CORRESPONDENCE

- *All letters must be typed with double spacing and signed by all authors.*
- *No letter should be more than 400 words.*
- *For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the BMJ.*
- *We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.*
- *Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.*

Community care and patients with progressive conditions

SIR,—The government's postponement of its plans for community care has been a bitter blow to many disabled people and their carers—the new arrangements will now come too late for many. Perhaps we can use the extra time to clarify one of the vaguer aspects of the new policy: how effective links are to be established between health authorities and local authorities. This is a critical issue because one of the paradoxes of the current situation is that the chance of gaining access to community care services seems to be considerably improved by a period of inpatient care. Will the new system manage things better? We hope so, especially for the sake of the many thousands of people suffering from progressive neurological conditions, some of whom fare badly in hospital.

Patients with these conditions may need few community services in the early stages, and their urgent need for information, support, and early assessment often goes unrecognised. They can become hidden in the community with few links to the services they need as their disabilities increase. They do not demand—and often do not receive—services as quickly as those with acute onset illnesses requiring admission to hospital or high tech medical intervention. They therefore have a special need for one of the features of the new proposals—"proper assessment of need and good care management as the cornerstone of good quality care."

In Romford, Essex, we have been trying to provide this since 1985, at first for patients with Parkinson's disease but since early 1990 also for people suffering from multiple sclerosis, motor neurone disease, ataxia, and dystonia. We believe that "good quality care" has to start from the time of diagnosis and requires special attention to "telling," to counselling, and to the provision of information—not only about available services but also about options for treatment and care. It is essential that people with incurable illnesses retain some control over their own lives; otherwise their medical and social care can become one more assault on their dignity and self esteem. They also need a key worker so that links between the patient and one member of the multidisciplinary care team can be forged right from the beginning and an individualised system of monitoring established.

Early feedback suggests that we are on the right lines. A higher proportion of the project group (12/28) than of a comparison group (4/24) believed that the drug treatment had been adequately explained ($p=0{\cdot}05$), and many more (27/31 *v* 12/24; $p=0{\cdot}01$) had heard of the existence of the Parkinson's Disease Society. We are listening to the patients and learning from them. Perhaps their experiences and ours will seem relevant to those planning the new patterns of community care.

LESLIE J FINDLEY
MARIE OXTOBY

Neuro-Care Team,
Harold Wood Hospital, Romford RM3 0BE

Anti-D immunoglobulin for bleeding in early pregnancy

SIR,—A memorandum from the Department of Health written in 1976 states that all rhesus negative women who have a threatened or spontaneous abortion should be given anti-D immunoglobulin. General practitioners were made responsible for this and have been criticised for their lack of enthusiasm.

In 1988 I argued that there was no evidence to support the department's recommendation and that the only women who required anti-D immunoglobulin before 16 weeks' gestation were those who had an evacuation, which is a hospital procedure.[1] Contreras and Tovey agreed that anti-D immunoglobulin would be necessary after 13 weeks.[2-4]

In July 1989 the immunoglobulin working party accepted this proposal, but a formal letter had to be sent to the chief medical officer asking if the 1976 recommendation could be reviewed. This was done with the support of Dr Iain Chalmers (National Perinatal Epidemiology Unit) on 6 September 1989. Two years after it seemed to be agreed that anti-D immunoglobulin was being given unnecessarily the department has not relieved general practitioners of the obligation to give it to all rhesus negative women who bleed in early pregnancy. It is extremely time consuming to administer and a waste of a rare resource.

Every year in England and Wales there are probably 100 000 women with bleeding in early pregnancy who are not admitted to hospital. The current recommendation means that the general practitioner has to determine the woman's blood group immediately, and only if she is rhesus negative can anti-D immunoglobulin be obtained from the nearest district general hospital for injection within 72 hours after the bleed. There is no record of how many (unnecessary) doses have been given since 1976.

It is ironic that the department is keen to reduce costs in general practice but could save money by cancelling advice that was imposed in 1976. How many millions of pounds might have been better used elsewhere in the NHS during the past 14 years?

I would be interested to learn what is happening in Europe and other countries. Is this problem of inappropriate advice purely an English one and is the Department of Health likely to resolve it in the near future?

C B EVERETT

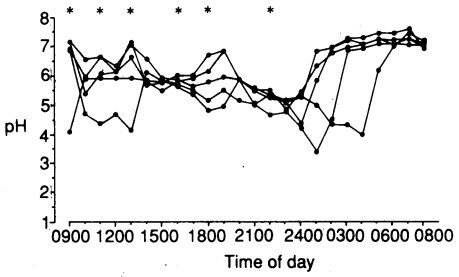
Alton,
Hampshire GU34 2QX

1 Everett CB. Is anti-D immunoglobulin unnecessary in the domiciliary treatment of miscarriages? *BMJ* 1988;**297**:732.
2 Contreras M. Is anti-D immunoglobulin unnecessary in the domiciliary treatment of miscarriages? *BMJ* 1988;**297**:733.
3 Tovey LAD. Anti-D and miscarriages. *BMJ* 1988;**297**:977-8.
4 Tovey LAD. Haemolytic disease of the newborn and its prevention. *BMJ* 1990;**300**:313-6. (3 February.)

Acid suppression

SIR,—Dr D G Colin-Jones is correct when he states that "As omeprazole is long acting, achlorhydria can be sustained throughout 24 hours,"[1] and Professor Richard H Hunt is wrong when he states that during dosing with omeprazole "the intragastric acidity profile reflects an intragastric pH profile that is unlike that of achlorhydria".[2]

The 24 hour intragastric pH when patients with pernicious anaemia are eating normal meals is within the range 4·2-7·7, with almost all values on the acid side of neutral.[3] This acidity probably comes from the ingested meals (pH range 5·1-6·4) together with the synthesis of organic acids within the stomach by bacterial overgrowth. Thus, patients with pentagastrin fast achlorhydria have low levels of intragastric acidity as assessed by a 24 hour profile.



Hourly intragastric pH in five of 18 patients with duodenal ulcer given omeprazole 20 mg every morning for 7-28 days. Asterisks indicate meals and snack times

We have measured profiles of 24 hour intragastric pH in 18 patients with duodenal ulcers dosed with omeprazole 20 mg every morning for 7-28 days.[4,5] The figure shows the hourly intragastric pH in the five patients with the lowest acidity (highest pH); about a third of patients given omeprazole 20 mg every morning have a profile of intragastric pH that "reflects achlorhydria" throughout the 24 hours. The proportion of patients having a profound decrease of acidity will rise when a dosage of 40 mg of omeprazole is used.[4]

Professor Hunt's interpretation of his meta-analysis of our data confuses the average results of a group with what may happen to the individual patient.

ROY POUNDER
EMILY PREWETT

Royal Free Hospital School of Medicine
London NW3 2PF

1 Colin-Jones DG. Acid suppression: How much is needed? *BMJ* 1990;**301**:564-5. (22 September.)
2 Hunt RH. Acid suppression. *BMJ* 1990;**301**:1047-8. (3 November.)
3 Lanzon-Miller S, Pounder RE, Hamilton MR, *et al*. Twenty-four hour intragastric acidity and plasma gastrin concentration in healthy subjects and patients with duodenal or gastric ulcer, or pernicious anaemia. *Alimentary Pharmacology Therapeutics* 1987;**1**:225-37.
4 Sharma BK, Walt RP, Pounder RE, Gomes M de FA, Wood EC, Logan LH. Optimal dose of oral omeprazole for maximal 24 hour decrease of intragastric acidity. *Gut* 1984;**25**:957-64.
5 Lanzon-Miller S, Pounder RE, Hamilton MR, *et al* Twenty-four hour intragastric acidity and plasma gastrin concentration before and during treatment with either ranitidine or omeprazole. *Aliment Pharmacol Therap* 1987;**1**:239-51.

SIR,—My letter on acid suppression[1] presented evidence for the degree to which acid should be suppressed for optimal duodenal ulcer healing, provided a scientific basis for the principles of acid suppression treatment, and attempted to correct a misconception relating to the pharmacology of omeprazole.

Unfortunately the letter underwent editorial revision, and the change of "by variation in the duration of the chosen antisecretory therapy" to "by varying the duration of treatment" in my final paragraph removed my clear acknowledgment of the wide choice of antisecretory drugs available.

Both papers to which I referred[2,3] have been widely recognised to consider the therapeutic principles that apply to all antisecretory drugs, regardless of the drug chosen. Their conclusions provide scientific evidence to balance the empirical advice presented in Dr Colin-Jones's editorial.[4] Furthermore, his interpretation of my final paragraph was entirely without foundation.[5] I singled out no drug by name nor, as he implied, in any way endorsed the widespread use of any product without first establishing a diagnosis.

Accurate diagnosis is not always available, and the benefits of endoscopy in patients under the age of 50 have been questioned.[6] The American College of Physicians advocates initial treatment with antacids or an H_2 receptor antagonist.[7] I agree with this pragmatic approach, which allows for those few dyspeptic patients who do not have ulcers and who respond to H_2 receptor antagonists. I do not believe, and have never advocated, that omeprazole should be considered at this point. If symptoms do not respond within two weeks endoscopy should be undertaken. For confirmed duodenal or gastric ulcer the doctor has a wide choice of drugs: if an antisecretory drug is described the duration of treatment will be determined by the degree of acid suppression. In gastro-oesophageal reflux disease conventional treatments provide relief of symptoms in mild to moderate disease, but only omeprazole so far has been found to be effective for healing grade 3 and 4 ulcerative oesophagitis.

As Dr Colin-Jones advocated endoscopy for all his patients it remains unclear why, for short term treatment, he would not offer those with confirmed duodenal or gastric ulceration or grade 3 to 4 gastro-oesophageal reflux disease treatment that would safely assure healing of most lesions in the shortest possible time.

RICHARD H HUNT

McMaster University Medical Centre,
Hamilton, Ontario L8N 3Z5,
Canada

1 Hunt RH. Acid suppression. *BMJ* 1990;**301**:1047-8. (3 November.)
2 Jones DB, Howeden CV, Burget DW, Kerr GD, Hunt RH. Acid suppression in duodenal ulcer: a meta-analysis to define optimal dosing with antisecretory drugs. *Gut* 1987;**28**:1120-7.
3 Burget DW, Chiverton SG, Hunt RH. Is there an optimal degree of acid suppression for healing of duodenal ulcers? A model of the relationship between ulcer healing and acid suppression. *Gastroenterol* 1990;**99**:345-51.
4 Colin-Jones DG. Acid suppression: How much is needed. *BMJ* 1990;**301**:564-5. (22 September.)
5 Colin-Jones DG. Acid suppression. *BMJ* 1990;**301**:1048 (3 November.)
6 Kahn KL, Greenfield S. The efficacy of endoscopy in the evaluation of dyspepsia. *J Clin Gastroenterol* 1986;**8**:346-58.
7 Health and Public Policy Committee, American College of Physicians. Position paper—endoscopy in the evaluation of dyspepsia. *Ann Intern Med* 1985;**102**:266-9.

**This correspondence is now closed.—ED, *BMJ*.

Forensic pathology

SIR,—Dr S Leadbeatter and colleagues claim that I was wrong to state that the police have exclusive access to the Home Office forensic science service because it "may undertake work for parties other than the police."[1] But, on their own admission, such work (for example, for the defence) has to be submitted to the service through the police and the results of any analysis have to be shown to the prosecution.

They imply that I should not have referred to the undergraduate teaching of forensic medicine as it "was not within the remit of the working party." That may be so, but it did not stop the working party reaching conclusions about how such teaching might be affected by changes in the forensic pathology service, and it is those conclusions that I criticised. They claim that Britain's forensic pathology service cannot be compared with those in Europe because the legal basis of the investigation of deaths differs among countries and that in at least one country the decision rests with the police. In fact the police or public prosecutor makes the decision in all European countries, but it is difficult to see how this is relevant. Furthermore, if comparisons of countries' forensic pathology services are invalid why did the working party, which visited at least one of them, not say so instead of ignoring their existence? Hence my title "A blinkered report."

They say that I failed to identify "the main problems" and "inevitable and radical reorganisation" to which I referred in the article. The main problem is that in this country there is no comprehensive, inclusive (that is, including forensic pathology), and unrestricted forensic science service available to all parties for all forms of legal action as exists in most of Europe. The inevitable and radical reorganisation is that which is needed to provide it.

Drs M A Green and A C Hunt take issue with my criticism of the working party's refusal to consider seriously the merging of forensic pathology with forensic science and medicine.[1] They say that "there is now little connection with the scientific aspects of forensic pathology and the techniques of forensic science." But the working party, of which they were both members, points out that "legal medicine now depends more than ever upon the close co-operation of experts in medicine and science." This is, of course, precisely what occurs in the European medicolegal institutes.

Professor D J Gee points out that forensic science was not in the remit of the working party and that as there are 600 forensic scientists and only 40 Home Office pathologists, the working party represented a "minority interest" (presumably forensic pathologists).[1] But this did not prevent the working party, of which the controller of the Home Office forensic science service was a member, taking important decisions about the relation between forensic pathology and forensic science in the conclusions that I criticised.

The Home Office's decision to set up separate working parties to review each of the two services must have seemed strange to our European colleagues and may have been aimed at avoiding the inconvenience and expense of the reorganisation that is needed to provide a service similar to that available in most of Europe. However carefully the Home Office worded the terms of reference, the working party took the view that it could not avoid referring to several issues outside its remit, including the relation of forensic science with forensic pathology. Having done so, it is difficult to see why the conclusions it reached on those issues should then be exempt from criticism as seems to be the view held by your correspondents.

J D J HAVARD

London N1 3DL

1 Correspondence. Forensic pathology. *BMJ* 1990;**301**:1160-1. (17 November.)

SIR,—At the recent meeting of the forensic medicine subcommittee it was reported that some readers believed that Dr J D J Havard's editorial represented BMA policy on the subject.[1] This is not the case, and I am anxious to set the record straight and to dissociate this committee from your editorial comments.

The association was represented on the Wasserman committee and fully supported its recommendations. We pressed repeatedly for the report's publication and then for its acceptance, both by direct contact with the Home Office and indirectly through the support of interested MPs.

Since the report's acceptance we have, through our regional services, supported members in drawing up local agreements with police authorities. Centrally, we have at last secured considerable increases in the derisory payments to forensic pathologists attending inquests. We deeply regret the timing of your editorial, which unbeknown to you came at a time when all these negotiations were nearing completion.

D E PICKERSGILL

Forensic Medicine Subcommittee,
British Medical Association,
London WC1H 9JP

1 Havard JDJ. Forensic pathology: a blinkered report. *BMJ* 1990;**301**:943-4. (27 October.)

Flat feet in children

SIR,—In dealing with flat feet in children in the short space available I realise that Mr M A Smith could give only a very general overview of this subject.[1]

Although he is correct that in the vast majority of children the infantile flat foot is "relentlessly corrected," it is important that the pathological flat foot should be identified at an early age and treated efficiently. The clue to this was provided by Harris and Beath, following their study of Canadian soldiers, in which they showed that the hypermobile flat foot was the commonest condition that caused downgrading or even discharge.[2]

It becomes important, therefore, to identify these pathological flat feet at the earliest possible age and to institute treatment. We have shown treatment to be effective and have defined the criteria for identification.[3,4] These include late walking (quite commonly not recognised by paediatricians), hypermobility of other joints, and a number of specific tests. The principle is not to make this judgment by appearance but by functional criteria.

The principle of treatment is equally important—namely, to maintain the foot in the corrected position until ligamentous laxity disappears, an event paralleling that in the normal child but very considerably delayed. Treatment, therefore, must be continued until the stability of the foot has been restored. If this condition is not identified ossification and hardening of the tarsal bones, to which Mr Smith refers, will occur with deformation, which perpetuates the condition.