LETTERS TO THE EDITOR

Dermatitis artefacta presenting as a basal cell carcinoma—an important clinical sign missed

EDITOR,—Dermatitis artefacta has not previously been reported presenting as a masquerade syndrome for basal cell carcinoma of the eyelid. We describe a patient who presented with a "typical" basal cell carcinoma of the lower eyelid, in whom the diagnosis only became apparent following its surgical excision.

CASE REPORT

A 43 year old right handed woman was referred by her general practitioner complaining of a 6 month history of a lesion on her left lower eyelid which had been increasing in size, and she had developed a red sticky eye. She had no ophthalmological history of note; however, she had previously worked in a beauty clinic and had used the sunbeds there with great regularity. She had a medical history of diverticular disease, anxiety, and was under investigation by a cardiologist for ectopic heart beats.

On examination she had a lesion which appeared typical of a basal cell carcinoma of her left lower eyelid with rolled edges, notching of the lid margin, and infiltration of the tarsal plate (Fig 1). The raised rolled edge of the lesion was indurated to palpation. In addition she had injection of the conjunctiva. She was referred to the oculoplastic clinic at Birmingham and Midland Eye Centre. At review the lesion appeared similar to Figure 1, although the conjunctival injection was absent. The lesion was excised with a 2 mm marginal clearance. The postoperative course was uneventful apart from the development of a mild papillary conjunctivitis—this was thought to be due to a chloramphenicol allergy and her topical antibiotic was discontinued, which led to resolution of this condition.

Histology of the excised specimen revealed "...no evidence of neoplasm..."; there was what appeared to be a keratinous cyst. There was stromal scarring and active inflammation, and histiocytes and giant cells were seen, some of which were clearly a reaction to free lipid. There were, in addition, a number of conjunctival epithelial inclusion cysts.

A diagnosis of dermatitis artefacta was made.

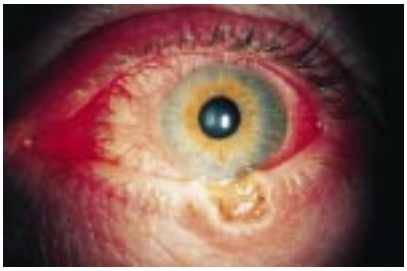

Figure 1 Dermatitis artefacta of the left lower eyelid imitating a basal cell carcinoma. Injection of the conjunctiva and the absence of subepithelial telangiectasia, in retrospect should have raised suspicion as to the certainty of the diagnosis of basal cell carcinoma.

COMMENT

In this case the "typical" features of what was thought to be a basal cell carcinoma of the lower eyelid overshadowed the subtle features in this patient's medical history that may have aided the development of this rare diagnosis. More importantly the presence of a patient with a red eye with a coexisting lesion of the eyelid should have alerted us to the possibility of excessive scratching/digitation after exclusion of more obvious causes such as molluscum. Also, in retrospect, the absence of fine subepithelial telangiectasia should have raised added suspicion.

Had the possibility of dermatitis artefacta been mooted, a short period of occlusive bandaging with the use of steroid cream may have aided confirmation of the diagnosis.

Dermatitis artefacta or self inflicted factitial dermatitis forms one of the spectrum of self inflicted dermatoses and also represents one of the spectrum of obsessive compulsive disorders. Typically, patients deny the self inflicted nature of the disorder. The disorder is seen more commonly in women (male to female ratio of at least 1:4) and has a broad and variable age of onset (9–73 years).[1 2] Patients frequently have an impulsive personality disorder.

Skin lesions are produced or significantly exacerbated by self inflicted trauma.[3] Recurrent excoriation produces inflammation and lichenification of the skin; the resultant irritation and pruritus leads to further self trauma and chronic dermatitis. The lesions have wide ranging morphological features and are often bizarre looking, with sharp geometric borders surrounded by normal looking skin.[4] In the right handed person, the left side is usually involved,[2] as in this case.

Self inflicted dermatoses vary greatly because of the wide range of methods that are used for inflicting the lesions: cutting, abrasion, burning applying chemicals, and injecting various products. This diversity makes it particularly difficult to diagnose dermatitis artefacta.[5] Histopathological diagnosis consists of features of acute inflammation with increased polymorphonuclear leucocytes with scattered erythrocytes. There may also be areas of necrosis with areas of healing with fibrocystic reaction.[6] We believe the characteristic rolled edge of the lesion was from such healing areas.

Patients with dermatitis artefacta are particularly sensitive to hostile feelings in medical practitioners, to which they react with renewed self mutilation.[7] The need for psychiatric referral should be balanced against the fact that the patient will interpret this referral as a rejection, which can intensify the self mutilation. Follow up studies have shown that most patients with dermatitis artefacta improve more significantly after changes in life situations and maturation than as a result of psychiatric treatment. In this case her continuing care was taken over by her general practitioner.

Our thanks to Mr Shun-Shin, Wolverhampton Eye Infirmary for his help in obtaining the documentation regarding this patient.

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Accepted for publication 27 August 1997

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Application of mitomycin C 0.02% for 2 minutes at the end of pterygium surgery

EDITOR,—Mitomycin C 0.02% has been shown to be highly effective in preventing the recurrence of pterygium following its surgical removal.[1 2] The safest method of application and the optimal concentration have yet to be determined.

In this retrospective study, we evaluated 45 consecutive patients who underwent surgical removal of pterygium at the Barzilai Medical Center between January 1995 and January 1996. Patients' ages at surgery ranged from 25 to 75 years (average 51 years). A bare sclera technique was employed in each case.

Patients were divided into two groups. At the end of surgery, patients in the treatment group (n=33) were treated by the application of a Weck cell, which was soaked in a solution of mitomycin C 0.02% and applied to the sclera at the site of the surgical bed for 2 minutes, followed by thorough irrigation with saline solution. Patients in the control group (n=12) did not receive treatment with mitomycin C.

Postoperative treatment included topical application of dexamethasone 0.1% four times a day, with gradual tapering off over the first month.

Between 5 and 13 months after surgery (average 8.3 (SD 3.3) months), 24 patients from the treatment group (73%) and 11 from the control group (92%) underwent a long term follow up examination. Recurrence of pterygium, defined as any growth of blood vessels crossing the limbus onto the cornea at the previous site of pterygium, was detected in six patients (25%) in the treatment group and in nine patients (82%) in the control group ($p<0.005$, Student's t test).

Closure of epithelial defects in the surgical bed was completed during the first 2 weeks after surgery in both groups. Mild punctate epithelial staining, which was found in patients in the treatment group, resolved within 2 weeks. Mild ocular discomfort lasting for 3 months was reported by six patients (25%) in the treatment group only.

COMMENT

This study showed that a single application of mitomycin C 0.02% for 2 minutes at the end of pterygium surgery is safe for the eye and reduces the rate of recurrence compared with untreated control eyes (25% *v* 81%). Even lower recurrence rates (4%) and very few complications, all of them mild, were obtained

by Frucht-Pery *et al* using a single application of mitomycin C 0.02% for 5 minutes at the end of surgery.[2]

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Accepted for publication 18 September 1997

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Indocyanine green angiography and idiopathic polypoidal choroidal vasculopathy

EDITOR,—Idiopathic polypoidal choroidal vasculopathy (IPCV) also known as "posterior uveal bleeding syndrome" or "serosanguineous detachments in black females" is an exudative macular degeneration characterised by a juxtapapillary branching choroidal network with surrounding polypoidal excrescences associated with serous and haemorrhagic detachments of the neurosensory retina and pigment epithelium.[1–4] The patients are almost always black females in the fifth to seventh decade.

We describe here the indocyanine green angiographic (ICGA) findings of a white male patient with IPCV, who had been misdiagnosed for exudative age related macular degeneration (AMD).

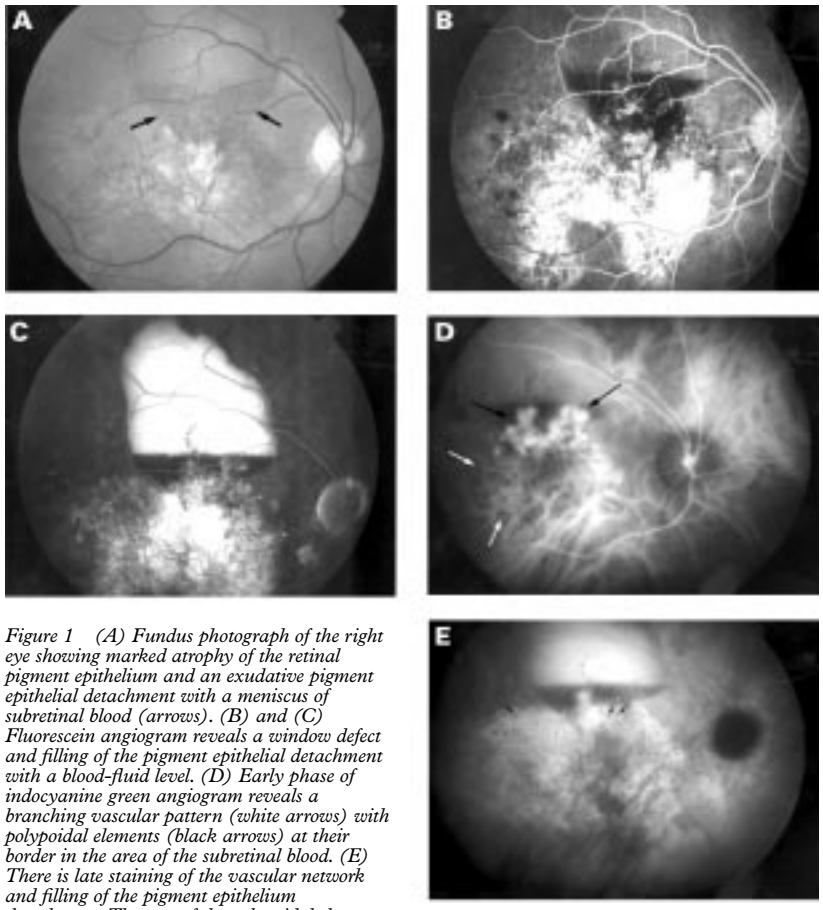

Figure 1 (A) Fundus photograph of the right eye showing marked atrophy of the retinal pigment epithelium and an exudative pigment epithelial detachment with a meniscus of subretinal blood (arrows). (B) and (C) Fluorescein angiogram reveals a window defect and filling of the pigment epithelial detachment with a blood-fluid level. (D) Early phase of indocyanine green angiogram reveals a branching vascular pattern (white arrows) with polypoidal elements (black arrows) at their border in the area of the subretinal blood. (E) There is late staining of the vascular network and filling of the pigment epithelium detachment. The core of the polypoidal elements became hypofluorescent (arrows).

CASE REPORT

A 54 year old white male patient was referred for an ICGA examination with the diagnosis of exudative AMD. He complained of decreased vision and metamorphopsia in his left eye of 10 days' duration. The past ocular history was remarkable for recurrent serosanguineous exudative maculopathy of his right eye over a period of 10 years. He did not have a history of systemic disease. Visual acuity of the right eye was reduced to 10/200. Visual acuity of the left eye was 25/200. There was no evidence of inflammation in either the anterior chamber or vitreous. Fundus examination of the right eye disclosed a pigment epithelial detachment with a meniscus of subretinal blood superior to the macula (Fig 1A). In addition, marked atrophy of the retinal pigment epithelium with visible choroidal vessels was seen in the centre of the macula. The left fundus revealed a neurosensory detachment of the macula associated with subretinal lipid deposits (Fig 2A). There were two serous pigment epithelial detachments inferior and temporal to the centre of the macula.

Fluorescein angiography (FA) of the right eye (Fig 1B and C) revealed a window defect with a visible choroidal vascular net and filling of the pigment epithelial detachment with a blood-fluid level. Early phase ICGA (Fig 1D) demonstrated a subfoveal vascular network in an oval configuration with nodular excrescences in the area of the subpigment epithelial blood. Later in the ICG study (Fig 1E) the vascular network showed staining. The core of the polypoidal structures became hypofluorescent. There was prominent leakage of ICG into the pigment epithelial detachment.

FA (Fig 2B and C) of the left eye revealed subfoveal mottled hyperfluorescence with late staining and filling of the two pigment epithelial detachments suggestive of occult choroidal neovascularisation (CNV). On ICGA (Fig 2D) a well defined hyperfluorescent network of branching vessels with polypoidal lesions at the inferior border became visible. Late phase ICG angiogram (Fig 2E) revealed staining of the vascular network and minimal leakage of dye into the pigment epithelial detachment.

COMMENT

AMD is currently the leading cause of severe visual loss in Western societies. The basic mechanism for severe visual loss is the ingrowth of CNV and its consequence: exudation, haemorrhage, and disciform scarring. Our patient demonstrated serous and haemorrhagic detachments of the retinal pigment epithelium and the neurosensory retina associated with lipid deposits, characteristics shared with CNV secondary to AMD. However, ICGA revealed a branching vascular network with polypoidal structures at their border characteristic of IPCV.

IPCV is rarely described. The few reported case series suggest that the exudative and haemorrhagic detachments arise from the polypoidal elements. These orange vascular structures may be difficult to visualise on slit lamp biomicroscopy.[5] While the polypoidal elements can be blocked by haemorrhage and fluid of the pigment epithelial detachments on FA as seen in our patient, ICGA clearly demonstrates the underlying pathology.[5]

Our patient is not typical of the clinical presentation and population in which this condition was originally described. Earlier studies have suggested that IPCV mainly occurs in middle aged black females and that the multifocal bilateral lesions, which initially often present with breakthrough vitreous haemorrhage, are found predominantly in the temporal juxtapapillary region.[1–4] Our male patient is white and he has a solitary lesion in the central macula which was unilateral over an extended course without a history of vitreous haemorrhage. However, our patient has features of IPCV with the vascular elements ending in aneurysmal or polypoidal lesions on ICGA which differentiates it from AMD. The ocular history with recurrent serous and haemorrhagic detachments of the retinal pigment epithelium and neurosensory retina is consistent as well. Within follow up the right eye went on to develop vitreous haemorrhage and the inferior serous pigment epithelial detachment of the left resolved. Whereas previously it was thought that IPCV was more or less exclusively in the peripapillary region, isolated lesions in the central macula may occur in 10% of patients. In addition, the lesions may be unilateral over an extended course (Yannuzzi, personal communication).

The differential diagnosis of IPCV includes the various causes of CNV. In our patient there were no "histo spots", angioid streaks, high myopia, history of trauma, or previous inflammations, nor was there any other systemic disease associated with CNV. No evidence of coexisting AMD such as focal hyperpigmentations or drusen were noted in both eyes of our patient. Our case report also differs from AMD in that the progression of the disease over a period of 10 years was relatively

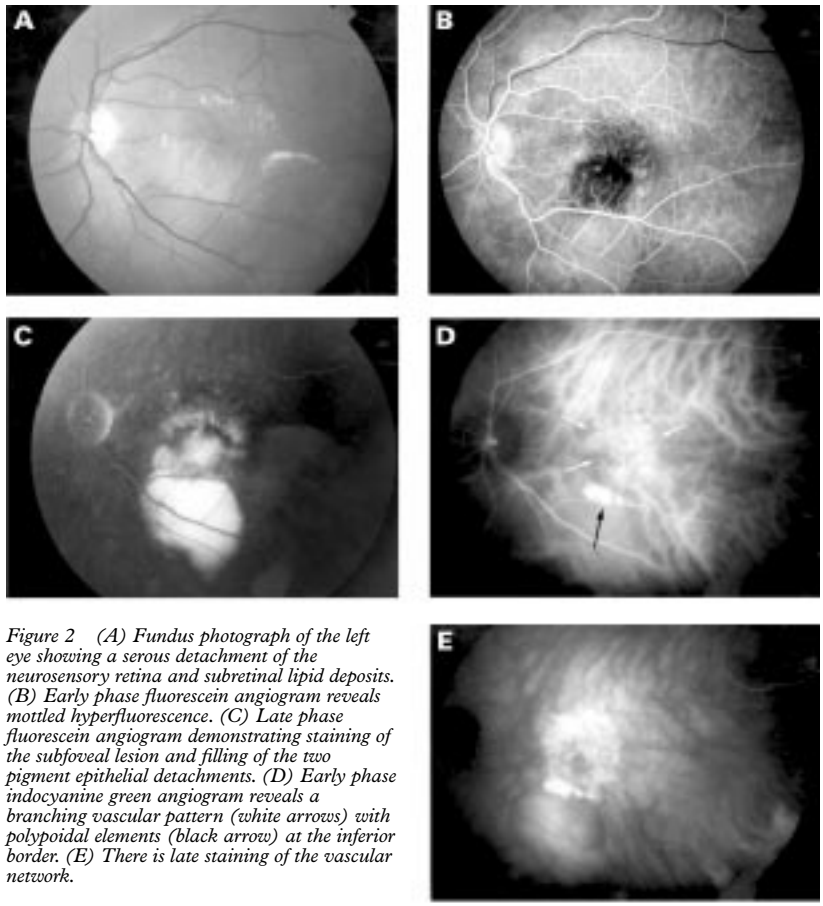

Figure 2 (A) Fundus photograph of the left eye showing a serous detachment of the neurosensory retina and subretinal lipid deposits. (B) Early phase fluorescein angiogram reveals mottled hyperfluorescence. (C) Late phase fluorescein angiogram demonstrating staining of the subfoveal lesion and filling of the two pigment epithelial detachments. (D) Early phase indocyanine green angiogram reveals a branching vascular pattern (white arrows) with polypoidal elements (black arrow) at the inferior border. (E) There is late staining of the vascular network.

slow and the exudative complications started in his early 40s.

The natural course of IPCV is generally associated with recurrent exudative and haemorrhagic episodes. While AMD causes large disciform scars, resolution of the exudative and haemorrhagic manifestations in IPCV may be associated with good visual outcomes in most cases. Involvement of the central macula, however, may result in severe loss of vision as described in our patient. Treatment of the polypoidal elements by laser photocoagulation may be taken into consideration in cases involving the central macula. In one report laser photocoagulation was performed in nine eyes with six showing resolution of the exudative manifestations. However, final visual acuities of the six treated eyes reached the same level as four of five untreated eyes.[1]

ICGA with its improved imaging of the choroidal circulation accentuates the vascular polypoidal lesion[5] and allows differentiation between IPCV and CNV, an important distinction concerning the natural course, visual prognosis, and management of these diseases. Additional clinical features such as a solitary lesion in the central macula and an unilateral course over an extended period may expand the clinical definition of IPCV.

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Accepted for publication 27 August 1997

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Rhinogenic optic neuropathy caused bilateral loss of light perception

EDITOR,—Rhinogenic optic neuropathy is a clinical entity including rhinogenous optic neuritis and optic neuropathy caused by a paranasal cyst. The damage to the optic nerve seems to be caused mainly by cyst compression and inflammatory changes. Rhinogenic optic neuropathy secondary to paranasal lesions is not so rare, and paranasal sinus mucoceles with unilateral blindness have already been reported. Bilateral loss of light perception caused by rhinogenic optic neuropathy, however, has not been reported in the literature.

CASE REPORT

A 48 year old man complained of acute visual loss in both eyes. His corrected visual acuities were light perception in both eyes, and the light reflex was defective. No remarkable finding was observed on routine ophthalmic examination. Optic atrophy was not present, and intraocular pressures were normal. He had a history of surgery for sinusitis 28 years ago and had been complaining of headache for a year. A computed tomogram showed a large high density area in the sphenoid sinus (Fig 1). Sphenoidotomy was undertaken and the mucocele was opened (Fig 2). It was confirmed that a large sphenoid mucocele was pressing both the optic nerves. Treatment with systemic corticosteroids and antibiotic was initiated upon admission. Despite this treatment, his visual acuities deteriorated to no light perception both eyes, and they never recovered. No remarkable finding was observed in the electroretinogram, but flash visually evoked potential was non-recordable.

COMMENT

Sphenoid sinus mucocele is a rare entity that can occur alone or as a result of ethmoid sinusitis and polyposis. A myriad of presentations is possible because of the presence of important contiguous neurological and vascular structures.[1]

Clinically, sphenoid sinus mucocele can cause a wide variety of signs and symptoms including bilateral visual loss, depending upon which adjacent structures are affected by the expanding cyst. The prognosis of visual disturbance is thought to be dependent on visual acuity preoperatively and the time from onset of the disease to the operation.[2] Recovery is quite difficult in patients who have severe preoperative disturbances such as no light perception. The prognosis is also poor in cases of sudden onset. In our case, the onset of visual disturbance was sudden, and preoperative visual acuity was only light perception in both eyes. Although the patient underwent

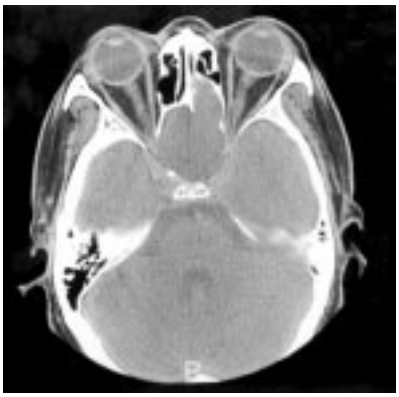

Figure 1 Axial computed tomograph of the orbits, demonstrating a homogeneous space occupying lesion in the sphenoid sinus (sphenoid sinus mucocele).

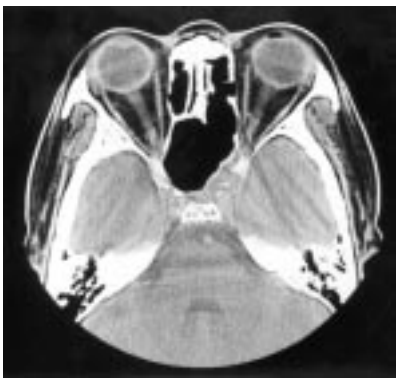

Figure 2 Postoperative computed tomograph show that the sphenoid sinus mucocele has been totally removed.

surgery within 24 hours from the onset, the recovery was unsatisfactory.

Vascular distribution to the optic nerve in the optic canal is less than in other sites, so that the optic nerve is very susceptible to a decrease in vascular supply. In this case, compression from the mucocele was thought to have caused ischaemia and/or venous congestion of the optic nerve. The presence of a headache for a year suggests long standing chronic inflammation of the mucocele. So in this case, besides compression by the mucocele, inflammatory disorders seemed to have contributed to irreversible damage to the optic nerve.

Patients with a sphenoid sinus mucocele often experience only ophthalmic symptoms such as visual impairment, external ophthalmoplegia, and diplopia.[3] Therefore, for early diagnosis and an improved prognosis, ophthalmologists must be alert to this as a possible cause of blindness.

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Accepted for publication 27 August 1997

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Bilateral subperiosteal haematoma after endoscopic sinus surgery

EDITOR,—Subperiosteal orbital haematoma as a complication during endoscopic sinus surgery is not well known to ophthalmologists.

It has the potential to increase intraocular pressure and subsequently cause blindness by optic nerve compression with central retinal artery occlusion.[1]

We report a case of a 13 year old boy who developed bilateral subperiosteal haematoma after bilateral endoscopic sinus surgery.

CASE REPORT

A 13 year old boy was referred for evaluation of decreasing visual acuity 3 days after bilateral endoscopic sinus surgery for pansinusitis.

He complained of blurred vision in both eyes, diplopia, and anhidrosis of the left side of his face. There was moderate swelling of the left upper eyelid. His best corrected visual acuity was 20/30 in the right and 20/40 in the left eye. Both pupillary responses showed sluggish reactions. Upward movement of left eye was limited, with inability to supraduct more than 30 degree past the midline because of pain. In the primary position he had a 5 prism dioptre left hypodeviation. Slit lamp examination, funduscopic examination, and confrontation visual field tests were normal in both eyes. Intraocular pressure by applanation was 14 mm Hg in both eyes. An orbital computed tomography (CT) scan demonstrated soft tissue swelling consistent with subperiosteal haematoma in both orbits (Fig 1). The patient had no history of systemic diseases such as coagulopathy or bleeding disorder.

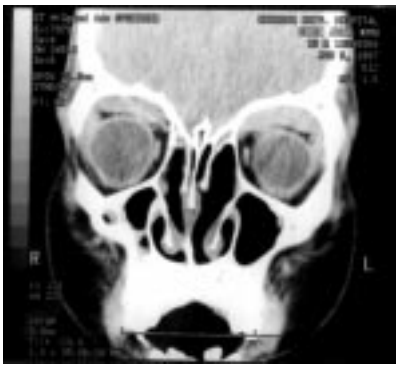

Figure 1 Coronal orbital computed tomogram reveals accumulation of blood in the both superior orbital subperiosteal spaces.

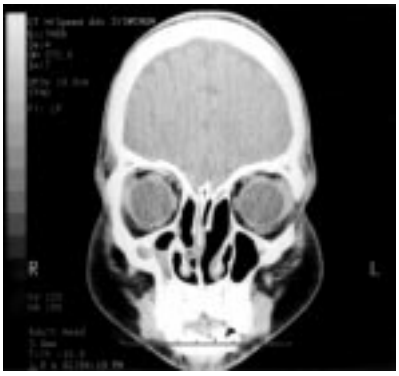

Figure 2 Coronal orbital computed tomogram obtained after operation shows clearance of haematoma in both the superior orbital subperiosteal spaces.

Treatment with systemic antibiotics and coagulant was tried, to promote the spontaneous absorption of the subperiosteal haematoma and prevent other complications, because of the patient's fear of more surgery.

Ten days after the sinus surgery, anhidrosis of the left side of the face persisted and left eye visual acuity decreased to 20/200. At that time, visual evoked potential (VEP) showed evidence of optic nerve damage in both eyes with prolonged latency and decreased amplitude. Follow up orbital CT revealed no decrease in the amount of blood present.

The same day an emergency operation was performed to evacuate the subperiosteal haematoma by bilateral sub-brow incision; the blood was evacuated through a periosteal incision.

The day after the operation his anhidrosis was resolved and visual acuity in both eyes recovered to 20/20 in 2 weeks. Orbital CT 2 weeks after operation demonstrated clearing of the subperiosteal haematoma (Fig 2).

COMMENT

Because of the relatively low complication rate from endoscopic surgery of the paranasal sinuses, orbital injury only occasionally presents to the ophthalmologist. These orbital complications include injury to the nasolacrimal duct, orbital emphysema, diplopia, orbital haematoma, and temporary or permanent blindness.[2] The most devastating complication is blindness resulting from optic nerve compression by haematoma or direct injury to the nerve itself. If injury of the anterior ethmoidal artery occurs during the ethmoidectomy, it may be difficult to control the bleeding owing to the contraction of the vessel into the orbit.

The pathogenesis of subperiosteal orbital haematoma is (1) traumatic tearing of an orbital vessel, (2) rupture of a subperiosteal vessel secondary to increased venous pressure transmitted by valveless orbital veins from congested sinus mucosa, or (3) erosion of a vessel by orbital extension of an infectious process. Its frequent occurrence in the roof of the orbit may be related to the loose attachment of the periosteum in this area.[3]

In this case, it was thought that the subperiosteal haematoma was caused by avulsion injury to the posterior ethmoidal arteries during endoscopic sinus surgery. There was no fat protrusion or anterior ethmoidal artery injury during surgery.

Herniation of the orbital fat is an important sign, indicating entry to the lamina papyracea during the removal of the polypoid tissue from the ethmoidal sinus.[2] If there is a suspicion of orbital bleeding, the patient should be observed for signs of increased intraorbital pressure such as pain, swelling of the eyelid, ecchymosis of the eyelid, proptosis, restriction of the eyeball movements, pupillary reaction, and diminished vision by the ophthalmologist in the recovery room.

Interestingly, this patient had anhidrosis of the left side of the face. The authors could not find any reports on anhidrosis associated with subperiosteal haematoma. Sweating over the ipsilateral side of the face is lost with sympathetic interruption below the bifurcation of the carotid artery, where the facial sweat fibres leave the artery.

Anhidrosis in this patient may be related to injury of sympathetic fibres in the optic foramen by pressure of a subperiosteal haematoma.

This case reports an unusual but dangerous simultaneous bilateral orbital complication of endoscopic sinus surgery and its treatment.

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CORRESPONDENCE

Reassessment of the PAS patterns in uveal melanoma

EDITOR,—We read with great interest the article by Foss and associates published in the March issue of the *BJO*.[1]

In discussing different techniques for the detection of vascular structures in uveal melanomas, it is obviously necessary to avoid a misinterpretation of the pertinent criteria:

(1) Foss and associates interpreted the areas 1 and 2 shown in Figure 1A of their article as "area of silence", and "area of normal vasculature". We would not have classified the region marked as 1 in Figure 1A as a "silent area", because many small vessels with a "white" lumen and a very thin PAS positive basement membrane can be identified, even at the magnification and the weak PAS stain given in their article. In addition, the two enlarged vessels marked as 2 would not have been classified as "normal" according to our original description.[2–4] Therefore, we were not surprised that the authors could demonstrate a positive staining for vascular endothelium using factor VIII in Figure 1B. In our view, Foss and associates just misinterpreted findings with their PAS technique, which led to a misleading conclusion.

(2) Furthermore, in our experience we find it relatively easy to discriminate between basement membrane material and connective tissue as shown in Figure 2. In cases of doubt, we always stained serial sections for connective tissue using the Gomori's trichrome stain. Blood vessels within a tumour contain endothelial cells, a basement membrane, and a lumen sometimes with detectable erythrocytes. This lumen can be identified in most of the tumour blood vessels using the PAS staining technique. In cases of doubt, the PAS stain could be compared with the adjacent haematoxylin and eosin stain of serial sections in order to identify a lumen and/or erythrocytes within a vessel lumen. The ultrastructure of the tumour blood vessels in melanocytic naevi and malignant melanomas of the uvea has been demonstrated in detail, previously.[4] The blood vessels in malignant melanomas usually had a thick, multilaminated, and fragmented basement membrane with excessive amounts of connective tissue. These findings were the morphological basis for the detection of tumour blood vessels using the modified PAS stain without haematoxylin counterstaining.[2–4]

(3) Finally, Foss and associates assume in their article that the detection of vascular networks in malignant melanomas using the fluorescein conjugated lectin, *Ulex europaeus* 1, was mainly due to autofluorescence. As we used appropriate control sections, dyes, and filter blocks we can rule out the possibility of autofluorescence in our studies.[2 5] For example, the highly vascularised choriocapillaris in tissue sections of surgically enucleated eyes served as an internal positive or negative control, and did not show any autofluorescence, but did stain positive with F-UEAI agglutinin. Moreover, Foss and associates also claimed that our fluorescent patterns "could partially resist bleaching". We only examined patterns demonstrated by fluorescein conjugated markers with laser scanning confocal microscopy, and we never bleached tissue thus examined because we demonstrated that the laser scanning confocal microscope can image through melanin.[2 5]

We feel that the routine detection of tumour blood vessels in malignant uveal melanomas can be performed easily using the simple PAS stain without haematoxylin counterstaining.[2–7] After learning how to interpret the results,[3 4] this staining technique is as sensitive as other cost intensive and time consuming immunohistochemical staining techniques of blood vessels in uveal melanocytic lesions. In our view, the detection of the microcirculation architecture is the most important prognostic factor for the survival of patients with uveal melanoma.

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1 Foss AJE, Alexander RA, Hungerford JL, Harris AL, Cree IA, Lightman S. Reassessment of the PAS patterns in uveal melanoma. *Br J Ophthalmol* 1997;**81**:240–6.
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Reply

EDITOR,—We are grateful for the opportunity to respond to the letter from Rummelt and Naumann. It must be unusual for differences in tinctorial staining to be discussed in the pages of an ophthalmology journal! They reiterate a number of points made by Folberg[1] to which we would respond as follows.

We do not believe that a PAS stain can be considered as specific or as sensitive a method for the detection of blood vessels as immunohistochemistry for endothelial cells. However, we would emphasise the fact that we did find the patterns to be statistically significant, as have other groups.[2] We stand by our histological interpretation of the "silent" areas and would point out that transverse sections of blood vessels can be identified within the silent areas shown in Folberg's original paper, Figure 2.[3] Of the defined Folberg patterns, we feel that this term best describes the area shown in our Figure 1.

We would argue that blood vessels with collagen around them are fibrovascular structures and that new canalised or non-canalised capillary buds are unlikely to show such structure. The Folberg patterns contain mature vessels but they are not the sole source of perfusion for which the tumour depends on for oxygenation—the "silent" areas in some tumours in the PAS stained sections are too large to support an actively growing tumour and we would expect much more necrosis in such tumours if no blood vessels were present.[4] The F8 stain shows that there are capillaries.

The autofluorescence we found was primarily associated with collagen, of which there is little around vessels in the choriocapillaris. Autofluorescence is a well known phenomenon which can easily be used to show fibrovascular trabeculae in unstained melanoma and other tissues.[5] We note that the legend of Figure 3 of Folberg *et al*'s original description[3] describes autofluorescence in their preparations. It is sometimes difficult to convince editors, but unstained controls are essential in fluorescence studies and should be illustrated. We note that the controls referred to by Rummelt and Naumann in their letter applied to their later work which used cyanine[6] and not fluorescein which was used in the original paper.[3]

We agree with Rummelt and Naumann that the detection of the microcirculation is of prime importance in determining prognosis in patients with choroidal melanoma. There is now a considerable body of published research indicating the importance of angiogenesis in uveal melanoma.[1–3 6–11] In contrast with other tumours in which angiogenesis is thought to be important,[12] there is no effective therapy for metastatic disease and death from uveal melanoma therefore reflects metastatic spread. There can be little doubt that the prognostic significance of tumour vascularity is due either to the ability of highly vascular tumours to metastasise more readily or the ability of metastatic cells, from such tumours, to be able to grow at sites of implantation.

It is quite clear that both the PAS patterns and the microvessel count have prognostic significance in uveal melanoma. It is not clear what the PAS patterns represent (we suggest hypotheses in our paper) but as the patterns do include mature blood vessels, it can be argued that they, in some way, represent the ability of the tumour to stimulate the formation of a mature blood supply. Microvessel density, by contrast, reflects the proliferation of capillary endothelium within the tumours and not all of the F8 stained structures may not be canalised. These facets of the biology of uveal melanoma are clearly related and important to tumour growth. However, at present we fail to see the point of routinely using either the Folberg or Foss methods for assessing angiogenesis in melanoma: there are no treatment decisions to be made! Nevertheless, we accept that an anti-angiogenesis strategy may be feasible and we hope that a measure of the angiogenic capacity of uveal melanoma will then prove useful in selecting patients for appropriate adjuvant therapy. Perhaps this is where we should now concentrate our efforts.

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Authors' reply

EDITOR,—The issues that separate us from Foss and Cree extend considerably beyond the subject of tinctorial staining of histological tissue sections. The overall goal of our research has been to design a non-invasive technique that would permit ophthalmologists to grade the biological behaviour of patients with ciliary body and choroidal melanomas at the time of diagnosis.[1 2] As we have noted previously, uveal melanomas are among the few forms of cancer that are treated before a pathologist can examine tissue and assign a biological grade. Our research strategy was designed to identify a characteristic from tissue sections of eyes removed for ciliary body or choroidal melanomas that would be a reliable and strong marker for metastasis and that would be detectable by a non-invasive imaging technique. To date, we have focused our attention on the architecture of the microcirculation of uveal melanomas as such a marker.

Foss and coworkers[3 4] have repeatedly challenged our assertion that patterns detected by our modification of the PAS stain actually detect blood vessels (an observation which, if correct, would invalidate the use of the microcirculation as a clinically relevant marker of malignant potential). At first, they declared that our microcirculation patterns were an artefact of autofluorescence. They repeat this assertion in their latest letter, but concede that their objection is restricted to a caption from one photomicrograph[1] (they do not contest other work[2 5] that supports our claim). Microvessels consist of more than tubules of endothelium. Even microvessels are associated with a extracellular matrix sheath. The modified PAS stain detects extracellular matrix elements associated with the microcirculation.[6] Perhaps the most conclusive evidence to support our claim that the modified PAS stain accurately reflects the microcirculation in choroidal and ciliary body melanomas comes from a new report that shows an excellent correlation between angiograms of patients which choroidal melanomas taken with indocyanine green angiography with laser scanning confocal microscopy just before enucleation: careful mapping of histological sections from these tumours showed a correspondence between microcirculation patterns detected clinically and microcirculation patterns demonstrated with our modified PAS stain.[7]

Angiogenesis is more than the production of new vessels: the microcirculation remodels and microcirculatory patterns in uveal melanoma therefore represent not only the new vessels but also the remodelling of this new vascular supply.[8] Study of microcirculation architecture is therefore different from the study of vascular density. Every independent investigator[9 10] who has studied microcirculation architecture in uveal melanomas has confirmed the association between the presence of microvascular loops or networks with metastasis (also M Jager, MD, personal communication, May 1997). However, at least two groups have now failed to confirm Foss' study linking microvascular density with metastasis[11] (also M Jager, MD, personal communication, May 1997).

Finally, we are dismayed by Foss and Cree's failure to see the point of routinely assessing angiogenesis in uveal melanomas because "there are no treatment decisions to be made". This would apply to all basic research concerning diseases that cannot be treated effectively today. Progress is being made in the laboratory, albeit slowly, in designing effective strategies to treat metastatic uveal melanoma,[12] even though no effective treatment is available presently. Until such treatment does become available, it is reasonable to gather as much information as possible from prognostic features such as the tumour's microcirculation architecture.

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1 Folberg R, Peter J, Gruman LM, Woolson RF, Jeng G, Montague PR, *et al*. The morphologic characteristics of tumor blood vessels as a marker of tumor progression in primary human uveal melanoma: a matched case-control study. *Hum Pathol* 1992;**23**: 1298–305.
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BOOK REVIEW

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Glaucoma: a colour manual of diagnosis and treatment. 2nd ed. By JJ Kanski, JA McAllister, JF Salmon. Pp 176. £55. Oxford: Butterworth-Heinemann, 1996. ISBN 0 7506 1820 5.

This book is the second edition of one of a series of colour manuals in ophthalmology, several of which have been written by Jack Kanski. As the preface states, this book is intended for trainee and general ophthalmologists and also for optometrists and other ophthalmic practitioners, and is intended to provide a systemic, detailed, and practical approach to the various forms of glaucoma. This is a medium sized book with 25 chapters covering most aspects of glaucoma including basic physiology, examination techniques, different types of glaucoma, and various treatments.

This book is written in the clear, didactic style, which combined with the many colour photographs and Terry Tarrant's illustrations, has made Jack Kanski's original book *Clinical Ophthalmology* the standard text for trainee ophthalmologists in the UK and many other countries. This book has also benefited from the contributions of the two co-authors James McAllister and John Salmon who have extensive clinical experience in glaucoma.

Inevitably, for many readers who have used the glaucoma chapter in clinical ophthalmology, there will be many familiar passages. However, this book also contains many new illustrations and there is generally more detail on every subject covered. There are also new, albeit brief, sections covering important growth areas such as new techniques in the early diagnosis of glaucoma. This book also contains practical details of diagnostic (for example, gonioscopy), laser, and surgical procedures including the management of complications of these procedures.

Inevitably, a book like this cannot be comprehensive, particularly in a field such as glaucoma which is moving so rapidly. For instance, the next edition would benefit from a section on contemporary methods of high resolution disc imaging and some mention of the recently described gene loci associated with various types of glaucoma.

In conclusion, this is an excellent primer book in glaucoma that is well illustrated and easy to read with many useful practical tips, particularly for the trainee ophthalmologist. It will hopefully interest and stimulate the reader to move on to more detailed textbooks on this fascinating group of disorders.

P T KHAW

NOTICES

Antipersonnel mines

The latest issue of the *Journal of Community Eye Health* (no 23) deals with injuries caused by antipersonnel mines. Editorial by Robin Coupland, and papers covering ocular trauma in Cambodia, Albania, Eritrea, Ethiopia, and Afganistan. For further information please contact Ann Naughton, ICEH, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL. (Tel: (44) 171 608 6910; fax: (44) 171 250 3207; email: eyeresource@ucl.ac.uk) Annual subscription: £25. Free to health workers in developing countries.

20th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 20th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 5–10 February 1998 at the Hyatt Regency Cerromar Beach Hotel, Dorado, Puerto Rico. 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)

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

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)

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).

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).

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

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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).

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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.

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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.

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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.*

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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.

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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.

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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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