Figure 3 Patient 4 weeks after onset.

Figure 4 Patient 6 weeks after onset.

tion with trauma or a concurrent debilitating process such as diabetes, alcoholism, or polymyositis.¹⁻⁵ The role of surgical exploration and debridement with irrigation using a hydrogen peroxide solution together with high dose antibiotics has been emphasised.² The pathogenesis of such rapidly spreading necrosis is uncertain. A hypersensitivity component such as an Arthus response has been suggested while an alternative hypothesis of activation of proteolytic enzymes with collagen necrosis has also been postulated.² Studies of the Arthus response in experimental animals have shown that it can be suppressed by depleting the animal of complement or neutrophils and the intensity of the response can be reduced in animals and humans by corticosteroids.⁶ The transient leucopenia has been reported previously and is due to convergence of granulocytes in affected areas, spleen, and lymphatics.

This patient showed some unusual features of the disease. There was no history of trauma or other underlying pathology, his general condition improved markedly after the introduction of steroids, and he settled without surgical intervention. The use of adjunctive steroids in the reported cases is not well described (except in the case associated with polymyositis) and in cases

where the underlying infection is poorly controlled their use might have serious consequences precipitating a potentially fatal bacterial septicaemia.

Appropriate high dose antibiotic therapy, often with surgical debridement, is the first line treatment in this disease. It may be, however, that in certain cases where the infection is controlled adjunctive steroids could limit the vasculitis and subsequent tissue damage which occurs. This patient's excellent recovery on conservative treatment may be due to the absence of any predisposing pathology but may also be a reflection of the effects of steroids on the pathogenesis of this aggressive disease.

I would like to thank Mr J J Kanski for his help and for allowing me to present this patient.

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Ocular complications associated with bungee jumping

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Bungee jumping originated in the islands of the South Pacific as a means of initiating young males into the realms of manhood. A length of vine was attached to their legs, but today this is replaced with a bungee rope. The sport is

increasing in popularity with a wide range of age groups taking part. It is reported to be dangerous, with lethal accidents caused by miscalculations of the extent to which the rope will stretch, and in one case, a jumper who forgot to attach his

Figure 1 Right fundus.

Figure 2 Left fundus.

rope.¹ Other reported complications include a non-fatal hanging injury and quadriplegia secondary to a locked facet joint. The only ophthalmic complication previously reported is that of periorbital bruising. We report a case of a man who presented with acute diminution in vision following a bungee jump.

Case report

A medically fit 31-year-old man bungee jumped, having been weighed, with an appropriate rope fastened to his feet and a harness attached to his body. He had been examined by his general practitioner 2 weeks previously and his unaided vision had been recorded as normal in both eyes. The patient jumped, head first, from a height of approximately 185 feet and he remained suspended in the air 'bobbing' up and down for approximately 1 minute. As soon as he was released from the harness he noticed that his vision was blurred.

He was seen in casualty at the eye hospital 2 hours following the jump and his unaided vision was then right 6/60, not improving with pinhole, and left 6/18, 6/9 with pinhole. The anterior segments were normal with clear media but his fundi showed scattered superficial retinal

Figure 3 Right eye.

and preretinal haemorrhages and numerous cotton wool spots in the macular area of each eye. These changes were more marked in the right eye and in this eye were associated with macular oedema (Figs 1 and 2). A general examination, including a full neurological examination, was normal and investigations including full blood count, erythrocyte sedimentation rate, plasma viscosity, clotting factors, and blood sugar were all within normal limits.

When the patient was seen the following day he had developed a subconjunctival haemorrhage in the right eye (Fig 3). One week after the jump the vision had improved in both eyes to right 6/18 and left 6/6.

One month after the injury the vision was 6/6 unaided in each eye with almost complete resolution of the retinal changes apart from minimal residual haemorrhages in his right eye.

Comment

This is a previously unreported complication of bungee jumping although these complications had been expected.³ The retinopathy we report is typical of Purtscher's traumatic retinal angiopathy which is thought to be due to an abrupt rise of intravascular pressure in the upper portion of the body, frequently following sudden compression of the chest.⁴ It is likely that the rise in intravascular pressure is due to the sudden deceleration which occurs when the downward momentum of the bungee jumper is overcome by the tensile strength of the cord.

We would advise caution in the sport of bungee jumping in view of the reported ocular complications in an otherwise fit man.

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