

LETTERS TO THE EDITOR

Traumatic ocular haemorrhage related to bungee jumping

EDITOR,—I read with interest the report by David *et al.*¹ I have been following a similar case of a patient who presented with sudden loss of vision immediately after bungee jumping.

A 26-year-old, healthy white female engaged in bungee jumping at the local county fair. Immediately afterwards, she experienced blurred vision, more so in the right eye, as well as a spontaneous nose bleed. Three days later, she presented to the retina service with a visual acuity of 20/400 in her right eye and 20/20-2 in her left eye. Her pupil response was normal.

On external examination, a monocular haematoma of the right eye could be seen, as well as a small subconjunctival haemorrhage in the left eye. Slit-lamp examination was unremarkable. Fundus examination showed an abnormal foveal reflex with oedema in both eyes, and small retinal haemorrhages in the right eye (Fig 1). Visual field examination showed a slight depression of her central sensitivity in both eyes, more so in the right eye. Amsler grid testing demonstrated a small area of central blurring, more so in the right than in the left eye. Fluorescein angiography showed a trace oedema in the late phase of the angiogram in the right eye. She had not taken any aspirin containing medications; she was taking oral contraceptives; there was no history of spontaneous nose bleeds or easy bruising before the ocular injury.

On return visits, there was a gradual improvement of vision, although 7 months from the initial trauma, the acuity had not improved to any better than 20/70. The macular changes had regressed and repeat fluorescein angiography showed only mild retinal pigment epithelium changes in the fovea.

The most likely explanation for her ocular findings is that the patient experienced a sudden increase in venous pressure during the bungee jump. The first part of the bungee jump consisted of a free fall, during which the patient dived down with her head in the lowest position. During this episode, there is an acceleration which creates a tendency of the blood to move from the head towards the feet. At the end of the free fall, there is a sudden deceleration which is caused by the stretching of the bungee cord. The reverse of direction of acceleration will create pooling of blood towards the head, causing a rapid rise in intravenous pressure. When the bungee cord is stretched maximally, it recoils

and causes an acceleration back towards the point of origin from where the patient jumped. This leads to an additional increase in movement of blood towards the head and eye, creating a further increase in venous pressure. The axial accelerating forces are furthermore affected by centrifugal forces created by the whiplash-like activity of the bungee cord at the time of maximal stretch.

Accelerating forces along the axial length of the body can be expressed as 'g' forces; 1 g is the force normally exerted by gravity. If the acceleration is towards the feet, then the g force is positive; accelerating towards the head is a negative g force. Each increase in negative g force is accompanied by an increase in venous pressure above the level of the heart.

Under testing conditions, conjunctival and retinal haemorrhages can be produced in a rapid deceleration with high g forces for a short duration. Retinal haemorrhages are produced by increasing the intravenous pressure to 100 mm Hg or more, which is achieved at approximately a minus 3 g force.²

A similar mechanism of sudden rise of venous pressure that leads to ocular haemorrhages can be seen in Purtscher's retinopathy and Valsalva retinopathy.^{3,4} Our patient had no evidence of spontaneous haemorrhages before the injury, and prothrombin time and partial thromboplastin time levels were normal, as was her bleeding time.

This underscores that bungee jumpers should be aware of the risk of loss of vision.

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- 1 David DB, Meart T, Quinlan MP. Ocular complications associated with bungee jumping. *Br J Ophthalmol* 1994; 78: 234-5.
- 2 Byrnes V. Elevated intravascular pressure as an etiologic mechanism in the production of eye injuries. *Trans Am Ophthalmol Soc* 1959; 57: 473-538.
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Reply

EDITOR,—Thrillseekers throughout the world are queuing up for the opportunity to bungee jump. Whereas previously this feat could only be performed in New Zealand it is now possible to do so at most local county fairs and charity fund raising events. This has allowed for greater access and perhaps less control on the operators.

Unfortunately, these adventurers may be trading a few seconds of sheer exhilaration for a lifetime of visual disability. There have been a few cases of retinopathy secondary to bungee jumping reported in the literature. In the case previously reported by David and colleagues the patient's visual acuity recovered to 6/6 in each eye.¹ Humphrey visual field analysis performed 6 months after the jump has, however, demonstrated a paracentral scotoma. In the case reported by Habib and Malik the patient's visual acuities improved from 6/60 to 6/12 right and from 6/9 to 6/5 left (12 weeks after the jump).² The patients reported by Jain and Talbot,³ and by Chan⁴ also appear not to have suffered any long term sequelae.

The report from van Rens adds another case to the literature. The patient, however, has not completely recovered the vision in the affected eye. The patient's visual acuity had been tested not long before the jump and was recorded as

'normal' (personal communication). Interestingly, in the previously reported cases the patients all had full body harnesses¹⁻³ — the patient described by van Rens was only anchored at the ankles. The compression of the chest by the harness may not be as significant as the rate of deceleration induced by the tensile strength of the bungee cord. The British Elastic Ropes Sports Association (BERSA) is a voluntary association of bungee jumping operators who promote safety and education. According to BERSA (personal communication) there are two different types of cord in use. There are latex cords which are specifically designed for bungee jumping and stretch to four times their length thus giving a gradual deceleration and there are shock cords which account for a more abrupt deceleration. They emphasise the importance of matching the jumper's weight to the elasticity of the bungee rope.

At present, guidelines are set by the health and safety executive, but no standards exist. It is important that the public is aware of the potential hazard.

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- 1 David DB, Mears T, Quinlan MP. Ocular complications associated with bungee jumping. *Br J Ophthalmol* 1994; 78: 234-5.
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- 3 Jain BK, Talbot EM. Bungee jumping and intracular haemorrhage. *Br J Ophthalmol* 1994; 78: 236-7.
- 4 Chan J. Ophthalmic complications after bungee jumping. *Br J Ophthalmol* 1994; 78: 239.

Infectious endophthalmitis after cataract surgery

EDITOR,—In the paper on infectious endophthalmitis after cataract surgery by Hughes and Hill,¹ the authors discuss the role of povidone iodine solution in preventing endophthalmitis. We question two statements: (1) 'the solution must remain in contact with the eye for several minutes', and (2) 'the ocular surface should be irrigated with saline before surgery as it has been suggested that endothelial toxicity may occur if significant amounts of povidone iodine enter the eye'. No references or evidence are given for these statements.

Povidone iodine solution is a potent antimicrobial agent. It kills most bacteria within 30 seconds² and thus the antiseptic need not remain in contact with the eye for several minutes.

Our experience is at variance with Hughes and Hill's recommendation to irrigate the eye with saline to remove the instilled povidone iodine because of fear of the solution entering the eye and causing endothelial toxicity. Firstly, we do not recommend irrigation of the eye with saline as part of the preoperative preparation (unless there is mucus or debris on the conjunctiva or cornea) because we found in our study that irrigation actually led to an increase in the number and species of bacteria on the conjunctiva.⁴

Secondly, as for the likelihood of causing endothelial toxicity if the instilled povidone iodine solution enters the eye, we feel this possibility is distinctly improbable. We invariably find that the solution instilled at the time of preoperative preparation disappears from the surface of the eye in a matter of a few minutes — certainly long before an incision is made into the eye. We further point out that



Figure 1 Fundus photograph right eye. Foveal oedema and intraretinal haemorrhage.

Wille⁶ found that when 20 to 30 drops of povidone iodine solution (instead of the one or two drops that we recommended) were placed in the conjunctival sac before surgery, there was no clinically significant effect on corneal thickness or endothelial cell count when compared with a control.

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- Hughes DS, Hill RJ. Infectious endophthalmitis after cataract surgery. *Br J Ophthalmol* 1994; 78: 227-32.
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Reply

EDITOR, — We thank Apt and Isenberg for their comments regarding our paper.¹

We are in agreement that 5% povidone iodine solution is a potent antimicrobial agent. In vitro, most bacteria are killed within 30 seconds to 1 minute.² However, Saggers' work also showed that at very high bacterial concentrations, several strains remained alive at 5 minutes and that the presence of organic material delays the rapid bactericidal effect.² While 5% povidone iodine solution significantly reduces the conjunctival bacterial population,^{3,4} it does not completely disinfect the conjunctiva. Therefore, it is probably not as effective clinically as the in vitro studies suggest. To our knowledge no study has shown the optimum duration of contact of 5% povidone iodine solution with the eye. In practice, if instilled during the preoperative preparation, the solution will remain in contact with the conjunctiva for several minutes.

Regarding irrigation of the eye; while we agree the saline irrigation alone increases the number of bacterial species isolated from the perilimbal area,⁵ Boes's study did not find an increase in bacterial isolates after preparation with 5% povidone iodine solution combined with saline irrigation when compared with the use of 5% povidone iodine alone. We feel that a few drops of solution normally used to moisten the cornea is probably adequate to remove residual povidone iodine, but in view of Boes's work⁴ it should be safe to irrigate more if deemed necessary.

The risk of endothelial toxicity resulting from povidone iodine inadvertently entering the eye has been repeatedly quoted for many years. We were aware of Wille's paper⁶ and had intended to qualify our statement on endothelial toxicity by adding that the risk remained theoretical rather than proved. We thank Apt and Isenberg for having pointed out this important omission.

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- Hughes DS, Hill RJ. Infectious endophthalmitis after cataract surgery. *Br J Ophthalmol* 1994; 78: 227-32.

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Horner's syndrome and Fuchs' heterochromic uveitis

EDITOR, — Several reports of Fuchs' heterochromic uveitis (FHU) accompanying both congenital and acquired Horner's syndrome can be found in the literature.^{1,2} However these cases are scarce, and the association remains in question. A sympathetic aetiology for FHU is unproved, and to date no convincing evidence exists.³

We report a case in which FHU and Horner's syndrome co-exist.

A 69-year-old white man presented with blurred vision in the right eye of rapid onset over the past 4 months. He had no other ocular symptoms. Systemically he was well except for a history of hypertension and an episode of vertebrobasilar insufficiency 10 years ago. Sixteen years earlier he was noticed to have a partial ptosis with a smaller pupil on the right side, and diagnosed as having Horner's syndrome clinically which was then confirmed pharmacologically. On examination his visual acuity was right counting fingers, and left 6/5. On the right he had a 4 mm ptosis, diffuse stellate keratic precipitates on the corneal endothelium, plus flare and plus cells in the anterior chamber, 2 mm miosis compared with the left, heterochromia iridis with iris stroma atrophy but no transillumination defects, no iris nodules, absence of posterior synechiae, and a moderate posterior subcapsular cataract. He had normal intraocular pressure, normal discs, and full fields. Both pupils responded normally to light and accommodation. Cocaine hydrochloride 4% failed to dilate the right pupil but fully dilated the left; however, the right pupil fully dilated after instillation of 1% phenylephrine.

This is a further report of Horner's syndrome occurring in association with FHU. Few such documented cases exist. This case differs from previously reported cases in that the diagnosis of Horner's syndrome was made some 16 years before the diagnosis of FHU. In most of the previously reported cases the diagnosis of Horner's syndrome was made in retrospect, once the signs of FHU were already present. Some authors⁴ have felt that such a diagnosis may be difficult or inaccurate because of the iris and pupillary changes which can occur in FHU itself, and the pharmacological tests for Horner's also become unreliable.

Whether this case illustrated a genuine association between FHU and Horner's syndrome remains unresolved, and the debate as to whether FHU has a sympathetic aetiology or not will continue.

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NOTICES

Keratoconus Self Help and Support Association

For some time keratoconus patients at Moorfields Eye Hospital have met as a self help and support group. On 10 March 1994 this was formally constituted as the Keratoconus Self Help and Support Association. Mr Roger Buckley, MA, FRCS, FRCOPhth, Director of Moorfields Contact Lens Department, accepted the Association's invitation to become its president. The Association aims to heighten awareness of keratoconus, its effects, and management, both within the medical and optical professions and generally. Funds are to be raised for publication of a pamphlet for this purpose. While the condition does not lead to blindness, for some the deterioration is such that a corneal transplant is the only option. Even then a contact lens may still be needed. There will be active support for research, regular meetings, and other activities including fundraising and a newsletter. Links are already being established with related societies and associations. Nor will the Association lose sight of its principal purpose, help and support for keratoconus sufferers. All keratoconus sufferers are welcome to join and associate membership is open to any interested non-sufferer. Further details: Mike Oliver (chairman), 39 Eversley Road, London SE7 7LF.

Office of Continuing Medical Education

A course entitled '1995 Update in the Management of Age-Related Macular Degeneration' will be held on 21 January 1995 at the Thomas B Turner Building, Johns Hopkins Medical Institutions, Baltimore, Maryland, USA, sponsored by The Wilmer Ophthalmological Institute of Johns Hopkins Medical Institutions. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical Education, 720 Rutland Avenue, Turner 20, Baltimore, MD 21205-2195, USA. (Tel: (410) 955-2959; Fax: (410) 955-0807.)

Photonics West '95

The International Society for Optical Engineering (SPIE) will hold a conference entitled 'Photonics West '95' on 4-10 February 1995 at the San Jose Convention Center, San Jose, California, USA. This meeting consolidates three established California meetings, OE/LASE, Biomedical Optics, and the IS&T/SPIE Symposium on Electronic Imaging Science and Technology. Further details: SPIE, PO Box 10, Bellingham, WA 98227-0010, USA. (Tel: 206/676-3290; Fax: 206/647-1445.)