who failed to transmit infection had long relationships in many cases, and their contraceptive or other "safe" practices were no different from those of the couples in whom both partners were infected.

This apparent non-infectivity may be an illusion as further testing of seronegative partners may show that they have become infected. It suggests, however, that many subjects who are positive for HIV antibody are, at least for a while, unable or unlikely to transmit their infection sexually. The transient nature of HIV antigenaemia in the early stages after an initial infection from contaminated blood products supports this hypothesis,¹⁶ but evidence suggests that infectivity recurs during the development of frank AIDS related complex or AIDS (J J Goedert *et al*, third international AIDS conference, Washington, 1987). Other factors must also play a part because transmission did not occur in 79% of the partnerships of more than 18 months' duration, and these relationships must have started shortly after the index partner became infected. The effect of enforced sexual abstinence during imprisonment for crimes related to drugs may be a factor.

The accuracy of the information given by the subjects may be questionable. Thus, although knowledge of a partner's HIV state seemed to be correct and accurately reported, 40% of couples disagreed about their sexual practices. This shows that they either did not understand the question or were too shy to admit such intimate details. In this group drug abuse carries no stigma and is freely discussed, but sexual practices are not. Notably, none of these couples attended a genitourinary medicine clinic for advice on HIV.

The evidence of heterosexual transmission of HIV in this country is strengthened by our findings, showing an infection rate of about 15% in the partners of seropositive drug addicts compared with 6.8%⁶ and 9.5%¹⁷ in the wives of infected haemophiliacs. Abusers of intravenous drugs in Edinburgh have travelled widely in Britain and shared needles with other drug abusers around the country.¹⁴ Thus the drug abusing community, both addicts and occasional users, presents a serious risk of infection to the heterosexual population. The lack of use of contraceptives was alarming, and emphasised by the large number of pregnancies in one of our study groups. Pregnancy did not, however, seem to increase the mother's risk of infection with HIV.

Although infection with HIV was transmitted heterosexually in only a small proportion of our subjects, the next phase of the local (Edinburgh) epidemic may result from transmission by this route as inadequate precautions are being taken and infected partners will probably become ill with AIDS in the near future. An epidemic of

sexually transmitted HIV infection may be expected to follow this development, and the problems of heterosexual and paediatric AIDS, more familiar to New York, may become commonplace.¹⁸

Many difficulties arise in studying heterosexual behaviour in young people, and the collection of data can never be complete. The circumstances of this study, particularly the availability of stored blood samples, allowed us to make retrospective observations on relationships and the duration of unprotected exposure to infection that may be less easy to obtain in future.

We thank the doctors and staff of the West Granton medical group and the City Hospital's screening clinic for helping to collect the data; Dr Martin Bland of St George's Hospital Medical School for providing the computer program for statistical analysis; Dr J R Peutherer of Edinburgh University's department of clinical bacteriology for the Western blot analyses; and the Scottish Home and Health Department, which funds the Edinburgh drug addiction study.

References

- 1 Biggar RJ. The AIDS problem in Africa. *Lancet* 1986;ii:79-83.
- 2 Mann JM, Bila K, Colebunders RL, *et al*. Natural history of human immunodeficiency virus infection in Zaire. *Lancet* 1986;ii:707-9.
- 3 Mann JM, Francis H, Quinn T, *et al*. Surveillance for acquired immunodeficiency syndrome in a central African city: Kinshasa, Zaire. *JAMA* 1986;255:3255-9.
- 4 Anonymous. *AIDS in the Third World*. London: Panos Institute, 1986.
- 5 Centers for Disease Control. Heterosexual transmission of human T-lymphocytic virus type III/lymphadenopathy-associated virus. *MMWR* 1985;34:561-3.
- 6 Redfield RR, Markham PD, Salahuddin SZ, *et al*. Frequent transmission of HTLV-III among spouses of patients with AIDS-related complex and AIDS. *JAMA* 1985;253:1571-3.
- 7 Allain JP. Prevalence of HTLV-III/LAV antibodies in patients with haemophilia and their sexual partners in France. *N Engl J Med* 1986;315:517-8.
- 8 Jones P, Hamilton PJ, Bird G, *et al*. AIDS and haemophilia: morbidity and mortality in a well defined population. *Br Med J* 1985;291:695-9.
- 9 Fischl MA, Dickson DM, Scott GB, *et al*. Evaluation of heterosexual partners, children and household contacts of adults with AIDS. *JAMA* 1987;257:640-4.
- 10 May RF, Anderson RM. Transmission dynamics of HIV infection. *Nature* 1987;326:137-42.
- 11 Piot P, Kreiss JK, Ndinya-Achola JO, *et al*. Transmission of HIV. *AIDS* 1987;1:199-206.
- 12 Robertson JR, Bucknall ABV, Welsby PD, *et al*. Epidemic of AIDS related virus (HTLV-3/LAV) infection among intravenous drug abusers. *Br Med J* 1986;292:527-9.
- 13 Brettell RP, Davidson J, Davidson SJ, *et al*. HTLV-3 antibodies in an Edinburgh clinic. *Lancet* 1986;ii:1099.
- 14 Brettell RP, Bisset B, Burns S, *et al*. Human immunodeficiency virus and drug misuse: the Edinburgh experience. *Br Med J* 1987;295:421-4.
- 15 Department of Health and Social Security. *Don't die of ignorance*. London: HMSO, 1987.
- 16 Allain J-P, Laurian Y, Paul DA, Senn D. Serological markers in early stages of human immunodeficiency virus infection in haemophiliacs. *Lancet* 1986;ii:1233-6.
- 17 Kreiss JUK, Kitchen LW, Prince HE, Kaspar CK, Essex M. Antibody to human T-lymphotropic virus type 3 in wives of haemophiliacs. *Ann Intern Med* 1985;102:623-6.
- 18 Weinberg DS, Murray HW. Coping with AIDS. The special problems of New York City. *N Engl J Med* 1987;317:1469-73.

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SHORT REPORTS

Benign multinodular goitre and reversible Horner's syndrome

Horner's syndrome due to thyroid disease has been reported before, and the possibility that benign conditions might be responsible has been suggested but not proved conclusively.^{1,2,3} We present a case of Horner's syndrome in which histological proof of a benign thyroid cause was obtained and in which the symptoms resolved after surgery.

Case report

A 63 year old man presented with a six week history of right sided ptosis and a gritty feeling in the right eye. He had a smooth non-tender goitre that had been present but untreated for many years. On examination his trachea was found to deviate to the left. The percussion note was dull over the right apex anteriorly, and breath sounds were diminished in this region. He had a complete right Horner's syndrome. There was a partial ptosis, with half of the pupil covered on forward gaze. The diameter of the right pupil was about half that of the left when viewed in normal room lighting. There was anhydrosis of the right side of his face. The results of neurological examination were otherwise normal. A chest x ray film

showed an opacity in the right upper zone, and Pancoast's tumour was provisionally diagnosed. A computed tomograph of the neck and upper thorax showed that the mass extended from the hyoid bone almost to the carina and that it arose from the thyroid. The contrast enhanced films showed the right carotid sheath being compressed by the mass (figure), thus causing Horner's syndrome. Tests of thyroid function all gave normal results.

A right thyroid lobectomy was performed through a standard collar incision. The thyroid gland was very tense within its capsule, compressing the surrounding structures at the level of the thoracic inlet. Histological examination confirmed that it was a benign multinodular goitre. The acini were distended with colloid, and the epithelium was flattened. There was evidence of haemorrhage in places. Six weeks after surgery there was a considerable improvement in his ptosis, the pupils were of equal size, and the discomfort in the eye had gone.

Comment

Horner's syndrome caused by thyroid masses has been reported before, but there is a tendency to assume that an invasive malignant process is responsible.^{1,2} In a review of 216 cases of Horner's syndrome 12 were reportedly due to thyroid adenoma.² No clinical details were given, so the diagnostic criteria are unknown. The subsequent management and outcome were also unreported. Recently a patient who had bilateral Horner's syndrome due to multinodular goitre has been described.³ In this case there

Computed tomograph of neck and upper thorax.

was no histological evidence that the goitre was benign, though clinically cancer seemed to be unlikely.

To our knowledge our patient is the first reported case of Horner's syndrome where there was histological proof of benign thyroid disease and where the condition resolved after surgery.

We thank Mr H R Matthews of East Birmingham Hospital for permission to report this case.

1 Billie JD, Wetzel WJ, Suen JY. Thyroid lymphoma with adjacent nerve paralysis. *Arch Otolaryngol* 1982;108:517-9.

2 Giles CL, Henderson JW. Horner's syndrome: an analysis of 216 cases. *Am J Ophthalmol* 1958;46:289-97.

3 Levin R, Newman S, Login I. Bilateral Horner's syndrome secondary to multinodular goitre. *Ann Intern Med* 1986;105:550-1.

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Nifedipine for epilepsy? A pilot study

Theoretical considerations suggest that blockers of calcium channels may have anticonvulsant activity, and this has been confirmed in seizure models in vitro¹ and in vivo.² Results with the sedative flunarizine in humans have been promising.³ The conventional calcium antagonists verapamil and diltiazem have been found to inhibit the biotransformation of carbamazepine and produce neurotoxicity.^{4,5} In this pilot study we assessed the antiepileptic potential of nifedipine, a dihydropyridine derivative.

Patients, methods, and results

Twelve patients who had intractable epilepsy (four women, eight men; age 20-58 years; duration of epilepsy 2-42 years) agreed to take part in the study, which was approved by the local ethical committee. Ten had complex partial epilepsy with secondary generalisation. One patient experienced only partial seizures and one generalised tonic-clonic fits. The patients were receiving the following treatment: carbamazepine (seven), carbamazepine with sodium valproate or phenobarbitone (two), phenytoin (two), and sodium valproate and phenobarbitone (one). For more than a year all had attended the epilepsy clinic at this hospital, where they routinely completed monthly "seizure frequency" charts.

The patients were admitted to this hospital for two weeks. They continued to take anticonvulsant drugs in unchanged dosages, and the concentrations were monitored. Blood for assaying carbamazepine and sodium valproate concentrations was drawn at 0800 ("trough"), 1100 ("peak"), and 1600 for three consecutive days before treatment with nifedipine was started and on days 1, 2, 3,

5, 7, and 10 of nifedipine treatment. The concentrations of phenytoin and phenobarbitone, whose elimination half lives exceed 24 hours, were obtained once daily. The mid-afternoon measurement of all drugs was repeated in the outpatient department after a further two and four months. The patients were treated with nifedipine 20 mg thrice daily in addition to their existing antiepileptic treatment. Their frequency of seizures was charted for three months before and after nifedipine treatment was started. Generalised tonic-clonic and partial seizures were recorded separately.

Two patients withdrew after six and 10 days because of headache and lightheadedness. Another was non-compliant, and a further patient stopped taking all his drugs after six weeks. Thus nine patients completed one month and eight all three months of the study.

In the nine patients taking carbamazepine the mean trough, peak, and mid-afternoon concentrations did not change significantly from the baseline concentrations in the first 10 days of nifedipine treatment, and there were no long term alterations in the six patients who continued taking nifedipine for four months (Student's *t* test; 95% confidence interval (CI) -12% to +6%). The protein binding capacity in the serum of carbamazepine and circulating concentrations of its active metabolite carbamazepine 10,11 epoxide were also unaffected by nifedipine. The concentrations of the other anticonvulsants were unaltered.

The overall frequency of seizures in patients receiving adjuvant nifedipine was significantly less at one ($p < 0.01$; 95% CI 23% to 36% reduction) and three months ($p < 0.01$; 95% CI 25% to 57% reduction) compared with previous months (Wilcoxon matched pairs test) (figure). Five and four patients had >50% decreases in the number of fits after one and three months, respectively, while seven reported >25% reductions at both time points. Two patients, for the first time in many years, remained free of seizures for the whole three months.

Total number of seizures one and three months before and after starting nifedipine treatment (20 mg thrice daily) in patients who have intractable epilepsy.

Comparisons were repeated on an "intention to treat" basis, and the differences remained significant ($p < 0.02$). It was not possible with such small numbers to separate out usefully differential effects on partial and generalised seizures, though a decrease in the frequency of partial events occurred in all patients after one ($p < 0.02$) and three months ($p < 0.01$) of nifedipine treatment.

Comment

This study was not controlled for the placebo effect of giving a new drug or for that of a short admission to hospital. Nevertheless, the reduction in frequency of seizures supports the hypothesis that nifedipine has an anticonvulsant action. Calcium antagonists may have important potential as adjuvant, non-sedative, antiepileptic drugs.

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1 Walden J, Speckmann E-J, Witte OW. Suppression of focal epileptiform discharges by intraventricular perfusion of a calcium antagonist. *Electroencephalogr Clin Neurophysiol* 1985;61:299-309.

2 Morocutti C, Pierelli F, Savarelli L, Stefano E, Peppe A, Mattioli GL. Antiepileptic effects of a calcium antagonist (nimodipine) on cefazolin-induced epileptogenic foci in rabbits. *Epilepsia* 1986;27:498-503.

3 Overweg J, Binnie CD, Meijer JWA, et al. Double-blind placebo-controlled trial of flunarizine as add-on therapy in epilepsy. *Epilepsia* 1984;25:217-22.