remained. He was discharged from hospital without continuing treatment. Two weeks later he developed severe deep otalgia and an increased blood glucose concentration. Repeat computed tomography showed more extensive temporal bone erosion. Intravenous ceftazidime was given for two weeks followed by oral ciprofloxacin 500 mg twice a day for three months as outpatient treatment. He steadily improved, and the facial palsy resolved almost completely. The vagal and hypoglossal palsies remained, but there was good functional compensation. He subsequently remained well 22 months after completing treatment.

In this case diagnosis of malignant otitis externa was delayed because of failure to culture pseudomonas from the meatus. In our opinion intravenous antibiotic treatment remains the best initial treatment because these patients need inpatient assessment and effective high blood concentrations of antibiotic are best guaranteed by systemic administration. Oral ciprofloxacin should be given as continuation outpatient treatment to prevent relapse. The optimum length of treatment has not yet been defined, but two to three months after the resolution of symptoms and signs seems to be adequate.

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1 Hickey SA, Ford GR, O'Connor AFF, Eykyn SJ, Sönksen PH. Treating malignant otitis with oral ciprofloxacin. *Br Med J* 1989;299:550-1. (26 August.)

Child abuse: case conferences

SIR,—The series on child abuse¹ was excellent; I was, however, disappointed to note the omission of community paediatric medical officers from the list of attendees at case conferences in Ms Jill McMurray's article.¹

In the London Borough of Richmond last year community medical officers attended 132 out of 150 case conferences and general practitioners 19. Doctors in the community paediatric service give general medical advice to the case conference, as well as specific descriptions of children's injuries that they have seen. Our doctors are called to see children in schools and clinics before case conferences and as a monitoring service after conferences, and I feel that their contribution is valuable.

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1 McMurray J. Case conferences. *Br Med J* 1989;299:500-2. (19 August.)

Athletes and drugs

SIR,—Dr Richard Feinmann's recent letter shows the widespread confusion about drug use in sport, within the sporting world and the medical profession.¹

In the example Dr Feinmann gave two of the athletes were "not allowed to take any medication" for hay fever. The Scottish Sports Council's recommendations for hay fever, however, permit inhaled steroids (beclomethasone dipropionate), several antihistamines (terfenadine, chlorpheniramine maleate, etc) and sodium cromoglycate eye drops.² We assume that all the sports bodies in the United Kingdom have similar guidelines.

It is unfortunate that all concerned in sporting events are not aware of the permitted treatments. We suggest that such information be widely distributed to all those in sport. In addition, it should be readily available to doctors so that appropriate treatment can be prescribed to athletes. A suitable place might be at the end of the *British National Formulary* in a separate appendix or in the "cautionary label" warnings.³ The Scottish Sports Council is to be commended for popularising such information by producing for athletes a handy credit card sized guide to banned and permitted drugs.²

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- 1 Feinmann R. Athletes and drugs. *Br Med J* 1989;299:570. (26 August.)
- 2 Scottish Sports Council. *Drug abuse in sport: banned classes and methods with examples*. Edinburgh: Scottish Sports Council, 1988.
- 3 Joint Formulary Committee. *British national formulary*. London: British Medical Association and Pharmaceutical Press, 1989.

Lambasted on heart disease

SIR,—In reporting the verdict of the Committee of Public Accounts on Britain's attempts to reduce coronary mortality Dr Tessa Richards states that in Scotland there is a lack of any campaign tackling the problem of heart disease.¹ This is inaccurate.

In December 1988, amid much publicity, SHARP (Scottish Heart and Arterial disease Risk Prevention) was launched at an international symposium on coronary risk factors. This organisation has been established to tackle Scotland's deplorable record of coronary morbidity and mortality and its main aim is to promote the prevention of cardiovascular diseases in primary care, in hospital practice, and in the community. Several major projects will soon be announced, but much of the resources of time, energy, and skill of the committee has initially had to be diverted to seeking financial support. So far all funding has come from pharmaceutical companies.

Dr Richards points out that £10m is spent on prevention while £500m is spent on treating cardiovascular diseases. If organisations such as SHARP were to be properly funded not only could the latter figure be drastically reduced but also the emotional and economic devastation to individual patients and families affected by cardiovascular diseases could be alleviated.

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1 Richards T. Lambasted on heart disease. *Br Med J* 1989;299:286. (29 July.)

Use of stents for treating obstruction of urinary outflow

SIR,—Recent correspondence has voiced reservation as to the advisability of implanting prostatic endoprostheses (the Wallstent), which cannot be moved or indeed removed without open surgery. As the main use of these stents would seem at present to be confined to unfit patients this criticism poses a potential limitation to the wider use of this technique.^{1,2} In a previous communication³ we reported our initial experience with the Wallstent, but at that time we had not had occasion to test the ease with which the stents could be removed. We have subsequently removed two stents, one being at the time of original placement as we were unsatisfied with the position of

the stent and the other being six weeks after implantation in a patient with advanced adenocarcinoma of the prostate. In both cases the stent was removed by grasping it under endoscopic control with alligator forceps and applying distal traction, thereby elongating the stent and reducing its diameter. In neither case did any urethral trauma result. To our knowledge a further three Wallstents have been removed without difficulty in other centres.

Although it is still too early to comment definitively on the use of this stent technique in managing prostate obstruction (owing to the short follow up time and small number of patients so far treated), we believe that it merits further consideration. Certainly the ability to remove these stents is of fundamental importance, and a potential application is the use of such stents to relieve obstruction due to the prostate on a trial basis in patients in whom dual pathology is suspected. The stent could then either be left in situ or be removed before a definitive transurethral prostatectomy.

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- 1 Parker C, Birch B, Gellister J, Grant DS, Miller R. Use of stents for treating obstruction of urinary flow. *Br Med J* 1989;299:120. (8 July.)
- 2 Buckley JF, Bowler GMR, Littlewood DG, Smith G, Fowler JW, Tolley DA. Treatment of obstruction of urinary outflow. *Br Med J* 1989;299:181. (15 July.)
- 3 Milroy EGJ, Chapple CR, Rickards D. Use of stents for treating obstruction of urinary outflow. *Br Med J* 1989;299:119-20. (8 July.)

Drug Points

Tachycardia precipitated by topical homatropine

DRS DUNCAN REID and JAMES D FULTON (Department of Geriatric Medicine, Stobhill General Hospital, Glasgow G21 3UW) write: Although rarely reaching concentrations that are clinically important, certain topical agents used in ophthalmological practice can be systemically absorbed.¹ For example, timolol eyedrops may rarely cause β adrenoceptor blockade² or interact with drugs such as verapamil to cause profound bradycardia.³ Topical homatropine also has been reported to cause confusion in an elderly man.⁴

An 84 year old woman was admitted to our wards after an episode of dizziness and palpitations culminating in virtual syncope. Her radial pulse rate was initially 160-180 beats/min but settled spontaneously to a rate of 80 beats/min. Physical examination showed partial blindness due to cataract and glaucoma, for which she was receiving topical homatropine eyedrops. She was taking no other medication and her only additional relevant history was of several similar episodes of palpitations and lightheadedness over the preceding four months. These attacks occurred in the morning or evening within an hour of instilling the drops, though she had been taking homatropine for about two years. Routine biochemical, haematological, and thyroid function tests; chest x ray film; and resting and 24 hour electrocardiograms were all normal. Homatropine was discontinued and she remained well when reviewed two and four months after discharge with no further symptoms suggestive of an arrhythmia. Rechallenge with homatropine was considered unethical.

Although possibly coincidental, the administration of homatropine eyedrops, at recommended therapeutic doses, seemed to precipitate tachycardia in this otherwise fit, elderly woman. Neither the Committee on Safety of Medicines nor the

manufacturer has received any reports of this reaction.

- 1 Selvin BL. Systemic effects of topical ophthalmic medications. *South Med J* 1983;76:349-58.
- 2 Hayes LP, Stewart CJ, Kim I, Mohr JA. Timolol side effects and inadvertent overdosing. *J Am Geriatr Soc* 1989;37:261-2.
- 3 Pringle SD, MacEwen CJ. Severe bradycardia due to interaction of timolol eyedrops and verapamil. *Br Med J* 1987;294:155-6.
- 4 Falbe WJ, Boyd DL. Homatropine-associated confusion in an elderly patient. *J Am Geriatr Soc* 1988;36:649.

Depression associated with diltiazem

Ms CECILIA BIRIELL (WHO Collaborating Centre for International Drug Monitoring, Uppsala, Sweden), Dr JOHN McEWEN (Adverse Drug Reactions Advisory Committee, Canberra, Australia), and Dr EMILIO SANZ (Swedish Adverse Drug Reactions Advisory Committee, Uppsala, Sweden) write: The World Health Organisation collaborative programme for international drug monitoring gathers details of suspected adverse drug reactions from 26 countries. We describe eight episodes of diltiazem associated depression identified in the programme's database.

Case 1—In 1985 a 52 year old Canadian man with angina, who was taking timolol 20 mg twice daily and isosorbide dinitrate 30 mg four times daily, started to take diltiazem 60 mg four times daily. After three days he developed "melancholia, weepiness, and general depression." These symptoms persisted for 14 days, when diltiazem was stopped. He then recovered.

Case 2—In 1987 a 56 year old man in the United Kingdom stopped taking nifedipine 10 mg capsules, which he had been taking for about 16 months for angina. On the same day he took 120 mg diltiazem. Six hours later he felt "unreal, depressed, and suicidal." These symptoms wore off. Two days later he again took 60 mg diltiazem and after six hours the symptoms returned. He continued to take diltiazem tablets 60 mg twice daily for three more days and the symptoms persisted. The symptoms resolved two days after he stopped taking diltiazem. He had been taking, and continued to take 200 mg metoprolol tartrate delayed release daily and up to 10 glyceryl trinitrate tablets sublingually daily.

Case 3—In 1987 a 64 year old Australian woman started to take diltiazem 30 mg three times a day for angina. After three days she developed insomnia and depression characterised by early morning waking and morbid thoughts. These symptoms persisted for 41 days, until she stopped taking diltiazem and recovered. She had been taking, and continued to take, one naproxen tablet daily for arthritis. About 10 days later she again started taking diltiazem, 90 mg daily, and the depression returned within two weeks. Recovery followed within a few days of stopping the drug.

Cases 4-9—Details are set out in the table.

Calcium channel blocking drugs are customarily classified in four groups, reflecting their chemical structures. The piperazine group, which includes cinnarizine and flunarizine, has adverse effects

strongly suggestive of a direct action on the central nervous system. Extrapyramidal symptoms and depression are well documented with flunarizine.^{1,2} Members of the other groups—phenylalkylamines (verapamil), dihydropyridines (nifedipine, felodipine, and nicardipine), and benzothiazepines (diltiazem)—have been used to treat neurological disorders including migraine,³ Gilles de la Tourette's syndrome,⁴ tardive dyskinesia and dystonia,⁵ and some psychiatric disorders. Verapamil, in particular, has been used to treat mania.^{6,7} However, evidence of direct adverse effects on the central nervous system is meagre. Single case reports have attributed akathisia⁸ and acute psychosis⁹ to diltiazem and acute psychosis to nifedipine.^{10,11} Recently four cases of substantial depression associated with nifedipine were reported.¹²

Spontaneous reports of suspected adverse reactions submitted to national centres often lack important information. None the less, we believe that these case reports show that diltiazem can cause depression. Cases 1 and 5 suggest that depression caused by diltiazem can be severe and may be accompanied by other symptoms. The latency of onset in these cases ranged from less than a day to one to two months. There was a positive rechallenge in two cases.^{2,3} In five of the eight cases the daily dose was 180 mg or more, and the pattern in case 4 suggested a dose related effect.

We thank the Committee on Safety of Medicines, United Kingdom, for details of cases 2, 6, and 7; Health and Welfare, Canada, for details of cases 1, 4, and 5; and the Food and Drug Administration, United States, for details of case 8.

- 1 Chouza C, Scaramelli A, Caamano JL, De Medina O, Aljanati R, Romero S. Parkinsonism, tardive dyskinesia, akathisia, and depression induced by flunarizine. *Lancet* 1986;i:1303-4.
- 2 Meyboom RHB, Ferrari MB, Dieleman BP. Parkinsonism, tardive dyskinesia, akathisia, and depression induced by flunarizine. *Lancet* 1986;ii:292.
- 3 Andersson KE, Brandt L, Hinfelt B, Ryman T. Migraine treatment with calcium channel blockers. *Acta Pharmacol Toxicol (Copenh)* 1986;58(suppl 2):161-7.
- 4 Berg R. A case of Tourette syndrome treated with nifedipine. *Acta Psychiatr Scand* 1985;72:400-1.
- 5 Falk WE, Wojcik JD, Greenberg AJ. Diltiazem for tardive dyskinesia and tardive dystonia. *Lancet* 1988;i:824-5.
- 6 Giannini AJ, Houser WL, Loiseleur RH, Giannini MC, Price WA. Antimanic effects of verapamil. *Am J Psychiatry* 1984;141:12.
- 7 Dubovsky SL, Franks RD, Schrier D. Phenelzine-induced hypomania: effect of verapamil. *Biol Psychiatry* 1985;20:1009-14.
- 8 Jacobs MB. Diltiazem and akathisia. *Ann Intern Med* 1983;99:794-5.
- 9 Bushe CJ. Organic psychosis caused by diltiazem. *J R Soc Med* 1988;81:296.
- 10 Ahmad S. Nifedipine-induced acute psychosis. *J Am Geriatr Soc* 1984;32:408.
- 11 Kahn JK. Nifedipine-associated acute psychosis. *Am J Med* 1986;81:705-6.
- 12 Hullett FJ, Potkin SG, Levy AB, Ciasca R. Depression associated with nifedipine-induced calcium channel blockage. *Am J Psychiatry* 1988;145:1277-9.

MMR and the nephrotic syndrome

Drs A S AHUJA and M WRIGHT (Paediatric Department, Royal Albert Edward Infirmary,

Wigan) write: In 1969 one of us (ASA) looked after a young child who developed the nephrotic syndrome two weeks after measles immunisation. In 1972 three cases of the nephrotic syndrome after measles vaccination were reported.¹ As yet none of the reports of adverse reactions to measles, mumps, and rubella (MMR) vaccination have included renal lesions (Committee on Safety of Medicines). It is of interest therefore that we have encountered a child who developed steroid responsive nephrotic syndrome after measles, mumps, and rubella vaccination.

A 13 month old girl received the vaccination in November 1988. She was well at the time and receiving no other medication. There was no history of contact with infectious disease. Six days later she became generally unwell and developed periorbital swelling. This proceeded to generalised swelling, in particular of the abdomen, hands, and feet. Her medical history was unremarkable. Her mother had asthma and hay fever, but the child showed no signs of atopy.

Examination showed an apyrexial child with periorbital, hand, and feet oedema and evidence of ascites. There was no lymphadenopathy and no rash. Examination of the respiratory and cardiovascular systems showed nothing abnormal, and in particular she was normotensive. Investigations showed: proteinuria 4+, midstream specimen of urine sterile, haemoglobin concentration 148 g/l, white cell count $14.5 \times 10^9/l$, platelet count $462 \times 10^9/l$, urea and electrolyte concentrations normal, total protein concentration 33.8 g/l, albumin concentration 17.3 g/l, aspartate aminotransferase activity <200 IU/ml, complement normal. She was treated with prednisolone (and penicillin). By the fifth day of treatment the proteinuria had diminished to 1+. The oedema settled by day 10 and she was discharged on the ninth day of treatment. Review 10 days later showed that the residual proteinuria had resolved and her albumin concentration had increased to 35.5 g/l (total protein concentration 60.9 g/l). Prednisolone was finally stopped in early March 1989, after which she remained well.

This case does not prove a causal relation between measles, mumps, and rubella vaccination and the nephrotic syndrome. We report it as an observation which may in future be substantiated by the observations of others and lead to further investigations.

- 1 Kuzemko JA. Measles vaccination and the nephrotic syndrome. *Br Med J* 1972;iv:665-6.

Correction

Evaluation of a call programme for cervical cytology screening

An error occurred in a letter with this title by Dr Katy J Shroff (26 August, p 567). In the last sentence the findings emphasise the need for accurate family practitioner committee registers and not family planning clinic registers as printed.

Case No	Age, sex	Daily dose (mg)	Latency of onset	Reported symptoms	Concomitant therapy	Outcome	Comment
4	55 F	240	36	Hallucinations at night, agitation, depression	Insulin, timolol maleate, glyceryl trinitrate	Recovered	Symptoms occurred after dose increased from 120 mg day, taken previously for 3 months
5	28 M	240	48-56	Slow onset of episodes of amnesia, general depression, despondency, violent verbal expressions	None	Recovered	Symptoms reported by patient after about 5 months' therapy; referred for psychiatric assessment and hospital admission
6	45 F	180	1	Light headed, impaired concentration, "talked gibberish," depressed, "odd sensations in chest"	Atenolol	Not recovered*	Symptoms did not resolve when dose lowered to 60 mg/day; symptoms persisted when atenolol stopped for 3 days
7	54 M	180	10	Arthralgia, depression	Enalapril, bendrofluazide, hydroxocobalamin	Recovered	Depression lifted within 24 hours of stopping treatment
8	60 M	90	1-14	Depression, severe fatigue, photosensitivity (after 3 months' treatment), nightmares (after 18 months' treatment)	Dipyridamole, aspirin	Recovered	Symptoms persisted during 20 months' therapy; nightmares ceased immediately and other symptoms within two weeks of stopping diltiazem

*At time of reporting.