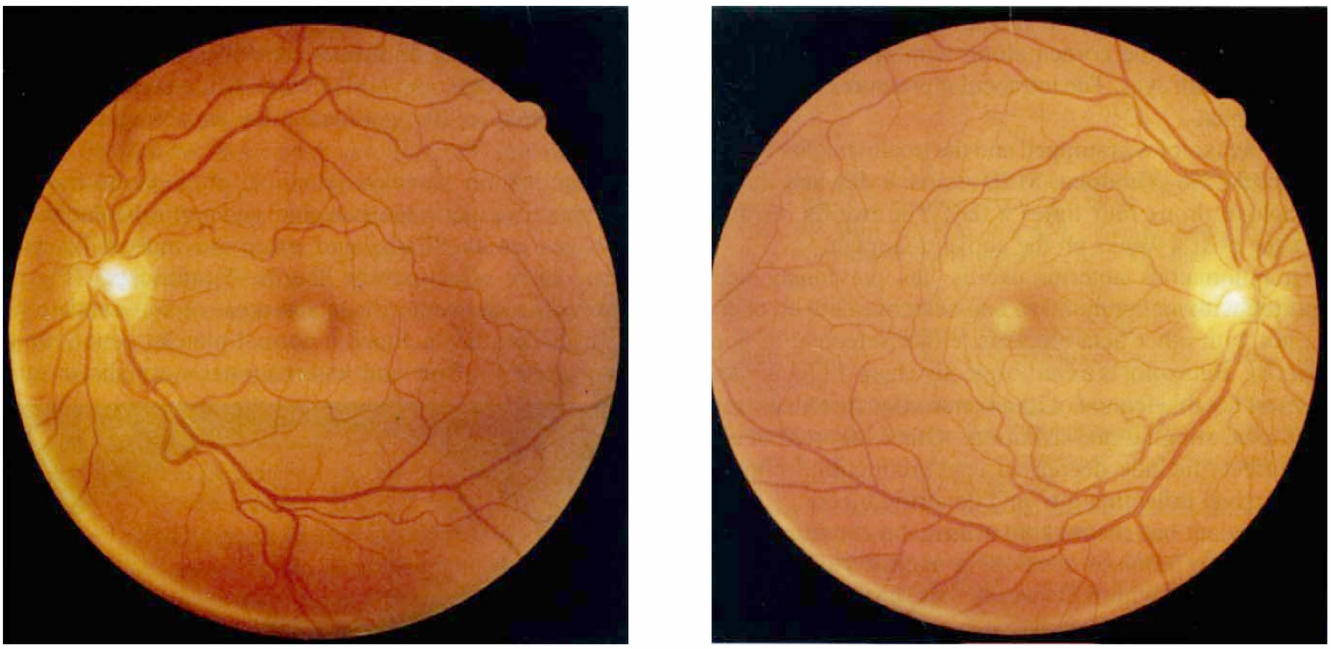

(a) (b)

Fig. 2. *Retinal photographs of the left (a) and right (b) eyes after 3 weeks of vitamin C treatment.*

(INR). The haemoglobin had fallen from 11.1 g/dl to 6.4 g/dl over 3 weeks with a normochromic picture. Serum ferritin, B_{12} and red cell folate were normal. The leucocyte ascorbic acid level was 42 nmol/10^8 WBC (reference range 60–300 nmol/10^8 WBC). A formal dietary assessment indicated that his daily vitamin C intake was very low at 4.5 mg (estimated average requirement 25 mg),[2] but that this was a remarkably selective deficiency and his folate intake was well above that recommended.

After vitamin replacement and dietary advice, his general health and retinal appearances returned to normal within 4 weeks (Fig. 2).

Comment

Scurvy in Britain is rare and now largely confined to elderly men in deprived circumstances, and drug addicts. Cases occur particularly in the spring because the vitamin C content of potatoes, an important source, wanes with overwinter storage.[3]

The main defect is impaired collagen synthesis in the basement membranes of capillaries. This leads classically to gum haemorrhages around teeth, perifollicular petechiae and bleeding into periostium, synovial joints and muscles. Death from cerebral haemorrhage is recorded.[3]

A variety of ocular changes in scurvy have been described[1] and include periorbital, conjunctival and retrobulbar haemorrhage, episcleritis and keratitis, central retinal vein thrombosis, papilloedema, optic atrophy and retinal detachment. Bloxham and colleagues[4] presented a case similar to ours with retinal haemorrhages and exudates. This case also had folate deficiency. In our case the satisfactory folic acid intake was due to the patient drinking beer.

We conclude that retinal haemorrhages and exudates are features of scurvy, presumably due to loss of integrity of the retinal vascular collagen support.

We would like to thank Mr Patrick Martin, FRCSE, for advice and Mrs Julie Hewitt for secretarial assistance.

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Sir,

Ocular Injury Resulting from Bungee-Cord Jumping

Three cases of serious ocular injuries resulting from the activity bungee jumping have been reported.[1–3] These comprised retinal haemorrhages in the form of a foveal haemorrhage,[1] an isolated retinal haemorrhage which broke through into the vitreous[3] and a type of Purtscher's traumatic angiopathy.[2] In these case reports the vision improved to 6/12 or better in the affected eyes.

We have encountered two further cases of ocular injury resulting from a bungee jump and in one case the visual loss was permanent.

Case Reports

Case 1. A 21-year-old man sustained a foveal haemorrhage following a jump. The visual acuity was reduced to 6/60 and had not improved when examined 18 months later. When the fundus was re-examined at this time atrophy was evident at the fovea and the remains of foveal haemorrhage was still evident.

Case 2. A 40-year-old woman who had diabetes of 6 years' duration that was treated with Humulin S (Lilly) 11 units in the morning and Humulin Initard (Nordisk Wellcome) 13 units in the evening developed headache and pain behind the eyes following a jump. This persisted and 6 days later she developed horizontal diplopia. She presented to the Eye Department 3 days later and was noted to have a small exophoria with slow recovery to binocular single vision. Nystagmoid jerks were noted on versions. A Hess chart demonstrated a small underaction of the right lateral rectus. There was no other ocular abnormality and both fundi were free of retinopathy.

Discussion

The rise in intravascular pressure within the head during a jump arises from a number of factors. Breath-holding and tensing abdominal muscles have been considered important.[2,3] The *G* forces at the end of the descent have been estimated to be 2.5–3.0 G^4 but could be higher (e.g. 7–8 *G*) in certain cases.[5] This is near the limits of tolerance as sustained by NASA astronauts on spacecraft takeoff.[6]

Current operator regulations exclude diabetics from bungee-cord jumping; however, it would appear that participants are not always aware of this exclusion.

The occurrence of serious ocular injuries with permanent impairment of vision gives rise to concern and participants should be aware of the risks involved. Further measures should be instituted to ensure that no diabetic patients participate.

We wish to thank Mrs M. Wardrop, Senior Orthoptist, Broomfield Hospital, for orthoptic assistance.

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Sir,

Retinal Vasculitis and Uveitis in IgA Nephritis

Retinal vasculitis with uveitis in IgA nephritis is reported for the first time to our knowledge.

Case History

An 80-year-old man presented to our eye unit in July 1993 with progressive painless reduction in vision in his right eye over 1 year and in his left eye over 2 months. He had not had previous eye problems.

Angina and peripheral vascular disease had been diagnosed 6 years previously and he had a long history of peptic ulceration and knee osteo-arthritis.

The patient developed acute renal impairment with a creatinine level of 475 μmol/l, nephrotic syndrome, anaemia (haemoglobin 93 g/l) and purpuric rash in January 1993 (6 months prior to presentation to our unit), and upper gastrointestinal bleeding in February 1993.

Gastroscopy showed a small hiatus hernia and mild gastritis. Examination did not show any evidence of neurological, pulmonary, malignant or infectious disease. He did not have abnormal C or P antineutrophil cytoplasmic antibodies.

Histology

Skin biopsy showed perivascular dermal neutrophil polymorphonuclear infiltrates with disruption of vascular architecture and copious numbers of extravasated erythrocytes and disrupted polymorph nuclear remnants (leucocytoclastic vasculitis). No direct immunofluorescence was detected, and the patient did not have thrombocytopenia.

Renal biopsy at the same time showed mesangial proliferation with vasculopathy and interstitial fibrosis. There was interstitial enlargement with mild fibrosis and subendothelial hyalinosis of arterioles and small arteries. There was no evidence of large vessel arteritis, tubular involvement or crescents. The renal biopsy did not show any significant cellular infiltrate. Immunofluorescence showed signs of active IgA nephritis including extensive granular IgA deposits together with IgM, C3 and fibrin in the mesangium and on basement of all glomeruli examined, whilst the blood vessels did not demonstrate immunofluorescence.

Systemic Response to Treatment

The patient responded well to intravenous methylprednisolone 500 mg and azathioprine 150 mg daily for 3 days. Azathioprine was then discontinued because of leucopenia, and the nephrotic syndrome improved on decreasing doses of oral prednisolone (slowly tapered