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Hazards of epilepsy

Bland words of reassurance do little to relieve the anxiety of parents fearful that a fit during sleep may kill their epileptic child. Familiarity with recurrent fits may eventually give them confidence—as they believe that the next fit will be as stereotyped and innocuous as the last. Some parents, however, do not get over the shock of witnessing the first convulsion, convinced that their child—apnoeic, stiff, and cyanosed—was dying; and it may lead to such a degree of watchful overprotection that epilepsy comes to dominate the life of the family.

A divided nightly vigil shared by parents is perhaps the extreme variant of this fear, but some worry about suffocation in a nocturnal fit is common, especially in parents of the very young. This may lead them to install a baby alarm, or share the same bed (usually with the willing collusion of the child, whose demanding behaviour may reflect the exceptional indulgence which parental anxiety breeds). This anxiety—and the restrictions of freedom which it generates—must be seen as counterproductive. Though difficult to measure, psychosocial factors are widely believed to influence the frequency and character of fits in children (as in adults), and in many children there appears to be a vicious circle of parental anxiety and poor control of fits. Yet much of our medical advice serves to consolidate rather than relieve these difficulties—for example, the recommendation of anticonvulsant medication is sometimes bolstered by the threat of brain damage through non-compliance, and swimming and cycling may be forbidden because of the harm that might accompany a fit occurring in potentially hazardous circumstances.

How valid are these well-intentioned but frustrating restrictions? Most studies of comparative risk rates for drowning, suffocation in bed, or trauma in patients with epilepsy have been based on patients in long-stay hospitals or mental subnormality units and have not included corrections for differing policies of management.[1-4] Simply because so many epileptics are dissuaded from swimming and driving, statistics of drowning and of road traffic accidents cannot identify the size of the potential hazard. Nevertheless, what evidence we have suggests that misadventure is relatively rare.

For example, the Brisbane Drowning Study[3] showed that, though the risk of drowning was four times greater in epileptic children who swam than in normal children, the absolute risk remained low.[6] Among the 107 deaths at all ages in the 10-year review of patients at the Norwegian State Hospital for Epilepsy and in the neurological unit of the University of Oslo three were due to accident and 11 to drowning.[2] Other population studies have shown few or no excess deaths due to epilepsy except in those patients (mostly children) who are mentally or physically handicapped,[7] who tend to have more severe epilepsy anyway. At a children's psychiatric hospital[1] there was no difference in the rate of injury among epileptic and non-epileptic children "engaged in a full athletic programme" (2·9% and 2·8% respectively). From the infrequency of reports of death during sleep[8] apparently due to suffocation during a fit the hazard may be presumed rare—especially in comparison with the frequency of nocturnal fits in children. Even so, substitution of a firm pillow might be a wise safeguard.

Generally, therefore, the picture is reassuring. Most of the traditional restrictions on epileptics, though well justified in theory, can be waived in practice, though not entirely abandoned. The enlightened medical director of the largest residential school in Britain for children with epilepsy has encouraged building a swimming pool at his school, arguing that the need to be able to swim is even greater for his pupils than for others. Swimming under supervision should be possible for all but the most poorly controlled patients. Nevertheless, for bathing a shower is preferable to the bath-tub for those children who demand privacy.

Cycling, too, is permissible for children with well-controlled fits or with "situation-dependent" or nocturnal attacks, if they have access to uncluttered highways. Sadly, for many (including normal) children this is an almost unattainable ideal in large conurbations. What constitutes good control of fits is an arbitrary judgment. Important factors in the decision whether or not to discourage cycling include not only the frequency of fits but also the amount of warning. School teachers often need guidance as much as parents. Their fearful prejudices may be unnecessarily restrictive, and they need an enlightened view of the management of epilepsy with a willingness to include rather than exclude sufferers in school activities.

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Epidemic myalgic encephalomyelitis

Outbreaks of the paralytic disease known as epidemic myalgic encephalomyelitis have puzzled doctors all over the world in the past 30 years. One of the best known of these epidemics was that at the Royal Free Hospital in London in 1955, which affected more than 300 people.[1] Most outbreaks tend to occur in the summer, young adults are predominantly affected, and the incidence is higher in women. The evidence suggests that infection is spread by personal contact, and young hospital personnel seem particularly at risk. The features common to

every epidemic include headache, unusual muscular pains (which may be severe), lymphadenopathy—often of the posterior cervical lymph nodes—and low-grade fever.[2 3] In a minority of cases frank neurological signs can be detected by careful clinical examination: there may be nystagmus, diplopia, myoclonus, bulbar weakness, motor weakness, increased or decreased tendon reflexes, disturbances of the sphincters, and extensor plantar responses.[2-7] Fasciculations, cranial nerve lesions, and extrapyramidal signs have also been reported. Most patients complain of paraesthesiae, and sensory loss is common.[1-4] One characteristic feature of the disease is exhaustion, any effort producing generalised fatigue. Often there are psychiatric abnormalities, especially emotional lability and lack of concentration.[1 3 4] The clinical outcome may take any of three courses: some patients recover completely, some follow a relapsing course, and some are permanently incapacitated.[3]

At a symposium held recently at the Royal Society of Medicine to discuss the disease and plan research there was clear agreement that myalgic encephalomyelitis is a distinct nosological entity. Other terms that have been used to describe the disease were rejected as unsatisfactory for various reasons: the cardinal clinical features show that the disorder is an encephalomyelitis; "Iceland disease" is not specific enough; and "neuromyasthenia" suggests a relation to myasthenia gravis whereas the muscle fatigability is different, as are the electrophysiological findings.[8] Indeed, the exhaustion and tiredness are similar to that described by patients with multiple sclerosis.[9] From the patient's point of view the designation benign is also misleading, since the illness may be devastating. Originally the term was used because no deaths had been recorded from myalgic encephalomyelitis. Two patients who had had the disease have now been examined post mortem: one was found to have multiple sclerosis. The adjective epidemic is correct, since most cases occur in an epidemic, but the disease may be endemic, and sporadic cases may occur.[10-12]

Some authors have attempted to dismiss this disease as hysterical,[13] but the evidence now makes such a tenet unacceptable. Some purely psychiatric symptoms may well occur, particularly in patients entering the chronic phase. No doubt, too, in an epidemic some hysterical persons will simulate the symptoms of the disease. Nevertheless, the organic basis is clear—from the finding that the putative agent can be transferred to monkeys[14]; the detection of an increased urinary output of creatine[2 15]; the persistent finding of abnormal lymphocytes in the peripheral blood of some patients[16]; the presence of lymphocytes and an increased protein concentration in the cerebrospinal fluid of occasional patients[3]; and the neurological findings. At this symposium more evidence was produced to support the organic nature of the disease. Increased serum concentrations of lactic dehydrogenases and transaminases have been found in several patients examined during the acute attack. In a recent outbreak in London immunological studies showed a high incidence of serum anticomplementary activity and the presence of ill-defined aggregates on electron microscopy of acute-phase sera.[17] A perplexing finding, suggesting the possibility of a persistent virus infection, was the ability of lymphocytes from patients to proliferate and survive in vitro for up to 19 weeks. The results of electroencephalographic studies were also stated to be abnormal, confirming other reports.[10]

We still know nothing about the nature and cause of epidemic myalgic encephalomyelitis, but outbreaks are still occurring. Future epidemics should be studied by a collaborative team of neurologists, epidemiologists, virologists, and immunologists. Its findings would be important not only for the study of epidemic myalgic encephalomyelitis but also for other neurological disorders, including multiple sclerosis.

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Respiratory complications of rheumatoid disease

The known respiratory complications of rheumatoid disease include diffuse pulmonary fibrosis,[1 2] lung nodules with[3] and without[4] pneumoconiosis, pleural effusions and fibrosis,[5] increased susceptibility to respiratory infections,[6] airways obstruction,[6] cricoarytenoid arthritis,[7] and pulmonary arterial obstruction.[8] Estimates of the frequency of these respiratory complications in patients with rheumatoid arthritis have varied from 1·6% when based on radiographic appearances alone[9] to over 45% when lung function tests were combined with chest *x*-ray films.[10]

The diffuse pulmonary fibrosis of rheumatoid arthritis is twice as common in men as women and may precede joint changes in 15-20% of affected patients. The common appearances on the chest film are of non-specific diffuse bilateral shadows in the lower zones (as in other forms of fibrosing alveolitis), though pleural changes are seen more often in patients with rheumatoid disease. The histological picture may include specific rheumatoid features such as fibroblast pallisading with hyaline and necrotic collagen as well as the alveolar wall fibrosis and desquamation of alveolar cells common to other forms of fibrosing alveolitis.

Initially these changes may produce no symptoms; by the time the chest film has become abnormal patients usually have breathlessness and cough with finger clubbing and basal crepitations. Again, lung function tests may show no abnormality in asymptomatic patients, but there is usually a restrictive ventilatory defect with reduced gas transfer when symptoms are present. Recent studies have shown impairment of carbon monoxide diffusing capacity in patients without symptoms[11] and in others with normal chest *x*-ray films.[12] Such changes could be due either to alveolar wall fibrosis or to airways obstruction with gas trapping. Pulmonary fibrosis has been reported[13] from the use of gold treatment. In one series half the patients with pulmonary fibrosis and rheumatoid arthritis had abnormal alpha$_1$-antitrypsin phenotypes associated with reduced serum tryptic inhibitory capacity.[14]

Caplan[3] first described small multiple peripheral lung nodules in coal workers with pneumoconiosis and rheumatoid