LETTERS TO THE EDITOR

Partial lipodystrophy with associated fundus abnormalities: an optical coherence tomography study

EDITOR,—A patient with acquired partial lipodystrophy (PLD), associated with drusen-like lesions of the posterior pole, underwent fluorescein angiography (FA), electroretinogram (ERG), pattern electroretinogram (PERG), and electro-oculogram (EOG). In addition, optical coherence tomography (OCT) scans of the posterior pole were performed. Visual acuity was normal and the patient did not reveal metamorphopsia. There were, however, diffuse alterations at the EOG, whereas the ERG results were normal.

Drusen-like lesions have already been described in PLD and the associated EOG abnormalities were supposed to be related to Bruch's membrane deposits.[1–3] This study, by means of OCT, confirms that drusen-like lesions correspond with accumulation phenomena under retinal pigment epithelium (RPE) probably causing EOG abnormalities.

CASE REPORT

A 40 year old woman with a clinical history of acquired PLD was referred to our department on 4 March 1997 for routine examination. The general physical examination showed loss of subcutaneous fat from the face. Previous blood tests had found low serum level of C3 (3–4 mg/dl; normal value 70–140 mg/dl) in the presence of normal values of other clinical variables.

The visual acuity was normal in both eyes. The Amsler test was negative. Fundus examination showed yellow, drusen-like lesions in the posterior pole and in the mid-periphery of the retina. The optic nerves were normal.

The patient underwent FA and electrophysiological examinations (ERG, PERG, EOG).

Fluorescein angiography showed numerous hyperfluorescent diffuse spots in the posterior pole and in the mid-periphery of the retina, corresponding to the drusen-like lesions. These lesions were more numerous and larger temporally to the macula (Fig 1). The electrophysiological examinations revealed a reduction of the EOG light peak to dark through the Arden ratio. Flash and flicker ERG and PERG values were essentially normal. The OCT line scans of the macular region showed that the hyperreflective band relative to the RPE (red pseudocolour) was not regular. More specifically, the scan of the areas corresponding to drusen-like lesions showed how the red band was slightly lifted (Fig 2).

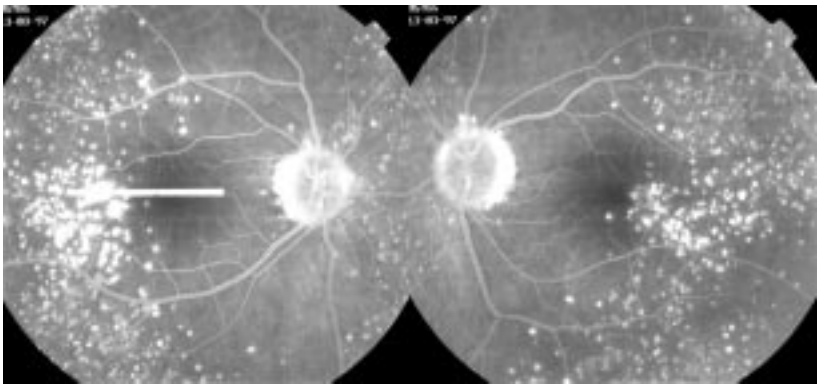

Figure 1 Fluorescein angiogram showing numerous hyperfluorescent drusen-like spots in the posterior pole. These lesions appear to be more numerous and larger temporally to the macula.

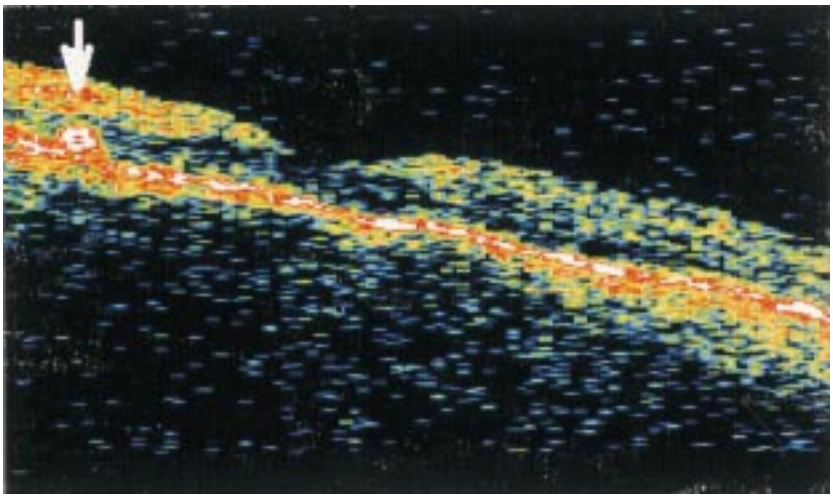

Figure 2 The OCT horizontal line scan of the macular region of the right eye (white line on Fig 1) shows that the hyperreflective band relative to the RPE (red pseudocolour) is not regular. The scan of the areas corresponding to drusen-like lesions shows how the red band is slightly lifted (white arrow).

COMMENT

O'Brien and colleagues described three cases of partial lipodystrophy associated with drusen-like lesions and alterations of EOG.[1] They hypothesised that EOG abnormalities could be related to substance accumulation phenomena under RPE. Our case showed clinical features similar to those described by O'Brien *et al.* The potential of OCT examination for detecting macular diseases has already been pointed out.[4 5] In this case, OCT examination found the presence of a non-regular pattern of RPE, associated with small solid liftings, corresponding to drusen-like lesions. This evidence confirms the presence of morphological alterations of the RPE, as was hypothesised, justifying the electrophysiological alterations.

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Unusual macular retinal detachment associated with vitreomacular traction syndrome

EDITOR,—Vitreomacular traction syndrome may result in macular pucker, retinal blood vessel avulsion, retinal hole formation, cystoid macular oedema, or traction retinal detachment.[1–6] Of these complications, traction retinal detachment is relatively uncommon and has not been well described. We report a case with an unusual convex macular detachment complicated by retinal pigment epithelial disorders caused by vitreomacular traction.

CASE REPORT

An 83 year old Japanese man was referred to our clinic for treatment of a retinal detachment in the right eye. The duration of retinal detachment was unknown, but he had been aware of decreasing vision in the right eye for more than 9 months. His medical history was unremarkable except for a cataract extraction and intraocular lens implantation in the left eye.

Ocular examination revealed his corrected visual acuities to be 20/200 in the right eye and 20/20 in the left. Intraocular pressure was 7 mm Hg in both eyes. Slit lamp examination disclosed a cortical cataract in the right eye and an intraocular lens in the left eye. Indirect ophthalmoscopy and slit lamp biomicroscopic examination of the right eye with a 90D lens and contact lens disclosed an unusual convex retinal detachment of the macula extending towards the disc in an arch-like configuration (Fig 1A). The posterior hyaloid was adherent to the detached posterior retina and separated from the attached retina. Ophthalmoscopic examination of the left eye revealed a tiny depigmented area in the macula. Scanning laser ophthalmoscopy (SLO) using an argon laser showed apparent radial traction striae of the posterior hyaloid surrounding the macula (Fig 1B). The vitreomacular traction produced by posterior hyaloid was clearly observed during eye movements.

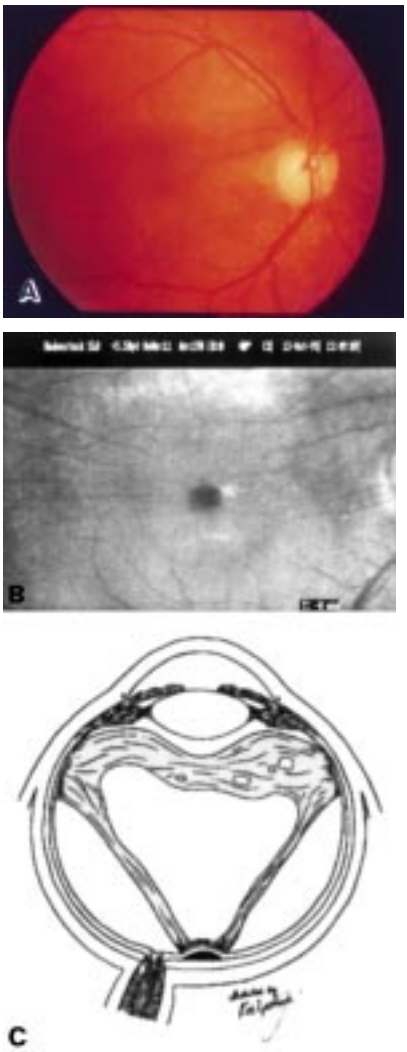

Figure 1 (A) An unusual convex macular detachment before surgery. (B) Scanning laser ophthalmoscopy showing radial traction striae attached to the macula produced by an incomplete posterior vitreous detachment. (C) Schematic cross section of the eye during the vitrectomy.

Vitrectomy was performed and abnormal vitreoretinal anatomical features were confirmed intraoperatively. There was marked syneresis of the vitreous cortex but Weiss's ring was not detected. The posterior vitreous face was detached in all but the macula and optic disc (Fig 1C). After core vitrectomy, the detached posterior hyaloid was removed to release the anteroposterior traction on the macula. The thickened posterior hyaloid was markedly adherent to the detached macula and it was necessary to use a microhooked needle to peel it off. Cataract extraction and intraocular lens implantation were simultaneously performed.

There were no remarkable changes in either visual acuity or the retinal detachment at 3 months postoperatively. Fluorescein angiography, performed to rule out subretinal neovascularisation, showed stainings and mild leakages through the retinal pigment epithelium beneath the detached retina and mild leakage at the disc (Fig 2). There were faint leakages from the retinal vessels as well. Four months after surgery, visual acuity began to improve. By 11 months after surgery, vision had been restored to 20/40 and the retinal detachment had disappeared.

COMMENT

Vitreomacular traction syndrome has recently been described as a distinct clinical entity, which develops secondary to persistent anterior to posterior traction on the macula via a directly observable persistent vitreomacular attachment.[2] It may cause a variety of abnormalities in macular appearance and function. Typically, the zone of vitreous attachment includes premacular tissues that result in a clinical appearance similar to that of idiopathic macular pucker. Although rare, traction retinal detachment can occur as a complication of vitreomacular traction syndrome.[1 2 4] We confirmed the diagnosis of vitreomacular traction syndrome in this case through preoperative fundus examinations including SLO and intraoperative observation. The attached posterior hyaloid was thickened and markedly adherent to the macula, thus necessitating membrane peeling. Strong vitreoretinal adhesion throughout the macula and peripapillary retina, in a "sheet-like" configuration, seemed to cause the broad based macular detachment.[4]

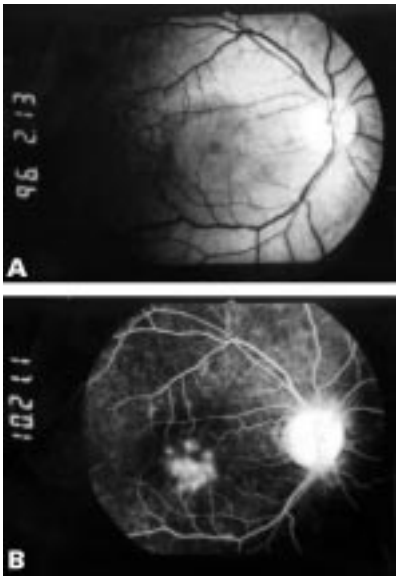

Figure 2 (A) Red-free photograph showing the residual dome macular detachment after vitrectomy. (B) Fluorescein angiography showing hyperfluorescence in the area corresponding to the detached retina.

The convex or dome-like detachment in the present case was unusual, in contrast with the commonly seen concave traction detachment. Melberg and colleagues[4] described the clinical characteristics of nine cases of vitreomacular traction syndrome with macular detachment. A similar unusual detachment was described in their series as a case report. They performed fluorescein angiography in their series which demonstrated mild intraretinal hyperfluorescence in all eyes, but there was no mention of retinal pigment epithelial changes. We observed mild leakage and staining at the retinal pigment epithelium beneath the detached retina. This retinal pigment epithelial degeneration may be secondary to vitreous traction. Long standing traction forces from the posterior hyaloid to the macula may have created a static pressure which resulted in leakage through the retinal pigment epithelium to produce the unusual concave retinal detachment seen in our case.

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Steroid responsive disc neovascularisation in uveitis associated with juvenile chronic arthritis

EDITOR,—Childhood uveitis is most frequently associated with juvenile chronic arthritis (JCA).[1] The uveitis associated with JCA is anterior with no posterior involvement, although cystoid macular oedema may occur after cataract surgery. We describe disc neovascularisation (NVD) in two children with JCA associated chronic anterior uveitis.

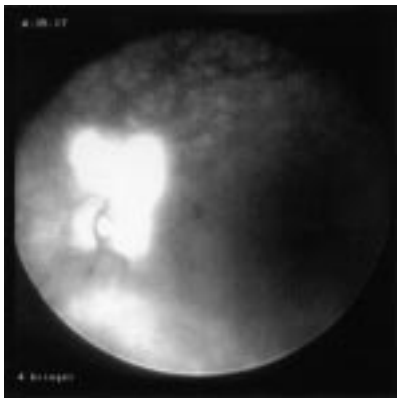

Figure 1 Left fluorescein angiogram demonstrating leakage from disc new vessels before systemic steroid treatment.

The possible aetiology of this and subsequent management are presented.

CASE REPORTS

Case 1

Left NVD was diagnosed in a white 13 year old girl with a 3 year history of bilateral anterior uveitis associated with pauciarticular onset JCA. There were no clinical or laboratory features to suggest any other disease process and she was completely asymptomatic apart from the involved joint. At presentation and throughout follow up she had a mild right anterior uveitis and moderate left anterior uveitis despite the constant use of topical steroids. No other form of immunomodulation had been used. A unilateral secondary cataract reduced left visual acuity to 6/12. There was no diabetes, pars planitis, vitritis or retinal vasculitis, or any other ocular pathology which may be associated with neovascularisation. The presence of unilateral left NVD without retinal ischaemia or retinal vascular leakage was confirmed angiographically (Fig 1).

Increased activity of her left iridocyclitis, and absence of ischaemia prompted the use of systemic steroids to control the neovascularisation. Oral prednisolone (1 mg/kg/day starting dose, reducing by 10 mg each week) was successful in causing significant regression of the NVD after 2 weeks of treatment (Fig 2). Attempted withdrawal resulted in increased NVD.

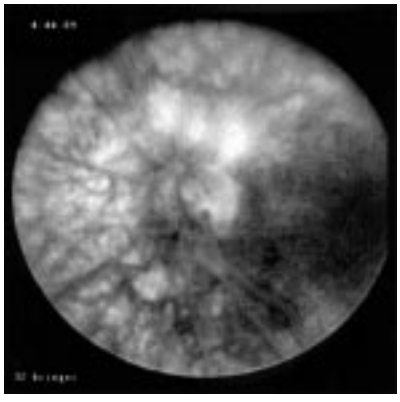

Figure 2 Fluorescein angiogram demonstrating less fluorescein leakage after 2 weeks of oral steroids. This photograph is of a later fluorescein circulation time than Figure 1 (by 9 seconds). Note clearer view resulting from reduced inflammatory media opacities.

Case 2

Bilateral burnt out NVD was observed in a 4 year old Kuwaiti boy. At presentation the patient had severe bilateral anterior uveitis, secondary cataracts, and ocular hypotony. Subsequently, he developed pauciarticular JCA and there was nothing to suggest any other systemic or ocular disease. When the cataracts were removed, bilateral NVD with gliosis was found. No other cause for neovascularisation was detected; in particular, there was no evidence of pars planitis, retinal vascultiis, or ischaemia. Before cataract extraction, no form of topical or systemic immunosuppression had been used.

COMMENT

This is the first report of posterior segment neovascularisation in patients with juvenile chronic iridocyclitis and JCA. The uveitis associated with JCA is entirely anterior and not associated with posterior segment disease.[2] Chevalley *et al* have described "complete regression" of both "subretinal and preretinal neovascularisation" in other types of uveitis after treatment with systemic steroids.[3]

It is presumed (but not proved) that the NVD found in these two cases of JCA associated chronic anterior uveitis have a causal relation. Certainly in case 1, the anterior segment inflammatory activity had been very difficult to control. In the absence of posterior segment ischaemia or inflammation, it would appear that angiogenic factors released from the anterior segment led to disc neovascularisation.

Angiogenesis (neovascularisation) occurs under both physiological (for example, wound healing, placental maturation) and pathological (for example, tumour growth, rubeosis) conditions. Regardless of aetiology, new vessel formation occurs in identical stages in response to various angiogenic factors. These angiogenic factors form an ever increasing group of compounds.[4]

Present experimental evidence of neovascularisation associated with uveitis would appear to support the angiogenic role of prostaglandins.[5–9] Primate immunological ocular inflammation produced by serum albumin[5] produces a clinical picture similar to that of proliferative diabetic retinopathy. Anti-inflammatory drugs that inhibit the effects of prostaglandins (for example, indomethacin[6] and methylprednisolone[7]) and irradiation (which induces leucopenia[8]), all reduce experimental neovascularisation in this primate model. Methylprednisolone is also well known for its potent antiangiogenic activity. This activity is common to a group of steroids collectively known as angiostatic steroids, including several glucocorticoid and mineralocorticoid steroids. Neither the glucocorticoid nor mineralocorticoid function is necessary for antiangiogenic activity.[9]

The regression of NVD after exposure to oral prednisolone in case 1 may therefore be a clinical example of the angiostatic role of steroids in uveitic neovascularisation.

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Scleritis associated with acute febrile neutrophilic dermatosis (Sweet's syndrome)

EDITOR,—Sweet's syndrome, or acute febrile neutrophilic dermatosis, is characterised by (1) fever, (2) neutrophilic leucocytosis, (3) the abrupt appearance of 0.5–12.0 cm painful erythematous nodules and plaques, especially on the face, neck, and limbs, and (4) a histological pattern of dense dermal infiltrates of mature neutrophils without vasculitis.[1 2] Patients are usually middle aged women and may have extracutaneous manifestations involving the eyes, kidneys, joints, liver, and lungs. Sweet's syndrome is associated with underlying malignancy, especially haematological, in 10% to 54% of patients[2 3] and with systemic inflammatory disorders. There may be a prodromal respiratory illness and an elevated erythrocyte sedimentation rate (ESR).

Ocular involvement is reported in 4% to 72% of cases and may manifest as conjunctivitis, episcleritis, subconjunctival haemorrhage, inflammatory glaucoma, iritis, or limbal nodules.[3–6] We describe a case of a patient with Sweet's syndrome and scleritis.

CASE REPORT

A 78 year old white man presented to his ophthalmologist with a 2 week complaint of a red, sore right eye without decreased vision. After a week of worsening symptoms on topical ciprofloxacin drops for presumed conjunctivitis, the ophthalmologist switched the patient to topical prednisolone acetate 0.125% for possible episcleritis. The persistence of ocular symptoms despite topical prednisolone acetate 0.125% as well as a 6 week history of numerous erythematous slightly tender papules over his extremities prompted referral to our hospital.

The patient's ocular history was significant for endophthalmitis in the left eye after extracapsular cataract extraction 3 years earlier. Additional history included a laryngeal mass biopsy 1 year earlier showing nonspecific inflammation, recurrent facial basal cell cancer, 4 months of episodic fever up to 102°F, and right mastoiditis and otitis media 1 month before initial presentation.

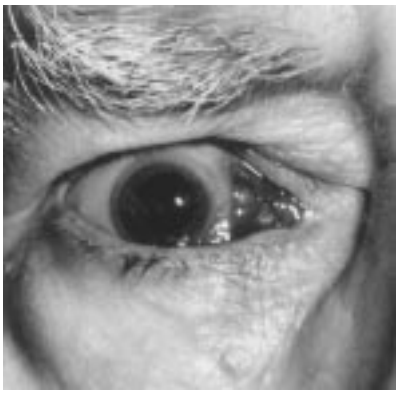

Figure 1 Photograph of right eye with medial scleral nodules.

At our initial examination, best corrected visual acuity was right eye 6/6 and left eye light perception with projection. Moderate injection was noted medially in the right eye, with pronounced tenderness to palpation. Application of 10% neosynephrine blanched the superficial vessels, revealing deep injection of scleral vessels as well as three broad scleral nodules with a central 3.0 mm yellow white avascular area (Fig 1). The left cornea had diffuse stromal oedema with an irregular superior pannus. The anterior chamber was deep and quiet in the right eye but flat in the left eye. The remainder of the anterior and posterior segment examinations were not significant. Examination of his arms and legs revealed multiple erythematous papules ranging from 2–10 cm in diameter.

Treatment with prednisolone acetate 0.125% four times a day to the right eye was continued, and oral prednisone 60 mg daily was added.

The possibility of Sweet's syndrome led to further examination and dermatology consultation. Significant findings included an ESR of 117 and a WBC of 16.4×10^9/l (neutrophils 31, lymphocytes 33, monocytes 34, bands 0). Blood cultures, uric acid, ANCA, ANA, RF, FTA-ABS, RPR, ACE, lysozyme, HLA-B27, and chest *x* ray were unremarkable. A bone scan showed intense uptake of the left knee suggesting osteomyelitis, inflammatory disease, or neoplasm. A skin biopsy revealed dermal neutrophilic infiltrates without vasculitis, consistent with Sweet's syndrome (Fig 2). Special stains for organisms (AFB, PAS, B&B, GMS stains) were negative.

A subsequent bone marrow biopsy demonstrated active cellular marrow with proliferation of all three cell lines and an excess of "blast" cells, most of which were myeloid and a minority of which were monocytes. This suggested a myelodysplastic syndrome, consistent with refractory anaemia with excess blasts (RAEB).

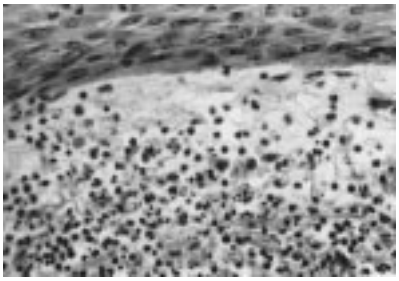

Figure 2 Skin biopsy with diffuse neutrophilic infiltrate and dermal oedema but without vasculitis (original magnification × 340).

Over the next 2 weeks, the patient's scleritis began to resolve with both prednisolone acetate 0.125% and oral prednisone 60 mg daily. Medications were tapered over 2 months; however, once a dose of 30 mg of prednisone was reached, he developed new skin lesions and the scleritis recurred. Increasing the prednisone to 40 mg daily resulted in partial improvement of the skin lesions and resolution of the scleritis. The skin lesions and scleritis have remained inactive for 16 months on prednisone 15 mg daily and prednisolone acetate 0.125% one drop every other day.

COMMENT

Most articles in the medical literature on Sweet's syndrome describe ocular involvement as benign.[2] There are only two case reports in the ophthalmic literature describing potentially vision threatening ocular complications associated with this disease.[5 6] We believe this is the first report of a patient with Sweet's syndrome presenting with scleritis. A physician seeing a patient with ocular inflammation, skin lesions, and fever must consider the diagnosis of Sweet's syndrome. This condition should not be missed, because it may be associated with an underlying haematological malignancy or solid tumour.

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Cornea plana—clinical features, videokeratometry, and management

EDITOR,—Cornea plana is a rare anomaly in which the corneal radius of curvature is larger than normal producing high hypermetropia with astigmatism and poor acuity in most cases. Myopic astigmatism can also occur. We describe three patients with this anomaly including videokeratometry images.

CASE REPORTS

Case 1

A 25 year old woman complained of a deterioration in visual acuity over recent years. Her parents, three brothers, and an older sister

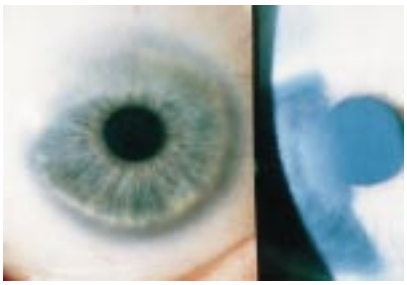

Figure 1 Right eye of case 1 shows the peripheral corneal changes of superficial pannus and deeper stromal opacification (left). There was also deep stromal opacification and thickening in the central cornea (right).

(examined by the authors) had no manifest ocular problems.

Corrected visual acuities were 6/24, N24 right eye and 6/60, N24 left eye with refractive errors of $-2.25/+1.50 \times 165°$ and $-7.50/+2.50 \times 20°$ respectively. Examination revealed apparently small corneas with peripheral opacification—particularly superiorly (Fig 1)—giving a horizontally oval configuration. There was central stromal thickening and faint opacity (Fig 1). Automated videokeratoscopy confirmed marked corneal flattening with regular astigmatism (Fig 2). There were no abnormal features affecting the iris or crystalline lens. The intraocular tensions (Goldmann tonometry) were right eye 11 mm Hg and left eye 10 mm Hg. Gonioscopy demonstrated open drainage angles with normal angle structures. Axial lengths were 26.90 mm right eye and 26.95 mm left eye. Funduscopy revealed healthy maculas and bilateral peripapillary chorioretinal degeneration consistent with marked axial myopia. She is managing well with a low visual aid but no additional improvement in vision could be achieved with contact lenses.

Case 2

A healthy 13 month old girl was noted to have bilateral corneal "haze" and a left exophoria of approximately 20 prism dioptres with and

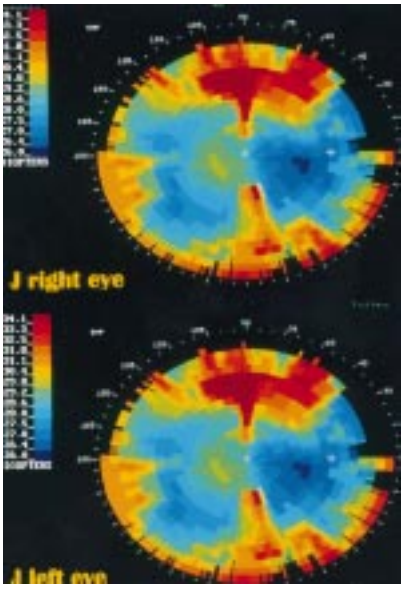

Figure 2 Computer assisted videokeratometry maps of case 1 showing marked flattening (see colour coded scale bar) with the characteristic "bow tie" pattern seen with regular astigmatism.

without optical correction. Examination under general anaesthesia showed refractive errors of +6.50/+2.00 × 110° right eye and +8.50/+2.00 × 70° left eye. Both corneas were small and horizontally oval with vertical corneal diameters of 8.75 mm right eye and 9.50 mm left eye. There was superficial pannus and deeper stromal opacification of the peripheral cornea and faint stromal opacification centrally. Intraocular pressure was 10 mm Hg in both eyes. The lenses, irides, and fundi were healthy. She was lost to follow up and was re-referred aged 4 years with manifest left convergent strabismus. Acuities were right eye 6/18, left eye 4/60 (Kay's pictures). Cycloplegic refraction revealed a refractive error of +5.50 DS in both eyes. Glasses were prescribed. Two months later the squint was much improved but acuity remained poor in the left eye—right eye 6/9, left eye 2/60 (Sheridan–Gardner test), and occlusion has been commenced.

Case 3

The 13 year old elder brother of case 2. Best corrected acuities were right eye 6/9, N5 and left eye 6/18, N5 with refractive errors of +7.50/+1.75 × 90° and +8.50/+1.75 × 110° respectively. He was orthophoric with good binocular function. Both corneas appeared horizontally oval with circumferential peripheral opacification. Vertical corneal diameters were 7.12 mm. There was superficial corneal opacification centrally but no vascularisation or thickening (Haag–Streit pachymeter readings: 0.42 mm right eye, 0.40 mm left eye). Both anterior chambers were moderately shallow. The drainage angles were open with no abnormal features. Intraocular pressures were normal.

Axial lengths were 23.15 mm right eye and 23.40 mm left eye. Computer assisted videokeratoscopy imaging revealed marked, bilateral corneal flattening (more prominent centrally) with regular astigmatism.

COMMENT

Cornea plana was first described by Rubel in 1912.[1] It is more often described along with the more common entity, sclerocornea,[2] in which there is peripheral opacification and vascularisation of the cornea. In cases with sclerocornea only a proportion are associated with an enlarged radius of curvature of the central cornea, cornea plana. Over 90% of cases are bilateral.[3] The peripheral corneal vascularisation may be superficial or deep and appears to be non-progressive. Differential diagnosis includes microphthalmia and microcornea.[4]

Conventional keratometry confirms the diagnosis but computer assisted videokeratometry has not been described previously. Other distinguishing features of the condition include the horizontally oval corneal appearance and a normal or enlarged (as in case 3) axial length. The usual refractive status is hypermetropia with astigmatism (cases 1 and 2) although myopia has also been described in eyes with marked axial lengthening.[5]

The gene responsible for cornea plana has recently been assigned linkage to the long arm of chromosome 12. This represents an exciting development in our understanding of this anomaly.[6]

Failure to diagnose and correctly manage individuals with cornea plana at an early age can result in the development of ametropic amblyopia.[7] Careful orthoptic assessment is vital (as demonstrated by case 1). There are several problems with spectacle correction using high plus lenses—poor cosmesis, restriction of peripheral visual field, magnification and peripheral ring scotomata—so rigid contact lenses, which overcome these optical problems, have been used with some success although the extreme corneal flattening in this condition can lead to a poor, unstable fit. This problem can be overcome (as in case 2) with the use of a soft (HEMA) lenticular contact lenses.[8] Penetrating keratoplasty has been advocated in a previous report[9] but seems unnecessary as contact lenses achieve good visual and cosmetic outcomes.

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Primary epithelial amyloid keratopathy with multiple recurrences in grafts

EDITOR,—Primary epithelial amyloid keratopathy (PEAK) is a newly described entity of subepithelial amyloidosis. PEAK is an aggressive variant of primary gelatinous drop-like dystrophy in patients of eastern origin. We describe the clinical and histopathological findings of two brothers suffering from PEAK. The opacities recurred in one after epithelial scrapping and in the other after repeated penetrating keratoplasty.

CASE REPORTS

Two patients of Iranian origin were treated in our department. Light microscopy, histochemistry, and electron microscopy were performed on epithelial scrapping, corneal buttons, and failed grafts.

Clinically, the two brothers presented with deterioration of vision, photophobia, and redness. Examination revealed corneal opacification of the central cornea, which also occurred in the grafts. Histopathological examination disclosed intraepithelial and stromal deposits of amyloid associated with inflammation.

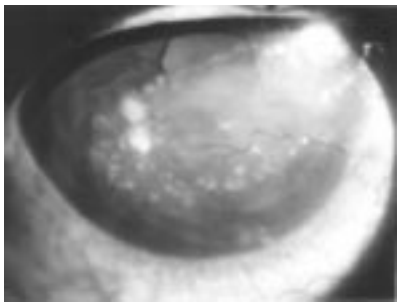

Figure 1 Corneal nodular opacification and neovascularisation seen in PEAK.

The elder brother, now a 50 year old, has been suffering from visual disturbances and photophobia in both his eyes from the age of 14. During the past 20 years he had undergone five penetrating keratoplasties on his right eye and four on his left eye. The indication for surgery was repeated recurrence of the opacities in the graft (Fig 1). In four instances the opacities in his right cornea reappeared in the graft within 1 year after the corneal transplantation. Deposits of amyloid were found by histopathological examination.

The younger brother, now 41 years old, had been complaining of epiphora and deteriorating visual acuity from the age of 18. He underwent right eye corneal scraping and left eye penetrating keratoplasty at the age of 39. There was a short period of relative improvement in visual acuity after the epithelial scraping. However, subsequently, more opacities developed at the centre of the cornea and right eye penetrating keratoplasty was performed 2 years later.

Histopathological examination of the corneal buttons removed during perforating keratoplasties revealed corneal epithelium and stromal amyloid deposits (Fig 2), confirmed by Congo red stain and birefringence. Electron microscopy demonstrated non-branching fibrils of 7.5–10 nm inside the epithelial cells and around them in the area of the amyloid deposit. The deposits in the stroma were associated with an inflammatory infiltrate including granulomatous inflammation showing multinucleated, foreign body giant cells. Amyloid was also recovered from the scrapping material.

There was no known parental consanguinity. They remembered having a grandmother with very poor eyesight, yet they did not know the cause. None of the other six brothers or their children (all are very young) suffer from corneal disease.

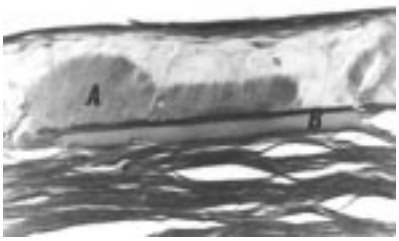

Figure 2 Histopathological picture showing the intraepithelial location of the amyloid deposition in PEAK at the margin of the corneal lesion (centrally it disrupts the basement membrane and the Bowman's layer and is located also in the stroma). A, amyloid; B, epithelial basement membrane and Bowman's layer (periodic acid Schiff, original magnification × 400).

These two brothers with PEAK, represent the relentless behaviour of this disease, with repeated recurrences, within a short period of time after scraping or penetrating keratoplasty.

COMMENT

Primary epithelial amyloid keratopathy (PEAK) is a variant of primary gelatinous corneal dystrophy, first described in 1994 by Edward and co-workers in patients who were of Asian-Indian or Iraqi origin[1]; another variant is primary drop-like corneal dystrophy which has been mainly described in Japan.[2 3] Patients with PEAK suffer from deterioration of vision, redness, and photophobia from childhood or adolescence whereas in primary gelatinous corneal dystrophy symptoms usually start at adulthood. On clinical examination there were corneal subepithelial nodules which tend to progress, associated with stromal opacification and neovascularisation. Histological examination of the corneas demonstrated multiple intraepithelial and subepithelial deposits at childhood while in adults the material was found also in the stroma.

Other patients suffering from PEAK[1 3] were noted to be from India and Iraq, our patients originated from the same geographical area, Iran. It seems that this variant of gelatinous drop-like corneal dystrophy might be more prevalent in mid-Asian countries than was previously known. This variant differs from the one described in Japan in that it manifests at a younger age, is more aggressive and tends to reappear in corneal grafts.

To conclude, PEAK is an aggressive variant of primary gelatinous corneal dystrophy, demonstrating a devastating corneal disease starting at young age and no remedy even after corneal scraping or repeated penetrating keratoplasty.

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CORRESPONDENCE

Shared care postoperative management of cataract patients

EDITOR,—Allan and co-authors raised an interesting question in their recent suggestion that alternatives to conventional postoperative care, including shared care with non-ophthalmologists, should be evaluated.[1] Previous studies looking at this issue have been contradictory. A study by the American Office of Technology looked at the differences in education and training between optometrists and ophthalmologists and concluded that the co-management of postoperative cataract patients may carry "potential risks".[2] In contrast, a large retrospective review of over 2000 co-managed patients concluded that co-managed postoperative care with optometrists "can be successfully organised, coordinated and delivered".[3] Similar suggestions have been made by other authors.[4 5]

In 1994 we carried out a prospective study to determine whether the postoperative management of cataract patients by optometrists is a safe and viable option. The optometrists involved in the study were a selected group who were not only already participating in our local glaucoma monitoring scheme, but also received education, by ourselves, on the care of postoperative cataract patients. In all, 121 patients, who had undergone uncomplicated extracapsular cataract extraction or phacoemulsification, were examined at 6 weeks postoperatively by an optometrist who performed both a refraction and a full ocular examination, following a strict examination protocol. A telephone "hotline" was available for the optometrist to seek advice if any abnormality was found. The patients were then examined by an ophthalmologist following the same examination protocol. The findings of the optometrist and ophthalmologist were compared. There was a high degree of consistency between the examination findings of the two groups, with only minor discrepancies in a few patients over the degree of posterior capsule thickening and age related macular degeneration. Most importantly, there were no clinically significant abnormalities missed by the optometrists. We therefore felt that, in our region, it was both safe and feasible for optometrists to carry out the postoperative management of uncomplicated postoperative cataract patients.

Since this initial study, we have routinely involved optometrists in the postoperative care of cataract patients. A recent audit confirmed the safety and feasibility of the practice. Patients welcome the opportunity to visit an optometrist for their postoperative care, citing improved access and convenience as major advantages. The benefits stemming from a reduction in our routine outpatient workload are obvious.

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Reply

EDITOR,—Booth *et al* highlight some interesting elements of the way in which shared care with non-ophthalmologists could work to reduce the burden of postoperative review after routine cataract surgery.

Open access to an ophthalmologist's opinion in problem cases is clearly a vital safety element for any shared care protocol. How often was their telephone hotline used? To justify shared care, it would be important to demonstrate a genuine cost saving. This would require details of time spent in telephone advice and any additional hospital visits.

No clinically significant complications were missed by optometrists in this study. In addition to screening for problems, review visits represent an opportunity for collecting outcome data (for example, visual acuity, refraction, etc). If routine review is to be devolved to optometrists or nurse practitioners, some mechanism for feeding this data back to the hospital should exist.

Another key issue is patient satisfaction. Booth *et al* note that their patients welcomed local optometric review. How was this assessed?

The complete findings of this and related studies should be published to expand debate in this important area.

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Peripapillary circle of Zinn–Haller revealed by fundus fluorescein angiography

EDITOR,—I read with interest the article by Ko *et al*,[1] in which the authors describe their findings on the morphology of the circle of Zinn–Haller (CZH) obtained with fluorescein angiography. It was not mentioned in the article that by using scanning laser Doppler flowmetry, a non-invasive clinical method, CZH can also be clearly identified.[2] Like Ko *et al*, the authors we found CZH easily visible in healthy myopic eyes within the temporal peripapillary area, especially if peripapillary atrophy was present.[2] Using Heidelberg retina flowmeter images focused on the retinal surface, the temporal part of CZH was seen even in moderate degree myopia (−5.0 D), as well as the centripetal arterial branches, which originate from the circle and lead to the optic disc.[2] It is even more interesting that the arterial perfusion in the CZH was also clearly detectable using scanning laser Doppler flowmetry even if the circle was not visible on the screen because of the retinal capillary network.

In healthy myopic eyes CZH was detected in 83% on the temporal side and in 8% on the nasal side of the disc.[2] This may suggests that CZH is frequently incomplete. In healthy non-myopic eyes the figure was 23% on both sides.[2] In glaucoma the temporal part of CZH was detected in 71% of the myopic eyes and in 75% of the non-myopic eyes. This suggests that retinal thinning or atrophic changes for any reason within the temporal peripapillary area may enhance the detectability of CZH.

Since fluorescein angiography reflects the plasma circulation while scanning laser Doppler flowmetry reflects the moving red blood cells the two methods may provide complementary information for anatomical and functional evaluation of peripapillary perfusion.

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Reply

EDITOR,—We greatly appreciate Dr Hollo's interest in our study. As was mentioned by some authors, the scanning laser Doppler flowmetry or indocyanine green angiography are valuable imaging methods for detecting the peripapillary circle of Zinn–Haller (CZH), especially when myopic crescent or glaucomatous peripapillary atrophy are present.[1 2] In this regard we agree that the chance to detect the CZH by any imaging methods can be enhanced by peripapillary chorioretinal thinning under any circumstances.

According to Olver and associates,[3] although the completeness of the vascular circle was about 77%, this did not mean the functional completeness. However, we should keep in mind the tremendous morphological variations of the CZH in the intraluminal diameter and its distance from the optic disc margin. The CZH does not have uniform intraluminal diameter during its course and does not run in the same plane within the sclera. In our study using flat section of human eyes,[4] the minimal diameter of the vascular circle was 20 μm.

This narrow portion of the CZH may be not detected by scanning Doppler flowmetry owing to resolution or functional incompetence. In addition, we think that the imaging methods have limitations in detecting the deep seated portion of the CZH with minimal peripapillary chorioretinal atrophy and in observing the detailed branching patterns. We agree that it will be interesting to study peripapillary perfusion in patients with normal or high tension glaucoma or anterior ischaemic optic neuropathy by scanning laser Doppler flowmetry.

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Sustained release intravitreal ganciclovir implant as salvage treatment in AIDS related cytomegalovirus retinitis

EDITOR,—Cytomegalovirus (CMV) retinitis affects 12%–46%[1–3] of individuals with AIDS and is frequently bilateral. The current management of CMV retinitis includes systemic anti-CMV therapy, local intravitreal injections of ganciclovir or foscarnet, and, more recently, intravitreal sustained release ganciclovir implant (Vitrasert) (Fig 1). High dose intravenous ganciclovir or foscarnet is effective in suppressing viral replication in the short term but breakthrough infection is common during the maintenance phase with up to 50% of patients experiencing recurrent disease.[4–6] Implantation can be done as primary therapy at the time of CMV retinitis diagnosis or as salvage therapy once the conventional treatment has failed. We describe our experience using Vitrasert as salvage treatment in patients with AIDS related recurrent CMV retinitis.

Data on patients with CMV retinitis treated with Vitrasert implants at two centres, Edinburgh and Belfast, were analysed retrospectively. Twenty five Vitrasert devices were implanted in 16 eyes of 11 patients between August 1995 and May 1997. Before implantation all patients showed recurrent CMV retinitis despite systemic treatment with ganciclovir and/or foscarnet, had central line sepsis, or were intolerant of these medications. The median post-implantation follow up time was 7 months (range 1–15 months). Following the diagnosis of CMV retinitis, median life survival of four patients now dead was 19.5 months (range 4–24), for seven surviving patients this period at the time of writing was 12 months (range 8–22). The median time interval between CMV retinitis diagnosis and initial implantation was 9 months (range 1–19). The median time interval for retinitis progression following first implantation was 6 months (range 2–13).

Post-implantation anti-CMV systemic treatment in the form of oral ganciclovir was continued at a reduced dose in eight (73%) patients and discontinued in three (27%) patients who received bilateral implantation and showed no clinical evidence of extraocular CMV disease. At the last follow up following implantation 13 (82%) eyes remained within one line of their preoperative visual acuity and three (18%) eyes had loss of 2 lines. A final visual acuity of 6/18 or more was retained in 56% eyes. Complications of the implant include mild vitreous haemorrhage in four eyes, cystoid macular oedema in three eyes, moderate anterior uveitis in one eye, and non-progressive macula on nasal rhegmatogenous retinal detachment in one eye, 6 months following implantation.

Figure 1 Ganciclovir implant (Vitrasert).

In this relatively small study the median time interval for retinitis progression was 6 months, similar to a previous study reported by Marx *et al*.[7] However, in an earlier study[8] done in patients with newly diagnosed CMV retinitis, this period was reported to be 7.5 months. Therefore, this preliminary study has shown that the intravitreal ganciclovir implant is effective as salvage treatment in recurrent CMV retinitis in the majority of patients. It avoids the need for repeated intravitreal injections and associated frequent hospital visits, provides a longer period of disease control, and may be used to retain useful vision in majority of patients helping to improve their quality of life. Although the implants are effective in controlling local CMV disease, systemic anti-HIV and anti-CMV therapy should be continued to prevent extraocular disease. The longer term efficacy and complications of this procedure should be studied prospectively in those individuals who fail "conventional therapies".

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Complications of fascia lata harvesting for ptosis surgery

EDITOR,—Wheatcroft *et al* are to be congratulated on their excellent article concerning complications of fascia lata harvesting for ptosis surgery in 24 consecutive patients.[1]

I do have some concern about their statement that leg scarring was considered unsightly in 38% of their patients, but was considered "a minor problem in all cases". Furthermore, they provided a graphic picture of a leg scar with a "poor cosmetic result". I believe that the more litigious American patient might not find this a minor problem. Also, if a keloid were to form at the incision used by the authors superior to the knee joint in the lateral aspect of the thigh (as the majority of surgeons do worldwide), the level of patient satisfaction might be quite low.

The authors did not encounter herniation of the muscle belly or haematoma formation in their series, but do provide references mentioning these problems.[2–4] A larger series of patients may have manifested these complications.

I have had conversations with a number of colleagues who have encountered one or more of the above complications following use of the traditional above the knee incision.

We have published a technique of harvesting fascia lata between the greater trochanter and iliac crest in an attempt to decrease the problems of a conspicuous scar, herniation of the muscle belly, and haematoma formation.[5] As was the case with the authors, we did not encounter these problems in our series.

With this technique a scar, keloid, or herniation of the muscle belly would be covered with short legged wearing apparel, undergarments, or a bathing suit.

With our technique, one can visualise the entire extent of the dissection, especially with the use of a fibreoptic retractor. In the event of a haematoma, increased exposure can aid in solving this problem and also provide visualisation of dissection of the tissue surrounding the fascia to be removed.

We suggest consideration of an incision between the greater trochanter and the iliac crest for harvesting fascia lata.

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Reply

EDITOR,—We are very grateful to Dr Naugle for his comments on our paper. We have read with interest his recent publication[1] which describes the approach to the fascia lata through a high leg incision. From the excellent results reported by Naugle and colleagues this approach is a good alternative to the conventional low incision placed above the knee. However, to date we have no experience with this approach.

Both papers agree that autogenous fascia lata is the best material for routine brow suspension procedures. The main disadvantage of the lower incision is that the scar can be conspicuous. The main advantage of the higher incision is a less conspicuous scar. Although we reported that few of our patients found the scar unacceptable, clearly they would prefer to avoid an obvious scar altogether.

One of the disincentives to the use of autogenous fascia lata is the perceived difficulty in harvesting it. The low incision approach is not difficult to learn and is reasonably quick. We shall certainly use the high incision technique in a series of our patients to assess the relative ease of the surgery.

We congratulate Naugle and colleagues on the introduction of this new technique.

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BOOK REVIEWS

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Ophthalmic Ultrasound—A Practical Guide. By Hatem R Atta. Pp 156. £35. Edinburgh: Churchill Livingstone, 1996. ISBN 044 304 7731.

Ophthalmic Ultrasound—A Practical Guide is a textbook for the practitioner of ophthalmic ultrasound. This 156 page monograph comprises nine chapters that address the use of diagnostic ultrasound in ophthalmology. The first two chapters provide a brief introduction and description of the basic requirements for the echographic examination, including instrumentation (A-scan, B-scan, and Doppler) recommended by the author. Although a chapter on the physics of ultrasound is not offered, some of the ultrasound principles are demonstrated in various areas of the book, mainly through line drawings that accompany the echograms.

Examination of the globe is covered in chapters 3 to 6. Screening techniques are explained through the use of plentiful line drawings, photographs and echograms, and a short section on evaluation of the anterior segment using a simple immersion technique is offered. The book also provides techniques for differentiating intraocular lesions as well as a chapter on measuring the axial length and corneal thickness (corneal pachymetry). In chapter 6, the reader will enjoy the correlation of clinical and ultrasound features for vitreoretinal disease, intraocular tumours and trauma in the presence of both opaque and clear ocular media.

The orbital evaluation is addressed in chapters 7 to 9. These chapters cover the detection and differentiation of vascular malformations and orbital mass lesions as well as evaluation of the extraocular muscles and optic nerves. Also included are brief sections on the lacrimal gland, Doppler ultrasound, and periorbital cavities. Those ultrasonographers who evaluate patients with orbital disease will have a particular interest in chapter 9 which is organised by the common signs and symptoms of orbital disease. Examples of lesions which can cause these signs and symptoms are shown along with a description of their echographic features.

The author, Dr Hatem Atta, is a respected practitioner of ophthalmic ultrasound who has condensed his many years of experience into a useful guide for both the aspiring and seasoned practitioner. His correlation of clinical and ultrasound findings and use of creative line drawings with clinical photographs and carefully selected echograms greatly enhance this book's value to the field of ophthalmic ultrasound.

SANDRA FRAZIER BYRNE

Recent Advances in Ophthalmology 9. Edited by Barrie Jay, Colin M Kirkness. Pp 224. £39.95. Edinburgh: Churchill Livingstone, 1995. ISBN 0443 051275.

With the ever increasing volume of literature on all subjects, this collection of 16 reviews of topical issues in ophthalmology provides a useful concise update for ophthalmologists: for those in training who require a "review lecture" on the subject; and for the more senior surgeon who may wish to keep abreast of subjects not necessarily in his field of special interest. The selection of subjects encompasses difficult clinical problems (for example, management of advanced glaucoma), visits newer techniques which are becoming more widespread (small incision cataract surgery, ocular surface reconstruction, use of botulinum toxin), and also presents reviews of newer diagnostic techniques requiring specialist instrumentation. Some of the chapters provide a wider overview of the subject, while other give detailed instruction for best clinical practice. Clearly written by recognised specialists in their fields, well illustrated and supported by extensive bibliographies, each article introduces the subject with a historical perspective, before describing the newer aspects and rationale for their approach. Tables and flow charts augment the text and provide easily remembered summaries and algorithms for approaching difficult diagnostic problems logically. Where individual techniques are recommended, a detailed method is provided to allow the reader to perform the task. Some techniques proposed by the authors may be familiar in principle, but different in detail and may vary from other contemporary opinions on the subject, or the reader's own experience—for example, the use of mitomycin in enhanced trabeculectomy under the scleral flap. The references in some cases may have been superseded by more recent articles published elsewhere since the production of this book, which is inevitable with books describing rapidly advancing medicine, but the ophthalmologist reading these review articles will have a good authoritative background article on which to build future knowledge.

J A SCOTT

NOTICES

1997 Lewis Rudin Glaucoma Prize

The 1997 Lewis Rudin Glaucoma Prize awarded for outstanding research has been given to Joseph Caprioli, MD, of UCLA School of Medicine, for glaucoma research published in 1996 in *Investigative Ophthalmology & Visual Science* (November). The research explored the possibility that the body's own defence mechanisms could be brought into play to protect the optic nerve cells against damage in times of stress.

Residents' Foreign Exchange Programme

Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, Secretary of the Foreign Exchange Committee, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.

The Cullen Course 1998. Clinical Advances in Ophthalmology for the Practising Ophthalmologist

Baylor College of Medicine, The Cullen Eye Institute, Department of Ophthalmology presents the Cullen Course 1998, Clinical Advances in Ophthalmology for the Practising Ophthalmologist, at the Houstonian Hotel and Conference Center, 111 North Post Oak Road, Houston, Texas on 6–8 March 1998. Further details: Carol J Soroka, Conference Coordinator, Office of Continuing Education, Baylor College of Medicine, One Baylor Plaza-S104, Houston, TX 77030, USA. (Tel: (713) 798-5600).

2nd International Glaucoma Symposium (IGS)

The 2nd International Glaucoma Symposium will be held on 15–20 March 1998 in Jerusalem, Israel. Further details: The 2nd IGS Secretariat, PO Box 50006, Tel Aviv 61500, Israel. (Tel: +972-3-514-0000; fax: +972-3-517-5674; email: glaucoma@kenes.com)

15th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 15th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 15–20 March 1998 at Manor Vail Lodge, Vail, Colorado. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical Education, Turner 20/720 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage:http://ww2.med.jhu.edu.cme)

American Institute of Ultrasound in Medicine

The American Institute of Ultrasound in Medicine is holding its 42nd annual convention on 22–25 March 1998 at the Hynes Convention Center, Boston, MA, USA. A pre-convention course entitled "Ultrasound and women's health" will take place on 21–22 March, and a pre-convention tutorial called "Vascular ultrasound" will be held on 22 March. Further information: AIUM, 14750 Sweitzer Lane, Suite 100, Laurel, MD 20707-5906, USA. (Tel: (301) 498-4100; fax: (301) 498-4450).

Globe 98—International Telecommunication Live-Surgery Event

Globe 98, the International Telecommunication Live-Surgery Event will be held on 27–28 March 1998 in Innsbruck, Austria. Further details: International Telecommunication Live-Surgery Network (ILSN), Fürstenweg 165, A-6020 Innsbruck, Austria. (Tel: 0043-512-286688 or 0043-512-581860; fax: 0043-512-264838; email: ilsn@net4you.co.at; homepage:http://www.carrier.co.at/ilsn/)

Leonard Klein Foundation

The Leonard Klein Foundation bestows the Leonard Klein Award for innovative scientific works in the field of development and application of microsurgical intruments as well as for microsurgical operating techniques. The award is endowed with 30 000 DM. Five copies of the work have to be submitted in English or German by 31 March 1998 to Stifterband fur die Deutche Wissenschaft e V, Herrn Peter Beck, Postfach 16 44 60, D-45224 Essen, Germany.

Wilmer Ophthalmological Institute

The Johns Hopkins Medical Institution/ Residents Association of the Wilmer Ophthalmological Institute is holding its 57th clinical meeting at the Baltimore-Turner Auditorium, JHH on 1–2 May 1998. Further details: Ms Sharon Welling, Conference Coordinator, Wilmer B20 - Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21287-5001, USA. (Tel: 410-955-5700; fax: 410-614-9632).

4th International Vitreoretinal Meeting

The 4th International Vitreoretinal Meeting will be held in Parma, Italy on 29–30 May 1998 at the University Eye Clinic. Further details: C Cantù and M A De Giovanni, Institute of Ophthalmology, University of Parma, Via Gramsci 14 - 43100 Parma, Italy. (Fax: ++39.521.292358; email: gnuzzi@rsadvnet.it)

11th Annual Meeting of German Ophthalmic Surgeons

The 11th Annual Meeting of German Ophthalmic Surgeons will be held on 28–31 May 1998 in the Meistersingerhalle, Nürnberg, Germany. Further details: Organisation Nürnberg GmbH, Wielandstrasse 6, D-90419 Nürnberg, Germany. (Tel: +49-911-393160; fax: +49-911-331204).

9th British Association of Day Surgery Annual Scientific Meeting and Exhibition

The 9th British Association of Day Surgery Annual Scientific Meeting and Exhibition will take place at the Harrogate International Centre on 4–6 June 1998. Further details: Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, W Yorks HD7 3AP. (Tel: 01484 854575; fax: 01484 854576; email info@kitecomms.co.uk)

XVIIIth International Congress of Ophthalmology

The XXVIIIth International Congress of Ophthalmology will be held in Amsterdam on 21–26 June 1998. Further details: Eurocongres Conference Management, Jan van Goyenkade 11, 1075 HP Amsterdam, Netherlands. (Tel: +31-20-6793411; fax: +31-20-6737306; internet http://www.solution.nl/ico-98/)

First Combined International Symposium on Ocular Immunology and Inflammation

The First Combined International Symposium on Ocular Immunology and Inflammation will be held in Amsterdam on 27 June–1 July 1998. The meeting is sponsored by the International Ocular Immunology and Inflammation Society, the International Uveitis Study Group, and the Immunology and Immunopathology of the Eye Organisation. Further details: Professor Aize Kijlstra, The Netherlands Ophthalmic Research Institute, PO Box 12141, 1100 AC Amsterdam, Netherlands (email: a.kijlstra@amc.uva.nl)

2nd International Conference on Ocular Infections

The 2nd International Conference on Ocular Infections will be held on 22–26 August 1998 in Munich, Germany. Further details: Professor J Frucht-Pery, 2nd International Conference on Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077; email: ocular@kenes.com)

ICOP 98

The next International Conference in Ophthalmic Photography (ICOP) will be held on 19–21 September 1998. Further details: Mrs Gillian Bennerson, Senior Ophthalmic Photographer, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX. (Tel: 0117-928-4677).

Correction

An author's error occurred in the editorial in the December issue of the *BJO* (1997; **81**:1025). In the fourth paragraph, line 9, in the sentence that states "The definitive surgical treatment seems to be a combination of ipsilateral inferior rectus strengthening, ipsilateral superior rectus weakening, and contralateral *inferior oblique* weakening, all muscles on adjustable sutures", the muscle specified should be *inferior rectus*. The author apologises for any confusion caused.

INSTRUCTIONS FOR AUTHORS

Adherence to the following guidelines is essential if efficient and expeditious processing of your manuscript is to be achieved. Manuscripts will be returned to authors for revision before peer review if they are submitted in incorrect format. Please indicate in a covering letter which category of paper your article represents.

The *British Journal of Ophthalmology* is an international journal covering all aspects of clinical ophthalmology and the visual/ophthalmic sciences. Contributors should consider the widely varying readership and write clear, simple articles with the minimum of technical detail. Space in the journal is limited and articles should therefore be as concise as possible. One page of text is approximately 1000 words.

Manuscripts should be sent to the editor who selects them on the basis of their suitability for the journal and of reports from independent referees. Manuscripts are acknowledged on receipt and the majority (>80%) are sent for review. Those that are not reviewed are returned to the author as rapidly as possible so that they may be submitted elsewhere.

Manuscripts may be processed by section editors who deal with specific areas of ophthalmology including surgical retina, medical retina, neuro-ophthalmology, glaucoma, paediatric ophthalmology, ocular motility, orbital disease, anterior segment disease, oncology, lens, optics and visual sciences, laboratory sciences, pathology, and immunology. A minimum of two referees, chosen for their specific expertise, review each article.

Papers are accepted on the understanding that they have not been and will not be published elsewhere, and that there are no ethical problems with the work described. If requested, authors shall produce the data upon which the manuscript is based for examination by the editor.

Categories of papers

ORIGINAL ARTICLES

(a) Clinical science

Articles on clinical topics are research reports of a general or specialised nature comprising approximately 3000 words and 4-6 display items (Figures and Tables).

(b) Laboratory science

Articles on ophthalmic or visual sciences are research reports of experimental work generally of the same size as clinical research reports. Laboratory science papers will be included in a designated section of the journal.

Both types of original article should include the following: title; key words (up to four); address and which author address for correspondence; structured abstract (approx 200 words, headings 'Aims/background', 'Methods', 'Results', and 'Conclusion'); introduction; materials and methods; results and discussion sections; references and acknowledgements; legends for display items (Figures and Tables).

REVIEW ARTICLES

Substantive review articles will be included under the section 'Perspective' and will address any aspect of clinical or laboratory ophthalmology. Review articles will be approximately 3000-5000 words in length including references and may contain display items (Figures and Tables). Most review articles are commissioned but uninvited reviews are welcomed. Prior discussion with the Editor is recommended. All reviews are subject to independent refereeing.

LETTERS TO THE EDITOR

Case reports will be published as 'Letters to the editor'. These are normally 500–600 words written in the form of a letter with a maximum of two display items (Figures and Tables). The letter should include an introductory section (without heading), the case report (heading: Case report) and a comment (heading: Comment), plus a maximum of 10 references.

CORRESPONDENCE

Letters are normally constructed in the form of scientific correspondence and are usually 200–300 words.

Preparation of manuscripts

Manuscripts will be received on the understanding that they have not been and will not be published elsewhere while under editorial review. Manuscripts may be subject to editorial revision with the author's agreement. All communications should be sent to the Editor, *British Journal of Ophthalmology,* Department of Ophthalmology, University of Aberdeen Medical School, Foresterhill, Aberdeen AB9 2ZD, Scotland, UK. (Tel: 01224 663812; Fax: 01224 663832.)

Manuscripts must be submitted in triplicate, and typed double spaced on one side of the paper only, with one inch margins. Each author must sign the covering letter as evidence of consent to publication. Revised manuscripts should be submitted as hard copy and on disk. Detailed instructions will be sent to authors on invitation to revise.

ILLUSTRATIONS

Illustrations must be submitted in triplicate. Transparencies must be accompanied by prints. Only salient detail should be included. All must be labelled with the author's name, numbered in the same order as they are cited in the text irrespective of whether they are in colour or black and white, and have the top indicated. Radiographs must be submitted as prints. Line drawings should be clearly labelled and will be redrawn to house style. The width of illustrations for the original articles should be 68 mm, 104 mm, 140 mm or, in exceptional circumstances, 176 mm, to fit the column layout of the journal. Illustrations for 'Letters to the editor' should be 56 mm or 116 mm. Stain used and a scale bar (or magnification) should be given. Legends must be typed on a separate sheet.

TABLES

Each table should be on a separate sheet, have a heading, and contain no vertical rules.

REFERENCES

In accordance with the Vancouver agreement references are cited by the numerical system. They must be *typed double spaced.*

References in the text must be cited in numerical order of first appearance. References in the list must be given in the numerical order in which they first appear in the text, not in alphabetical order of authors' names. References with one to three authors must include all authors' names; for references with more than three authors the first three should be given and then *et al*. Titles of journals should be abbreviated in accordance with the *Index Medicus* or given in full.[1] References to books must include names of editor(s) if there is one, town where published, name of publisher, year, volume, page numbers.[2]

1 Kaye SB, Shimeld C, Grinfield E, *et al.* Non-traumatic acquisition of herpes simplex virus infection through the eye. *Br J Ophthalmol* 1992; **76**:412-8.
2 Jakobiec FA, Font RL. Orbit. In: Spencer WB, ed. *Ophthalmic pathology: an atlas and textbook.* 3rd ed. Philadelphia: Saunders, 1986:2461-76.

References will not be checked in the editorial office. Responsibility for their accuracy and completeness lies with the author.

SI UNITS

The work should be reported in the units used. If these were not SI units, the equivalent in SI units should be given in parentheses.

STATISTICS

Particular attention should be paid to the description of any sample selection process; in particular, the representativeness of the sample should be argued and the handling of any missing data justified. Authors are asked to check tables etc to ensure that missing data are accounted for, that percentages add up to 100 and that numbers in tables are not at variance with those quoted in the text. The policy of the *British Journal of Ophthalmology* is based on the statistical guidelines published in the *British Medical Journal* in 1983 and these are a useful source of information for authors (Altman DG, Gore SM, Gardner MJ, Pocock SJ. Statistical guidelines for contributors to medical journals, *BMJ* 1983; **286:** 1489-93). Blanket statements on the use of statistical techniques should be avoided; it must be made quite clear in context which procedure is being used. Authors should bear in mind that relatively simple analyses are often quite adequate to support the arguments presented.

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