enriched with eicosapentaenoic acid given to patients with asthma over 10-12 weeks did not, however, lead to any
symptomatic improvement or to objective changes in lung function and non-specific bronchial hyperresponsiveness.
22,23 Although dietary fish oil lipids produced no change in most patients with asthma, eicosapentaenoic acid may modulate the disease in a few people. Picado et al showed that a diet containing 3 g of eicosapentaenoic acid daily for six weeks worsened airflow obstruction in 10 patients with asthma and aspirin intolerance.14 These effects were attributed to inhibition of the cyclo-oxygenase pathway.

Thus adding eicosapentaenoic acid to the diet will lead to it being incorporated into membranes and tissues, which may result in important changes in cellular biochemistry and function and may provide some benefit in selected diseases. Coronary artery disease is the condition that is most amenable to this dietary manipulation, but whether the benefit is sufficient to replace or reduce drug treatment in any condition remains to be seen.

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441-53.
3 Hirai A, Hamazaki T, Terano T, et al. Eicosapentaenoic acid and plasminogen function in
6 Harris WC, Connor WE, McMurry MP. The comparative reduction of the plasma lipids and lipoproteins by dietary polyunsaturated fats: salmon oil vs. vegetable oils. Metabolism 1983;32:
179-84.
7 Needham P, Raz A, Minkes MS, Ferrendelli JA, et al. Tissue prostaglandins: prostaglandin and thromboxane biosynthesis and unique biological properties. Proc Natl Acad Sci USA 1979;76:
944-8.
8 Gorey EJ, Shih C, Cashman JR. Docosahexaenoic acid is a strong inhibitor of prostaglandin but not leukotriene biosynthesis. Proc Natl Acad Sci USA 1983;80:1581-4.
9 Whitaker MO, Weche A, Fitzpatrick F. Prostaglandin D3 and eicosapentaenoic acid: as potential
10 Frischer S, Weber P. Prostaglandin E1 is formed in vivo in man after dietary eicosapentaenoic acid.
13 Samuelsson B. Leukotrienes: mediators of immediate hypersensitivity reactions and inflammation.
14 Lee TH, Hoover RL, Williams JD, et al. Effect of dietary enrichment with eicosapentaenoic and
docosahexaenoic acids on in vitro neutrophil and monocyte leukotriene generation and
15 Spering RL, Robin J-L, Kylander K A, et al. The effects of N-3 polyunsaturated fatty acids on the

A single seizure

Likely to recur

In 1881 Gowers concluded that when a single seizure has occurred others usually follow, but this view has recently come under scrutiny, with some ensuing controversy.1-4 General practitioners, casualty officers, neurologists, and paediatricians commonly see patients who have had a single seizure. Sometimes it has occurred because of alcohol or drug abuse, acute metabolic disturbance, acute cerebral disease or injury, or (especially in children) fever. More often, however, none of these factors are present and the seizure is regarded as unprovoked, although various reflex mechanisms, changes in sleep pattern, and emotional stress may be overlooked. Recent reports on the prognosis of a single unprovoked attack have seemed to conflict and management remains uncertain.

In Britain most patients are not treated after a single seizure on the principle that a single seizure is not epilepsy, though in the United States two thirds of patients are treated, perhaps for medicolegal reasons. Both the British and the American practice have recently been questioned.

In a multicentre study of patients presenting to neurological departments in Britain Hopkins et al confirmed that most single seizures (94%) are tonic-clonic attacks.1 Partial attacks are usually more frequent, may occur in clusters, and initially are often not recognised as seizures; they thus present to doctors as a single event much more rarely. A substantial minority of patients with tonic-clonic attacks also seek advice only after two or more seizures.1 In patients presenting with a single seizure the rate of recurrence has been reported to vary between 27% and 71% after three years of follow up.7 10 Two recent studies based on children referred to electroencephalography departments found rates of relapse of 59%6 7 and 52%.11 In a retrospective community study based on the records linkage system of the Mayo Clinic Annegers et al reported recurrence of seizures in 56% of patients after five years.12 All the studies agree that relapse occurs most often within the first year of follow up. In 408 adults over 16 the risk of recurrence was greater if the seizure occurred between midnight and 9 am, and older patients with a family history of seizures also seemed more likely to have a recurrence, but electroencephalography was of no predictive value.7 Computed tomography showed tumours in only 3%, and these subjects have a higher rate of relapse.

Some of the variation in the reported rates of relapse may be attributable to differences in the ages of the populations studied, differences in the types and causes of the single seizures, whether the studies were prospective (most were retrospective), and whether some of the patients were treated with antiepileptic drugs. The most important factor, however, is the interval between the seizure and the time of presentation and entry into the study. In patients with established epilepsy the second attack follows the first within one month in one third of patients.1 4 Therefore if one month elapses before a patient with a seizure is seen in a neurological clinic those with early recurrence will be selected out because they have already developed epilepsy. In the study of patients
with single seizures by Elwes et al, which had the highest rate of relapse (71%), all the 133 patients were identified within one day of the attack. Only 103 of them, however, had had a single seizure by the time they were seen in a neurological clinic within one month; their rate of relapse was 57%. Similarly Hopkins et al reported a rate of relapse of 52% in patients seen in neurological clinics within one week but of only 22% of those seen after eight weeks. The recurrence rate of 39% of Cleland et al was in patients seen at least six weeks after the event. 13

Epidemiological and general practice studies support the view that in most patients seizures are recurrent. In Rochester, Minnesota, only 23% of patients had a single seizure7; the corresponding figure from Greater Aarhus, Denmark, 14 was 16%. In a general practice in Kent only 18% of seizures were isolated. 15

It thus seems that Gowers was right as usual. Overall there is a high rate of relapse after a single seizure; but for the individual patient the probability of recurrence falls in proportion to the time between the seizure and presentation to a hospital clinic. Early relapse results in presentation to neurologists or paediatricians with epilepsy. Whether the patient with a single attack should be treated with an antiepileptic drug has never been adequately investigated. Among other factors, 19 the decision may be influenced by how soon the patient is seen: the earlier the patient is seen the higher the probability of recurrence and so perhaps the greater the need for treatment. Because of the suggestion that early treatment may prevent chronic epilepsy 11 16 there is an urgent need to study the management of patients with a single attack.

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Torture

Research needed into how to help those who have been tortured

In 1975 the World Medical Association defined torture as “the deliberate, systematic, or wanton infliction of physical and mental suffering by one or more persons acting alone or on the orders of any authority to force another person to yield information, to make a confession, or for any other reason.” Amnesty International stated in 1984 that some 66 governments either practised or condoned torture. 1 It estimates that hundreds of thousands of people are tortured, but gathering exact figures is obviously impossible.

The medical understanding of the consequences of torture has come mainly from rehabilitation centres in Canada and Denmark, and a post-torture syndrome is now well recognised. 12 Dr Inge Kemp Genefke, the founder and medical director of the Danish centre, will next week be presented in the Swedish parliament with one of this year’s right livelihood awards, which are also known as “the alternative Nobel prizes” (p 1428).

Patients may experience psychosomatic symptoms (pains, headaches, nightmares, night terrors, insomnia, tremor, dizziness, fainting, sweating, and diarrhoea); affective symptoms (depression, anxiety, fears, and phobias); behavioural symptoms (withdrawal, irritability, aggressiveness, impulsiveness, sexual dysfunction, and attempts at suicide); and intellectual and mental symptoms (confusion, disorientation, and loss of memory and concentration). 21-11 Cortical and central atrophy have also been reported in five previously healthy young men who had been tortured. 14

The Danish rehabilitation centre has a team of specialist physicians, psychiatrists, psychologists, social workers, physiotherapists, and lawyers. 15 Treatment deals with the physical and psychological sequelae of torture, tackles social, work, and family problems, and aims at integration into a new society. Although torture is so common, an effective response to psychological problems after torture has been slow. There are no controlled studies of the components of treatment. Reports on the outcome of treatment are all uncontrolled and anecdotal. 13-17

The symptoms seen after torture commonly resemble the focal phobias that may follow trauma and thus could be termed “traumatic phobias.” 18 Systematic exposure to cues that evoke fear until the ensuing emotion subsides is an effective treatment for phobias and this might also work in those who have been tortured. 19 Although there are no controlled studies of exposure in traumatic phobias, some case reports of effective treatment with fantasy desensitisation or imaginal exposure to cues and memories related to the trauma are published. 20,22 One of us (MB) has seen a woman with agoraphobia induced by torture who was almost house-bound because of an unrealistic fear of nearest and further torture. She was successfully treated with 10 sessions of graded exposure to circumstances related to her torture that induced anxiety—for example, the sight of a policeman.

Research on treatment of problems after torture is difficult for many reasons. Torture is a highly political issue with international implications. Governments almost never acknowledge its practice. This factor alone impedes any systematic efforts at treatment within those countries. But as