previously healthy person. Similarly, the girl in case 2 did not have status epilepticus. In this and the other cases it is difficult to prove that diazepam was the sole cause of respiratory failure, but as she was regaining consciousness after the fit and seemed to be breathing adequately before diazepam was given we believe that the drug was a major contributory factor. The man in case 3 was given diazepam when there was no evidence of seizures.

Staff in neurosurgical units are often asked how to manage fairly simple procedures in agitated or totally uncooperative patients. If the problem must be dealt with urgently (as may be the case with a scalp laceration) the patient can be given either a local anaesthetic and nursing care or a formal general anaesthetic by neuroanaesthetic techniques. Clearly, problems that are not urgent should be left until the patient's condition has improved.

In many cases emergency measures were taken for conditions that could have been dealt with less urgently. Delaying treatment would have ensured a higher standard of treatment and that other work in a busy hospital was not disrupted. For example, one woman (case 5) required prompt treatment for a subdural empyema; it was undesirable to have to provide treatment urgently as diazepam had put her on the brink of respiratory failure. A similar situation occurred in case 4. The child required urgent intubation and ventilation but a crisis was created out of urgency when respiratory arrest occurred; a cardiorespiratory arrest team had to be called and had to abandon the work it was doing at that time.

In several patients blood gas estimations showed respiratory failure and high carbon dioxide pressure. This would result in a rise in intracranial pressure and account for the fixed dilated pupils in one patient (case 10) and possibly the need for ventilation in another (case 8). Respiratory problems occurred in all of our cases, and six patients required ventilation.

Although many factors may be blamed for the deterioration of the patients' condition, in all cases diazepam was given inappropriately and was likely to be harmful.

In such cases we control seizures with phenytoin; the success rate is equal to that obtained with diazepam, control is long lasting, and respiratory depression or depression of the level of consciousness is not a problem.[5] Phenytoin is thought to act too slowly to be useful in controlling seizures. If, however, an adequate dose is given (15-20 mg/kg at a rate not exceeding 1 mg/kg/min) seizures are controlled within 20 minutes in half of cases of status epilepticus due to an acute brain lesion.[5] Peak phenytoin concentrations are achieved at the end of the infusion; or, well before brain damage from status epilepticus might be expected to occur.[2,6] Care must be taken with the infusion as the vehicle is an irritant and the treatment has cardiological side effects. Severe acute toxic effects, however, are rare and noticed only when a loading dose is given to a patient whose serum concentration exceeds 20 μg/l (above normal limits).[5] The risk of giving a loading dose to a patient known to be receiving phenytoin is therefore less than the risk of continued status epilepticus. In addition, phenytoin is recommended to be given at the same time as diazepam to prevent recurrent seizures when the diazepam wears off. Leppik *et al* have shown no difference between giving diazepam and phenytoin and giving phenytoin alone.[5]

In conclusion, we believe that patients with head injuries or other acute neurological lesions who have status epilepticus or recurrent seizures can be managed effectively with phenytoin. Compared with benzodiazepines the risk of respiratory failure or loss of consciousness is reduced, and control of seizures is long lasting.

1 Browne TR. Status epilepticus. In: Browne TR, Feldman RG, eds. *Epilepsy: diagnosis and management*. Boston: Little, Brown, 1983:341-54.
2 Ramsay R, Hammond BJ, Perchalsk RJ, Wilder BJ. Brain uptake of phenytoin, phenobarbital and diazepam. *Arch Neurol* 1979;**36**:535-9.
3 Bell GD, Reeve PA, Moshiri M, *et al*. Intravenous midazolam: a study of the degree of oxygen desaturation occurring during upper gastrointestinal endoscopy. *Br J Clin Pharmacol* 1987;**23**:703-8.
4 Eldridge PR, Hope DT, Yeoman P, *et al*. Influence of hypoxia on somatosensory evoked potentials in intracranial hypertension [Abstract]. *J Neurol Neurosurg Psychiatry* (in press).
5 Leppik IE, Patrick BK, Cranford RE. Treatment of acute seizures and status epilepticus with intravenous phenytoin. *Adv Neurol* 1983;**34**:447-51.
6 Wilder BJ. Efficacy of phenytoin in treatment of status epilepticus. *Adv Neurol* 1983;**34**:441-6.

(Accepted 8 December 1989)

Scientifically Speaking

When going to work makes you ill

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Br Med J 1990;**300**:1190-1

"If earnest researchers go around with clip-boards, positively *asking* about these things, most people will say that they are a little under the weather at the moment, with a bit of a sore throat, a slight headache and a certain amount of tiredness," the late, much missed Henry Miller once said. "They will," he added, "be quite enthusiastic about revealing these medical facts to anyone who will listen—particularly if they are experts, and especially if they carefully write down what they are told."

Henry was talking about "suburban neurosis," a supposedly specific condition that had recently been identified among young housewives living on featureless housing estates. But his remarks could well apply (at least as a methodological caution for research workers) to several other maladies and syndromes that have erupted into the headlines from time to time.

Miller's dismissal certainly came to my mind when I first began to read claims, in newspapers and the scientific literature, about so called sick building syndrome. I had worked in at least one building that was blamed by some of its occupants for both winter sniffles and summer lethargy. But each of these complaints seemed to be amply explicable on conventional grounds. I was not, therefore, immediately converted even by carefully considered papers such as that published six years ago in the *BMJ* in which Michael Finnegan and colleagues delineated the characteristic features of this modern malady.[1] Since then my scepticism has if anything been reinforced by successive reports attributing sick building syndrome to everything from mould spores in the air ducts to bacteria in the central heating, from radon gas seeping through the walls to invisible rays emanating from the VDU screen, from bad psychodynamics related to inept open plan office design to infrasound

waves caused by high rise buildings swaying in the wind.

Now, however, we may be seeing rather more concrete progress in validating this condition and revealing its true aetiology. The evidence comes in the form of the second report of an outbreak in the United Kingdom in which legionnaires' disease was closely associated with the appearance in employees of symptoms suggestive of sick building syndrome. Written by Mary O'Mahony and colleagues at the Public Health Laboratory Service's Communicable Diseases Surveillance Centre in Colindale, London, it appeared recently in *Epidemiology and Infection*.[2]

The incident first came to light when a 41 year old man employed by one of the county police forces went down with legionnaires' disease. He worked at the police headquarters in the operations room of the communications wing—the only air conditioned wing in a five wing, three storey building constructed just a few years earlier. Thorough investigations among the 273 employees at the headquarters, including retrospective reviews of illnesses occurring up to four months earlier, soon led to the identification of six cases of legionnaires' disease. Four of the victims were members of staff who had worked in or visited the communications wing and two were members of the local community. O'Mahony and her coworkers then conducted a case-control study, which implicated the operations room as the main area associated with the infection.

Blowing in the wind

Samples taken from microbiological screening showed that *Legionella pneumophila* was present in water in the cooling tower at the headquarters and in the sludge in its pond, but not in taps or showers that were examined throughout the building. Smoke tests then confirmed that both the exhaust at the top of the tower and condensate from the base could enter the main air intake that serviced the air conditioning system and thus circulate throughout the communications wing. The two victims in the local community—one regularly walked her dog in the grounds and the other lived only a quarter of a mile away—had probably been infected by exhaust blowing in the wind. No further cases of the disease occurred after the cooling tower, which had not been drained for two years, was thoroughly cleaned and disinfected.

But this was not all. From pilot interviews conducted at the outset the investigators learnt that there was a history of minor complaints, chiefly headaches and eye strain, among staff in the communications wing. The case control study then showed that individuals working in this wing had had more frequent chest infections and influenza-like illnesses or both, and were more likely to have been on sick leave compared with those based in other parts of the building. Dry cough and eye strain were strongly associated with working in the communications wing. Those employed there also experienced more sore throats—over a third of them noticing the soreness immediately on starting work in the morning. Employees reported that all of these symptoms improved markedly when they were away from the police headquarters at weekends and during holidays.

No association with drinking water

Within the communications wing the Public Health Laboratory Services investigators found that there was no association between illnesses and the use of toilets or drinking water facilities. There was also no correlation between possible sick building symptoms such as eye strain and cough and the presence of antibodies to *L pneumophila* in the employees' serum. It seemed unlikely, therefore, that these symptoms were linked directly with the outbreak of legionnaires' disease.

Nevertheless, Mary O'Mahony and her coworkers believe that their findings raise the possibility that microorganisms, proliferating inside an inadequately maintained cooling tower or airconditioning system, can cause the symptoms of sick building syndrome. They cite an earlier episode in which an investigator pinpointed a cooling tower as the source of an infective aerosol of *L pneumophila* and in which individuals sitting near an air vent were more likely to develop soreness of the eyes. This incident was not studied in detail, however, and the findings have not been reported in the scientific literature.

Two such correlations certainly do not establish the reality or aetiology of an inherently vague condition such as sick building syndrome. They do, however, indicate a clear strategy for further investigation—not least by looking more closely at the circumstances surrounding other past and future outbreaks of legionnaires' disease in this country and abroad.

1 Finnegan MJ, Pickering CAC, Burge PS. The sick building syndrome: prevalence studies. *Br Med J* 1984;**289**:1173-5.
2 Mahony M, Lakhani A, Stephens A, Wallace JG, Youngs ER, Harper D. Legionnaires' disease and the sick-building syndrome. *Epidemiology and Infection* 1989;**103**:285-92.