neuroleptic malignant syndrome, and this presentation of carbon monoxide poisoning led us to treat the patient with dantrolene,[1] with good effect. Thermogenesis was probably due to skeletal muscle rigidity, and the relaxant action of dantrolene produced a fall in temperature as a secondary benefit.

Our patient may have recovered spontaneously, but the responses to dantrolene merit notice. Further investigation is needed to see whether dantrolene is effective in treating severe cases of carbon monoxide poisoning. Moreover, its use in other hypermetabolic states could be beneficial.

1 Guze BH, Baxter LR. Neuroleptic malignant syndrome. *N Engl J Med* 1985;313:163-6.
2 Clark CJ, Campbell D, Reid WH. Blood carboxyhaemoglobin and cyanide levels in fire survivors. *Lancet* 1981;i:1332-5.
3 Dolan MC. Carbon monoxide poisoning. *Can Med Assoc J* 1985;133:392-9.
4 Myers RA, Linberg SE, Cowley RA. Carbon monoxide poisoning: the injury and its treatment. *Journal of the American College of Emergency Physicians* 1979;8:479-84.
5 Boutros AR, Hoyt JL. Management of carbon monoxide poisoning in the absence of hyperbaric oxygenation chamber. *Crit Care Med* 1976;4:144.

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Photosensitive epilepsy in children who set fires

The flickering light of television is recognised as epileptogenic. Some children with photosensitive epilepsy find viewing television compulsively attractive[1] and even induce seizures by viewing it (C D Binnie, Compliance in epilepsy, Salzburg, 1987). Flames may be a self induced stimulus in those children with photosensitive epilepsy who repeatedly set light to things (fire setting).

Case reports

CASE 1

A man aged 31 had his first attack of epilepsy at the age of 7, when he saw "hundreds of tiny silver dots, then blackness." Minutes later his sight returned. He had innumerable similar attacks for eight years. As his vision cleared he was overtaken by anger and violence commonly followed: he punched bystanders, broke windows, and burned sheds and hedgerows. Recognising the sequence, he tried to limit the damage, running off alone whenever he saw the silver dots.

His behaviour antagonised his parents. He was seen in child guidance clinics, put on probation, taken into care, placed in children's homes, and sent to approved schools, but his disruptive behaviour continued, often after viewing television. At age 13 his epilepsy was diagnosed. Electroencephalography without photic stimulation showed diffuse abnormalities, arrhythmia and sharp and slow components that were maximal in the posterior left temporal region. Although prescribed phenytoin, he palmed the tablets. His attacks stopped without treatment at age 15, and electroencephalographs were subsequently normal.

CASE 2

From babyhood the 6 year old daughter of the man in case 1 had thrown paper on to fires and watched it burn. She set fire to her toys, singed her rabbit's fur, posted lighted matches through the letterbox, and fused an electric circuit with burning papers. She watched television from a distance of 50 cm with maximum colour and contrast. Her mother noticed repeated short episodes, while she was viewing television when she suddenly stared at the wall, white faced and withdrawn. Tears or pranks followed; once she set fire to the sofa. Epilepsy was suspected, and while awaiting neurological investigation she stole a cigarette lighter. Her father, fearing a fire, caned her, and she was admitted to hospital. Compulsory care proceedings were started. On transfer to another hospital an electroencephalogram showed typical photoconvulsant responses during photic stimulation at flash rates above 10 cycles/second, particularly with red and orange light; the recording was otherwise normal. She returned home and complied with instructions to stay three metres from the television screen.[2] Her behaviour was exemplary. Three months later a 24 hour ambulatory electroencephalogram showed that abnormalities occurred immediately when she viewed television from her former position.

After six months of normal behaviour her parents reported renewed moodiness, then discovered caches of spent matches. Meanwhile her teachers, misled by her episodic pallor and abstraction, suspected parental cruelty. Electroencephalography showed sensitivity to fluorescent light, to which she was exposed at school, and to patterns. She was admitted to hospital for drug treatment under electroencephalographic control. Photic spikes lessened with phenytoin, returned with carbamazepine, then disappeared with sodium valproate 200 mg twice daily. She continued to take this drug, and her behaviour was normal during the next six months.

Comment

Fire setting and photogenic epilepsy might coexist by chance but were closely linked in these two cases. The aggressive conduct of the patient in case 1 followed his seizures, and he continued to set fires until he outgrew his epilepsy. His daughter (case 2) behaved normally after avoiding the photic stimulus of close viewing of the television. She began to set fires again in response to photic stimulation by fluorescent lights. Both her fire setting and electroencephalographic abnormalities resolved on treatment with sodium valproate.

Fire setting inevitably causes family disturbances, and blaming the child or the family may lead to inappropriate attempts at containment instead of treatment. Sensitivity to television as a cause of epilepsy is easily missed because the family watches the screen, not each other. Flames lit during preictal compulsion or postictal confusion may kindle further epilepsy.[3] Those who set fires, particularly younger, solitary children,[4] should undergo electroencephalography.

We thank Professor V Dubowitz for allowing publication of case 2; Oxford Medical Systems for the loan of an ambulatory electroencephalographic recorder; and Elizabeth Wallace of the British Epilepsy Association for helpful advice.

1 Jeavons PM, Harding GFA. *Photosensitive epilepsy*. London: William Heinemann, 1975.
2 Anonymous. Television-induced epilepsy and its prevention. [Editorial.] *Br Med J* 1978;i:1301-2.
3 Reynolds EH. Biological factors in psychological disorders associated with epilepsy. In: Reynolds EH, Trimble MR, eds. *Epilepsy and psychiatry*. Edinburgh: Churchill Livingstone, 1981:264-90.
4 Jacobson RR. Child fire setters: a clinical investigation. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 1985;26:759-68.

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An offer of rheumatology training: failure to influence clinic referrals

It has been suggested that teaching of small groups of general practitioners by consultants in the general practitioners' surgeries may be a more useful approach than lectures to large audiences in hospitals.[1] Such teaching can influence the behaviour of general practitioners volunteering for educational sessions,[2] but education does not necessarily decrease the demand for specialist opinions.[3] This study reports the responses of general practitioners referring patients with soft tissue lesions of the shoulder and elbow when they were offered individual clinical tutorials by a consultant.

Patients, methods, and results

From 1 August 1984 until 31 July 1987 every general practitioner who referred a patient with an uncomplicated soft tissue lesion of the shoulder or elbow to a rheumatology clinic was sent an offer of individual teaching by a consultant with the relevant patient's clinic letter. This offer suggested that should the general practitioner have any further patients with shoulder or elbow lesions he or she should contact the consultant, who would come to the surgery and, with the general practitioner, examine, diagnose, and, where appropriate, inject the patient's soft tissue lesion. Further letters were sent to the general practitioners after each subsequent referral. These educational offers had no implications with respect to section 63 or domiciliary visit payment. In all 120 offers were made (19 in relation to elbow and 101 in relation to shoulder lesions) to a total of 41 principals and four trainees. Twenty two doctors received two or more offers, and six sessions were arranged, with nine general practitioners receiving teaching.

A lunchtime meeting entitled "shoulder pain—the GP's role," accounting for half of a section 63 session, was organised two and a half years after the start of the study at this hospital, and all general practitioners in Leeds were invited to attend. The potential effects of the educational exercise on clinic load were examined by observing how many referrals in the last six months of the study might have been avoided if general practitioners who had received two or more educational offers had responded to them and managed the patient themselves.

In a follow up period of 5-33 months only one of the general practitioners who had received teaching continued to refer patients with shoulder or elbow lesions to the clinic. The percentages of all clinic referrals relating to simple shoulder or elbow lesions were 6·5%, 7·9%, and 8·6% in the first, second, and third years of the study, respectively (32, 41, and 47 patients). The waiting time for new clinic appointments rose from two to six weeks during the study. Of the 24 general practitioners who attended the lecture, 17 had never referred patients with shoulder or elbow lesions to the clinic and had thus never been offered individual teaching, five had made one referral, and two had made four referrals. None of the three general practitioners who referred the most patients (9, 10, and 15) attended the lecture or requested educational sessions. These three general practitioners had qualified in the 1960s and 1970s. All except one of those who did arrange sessions had qualified in the 1970s and 1980s. In the last six months of the study 37 patients were referred; 27 of these referrals were from general practitioners who had been offered two or more educational sessions.

Comment

This study supports the view that there are different types of general practitioner, one of which refers a large proportion of patients and does not attend educational sessions or respond to educational offers. Some studies ignore this group.[2,3] It is recognised that as treatment for soft tissue lesions is not ideal referral to a consultant clinic, if only for a second opinion, may on occasions be necessary.[4] Reduction in referral by trained general practitioners in this study was outweighed by increased referral from elsewhere.

It seems likely that there is a lack of consensus among specialists, general practitioners,[5] and patients about the role of the general practitioner in the treatment of minor musculoskeletal disease.

I thank Mrs Susan Briggs for secretarial help.

1 Badley EM, Lee J. The consultant's role in continuing medical education of general practitioners: the case of rheumatology. *Br Med J* 1987;**294**:100-3.
2 Grahame R, Gibson T, Dale E, *et al*. An evaluated programme of rheumatology training for general practitioners. *Br J Rheumatol* 1986;**25**:7-12.
3 Ross AK, Lawton WA. Evaluation of a course for general practitioners on muscles and joints. *Br Med J* 1984;**294**:100-3.
4 Bulgen DY, Binder AI, Hazleman BL, Dutton J, Roberts S. Frozen shoulder: a prospective clinical study with an evaluation of three treatment regimens. *Ann Rheum Dis* 1984;**43**:353-60.
5 Whitfield MJ. What do consultants think of general practice? *J R Coll Gen Pract* 1980;**30**:228-9.

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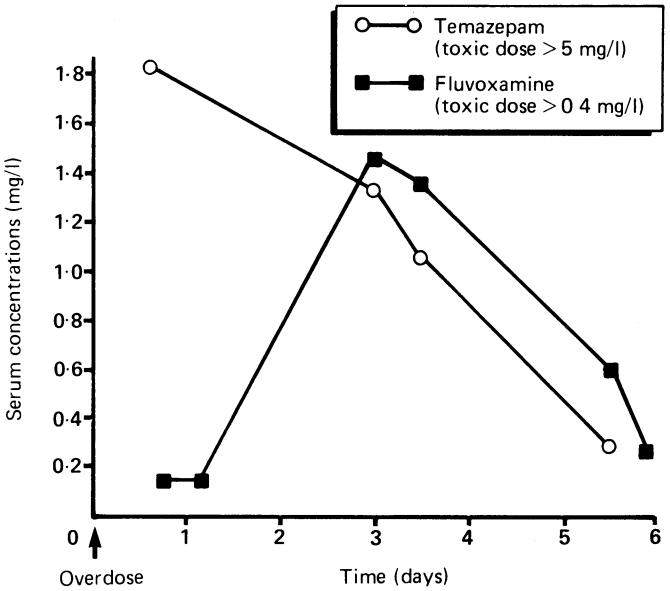
Recovery from prolonged cerebral depression after fluvoxamine overdose

Fluvoxamine maleate [5-methoxy-4-(trifluoromethyl) valerophenone (E)-0-(2 aminoethyl) oxime maleate (1:1)] is a new antidepressant which is thought to act by inhibiting the neuronal uptake of 5-hydroxytryptamine with little or no effect on the catecholamine system.[1] Fluvoxamine has a low incidence of anticholinergic side effects and, unlike tricyclic antidepressants, has no clinically important cardiovascular effects.[2] Minor reported side effects include nausea, vomiting, dizziness, somnolence, dyspepsia, headache, anxiety, palpitations, diarrhoea, and a rash.[3] Fluvoxamine has been implicated in 41 cases of self poisoning, the maximum dose taken being 6·5 g. One patient died, but necropsy showed the fluvoxamine tablets to be intact in the patient's stomach and the death was attributed to overdose of propranolol. I report here a case of fluvoxamine overdose which resulted in coma, the first reported case of prolonged cerebral depression caused by an overdose of fluvoxamine.

Case report

A 74 year old white woman with a two year history of depression treated with 100 mg of fluvoxamine a day for one year was admitted unconscious after attempted self poisoning with 60, 50 mg fluvoxamine tablets (total 3 g) and 25, 10 mg temazepam tablets. A stomach washout was performed in the casualty department. On examination the patient was afebrile and unconscious with a pulse of 70 beats/min, sinus rhythm, blood pressure of 80/50 mm Hg, and a respiratory rate of 20. She was unresponsive to pain, had a sluggish pupillary light reflex, and an absent gag reflex. Tone was normal with normal reflexes and flexor plantar responses. Power could not be assessed. Urea and electrolyte concentrations, liver function tests, blood glucose concentration, routine chest radiograph, and electrocardiogram were all normal. Paracetamol, salicylate, and barbiturates were not detectable in the plasma. Serial fluvoxamine and benzodiazepine concentrations were measured (figure).



Serum concentrations of fluvoxamine and temazepam after overdose.

The patient was treated with supportive measures and her blood pressure returned to 110/70 mm Hg within 24 hours. She remained unconscious for five days with no sign of improvement until day 6, when she opened her eyes spontaneously and began to show signs of having returned to her normal state. On questioning she admitted to having taken the tablets of fluvoxamine and temazepam.

Comment

Fluvoxamine is reported to be a very safe antidepressant with few side effects. Peak plasma concentrations are usually reached after two to eight hours after an oral dose of the drug. The mean plasma half life is 15 hours, and pharmacokinetics of the drug are not altered in the elderly (N Hockings *et al* unpublished).

The initial fluvoxamine concentration (0·16 mg/l) in our patient was less than the normal mean steady state value (0·4 mg/l) that would occur on regular treatment of 100 mg/day, and the delay in reaching a peak in our patient is hard to explain. An initial delay in absorption might be related to posture since serum concentrations of drugs may be reduced when lying down owing to reduced gastric emptying in that position.[4] In addition, fluvoxamine is known to induce nausea and this itself is associated with reduced gastric emptying and may have also contributed to the delayed absorption of the drug.

Our patient recovered from her coma when the fluvoxamine concentration fell below 0·7 g/l. According to the Committee on Safety of Medicines and the manufacturers, previous cases of self poisoning with fluvoxamine have not been associated with prolonged coma, and recently a patient survived an overdose of 6·5 g of fluvoxamine without losing consciousness. The prolonged cerebral depression in our patient might have been due to an interaction between fluvoxamine and temazepam and its metabolites, but I am unaware of any data on this possible interaction.

Although I cannot explain the mechanism for the prolonged cerebral depression, clinicians should be aware of this potential side effect of overdose, especially when fluvoxamine is taken in conjunction with benzodiazepines.

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1 Claassen V, Davies JE, Hertting G, *et al*. Fluvoxamine, a specific 5-hydroxytryptamine uptake inhibitor. *Br J Pharmacol* 1977;**60**:505-16.
2 Roos JC. Cardiac effects of antidepressant drugs. A comparison of the tricyclic antidepressants and fluvoxamine. *Br J Clin Pharmacol* 1983;**15**:435S-95.
3 Mullin JM, Pandita-Gunawardena VR, Whitehead AM. A double-blind comparison of fluvoxamine and dothiepin in the treatment of major affective disorders. *Br J Clin Practice* 1988;**422**:51-6.
4 Warren JB, Cuss F, Barnes PJ. Posture and theophylline kinetics. *Br J Clin Pharmacol* 1985;**19**:707-9.

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