developed upper gastrointestinal haemorrhage, 33% of those in the study of Chisholm *et al* (1977), and 44% of those in the study of Hadjiyannakis *et al* (1971) were women, which suggests that other factors play a part.

It has been suggested that administration of corticosteroids may damage the gastric mucosa[29] or impair the resistance of the mucosal barrier to injury.[3] A significantly higher incidence of peptic ulceration was observed in a large series of patients with rheumatoid arthritis treated with steroids,[27] and the recent findings of McGeown *et al* (1977) in renal transplant recipients reinforces this view. With a starting dose of prednisone of only 20 mg/day after transplantation only one out of 100 patients died from upper gastrointestinal haemorrhage.

The treated and untreated groups were well matched for age, sex, and blood group and the immunosuppressive regimen was standardised throughout the study. The high incidence (18%) of gastrointestinal haemorrhage in the untreated group may reflect the steroid dosage used. No other symptomatic upper gastrointestinal tract complications occurred in either group, however, which compares favourably with the results in other series, in which symptomatic peptic ulceration has occurred in up to 22% of patients.[6] The overall mortality in the untreated group was low at 3%, and among those who bled, the mortality was only 16%.

None of the patients who had previously diagnosed (and medically treated) peptic ulceration but did not receive cimetidine bled, but the patient with giant rugal hypertrophy died from his gastrointestinal haemorrhage. The endoscopic confirmation of healing in the patients in the treated group who were known to have active lesions shortly before transplantation is encouraging, and cimetidine may have a therapeutic as well as prophylactic part to play in managing established peptic ulceration in transplant recipients.

Cimetidine is excreted by the kidneys; in the presence of delayed graft function 200 mg of cimetidine given every 12 hours will produce therapeutic blood concentrations until renal function begins to improve.[30] The drug appears to be completely cleared by haemodialysis.[30] Recent reports[31 32] of central nervous system and other side effects (mental confusion, agitation, flushing, and sweating), possibly related to overdose, are worrying, but none were observed in our patients. No other adverse effects attributable to the drug were observed, either clinically or in terms of haematological or biochemical values.

We therefore consider that cimetidine, administered routinely after renal transplantation, promises to be an effective and safe prophylactic agent against upper gastrointestinal complications. Further trials and longer follow-up of its efficacy are indicated.

References

1 Moore, T C, and Hume, D M, *Annals of Surgery*, 1969, **170,** 1.
2 Hadjiyannakis, E J, *et al*, *Lancet*, 1971, **2,** 782.
3 Coleman, M J, *et al*, *Medical Journal of Australia*, 1972, **1,** 546.
4 Penn, I, *et al*, *Annals of Surgery*, 1968, **168,** 865.
5 Libertino, J A, Zinman, L, and Dowd, J B, *Surgical Clinics of North America*, 1971, **81,** 733.
6 Chisholm, G D, *et al*, *British Medical Journal*, 1977, **1,** 1630.
7 Owens, M L, *et al*, *Annals of Surgery*, 1977, **186,** 17.
8 Gurland, H J, *et al*, *Proceedings of the European Dialysis and Transplant Association*, 1973, **10,** 17.
9 Hampers, C L, *et al*, *Long-Term Haemodialysis*, p 144. New York, Grune and Stratton, 1973.
10 Peterson, W L, *et al*, *New England Journal of Medicine*, 1977, **297,** 341.
11 Silen, W, and Skillman, J J, *Advances in Internal Medicine*, 1972, **19,** 195.
12 Opolon, P, and Caroli, J, *Médicine et Hygiène*, 1974, **32,** 159.
13 MacDougall, B R D, *et al*, *Lancet*, 1977, **1,** 617.
14 Black, J W, *et al*, *Nature*, 1972, **236,** 385.
15 Pounder, R E, *et al*, *Gut*, 1975, **16,** 397.
16 MacDougall, B R D, Bailey, R J, and Williams, R, in *Cimetidine*, ed W R Burland and M A Simkins, p 329. Amsterdam, Excerpta Medica, 1977.
17 Moody, F G, *et al*, *American Journal of Digestive Disease*, 1976, **21,** 148.
18 Bodemar, G, and Walan, A, *Lancet*, 1976, **2,** 161.
19 Gray, G R, *et al*, *Lancet*, 1977, **1,** 4.
20 Frost, F, *et al*, *British Medical Journal*, 1977, **2,** 795.
21 Johnson, L R, *Gastroenterology*, 1968, **54,** 8.
22 Davenport, H W, in *Progress in Gastroenterology*, vol II, ed G Jerzy-Glass, p 42. New York, Grune and Stratton, 1970.
23 Moore, T C, Thompson, D F, and Glassock, R J, *Annals of Surgery*, 1971, **173,** 381.
24 Comty, C M, Baillod, R A, and Shaldon, S, *Proceedings of the European Dialysis and Transplant Association*, 1965, **2,** 88.
25 Pendras, J P, and Erickson, R U, *Annals of Internal Medicine*, 1966, **64,** 293.
26 Wieners, S N, Veacs, V, and Shapiro, H, *Radiology*, 1969, **92,** 110.
27 Spiro, H M, and Miles, S S, *New England Journal of Medicine*, 1970, **263,** 286.
28 Gordon, E M, Johnson, A G, and Williams, G, *Lancet*, 1972, **2,** 226.
29 West, H F, *British Medical Journal*, 1959, **2,** 680.
30 Canavan, J S F, and Briggs, J D, in *Cimetidine*, ed W R Burland and M A Simkins, p 75. Amsterdam, Excerpta Medica, 1977.
31 Grimson, T A, *Lancet*, 1977, **1,** 858.
32 Delaney, J C, and Ravey, M, *Lancet*, 1977, **2,** 512.

(*Accepted 28 November 1977*)

Hang-gliding accidents

RAIMUND MARGREITER, LOIS-JÖRG LUGGER

British Medical Journal, 1978, **1,** 400-402

Summary

Seventy-five known hang-gliding accidents causing injury to the pilot occurred in the Tyrol during 1973-6. Most occurred in May, June, or September and between 11 am and 3 pm, when unfavourable thermic conditions are most likely. Thirty-four accidents happened during launching, 13 during flight, and 28 during landing, and most were caused by human errors—especially deficient launching technique; incorrect estimation of wind conditions, altitude, and speed; and choice of unfavourable launching and landing sites. Eight pilots were moderately injured, 60 severely (multiply in 24 cases), and seven fatally; fractures of the spine and arms predominated. Six of the 21 skull injuries were fatal.

The risk of hang-gliding seems unjustifiably high, and safety precautions and regulations should be adopted to ensure certain standards of training and equipment and to limit flying to favourable sites and times.

Chirurgische Universitätsklinik, Anichstrasse 35, A-6020 Innsbruck, Austria
RAIMUND MARGREITER, MD, surgeon
LOIS-JÖRG LUGGER, MD, surgeon

Introduction

Hang-gliding accidents are increasing with frightening rapidity, but there have been few reports on their epidemiological and

medical aspects.[1 2] We have therefore analysed data on accidents in the Tyrol as a basis for formulating precautions to reduce the high risk of this fashionable new sport.

Methods

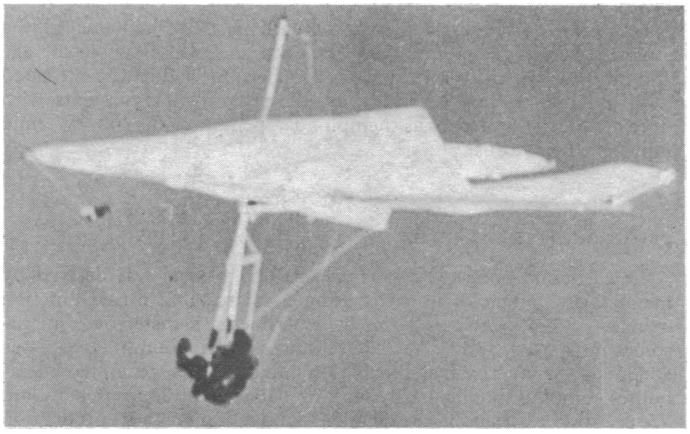
We asked the departments of emergency surgery in all Tyrolean hospitals, police stations near the known flying areas, and several hang-gliding clubs for details of accidents occurring in 1973-6 in which the pilot was injured. Hang-gliding accidents, however, are not always reported. Up to the end of 1976 the victims of such accidents were not covered by sickness insurance in Austria and many injuries —we estimate about a quarter—are likely to have been falsely registered for financial reasons. Moreover, until the beginning of 1977 Austria had no hang-gliding association and fliers were not obliged to report accidents. Our figures therefore must be considerable underestimates.

Results

Between 1 January 1973 and 31 December 1976 there were 75 reported accidents in which the pilot was injured or (in 7 cases) killed; two men were injured twice. The number increased steeply during these years, being 1, 3, 27, and 44 in the years 1973 to 1976 successively. The age range was 19-61 (mean 31·7), and three of the pilots were women. Many were experienced hang-gliders, the mean number of previous flights being 94 (range 7-200). None were known to be abusing drugs or alcohol.

Time of accidents—Most accidents happened during May, June, or September (table I), months when hot weather in the Alps creates more turbulent conditions that would partly explain these peaks. Convection currents may help to account for the fact that 73% occurred between 11 am and 3 pm, though this is also the time when most pilots fly.

Circumstances of accidents—Thirty-four accidents took place during the launching, 13 during the flight, and 28 during the landing. Over 90% were caused by human errors. The commonest were deficiencies in launching technique, generally inadequate speed or too sharp an angle of take-off (both resulting in a "pancake" crash analogous to an aircraft stall). Turbulent side-winds, however, can also cause a crash by bringing the kite out of balance when it is too near the ground, and unfavourable launching sites were sometimes chosen. During flight and landing the main errors were incorrect estimation of wind conditions, altitude, and speed, and choice of unfavourable landing sites. Two accidents were caused by equipment failure. In six cases wind conditions were the sole cause—the experienced pilots had no opportunity to prevent a crash when their gliders were pushed straight down by a down-draught (fig). The most remarkable accident was a collision between two kites during flight; one of the pilots was killed and the other broke his spine.



Hang-glider photographed during a vertical fall.

TABLE II—*Injuries sustained by 73 pilots**

	No of injuries	No fatal
Skull:		
Fractures with brain contusion	3	3
Brain contusion	4	3
Concussion	14	
Fractures of:		
Ribs	4	
Spine	24	1
Humerus	2	
Radius	8	
Ulna	4	
Hand	7	
Pelvis	3	
Sacrum	2	
Femur	3	
Knee	1	
Tibia and fibula	8	
Ankle	5	
Foot	10	
Elbow luxation	6	
Major soft-tissue injury	6	
Blunt abdominal trauma with:		
Ruptured kidney	3	
Ruptured spleen	1	
Ruptured spleen and kidney	1	
Thorax contusion	3	
Total	122	7

*Some pilots had multiple injuries, and two had two separate accidents.

Models and flying positions—We could not assess whether a particular model of hang-glider or a particular flying position carried special risk. Three-quarters of the hang-gliders were standard models and only a quarter competition models; similarly, over 80% of the pilots flew in a sitting position and under 20% in a prone position. It is not clear, however, how far these figures reflect the usual proportions.

Injuries—Eight of the injuries were moderate, 60 serious (multiple in 24 cases), and seven fatal. The types of injury were varied, with spinal trauma predominating (see table II). In one case a cervical subluxation fracture was immediately fatal, and in two a temporary paraplegia occurred at the level of a fractured vertebra. There were only 21 skull injuries, probably because most fliers wore crash helmets; but six were fatal. The numerous severe arm injuries were probably due to holding the control bar during the crash.

Conclusions and recommendations

Without giving any definitive figures, Yuill[3] claims that hang-gliding is no more dangerous than are horse riding, rock climbing, potholing or motorcycle racing. Our findings do not support this view. We could not make any precise calculation of risk because the exact numbers of fliers and flights were not known. At the end of 1976, however, an estimated 350 pilots were active in the Tyrol, five times as many as two years previously. Since 36 Tyroleans had an accident in these two years (the other 37 were foreigners), the risk of hang-gliding seems remarkably high.

We therefore recommend the following precautions that would lessen the risk:

(1) Practical and theoretical training in aerodynamics, meteorology, flight regulations, and first aid in authorised schools.

(2) A system of compulsory flight licences.

(3) The use of crash helmets and protective dress and equipment (including gloves and ankle boots).

TABLE I—*Mean monthly distribution of hang-gliding accidents 1974-6*

	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec
No of accidents	1	2	9	6	13	15	4	6	11	6	1	1

(4) Control by a public authority of the manufacture and selling of hang-gliders, which should be licensed (at present any manufacturer can produce them).

(5) The mandatory use of altimeters and air-speed indicators.

(6) Designation of flying areas with favourable launching and landing sites.

(7) Avoidance of flying between 11 am and 3 pm on hot days, when dangerous convection currents are likely.

(8) The establishment of a central information office to collect facts and figures about accidents so that safety precautions can be formulated.

Even with proper safety precautions accidents will inevitably occur, and full insurance cover should be compulsory. In our series even experienced pilots following all the rules had accidents caused by unforeseen turbulence, and we believe that hang-gliding will remain a dangerous sport.

References

[1] Eiseman, B, *Journal of the American Medical Association*, 1975, **233**, 171.
[2] Krissoff, W B, and Eiseman, B, *Journal of the American Medical Association*, 1975, **233**, 158.
[3] Yuill, G M, *British Medical Journal*, 1977, **1**, 823.

(*Accepted 9 December 1977*)

Possible role of antibody specific for a practolol metabolite in the pathogenesis of oculomucocutaneous syndrome

H E AMOS, B G LAKE, J ARTIS

British Medical Journal, 1978, **1**, 402-407

Summary and conclusions

The clinical distribution of an antibody to a metabolite of practolol was investigated, particularly in relation to the oculomucocutaneous syndrome. Serum samples were obtained from patients with and without a history of adverse reaction to practolol and two groups of control patients who had never taken the drug. Five patients also participated in a challenge study.

The presence of the antibody was found to be related to practolol administration, and antibody activity could be increased by antigenic challenge. The role of this antibody in the pathogenesis of the oculomucocutaneous syndrome remains uncertain. The lesions may be the result of a hitherto unknown type of hypersensitivity response to practolol.

Introduction

The serious adverse effects associated with practolol are perhaps the biggest single medical tragedy since the thalidomide disaster and they emphasise the problem of assessing drugs for unexpected untoward reactions. Extensive epidemiological studies have shown that the tissue damage associated with practolol is probably confined to this one drug and is not seen in patients receiving other beta-blocking agents. The pathology of the lesions, however, is unusual for a drug-induced adverse effect, and the available data do not explain their pathogenesis. Further data are needed to clarify the pathological processes. The experimental effort necessary for such investigations is justifiable, if only to establish that the adverse responses are related to the molecular structure of practolol rather than to its specific pharmacological and therapeutic actions.

The British Industrial Biological Research Association, Carshalton, Surrey SM5 4DS

H E AMOS, MB, FIBIOL, immunologist
B G LAKE, BSC, PHD, scientific officer
J ARTIS, BSC, HNC, research assistant

We have described a method for detecting an antibody with specificity for a phase-one metabolite of practolol,[1] and this paper concerns the clinical distribution of the antibody, particularly in relation to the so-called oculomucocutaneous syndrome.

Patients and methods

Serum samples for the investigation were obtained from four groups of patients.

Group 1—This group comprised 24 patients who had taken practolol for various periods and developed some type of adverse effect. Most had conjunctival signs (as assessed by Mr P Wright, Moorfields Eye Hospital). Also included were four patients with sclerosing peritonitis that had been confirmed by laparotomy.

Group 2—This group of 15 patients had also been treated with practolol, but when serum samples were taken they had no adverse signs or symptoms attributable to their treatment.

Group 3—These 10 patients were being treated with either propanolol or atenolol and were free from any untoward effects of the drugs.

Group 4—Two control groups of patients were included in the study: (*a*) 10 patients who were matched so far as possible for age and physical disability with those in group 1, but had not been treated with practolol or any beta-blocking drug; and (*b*) 11 patients with unrelated diseases such as pemphigus, pemphigoid, and systemic lupus erythematosus.

DETECTION OF SERUM ANTIBODY

Production of antigenic determinants—The antigen was derived by metabolising practolol in vitro using a microsomal mixed-function oxidase system.[1] For this series of experiments hamster-liver microsomes were used, and ^{125}I-labelled human serum albumin (HSA) was added to the microsomal incubation mixture; the generated metabolites combine spontaneously with the labelled albumin preparation. We are still investigating the detailed kinetics of metabolite production and type of macromolecular binding as well as the molecular structure of the metabolites. Two antigen controls were used, one omitted essential cofactors from the microsomal incubation mixture and the other omitted practolol until the microsomal incubations ended.

Antibody assay—A modification of the technique described by Farr[2] was used. The HSA-metabolite complex was freed from unmetabolised drug and uncoupled metabolites by Sephadex G-25 chromatography and then added to dilutions of sera from patients in the four groups. Complexes of HSA metabolite were precipitated by antibody specific for the metabolite in the presence of saturated