

sharp edge of the probe cutting conjunctival vessels and causing bleeding. Thin adherent debris was then carbonized, allowing the laser tip temperature to rise and causing scleral perforation. The defect required suturing. This led to the redesigning of the laser probe tip. In our case, there was no such carbonized debris seen and we think it is unlikely to be the reason for our perforation.

Sabri and Vernon<sup>10</sup> reported a case of scleral perforation using the new contact G-probe. The defect required suturing with two 10-0 vicryl sutures. However, 1 week later, the scleral leak recurred and further suturing was needed.

In our case, with crepe bandage and oral acetazolamide for 1 day, we were able to stop the leakage. During subsequent follow-up, the scleral hole healed and was covered by intact conjunctival epithelium. This demonstrated that suturing may not always be necessary, especially when the perforation is small.

Pre-existing scleral thinning is a common risk factor in the two previously reported cases, and also in our patient. Hence, Sabri and Vernon<sup>10</sup> suggested the use of a lower laser power setting (50%), though there is no good proof that such a lower power could prevent perforation and is still as effective.

With heightened awareness of this complication, and appropriate management when it occurs, we believe the risk of scleral perforation and its consequences could be minimized.

## References

- Mastrobattista JM, Luntz M. Ciliary body ablation: where are we and how did we get here. *Surv Ophthalmol* 1996; **41**: 193–213.
- Gupta N, Weinreb RN. Diode laser transscleral cyclo-photocoagulation. *J Glaucoma* 1997; **6**: 426–429.
- Threlkeld AB, Johnson MH. Contact transscleral diode cyclophotocoagulation for refractory glaucoma. *J Glaucoma* 1999; **8**: 3–7.
- Kosoko O, Gaasterland DE, Pollack IP, Enger CL. Long-term outcome of initial ciliary ablation with contact diode laser transscleral cyclophotocoagulation for severe glaucoma. The Diode Laser Ciliary Ablation Study Group. *Ophthalmology* 1996; **130**: 1294–1302.
- Bock CJ, Freedman SF, Buckley EG, Shields MB. Transscleral diode laser cyclophotocoagulation for refractory pediatric glaucomas. *J Pediatr Ophthalmol Strabismus* 1997; **34**: 235–239.
- Bloom PA, Tsai JC, Sharma K, Miller MH, Rice NS, Hitchings RA *et al*. 'Cyclodiode': trans-scleral diode laser cyclophotocoagulation in the treatment of advanced refractory glaucoma. *Ophthalmology* 1997; **104**: 1508–1520.
- Schlote T, Derse M, Thiel H, Benedikt J. Pupillary distortion after contact transscleral diode laser cyclophotocoagulation. *Br J Ophthalmol* 2000; **84**: 337–339.
- Bhola RM, Prasad S, McCormick AG, Rennis IG, Talbot JF, Parsons MA. Pupillary distortion and staphyloma following trans-scleral contact diode laser cyclophotocoagulation: a clinicopathological study of three patients. *Eye* 2001; **15**: 453–457.
- Gaasterland DE, Pollack IP. Initial experience with a new method of laser trans-scleral cyclo-photocoagulation for ciliary ablation in severe glaucoma. *Trans Am Ophthalmol Soc* 1992; **90**: 225–246.
- Sabri K, Vernon SA. Scleral perforation following trans-scleral cyclodiode. *Br J Ophthalmol* 1999; **83**(4): 502–503.
- Schlote T, Derse M, Rassmann K, Nicaeus T, Dietz K, Thiel HJ. Efficacy and safety of contact transscleral diode laser cyclophotocoagulation for advanced glaucoma. *J Glaucoma* 2001; **10**: 294–301.

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## Sir, Vitreous haemorrhage following cardiopulmonary resuscitation

Vitreous haemorrhage is known to occur by a wide variety of mechanisms. We present the case of a 27-year-old gentleman who developed a vitreous haemorrhage following cardiopulmonary resuscitation (CPR). We postulate that this occurred as a result of the chest compressions performed, through a mechanism similar to valsalva retinopathy. To our knowledge this is the first reported case of vitreous haemorrhage arising in this way.

### Case report

A 27-year-old man presented to the accident and emergency department with sudden onset of severe retrosternal chest pain. There was no relevant past medical or ocular history and no previous history of chest pain. He was a cigarette smoker. Initial electrocardiography (ECG) showed ST segment elevation in the anterior chest leads. Shortly afterwards the patient collapsed on a trolley having had a ventricular fibrillation cardiac arrest. He was shocked into asystole and after 1 mg of adrenaline intravenously and CPR, he regained a perfusing rhythm. He was transferred for cardiac angiography, which showed 100% occlusion of his left anterior descending (LAD) coronary artery. The decision was taken not for thrombolysis but angioplasty instead. A proximal occlusion of the LAD was readily canalised and two stents were inserted. The patient was then transferred to the intensive care unit where he received intravenous dobutamine and noradrenaline for resuscitation of presumed stunned myocardium.

Following extubation 8 days later he complained of blurred vision in his right eye. He was subsequently reviewed in the ophthalmology department. Snellen visual acuities were 3/60 right eye and 6/4 left eye. He was found to have a dense vitreous haemorrhage and no fundal view in the right eye. Ultrasound revealed a flat retina. The haemorrhage failed to resolve and 3 months following myocardial infarction he underwent right vitrectomy. At surgery, dilatation of peripapillary vessels with adherent vitreous haemorrhage was noted, but no other retinal abnormalities. He made an uneventful recovery and postoperative visual acuity in his right eye was 6/6. Dilated funduscopy since the surgery has not revealed any associated pathology.

### Comment

This case describes a presumed iatrogenic vitreous haemorrhage. In the absence of contributing ocular pathology, the only causative factor we can identify is the cardiac compressions the patient received during his resuscitation.

Chest compressions during CPR are known to cause several complications, for example rib fractures, pulmonary haemorrhages and liver lacerations. However, ocular complications are more unusual. Chest compression by safety belt in automobile accidents is known to cause traumatic retinal angiopathy (Purtscher's disease) – even from a minor compression injury from a lap-shoulder belt<sup>1</sup> – and retinal detachment.<sup>2</sup> We also note the recent report by Chandra *et al*<sup>3</sup> of a case of Purtscher's retinopathy in one eye, and Valsalva retinopathy in the other, following a compressive chest injury.

The mechanism and efficacy of chest compressions as part of CPR have become better understood in recent years. There has been deliberation as to whether sternal compression generates a direct increase in arterial forward flow by cardiac compression, or whether it is the relaxation phase that encourages venous return through negative intrathoracic pressure. However, it is now generally thought that both mechanisms are acting to provide some cerebral perfusion, though the flow generated is insufficient to force vessel aneurysm or rupture. Babbs<sup>4</sup> used a mathematical model to show that even the most efficient compression technique would only generate a maximum of 3.1 l/min and 58 mmHg of systolic perfusion pressure. Pinning *et al*<sup>5</sup> confirmed this by performing transoesophageal echocardiography during closed-chest CPR, in cardiac arrest. Peak forward aortic flow was measured at  $58.8 \pm 11.6$  cm/s; less than half of normal peak aortic flow.

Valsalva retinopathy is a well-described phenomenon. A preretinal haemorrhage in the macula area is the usual finding at presentation. The cause is thought to be a rupture of a retinal vessel, following physical exertion – usually a sudden and rapid rise in intrathoracic pressure (such as when coughing, heavy lifting or straining at stool). This is thought to cause a rise in the intravenous pressure, then a rise in retinal vessel intraluminal pressure, with consequent rupture of a retinal venule or capillary, perhaps at the site of a pre-existing vessel wall weakness.<sup>6</sup> Extension of the haemorrhage into the vitreous is rare, but is thought to occur if the force of the intrathoracic pressure rise is sufficiently high<sup>7</sup> or in the presence of a blood disorder such as sickle cell disease.<sup>8</sup>

In our case, we can rule out other causes of vitreous haemorrhage. There was no previous ocular pathology, no retinal pathology was identified during vitrectomy surgery and the patient had not been thrombolysed, or given any anticoagulant therapy. We therefore hypothesise that this patient's vitreous haemorrhage was due to the chest compressions received during CPR, via a mechanism similar to Valsalva retinopathy.

### References

- 1 Hoare GW. *Br J Ophthalmol* 1970; **54**(10): 667–669.
- 2 Oksala A. Traumatic retinal angiopathy and retinal detachment as result of traumatic compression of the thorax. *Nord Med* 1958; **59**(4): 142–145.
- 3 Chandra P, Azad R, Pal N, Sharma Y, Chhabra MS. Valsalva and Purtscher's retinopathy with optic neuropathy in compressive thoracic injury. *Eye* 2005; **19**: 914–915.
- 4 Babbs CF. CPR Techniques that combine chest and abdominal compression and decompression. *Circulation* 1999; **100**(21): 2146–2152.

- 5 Pinning LIU, Yan GAO, Xiangyang FU, Junhao LU, Ying ZHOU, Xianglong WEI *et al.* Pump models assessed by transoesophageal echocardiography during cardiopulmonary resuscitation. *Chin Med J* 2002; **115**(3): 359–363.
- 6 Kadrmas EF, Pach JM. Vitreous haemorrhage and retinal vein rupture. *Am J Ophthalmol* 1995; **120**(1): 114–115.
- 7 Jones WL. Valsalva maneuver induced vitreous haemorrhage. *J Am Optom Assoc* 1995; **66**(5): 301–304.
- 8 Konotey-Ahula F. Valsalva vitreous haemorrhage and retinopathy in sickle cell haemoglobin C disease. *Lancet* 1997; **349**(9067): 1774.

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Sir,  
**An unusual case of proptosis**

We report a rare case of orbital xanthoma in a 38-year-old man presenting as droopy eyelid and proptosis. The only clue to the diagnosis was the presence of eyelid xanthelasma.

#### Case report

A previously well 38-year-old Bangladeshi labourer presented with a 2-month history of worsening right ptosis, proptosis, and dull ache over the forehead (Figure 1). Examination revealed a right non-axial proptosis which measured 23 mm on Hertel's exophthalmometer (compared with 21 mm on the left). The right palpebral fissure measured 5 mm (compared with 7 mm on the left). The right upper eyelid had an area of xanthelasma in the nasal aspect; otherwise, it is not erythematous or tender to palpation. The ocular movement was normal except for dull ache and slight restriction on upgaze. The vision was normal in both eyes. An urgent CT scan revealed a mass which appeared

to involve the superior rectus and levator superioris complex (Figure 2). A possible diagnosis of myositis was made and he was started on oral 100 mg diclofenac b.d. The blood tests for autoimmune antibodies, ESR, and thyroid function tests were normal. The only abnormal result was raised cholesterol level of 6.2 mmol/l. There was no clinical improvement after 2 weeks. A MRI scan was performed to elucidate the lesion. This showed the mass to be distinct from the superior rectus and appeared to involve only the levator superioris (Figure 3). An open biopsy through the skin crease revealed an uncapsulated orange coloured lesion between the orbicularis oculi and the orbital septum with infiltration of the levator aponeurosis (Figure 4). The lesion had a rubbery consistency and extended posteriorly beneath the orbital roof. The mass was removed *en bloc*. It had a uniform yellow surface



**Figure 1** A 38-year-old Bangladeshi labourer with right eye ptosis and proptosis.



**Figure 2** A CT coronal scan of the orbit revealed a mass at the right eye superior rectus and levator superioris complex.