LETTERS TO THE EDITOR

Linear scleroderma with orbital involvement: follow up and magnetic resonance imaging

EDITOR,—Scleroderma is a chronic connective tissue disease of unknown aetiology.[1] Linear scleroderma is an uncommon dermatological disorder which produces an unilateral and localised band-like linear atrophy of the skin and its underlying structures.

Orbital involvement has been described, but is very rare.[1 2]

CASE REPORT

A 21-year-old woman first presented to an ophthalmologist because of a progressive narrowing of the right palpebral fissure over a period of 3 years with loss of lashes of the eyelids. Further ophthalmic examination was normal. An orbital computed tomogram (CT) was normal.

Three years later there was additional enophthalmia and an entropion of the lower right eyelid was seen (Fig 1). Visual acuity was 8/10 in the right eye and 10/10 in the left. Visual field examination and colour vision were normal.

With the exception of a localised area of parietotemporal alopecia on the right side, no other skin defects could be detected. General examination revealed no muscular pain, no arthralgia, no fatigue, and no other systemic symptoms. Laboratory examination of muscle enzymes, tissue antibodies and complement factors was normal. There were no inflammatory factors and thyroid tests were within normal limits.

On an orbital CT the right eye was displaced laterally by a band-like mass. The medial rectus muscle and oblique superior muscle could not be identified. Muscle and conjunctival biopsy only showed some oedema in these structures.

The presumed diagnosis of localised scleroderma was confirmed by a dermatological examination. Initially no treatment was established.

An orbital magnetic resonance image (MRI) confirmed the CT findings but also demonstrated that the right superior rectus muscle was involved in the mass (Fig 2A and B). A new biopsy only showed muscular and fibroid tissue without signs of inflammation.

Two years after the first admission the patient complained of increasing right sided headache, photophobia, and progressive decrease of the visual acuity on the right side from 8/10 to 3/10. Visual field examination and colour vision were normal. Biomicroscopic examination showed a punctate keratitis on the right side. Funduscopy revealed a hyperaemic optic disc. Follow up MRI showed a progressive increase in the size of the orbital mass with extension into the apex. The optic nerve could only be identified following intravenous gadopentetate dimeglumine injection (Fig 2C–F). A canthotomy and an eyelid plasty were performed for restoration of the right upper eyelid (Fig 3).

The unilateral loss of vision progressed slowly to finger counting at 1 metre the following year. The visual fields showed normal peripheral limits, but colour vision was absent in the right eye. Cortisone therapy (Medrol, 32 mg/day) and radiotherapy (4 Gy on the right orbit, followed by 20 Gy in fractions of 2 Gy) were initiated, but the patient complained of increasing pain in and around the right eye. The patient became blind on the right side. Ophthalmic examination showed a mydriatic non-reactive pupil and a pale optic disc on the right side. Two follow up MR examinations did not show a further progression of the orbital abnormalities.

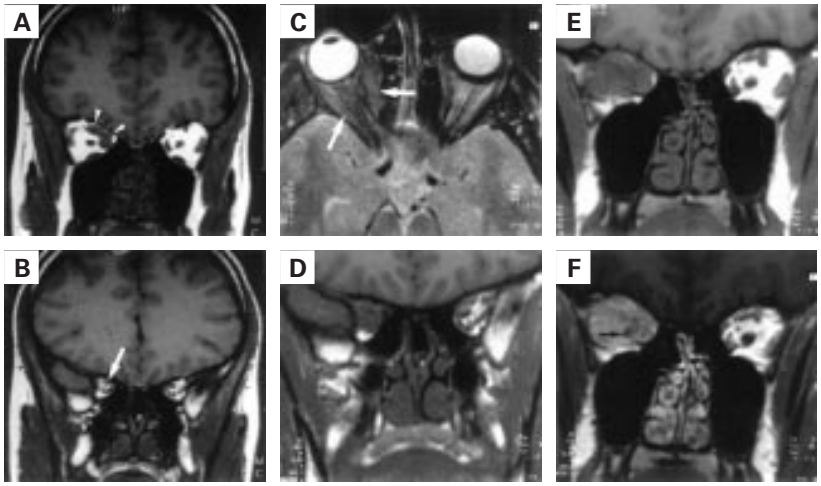

Figure 2 (A,B). Coronal T1 weighted MRI (TR/TE = 520/15 ms). The superior rectus muscle, the superior oblique muscle, and the medial rectus muscle on the right side appear enlarged and cannot be distinguished from each other (A) (arrowheads). The orbital apex is not involved (B) (arrow). (C) Axial T2 weighted MRI (TR/TE = 2200/80 ms). (D, E) Coronal T1 weighted MRI (TR/TE = 500/15 ms). (F) Coronal T1 weighted MRI after gadopentetate dimeglumine injection (TR/TE = 500/15 ms). The orbital abnormalities return a low signal on T2 weighted images (C) (arrows). The apex is involved and the entire orbital fat is infiltrated (D,E). Note that the optic nerve is only visible after the contrast administration (arrow) (F).

COMMENT

Scleroderma may occur as a systemic disease or as a localised form. Localised scleroderma presents in three clinical forms: generalised, morphoea (atrophic and sclerotic skin lesions), and linear scleroderma. In the linear form the lesions are arranged in a band-like linear distribution and may involve and fix the deeper layers of the skin and underlying structures. If it involves the frontoparietal region it is called scleroderma 'en coup de sabre' because of the depression that extends below the level of the skin. In patients with morphoea or linear scleroderma, the disease progresses slowly and may last from a few months to many years, but the prognosis is worst in the en coup de sabre type. According to some authors, it may lead to homolateral facial hemiatrophy (Parry–Romberg syndrome).[1] However, the relation between linear scleroderma en coup de sabre and Parry–Romberg syndrome remains unclear.[3]

Olver *et al* have reported a patient with bilateral acquired Brown's syndrome and combined lichen sclerosus et atrophicus and morphoea.[4] They suggest that the deep subdermal fibrosis may limit the passive movement of the superior oblique tendon. The clinical features of Brown's syndrome were not present in our patient, but can be seen in localised scleroderma.

The cutaneous pathological features depend upon the stage of the disease: the striking accumulation of inflammatory cells among collagen bundles of the lower dermis and among the fat cells and collagen bundles of the subcutaneous tissue is followed by the replacement of the subcutaneous tissue by hyalinised connective tissue.[1]

Involvement of the orbit and the eye is uncommon but has been described.[1 2] Involvement of the extrinsic eye muscles is extremely rare and only CT findings have been reported.[2]

The clinical findings in this case are typical of localised linear scleroderma: the age and sex of the patient, the slow progression, the distribution and nature of the superficial

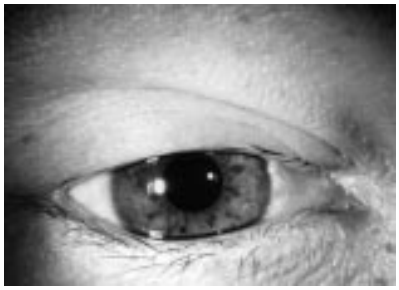

Figure 1 Right eye: note the narrowing of the palpebral fissure and the loss of lashes of the eyelids.

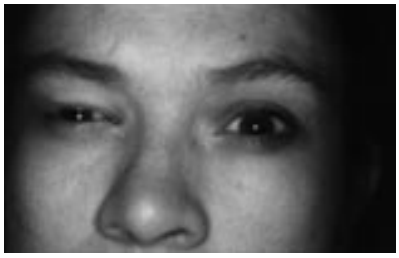

Figure 3 Clinical photograph taken after the two minor surgical procedures shows the progression of the disease.

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defects, and the unsuccessful therapy. This patient has a significant induration of the scalp but there is no extension below the forehead and is therefore not considered as a Parry–Romberg syndrome. The term pseudotumour is misleading and inappropriate because of the presence of enophthalmia and the absence of mass effect.[5 6] The latter can be explained by the atrophy of orbital fat that is only partially replaced by fibrosis. Orbital fat atrophy is a basic mechanism of enophthalmia, and its relation to linear scleroderma has already been reported.[7 8] The thickening of the orbital muscles in this case can probably be secondary to orbital fat atrophy and fibrosis.[2]

Although the imaging findings are not specific, important information can be obtained. A precise localisation of the orbital abnormalities, a clinicoradiological correlation and the follow up of the abnormalities can be achieved.

The CT findings are non-specific.[9] In this patient the lesion is of the chronic and infiltrative or diffuse type. It does not invade or distort the globe and there is no bone erosion.[5 6]

MRI findings in patients with a non-specific orbital inflammatory condition have been described as hypointense to fat and isointense to muscle on T1 weighted images. On T2 weighted images the abnormalities were isointense or slightly hyperintense to fat in nine out of 10 cases.[10] We found similar signal characteristics in this patient. These appearances were markedly different from those in other diseases, including malignancy and haematoma, which appeared markedly hyperintense to fat on T2 weighted images.[10] The use of gadopentetate dimeglumine was helpful in assessing the compression by the lesion on the optic nerve.

To our knowledge, this is the first report on follow up of localised linear scleroderma by MRI. The abnormalities on MRI correlate with the clinical progression. MRI might be better to distinguish between orbital fat atrophy, oedema, and fibrosis but more experience is needed to know if MRI can distinguish the type and the activity of an inflammatory condition.

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Penetrating eye injury caused by a golf tee

EDITOR,—We report a case of penetrating eye injury caused by a golf tee. We feel that this may have been a one off event; however, the paucity of information in the literature regarding ocular golf injuries makes it difficult to comment on its significance.

CASE REPORT

A 16-year-old male was playing pitch and putt, and while teeing off he felt something go into his right eye. The eye became painful immediately and the vision blurred. He removed the object from his eye, and it turned out to be his golf tee. The club he was using was a medium lofted (seven) iron and the surface underfoot was a rubber mat. The tee was a standard plastic golf tee (Fig 1).

On examination of the right eye there was a 2 mm penetrating wound at the limbus inferonasally with a prolapsing knuckle of iris. He was brought to theatre where the conjunctiva was opened and the wound explored. The wound did not extend beyond the limbus and the lens did not appear to have been injured. The iris was reposited and the wound repaired with two 10-0 nylon sutures (Fig 2). The postoperative recovery was uneventful and he was discharged home 2 days later. The eye healed up well over the ensuing weeks with a rapid return of vision to 6/5.

COMMENT

In recent times golf has become an increasingly popular pastime among people from all walks of life in many developed countries.[1] The number of 'teeings off' every year in Ireland alone probably runs into several millions or even hundreds of millions. In this unit we have seen many golf injuries though these are usually from golf clubs or balls, but we have never before seen one caused by a golf tee. We could find only limited references to eye injuries among golfers in the literature. Most were part of larger series of penetrating eye injuries[2] or sports injuries.[3] Only one recent article specifically relates to ocular golf injuries.[4] Cole *et al* in a series of 378 penetrating eye injuries reported that two of these occurred in golfers; however, no further details are provided.[2] A study of sporting eye injuries from Sussex Eye Hospital found two eye injuries as a result of golfing accidents over an 18 month period; golf injuries were among the most severe injuries seen and caused the greatest degree of visual loss.[3] This article mentions that the single worst injury seen was caused by a golf ball; the nature of the second golfing injury is not specified. A series from the USA reported nine golf related ocular injuries over a 6 year period.[4] Seven of the injuries were caused by golf balls and the remainder by clubs. Six out of the nine eyes were enucleated, two as a primary procedure.

Golf related eye injuries are uncommon. The United States Eye Injury Registry stated in its 1994 report of 6500 injuries that 7% (455) were sports related.[5] Of these sporting injuries 4% (18) were golf related. All the eye injuries that have been reported were caused by either golf balls or clubs, none were caused by tees.[3 4] Using lofted irons to tee off from rubber mats may be hazardous though this may have been a unique event. It is also possible that plastic tees are somehow less safe than wooden ones. Accurate reporting would help establish the incidence of eye injuries among golfers as well as demonstrating which ones are freak events and which are repeatable and so requiring preventative measures.

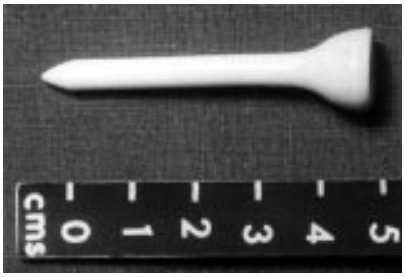

Figure 1 Standard plastic golf tee responsible for the penetrating injury.

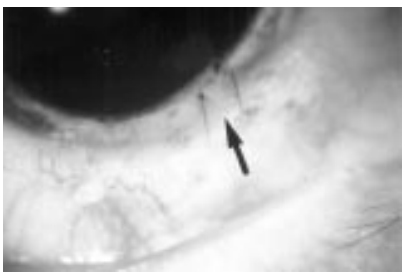

Figure 2 The right eye showing the injury site 24 hours after repair (arrow).

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Preretinal 'hypopyon'

EDITOR,—Retinal inflammations generally present in varied but fairly characteristic patterns. The advent of penicillin almost wiped out syphilis; however, there has been an increase in the incidence of syphilitic infections reported over the past decade. This protean infection can mimic many others and produces unusual lesions anywhere in the eye.

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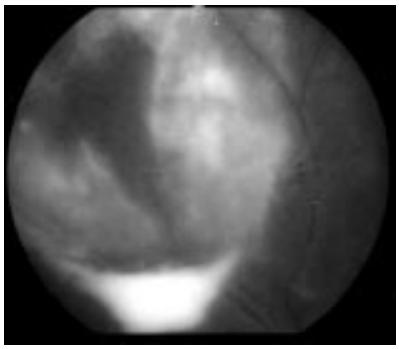

Figure 1 Fundus photograph of the preretinal 'hypopyon', right eye. Large yellow white preretinal 'hypopyon' obscuring the superotemporal retinal vessels. Vessels above this are completely or partially sheathed and have an overlying preretinal exudation and vitritis.

CASE REPORT

A 20-year-old male patient presented with hazy vision and pain in the right eye for 2 weeks. There was no history of similar complaints in the past. The patient did not suffer from oral or genital ulcers or any other systemic illnesses.

On examination, the visual acuity was 20/200 in the right eye and 20/20 in the left. The anterior segment was essentially within normal limits.

On examination of the right fundus (Fig 1) the patient was found to have a yellow-white preretinal exudation and vitritis over 5–6 disc diameters above the macula and over the superotemporal retinal vessels. The inferior part of this exudation was in the form of a preretinal 'hypopyon'. The surrounding vessels, both arterial and venous, showed active vasculitis, and the disc was congested and full, with peripapillary retinal oedema. There were folds around the macula.

The patient was investigated serologically before starting any medication. His total leucocyte count was 9.7×10^6/l, the differential count had 87% neutrophils and his erythrocyte sedimentation rate was 14 mm in first hour. The Mantoux test and IgG/IgM for *Toxoplasma* were negative. The fluorescent treponemal antibody absorption test was positive and the VDRL was positive in a titre of 1:16. All tests for HIV were negative. The cerebrospinal fluid analysis was normal. The fluorescein angiogram showed an early and marked leak from the disc. The superotemporal vessels were tortuous and irregular with a perivascular leak which increased over time leading to hyperfluorescence in the area of the preretinal 'hypopyon' (Figs 2–4).

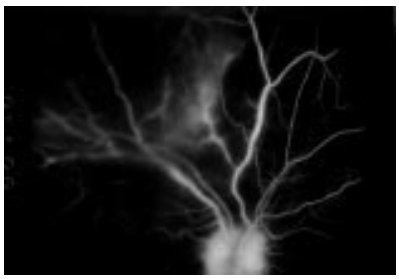

Figure 2 Fluorescein angiogram at 14.5 seconds shows a marked leak from the disc and tortuous, irregular vessels in the superotemporal fundus with perivascular leakage of fluorescein.

Figure 3 Fluorescein angiogram at 31.7 seconds shows increased hyperfluorescence in the superotemporal fundus and blocks fluorescence above the macula.

The patient was given penicillin G benzathine 2.4 million units intramuscularly on the first day and weekly for 2 weeks. After 2 days of the penicillin therapy the vision in the right eye improved to 20/40 and the vitreous exudation had cleared, revealing extensive sheathing of blood vessels and focal infiltrates in the preretinal region. The preretinal 'hypopyon' was also more localised. One week after institution of the penicillin therapy the 'hypopyon' had resolved completely leaving the superotemporal retinal oedematous with narrowed and sheathed venules and arterioles. Preretinal focal infiltrates persisted above the macula, temporal to the disc and in the superotemporal retina. The disc oedema had also resolved to a large extent, though the margins were still blurred and minimal peripapillary oedema persisted (Fig 5). After 4 weeks the vision was 20/20 with no deficit on an Amsler grid evaluation and the focal infiltrates had completely resolved, leaving a mottled superotemporal retina, narrowed and sheathed vessels superotemporally, and a pale disc with ill defined margins.

COMMENT

The clinical manifestations of acquired syphilitic neuroretinal involvement are described as a patchy diffuse retinal oedema with associated vitritis, vasculitis (generally arterial), and occasionally haemorrhages.[1–5] Preretinal exudates were described by Calmettes and Deodati in 1947, as quoted by Duke-Elder, but are not a common feature of this disease.

Behçet's disease generally presents with a hypopyon in the anterior chamber but involvement of the posterior segment in the form of an arterial occlusion can lead to infarction of large areas of the retina and is accompanied by intense retinal oedema, haemorrhage, and vitreous opacification which resemble the retinitis and vitritis commonly seen in secondary syphilis.[6]

A retinal pseudohypopyon-like picture has been described in patients with Best's vitelliform dystrophy where partial resorption of the premacular yellowish material simulates an exudative reaction. A subretinal hypopyon was reported in a patient with prolymphocytic leukaemia, wherein the contents were presumed to be an accumulation of abnormal lymphocytes.[7]

Figure 4 Fluorescein angiogram at 193.9 seconds shows a further increase in the hyperfluorescence, now setting down into the area of the hypopyon.

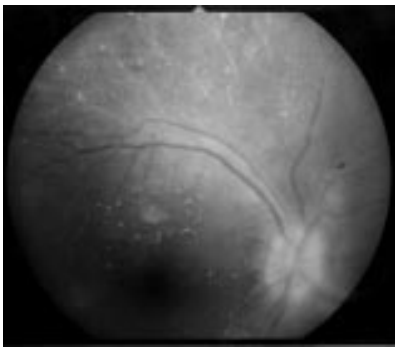

Figure 5 Fundus photograph of the right eye a week after penicillin therapy. There are localised, white preretinal infiltrates seen scattered over the temporal and superotemporal fundus. Narrowing and sheathing of both arterioles and venules is present above the superotemporal arcade. The disc is still congested and oedematous.

Our patient had severe syphilitic retinal vasculitis with a hitherto unreported picture of a preretinal 'hypopyon' which cleared dramatically in response to penicillin therapy.

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CORRESPONDENCE

'Visual fields by numbers' for the detection of glaucoma

EDITOR,—In his interesting article on the seven million people blind from glaucoma worldwide, Quigley highlights the need for better detection of cases with moderate and severe glaucoma damage and hence the greatest risk of blindness.[1] In his editorial, Rait correctly states that the costs of standard methods of glaucoma screening, including computerised perimetry, are prohibitive in most developing countries so that glaucoma remains undiagnosed until very late in its course.[2] A simple and inexpensive visual field test has been designed[3] specifically for the detection of moderate and severe glaucomatous visual field loss in situations precluding other methods, but Rait seems to dismiss this technique as 'less reliable', quoting an article[4] that is not representative of the published literature.

The oculokinetic perimetry (OKP) glaucoma test, to which Rait referred, is a handheld card, with 26 numbered fixation targets arranged in a spiral around a central, black spot. The patient is asked to look at each number for 1 second and to state whether or not the spot is seen. If any number is associated with disappearance of the spot, the test result is abnormal. Wishart reported only a 60.5% sensitivity and 61.5% specificity when 56 patients were examined by a technician in hospital.[4] In contrast, Greve and Chisholm in Canada reported 95% and 93% concordance rates in 584 sector for sector comparisons with the Friedmann analyser and 994 sector comparisons with the Humphrey analyser respectively.[5] The 26 point OKP test took under 1 minute in normal eyes, which was about a third of the time taken with the other methods. Sponsel and colleagues from Prevent Blindness America reported that the 26 point test detected 92% of 48 patients with moderate to severe visual field loss, with a false positive rate of 3.6%.[6] Statistically, these results were not significantly different from those obtained with the Henson visual field analyser. Christoffersen *et al* reported a 94% specificity, when 185 patients were examined by medical secretaries in a general practice in Norway, mentioning two cases detected by the secretaries with OKP but missed by general practitioners with the Bjerrum tangent screen.[7]

The OKP chart used in these studies had a single, black stimulus to make the cost low enough for worldwide distribution free of charge by a pharmaceutical company. This device (now called the 'multifixation campimeter') has been improved, now incorporating a disc, which is dialled by the examiner so as to make a 3 mm grey spot of the desired contrast (10% or 25%) appear briefly in a central window. Twenty points in the central 28° field are examined, with two extra points in the normal blind spot to check reliability. Compared with the version it supersedes, this test is easier (for both patient and examiner) and more 'foolproof', with interim results of an ongoing evaluation indicating greater sensitivity and specificity (C H Fenerty and B E Damato, unpublished data).

Independent evaluations of the new multifixation campimeter are required, including studies performed under normal working conditions.

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Reply

EDITOR,—Damato and Fenerty are to be congratulated on providing a new and improved multifixation campimeter, 'easier' and 'more foolproof' than its predecessor. I have no doubt that 95%/93% sector concordance is possible with the original device[1] but it can only detect moderate to severe visual field loss with any reliability.[2 3]

I understand that the original Damato stimulus is roughly equivalent to a size III Humphrey stimulus of 19dB (Patella M, Zeiss–Humphrey Instruments, personal communication). This calculation is based on the contrast ratio between the white card and the black ink, plus a Goldmann type conversion for size difference. Such a stimulus value leads to a very specific but not very sensitive screening level when applied to a population at large. Obviously it is useful to have a specific but not very sensitive test to detect moderately severe glaucoma but it is not ideal. It is hoped that the sensitivity of disease detection has been improved in the new version and ultimately field trials on an appropriate population should confirm or deny this.

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Association of HLA type with pseudoexfoliation of the lens capsule

EDITOR,—I read with interest the recent article by FitzSimon *et al*.[1] The finding of association of particular HLA types in pseudoexfoliation assumes importance in view of reports of possible cross reaction between antigens of infectious agents and particular HLA types in disorders such as ankylosing spondylosis.[2] Genetic predisposition in association with infectious disease in the aetiology of anterior uveitis is another example.[3] Hence, the following points need attention in drawing any conclusions from the study.

In apprehension of bias towards null hypothesis, sampling bias has led to systematic differences between study populations and general populations, posing a threat to external validity. This could prevent generalisation in the population under study. Deliberate introduction of selection bias between cases and controls also resulted in systematic differences between comparison groups posing a threat to internal validity and thus invalidating the conclusions of the study with regard to the Irish population. No control measures were employed for confounding variables such as solar radiation; the association between HLA type and solar radiation thus confounding one another. Though the type of solar radiation is the same in both cases and controls, the duration of solar radiation is not the same in both groups.

By selecting age-matched controls without pseudoexfoliation and performing ocular examination, correlation could have been more acceptable. It is preferable to risk bias towards a null hypothesis rather than introducing sampling or selection bias in a controlled study. In an earlier study both sampling and selection biases were avoided.[4]

The authors have mentioned multifactorial inheritance of pseudoexfoliation. No hereditary pattern has been clearly established so far.[5] Rather, association of HLA typing with multifactorial environmental influences, including solar radiation, and probably an association with infectious disease is more plausible. Finally, the reason for quoting a prevalence of 1.33% in the general population over 50 years on p 402 and in the population over 40 years on p 404 is not clear.

A carefully planned case-control study or, preferably, a prospective cohort study could establish a cause and effect relation of HLA status and pseudoexfoliation.

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1 FitzSimon JS, Mullvihill A, Kennedy S, Finch A, Collum LMT, Eustace P. Association of HLA type with pseudoexfoliation of the lens capsule. *Br J Ophthalmol* 1996;**80**:402–4.
2 Ebringer A. The link between genes and disease. *New Scientist* 1978;**79**:865.
3 Forrester JV. Endogenous posterior uveitis. (Mini review) *Br J Ophthalmol* 1990;**74**:620–3.
4 Slagsvold JE, Nordhagen R. The HLA system in primary open-angle glaucoma and in patients with pseudoexfoliation of the lens capsule (exfoliation of fibrillopathia epitheliocapsularis). *Acta Ophthalmol* 1980;**58**:188–92.
5 Layden WE. Exfoliation syndrome. In: Ritch R, Shields MB, eds. *The secondary glaucomas.* St Louis: CV Mosby, 1981:99.

Reply

EDITOR,—We thank Dr Seshubabu for his interest in our work and would like to make the following points.

In all, 128 cases with pseudoexfoliation (PXF) were identified on ophthalmic examination and the control group was 11 805 blood donors. While we did not specifically ask patients about solar or outdoor exposure, we believe our large numbers and representatives from both sexes and rural and town populations in each group reduced the risk of sampling a subset of the population. We believe that possible differences in the case

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and control populations are not valid in the small, uniform, and racially homogeneous population of this island.

Solar radiation may be a factor in the pathogenesis of PXF but it is difficult to explain the different frequencies in populations of the world. It is possible that solar radiation in an individual with a certain HLA profile may lead to PXF. Likewise, specific HLA antigens may confer disease susceptibility and infection could lead to development of PXF, but this is speculative only. An HLA association would be an integral part of the pathogenesis.

In the earlier study[1] age-matched controls were used but the study numbers were much smaller. Age-matched controls in such a study would have been ideal but this was an opportunistic study in a genetically relatively isolated population.

A population based study in Ireland documented the prevalence of PXF as 1.33% in cases over 40 years old; therefore some controls may have had undetected PXF but, as stated, this biases towards the null hypothesis.

We agree that prospective cohort studies would clarify the cause and effect relation between PXF and HLA and emphasise that these should be in different populations in the world where PXF is a relatively frequent finding.

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1 Slagsvold JE, Nordhagen R. The HLA system in primary open-angle glaucoma and in patients with pseudoexfoliation of the lens capsule (exfoliation of fibrillopathia epitheliocapsularis). *Acta Ophthalmol* 1980;**58**:188–92.

BOOK REVIEW

Orbital Surgery. A Conceptual Approach. By Jack Rootman, Bruce Stewart, Robert Alan Goldberg. Pp 416. £167. Philadelphia, Lippincott-Raven, 1995.

The accurate description of orbital surgery is extremely difficult, if not impossible. This difficulty arises because of highly variable presentation and extent of orbital disease, from the ready distortion of normal orbital anatomy by disease or during surgery, and as a result of complexities in the three dimensional portrayal of orbital conditions.

Jack Rootman, with his co-authors Bruce Stewart and Robert Goldberg, has approached this task with the superb skill—both surgical and artistic—for which he is internationally renowned.

In the early chapters, the authors lay the principles for surgical approaches to various types of lesion in different orbital locations, highlighting that surgery should be variations on a theme and not a rigid, unerring process. Having comprehensively presented the concept of themes for management, the normal anatomy is beautifully portrayed in a superb series of illustrations by Bruce Stewart; most of the illustrations are new, there being only minimal overlap with those in Jack Rootman's previous epic, *Diseases of the Orbit* (Lippincott, 1988).

The later, major, part of the work is devoted to details of surgical approaches to regions of the orbit, as related to the treatment details for specific conditions. The comprehensive text is complemented by drawings of the highest standard and by radiographs and clinical photographs of illustrative clinical cases; this combination of details making for delightful reading. The reviewer approached review of the book with several varied (hypothetical) clinical problems to be answered: the book provided useful details or ideas for the treatment of all of the set problems, although obviously it cannot be a substitute for practical experience.

The quality of publication is immediately evident. The illustrations are of excellent reproduction, the text layout pleasing, and the binding good. The book is essential to all ophthalmic libraries and is very warmly recommended for the personal libraries of all orbital surgeons.

GEOFFREY E ROSE

NOTICES

European Board of Ophthalmology

The examination of the European Board of Ophthalmology (EBO) will be held in Fondation Ipsen, Paris on Friday 23 May and Saturday 24 May 1997.

This examination is optional.

The successful candidates will be awarded the Diploma of the EBO (EBOD) as a mark of excellence.

The multiple choice question examination (MCQ) will be held on Friday 23 May 1997 and the oral examination will be held on Friday 23 and Saturday 24 May 1997.

Closing date for preliminary applications: 31 January 1997.

All candidates will have the option of sitting the examination in their own national language.

For further information please contact your national delegates to the EBO or Professor Peter Eustace, Secretary General, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland. (Tel: 00353 1 8600254; Fax: 00353 1 8305693.)

All applicants must have unrestricted licence to practise medicine in a country of the European Union (EU) and must have satisfactorily completed a residency training programme in ophthalmology of at least 48 months' duration in a unit recognised for training by the EBO. They must be recognised as ophthalmologists within one country of the EU and have passed the appropriate national examination of the EU country in which they trained if there is a national examination in that country, otherwise they must have met the certifying requirements of that country. Candidates from Norway and Switzerland, who are full members of the Union Européene Des Médecins Spécialistes (UEMS) and the EBO, are also eligible under the same terms.

Three places will be reserved for each country of the EU. To be eligible to sit the examination applicants must have returned the application form fully completed and have paid the examination fee of 250 ecu.

Conferences on Angiography in Créteil

A conference on retinal telangiectasia will take place on 13 January 1997 at the University of Créteil. Further details: Professor Gisèle Soubrane, Clinique Ophtalmologique Universitaire de Créteil, 40 Avenue de Verdun, 94010 Créteil Cédex, France. (Tel: 45 17 52 22.)

XVI Congress of the Asia Pacific Academy of Ophthalmology

The XVI Congress of the Asia Pacific Academy of Ophthalmology will be held in Kathmandu, Nepal from 2–6 March 1997. Further details: The Secretariat, XVI Congress of APAO, Nepal Eye Hospital Building, Tripureswor, PO Box 335, Kathmandu, Nepal. (fax: +977 1 227505/518.)

Conferences on Angiography in Créteil

A review of the literature on retinal and choroidal vascular pathology will take place on 10 March 1997 at the University of Créteil. Further details: Professor Gisèle Soubrane, Clinique Ophtalmologique Universitaire de Créteil, 40 Avenue de Verdun, 94010 Créteil Cédex, France. (Tel: 45 17 52 22.)

Glaucoma Meeting Basel '97

A teaching meeting on NO and endothelin in the pathogenesis of glaucoma will be held on 21–22 March 1997 at the Zentrum fur Lehre und Forschung (ZLF), Kantonsspital Basel, Hebelstrasse 20, Basel, Switzerland. Further details: Daniela Stumpfig, University Eye Clinic Basel, Mittlere Strasse 91, PO Box, CH-4012 Basel, Switzerland. (Tel: ++41-61-321 59 62 or ++44-61-321 77 77; fax: ++41-61-322 40 01.)

International Symposium on Ocular Tumors

The International Symposium on Ocular Tumors will be held on 6–10 April 1997 in Jerusalem, Israel. Further details: Professor J Pe'er, Tumors, PO Box 50006, Tel Aviv 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 514007.)

2nd International and 4th European Congress on Ambulatory Surgery

The 2nd International and 4th European Congress on Ambulatory Surgery will be held at the Queen Elizabeth II Conference Centre, Westminster, London on 15–18 April 1997. Further details: Congress Secretariat, Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, West Yorkshire HD7 3AP. (Tel: +44 1484 854575; fax: +44 1484 854576.)

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Second European Forum on Quality Improvement in Health Care

The Second European Forum on Quality Improvement in Health Care will take place on 24–26 April 1997 in Paris, France. The forum will consist of one day teaching courses, invited presentations, posters and presentations selected from submissions, and a scientific session. Further details: BMA, Conference Unit, PO Box 295, London WC1H 9TE. (Tel: +44 (0) 171 383 6478; fax: +44 (0) 171 383 6869.)

ARVO

The Association for Research in Vision and Ophthalmology (ARVO) is holding its annual meeting on 11–16 May 1997 at the Fort Lauderdale Convention Center, Fort Lauderdale, Florida, USA. Further details: ARVO, 9650 Rockville Pike, Bethesda, MD 20814-3998. (Tel: (301) 571-1844; fax: (301) 571-8311.)

30th Panhellenic Ophthalmological Congress

The 30th Panhellenic Ophthalmological Congress organised by the Hellenic Ophthalmological Society will be held at the Astir Palace Hotel, Vouliagmeni on 28 May to 1 June 1997. Further details: T Kouris, CT Congress, Creta Travel, 19 Amerikis 106 72 Athens, Greece. (Tel: (01) 3607 120, 3635 104; fax: 3603392.)

Conferences on Angiography in Créteil

A conference on clinical cases in ICG will be held on 9 June 1997 at the University of Créteil. Further details: Professor Gisèle Soubrane, Clinique Ophtalmologique Universitaire de Créteil, 40 Avenue de Verdun, 94010 Créteil Cédex, France. (Tel: 45 17 52 22.)

British Council International Seminar

A British Council international seminar (number 97031) entitled 'Corneal and external eye disease: new surgical techniques' with Professor D L Easty as director will be held on 29 June to 5 July 1997 in Bristol, UK. The seminar will be of particular interest to all young eye surgeons from the developing and developed world. Further details: Promotions Manager, International Seminars, The British Council, 1 Beaumont Place, Oxford OX1 2PJ, UK (Tel: +44 (0) 1865 316636; fax: +44 (0) 1865 557368/516590; e-mail: International.Seminars@britcoun.org).

5th International Symposium on Ocular Circulation and Neovascularisation

The 5th International Symposium on Ocular Circulation and Neovascularisation will be held on 15–19 September 1997 in Kyoto, Japan. Further details: Professor Dr Masanobu Uyama, Secretary General of the Organising Committee, Department of Ophthalmology, Kansai Medical University, Moriguchi, Osaka 570, Japan. (fax: 81-6-997-3475.)

2nd International Symposium on ARMD

The 2nd International Symposium on ARMD will be held at Glasgow University, Scotland under the auspices of the Royal College of Ophthalmologists on 16–18 September 1997. Further details: Dr G E Marshall, Eye Department, Western Infirmary, 38 Church Street, Glasgow G11 6NT, UK. (Tel: 0141 211 2094; fax: 0141 339 7485; email: gem1b@clinmed.gla.ac.uk)

XXVIIIth 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, the Netherlands. (Tel: +31-20-6793411; fax: +31-20-6737306; internet http://www.solution.nl/ico-98/)

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, Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077.)

Correction

The letter by Ramboer *et al*, which appeared in the October issue of the *BJO* (1996;**80**:930–2), in cluded an incorrect figure (Fig 2). The letter is republished in full on p 90 of this issue with the correct figure. We apologise to the authors for this error.

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