black market dealings, but it does not stop them. (2) Does it cure? In my humble opinion, No. And I doubt if it ever will. It does lead to trading of NHS drugs. (3) Is it a good thing for us to let them receive sick pay and supplementary benefits, and stay at home on their drugs? (4) These people are a potential hazard to others. They have an "infectious," almost incurable, and very dangerous disease which will probably cause many early deaths. (5) Anyone who spreads addiction, sorrow, early death, for personal gain is far worse than someone who kills in the heat of the moment. They merit the most severe penalty. It must be made impossible for them ever to do this again. (6) Other forms of treatment: Although "patients" they must be restricted by law and treated as compulsory inpatients until the severe withdrawal symptoms are past. They should then be sent to hostels, work camps, etc, where the regimen would have to be kind but strict, administered by a dedicated staff who could not be "bought." No contact with the outside world would be allowed, no parcels, no unopened letters, visitors very rarely, and strictly supervised, only, say, a quarter of the inmates being visited at any one time.

This re-education in the broadest sense would have to continue for quite a time, and when patients were allowed to go home a kindly but strict and frequent supervision would have to be kept over them until they were considered completely and permanently cured. I realise how difficult all this would prove, and how expensive to institute, but not much less would be effective. Dedicated people are hard to find nowadays and they would be likely to have to face vilification by folks of small understanding in the outside world and vituperation from those inside. The possibility of litigation could not be ruled out. They would need wholehearted and firm backing by the law. If *real* cures, *permanent* cures, resulted, perhaps this difficult and expensive treatment would prove worth while.

C R BARRINGTON

Bromley, Kent

Toxic megacolon in Crohn's disease

SIR,—Your leading article on the above subject (27 September, p 723) refers to a recent report from this institution.[1] Our preferred method of primary surgical treatment for toxic megacolon is that of loop ileostomy and decompression blow-hole colostomy.[2] In a group of 49 patients recently reported[3] there was one death, a mortality of 2%. All patients in the group detoxified and decompressed following this procedure, the average hospital stay being 18 days. Two patients required early colectomy during the original hospitalisation for persistent colonic haemorrhage. Colectomy was subsequently performed on 41 of these patients at a mean interval of six months, and in eight patients an ileorectal anastomosis was able to be performed. Pathological examination of the colectomy specimens showed a 2:1 predominance of transmural colitis 66% to mucosal ulcerative colitis 29%, with 5% being in the non-specific group.

On the basis of these results we feel that less than colectomy is indicated in the surgical management of the toxic megacolon phase of inflammatory bowel disease, and that total proctocolectomy can hardly ever be justified in this condition. It seems logical to us to perform a comparatively simple and effective operative procedure in an acutely ill patient, and in this way to prepare the patient for colectomy at a time when the acute illness has passed.

D G JAGELMAN
V W FAZIO
R B TURNBULL

Department of Colon and Rectal Surgery,
Cleveland Clinic Foundation,
Cleveland, Ohio, USA

1 Farmer, R G, Hawk, W A, and Turnbull, R B, *Gastroenterology*, 1975, **68**, 627.
2 Turnbull, R B, *et al*, *Surgical Clinics of North America*, 1970, **50**, 1151.
3 Fazio, V W, *Gastrointestinal Emergencies*. Philadelphia, W B Saunders. In press.

Serum α-fetoprotein in cystic fibrosis

SIR,—Professor R K Chandra and others (29 March, p 714) reported significantly increased levels of α_1-fetoprotein (AFP) in the serum of patients with cystic fibrosis (CF), in the parents of the patients, and in some of their siblings in Newfoundland, Canada. These findings were confined by Dr J A Smith (17 May, p 392) in the United States for both homozygote and heterozygote carriers of CF genes. In contrast to Professor Chandra, Dr D J H Brock and others in Scotland and Dr J C Wallwork and others in England (17 May, p 392) were unable to detect an increased serum concentration of AFP in any of their CF patients or their relatives. So far as we know, serum AFP levels have not been measured in homozygote and heterozygote carriers of CF genes in central Europe.

Over the past four months we have examined the sera of 38 patients (aged 2 to 18 years) with CF of various severity of the disease for the presence of raised serum levels of AFP. Except for one Turkish boy, all patients with CF were of German origin. The diagnosis of CF was based on both clinical symptoms of pulmonary and pancreas involvement and sweat sodium-chloride levels above 80 mmol/l on pilocarpine iontophoresis. All patients with CF except one 7-year-old boy had normal values of SGOT, SGPT, and the other liver-specific serum enzymes.

The determination of AFP in the serum was carried out by radioimmunoassay with ^{125}I-labelled AFP in the double antibody technique. The first antibody was a rabbit anti-human AFP-serum, the second antibody a goat-anti-rabbit-γ-globulin. The test was carried out in 0·1 M borate buffer containing 0·5% beef albumin as described by Nishi and Hirai[1] and Ishii.[2] Incubation periods were always 24 hours for the first antibody, the radioactive labelled AFP, and the second antibody. ^{125}I-labelled AFP and the antisera were obtained from Dianabot-Laboratories, Tokyo, Japan. Crystalline AFP, isolated as described by Lehmann,[3 4] was used as standard by Mancini's radial immunodiffusion technique. The concentration of crystalline AFP (local standard) was 90 μg/ml in the International Standard Preparation 72/225 of the World Health Organisation (Lyon, France).[7] The normal range was calculated from the values of 62 healthy adults: the mean value of AFP in the serum was 4·52 ng/ml, the standard deviation 1·92 ng/ml, the upper limit of the 2·57-ς-range, including 99% of all values, 9·45 ng/ml.

The serum AFP levels in our 38 patients with CF were not different from those in healthy adults. The average serum AFP level of all patients with CF was 3·08 ng/ml (range 1-10 ng/ml). The standard deviation was 2·27 ng/ml. In infants and children the normal range of the serum AFP levels is even higher than in healthy adults. Therefore the serum AFP levels in our patients with CF are within the normal range for healthy children of the same age. In addition, the sera of all 38 patients with CF were examined both by electroimmunoosmophoresis with anti-human AFP-serum (limit of sensitivity 3·6 μg/ml) and by double diffusion (limit of sensitivity 10 μg/ml) as described by Lehmann and Lehmann.[6] Our findings correspond with those of Dr Wallwork and others and of Dr Brock and others. These authors also, in contrast to both Professor Chandra and others and Dr Smith, have been unable to detect an increased serum concentration of AFP in any of the CF patients.

There is no explanation for this discrepancy, as Professor Chandra and his colleagues have apparently used the same rabbit antiserum and standards as Dr Brock and others. In our opinion, genetic differences cannot explain the different results concerning the serum AFP levels in patients with CF. The majority of the ancestors of the patients with CF who have been tested in the USA and in Canada may have their origin in central Europe and the northwestern part of Europe.

G KNÖPFLE
H W ROTTHAUWE

University Kinderklinik,
Bonn, West Germany

F G LEHMANN

University Medical Klinik,
Marburg, West Germany

1 Nishi, S, and Hirai, H, *Cancer Research*, 1973, **14**, 79.
2 Ishii, M. *Cancer Research*, 1973, **14**, 89.
3 Lehmann, F-G, Lehmann, D, and Martini, G A, *Clinica Chimica Acta*, 1971, **33**, 197.
4 Lehmann, F-G, and Lehmann D *Zeitschrift für klinische Chemie und klinische Biochemie*, 1971, **9**, 309.
5 Mancini. G, *et al*, *Colloquium on the Protides of the Biological Fluids*, 1965, **11**, 370.
6 Lehmann, F-G, and Lehmann, D, *Zeitschrift für klinische Chemie und klinische Biochemie*, 1973 **11**, 399.
7 Sizaret, P, *et al*, *Journal of Biological Standards*, 1975, **3**, 201.

Whooping-cough vaccine

SIR,—The statement by the Joint Committee on Vaccination and Immunisation (20 September, p 687) concerning the continued use of whooping-cough vaccine is important, if only for the data it contains. These show that as in previous epidemics mortality from whooping cough in the 1973-4 epidemic occurred almost entirely in infants under 12 months. Current DHSS inoculation policy leaves this age group relatively or completely unprotected by direct vaccination, and any benefits of the inoculation programme appear to depend on herd immunity promoted among children in whom mortality from pertussis has always been much less. The success of this recently modified inoculation policy operating in the context of an increasing proportion of parents born after systematic pertussis inoculation was introduced, and who probably do not now have significant protection against pertussis,[1] remains to be seen.

It is not possible to judge from the committee's data the extent to which the promotion of community protection may be offset by the hazard from a severe encephalopathic reaction in an inoculated child past the age of maximum hazard from the natural