Fitness for diving

SIR,—In answer to a recent question (17 April, p 949) whether it would be safe for a young person to dive if he had a history of an abnormal electroencephalogram (EEG) and had been treated for epilepsy until the age of 11 your expert's reply was that "it is as safe for this young man to do underwater diving as it is for anyone."

We believe this guidance to be potentially dangerous. In fact, this guidance is immediately contradicted by the next sentence of the answer, which states that in various circumstances under water "he would be more likely to have a fit than someone without his previous history." It needs to be realised that a fit occurring under water, whatever its cause, can be rapidly fatal.

Standards of fitness to dive are laid down by various authorities. Current British naval practice would not accept an individual with an EEG suggestive of epilepsy even if he had never had a fit. United States Navy regulations state that "organic brain disease seizure disorders of any sort . . . shall be disqualifying." The memorandum for medical officers who have been approved by the Secretary of State, Department of Energy, to undertake the medical examination of commercial divers in the North Sea states that epilepsy is among the conditions which disqualify a man from diving. The British Sub-Aqua Club has recently decided to exclude from diving those with a history of epilepsy.

Under the weight of these separate opinions it would seem somewhat more responsible for the physician to recommend that this particular individual should seek some other sport or occupation.

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****Our expert writes: "It is true that if someone were to have a fit under water his chances of dying would be high. The first point to make is that this young man is aged 21 or more; and, this being so, he has had no fits for 10 years. To all intents and purposes such a person should not be regarded as suffering from epilepsy and he should lead a normal life, doing any job he wants to take and driving motor-cars. When I read the question I thought only of diving for sport with aqualungs and was not thinking of commercial or naval diving. Aqua-lung diving was, I presume, what the question was about. I quite see that, whereas a young man who is keen on aqua-lung diving would be willing to take the very small risk of ever having a fit while diving, the Navy cannot take this responsibility for someone else."—ED, *BMJ*.

Guillain-Barré syndrome

SIR,—The essential contribution of Guillain, Barré, and Strohl[1] to the study of peripheral neuropathy was the discovery, in their cases, of a raised cerebrospinal fluid (CSF) protein with a normal cell count—the "cytoalbuminologic dissociation." The three patients described by Drs A Royston and B J Prout (17 July, p 150) are of considerable interest, but as the CSF was normal in two they cannot be properly described as simulating the Guillain-Barré syndrome.

Eponyms change meaning over the years, but it is a pity to forget the basis of the original definition.

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[1] Guillain, G, Barré, J A, and Strohl, A, *Bulletin de la Société Médicale des Hôpitaux de Paris*, 1916, **40**, 1462.

Diet and malignant disease

SIR,—In answer to a question on the treatment of histiocytic lymphoma (19 June, p 1522) your expert says, "I know of no good evidence that diet can influence the course of this or any other malignant disease in man." May I refer you to your leading article entitled "Small bowel tumours" (18 January 1975, p 115), where it is stated with reference to coeliac disease and malignancy that "both lymphoma and carcinoma occur, but the incidence of carcinoma is reduced if the patient is treated with a gluten-free diet"?

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****Our expert writes: "There is no conflict between what I said in my answer and what was said in the leading article mentioned by Mr Smith. My answer referred to the possibility that the course of malignant disease might be influenced by diet. The leading article refers to the incidence. There is quite a lot of evidence to suggest that the remarkable geographical variations in incidence of various kinds of malignant disease, especially bowel cancer, might be related to differences in diet, but I repeat that I know of no good evidence that diet can influence the course of any malignant disease. Of course, this is not to say that it does not do so. It may well do so. I am just not aware of any good evidence that it does."—ED, *BMJ*.

Mechanism of action of antiallergic drugs

SIR,—Drs J C Foreman and L G Garland (3 April, p 820) have provided an excellent summary of current thinking on the mechanism of the anaphylactic reaction and on the way in which cromoglycate and similar compounds may inhibit this process. Their experiments[1] and other recently published evidence[2] support the hypothesis that drugs of this type inhibit the entry of calcium into the cell. We would, however, disagree with their suggestion that the inhibition of the anaphylactic reaction caused by some of these compounds is attributable to their inhibition of cyclic adenosine monophosphate phosphodiesterase (cAMP-PDE). Our disagreement is based on the following observations.

(1) We have found no correlation between the ability to inhibit anaphylactic release of histamine from rat mast cells.[4] This is illustrated for cromoglycate, AH7725, and theophylline in the table. All have the same order of activity as inhibitors of cAMP-PDE but not as inhibitors of histamine release.

(2) The kinetics of inhibition of histamine release by cromoglycate, AH 7725, and many other antianaphylactic compounds are not those expected of inhibitors of cAMP-PDE.

	Inhibition of cAMP-PDE (human lung) (k_i)	Inhibition of histamine release (rat mast cell) (ID_{50})
Cromoglycate	$1{\cdot}1 \times 10^{-4}$ mol/l	$9{\cdot}8 \times 10^{-6}$ mol/l
AH 7725	$2{\cdot}5 \times 10^{-4}$ mol/l	$4{\cdot}5 \times 10^{-5}$ mol/l
Theophylline	$2{\cdot}3 \times 10^{-4}$ mol/l	$2{\cdot}8 \times 10^{-3}$ mol/l

Cromoglycate and similar drugs must be added to the mast cell simultaneously with antigen in order to inhibit effectively the release of histamine.[4-6] Preincubation of the drugs with the cells for as little as 10 minutes before adding antigen renders the drugs much less active. In comparison, theophylline and more potent inhibitors of cAMP-PDE must be preincubated with the cells before adding antigen to produce significant inhibition of the release of histamine. This is understandable as inhibitors of cAMP-PDE require time to raise the intracellular concentration of cyclic AMP.

Even if cromoglycate in high concentration was found to raise the concentration of cyclic AMP in mast cells it would not be good evidence that this is its primary mode of action in inhibiting anaphylaxis because of its obvious activity against the cells at much lower concentrations. Similarly the modest degree of synergism reported for mixtures of cromoglycate and isoprenaline[7] is less than we would expect for a mixture of an effective inhibitor of cAMP-PDE and a beta-stimulant. Thus we would not dispute the fact that increases in the intracellular concentration of cyclic AMP reduce the release of histamine from mast cells, but we do not believe that cromoglycate and similar compounds exert their inhibitory action in this way.

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[1] Garland, L G, and Mongar, J L, *International Archives of Allergy and Applied Immunology*, 1976, **50**, 27.
[2] Spataro, A C, and Bosmann, H B, *Biochemical Pharmacology*, 1976, **25**, 505.
[3] Brooker, G, *et al*, *Biochemistry*, 1968, **7**, 4177.
[4] Fullarton, J, Martin, L E, and Vardey, C J, *International Archives of Allergy and Applied Immunology*, 1973, **45**, 84.
[5] Kusner, E J, Dubnick, B, and Herzig, J, *Journal of Pharmacology and Experimental Therapeutics*, 1973, **184**, 41.
[6] Thomson, D S, and Evans, D P, *Clinical and Experimental Immunology*, 1973, **13**, 537.
[7] Taylor, W A, *et al*, *International Archives of Allergy and Applied Immunology*, 1974, **46**, 104.

Alcohol and the brain

SIR,—Your leading article on this subject (15 May, p 1168) draws attention to damage to brain function related to alcoholism. A study carried out by Mary Marshman[1] on a series of patients admitted under my care at this hospital further emphasises the degree of cerebral dysfunction demonstrable by psychological studies in people seeking treatment for alcoholism. The findings were that of this particular group 76% showed signs of cerebral dysfunction on psychometric testing. This was associated with a very much higher level of reported hostility and personality disturbance, combined with social disruption, than is usually reported in the literature.

I wonder how much this degree of cerebral dysfunction influences the ability of these people to take advantage of the psychotherapeutic methods of "treatment" which are the principal approach to the management of