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.

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.

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 facitral dermaitis 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). Patients frequently have an impulsive personality disorder.

Skin lesions are produced or significantly exacerbated by self inflicted trauma. Recurrent excoriation produces inflammation and lichenification of the skin; the resultant irritant 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. In the right handed person, the left side is usually involved.

Self inflicted dermatoses vary greatly because of the wide range of methods that are used for inflicting the lesions: cutting, abrasion, burning, application of chemicals, and injecting various products. This diversity makes it particularly difficult to diagnose dermatitis artefacta.

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. 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. 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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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 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.

REFERENCES
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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Indocyanine green angiography and idiopathic polypoidal choroidal vasculopathy

EDITOR,—Idiopathic polypoidal choroidal vasculopathy (IPCV) also known as “posterior uveal bleeding syndrome” or “serosanguineous exudative maculopathy” is a relatively uncommon clinical entity (CNV) with a well-defined angiographic pattern. The presence of IPCV is associated with systemic hypercoagulability. The angioarchitecture of IPCV is characterized by a well-defined vascular network that includes the following features: (1) a well-defined perfused vascular cavity; (2) a well-defined vascular boundary; (3) a well-defined perfused vascular network; and (4) a well-defined perfused vascular cavity. In this study, we describe 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.² Our 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 including 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 neurosensorv 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 “histro 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 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 at the inferior border 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 become hypofluorescent (arrows).
polypoidal lesion
choroidal circulation accentuates the vascular thesamelevelasfouroffiveuntreatedeyes.
visual acuities of the six treated eyes reached exudative manifestations. However, finalinnineeyeswithsixshowingresolutionofthe report laser photocoagulation was performed cases involving the central macula. In one vision as described in our patient. Treatment macula, however, may result in severe loss of in most cases. Involvement of the central distinction concerning the natural course, between IPCV and CNV, an important
intraocular pressures were normal. He examination. Optic atrophy was not present,
lightreflexwasdefective.Noremarkablefind-
lossinbotheyes.Hiscorrectedvisualacuities 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 electoretinogram, but flash visually evoked potential was non-recordable.

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. ICVA with its improved imaging of the choroidal circulation accentuates the vascular polypoidal lesion and allows differentiation between IPCV and GNV, 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.

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

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

Artery occlusion.

Optic nerve compression with central retinal pressure and subsequently cause blindness by eye was limited, with inability to supraduct sluggish reactions. Upward movement of the left upper eyelid. His best corrected visual eye was limited, with inability to supraduct sluggish reactions. Upward movement of the left upper eyelid. His best corrected visual eye was limited, with inability to supraduct sluggish reactions.

**CASE REPORT**

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

**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. 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.

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 papryacea during the removal of the polyoid tissue from the ethmoid sinus. 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 report an unusual but dangerous simultaneous bilateral orbital complication of endoscopic sinus surgery and its treatment.

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

**Figure 2** Coronal orbital computed tomogram obtained after operation shows clearance of haematoma in both the superior orbital subperiosteal spaces.
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 British Journal of Ophthalmology (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 by Foss. The enlarged vessels marked as 2 would not have been classified as “normal” according to our original description. Therefore, we were not surprised that the authors could demonstrate a suggestive role for vascular endothelial growth 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, which would allow easy identification of basement membrane materials, and connective tissue as shown in Figure 2. In contrast with the patterns to be statistically significant, as we would expect much more necrosis in such tumours if no blood vessels were present. The F8 stain shows that there are capillary buds which are unclassified vessels. The autofluorescence we found was probably associated with collagen, of which there is little around vessels in the choriodiopilars. Autofluorescence is a well known phenomenon which can only be used to show fibrovascular trabeculae in unstained melanomas and other tissues. We note that the legend of Figure 3 of Foss et al’s original description 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 Rummel and Naumann in their letter applied to their manuscript work which used the.Ulex europaeus lectin, and not the fluorescein which was used in the original paper.

We agree with Rummel and Naumann that the detection of the microcirculation of significance is of prime importance in determining prognosis in uveal melanoma patients with choroidal melanoma. It is now a considerable body of published research indicating the importance of angiogenesis in uveal melanoma. In contrast with other tumours in which angiogenesis is thought to be important, 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 vasculature 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 is 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. Moreover, contrast, reflects the proliferation of capillary endothelium within the tumours and not all of the F8 stained structures may 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 uveal melanoma; there are no treatment decisions to be made! Nevertheless, we accept that an antiangiogenesis 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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8. Pe’er J, Rummelt V, Mawn L, Hwang T, Woolson RF, Folberg R. Mean of the ten largest nucleoli, of fibrovascular 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 (also M Jager, MD, personal communication, May 1997).
9. 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 be inconsistent with 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, even though not all 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.

Editors’ reply—The issues that separate us from Foss and Cree extend considerably beyond the subject of tumoral staining of histological tissue sections. The overall goal of our research has been to design a non-invasive technique that would permit ophthamologists to grade the biological behaviour of patients with ciliary body and choroidal melanomas at the time of diagnosis. As we have noted previously, uveal melanomas are among the few cells not in a large number of cancer that are treated before a pathological exam can test examine and assign a clinical grade 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, even though not all 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.

**BOOK REVIEW**

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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 systematic, 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 been 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 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 Cerro Mar 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: 410 955-8087; email: cmenet@som.adm.jhu.edu; homepage: http://www2.med.jhu.edu/cme)

The 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 will have to be submitted in English or German by 31 March 1998 to Stifterverband für die Deutsche Wissenschaft e.V., Herrn Peter Beck, Postfach 16 44 60, D-45224 Essen, Germany.

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: 0043-512-264683; email: lsn@net4you.co.at; homepage: http://www.carrier.co.at/lsn)

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.

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

REVIEWS

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

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