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.

(*Accepted 2 May 1984*)

Bristol Eye Hospital, Bristol BS1 2LX
A J ELLIOTT, MA, MRCP, senior house officer
DYLAN JONES DO, FRCSED, senior house officer

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.

(*Accepted 10 May 1984*)

Department of Medicine, Royal Hallamshire Hospital, Sheffield S10 2JF
J S DUNCAN, MA, MRCP, registrar in medicine
H J KENNEDY, MD, MRCP, lecturer in medicine
D R TRIGER, DPHIL, FRCP, reader in medicine

Correspondence to: Dr D R Triger.