function during prolonged treatment with captopril suggests that some of the renal impairment observed before treatment might be due to a functional disorder.

We recommend close monitoring of renal function at the start of treatment with captopril in patients with chronic renal failure and severe arterial hypertension.

[1] Anonymous. Captopril: benefits and risks in severe hypertension [Editorial]. *Lancet* 1980;ii:129-30.
[2] Coulie P, De Plaen JF, van Ypersele de Strihou C. Captopril induced acute reversible renal failure. *Nephron* 1983;**35**:108-11.
[3] Blythe WB. Captopril and renal autoregulation. *N Engl J Med* 1983;**308**:390-1.
[4] Hollenberg NK, Adams DF. The renal circulation in hypertensive disease. *Am J Med* 1976;**60**:773-84.
[5] Brunner HR, Wauters JP, McKinstry D, Waeber B, Turini G, Gavras H. Inappropriate renin secretion unmasked by captopril in hypertension of chronic renal failure. *Lancet* 1978;ii:704-7.

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Potassium supplements during treatment of glaucoma with acetazolamide

Drugs that inhibit carbonic anhydrase, such as acetazolamide, lower the pressure in most glaucomatous eyes. The ocular hypotensive effects of the drugs appear to be independent of their diuretic action.[1] A common side effect of treatment with acetazolamide is paraesthesia in the legs and arms.[2] This is a symptom of hypokalaemia, so patients suffering from this side effect are often given potassium supplements. This study aimed at determining the necessity for potassium supplements.

Patients, methods, and results

Of a random sample of 150 patients attending hospital ophthalmic clinics and receiving acetazolamide to treat glaucoma, 145 (96·9%) also received potassium supplements. In almost all cases the supplement was one potassium chloride slow release tablet (slow K; 600 mg (8 mmol)) daily. We randomly selected 16 patients with glaucoma who had been treated with acetazolamide and potassium supplements for more than three months. They were questioned about their diet, drug compliance, drug history, and unwanted side effects. Any factor in their medical history that might influence their serum potassium concentrations was taken into consideration. Serum potassium and bicarbonate concentrations were measured, and potassium supplements were then withdrawn from all patients. Serum potassium concentrations were measured monthly, and the patients were asked about relevant changes in dietary habits and side effects.

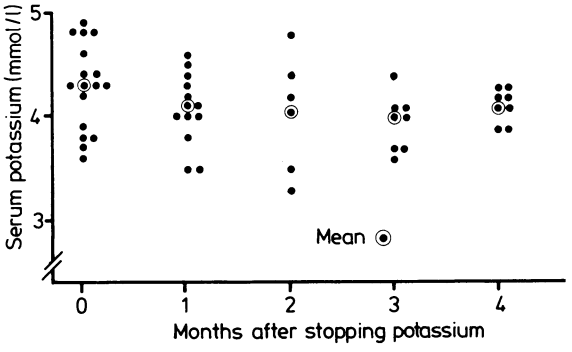
Fifteen patients were taking 8 mmol (600 mg) and one 16 mmol (1200 mg) potassium daily. None had a medical history that might affect their serum potassium concentration. The daily dose of acetazolamide ranged from 500 mg to 1000 mg. Of the patients studied, 15 were white and one Asian; their mean (SD) age was 64·3 (12·1) years. The serum bicarbonate concentration was used as an estimate of compliance, as acetazolamide is a carbonic anhydrase inhibitor, and all patients had serum concentrations below the normal limit of 24 mmol(mEq)/l. They were all also assessed after interview as being good compliers.

The diet of 15 of the 16 patients did not change; the remaining patient received a poor diet throughout the study. The incidence of paraesthesia did not alter after potassium was stopped. but nausea stopped in two patients who had previously suffered from it. The results are summarised in the figure.

Analysis of variance showed that serum potassium concentrations in patients who had stopped taking potassium supplements were not significantly different from those in patients taking potassium or from concentrations in a standard population of a similar age. Two patients who had never taken potassium supplements and had been taking acetazolamide for more than five years were found to have normal potassium concentrations.

Comment

The diuretic effect of acetazolamide is due to inhibition of carbonic anhydrase in the kidney tubules, which reduces the formation of hydrogen and bicarbonate ions. This increases the volume of urine, and metabolic acidosis follows. Further acidosis does not occur with continued acetazolamide treatment as there is a compensatory increase in reabsorption of bicarbonate in the proximal tubule. An initial loss of potassium occurs, but this is self limiting, as is the diuresis.[3] Thus hypokalaemia may occur during the first two weeks of treatment with acetazolamide, and during this period potassium supplements may be required. Further work is needed to determine their necessity.



Serum potassium concentrations in patients with glaucoma treated witha cetazolamide after they stopped taking potassium supplements.

Conversion: SI to traditional units—Potassium: 1 mmol/l = 1 mEq/l.

From the results obtained and the recent reports of the side effects of potassium[4] it seems logical to recommend potassium supplementation only in patients with proved hypokalaemia. If a supplement is required this should be given in a dose that is sufficient to have a therapeutic effect.

[1] Maren TH. Effects of Diamox on electrolyte metabolism. *Bulletin of the Johns Hopkins Hospital* 1954;**9**:277-321.
[2] Epstein DL. Carbonic anhydrase inhibitor side effect, serum chemical analysis. *Arch Ophthalmol* 1977;**95**:1378-82.
[3] Ellis PP. Carbonic anhydrase inhibitors: pharmacologic effects and problems of long therapy. *Symposia on Ocular Therapy* 1960;**4**:32-8.
[4] McMahon FG. Upper gastrointestinal lesions after potassium chloride supplements; a controlled clinical trial. *Lancet* 1982;ii:1059-61.

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Major ocular trauma: a disturbing trend in field hockey injuries

Injuries due to blows from sticks in hockey have not been reported as contributing greatly to ocular sports injuries.[1] We describe three perforating eye injuries seen in the first half of one season.

Case reports

Case 1—A 13 year old girl was hit in the left eye by the stick of an opponent who prematurely played the ball as she ran past. She suffered immediate loss of vision and was admitted to hospital. Examination under general anaesthesia showed a large horizontal corneoscleral laceration with prolapsing iris and ciliary body. The lens could not be identified. A primary repair was made, but six weeks later total hyphaema was still present and an ultrasound scan showed retinal detachment. Vitrectomy was carried out, but a fibrotic retina disinserted through 270° proved impossible to replace. At follow up the eye had no perception of light and was becoming phthisical. Unaided distance acuity in the right eye remained normal (6/5).

Case 2—A 14 year old girl sustained a blow to her left eye when she ran into a high follow through stroke of a stick during a tackle. On admission a perforating injury was obvious and she had no perception of light in this eye. At operation a long laceration extended vertically from the cornea well

into the sclera above. Retina and vitreous protruding from the wound were excised, and the laceration was sutured. Iris tissue was identifiable, and the anterior chamber was repaired. Several weeks later the eye was still blind and was becoming soft and shrunken. The right eye was normal.

Case 3—A 29 year old woman was admitted after having been hit in the right eye by a stick during a mixed match: she had been caught by a follow through stroke of an opposing player. An extensive scleral rupture was noted, which extended posteriorly from the limbus with prolapse of uveal tissue. Avulsions of the upper and lower lids, affecting both canaliculi and the levator aponeurosis, were also noted. Primary repair was undertaken. Postoperatively, extensive intraocular haemorrhage persisted and the eye became painful; it was eviscerated three days later. The left eye was normal.

Discussion

Hockey does not have the bad reputation for eye injuries of games such as squash and hurling.[2] When injuries do occur, however, they can be devastating. Contributory factors include the aggressive nature of the sport, the almost universal absence of face protection, and a stick whose shape permits orbital penetration.

Before 1982 the rules of hockey penalised players for raising the stick above shoulder height. This was then changed, and now the stick may be raised above the shoulder unless the umpire considers this to be dangerous or intimidating to another player.[3] Ironically, in Canadian ice hockey a "high sticks" rule was introduced in 1975 in response to a growing awareness of the high incidence of injuries, most of which were caused by sticks. This change was associated with a noticeable reduction in blinding injuries in the following season.[4] Furthermore, face protectors became compulsory in 1978, resulting in a further distinct reduction in eye injuries (T J Pashby. Paper presented at the international ophthalmic congress, San Francisco, 1982). The recent modification of the rule on high sticks appears to be a retrograde step where safety is concerned, especially as face protection is almost never worn. The injuries described here had disastrous effects on three otherwise healthy young people: that they might have been preventable is a tragedy.

We thank Mr R H B Grey, Mr R H Markham, and Mr D S Thomson for allowing us to review their patients.

[1] Canavan YM, O'Flaherty MJ, Archer DB, Elwood JH. A 10 year survey of eye injuries in Northern Ireland. 1967-76. *Br J Ophthalmol* 1980;**64**:618-25.
[2] Kelly S, Nolan J. Eye injuries in organised sport in a rural area. *Br J Ophthalmol* 1983;**67**:837-9.
[3] Hockey Rules Board. *Rules of the game of hockey with guidance for players and umpires and advice to umpires*. Hockey Rules Board, 1981, 1983. (Available from Mr G Croft, 26 Stompond Lane, Walton-on-Thames, Surrey.)
[4] Pashby TJ. Eye injuries in Canadian hockey. Phase II. *Can Med Assoc J* 1977;**117**: 671-3.

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Treatment of pruritus due to chronic obstructive liver disease

Pruritus in chronic obstructive liver disease is difficult to treat. Antihistamines are of questionable value and cause drowsiness,[1] and cholestyramine commonly gives rise to diarrhoea. We conducted a single blind, randomised crossover trial in which we compared the antipruritic activity of cholestyramine, chlorpheniramine, and placebo with that of terfenadine, a new H_1 specific antihistamine reportedly free of sedative effect.[2-4]

Methods and results

Eight ambulant patients with pruritus due to liver disease (seven with primary biliary cirrhosis and one with sclerosing cholangitis) were selected. None showed signs of encephalopathy. All antipruritic drugs were stopped for at least one week. We obtained the informed consent of each patient and the approval of the local ethical committee.

Each drug was administered for two weeks. A single assessor (JSD) reviewed the patients at the beginning and end of each period of treatment. Treatment was supplied in unlabelled bottles by the hospital pharmacy. The order of administration of the drugs was randomised, different for each patient, and concealed from the assessor. The patients were given diaries in which they entered a daily score for the severity of pruritus. They were advised to reduce or stop medication if any side effects became troublesome. Treatment was started at one dose at night on day 1 (cholestyramine 4 g, terfenadine 60 mg, chlorpheniramine 4 mg, and lactose 200 mg), increasing if tolerated to one dose twice daily on day 3. Chlorpheniramine, terfenadine, and placebo were increased further to one tablet thrice daily on day 5 if tolerated. We analysed the scores for only the last 10 days of each treatment period.

Psychometric testing and electroencephalography were carried out before and after each treatment period. Tests used were standard number connection tests,[5] digit span test, digit symbol matching test, and deletion of e test. Testing was carried out at the same time of day, two to five hours after the last medication. Pruritus scores were assessed with the Wilcoxon rank sum test on paired data, and the results of the psychometric tests with Student's paired *t* test.

Tablet counts confirmed the patients' records of treatment. The table shows that the mean cumulative pruritus scores were significantly lower during treatment with cholestyramine and terfenadine than with placebo ($p < 0.05$) and chlorpheniramine.

Side effects occurred with each drug. One patient stopped taking placebo because of nausea and cutaneous burning; chlorpheniramine was reduced to twice daily by two patients because of drowsiness and to once daily by one because of headache; terfenadine was reduced to twice daily by one patient because of emotional lability; and two patients stopped taking cholestyramine because of diarrhoea and vomiting and two reduced the dosage to once daily because of diarrhoea.

Results of psychometric testing showed that the patients remained stable throughout the treatment regimens. The baseline electroencephalogram was mildly abnormal in two patients (dominant rhythm 7-8 Hz) but no appreciable deterioration was noted after any of the treatment schedules. Patients who developed cerebral side effects spontaneously reduced their dosages and had lost their symptoms by the time they were formally tested.

Cumulative pruritus scores over 10 days in eight patients with liver disease treated with each of cholestyramine, terfenadine, chlorpheniramine, and placebo. (0=no pruritus, 1=mild, 2=moderate, 3=severe)

	Case No								Mean score
	1	2	3	4	5	6	7	8	
Cholestyramine	20	9	10	13	0	10	30	11	12·9
Terfenadine	8	12	10	16	22	24	21	13	15·8
Chlorpheniramine	19	12	13	13	30	27	26	14	19·3
Placebo	20	16	13	19	20	26	30	18	20·3

Comment

To our knowledge this is the first time a randomised crossover trial has been used to assess the effect of drugs in treating chronic pruritus in liver disease. Our results confirm that cholestyramine and chlorpheniramine are associated with a high incidence of side effects and that chlorpheniramine is ineffective. Terfenadine had a significant antipruritic effect and was well tolerated. Cerebral side effects were observed only when patients were taking a higher dose than that recommended (60 mg twice daily). Patients with overt encephalopathy were excluded from our study.

Certain patients benefited strikingly from either cholestyramine or terfenadine. Others experienced a modest improvement with both drugs, which suggests that there may be a place for using them concurrently. They should not, however, be administered simultaneously because of the binding properties of cholestyramine.

We are grateful to Dr E J W Gumpert and the staff of the electroencephalography department and the pharmacy of the Royal Hallamshire Hospital for their help.

[1] Greaves MW. The nature and management of pruritus. *Practitioner* 1982;**226**: 1223-5.
[2] Clarke CH, Nicholson AN. Performance studies with anti-histamines. *Br J Clin Pharmacol* 1978;**6**:31-5.
[3] Nicholson AN, Stone BM. Performance studies with the H_1-histamine receptor antagonists, astemizole and terfenadine. *Br J Clin Pharmacol* 1981;**13**:199-202.
[4] Fink M, Irwin P. CNS effects of the anti-histamines diphenhydramine and terfenadine. *Pharmakopsychiatr Neuropsychopharmakol* 1979;**12**:35-44.
[5] Conn HO, Lieberthal MM. *The hepatic coma syndromes and lactulose*. Baltimore and London: Williams and Wilkins, 1978:182-4.

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