol Head Neck Surg* 1988;114:856-9.

Vitamin A supplementation in preschool children with acute diarrhoea

EDITOR,—Nita Bhandari and colleagues report the results of a trial of vitamin A in the prevention of respiratory and diarrhoeal morbidity in a population of children likely to be clinically or subclinically deficient in vitamin A.¹ We do not believe that the conclusions are justified by the results described, and the public health implications contained in the box are misleading.

The first two points in the box are not contentious and are based on previous research. The third point, referring to the authors' study, states that in children aged 1-5 vitamin A supplementation reduces the prevalence of diarrhoea associated with fever by 36%. This finding applies only to those aged 23 months and over, thus excluding some children aged 1 year, and was based on subgroup analysis of the data. The P value for this observation was only 0.05, and given that 20 subgroup analyses were carried out, we do not believe that this can be considered to be significant. The fourth point in the box is that children given supplements suffered less measles. This finding was not significant for the whole study population, and the significant reduction in the younger age group was countered by an opposite (and not similar as stated in the text) trend in the older age group. The main findings of this study are not emphasised in the box—that is, there was no difference in the incidence or prevalence of diarrhoea or respiratory infections between the treatment and control groups.

In the final paragraph the authors recommend that vitamin A supplementation should be provided to children aged 6 months to 5 years in settings in

which vitamin A deficiency is a public health problem. Given that this conclusion could not be drawn from the results of this study and depends on the results of previous trials, an important ethical question arises. The study was carried out on a subgroup of children, many of whom were likely to have subclinical vitamin A deficiency; given the conclusion derived from literature that predated this study, it might be considered unethical to withhold vitamin A in these circumstances. Because vitamin A may be harmful it might be argued that a trial is justified, but a different trial design would have to be used to have sufficient power to detect adverse effects.

PAUL PHAROAH
Senior registrar
JULIUS WEINBERG
Senior registrar
ANDREW HAYWARD
Registrar

PHLS Communicable Disease Surveillance Centre,
London NW9 5EQ

- 1 Bhandari N, Bhan MK, Sazawal S. Impact of massive dose of vitamin A given to preschool children with acute diarrhoea on subsequent respiratory and diarrhoeal morbidity. *BMJ* 1994;309:1404-7. (26 November.)

Authors' reply

EDITOR,—Several of the issues raised by Paul Pharoah and colleagues are pertinent. The reduction in the prevalence of diarrhoea with fever and the incidence of measles was observed only in subgroup analysis and must be considered to be only suggestive. As there was a significant interaction of age on the effect of vitamin A and diarrhoeal morbidity, in this instance analysis within age subgroups was justified and, in a way, more meaningful than the overall analysis.

Several recent publications have reported the impact of vitamin A supplementation on morbidity. The findings ranged from no reduction in children given supplementation in Haiti¹ through a decrease in visits by a doctor because of diarrhoea in Ghana² to an overall reduction in the incidence of diarrhoea by 20% in Brazil.³ In the last study in Brazil the benefit of supplementation was greater for severe episodes of diarrhoea. When diarrhoea was defined as the passage of three or more liquid stools a day the overall reduction in the prevalence of diarrhoea was 8%; the reduction increased to 20% when a more stringent definition of six or more liquid stools a day was used. The variable results reported may be related to the differences in severity of vitamin A deficiency across studies and the different methods of analysis.

When these studies are viewed together vitamin A supplementation seems to reduce the incidence of measles and to affect the severity of diarrhoea favourably but to have no impact on pneumonia. This is consistent with findings on cause specific mortality after vitamin A supplementation in the few studies providing such data.⁴

NITA BHANDARI
Senior research officer
M K BHAN
Additional professor
SUNIL SAZAWAL
Senior research officer

Division of Gastroenterology and Enteric Infections,
Department of Paediatrics,
All India Institute of Medical Sciences,
New Delhi 110029,
India

- 1 Stansfield SK, Louis MP, Lerebours G, Augustin A. Vitamin A supplementation and increased prevalence of childhood diarrhoea and acute respiratory infections. *Lancet* 1993;342: 578-82.
- 2 Ghana VAST Study Team. Vitamin A supplementation in northern Ghana: effect on clinic attendances, hospital admissions, and child mortality. *Lancet* 1993;342:7-12.
- 3 Barreto ML, Santos LMP, Assis AMO, Araújo MPN, Farenzena GG, Santos PAB, et al. Effect of vitamin A supplementation on diarrhoea and acute lower-respiratory-tract infections in young children in Brazil. *Lancet* 1994;344:228-31.

- 4 Beaton GH, Martorell R, L'Abbé KA, Edmonston B, McCabe G, Ross AC, et al. Effectiveness of vitamin A supplementation in the control of young child morbidity and mortality in developing countries. 1992. Report submitted to CIDA Faculty of Medicine, University of Toronto. Toronto: University of Toronto, 1992.

Carrier testing in children for cystic fibrosis

EDITOR,—Your recent article debating screening for carriers of cystic fibrosis raises important issues.¹ One aspect, however, that has received relatively little attention is whether it is justified to test for carrier status in siblings of patients with cystic fibrosis during childhood. A recent report from the Clinical Genetics Society (UK) was not in favour of testing children for recessive disorders where this is of purely reproductive significance to that child in the future.²

However, we found that 90% (103/114) of parents of patients with cystic fibrosis wanted to know the carrier status of their other children; furthermore, 91% (104/114) believed that they had a fundamental right to know.³ In contrast, we found that most clinical geneticists would try to dissuade the parents or postpone testing until the child was older; 41% (11/27) thought that the parents had no rights to all to know their children's carrier status. Out of 78 doctors working in child health for their views, we found that 76% (59/78) would want to know and 69% (54/78) thought that it was their right to know.

Clearly there is a divergence of opinion, and a consensus probably cannot be reached. As with many difficult ethical issues in clinical paediatrics, the problem of who should make these sorts of decisions remains. Should it be the experts (clinical geneticists), the clinicians who regularly see the families (paediatricians and general practitioners), or the families themselves once properly informed? While it may be prudent to wait until the siblings are old enough to express an opinion, problems may arise when parents of younger children are insistent as carrier testing is becoming more readily available. Commercial pressures will eventually make such testing available both in private laboratories and from local chemists, and the worry is that results will not be accompanied by adequate genetic counselling.

IAN BALFOUR-LYNN
Clinical research fellow
SU MADGE
Clinical nurse specialist
ROBERT DINWIDDIE
Consultant paediatrician

Respiratory Unit,
Great Ormond Street Hospital for Children NHS Trust,
London WC1N 3JH

- 1 Raeburn JA, Marteau T. Screening for carriers of cystic fibrosis. *BMJ* 1994;309:1428-30. (26 November.)
- 2 Clinical Genetics Society (UK). The genetic testing of children. Report of a working party of the Clinical Genetics Society (UK). *J Med Genet* 1994;31:785-97.
- 3 Balfour-Lynn IM, Madge S, Dinwiddie R. Testing carrier status in siblings of patients with cystic fibrosis. *Arch Dis Child* (in press).

Management of childhood nephrotic syndrome

EDITOR,—P D Mason and C D Pusey's review article on diagnosing and treating glomerulonephritis does not refer, in the paragraph on minimal change nephropathy in children, to the recently published consensus statement of the British Association for Paediatric Nephrology on the management of steroid responsive nephrotic syndrome.^{1,2} In particular, Mason and Pusey's recommendations on treatment differ from those of paediatric nephrologists.

It is inaccurate to state that the use of alkylating

agents in minimal change nephropathy is controversial. This implies that the benefits are unproved, whereas a definite, sustained effect has been shown in controlled trials.³ There are, however, side effects associated with these agents, and the authors are correct in stating that their use should be carefully considered. The consensus statement lists two clear indications for second line treatment. (1) Relapse while taking prednisolone >0.5 mg/kg on alternate days plus one or more of (a) unacceptable side effects of corticosteroid treatment; (b) high risk of steroid toxicity—for example, in boys approaching puberty or in diabetes; (c) unusually severe relapses (hypovolaemia or thrombosis); and (d) inadequate facilities for follow up or concern about compliance. (2) Relapse while taking prednisolone >1 mg/kg on alternate days. The recommended dose of cyclophosphamide is 3 mg/kg/day for eight weeks. The report describing fewer relapses in children treated with cyclophosphamide for 12 weeks was limited to patients with steroid dependent disease.⁴

GRAHAM SMITH
Senior registrar in paediatric nephrology
Royal Hospital for Sick Children,
Yorkhill,
Glasgow G3 8SJ

- 1 Mason PD, Pusey CD. Glomerulonephritis: diagnosis and treatment. *BMJ* 1994;309:1557-63. (10 December.)
- 2 Report of a workshop by the British Association of Paediatric Nephrologists and Research Unit, Royal College of Physicians. Consensus statement on management and audit potential for steroid responsive nephrotic syndrome. *Arch Dis Child* 1994;70:151-7.
- 3 International Study of Kidney Disease in Children. Prospective controlled trial of cyclophosphamide in children with nephrotic syndrome. *Lancet* 1974;ii:423-7.
- 4 Arbeitsgemeinschaft für Pädiatrische Nephrologie. Cyclophosphamide treatment of steroid-dependent nephrotic syndrome: comparison of eight weeks with 12 weeks course. *Arch Dis Child* 1987;62:1102-6.

Eradication of *Helicobacter pylori* in management of peptic ulceration

Evidence supports eradication only in patients who have helicobacter and peptic ulceration

EDITOR,—C O Record emphasises the prevalence of *Helicobacter pylori* infection in patients without peptic ulcer disease,¹ but this in no way diminishes the possibility that the bacterium is an important causal factor in ulcer disease. Secretion of acid is also common in patients without peptic ulceration, but we doubt if that has prevented Record from successfully using drugs that suppress such secretion. Neither acid nor helicobacter infection are the sole cause of peptic ulceration, but both are likely to be important causal factors in a multifactorial disorder. Most ulcers can be healed and kept healed by the suppression of either helicobacter or acid.

Record uses hypochlorhydria in the early stages of helicobacter infection as an argument against a role for the bacterium in the genesis of duodenal ulcer disease, which occurs in patients with normal or increased production of acid. In chronic helicobacter infection, however, evidence suggests that the bacterium promotes secretion of both gastrin and acid.²

We agree that there is no ideal regimen for eradicating helicobacter, but Record's assumption that four weeks' treatment is required is unnecessarily pessimistic. Standard triple treatment is recommended to be given for two weeks,³ as is the licensed dual treatment of omeprazole and amoxycillin. Promising one week regimens have also been reported.⁴

Record's discussion of whether eradication cures the disease is superficial. Record cites a single study, published only as a letter.⁵ Since 94% of the patients were positive for helicobacter at the time

of relapse the study is irrelevant to the efficacy of eradication of helicobacter in preventing relapse. Record neglects the substantial body of trial data showing that eradication of the organism is followed by a major reduction in, or even absence of, relapse in patients with both duodenal and gastric ulceration.⁴

Record implies that all 20 million carriers of helicobacter would have to be treated for the burden of gastroduodenal disease in the community to be altered. The evidence, however, supports eradication only in patients positive for the bacterium who have peptic ulceration. Results in non-ulcer dyspepsia are unpredictable and often disappointing, and the efficacy of eradicating the bacterium as prophylaxis against gastric cancer remains to be established. We believe that Peter C Rubin's commentary is correct: "If a person with peptic ulcer disease is shown to have *H pylori* then eradication is indicated."¹

J P MILLER
Consultant gastroenterologist
S G W JONES
Registrar in gastroenterology
Department of Medicine,
Withington Hospital,
Manchester M20 2LR

- 1 Hawkey CJ, Record CO. Eradication of *Helicobacter pylori* should be pivotal in managing peptic ulceration. *BMJ* 1994;309:1570-2. (10 December.)
- 2 El-Omar E, Penman I, Dorrian CA, Ardill JES, McColl KEL. Eradicating *Helicobacter pylori* infection lowers gastrin mediated acid secretion by two thirds in patients with duodenal ulcer. *Gut* 1993;34:1060-5.
- 3 Tytgat GNJ, Axon ATR, Dixon MF, Graham DY, Lee A, Marshall BJ. *Helicobacter pylori*: causal agent in peptic ulcer disease? In: *Working party reports of the world congresses of gastroenterology, Sydney, Australia, 26-31 August 1990*. Melbourne: Blackwell, 1990:36-45.
- 4 Tytgat GNJ. Treatments that impact favourably upon the eradication of *Helicobacter pylori* and ulcer recurrence. *Aliment Pharmacol Ther* 1994;8:359-68.
- 5 Bianchi Porro G, Lazzaroni M. *Campylobacter pylori* and ulcer recurrence. *Lancet* 1988;ii:593.

Gastric metaplasia should be considered too

EDITOR,—In maintaining that *Helicobacter pylori* does not cause peptic ulceration C O Record draws an analogy with the presence and effect of bacteroides in diverticular disease: the presence of bacteroides does not mean that the organism is the initial cause of the diverticulitis, the initial cause being the presence of diverticulosis.¹ Likewise, *H pylori* cannot exist in the duodenum without pre-existing gastric metaplasia of the duodenal mucosa and duodenal ulceration does not occur in the absence of gastric metaplasia. Despite *H pylori* being eradicated, gastric metaplasia remains unchanged, leaving a suitable environment for possible recolonisation.² More attention should be drawn to the elimination of gastric metaplasia and restoration of normal duodenal mucosa as well as the successful eradication of *H pylori*.³

FRANK I TOVEY
Research fellow
Department of Surgery,
University College London Medical School,
London W1P 7LD

- 1 Hawkey CJ, Record CO. Eradication of *Helicobacter pylori* should be pivotal in managing peptic ulceration. *BMJ* 1994;309:1570-2. (10 December.)
- 2 Noach LA, Rolf TM, Bosma NB, Schwartz MP, Oosting J, Rauws EAJ, et al. Gastric metaplasia and *Helicobacter pylori* infection. *Gut* 1993;34:1510-4.
- 3 Tovey FI, Yiu YC, Husband EM, Baker L, Jayaraj AP. *Helicobacter pylori* and peptic ulcer recurrence. *Gut* 1992;33:1293.

Management of stroke patients

EDITOR,—The two points of view in your debate on community care for stroke patients¹ are not necessarily in opposition. Organised stroke care is now recognised as desirable²; purchasers and

providers must ensure that a "seamless service" exists for patients with stroke.³ John Young does not seem to be arguing for a stand alone community stroke service but for improvement in the current disorganised state of community rehabilitation. While up to 70% of stroke patients are admitted to hospital, at least 30% are not, and some of the patients admitted could probably remain at home if better coordinated services existed.

To allow optimal outcomes all stroke patients need access to specialised rehabilitation, coordinated care, and secondary prevention; acute medical care may also increase in importance in the light of the international stroke trial and other current drug trials. Surely the answer is to have a comprehensive stroke rehabilitation service for each geographical area, with both hospital based and community based components? The hospital stroke rehabilitation unit would probably act as the base but should be coordinated with outreach services providing immediate assessment and ongoing rehabilitation for stroke patients in the community and follow up for people discharged from hospital whose needs are not met by other services such as day hospitals. The unit should also provide information for patients, carers, and other professionals about stroke care and local services and should be able to deal with the problems that patients and carers face. Clearly such a service would require close liaison between hospital services, primary care services, and community care teams, but such a service should largely answer the points raised by both Young and Lincoln.

CHRISTINE H MCALPINE
Consultant physician in geriatric medicine
Stirling Royal Infirmary,
Stirling FK8 2AU

- 1 Young J, Lincoln N. Is stroke better managed in the community? *BMJ* 1994;309:1356-8.
- 2 Dennis M, Langhorne P. So stroke units save lives: where do we go from here? *BMJ* 1994;309:1273-7.
- 3 Working group of the National Medical Advisory Committee. *The management of patients with stroke*. Edinburgh: HMSO, 1993.

BMA's representation of clinical and medical directors

Medical directors in Wales not represented

EDITOR,—I wish to correct the erroneous impression given in a report in *Medicopolitical Digest* that the BMA represents medical directors.¹ The new subcommittee of the BMA's Central Consultants and Specialists Committee may represent some medical directors of NHS trusts in England, but it represents none in Wales. At a conference held on 8 November the possibility of the BMA not representing medical directors was rapidly disregarded by the chairperson, although the obvious difficulties of such a relation were clearly recognised by many of those present from trusts outside Wales. The BMA is unwise to try to form a joint body of clinical and medical directors since their functions are as disparate as those of any two branches of our profession represented separately in the BMA's present structure.

Individual medical directors may be members of the BMA (as I am) and therefore represented by the organisation. In our official capacity, however, we in Wales believe that it would be a conflict of interest for us to be subsumed into a subcommittee of the body that represents the trade union interests of those with whom we are employed to negotiate on pay and conditions and whom we may even have to discipline. Besides this, some of the medical directors are not able to be represented directly by the Central Consultants and Specialists Committee, being general practitioners or academics. Perhaps the entire structure of the BMA needs alteration so that it matches all the changes taking place.