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]

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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 hypochlohydria 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]

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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]

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Management of stroke patients

EDITOR,—The two points of view in your debate on community care for stroke patients[1] are not necessarily in opposition. Organised stroke care is now recognised as desirable[2]; 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.

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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.