The iris root was displaced into the bleb through the break and incarcerated, thus blocking the aqueous humor outflow and causing the IOP to rise rapidly, which is one of the complications of NPTS.[1]

UBM can clearly image the anterior segment of the eye, so it is widely used to evaluate the bleb and to explore the potential reasons for failure of the bleb after NPTS.[2,3] However, in this case, from the UBM picture, we could not identify the iris root location and its relationship with ciliary. In addition, there was a strong reflective cycle in the bleb, which could easily be misinterpreted as a bleb encapsulation. However, an encapsulated bleb would not be associated with an acute increase in IOP, as encapsulation is a slow process. In combination with the supplementary clinical examination results, we concluded that the strong reflective cycle was actually the incarcerated iris. To the best of our knowledge, this exceptional phenomena has not been reported before.

NPTS, which does not enter the anterior chamber during the operation, and in the absence of an iridectomy, would ensure little postoperation inflammation. However, a small percentage of patients were observed with an increasing IOP at prolonged periods after NPTS, which may be due to rupture of the trabeculo-decemet's membrane or adherence of iris root to the membrane.[1] These complications, followed up with NPTS, were induced by a consistent existing pressure difference between the upper and lower iris surface after the surgery. Therefore, it is worth considering to perform a laser iridectomy at the surgical area, before the NPTS, as a means of preventing these surgical complications.

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Sir,

**Argon laser photocoagulation for diabetic macular oedema**

### *Case report*

The Early Treatment of Diabetic Retinopathy Study (ETDRS) showed that laser photocoagulation reduced moderate visual loss (MVL) in patients with clinically significant diabetic macular oedema (CSME).[1] The recent UK National Diabetic Retinopathy Laser Audit showed some effect with laser treatment, otherwise there is little data from UK units.[2–4] We reviewed the outcome of patients treated for CSME in the Southampton Eye Unit.

All the patients referred with CSME in 1998 from our screening service were included. The Snellen visual acuity (converted to LogMAR),[5] number of treatments, and fluorescein angiographies performed were recorded. The patients were followed for 3 years, and the proportion of patients with MVL at 1, 2, and 3 years was compared to ETDRS data (Figure 1).

One-hundred and thirty eight eyes from 106 patients with untreated diabetic maculopathy received laser treatment. The average visual acuity at referral was between 6/7.5 and 6/9. Only four eyes developed proliferative diabetic retinopathy during the period audited. Ten eyes were excluded for coexisting ocular pathology; seven eyes underwent cataract surgery, two had vitrectomies, and one eye amblyopia. MVL occurred in 5.1% of patients at 1 year, 8.2% at 2 years, and 14.7% at 3 years. There was no statistically significant difference in

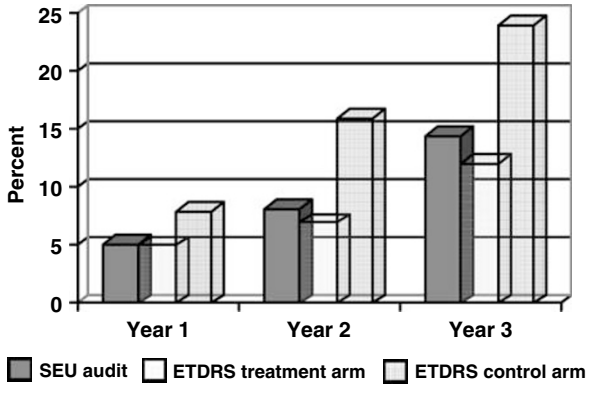


**Figure 1** Graph showing incidence of MVL.

MVL between the ETDRS immediate treatment arm and our patients ($P = 0.11$). No patient underwent fluorescein angiography.

**Comment**

The recent UK audit of diabetic maculopathy treatment found that 9.2% of patients doubled their visual angle by 9 months.[2,3] Our outcomes were more positive, possibly as a result of these being newly diagnosed cases. This was achieved without fluorescein angiography. There is little evidence that patients having fluorescein angiograms have better acuity outcomes than individuals treated on clinical grounds alone. A randomized controlled trial to conclusively demonstrate visual benefit would be prohibitively expensive.[6] Could other noninvasive methods of assessing macular morphology such as optical coherence tomography be used to target laser treatments in the early stages of the disease?

At present, UK audit standards for diabetic maculopathy treatment require assessment of waiting times and access to treatment but not of the visual outcome. This audit demonstrates that measurement of visual outcomes can be achieved, and are essential for the assessment of retinal services, from screening to treatment.

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Sir,

**Horner's syndrome: an atypical presentation in a child with neurofibromatosis type 2**

Atypical manifestation of neurofibromatosis type 2 (NF2) in a 10-year-old boy was recently reported in *Eye*.[1] I would like to present another 10-year-old child with the atypical presentation of Horner syndrome. Although a diagnosis of NF2 is most often made in adults, both cases highlight that onset is also possible in childhood. Further, eye manifestations may be the presenting symptoms.

***Case report***

Seven years ago, a 10-year-old girl was referred for ophthalmic opinion as part of a general investigation because of heredity of NF2 in her father and half-brother. At that time there was no multidisciplinary follow-up program of relatives of NF2 patients in our hospital and her eyes had not previously been examined. Audiology examination had not revealed any vestibular schwannomas. She had no eye complains. Visual acuity was normal (6/6) both eyes. The anterior segment was normal in her right eye, but there was a posterior subcapsular opacity in her left eye, not affecting the visual acuity. No Lisch noduli were found. Horner's syndrome was noted in the left eye with a moderate ptosis and an anisocoria, most prominent in dark. Both fundi were normal. She had no strabismus.

The girl was investigated by the paediatric neurologists, and a magnetic resonance imaging (MRI) scan revealed an intraspinal/thoracic tumour with an extension from CVI to ThII and extrapleurally left side (Figure 1). The tumour was operated in two sessions when she was 11 years old. Histological examination showed neurofibroma. Minor vestibular schwannomas were detected at 15 years of age and are followed up, but still not operated at the age of 17 years.

The girl is followed up regularly by audiologist, paediatric neurologist, and ophthalmologist. Her posterior subcapsular cataract remains similar and she still has a normal visual acuity in both eyes.

NF2 is an autosomal, dominantly inherited disease and is associated with mutations in the NF2-gene, localized to chromosome 22.[2] It is characterized by bilateral