counselling. Fellow workers as well as employers should be well informed and concerned. A protective code of practice was thought desirable; but the Manpower Services Commission produced a good one on disability in general last year, and that is not well enough known.[7] It is highly relevant to epilepsy both on recruitment and on promoting the interests of existing employees. In the spirit of this code the basis for placing a person in a job should be, as someone put it, his ability rather than his disability.

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1 Office of Health Economics. *Epilepsy in society*. London: Office of Health Economics, 1971.
2 Rodin EA, Shapiro HL, Lennox K. Epilepsy and life performance. *Rehabil Lit* 1977;**38**:34-9.
3 Udel MM. The work performance of epileptics in industry. *Arch Environ Health* 1960;**1**:91-8.
4 Kettle M. *Disabled people and their employment*. Banstead, Surrey: Association of Disabled Professionals, 1979.
5 Scambler G, Hopkins A. Social class, epileptic activity, and disadvantage at work. *J Epidemiol Community Health* 1980;**34**:129-33.
6 Raffle PAB. The HGV/PSV driver and loss or impairment of consciousness. In: Godwin-Austen RB, Espir MLE, eds. *Driving and epilepsy—and other causes of impaired consciousness*. London: Royal Society of Medicine, 1982. (International congress and symposium series No 60; proceedings of Labaz symposium.)
7 Manpower Services Commission. *Code of good practice on the employment of disabled people*. Sheffield: Manpower Services Commission, 1984.

Quinine for night cramps

Quinine has a long and colourful history. For centuries cinchona bark, from which quinine was first isolated, was used in treating malaria and other feverish illnesses, and quinine remains the drug of choice for chloroquine resistant falciparum malaria.[1] Because of its bitter taste it is used in tonic water and other drinks, and it is also added to illicit heroin to mask the presence of tasteless adulterants. In the past it also achieved notoriety as an abortifacient. Today the main medical use for quinine in Britain is in treating nocturnal cramp. Little has been published on the complications which occur with regular treatment with quinine at the low doses in which it is used in night cramp. By far the most important problem is the possibility that it might be taken in overdose.

The paper from the Edinburgh regional poisons treatment centre is a salutary reminder of the problems posed by quinine overdose (p 31). The patient may become blind (but will usually recover some vision) and cardiac arrhythmias and deaths may and do occur. A recent report on quinine poisoning from London recorded similar complications, showing that this experience is nation wide.[2] The management of quinine poisoning is controversial. A large and careful study by Bateman and colleagues throws doubt on claims that the drug is effectively removed by haemodialysis, haemoperfusion, or forced diuresis, and the use of these treatments in quinine overdose does not appear to be justified.[3] Recovery of vision shortly after the start of these procedures has been reported,[4-6] but the duration of blindness due to quinine poisoning is often brief, and the recovery might have been coincidental. Certainly it cannot be attributed to the quantity of drug removed.

Stellate ganglion blockade was proposed almost 40 years ago as a treatment for quinine induced blindness and has been widely recommended.[7] The rationale behind this treatment is to reduce the sympathetic supply to the eye and thus produce vasodilatation. As the Edinburgh team points out, however, the blindness seems to be due to a direct effect on the retina, while arteriolar constriction tends to appear later, often as the blindness is recovering. The procedure is clearly frought with complications, and the evidence for its effectiveness is so meagre there no longer seems to be any indication for its use. Many doctors, however, will not find it easy to stand by and watch their patients going blind. In those moments the natural emotional reaction is to use any treatment which has the remotest possibility of success. The advice must be to stand firm, use commonsense measures aimed at removal of quinine from the gut, give activated charcoal, and provide full supportive treatment.

Because of the potential seriousness of quinine poisoning and the paucity of available remedies prevention is worth every effort. Dyson and colleagues suggest that quinine should not be sugar coated and that it should be dispensed in child resistant containers. This should help prevent accidental quinine poisoning in small children—which may have a sudden tragic outcome owing to fatal cardiac arrhythmias.[8] Recent evidence that these containers do indeed reduce the incidence of accidental poisoning[9 10] reinforces the argument that quinine should be dispensed in child resistant containers by legislation rather than by the present voluntary agreement.[11]

Night cramps may be regarded as trivial, but this is not the case for the many who suffer from them. They may not be a threat to life, but they are painful, distressing, and disruptive of sleep and composure. We do not know their cause, and unfortunately this condition cannot be studied in animals. They may be provoked by medication,[12-14] and in this case the remedy is to change the medication rather than falling into the trap of adding quinine to the patient's regimen. They should be distinguished from miner's or stoker's cramp, where sodium loss is the cause and sodium replacement may be curative.[14] There have been few comparative trials of quinine's effectiveness in relieving night cramps—presumably because it is so clearly successful—and its place seems established until a better treatment is found.[15] We do, however, need to understand the nature and causes of night cramps and to find effective remedies which are less toxic than quinine.

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1 Reynolds JEF, ed. *Martindale; the extra pharmacopoeia*. 28th ed. London: Pharmaceutical Press, 1982:394-407.
2 Boland ME, Brennand Roper SM, Henry JA. Complications of quinine poisoning. *Lancet* 1985;i: 384-5.
3 Bateman DN, Blain PG, Woodhouse KW, *et al*. Pharmacokinetics and clinical toxicity of quinine overdosage: lack of efficacy of techniques intended to enhance elimination. *Q J Med* 1985;**54**:125-31.
4 Floyd M, Hill AVL, Ormston BJ, Menzies R, Porter R. Quinine amblyopia treated by haemodialysis. *Clin Nephrol* 1974;**2**:44-6.
5 Sabto J, Pierce RM, West RH, Gurr FW. Haemodialysis, peritoneal dialysis, plasmapheresis and forced diuresis for the treatment of quinine overdose. *Clin Nephrol* 1981;**16**:264-8.
6 Gibbs JL, Trafford A, Sharpstone P. Quinine amblyopia treated by combined haemodialysis and activated resin haemoperfusion. *Lancet* 1985;i:752-3.
7 Redslob E, Warter J, Isch F. Intoxication par la quinine. Traitement de la surdité et de l'amaurose consécutives par des infiltrations stellaires. *Annales d'Oculistique* 1946;**179**:218-20.
8 Garrod GD, Judson JA. Fatal quinine poisoning: a case report. *NZ Med J* 1981;**94**:215-6.
9 Clarke A, Walton WW. Effects of safety packaging on aspirin ingestion by children. *Pediatrics* 1979;**63**:687-93.
10 Jackson RH. Childhood poisoning: perspectives and problems. *Hum Toxicol* 1983;2:285-93.
11 Craft AW. Circumstances surrounding deaths from accidental poisoning 1974-80. *Arch Dis Child* 1983;**58**:544-6.
12 Zelman S. Terbutaline and muscular symptoms. *JAMA* 1978;**230**:930.
13 Keider S, Binenboim C, Palant A. Muscle cramps during treatment with nifedipine. *Br Med J* 1982;**285**:1241-2.
14 Joekes AM. Cramp: a review. *J R Soc Med* 1982;75:546-9.
15 British Medical Association and Pharmaceutical Society of Great Britain. *British national formulary. No 9*. London: British Medical Association, 1985:324.