Numerous subretinal hypopigmented tracks with small haemorrhages were noted in the superior retina, and perivascular haemorrhages were observed around the inferior branch of the central retinal vein (Fig 2). Fluorescein angiography showed numerous hyperfluorescent tracks without dye leakage. The patient’s blood test revealed a slightly elevated white blood cell count (10 200 × 10^9/l; normal <9000) and elevated IgE in the serum (680 U/ml; normal <250).

On the following day pars plana vitrectomy was performed and the worm was found partially migrated into the subretinal space of the macula. The worm was aspirated successfully through a 20 gauge silicone tipped needle and submitted for parasitological study; however, it was lost during transportation to a different laboratory. Parasitological study of the patient’s serum detected an antibody to *Dirofilaria*. Six months after surgery, the patient’s visual acuity was still 20/50. Ophthalmoscopic examination disclosed a white worm, approximately 3 disc diameters in length, equivalent to about 4.5 mm, moving slowly in the macula at the epiretina. Cone and rod electroretinograms to full field stimuli were normal bilaterally.

**COMMENT**

Our patient owned two dogs, and her ocular findings could be differentiated from toxocariasis, in which only larvae of *Toxocara canis* can infect humans. The length of the *Toxocara* larva is about 400 µm, and grows no longer. Although no direct microscopical evidence of *Dirofilaria* infection was obtained in our patient, a positive antibody to *Dirofilaria* in the serum indicated its infection. There have been many reports of intraocular filariasis; however, the filariae were removed and identified in only six of 56 cases reviewed by Beaver in 1989. Direct parasitological microscopic examination is necessary for accurate diagnosis, but serological study can be helpful, as shown in the present case.

Various types of management for intraocular parasites have been reported. Direct photocoagulation to the worm body has been successfully reported in cases with filaria-like worms and in one case with insect parasites. It has been suggested that photocoagulation denatures the parasite proteins and mitigates the immune reaction. If the parasite is located in the posterior pole of the retina, however, photocoagulation may cause permanent visual impairment, and surgical removal should be selected. Furthermore, photocoagulation would make parasitological identification impossible. Preretinal or subretinal parasites were retrieved successfully by pars plana vitrectomy in several reported cases.

In the present case, the visual acuity was still 20/50 6 months postoperatively. The migrating worm may have caused considerable damage to the macular function, therefore, we believe that intraocular parasites should be removed as soon as possible.

The authors are grateful to Dr Keizo Yamaguchi for parasitological examination and Mr Shingo Yamazaki for fundus photographs.

The cornea showing diffuse punctate staining with linear epithelial infiltrates.

**Surgically removed submacular nematode**

**EDITOR**—Intraocular filariasis is an uncommon disease in civilised countries. In only a few cases has the worm been surgically removed from the posterior portion of the eye and identified. In this report we describe a patient who had a filaria-like worm in an epimacular and/or submacular lesion, which was successfully removed surgically.

**CASE REPORT**

A 38 year old woman complained of decreased visual acuity in her left eye of 5 days’ duration. Her medical history was unremarkable. The patient had two pet dogs, and she had not travelled to foreign countries in recent years. On examination, her corrected visual acuity was 20/20 in the right eye and 20/100 in the left. No inflammation was present in the anterior segment or vitreous cavity bilaterally. Ophthalmoscopic examination disclosed a white worm, approximately 3 disc diameters in length, equivalent to about 4.5 mm, moving slowly in the macula at the epiretina. Epiretinal and intraretinal haemorrhages were observed in and around the macular region. The body of the worm was tapered at one end and slightly rounded at the other (Fig 1). A round, preretinal haemorrhage was observed on a branch of the superonasal retinal artery, which could have been the route of entry into the eye.

**Figure 1** Fundus photograph of the left posterior pole demonstrates a white worm and preretinal haemorrhage.

**Figure 2** Fundus photograph of the left eye shows a white worm at the macula, a round, preretinal haemorrhage along the superonasal retinal artery, and numerous subretinal hypopigmented tracks in the superior retina.
Corneal epithelial cells were strongly suspected. Corneal epithelium was removed for microscopy, culture, and histology. Her contact lens case and solutions were also sent for culture. Acanthamoeba was cultured from both corneal epithelium and lens case, but not the lens solutions. She was treated with topical polyhexamethylene biguanide 0.02% (PHMB) and prednisolone 0.3% as well as oral fluoro-
profen (Frobén, Knoll Ltd, Nottingham). Six weeks after diagnosis she was asymptomatic with a visual acuity of 6/9. The eye was quiet, though some corneal infiltrates remained.

COMMENT
Acanthamoeba keratitis is an uncommon but potentially devastating condition. The number of cases diagnosed in the United Kingdom has steadily risen over the past 20 years owing to increased contact lens wear. A case control study by Radford et al. found that daily wear disposable contact lenses, in which the lens is discarded after 1 day’s wear only, is an uncommon but potentially devastating condition. The number of cases diagnosed in the United Kingdom has steadily risen over the past 20 years owing to increased contact lens wear. A case control study by Radford et al. found that daily wear disposable contact lenses, in which the lens is discarded after 1 day’s wear only, is an uncommon but potentially devastating condition. The number of cases diagnosed in the United Kingdom has steadily risen over the past 20 years owing to increased contact lens wear. 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Clinical course of acute zonal occult outer retinopathy

EDITOR—Patients with acute zonal occult outer retinopathy (AZOOR) may present with a normal fundus examination and almost normal fluorescein angiography (FA), despite severe loss of visual field and electroretinogram (ERG) abnormalities. There is no specific evidence for an immune abnormality. There is also no established conclusion about progression of visual field loss. With a multifocal ERG (m-ERG), a large number of retinal locations can be stimulated simultaneously and local responses can be extracted independently in a single recording session. High resolution topographic mapping of retinal function also is possible. A previous report indicates its efficacy in the diagnosis of AZOOR. There are no reports about the clinical course. Thus, using m-ERG and static perimetry (Humphrey 30–2), the alteration of retinal function in a clinical course of a patient with AZOOR was investigated.

CASE REPORT
A healthy 60 year old woman presented to our outpatient clinic complaining of acute onset of visual disturbance in her left eye. Her corrected visual acuity was 20/20 in her right eye, and 20/100 in her left. The pupils were equal and reactive normally. Silt lamp and fundus examination, computed tomography, magnetic resonance imaging scan, and general examination were normal. HVF 30–2 demonstrated blind spot enlargement breaking out to the inferotemporal periphery in the left eye (Fig 1, top). Full field ERG showed grossly reduced A and B waves in the left eye. The FA showed slight leakage from peripapillary capillaries. Indocyanine green (ICG) angiography showed slight hypofluorescence of the macular area at a late phase. Analysis of the mean deviation in HVF over the clinical course corresponded with the visual acuity (Fig 1, bottom). In addition, m-ERG (Veris III, Tomey, Nagoya, Japan) was analysed during the clinical course. In this examination, the fundus was divided into four foci and the sum of amplitudes in each group was measured (Fig 2, top). In the left eye, the sum of amplitudes was altered individually but all of them were affected during the clinical course. Only in the inferotemporal area did it correspond with HVF. The values in the right eye were about 5000 μV in each focus. This is almost the same as normal volunteers in our clinic (data not shown).

COMMENT
AZOOR may be precipitated by various retinal disorders and is characterised by rapid visual field loss which cannot be explained by the ophthalmoscopic changes resulting from the initiating disease. The ERG is abnormal, indicating that the field loss is due to retinal dysfunction. The cause of the acute damage to sharply defined zones of the retinal receptor cells in the absence of visible fundus changes in patients with AZOOR is unknown. In some previous reports, an apparent response to corticosteroid therapy suggested that an inflammatory and perhaps an immune reaction may play a part in the disease; however, there is no specific evidence for an immune abnormality. An infectious aetiology could also be the cause of AZOOR.

Figure 1 (Top) Ratio images of Humphrey 30–2 visual fields in the left eye. (Bottom) The relation between clinical course and mean deviation (MD) of Humphrey 30–2 visual field and visual acuity in the left eye. Asterisks indicate the day in which multifocal ERG was analysed. The roman numerals correspond with raw images in the upper part of the figure.
The findings of AZOOR that we observed in our patient suggest that the retinal recovery assessed by m-ERG was different for the inferotemporal retina and was delayed compared with visual acuity and HVF. Perhaps this delayed retinal recovery reflects a subtle microcirculatory disturbance that can not be clearly detected by FA or ICG. Slight leakage in FA and slight hypofluorescence on the late phase in ICG would suggest such a microcirculation disorder.

When better understanding of the aetiology and pathophysiology of AZOOR is available, the clinical response to appropriate therapy may perhaps be best followed by m-ERG.

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Hypotonic maculopathy following pneumatic retinopexy: a UBM study

EDITOR,—Pneumatic retinopexy is a promising surgical alternative in selected cases of retinal detachment.

We report a rare complication, to our knowledge the first ever reported, of this procedure—namely, hypotonic maculopathy, caused by a wound dehiscence in a patient who had previously undergone a standard extracapsular extraction with posterior chamber implant. Ultrasound biomicroscopy (UBM) proved to be a valuable adjunct in both the diagnosis and management of this complicated case.

CASE REPORT

A 63 year old white man, complaining of a shadow in his right visual field, was referred to our department in April 1997 for further management. Ocular history was notable for bilateral extracapsular cataract extraction with posterior chamber lens implantation (PC/ IOL), right eye in January 1997 and left eye in June 1996. Visual acuities were 20/25 in both eyes. IOPs were within normal limits. Anterior segment examination was unremarkable with well positioned IOLs. Fundal examination of his right eye revealed a superotemporal macula on rhegmatogenous retinal detachment, produced by a single horseshoe retinal break around 10 o’clock. After the risks and benefits of pneumatic retinopexy were explained to the patient he chose to have the procedure.

Cryopexy was applied to the tear and after an anterior chamber paracentesis was performed 0.7 ml of 100% SF, gas was injected into the vitreous cavity 3.5 mm behind the limbus inferotemporally.

He tolerated the procedure well and made an excellent recovery with the retina totally attached. He maintained a 20/30 visual acuity right eye. IOP remained within normal limits until 3 months later. At that time an IOP of 6 mm Hg and a deterioration of visual acuity to 20/70 was noted. Initially this visual loss was attributed to a posterior capsule opacification and a YAG capsulotomy was performed.

Between October 1997 and November 1997 he was presented to our unit on three occasions complaining of further deterioration of vision to 20/200, IOPs between 3–5 mm Hg, and a full blown hypotonic maculopathy. After 3 months of hypotony of "unknown origin", a diagnosis was made by a glaucoma specialist using indentation gonioscopy revealing a dehiscence of the cataract wound. A preoperative UBM study confirmed the presence of an internal wound gap, behind the limbus, superotemporally, 3 mm in circumference. Ultrasonically the wound dehiscence was depicted as a narrow slit (Fig 1) with a flat inadvertent bleb above which was not apparent clinically. A surgical repair of the wound was decided upon. Intraoperatively no definite dehiscence could be clinically identified. Balanced salt solution through a 30 gauge needle was repeatedly injected under pressure to the anterior chamber but this failed to localise any suspicious area. At this point, based on the ultrasonic study, two 10-0 nylon interrupted sutures were placed through the sclera parallel to the limbus in the suspicious area. These bites were moderately deep in an attempt to engage the internal flap of the cataract wound.

Two weeks postoperatively, the pressure normalised (IOP 12 mm Hg), maculopathy was reversed, and visual acuity improved to 20/50.

Hypotonic maculopathy is an unusual and, to our knowledge, the first reported complication of this kind after pneumatic retinopexy. We hypothesise that the original cataract wound did not heal properly and the additional cryoprobe manipulation caused the dehiscence.

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wound to leak. The UBM study provided us with an interesting insight into how the scleral suture repair may have worked to correct the wound leak as shown in Figure 2. The fact that hypotony resolved after suturing the wound, indicated that the external part of the wound was secure (no slit is apparent) despite the fact that the internal part of the wound was gapping even more postoperatively. This finding implies, therefore, that only minimal overall alteration of the wound architecture postoperatively, sufficient to rectify the leak.

We believe that UBM is a valuable adjunct in the management of similar cases by clearly identifying both the presence and exact location of leak. Finally, pneumatic retinopexy should be performed with caution, especially in cases of previously operated eyes with large incision wounds.

**COMMENT**

Involvement of the lens exclusively, sparing other ocular structures is rare. This case documents such a possibility and also highlights the salient features involving electric trauma to the lens. The scarlet burn in this case represents the entrance wound for the electrical energy but the lack of an exit wound makes this case particularly peculiar. Both entry and exit sites for the electric current have been reported by all previous authors.

The excellent surgical results noted in both eyes of this patient are in keeping with the similar result reported by Portellos et al. This observation should encourage the ophthalmologist to undertake surgery for electric cataract, where necessary, without any undue concern.

**Bilateral electrical cataract**

**EDITOR—**Electric trauma is not uncommon in India where majority of the population lives in the rural setting. Few cases of electric cataract have been reported in literature probably because few patients survive the high voltage of current that induces cataract formation. Most patients with electric cataract have no subjective complaints early on but become aware of the reduced visual function several months later.  

**CASE REPORT**

A 26 year old man complained of gradual and painless diminution of vision in both eyes, right more than left, for the past 2–3 years. There was a history of an overhead high tension electric transmission cable accidentally falling on the patient’s head 4–5 years earlier. This had resulted in immediate burn to the scalp. The patient was visually asymptomatic till about 1 year after the mishap, when he began to notice the gradual fall in vision that had progressed to its present state.

Corrected visual acuity was 20/200 right eye and 20/60 left eye. A 15 × 2.5 cm linear, sagittal scar extending from the frontal to the occipital region of the head was noticed. The lids, conjunctiva, cornea, and pupils showed no abnormality in either eye. Fundus examination was unremarkable. Slit lamp examination revealed multiple, mid-peripheral snowflake-like anterior subcapsular lens opacities in both eyes, right greater than left. In the right eye some of these opacities were seen encroaching into the visual axis and additionally a few posterior subcapsular opacities were noticed (Fig 1).

In view of the history of electrical injury and classic location and typical appearance of the lens opacities, a diagnosis of bilateral electric cataract was made. Extracapsular cataract extraction (ECCE) with posterior chamber intraocular lens implantation was undertaken in both eyes, right eye first followed 3 months later in left eye. The intraoperative and postoperative course were uneventful and the patient has achieved corrected visual acuity of 20/20 in both eyes.

**References**


**Figure 1** Cross section through temporal artery showing disrupted internal elastic lamina (arrow) and large calcific plaques (C) in the tunica media (haematoxylin and eosin; original magnification ×40).
nal elastic lamina should not be erroneously interpreted as sequelae of previous arterial inflammation. The pathophysiology of Monckeberg's arteriosclerosis is still unclear, but it can be induced in animal models by injecting adrenalin, nicotine, parathyroid hormone, and vitamin D. In addition, lumbar sympathectomy has been shown to promote occurrence of Monckeberg's arteriosclerosis of the lower extremities in humans.1 Automatic dysfunction from diabetic neuropathy is thought to be responsible for the occurrence of Monckeberg's in diabetic patients.1 Unlike atherosclerosis, Monckeberg's arteriosclerosis is a benign condition and does not cause vascular thrombosis. In conclusion, Monckeberg's arteriosclerosis of the temporal artery may be seen occasionally in the temporal artery. It is an interesting histological diagnosis that has little clinical significance but could be recognised in temporal biopsies.

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Retinal vascular abnormality in Poland’s syndrome

EDWARD.—Poland’s syndrome is a congenital anomaly first described in 18411 consisting of unilateral hypoplasia or aplasia of the pectoralis major muscle and ipsilateral upper extremity abnormalities which often include ipsilateral syndactyly. Since then a number of associated anomalies have been reported. These include absence of the pectoralis minor muscle, absence or atrophy of ipsilateral ribs two to five, aplasia of the ipsilateral breast or nipple, and simian crease of the affected extremity. Although vascular alterations associated with this syndrome have been described,1 no involvement of eye vasculature has been reported so far.

CASE REPORT

We examined a 39 year old man previously diagnosed with Poland’s syndrome who came to our clinic because he had experienced blurred vision in the right eye for 2 months. Computed axial tomography, arteriography, and abdominal echography revealed absence of the left kidney. There was no history of diabetes or hypertension. The ophthalmological examination of the right eye revealed a visual acuity of 20/50 and a paracentral relative scotoma. The right eye fundus showed perimacular capillary telangiectasis, retinal vascular distortion, moderate perimacular hard exudates, and retinal swelling, all of them more prominent in the temporal perimacular area. Fluorescein angiography clearly showed the vascular abnormalities (Fig 1). The left eye fundus was normal. A diagnosis of juxtafoveal retinal telangiectasia was made.

COMMENT

Juxtafoveal telangiectasis is difficult to detect ophthalmoscopically and therefore can be neglected by routine eye fundus examinations made before the first visual symptoms appear. Unilateral renal agenesis occurs in approximately 1 per 1000 births and can be associated with vascular anomalies.1 The aetiology of juxtafoveal telangiectasis is unknown. The patient we examined had retinal vascular malformations that may have originated in the early stages of life. Indeed, the deformed capillaries may function for many years before endothelial decompensation causes retinal swelling.1 While the exact pathogenesis of Poland’s syndrome is not well known, it has been postulated that the original cause may be a vascular abnormality at the embryonic stages.2 The vascular abnormalities, mostly of the diseased hemithorax, reported in this syndrome1 and our finding of retinal vascular abnormalities in this patient support this hypothesis.

To our knowledge, this is the first reported case of coexistent juxtafoveal telangiectasia, renal agenesis, and Poland’s syndrome. Although we are not aware of direct evidence reported in the literature indicating any retinal involvement linked to this syndrome, it is plausible that the three anomalies found in this patient may have had common original vascular causative factors. Therefore, we recommend a careful eye fundus examination of patients presenting with this syndrome.

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Bilateral optic disc oedema associated with latanoprost

EDITOR.—Latanoprost is a recent addition to the medical management of raised intraocular pressure in chronic open angle glaucoma and ocular hypertension. It is a potent ocular hypotensive agent with few ocular or systemic side effects.

We report a case of bilateral optic disc oedema developing soon after commencing treatment with latanoprost which resolved once therapy was stopped.

CASE REPORT

An asymptomatic 64 year old woman presented with raised intraocular pressure. She maintained good general health, had no significant past medical or family history, and was not on any medication. Snellen visual acuities were 6/5 both eyes. The intraocular pressures were 28 mm Hg right eye and 26 mm Hg left eye. Ocular examination was otherwise unremarkable with open angles, normal optic nerves, and full Humphrey 24-2 visual fields. She was thus diagnosed as having ocular hypertension and consented to enter a prospective double masked trial comparing some of the intraocular pressure lowering drops. Therapy was commenced with one of the drugs involved in the study and at a 1 month review she reported no problems with the drops. The intraocular pressures had lowered to 16 mm Hg in both eyes and the examination was otherwise unchanged. At her third visit 2 months later, she was again asymptomatic with visual acuities of 6/5 in both eyes and intraocular pressures of 15 mm Hg. However, examination of the optic nerves revealed bilateral oedema which was more prominent in the left eye. There were no signs of uveitis in either eye, papillary reflexes were normal, colour vision and Amsler testing were not affected, and the visual fields were full. At this point the code for the trial drug was broken and it was seen that she had been using latanoprost 0.005% eye drops at night to both eyes over the 3 month period. A neurological consultation failed to find any neurological abnormality and all haematological and biochemical analyses were normal. A computed tomography scan with contrast showed no abnormality and she was discharged from neurological review. Follow up in the eye clinic revealed no change after 72 hours. The latanoprost was stopped and the disc swelling had largely resolved at 1 week. By 10 weeks
both optic nerves looked normal. Visual acuities were still 6/6 in both eyes and there was no loss of colour vision or visual field. The intraocular pressures had increased to 22 mm Hg in both eyes.

**COMMENT**

Latanoprost is a prostaglandin F₂ₐ analogue which acts by increasing uveoscleral outflow. Side effects include increased iris pigmentation, hypotrichosis and increased eyelash pigmentation, anterior uveitis in patients with complicated glaucoma or in those having had previous incisional surgery, and cystoid macular oedema occurring soon after beginning latanoprost in pseudophakic or aphakic eyes. Ocular hypotony with choroidal effusions and facial rash have also been attributed to latanoprost. To the best of our knowledge, optic disc oedema associated with latanoprost has not previously been described. The mechanism behind this association is unclear. One may not be surprised to see optic nerve swelling in association with signs of posterior uveitis or hypotony but in this case it occurred without any sign of uveal inflammation and the lowest recorded intraocular pressure was 15 mm Hg. It may be feasible that the perfusion to the optic nerve heads via the short posterior ciliary arteries was compromised by a prostaglandin-like action manifesting as disc oedema. It was found that latanoprost acid and prostaglandin F₂ₐ at high concentrations could cause vasoconstriction of bovine ciliary arteries and a similar action cannot be discounted in this case. The rapid resolution of the swelling with seemingly no long term sequelae once latanoprost was stopped would perhaps support this hypothesis.

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**Letters**

**Somatostatin scan positive gastrinoma ocular metastasis**

**Editor,—** Symptomatic ocular metastases are uncommon despite the 4% prevalence in patients dying of all types of malignancy in postmortem series. We report a case of ocular metastasis from a gastrinoma, which was part of the Wermer’s syndrome (multiple endocrine neoplasia (MEN) type 1), diagnosed by indium labelled octreotide scanning.

**CASE REPORT**

A 57 year old man presented with a 1 week history of blurring in his peripheral vision in his right eye and severe loss of visual acuity, worse early morning. He had been diagnosed with MEN type 1, 8 years previously after two perforated jejunal ulcers (1978, 1990) led to a diagnosis of Zollinger–Ellison syndrome, and a hyperplastic parathyroid gland had been removed for hypercalcaemia (1990). His mother had MEN type 1. Ophthalmic examination revealed 6/12 acuity in the right eye and 6/5 in the left. His anterior segments were unremarkable. His right fundus showed a small amelanotic lesion about one disc diameter above the right disc. Ultrasonography demonstrated a base of 13 mm and a height of 8 mm. He also had bilateral inferior retinoschisis. One month later the tumour base measured 14.5 mm and the thickness measured 7.9 mm. The posterior edge now practically abutted the optic disc (Fig 1). There was subretinal fluid accumulation.

A liver ultrasound scan showed multiple lesions and a tumour biopsy was composed of small islands of polygonal cells with granular cytoplasm, diagnostic of metastatic neuroendocrine carcinoma (immunostaining positive for chromogranin, neuron specific enolase, and NCAM, negative for S-100 and HMB45 (melanoma markers)). His urinary 5HIAA was marginally raised at 133 pmol/24 hours (normal up to 75), and a fasting intestinal peptide screen, showed a highly elevated gastrin level (on omeprazole 40 mg per day) of 343 pmol/l (normal range 0–40 pmol/l) but normal levels of other polypeptides.

No prior tumour or further metastases were seen using body computed tomograph and magnetic resonance imaging scans and the I-123 MIBG scan (meta-iodobenzyl guanidine) was also negative. An indium (In-111) labelled octreotide scan at 1 and 4 hours +PET showed focal areas of increased uptake in the right orbit (Fig 2), the nasal region, mediastinum, multiple sites in the liver, and possibly other abdomen sites. However, a positive octreotide scan is not exclusively seen with neuroendocrine tumours, since other tissues have somatostatin receptors.

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**Oncological Imaging**

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Retinal neovascularisation in Goltz syndrome (focal dermal hypoplasia)

EDITOR,—This is the first reported case of Goltz syndrome with documented peripheral retinal non-perfusion with subsequent retinal neovascularisation and vitreous haemorrhage. In the eye this represents solely a mesodermal neovascularisation and vitreous haemorrhage. Goltz syndrome with documented peripheral macula, and temporal equatorial fibrotic right myopia. Right eye 6/9 and left eye 6/5, with a minor was negative for skin, skeletal, and ocular genital absence of one deciduous incisor. Dental screening revealed the con-ophthalmic screening revealed no ocular anomalies. Other reported ocular anomalies include ano- strabismus, nystagmus, and ectopia lentis. Colobomata have been reported in one third regression within weeks.

COMMENT
Focal dermal hypoplasia is a rare disorder of ectodermal and mesodermal dysplasia originally described by Goltz et al. It is characterised by congenital atrophic skin changes often associated with herniation of the subcutane- ous fat; skeletal anomalies, in particular syn- dactyly, polydactyly, or adactyly as well as sco- liosis, kyphosis, spina bifida occulta, rib and scapula anomalies; and dental anomalies, especially hypodontia. Ocular anomalies occur in 40% of cases. Colobomata have been reported in one third of cases, then less frequently microphthalmia, strabismus, nystagmus, and ectopia lentis. Other reported ocular anomalies include ane- phthalmitis, corneal clouding, aniridia, hetero- chromia, and optic atrophy. Rarely ectropion and ptosis may occur as well as lid margin or chro- miation and vitreous haemorrhage but usually manifests as severe choroidal and reti- nal rupture associated with haemorrhage but not pen- etrating it. It is a concession injury, which usually manifests as severe choroidal and reti- nal rupture associated with haemorrhage but an intact sclera. We report a case of chorioretinitis sclopetaria resulting from de- ployment of an airbag in a stationary motor vehicle.

CASE REPORT
A 32 year old pregnant woman was the driver in a car accident in which the airbag failed to deploy at the time of impact but inflated when she returned to sit in the drivers seat approxi- mately 5 minutes after the accident occurred. The patient was not wearing spectacles and there was no evidence of injury from other sources.

At presentation the visual acuity was hand movements in the left eye and 6/6 in the right eye. Examination revealed evidence of swollen eyelids and marked chemosis and subconjunc- tival haemorrhages on the left, with no bony injury and a full range of eye movements. Anterior segment examination revealed a clear left cornea and a quiet anterior chamber with a microhyphaema. The lens was clear and there was no evidence of a cataract. There was no angle recession and the intraocular vessels and haemorrhage. Fluorescein angiography showed bilateral peripheral retinal non-perfusion and right temporal neovascularisation (see Figs 1 and 2). This was treated conservatively for 2 years; however, after six bleeds within 3 months indirect retinal photo- coagulation to the areas of non-perfusion was performed with subsequent new vessel regression within weeks.

Retinitis sclopetaria associated with airbag inflation

EDITOR,—Chorioretinitis sclopetaria is a se- vere form of blunt trauma, caused by a high velocity object grazing the globe but not penetr- etrating it. It is a concussion injury, which usually manifests as severe choroidal and reti- nal rupture associated with haemorrhage but an intact sclera. We report a case of chorioretinitis sclopetaria resulting from deployment of an airbag in a stationary motor vehicle.

CASE REPORT
A 32 year old pregnant woman was the driver in a car accident in which the airbag failed to deploy at the time of impact but inflated when she returned to sit in the drivers seat approxi- mately 5 minutes after the accident occurred. The patient was not wearing spectacles and there was no evidence of injury from other sources.

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pressure was recorded at less than 4 mm Hg. Fundal examination revealed evidence of a retinal tear although details were obscured by a diffuse vitreous haemorrhage. An ultrasound examination showed a vitreous haemorrhage and large retinal tear but no evidence of a scleral perforation.

An examination under anaesthesia performed the following day confirmed that there was no scleral rupture and indirect ophthalmoscopy confirmed the findings of diffuse vitreous haemorrhage and a retinal tear.

At the 2 week postoperative clinic visit, visual acuity had improved to 6/18. There was 2+ cells in the anterior chamber and the intraocular pressure was 24 mm Hg. Posterior segment findings were an intragel haemorrhage and retinitis scopolertia with no evidence of retinal detachment.

Four months later, the best corrected visual acuity was 6/36. The anterior chamber activity had settled; the intraocular pressure was 10 mm Hg, and fundal examination was unchanged. In view of the persistent vitreous haemorrhage the patient was listed for routine pars plana vitrectomy in the postpartum period.

COMMENT

Airbags are designed to protect the driver from direct impact from the steering wheel, dashboard, and windscreen. They are designed to inflate in 10 ms in response to sudden deceleration and during deployment, the airbag is propelled out of its storage compartment at speeds of more than 100 mph. Following inflation the airbag deflates slowly within seconds.

Facial and ocular injuries associated with airbags have been reported in the literature. Skin abrasions, burns, and eyelid ecchymoses are the most common facial injuries. Reported ocular injuries include orbital fractures, keratitis, corneal abrasions, hyphaema, angle recession, and lens subluxation. In the posterior segment, vitreous and retinal haemorrhages, commotio retinae, retinal tears and dialyses, and choroidal ruptures have been reported. To our knowledge, this is the first reported case of retinitis scopolertia secondary to airbag inflation.

Although airbags have clearly been shown to reduce serious morbidity and mortality associated with road traffic accidents, they are associated with a number of injuries directly attributable to their inflation. Some of these are serious ocular injuries and it is important for ophthalmologists and others involved with trauma cases to be aware of these complications. A full ophthalmic assessment is mandatory in all cases and this should include indentation ophthalmoscopy. A variety of posterior segment injuries have been reported in the literature but this is the first report of retinitis scopolertia resulting from airbag deployment.

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Figure 1  Airbag injury with retinitis scopolertia, an intragel haemorrhage, and attached retina.

Specular microscopic findings of corneal deposits in patients with Bietti’s crystalline corneal retinal dystrophy

Editor,—In 1937, Bietti first described three cases of tapetoretinal degeneration characterised by yellowish glistening retinal crystals, tapetoretinal degeneration with choroidal sclerosis, and marginal crystalline deposits of the cornea. Although more than 100 cases of crystalline retinopathy have been reported, crystalline deposits of the corneal limbus have been observed in only four out of 52 Japanese patients with crystalline retinopathy. Recently, observation using specular microscopy has been reported to be useful in detecting crystalline deposits at the limbus of patients with crystalline corneal retinal dystrophy. Therefore, in this study, we examined four patients with crystalline retinopathy using specular microscopy under a “con-surface” mode, which is used for the observation of the corneal surface, and we detected the deposits at the limbus.

Twelve months after the initial specular microscopic examination, we reinspected the crystalline deposits of two cases. Interestingly, the changes in the locations and forms of the crystalline deposits in the corneal limbus were exposed over time (Fig 1). More crystalline deposits were found in the patients with more advanced retinopathy. It is supposed that corneal deposits and fundus deposits are essentially the same and it is suggested that crystalline retinopathy is caused by systemic abnormality. Although the exact pathogenesis of crystalline deposits is still uncertain, it is possible that destroyed fibroblasts appear to glitter or fibroblasts with crystalline-like deposits look glittering during breakdown. Further biochemical or cellular biological studies are needed to clarify these possibilities.

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