mm Hg on three occasions and atenolol therapy was started on 11 September (general practitioner's records). He presented with abdominal pain in April 1979 and was referred to hospital. Therefore he took atenolol for seven and a half months before the onset of symptoms that were found to be due to retroperitoneal fibrosis at operation in July 1979. The authors state that the "patient had taken no other drugs in the six months prior to his presentation with abdominal pain." This is not so. According to the general practitioner the patient had been on clofibrate for a long time and prescriptions were still being issued at appropriate intervals.

In November 1975 the erythrocyte sedimentation rate (ESR) was raised to 24 mm in one hour (general practitioner's records). Before operation, on 11 July 1979, the ESR was found to be raised on two occasions, 91 mm in one hour in May and 106 mm just before operation (information from surgeons). The letter by Mr Johnson and Mr McFarland does not state that hypertension and raised ESR are known to be associated with idiopathic retroperitoneal fibrosis.¹ Two expert pathologists have examined the histological appearances of the tissue removed at operation and both state plainly that they are those of idiopathic retroperitoneal fibrosis. The histological appearances of proctolol peritonitis are different and distinguishable. The surgeons recorded post-operative blood pressure (no antihypertensive therapy) of 120/80 mm Hg on 17 July 1979, 110/70 on 18 July 1979, and 160/100 on 6 September 1979. This last reading was taken in outpatients after the patient had made a long journey to the hospital. The blood pressure was normal again a month later when he was seen by the general practitioner, who recorded a reading of 120/80 mm Hg.

It is reasonable to conclude that the abdominal symptoms and hypertension were caused by idiopathic retroperitoneal fibrosis and that both were cured by surgery.

In the concluding paragraph there is an implication that retroperitoneal fibrosis associated with atenolol may bear a similar causal relationship to the sclerosing visceral peritonitis brought about by proctolol. There is no evidence that the peculiar and unique fibrotic reactions caused by proctolol have ever been shown to arise in connection with the use of any other beta-blocker. In view of the widespread use of this class of medicines it is scarcely surprising that, from time to time, a chance association with the idiopathic form of retroperitoneal fibrosis should be reported. It would be unfortunate if physicians who regularly use beta-blockers should be given a wrong impression.

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¹ Hawk WA, Hazard JB. *Am J Clin Pathol* 1959;32:321.

ECT: balancing risks and benefits

SIR,—It is difficult to counter the unpromising denunciation of unmodified electric convulsion therapy (ECT) by Dr P E Bebbington and others (15 March, p 792) by reasoned argument, since their letter contains so little. They declare that unmodified ECT "should be removed from consideration as a treatment approach in any circumstances" (my italics). It is not clear why this should be a sacred principle.

They appeal to the Royal College of Psychiatrists' guidelines. But guidelines are guidelines, not laws carved in stone; and these guidelines do not make any such unpromising declaration. Dr Bebbington and others express concern for the peace of mind of their patients and their relatives. I do not know about the patients and the relatives of the patients of Dr Bebbington and his nine colleagues; but mine are inclined to demand explanations. I try to promote their peace of mind by explaining the purposes and risks of a proposed treatment on the basis of up-to-date medical knowledge and balanced clinical judgment, not on the basis of an ideology recited in unison. Dr Bebbington and his colleagues are jealous of the good name and reputation of British psychiatry, and so they should be: British psychiatry has a reputation for common sense, clinical and scientific objectivity, and freedom from dogma. These things are valued also by some of the lesser breeds beyond the law.

Professor D A Pond (9 February, p 403) presents a moderate, balanced, reasoned point of view which makes good clinical sense. He reminds us that the reason for modifying ECT is prosaic and practical: to reduce the risk of fracture. He wishes to point out merely that, on occasion, a general anaesthetic may be contraindicated or not available, although there remains a strong indication for ECT. Once in a while the balance of risks may be in favour of unmodified ECT.

There is an adage in medicine: "Never say always; never say never." When, at rare moments in medical history, we do have to say "never," when treatment that has until that moment been a part of clinical practice is to be totally banned without room for clinical judgment, the evidence must be strong and the reasoning conclusive. Dr Bebbington and his colleagues have presented no evidence and little reasoning.

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Compliance and epilepsy

SIR,—In reply to Dr P T White and Dr E G Buckley (22 March, p 864) we would draw their attention to the fact that there have been several studies of drug compliance with epileptic patients.¹⁻⁵ All suggest that poor compliance is a major problem. Our own observations (16 February, p 484), which are based on low or falling blood drug concentrations in association with a recurrence of seizures, are merely in line with previous experience, albeit in a different patient population—new hospital referrals. All the published studies, however, have been in hospital-based populations, and we therefore await with interest the details of their seemingly encouraging and desirable results in a practice-based study.

We cannot agree that we have shown that "the responsibility for failure in the treatment of epilepsy usually lies neither with the disease itself nor with the patient, but with the doctor." In our experience all three make their contribution in varying degrees. A long history of untreated or poorly treated attacks, or seizures associated with brain lesions (both static and, especially, progressive ones) detectable on computed tomography are

frequently associated with failure of optimum therapy with one or more drugs. Certainly, many doctors could do much more to improve the results of treatment especially in the early stages by applying modern knowledge of the clinical pharmacology of the drugs, or by explaining more carefully to patients the implications of starting therapy, the need for good compliance and regular supervision, and the pitfalls to be avoided (both with respect to drug therapy and to other factors which may provoke seizures). Part of the problem is that epilepsy is usually a very long term, often lifetime, disorder. Indeed, in hospital practice it is remarkable how patients accumulate even more doctors than drugs, especially as they are often seen by a succession of registrars. We agree there is a need for much more education in this matter.

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¹ Lund M, Jorgensen RS, Kuhl V. *Epilepsia* 1964; 5:51-8.

² Gibberd F, Dunne J, Hadley A, Hazleman B. *Br Med J* 1970;i:147-9.

³ Lund M. *Acta Neurol Scand* 1973;49:295-306.

⁴ Wilson JT, Wilkinson GR. *Neurology* 1974;24: 614-23.

⁵ Eisler J, Mattson RH. *Epilepsia* 1975;16:203.

Topical corticosteroids on the face

SIR,—Dr M Whitefield's letter (29 March, p 941) regarding the possible link between perioral dermatitis and topical corticosteroids lacking a free 17-OH group ignores two points.

Firstly, hydrocortisone 17-butyrate is readily converted to 21-butyrate outside the liver, even to the extent of conversion in standard vehicles if the pH is not carefully controlled (pH 3.5-4.2). Secondly, Wilkinson cites two cases of perioral dermatitis occurring after hydrocortisone itself.¹ It would therefore be more logical to suppose that perioral dermatitis is a manifestation of topical corticosteroid therapy in general. Dr Whitefield's argument that hydrocortisone does not form a reservoir in the epidermis may partly explain why it is relatively inactive in the treatment of steroid-responsive dermatoses.

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¹ Wilkinson D. *Br J Dermatol* 1979;101:245-57.

Sulphasalazine in rheumatoid arthritis

SIR,—The letters of Professor H L F Currey and his co-workers at the London Hospital (22 March, p 861) are of considerable interest. I am sure there is general sympathy with the expressed view that before a new drug is generally accepted as a useful drug it should be subjected to rigorously controlled studies.

I am less in sympathy with the implied suggestions that editorial policies should not allow uncontrolled studies to be published in reputable medical journals. Without access to studies such as that of Dr B McConkey and others (16 February, p 427), no statistician would be able to predict the number of patients required for a rigorously controlled