genuine absent-mindedness which can beset any of us under pressure.

Shoppers are well advised not to take bags or commodious purses into stores, but rather easily-carried expansible net or plastic carriers to be loaded from baskets at the check-out point.

Thus in shoplifting cases and sometimes those where medical issues might reasonably be involved—for example, sexual offences, non-accidental injury to children, baby-stealing, amnesic syndromes, automatism, alcoholism, compensation cases, etc, a deal can often be done, particularly where relevant medical notes antedating the legal issue are available. One can attempt to spare the individual and the family concerned often additional, unnecessary, and unjustifiable trauma, as this might have devastating and potentially avoidable effects upon the emotional, marital, social, and possibly employment conditions and future stability of those concerned.

MONTAGUE SEGAL

Department of Psychological Medicine,
Halifax General Hospital,
Halifax, West Yorks

Epilepsy

SIR,—I am writing in support of the letter from Dr D P Addy (22 November, p 460) which contained several meritorious and significant criticisms relative to Dr F B Gibberd's article on the treatment of epilepsy (1 November, p 270). I am in complete agreement that Dr Gibberd's presentation of the minor motor epilepsies and their treatment is less than satisfactory and am gratified to note that Dr Addy recommends the ketogenic diet as a therapeutic regimen for this disorder. While Dr Addy limits its usage to "resistant and severe cases," we employ the ketogenic diet regimen as our therapeutic instrument of first choice for the control of minor motor seizures.

Minor motor attacks are exceedingly refractory to the available anticonvulsant drugs, which, in our experience, have been essentially unrewarding. We have not obtained beneficial results with the standard antiepileptic agents—that is, ethosuximide, methsuximide, phenobarbitone, phenytoin, primidone, and troxidone—nor with other drugs recommended by some physicians—for example, acetazolamide, chlortetracycline, and pyridoxine. In addition, our experience with the use of the benzodiazepines (clonazepam, diazepam, nitrazepam) in the treatment of approximately 500 children with minor motor seizures indicates that they are of limited value in most cases. We achieved prolonged seizure control with these medications in only rare instances.

Our 40 years' experience with the use of the ketogenic diet regimen (4:1) in the treatment of minor motor seizures has consistently demonstrated it to be the most efficacious form of therapy for this type of epilepsy.¹⁻³ During this period we have studied and treated approximately 1500 children with minor motor epilepsy. We prescribed the ketogenic diet regimen to 915 of these patients with the following results: seizures were controlled in 485 (53%); there was marked improvement in 238 (26%); and 192 (21%) did not respond to this therapy. Anticonvulsant medication had been given adequate trial in 732 of this group and 183 had received no previous treatment.

In addition to its anticonvulsant properties,

the ketogenic diet produces a marked tranquillising effect in some children, does not impair mental acuity as antiepileptic drugs frequently do, and is singularly free of toxicity. Details relative to our method of prescribing and managing the ketogenic diet regimen are presented in a recent publication.³

SAMUEL LIVINGSTON

Epilepsy Clinic,
Johns Hopkins Hospital,
Baltimore, Maryland

¹ Livingstone, S, *The Diagnosis and Treatment of Convulsive Disorders in Infants and Children*. Springfield, Thomas, 1954.

² Livingstone, S, in *Advances in Pediatrics*, ed S Z Leoine. Chicago, Year Book Publishers, 1958.

³ Livingston, S, *Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence*. Springfield, Thomas, 1972.

Cell-mediated immunity in patients with cystic fibrosis

SIR,—In their paper on this subject (17 January, p 120), Mr A R Gibbons and his colleagues make the following statement: "In those patients with cystic fibrosis (CF) who responded well to prednisolone treatment their initially depressed migration index (MI) returned to normal, which suggests that impaired cell-mediated immunity plays a part in the pathogenesis of CF."

A low MI signifies sensitivity to an antigen. The expression of such sensitivity can be achieved only by functionally active immunocompetent cells. It is misleading to suggest that such a migration index represents an impairment of cell-mediated immunity (CMI).

It is also invalid to suggest that steroids could reverse an impaired state of CMI. Rather the reverse is true, that steroids might cause impairment of CMI.

CONLETH FEIGHERY
ROBERT WOODS

Central Immunology Laboratory,
School of Pathology,
Trinity College,
Dublin

* * * We sent a copy of this letter to Mr Gibbons and his colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—We agree with the comment of Dr Feighery and Mr Woods that a low migration index signifies sensitivity to an antigen which can be achieved only by functionally active immunocompetent cells. However, we must emphasise that the functions of active immunocompetent cells may or may not be impaired, depending upon the antigens to which such cells are sensitised. Since comparative cells from age-matched controls and from blood donors did not show such sensitisation to those antigens to which the CF cells were sensitised then there is no doubt that the normal function of these immunocompetent cells in the patients with CF is indeed impaired.

When one considers that steroids are known immunosuppressants it is certainly not surprising that treatment of the CF patients with prednisolone caused a decreased sensitivity of these sensitised CF cells to the antigens. Indeed, therapeutic concentrations of steroids were able to reduce the ability of sensitised spleen cells and of lymph-node cells to destroy target cells *in vitro*.¹ Apart from this inhibition of sensitised cells by steroids, 90% of sensitised lymphocytes were killed when prednisolone

1 mg/l was present during the sensitisation phase.² In addition it was observed in man that if cortisol or prednisolone was given two hours before abrading the skin, then there occurred a marked reduction of emigration of both mononuclear cells and polymorphs into the area, thus inhibiting the mobilisation of macrophages.³ We believe that the situation with regard to steroid therapy and inhibition of migration of leucocytes from CF patients is similar.

HYLTON MCFARLANE
ALAN R GIBBONS

Department of Medical Biochemistry
University of Manchester

¹ Rosenau, W, and Moon, M D, *Journal of Immunology*, 1962, **89**, 422.

² Cohen, I R, Stauby, L, and Feldman, M, *Journal of Experimental Medicine*, 1970, **132**, 1055.

³ Boggs, D R, et al, *American Journal of Pathology*, 1964, **44**, 763.

Diagnosis of invasive aspergillosis

SIR,—Opportunist fungal infections in recipients of organ transplants, open heart surgery patients, and others¹⁻¹⁰ are becoming an increasing problem. Aspergillosis is particularly troublesome because the infecting fungus is seldom cultured, the disease progresses very rapidly, and consequently diagnosis is most commonly made post mortem.²⁻⁵⁻⁸ Where treatment (amphotericin B) is given it is often unsuccessful, although there is evidence that early diagnosis would improve its effectiveness.³

Detection of fungal antigens or metabolites in patients' serum would aid the early diagnosis of infection, and monitoring serum antigen or metabolite levels might also assist in determining the effectiveness or required duration of treatment. Cryptococcal antigen detection is a routine test for the diagnosis of cryptococcosis, and detection of candida metabolites in serum shows promise as a means of rapidly diagnosing systemic candidiasis.⁷

We are currently looking for circulating aspergillus antigens and metabolites in the sera of infected animals which may find application in the improved diagnosis of systemic human infections. Because of the poor live diagnosis rate and, in absolute terms, the rarity of the disease in humans the difficulty we face is obtaining sera from known positive cases of invasive aspergillosis. If any readers have any such sera or would be willing to co-operate in storing sera from possible cases until diagnosis were made we would very much like to hear from them.

L O WHITE
M D RICHARDSON
R C WARREN

Department of Microbiology
University of Birmingham,
Birmingham B15 2TT

¹ *British Medical Journal*, 1975, **3**, 264.

² Bach, M C, et al, *Lancet*, 1975, **1**, 180.

³ Burton, J R, et al, *Annals of Internal Medicine*, 1972, **77**, 383.

⁴ Gallis, H A, et al, *Archives of Internal Medicine*, 1975, **135**, 1163.

⁵ Herberston, B M, Royal College of Pathologists symposium on systemic fungal infections, 1974 (unpublished).

⁶ Kammer, R B, and Utz, J P, *American Journal of Medicine*, 1974, **56**, 506.

⁷ Murray, H W, et al, *Johns Hopkins Medical Journal*, 1975, **137**, 235.

⁸ Rubenstein, E, et al, *Medicine*, 1975, **54**, 331.

⁹ Turner, E, et al, *Chest*, 1975, **67**, 262.

¹⁰ Zazgornik, et al, *Deutsche medizinische Wochenschrift*, 1975, **100**, 2082.

¹¹ Miller, G G, et al, *Journal of Clinical Investigation*, 1974, **54**, 1235.