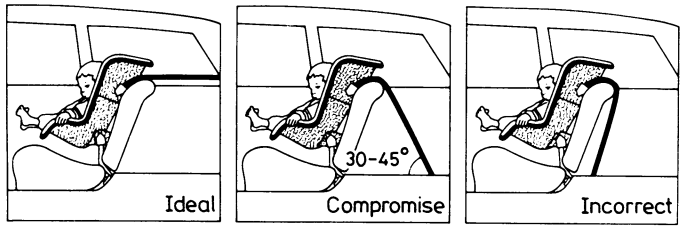
Children in cars: how are they being restrained?

The Motor Vehicles (Wearing of Seat Belts) Regulations state that anyone travelling in the front seat of a car must wear a seat belt and any child under the age of 1 in the front seat must be in an approved restraint designed for his age and weight. This legislation does not ensure the safety of children in cars, as any child may travel in the back seat without any restraint, a child over the age of 1 may travel in the front seat in an inappropriate restraint, and there is no control over the anchorage of back seat restraints.

We studied how children are usually restrained in cars. The British Standards Institution[1] and the Department of Transport[2] recommend the following restraints for children: for those aged under 9 months (< 10 kg) a carrycot either with a harness or wedged between front and back seats; for those aged 9 months to 4 years (10-20 kg) a child seat; for those aged 5-9 years (20-40 kg) a child seat or adult belt with booster cushion; and for those aged over 9 (> 40 kg) an adult seat belt. Alternatively, a specifically designed adjustable belt can be used for ages 5-13 years.

We also considered the anchorage of back seat restraints. Most people do not realise that if the rear anchorage point is too near the back of the seat in an estate or hatchback car the wearer can move within the restraint (figure).



Positioning of rear anchorage points.

Method and results

We interviewed 130 parents using a standard questionnaire to determine the number and ages of children carried in each car; the type, make, and anchorage of restraints used; and whether the restraints were fitted by the owner of the car or a "professional."

Eight drivers could not give sufficient data for analysis. The remaining 122 carried a total of 268 children. Only 91 children (34%) were correctly restrained, 117 (44%) were not restrained, 28 (10%) were in inappropriate restraints, and 34 (13%) were in incorrectly anchored restraints. Full information on anchorage was obtained for 95 of the 108 back seat restraints fitted, of which 34 were incorrectly anchored. There was no difference between those fitted by owners (29 correct out of 55 (53%)) and those fitted by professionals (nine correct out of 24 (38%)).

Only two children were carried in the front seat in contravention of the current law. One was not restrained at all, and the other was held on an adult's lap with a seat belt around both.

Comment

In 1983, 577 car and van occupants under the age of 14 were killed or seriously injured.[3] This figure is 50% less than that of 1982, when the seat belt regulations were not in force. The drop is due mainly to a decrease in injury to front seat passengers (48% down), with only a minimal decrease in injuries to rear seat occupants (3% down).

We found that only one third of children under the age of 14 were correctly restrained, yet only two of our cases contravened the present law. Several reasons were given by parents for not using restraints: firstly, the cost; secondly, restraints decrease the amount of space in a car; and, finally, some children dislike being carried in restraints and climb out or suffer from travel sickness when restrained.

What can be done to promote the use of child restraints? Several studies have emphasised the role of the doctor in educating parents,[4 5] but, though counselling helps, it does not appreciably increase the use of child restraints. More effort is needed from those concerned with community health, the media, and those responsible for opinion making and, ultimately, legislation.

1 British Standards Institution. *Specification for seat belt assemblies for motor vehicles. Section 5: restraining devices for children.* Milton Keynes: British Standards Institution, 1960:14-20. (BSI 3254.)
2 Department of Transport. *Child safety in cars 1982.* London: HMSO, 1983.
3 Department of Transport, Scottish Development Department, Welsh Office. *Road accidents Great Britain 1983.* London: HMSO, 1984.
4 Pless IB. Accident prevention and health education: back to the drawing board? *Pediatrics* 1978;**62**:431-5.
5 Reisinger KS, Williams AF, Wells JK, John CE, Roberts TR, Podgainy HJ. Effect of pediatricians' counselling on infant restraint use. *Pediatrics* 1981;**67**: 201-6.

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Control of an outbreak of systemic Candida albicans

Recently an outbreak of systemic candidiasis on the intensive care unit at the London Hospital was reported.[1] From July 1983 to March 1984 a single strain of *Candida albicans*, defined as serotype A, morphotype A1, biotype 0/1 5 5/7, caused 12 cases of proved systemic candidiasis, and one suspected case, as defined previously.[1] The staff transmitted the strain between patients on their hands, with their mouths and perineums acting as secondary reservoirs of infection. Four of the 65 staff were oral carriers, and one nurse carried the strain on her hands.[1] We report here the outcome of using oral ketoconazole in an attempt to control this outbreak; ketoconazole has been reported as superior to amphotericin B and nystatin[2] and has been used successfully to treat chronic mucocutaneous candidiasis[3] and systemic candidiasis.[4]

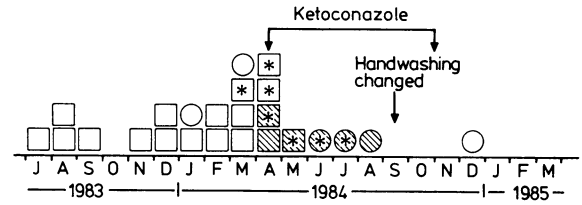
Patients, methods, and results

All patients admitted to the intensive care unit from May to October 1984 received oral ketoconazole 400 mg daily until they were discharged. Patients were swabbed orally and perineally on admission and discharge and weekly for carriage of yeast. Oral and perineal carriage in the staff was assessed three months after the treatment with ketoconazole was begun and also at six months, just before it was stopped. All isolates were typed as described previously and confirmed by sonityping.[1]

The outbreak strain proved fairly resistant to Hibiscrub, the disinfectant used at the time of the outbreak.[1] Thus either Betadine (Napp, United Kingdom) or Hibisol (Imperial Chemical Industries, United Kingdom) was used instead of Hibiscrub when patients with heavy candidal infection were being nursed. The strain proved fully sensitive to both these reagents in handwashing experiments (W Lee, J P Burnie, R C Matthews, unpublished observations).

In April 1984, when an increasing proportion of patients were receiving ketoconazole, there was one proved and three suspected cases of systemic candidiasis, all due to the strain that caused the outbreak. In the next six months, when all patients were receiving ketoconazole, one proved and three suspected cases occurred, and only one of these was caused by the outbreak strain. After treatment was stopped there was one other case, which was caused by a different strain (figure).

The proportion of patients in the unit for over three days who were colonised with the outbreak strain also fell sharply, from 39% (16 out of 41 patients) before treatment with ketoconazole to 15% (seven of 47 patients) during treatment. The incidence of colonisation remained low at 17% (four of 24) after ketoconazole was withdrawn. At this time only two of the oral swabs taken from the 65 staff grew yeasts, and neither was the outbreak strain.



Distribution of cases of systemic candidiasis due to outbreak strain (□) and non-outbreak isolate (○) over time. Shading indicates prophylaxis with ketoconazole.
*Suspected cases.

Comment

Oral ketoconazole appreciably reduced the rate of isolation of the outbreak strain from both systemically infected and colonised patients. The outbreak strain did not reappear when ketoconazole was withdrawn. All cases of systemic candidiasis acquired in the unit before April 1984 were caused by the outbreak strain, whereas after May 1984 all cases were caused by other strains.

Control of an outbreak depends on its identification and the prevention of cross infection. Existing handwashing reagents can be replaced with fungicidal disinfectants such as Hibisol or Betadine, and antifungal prophylaxis can be given. Recent work in neutropenic patients in whom infection was probably due to an endogenous isolate showed that ketoconazole was as effective as amphotericin B, and prophylaxis with either agent failed.[5] In this study ketoconazole failed in six cases, perhaps because of poor absorption in the gut as five patients had undergone major gastrointestinal surgery. Treatment with ketoconazole resulted in the virtual elimination of the outbreak strain, the incidence of cases returning to its former value, with occasional cases caused by the patients' own yeast flora.

A much shorter course of prophylaxis might have been equally effective and could be considered in any unit where the incidence of candidal sepsis is unacceptably high and cross infection a problem.

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1 Burnie JP, Odds FC, Lee W, Webster C, Williams JD. Outbreak of systemic Candida albicans in intensive care unit caused by cross infection. *Br Med J* 1985;**290**:746-8.
2 Hann IM, Prentice HG, Corringham R, *et al.* Ketoconazole versus nystatin plus amphotericin B for fungal prophylaxis in severely immunosuppressed patients. *Lancet* 1982;i:826-9.
3 Hay R. Ketoconazole: a reappraisal. *Br Med J* 1985;**290**:260-1.
4 Drouet E, Dupont B. Laboratory and clinical assessment of ketoconazole in deep seated mycoses. *Am J Med* 1983;**74**:30-47.
5 Donnelly JP, Starke ID, Galton DAG, Catovsky D, Goldman JM, Darrell JM. Oral ketoconazole and amphotericin B for the prevention of yeast colonization in patients with acute leukaemia. *J Hosp Infect* 1984;**5**:83-91.

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Controlled trial of arbaprostil in bleeding peptic ulcer

In patients with acute upper gastrointestinal bleeding no convincing effect on the bleeding has been reported after treatment with antisecretory drugs such as histamine H_2 receptor antagonists,[1] antifibrinolytic agents such as tranexamic acid,[1] or somatostatin,[2] which reduces both splanchnic blood flow and acid secretion. "Cytoprotective" prostaglandin analogues may be an innovation in treatment[3] and were claimed to be beneficial in a single patient.[4] We have carried out a prospective double blind, placebo controlled trial of the efficacy of 15(*R*)-15-methylprostaglandin E_2 (arbaprostil[4]; 50 μg by mouth every six hours for seven days) in stopping acute bleeding from erosive or ulcerative lesions of the stomach and duodenum and preventing rebleeding.

Patients, methods, and results

During one year 237 consecutive patients admitted for haematemesis or melaena were considered for the study, which was approved by the ethical committee of Funen and Vejle Conties, Denmark. Criteria for entry were evidence of bright red or coffee ground gastric aspirate on lavage, gastritis or gastric or duodenal ulcer with active bleeding or fresh clot formation seen during the subsequent endoscopy, a transfusion requirement of 2 units or more and packed cell volume of 0·30 or less, or a postural change in diastolic blood pressure of 10 mm Hg or more. Of 87 patients aged over 18 who satisfied these criteria, four needed emergency surgery, one died before endoscopy, and in one case informed consent was not obtained. The remaining 81 patients were given at least four doses of trial medicine and were considered suitable for analysis (table). Arrest of bleeding was defined as stabilisation of the packed cell volume and vital signs, clearance of nasogastric aspirate, and a reduced requirement for or no further need of transfusion.

The 95% confidence interval for the observed difference[5] between arbaprostil and placebo in stopping bleeding within 48 hours ranged from 6% in favour of arbaprostil to 38% in favour of placebo. As regards the frequency of rebleeding the same interval ranged from 6% in favour of arbaprostil to 32% in favour of placebo. These estimates suggest that we had not overlooked any major therapeutic difference. Endoscopic assessment of 72 patients at completion of the trial disclosed no difference in the rate of ulcer healing. A statistically significant ($p < 0·05$) improvement in concomitant gastritis and duodenitis, however, was shown by means of a scoring system. Four patients aged 73-81 years died. Necropsy was done in three cases and showed concomitant acute leukaemia and ovarian and renal carcinoma, respectively.

Comparability of groups and outcome of treatment

	Arbaprostil (n = 40)	Placebo (n = 41)	Significance
Sex (M/F)	20/20	24/17	NS*
Mean age (years)	70·1	68·8	NS†
No (%) of smokers	16 (40)	21 (51)	NS*
No (%) taking aspirin like drugs	19 (48)	23 (56)	NS*
Mean systolic blood pressure (mm Hg)	131	132	NS†
Mean pulse rate/min	91	91	NS†
Mean serum urea (mmol/l)	14·9	13·1	NS†
Mean packed cell volume	0·26	0·25	NS†
No (%) with bleeding gastric ulcer	27 (68)	24 (59)	NS*
No (%) with bleeding duodenal ulcer	12 (30)	16 (39)	NS*
No (%) with haemorrhagic gastritis	1 (2·5)	1 (2·4)	NS*
No (%) with visible vessel	26 (65)	24 (59)	NS*
No (%) whose bleeding stopped after 24 h and did not recur	19 (48)	22 (54)	NS*
No (%) whose bleeding stopped after 48 h and did not recur	20 (50)	27 (66)	NS*
Mean transfusion requirements (units)	6·7	6·7	NS†
No (%) with rebleeding	12 (30)	7 (17)	NS*
No (%) operated	9 (23)	7 (17)	NS*
No (%) of deaths	2 (5)	2 (5)	NS*

*χ^2 test: $p > 0·05$.
†Student's t test: $p > 0·05$.
Conversion: SI to traditional units—Urea: 1 mmol/l ≈ 6 mg/100 ml.

Comment

These findings show that arbaprostil is unlikely to have a substantial effect on outcome in patients with acute bleeding from ulcerative lesions in the stomach or duodenum. Although our sample was too small to exclude a marginal beneficial effect on mortality, the results challenge the belief that "direct cytoprotection"[3] provides a major breakthrough in medical treatment of patients with upper gastrointestinal bleeding and for the prevention of rebleeding.

We thank the Upjohn Company, Kalamazoo, Michigan, USA, for supplying the arbaprostil and placebo solutions.

1 Barer D, Ogilvie A, Henry D, *et al.* Cimetidine and tranexamic acid in the treatment of acute upper-gastrointestinal-tract bleeding. *N Engl J Med* 1983;**308**:1571-5.
2 Somerville KW, Henry D, Davies JG, Hine KR, Hawkey CJ, Langman MJS. Somatostatin in treatment of haematemesis and melaena. *Lancet* 1985;i:130-2.
3 Robert A. Regulatory systems: prostaglandins. In: Berk JE, ed. *Bockus gastroenterology*. Philadelphia: W B Saunders, 1985:4480-6.
4 Weiss JB, Peskin GW, Isenberg JI. Treatment of hemorrhagic gastritis with 15(R)-15 methyl prostaglandin E_2: report of a case. *Gastroenterology* 1982;**82**:558-60.
5 Elashoff JD. Statistical considerations in drug trials of peptic ulcer. *J Clin Gastroenterol* 1981;**3** (suppl 2):135-40.

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