the headings "Danger of overreaction" and "Two sides of the coin" he ends by white-washing one of the worst abuses of our century by comparing, with some sympathy, the dilemma of the Soviet government in its abuse of psychiatry with that of the Catholic church in dealing with heretics or the Romans in dealing with the early Christians. Surely that is the point, that we are now faced with an evil of similar dimensions which should be vigorously condemned, not minimised because brutal suppression by the establishment has occurred before.

In his first paragraph Dr Hare infers that oppression in modern Russia is merely a continuation of that under the Tsars. Yet has he really taken into account the sheer scale of terror in Russia since the communist régime came to power?

We should avoid doublespeak and condemn oppression unequivocally wherever it occurs.

C H MAYCOCK

Crediton, Devon

Pneumoconiosis and chronic bronchitis

SIR,—We regret that in our recent paper "Pneumoconiosis and chronic bronchitis" (13 August, p 424) we wrongly attributed to Hart and Aslett¹ the suggestion that chronic bronchitis might protect against pneumoconiosis. In fact they mentioned the equally pertinent but contrary hypothesis, that bronchitis might impede dust elimination, but were unable to reach definite conclusions on this from the data available. Our paper showed no interaction either way.

D C F MUIR

J BURNS

M JACOBSEN

W H WALTON

Institute of Occupational Medicine,
Edinburgh

¹ Hart, P D'A, and Aslett, E A, *Chronic Pulmonary Disease in South Wales Coalminers*, Special Report Series No 243. London, Medical Research Council, 1942.

Oestrogens for menopausal flushing

SIR,—Since our study¹ has been quoted in the controversy between Mulley *et al*^{2,3} and Campbell *et al*^{4,5} may we be allowed to comment on some points of difference between our trial and that of Campbell *et al*?⁶

We did not separate our patients into "mild" and "severe" groups; patients were selected who complained of symptoms listed in the Kuppermann Index⁷ and included some women with a few flushes and three with none. Dilution of the group of severe cases by these milder ones would cause an apparent reduction in the difference between oestrogen and placebo. However, after excluding the three patients with no flushes the proportional reduction in mean flushes on oestrogen compared with placebo reached significance ($P < 0.05$).

Campbell informed his patients that "their tablets would be of different strengths"; ours were told that they would receive either an inert preparation or oestrogen for three months each. Campbell found a similar placebo effect whether it was given before or after Premarin. We found a significant difference: patients changing to placebo after oestrogen complained of flushes which were often more

severe than at the start of the trial. This informed them that they had stopped taking oestrogen and the trial became no longer "blind" in this group. Consequently for analytical purposes placebo in the first three months only was compared with oestrogen in the first or the second periods.

We conclude that oestrogens are highly effective in relieving severe menopausal flushing and that placebos are effective for mild cases only if given *de novo* because of the "rebound phenomenon" of severe flushes in women who go on to take placebo after three months on oestrogen.

A cautious approach to long-term therapy is justified on the grounds of economy in prescribing and also to prevent heavy expenditure of scarce time and money on medical supervision and regular curettage in patients at risk of endometrial cancer. Short-term therapy is helpful and safe particularly in hysterectomised patients.

JEAN COOPE

L POLLER

Bollington Medical Centre,
Macclesfield, Cheshire

Haematology Department,
Withington Hospital,
Manchester

¹ Coope, J, Thompson, J M, and Poller, L, *British Medical Journal*, 1975, **4**, 139.

² Mulley, G, and Mitchell, J R A, *Lancet*, 1976, **1**, 1397.

³ Mulley, G, and Mitchell, J R A, *British Medical Journal*, 1977, **2**, 766.

⁴ Campbell, S, and Whitehead, M J, *British Medical Journal*, 1977, **1**, 104.

⁵ Campbell, S, *et al*, *British Medical Journal*, 1977, **2**, 1218.

⁶ Campbell, S, *et al*, in *Management of the Menopause and Post-Menopausal Years*, ed S Campbell, p 149. Lancaster, Medical and Technical Publishing, 1976.

⁷ Kuppermann, U S, Blatt, M U G, and Wiesbaden, U, *Journal of Clinical Endocrinology*, 1953, **13**, 88.

Ischaemic colitis and drug abuse

SIR,—Mr A R Turnbull and Dr P Isaacson (15 October, p 1000) rightly stress the role of hypotension in the development of bowel necrosis in young adults. We have seen a similar case of necrotising colitis after self-poisoning.

A 27-year-old woman attempted suicide by taking approximately 75 tablets of Distalgic (dextropropoxyphene and paracetamol). She was found unconscious and developed fits and apnoea. Her blood pressure was unrecordable on admission and remained below 80 mm Hg (systolic) for the next 20 hours. In addition to routine measures she required intermittent positive pressure ventilation and she was haemodialysed. She was also given nalorphine and, to control fits, intravenous diazepam. She regained consciousness and apparent wellbeing within eight hours of admission.

Twenty-four hours later she complained of lower abdominal pain. This became worse, she developed generalised peritonitis, and laparotomy was performed. Patchy gangrene was found in the caecum and ascending, transverse, and proximal part of the descending colon. There was a small amount of free gas. The main vascular supply of the colon was quite normal and the vascular arcades were pulsating. The gangrenous colon was excised and ileosigmoid anastomosis was carried out.

Histological examination of the excised colon showed necrosis of the whole bowel wall, with inflammatory infiltration and arteritis of small vessels. The larger vessels were normal. No clostridial organisms were seen.

Necrosis of the colon without vascular obstruction was termed necrotising colitis in 1961.¹ The aetiology has been attributed to *Clostridium welchii* organisms found in the lumen of the large bowel and invading the muscularis of the colon.² However, it is clear

that in our patient the cause was prolonged hypotension consequent on a drug overdose.

R S J BRIGGS

K G BARRAND

M LEVENE

Royal Sussex County Hospital,
Brighton, Sussex

¹ Killingback, M J, and Lloyd Williams, K, *British Journal of Surgery*, 1961, **49**, 175.

² Tate, G T, Thompson, H, and Willis, A T, *British Journal of Surgery*, 1965, **52**, 194.

ECT and the media

SIR,—May we comment briefly on Mr K Sabbagh's letter (5 November, p 1215)?

That he has to produce excerpts in favour of ECT from a single programme is surely evidence of the bias he denies. It has become fashionable for the media to focus on only one aspect of a problem or to present only one side of the argument. We know the programme lamenting the fate of the thousands languishing in mental hospitals will be followed by one criticising the policy of discharging schizophrenic patients into the community, and the programme attacking social workers who failed to protect battered children will be followed by one deploring their readiness to use compulsory powers.

Mr Sabbagh may feel that his need to "worry" people justifies such a loaded approach to society's problems, but if the BBC has come to believe that there are no absolute values and no ultimate truths it would at least be more honest and responsible to present conflicting views in the same programme.

J F ANDERSON

Secretary,

Stratheden Hospital Medical Committee

Cupar, Fife

Susceptibility of bacteroides to noxythiolin

SIR,—We were interested in the report by Dr B Chattopadhyay (29 October, p 1121) of noxythiolin-resistant *Pseudomonas aeruginosa* in which he emphasises the importance of checking the sensitivity of pathogenic organisms before using this preparation.

Noxythiolin is widely used for peritoneal lavage and instillation into other sites which may be infected with *Bacteroides fragilis*. Despite an extensive literature search including the work of Haler and Aebi,¹ which is incorrectly cited in the list of references given by Dr Chattopadhyay, we have been unable to find a report of the susceptibility of this organism. We present here the results of our own studies.

Fifty-one clinical isolates of *B. fragilis* from different patients were tested by an agar dilution technique on DST agar (Oxoid) with 5% lysed horse blood using an inoculum of approximately 10⁶ organisms. Plates were incubated overnight in Gaspak jars (Baltimore Biological Ltd) at 37°C and the minimum inhibitory concentration (MIC) recorded. The minimum bactericidal concentration (MBC) was determined by replica plating on to antibiotic-free medium and reincubating. The MIC was 0.125 g/l for 18 isolates, 0.25 g/l for 28 isolates, and 0.5 g/l for the remaining five. The MBC was four times the value of the MIC.

The rate of killing was also determined on three isolates by a method similar to that described by Dr Chattopadhyay but using doubling dilutions from 20 g/l to 1.25 g/l and also including a

noxythiolin-free control. A 10- μ l sample from each tube was plated on blood agar at 0, $\frac{1}{2}$, 1, 2, 4, and 24 h. The results for one of the organisms tested with 10^6 bacteria added to 1 ml of solution are shown in the table. It can be seen that 2 h exposure was required for killing by a 10 g/l solution of noxythiolin, although the MBC as previously determined was 2 g/l. Two other organisms (MBC 1 g/l) when tested with 10^6 organisms/ml showed complete killing by 20 g/l in 1 h, by 10 g/l in 2 h and by both 5 g/l and 2.5 g/l in 4 h.

Presence of persisting Bacteroides fragilis after exposure to noxythiolin

Time of exposure (hours)	Noxythiolin concentration (g/l)					
	20	10	5	2.5	1.25	0
0	+++	+++	+++	+++	+++	+++
$\frac{1}{2}$	+++	+++	+++	+++	+++	+++
1	++	++	++	++	++	++
2	—	—	+	++	++	++
4	—	—	—	++	++	++
24	—	—	—	—	—	—

The results of our own studies on *B. fragilis* and those of Dr Chattopadhyay on *Ps. aeruginosa* lead us to believe that the disc sensitivity test or determination of MIC or MBC as routinely applied to the study of antibiotics may be of limited value. We feel that assessment of the rate of killing is the only method likely to give a meaningful result for topical agents which may otherwise be instilled for too short a period of time, with long periods of time between treatments during which regrowth could occur. We doubt, however, whether laboratories could devote the time needed to assess the rate of killing in every case.

R H GEORGE
D E HEALING

Department of Microbiology,
Birmingham Children's Hospital,
Birmingham

¹ Haler, D, and Aebi, A, *Nature*, 1961, **190**, 734.

Drinking behaviour

SIR,—Your leading article entitled "Drinking behaviour" (8 October, p 914) is rather misleading in its emphasis. Alcohol is rarely a main cause of the conditions attributed to its abuse^{1, 2}; only a tiny minority of British drinkers (perhaps 0.05-0.1%) develop serious complications. Where there is an association with cirrhosis, marital strife, dementia, work-time lost, etc, the alcohol ingestion may well be a correlate or even a consequence rather than a cause.

Mental, physical, and social illnesses cause alcoholism rather than the other way round. Thus even when alcoholics abstain totally they tend to be overtly disturbed.⁴ If nuclear alcohol dependency is usually a symptomatic phenomenon the crucial lesson is that we must search exhaustively for underlying, often treatable, pathology. If alcoholism is disease-based the present expensive educational drive by David Ennals in the north-east to reduce the Geordies' intake is doomed to failure.

Excessive drinkers might wreak even more havoc on themselves or society without the refuge of the bottle⁵ which serves as a "sinister but effective" peacemaker. This pain-relieving model suggests a more heuristic, treatment-oriented typology than the rather ineffective WHO restatement of Jellinek's excellent descriptive classification:

(Ia) Those "normals" whose life problems, leading to excessive intake, may be soluble by a

non-organic approach—for example, family counselling. This type, often a middle-class minority, has traditionally and, I feel, most unfortunately been confused with the following categories.

(Ib) Those "normals" for whom sociopsychological measures alone are insufficient and who therefore need pharmacological help while their underlying stress continues. A relevant analogy is the snoker who starts smoking much more when subjected to stress. Both may continue to indulge out of habit when the stress is removed, but not to excess any more. This group can return to "social" drinking, as an increasing number of studies is now showing.⁷

(II) The "core" alcoholics needing long-term drug help in their effort at tension-discharge. They typically suffer from chronic physical and/or psychiatric illness. Female alcoholics are over-represented here in that they tend to be more psychologically disturbed.⁸

Oral medication is usually unsuitable for maintenance treatment.⁹ The obvious answer is the depot phenothiazine,^{10, 11} which is not only our most effective anti-aggressive agent but may even block a possible toxic-addictive complex of dopamine with a tetrahydroisoquinoline derivative of ethanol.

If this tension-reduction approach should prove scientifically fruitful we will have achieved a breakthrough in the exorcism of a very nasty "demon." The bell may toll only for those not given the chance of treatment with a depot neuroleptic.

H G KINNELL

Northgate Hospital,
Morpeth, Northumberland

¹ Benjamin, I S, Inrie, C W, and Blumgart, L H, in *Alcoholism: New Knowledge and New Responses*, ed G Edwards and M Grant, p 198. London, Croom Helm, 1977.

² *World Health Statistics Annual*, 1973-1976. Geneva, WHO, 1976.

³ Galambos, J T, in *Progress in Liver Diseases*, ed H Popper and F Schaffner, vol 4, p 567. New York, Grune and Stratton, 1972.

⁴ Gerard, D L, Saenger, G, and Wile, R, *Archives of General Psychiatry*, 1962, **6**, 83.

⁵ *Sunday Express*, 18 September 1977, p 21.

⁶ Proudfoot, A T, and Park, J, in *Alcoholism: New Knowledge and New Responses*, ed G Edwards and M Grant, p 224. London, Croom Helm, 1977.

⁷ Litman, G K, *Once an Alcoholic, Always an Alcoholic: A Review and Critique* (Addiction Research Unit paper). London, Institute of Psychiatry, 1977.

⁸ Beckman, L J, *Quarterly Journal of Studies on Alcohol*, 1975, **36**, 797.

⁹ Proudfoot, A T, and Park, J, in *Alcoholism: New Knowledge and New Responses*, ed G Edwards and M Grant, p 222. London, Croom Helm, 1977.

¹⁰ Kinnell, H G, *Lancet*, 1977, **2**, 659.

¹¹ Bartholomew, A A, *Quarterly Journal of Studies on Alcohol*, 1966, **27**, 510.

Cimetidine and gastric ulcer healing

SIR,—Dr R J Machell and his colleagues suggest (15 October, p 1023) that the great effect of cimetidine found in our double-blind gastric ulcer trial (24 September, p 795) may partly have been due to unsuccessful randomisation, especially because the healing rate in the placebo group was very low. We should of course have been happier if the retrospective stratification had not shown that the treatment group and the control group were, by chance, not comparable in all respects, but as discussed in the paper this fact probably does not bias the results in favour of cimetidine. Also properly conducted randomised therapeutic trials are subject to stochastic variation, but it is worth pointing out that our findings are in very good agreement with those of the only previously published complete trial.¹

It is necessary that we learn the truth about the effect of cimetidine in a disease which is as common as gastric ulcer disease, but the full truth will emerge only when more trials have been published. Dr Machell and his colleagues

mention that their trial is not complete and that a significant effect of cimetidine "has yet to emerge." This statement worries us because usually it is assumed that the results of a randomised trial are not known until the completion of the study. Otherwise it may be difficult to decide when to stop the trial.

We agree very much with Mr R S Arnot (15 October, p 1022), who states that treatment with cimetidine may lead to an increased incidence of missed gastric carcinomas if the patients are not examined sufficiently early.

K GOTLIEB JENSEN
S RUNE
H R WULFF

Ribe County Hospital,
Glostrup University Hospital,
Herlev University Hospital,
Denmark

¹ Multicentre Trial (France), in *Cimetidine*, ed W R Burland and M Alison Simkins, p 287. Amsterdam and Oxford, Excerpta Medica, 1977.

Depression in men and women

SIR,—Your recent leading article on this subject (1 October, p 849) very rightly focused attention on the significantly higher incidence of depression in women during their reproductive years as compared with men of comparable age and also older women. The two articles which are discussed in detail^{1, 2} make some mention of the possible reasons for this, but come to no very firm conclusions on the relative importance of endocrine and other factors.

We should like to support the view that depression occurring in a cyclical manner during the premenstrual days has an endocrine component in many cases, and to advance the hypothesis that subtle neuroendocrine imbalance may well exist in the cases in which it is at present not possible to detect endocrine abnormality. Recent work from this department on patients with the premenstrual syndrome (PMS) has demonstrated a deficiency of plasma progesterone during the luteal phase in 40% of the patients investigated^{3, 4} and preliminary studies suggest a tendency to increased prolactin levels when progesterone levels are significantly decreased.⁵ Serum oestradiol levels may also be slightly increased in the late luteal phase.⁶ These findings are in general agreement with those of Carstensen in Uppsala.^{7, 8} A tendency to increased prolactin levels in PMS has also been reported by others.^{9, 10}

It is encouraging that several recent approaches to the treatment of PMS have given beneficial results which are clearly well above the maximum (30%) placebo effect likely to be encountered. These various approaches have recently been discussed¹¹ and their rationale in PMS considered. They include the use of dydrogesterone, especially in patients with a luteal deficiency of progesterone,¹² and high-dose pyridoxine therapy.¹³ Both these treatments may give complete or partial relief of symptoms in about 75% of selected cases. Research currently in progress suggests that further improvement in relief of symptoms can be obtained by individualising dose regimens and sometimes by combining dydrogesterone and pyridoxine therapy. A single-blind trial on the use of dydrogesterone in PMS is now almost completed and a similar trial using pyridoxine is about to start.

We therefore feel that there are grounds for