available in a statistically valid form for doctors. Nevertheless, an examination of the major settings in which heavy drinking began may provide some clues on why doctors have a high risk of becoming alcoholics. In seven cases abnormal drinking began in the wake of well-established drug dependence, which suggests that doctors' increased access to drugs may predispose them not only to drug dependence but also to later alcoholism: the addicted doctor suspected of overprescribing for himself may substitute alcohol for drugs. As any reader of Richard Gordon knows, an ability to hold one's liquor is supposed to be almost mandatory for medical students. That eight of the alcoholic doctors had started their abnormal drinking as undergraduates or housemen suggests that attempting to measure up to this caricature is not without risk. Service in the armed forces can also be a significant factor in the aetiology of alcoholism,¹⁴ but, since doctors had to spend no more time in the services than other men, it is difficult to relate this to their greater liability to alcoholism.

Once heavy drinking patterns are established they may be reinforced by the social and economic milieu in which doctors live and work. A further factor may be the surprising ignorance of the dependent doctors about how much it is safe to drink day in day out. In a recent epidemiological survey Cartwright *et al*¹⁵ found that below a level of 60 cl of absolute alcohol per week there was no relation between consumption and drink-related problems, but above that intake increased consumption led to more and more problems. This level represents an average daily consumption of just over eight single whiskeys or four pints of beer—amounts considerably below those which the dependent doctors in this study had thought it safe to drink. Medical colleagues, too, had rarely showed concern about the amounts these doctors had been consuming. Indeed, colleagues, patients, and, at times, even police appeared to collude with the drinking doctor to prevent his dependence being recognised and treated. This natural reluctance to interfere only too often allowed the dependent doctor to proceed unhindered down the road to professional and social ruin.

The suicide rate of English doctors is 176% of that of the general population,³ while that of all alcoholics admitted to hospital is about 25 times the expected rate.¹⁶ The high risk of suicide among the alcoholic doctors in this study is, therefore, hardly surprising. That so few completely overcame their dependence on alcohol is, none the less, disappointing since their successful completion of medical training indicated that they once had considerable resources of personality and intellect.

There were no reliable predictors of outcome, and the two consultants who specialised in alcoholism were no more successful than their colleagues in making a prognosis. They did, however, take a more realistic view of their doctor-patients' competence to practise medicine, and one regularly recommended that his patients should be sober for six to 12 months before returning to medical practice. The Merrison Committee¹⁷ has concluded that the present constraints that prevent mentally sick doctors from practising are inadequate. Several incidents when alcoholic doctors returned to practice against medical advice with disastrous consequences highlighted the inadequacy. In particular, the ease with which drinking doctors could take on and then lose a succession of general practice locums was disquieting.

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References

- Shaw, G B, *The Doctor's Dilemma*, preface. London, Bodley Head, 1931.
- Murray, R M, *Lancet*, 1976, **2**, 729.
- Registrar-General's Decennial Supplement 1961. *Occupational Mortality Tables*. London, HMSO, 1971.
- American Medical Association, Council on Mental Health, *Journal of the American Medical Association*, 1973, **223**, 684.
- General Medical Council, *Annual Report*. London, GMC, 1974.
- Edwards, G, *Lancet*, 1975, **2**, 1297.
- Murray, R M, *British Journal of Psychiatry*, in press.
- Dight, S E, *Scottish Drinking Habits*. Office of Population Censuses and Surveys. London, HMSO, 1976.
- Edwards, G, Kyle, E, Nicholls, P, *Quarterly Journal of Studies on Alcohol*, 1974, **35**, 499.
- Vaillant, G E, Brighton, J R, and McArthur, C, *New England Journal of Medicine*, 1970, **282**, 365.
- Modlin, H C, and Montes, A, *American Journal of Psychiatry*, 1964, **121**, 358.
- Murray, R M, *Journal of Alcoholism*, 1975, **10**, 23.
- Plant, M, *British Journal of Addiction*, in press.
- Carney, M W P, and Lawes, T G G, *Quarterly Journal of Studies on Alcohol*, 1957, **28**, 59.
- Cartwright, A K J, Shaw, S J, and Spratley, T A, *Designing a Comprehensive Community Response to Problems of Alcohol Abuse*. London, Maudsley Hospital, 1975.
- Nicholls, P, Edwards, G, and Kyle, E, *Quarterly Journal of Studies on Alcohol*, 1974, **35**, 841.
- Committee of Inquiry into the Regulation of the Medical Profession, *Report*. London, HMSO, 1975. (Merrison Report.)

SHORT REPORTS

Herbal cigarettes for kicks

The anticholinergic effects of *Datura stramonium* have been recognised and utilised for centuries. The dried leaves, in powder or tobacco form, are still the basis of some traditional asthma remedies. Several instances of poisoning from the misuse of such preparations have been reported in the USA,¹⁻³ but only one has been recorded in Britain.⁴ We report here two further cases.

Case reports

Case 1—A 25-year-old art student was admitted to hospital after collapsing on a bus. At the time no other information was available. He was uncommunicative and made jerky, uncoordinated movements. The skin and mucous membranes were warm and dry, the pulse was 110/min, and the bladder was distended. The pupils were widely dilated and unresponsive to light, and there was rotary nystagmus. All tendon reflexes were exaggerated, and the plantar responses were extensor. The clinical features were consistent with poisoning by an atropine-like compound. During recovery he experi-

enced visual hallucinations and was sedated with diazepam, but he was rational within 12 hours of admission. He then admitted eating one and a half Surama herbal cigarettes.

Case 2—Ten days after case 1 a 16-year-old girl was admitted with a six-hour history of bizarre behaviour. She was seen by the casualty officer, a neurosurgeon, and a psychiatrist before the possibility of poisoning was considered on the clinical presentation of an acute psychosis with disorientation. She was agitated, talking incoherently, and plucking at the bedclothes. The skin and mucous membranes were dry, the pulse was 110/min, and the bladder was distended. The pupils were dilated and reacted sluggishly to light, rotatory nystagmus was present, and the tendon reflexes were hyperactive with flexor plantar responses. Her agitation was controlled with diazepam, and within 24 hours of admission she was rational and admitted eating two Surama cigarettes. She described vivid and frightening hallucinations of dead babies and of a satanic black mass. Two friends also ate some of the cigarettes but were not referred to hospital.

Comment

D stramonium, known as the Jimson-weed or thorn-apple, is distributed worldwide. The leaves and seeds contain hyoscyamine,

atropine, and hyoscine and have been ingested, especially in the USA, for their hallucinogenic properties. Deaths have resulted.⁵

Our patients ate Surama cigarettes, which contain 92.5% stramonium leaf (equivalent to 1.5-2.5 mg alkaloids per cigarette). They displayed many features typical of anticholinergic poisoning but recovered without specific treatment. In severe poisoning physostigmine salicylate may be of value.

The occurrence of two or possibly four cases in so short a time suggests that the potentially lethal practice of ingesting such cigarettes for kicks may be enjoying a vogue.

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¹ Dean, E S, *Journal of the American Medical Association*, 1963, **185**, 882.

² Goldsmith, S R, Frank, I, and Underleider, J T, *Journal of the American Medical Association*, 1968, **204**, 169.

³ Gowdy, J M, *Journal of the American Medical Association*, 1972, **221**, 585.

⁴ Harrison, E A, and Morgan, D H, *British Medical Journal*, 1976, **2**, 1195.

⁵ Mikolich, J R, Paulson, G W, and Cross, C J, *Annals of Internal Medicine*, 1975, **83**, 321.

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Postural proteinuria and lipid nephrosis of 58 years' duration

Lipid nephrosis of childhood usually undergoes remission at least during adult life and is not associated with accentuation of proteinuria with the upright position.¹ Postural proteinuria, on the other hand, is defined with normal quantitative protein on recumbency. The following case is therefore interesting.

Case report

In August 1968 a 60-year-old woman was admitted to the Thomas Jefferson University Hospital. Her history had begun in 1919 when, aged 11, she presented with intermittent swelling of the legs and face that was diagnosed as "nephritis." She was next seen in 1941, again because of swollen legs, and was told she had severe proteinuria. Oedema was nearly always present. For as long as she could remember she had had nocturia; she had to rise two to three times nightly for copious voiding. In 1965 she began taking diuretics.

Examination in 1968 showed pedaloedema, blood pressure 146/90 mm Hg, urine analysis 3+ - 4+ protein with daily excretion varying from 1.5 to 3 g depending on her position, and no urinary sediment. Serum urea was 1.6 mmol/l (10 mg/100 ml), serum creatinine 44.2 μ mol/l (0.5 mg/100 ml) (repeated) with a creatinine clearance rate of 110 ml/min, and two-hour postprandial blood sugar 74 mg/100 ml. Plasma albumin:globulin ratio was 21:27 g/l, cholesterol 4.7 mmol/l (183 mg/100 ml). Chest radiograph and ECG showed normal findings. (On admission at other hospitals serum albumin concentrations below 21 g/l had sometimes been recorded.)

She was readmitted in August 1969. She had mild oedema of the legs and there was no venous insufficiency. Blood chemistry showed serum urea 2.5 mmol/l (15 mg/100 ml); creatinine 53 μ mol/l (0.6 mg/100 ml); phosphate 1.42 mmol/l (4.4 mg/100 ml); Ca 4.4 mmol (mEq)/l, CO₂ 28, K 4.5, Na 138, and Cl 100 mmol (mEq)/l. Protein electrophoresis showed albumin 3.0, globulins: α_1 0.3, α_2 0.9, β 0.6, and γ 0.4. Twenty-four hour urinary protein was about 2 g. Plasma immunoglobulins were: IgG 6.9 g/l (690 mg/100 ml),

IgA 1.8 g/l (180 mg/100 ml), IgM 1.92 g/l (192 mg/100 ml). Blood volume was 2960 ml (normal for patient's height and weight). X-ray films of stomach and small bowel and jejunal biopsy were all normal. Labelled chromium study did not show any protein wasting in the bowel. An excretory urogram did not show ptosis of the kidney in an upright position.

Special studies on the effect of posture on protein and NaCl excretion showed reversal of normal diurnal cycle of NaCl excretion, which was restored to normal by 24-hour recumbency. Protein excretion averaged 1.5 g (7 am-7 pm) while she was up and about and 0.5 g (7 pm-7 am) while she was recumbent. The same pattern (but not degree) occurred with 24-hour recumbency. The renal biopsy result is shown in the figure. Light microscopy showed nearly normal glomeruli with occasional minimal focal changes. Electron microscopy (only one compressed glomerulus could be found in the sample) showed some fusion of foot processes but no thickening of basement membrane nor deposits.

Follow-up to 1976 showed that she was in excellent health with oedema well controlled on diuretics, a high-protein diet, and thermelastic stockings.

High-power view of a representative glomerulus. (PAS stain. $\times$ 310.)

Comment

This case of proteinuria is interesting because of the condition's unusually long course (58 years) with normal renal function and histology. The long duration and severe postural accentuation of proteinuria is unusual for lipid nephrosis,¹ yet it is equally unusual for postural proteinuria because of considerable losses during recumbency.

This reopens the controversy as to the lesion in lipid nephrosis, and whether "pure" lipid nephrosis is not a group of disorders. Rich² suggested many years ago that the lesion of focal glomerular sclerosis may first become manifest in the juxtamedullary glomeruli and thus could easily be missed with a conventional renal biopsy. Furthermore, some recorded cases of lipid nephrosis with progressive renal insufficiency are really examples of focal glomerular sclerosis² or very early stages of membranous³ or proliferative glomerular nephritis. The precise diagnosis in this case is still not clear.

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¹ Saito, H, *Tohoku Journal of Experimental Medicine*, 1968, **95**, 207.

² Rich, A R, *Johns Hopkins Hospital Bulletin*, 1957, **100**, 173.

³ White, R H R, Glasgow, E F, and Mills, R J, *Lancet*, 1970, **1**, 1353.

⁴ Churg, J, et al, *New England Journal of Medicine*, 1965, **272**, 165.

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