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.

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

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 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 were not present in our patient, but can be seen in localised scleroderma.

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 some oedema in these structures.

The cutaneous pathological features described from the lesions are arranged in a band-like distribution and nature of the superficial dermis. In patients with morphea 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.

Olver et al have reported a patient with bilateral acquired Brown’s syndrome and combined lichen sclerosus et atrophicus and morphea.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 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 dermis were not present in our patient, but can be seen in localised scleroderma.

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

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.

COMMENT
Scleroderma may occur as a systemic disease or as a localised form. Localised scleroderma presents in three clinical forms: generalised, morphea (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 morphea 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.

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Involve-
defects, and the unsuccessful therapy. This patient has a significant indentation 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 encephalomalacia and the absence of mass effect.14 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 encephalomalacia, and its relation to linear scleroderma has already been reported.14 The thickening of the orbital muscles in this case can probably be secondary to orbital fat atrophy and fibrosis.15

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.16 In this patient the lesion is the chronic and infiltrative or diffuse type. It does not invade or distort the globe and there is no bone erosion.17

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 more than 90% of cases.10 We found similar signal characteristics as hypointense to fat and isointense on T1 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 beneficial in distinguishing 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.

K RAMBOER
PH DEMAEREIL
A L BAERT
Department of Radiology,
University Hospitals, Leuven, Belgium

J CASTEELS
G DRLANDS
Department of Ophthalmology,
University Hospitals, Leuven, Belgium

Correspondence to: Ph Demaereil, MD, Department of Radiology, University Hospitals, Herestraat 49, B-3000 Leuven, Belgium

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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 standard wooden club (seven iron) and the surface underneath 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 repotted 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 'teeing 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 injuries2 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.4 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.5 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.6 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.7 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.15 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.

ALAN MULLVILLII
PATRICIA LOGAN
Eye Department, Mater Misericordiae Hospital,
Dublin, Ireland

Correspondence to: Mr Alan Mulvhill, Institute of Ophthalmology, 60 Eccles Street, Dublin 7, Republic of Ireland

Accepted for publication 16 September 1996

1 Royal and Ancient Golf Club of St Andrews' Development Panel. The Demand for Golf. 1989.

Preterential ‘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 pro-thrombin infection can mimic many others and produces unusual lesions anywhere in the eye.

References


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

Figure 2 The right eye showing the injury site 24 hours after repair (arrow).
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 vitreitis 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 peri papillary retinal oedema. There were folds around the macula.

The patient was investigated serologically before starting any medication. His total leucocyte count was 9.7 × 10^3/μl, 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).

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 vessels 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 neuroretinitis are described as a patchy diffuse retinal oedema with associated vitritis, vasculitis (generally arterial), and occasionally haemorrhages. 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.

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 proliferoophthalmic leukema, wherein the contents were presumed to be an accumulation of abnormal lymphocytes.

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

R SIHOTA
Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India

A H PAUL
Bausch and Lomb, India

R CHANDNA
Escorts Medical Centre, Faridabad, India

Correspondence to: Dr R Sihota, Dr Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi 110029, India.

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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 perimeter, 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 designed3 specifically for the detection of moderate and severe glaucomatous visual field loss in situations precluding ophthalmic examination.4 Rait seems to dismiss this technique as ‘less reliable’, quoting an article 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.5 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.6 The 26 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%.7 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.8 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 ‘multifraction 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 nasals 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).

4 Wishart PK. Oculokinetic perimetry compared with Humphrey visual field analysis in the detection of glaucomatous visual field loss. Eye 1993;7:113–21.

Reply

Editor,—Damato and Fenery are to be congratulated on providing a new and improved multifraction campimeter9 and ‘more foolproof’ than its predecessor. I have no doubt that 95%/93% sector concordance is possible with the original device1 but it only detect moderate to severe visual field loss with any reliability6,8.

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.

JULIAN L RAIT
University of Melbourne,
Royal Victoria Eye and Ear Hospital,
Melbourne, Australia


Association of HLA type with pseudoxefoliation 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 pseudoxefoliation assumes importance in view of reports of possible cross reaction between antigens of infectious agents and particular HLA types in disorders such as ankylosing spondylitis.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.


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 pseudoxefoliation (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

G SESHUBABU
D I I I, Dhanvantari Nagar,
JIPMER, Pondicherry 605 006, India
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 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 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.

J S FITZSIMON
A MULVILHHILL
S KENNEDY
A FINCH
L M T COLLUM
P EUSTACE
Mater Misericordiae Hospital, Eccles Street, Dublin 7, Ireland

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 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 Européenne 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 Stumpfeg, 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 61300, 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, Mether, West Yorkshire HD7 3AP (Tel: +44 1484 854575; fax: +44 1484 854576.)
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 Ophthalmologique 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. (Tel: 81-6-997-3475.)

2nd International Symposium on ARM D

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.

Correction

The letter by Ramboer et al, which appeared in the October issue of the BJ O (1996;80:930–2), included 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.
Linear scleroderma with orbital involvement: follow up and magnetic resonance imaging

K RAMBOER, PH DEMAEREL, A L BAERT, I CASTEELS and G DRALANDS

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