

## Febrile convulsions

## Further reassuring news about prognosis

Among children in the United Kingdom 2.7% have at least one febrile convulsion, meaning a fit associated with fever in infancy or childhood and without evidence of intracranial infection or defined cause (other than infection outside the central nervous system). Just over one third will have at least one further febrile convulsion, but the majority stop having seizures of any kind, and only 2.4% of those who were previously normal develop epilepsy (defined as two or more seizures without fever). In the few children who develop epilepsy there is little evidence that this has been caused by the febrile convulsions. These are the reassuring findings reported by Verity and Golding on p 1373.1 They are based on the cohort of nearly 15 000 children in the British birth survey, who were born in one week in April 1970. Two other large cohort studies in the United States reached remarkably similar conclusions.2-4 The new study, based on an unselected nationwide cohort of children, removes any doubts that the American studies are applicable to British children.

Febrile convulsions were traditionally regarded as benign, so the news from this study may appear welcome but unsurprising. Its importance lies in the context of the changing views and controversies which have surrounded this common disorder in the past 20 years. In the 1970s concern arose that febrile convulsions were less benign than had been thought. Prolonged febrile convulsions might cause brain damage, particularly in the temporal lobes, leading much later to temporal lobe epilepsy (now more usually called complex partial epilepsy) as well as to other neurological problems. Patients with temporal lobe epilepsy frequently had a history of a prolonged febrile convulsion in early childhood<sup>5</sup>; and in those undergoing temporal lobectomy for intractable temporal lobe epilepsy the commonest pathological lesion was mesial temporal sclerosis, again frequently associated with a history of a prolonged febrile convulsion in early childhood.6 A view developed that it was important to prevent febrile convulsions, particularly those which might become prolonged, and that regular anticonvulsant treatment should be given, at least to selected groups of children who had had a febrile convulsion.7

Three factors ended the phase of enthusiasm for anticonvulsant prophylaxis, about which some paediatricians had always remained sceptical. Firstly, the United States collaborative perinatal project, a large cohort study, showed that the majority of children did well following febrile convulsions, with only a slightly increased risk of later epilepsy compared with children who had not had them.<sup>23</sup> Secondly, regular

anticonvulsant prophylaxis had questionable benefits. The two most promising drugs, phenobarbitone and sodium valproate, both have appreciable unwanted effects, and their efficacy in preventing recurrences is uncertain when trials are analysed, as they should be, on an intention to treat basis.<sup>89</sup> Thirdly, even if they did reduce the risk of further febrile convulsions there was no evidence that they reduced the small risk of later epilepsy—a limitation which also applies to rectal diazepam given at the time of fever to prevent convulsions.<sup>10</sup>

Recently, a joint working group of the Royal College of Physicians of London and the British Paediatric Association produced guidelines for managing febrile convulsions. The working group was convinced that long term drug prophylaxis was rarely indicated, and though it may be used in the child with frequently recurring febrile convulsions, there was no evidence that it would prevent later epilepsy. Verity and Golding's paper supports this view.

The outcome is usually good for children who have had a febrile convulsion, but two questions remain. Firstly, how do we now interpret the earlier reports suggesting that febrile convulsions might cause later temporal lobe epilepsy? The cohort studies suggest that this sequence, if it occurs at all, is rare. The earlier studies were retrospective, and though they demonstrated a sequence of events, they could not show how often it happened or that it was necessarily causal. They were also based on children whose febrile convulsions had happened at least 40 years ago, when seizures may have been allowed to continue much longer than would be the case now. Very prolonged seizures, lasting much more than half an hour, may indeed be damaging.<sup>12 13</sup>

Secondly, why are children who have had a febrile convulsion more prone to later epilepsy than those who have not? All the cohort studies show the extra risk to be small, though it may increase to 7% with follow up to the age of 25. They also show that the risk is greater if the child has had a "complex" febrile convulsion (lasting more than 15 minutes, or focal, or repeated in the same illness), if there is a family history of epilepsy, or if the child had a pre-existing neurological abnormality (though many would not regard this as consistent with the label of febrile convulsion). The most probable explanation is that among children who have febrile convulsions there is a small minority with either an inherited predisposition to epilepsy or pre-existing minor cerebral abnormalities, such as focal cortical microdysgenesis,14 which predispose both to complex features in the febrile convulsion and also to later epilepsy. 4 15 In Verity and Golding's study the

complex feature most strongly associated with later epilepsy, particularly with complex partial seizures, was a focal febrile convulsion, which seems most likely to reflect a pre-existing focal abnormality.

Three practical messages emerge. Parents should be reassured about the generally excellent prognosis of febrile convulsions. Prolonged convulsions should still be prevented,15 and to this end parents may give rectal diazepam, though the working party did not resolve whether this should be done as soon as a convulsion begins or only after five minutes.11 Prophylactic anticonvulsants are rarely needed, and there is no evidence that they have any long term benefit in febrile convulsions.

ROGER J ROBINSON

Emeritus Professor of Paediatrics, BM3, London WC1H 9JR

1 Verity CM, Golding J. Risk of epilepsy after febrile convulsions: a national cohort study. BM3

- 2 Nelson KB, Ellenberg JH. Predictors of epilepsy in children who have experienced febrile seizures. N Engl J Med 1976; 295:1029-33.
  Nelson KB, Ellenberg JH. Prognosis in children with febrile seizures. Pediatrics 1978; 61:720-7.
- 4 Annegers JF, Hauser WA, Shirts SB, Kurland LT. Factors prognostic of unprovoked seizures after febrile convulsions. N Engl J Med 1987;316:493-8.
- 5 Ounsted C, Lindsay J, Norman M. Biological factors in temporal lobe epilepsy. London: Spastics
- Society with Heinemann, 1966 6 Falconer MA. Genetic and related aetiological factors in temporal lobe epilepsy; a review. Epilepsia 1971;**12**:13-31.
- 7 Robinson RJ. Febrile convulsions. Guv's Hospital Reports 1973;122:43-51
- Newton RW. Randomized controlled trials of phenobarbitone and valproate in febrile convulsions.

  Arch Dis Child 1988;63:1189-91.
- 9 McKinlay I, Newton R. Intention to treat febrile convulsion with rectal diazepam, valproate or phenobarbitone. Dev Med Child Neurol 1989;31:617-25.
- 10 Knudsen FU. Intermittent diazepam prophylaxis in febrile convulsions: pros and cons. Acta Neurol Scand 1991;135(suppl):1-24
- 11 Joint Working Group of the Research Unit of the Royal College of Physicians of London and the British Paediatric Association. Guidelines for the management of convulsions with fever. BM7
- 12 Meldrum BS. Secondary pathology of febrile and experimental convulsions. In: Brazier MAB,
- Coceani F, eds. Brain dysfunction in infantile febrile convulsions. New York: Raven, 1976:213-22.

  13 Vannucci RC. Metabolic and pathological consequences of experimental febrile seizures and status
- epilepticus. In: Nelson KB, Ellenberg JH, eds. Febrile seizures. New York: Raven, 1981:43-57.

  14 Hardiman O, Burke T, Phillips J, Murphy S, O'Moore B, Staunton H, et al. Microdysgenesis in resected temporal neocortex: incidence and clinical significance in focal epilepsy. Neurology 1988;38:1041-7.
- 15 Wallace SJ. The child with febrile seizures. London: John Wright, 1988:81,126.

## Rescuers' psychological responses to disasters

## Rescuers need support as well as victims

Many people other than the primary victims may be affected psychologically after a major disaster. Despite their training, emergency workers may fall victim to stressors created by the work they have to do. The findings of an early study after a rail crash in Sydney have been confirmed many times—that over 70% of rescuers may experience transient symptoms of posttraumatic stress: nightmares, anxiety, and flashbacks.2 In most cases these settle, and indeed in 35% of the workers in Sydney more positive feelings about the value of life emerged. Nevertheless, in some the distress does not settle and severe morbidity develops. Valuable research has been done to clarify the impact of disasters on rescue workers and suggest ways of preventing long term morbidity.

During rescue operations workers may confront scenes that bring physical revulsion, even vomiting; transient physical, emotional, and behavioural reactions are common. Sometimes when confronted with overwhelming trauma or the strangeness of foreign cultures rescuers may feel helpless and retreat from or misinterpret what they find. On the other hand, the excitement of involvement may generate a "high," which may extend to overinvolvement and a sense of omnipotence—the counterdisaster syndrome.

The most sensitive indicators of continuing impairment are cognitive impairment and disturbed interpersonal relationships as well as increased arousal, irritability, and loss of interest or withdrawal. Workers may resort to excessive drinking in an effort to forget or dampen distress or to sleep. Marital and family relationships often suffer, and families, especially spouses, may also need support, particularly if the worker remains locked into his or her experience.

Characteristics of the disaster, the rescue operation, and the rescuer himself may all affect the degree of stress experienced. For example, gruesome tasks, particularly when there are multiple deaths, mutilated bodies, or the deaths of children, are stressful for most workers. Even trained body handlers were stressed by recovering bodies after the Mount Erebus air disaster, with a quarter still showing stress 20 months later.<sup>3</sup> Similarly, after the mass suicide in Jonestown experienced servicemen were distressed by the large numbers of dead, the rotting bodies, and the futile deaths of children.4

Emergency workers are as susceptible as any others to ordinary work related stress arising out of organisational or management issues.5 A massive disaster is likely to aggravate many of these and provide an additional psychological burden.

Conversely, careful organisation and management may be powerful antidotes to stressor effects.6 The police who helped retrieve bodies after the explosion on the Piper Alpha oil rig were provided with detailed induction to their tasks, explaining the importance of what they were doing, their possible reactions, and the need to attend to their own welfare. They worked in pairs with an older, experienced officer in each, their shifts were limited, they were debriefed each day, and informal support was available from a psychiatrist. They showed no long term effects from their stress. Even when stress is experienced at "caseness" levels by disaster workers, as in the fire at Bradford football stadium, brief counselling sessions can facilitate recovery.

From their extensive experience with disasters in the United Kingdom, Hodgkinson and Stewart have identified personal loss or injury, encounters with death, and "mission failure" as the primary stressors for rescue workers. Frustration at lives that cannot be saved, failure of equipment, delays, and overwhelming demands all contribute to psychological distress. Symptoms may reflect this conflict, with guilt, reconstruction anxiety, general irritability, focused resentment, and loss of interest in work.

Personality characteristics also affect rescue workers' vulnerability to stress. Simply being older and more experienced in itself is protective. "Hardiness"—a sense of commitment, challenge, and control—is a protective personality style for many workers.8 Coping styles that emphasise sharing problems, constructive use of humour, and the use of social support also seem to be helpful.8 Conversely, those who are drawn to action but deny their vulnerability will find it difficult to admit to stress or seek help. Fear that workmates will think them inadequate or that their career prospects will be damaged are the commonest reasons for distressed workers not taking advantage of stress counselling. Among volunteer bushfire fighters in Australia neuroticism and past psychiatric