Dense Kayser-Fleischer ring in asymptomatic Wilson’s disease (hepatolenticular degeneration)

The Kayser-Fleischer ring is the single most important diagnostic sign in Wilson’s disease; it is found in 95% of patients. Virtually all patients with Kayser-Fleischer rings have neurological manifestations. Pseudo rings have been described in other conditions. The density of a Kayser-Fleischer ring correlates with the severity of Wilson’s disease. We describe a rare case of a dense Kayser-Fleischer ring in an asymptomatic patient with an extremely high liver copper content. The recent significant genetic advances, and the clinical implications are discussed.

Case report

At the age of 14 this 23 year old white woman had an evisceration of her left eye for painful rubeotic glaucoma following chronic retinal detachment, resulting from ANA positive iridocyclitis. Her optician referred her because over the past few years, her left prosthesis had repeatedly needed an increasingly dense brown ring painted onto it, to match her remaining eye. On examination the visual acuity in the right eye was 6/6 with a heavily pigmented red brown Kayser-Fleischer ring (Fig 1). Her systemic and neurological examination was normal.

Baseline biochemistry, liver function, hae-matology, and cerebral magnetic resonance image (MRI) were normal. She was ANA positive 1 in 20, serum copper 11.6 µmol/l (normal 11–22), and ceruloplasmin 0.15 g/l (normal 0.15–0.45). Twenty four hour urinary copper 9.2 µmol/24 h (normal 0.9 µmol/24 h). Haematoxylin and eosin stained liver biopsy showed normal architecture. Ornithine and rhodanine stains showed patchy excessive copper binding protein, and copper within the hepatocytes. The quantified copper content of the sample was hugely elevated at 3000 µmol/l (normal 20–50 µmol/l).

She was treated with trientine dihydrochloride. The Kayser-Fleischer ring initially became denser but has faded considerably over the past 5 years; a thin rim still persists (Fig 2).

The patient remains well with no hepatic or neurological manifestations.

Comment

This patient had an extremely elevated liver copper level, 60 times the normal level. She had no hepatic or neurological abnormalities. Despite the severity of her condition she remained neurologically asymptomatic with normal liver architecture.

Wilson’s disease is inherited as an autosomal recessive trait. The defect has been mapped and sequenced to the long arm of chromosome 13 (13q14.3). The Wilson’s gene encodes a P-type ATPase bound to the endoplasmic reticulum (ER), ATP7B. The patient was found to have a deletion in exon 1 of ATP7B.

There are a large number of copper binding ATP7B mutations. The clinical heterogeneity and overlap of clinical manifestations suggest that locus heterogeneity alone is unlikely to be responsible. It has been hypothesised that there is a subset of pedigrees in which an additional gene is affected other than that for ATP7B. Genes encoding proteins for detoxification of stored copper—for example, metallothionein, and neutralisation of free radicals such as super oxide dismutase— this could be a plausible explanation as to why such an extraordinarily high level of copper was bound safely in this patient’s liver.

Untreated Wilson’s disease has progressive, irreversible consequences, and ultimately causes death. The identification of a Kayser-Fleischer ring remains the most important clinical sign for the diagnosis of Wilson’s disease.

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References


Rapidly developing intimal fibrosis mimicking giant cell arteritis

Temporal headache associated with a tender superficial temporal artery and decreased pulse on palpation are characteristics of giant cell arteritis. We report the clinical and biopsy findings in a patient in whom these symptoms were caused by a rapid developing intimal fibrosis.

Case report

A 51 year old woman presented with a 2 month history of a tender and painful left superficial temporal artery (STA). First she noticed a “thickened cord” on the left temple which felt pulsatile on palpation. Within 6 weeks the throbbing pulse disappeared and was followed by tenderness and pain. She also reported arthritic pain in the limbs and a morning cough over a 2 year period. She had smoked 40 cigarettes per day for 30 years. On examination the left STA felt hardened, knot-ted, non-pulsatile, and was slightly tender over a 2 cm distance (Fig 1, cross). There were no bruits on auscultation of major arteries. The facial and maxillary arteries were soft and
CRP were normal. She continued having diffuse body pain.

Comment

GCA is a neurological emergency which, when left unrecognised and untreated frequently leads to permanent blindness. 3 ESR can be normal in 5–30% of patients,3 but this is an exceptionally rare diagnosis in middle aged patients.

In summary, our patient presented with a clinical picture suggestive of vasculitic occlusion of the superficial temporal artery, polymyalgia, and cough. We present this case as an illustration that this clinical picture can result from rapidly developing intimal fibrosis without any evidence of inflammation.

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References


Fixed dilated pupil following deep lamellar keratoplasty (Urrets-Zavalia syndrome)

A fixed dilated pupil following penetrating keratoplasty is a well recognised if rare postoperative complication. We report a case of Urrets–Zavalia syndrome following a deep lamellar keratoplasty (DLK).

The mydriasis following penetrating keratoplasty was first described by Castroviejo (Castroviejo R, personal communication) but it was Urrets-Zavalia who first published his observations on a series of six cases and suggested an association of fixed dilated pupil, iris atrophy, and secondary glaucoma.3

The incidence of this syndrome is estimated at 5.8%, from pooled data on 445 eyes undergoing penetrating keratoplasty for keratoconus, of which 24 eyes developed a fixed dilated pupil. Davies and Ruben also found a similar incidence.4 However, other more recent studies find no cases,5–7 and some even question its continued existence. This may in part reflect improved surgical technique and differing diagnostic criteria.

The pupil can become abnormally dilated following penetrating keratoplasty for keratoconus, particularly if dilating drops are used. There are three main groups of pupillary dilatation:

1. A pupil with normal light and near reaction which is at least 1.5 mm larger than its fellow unoperated eye. It fully constricts with topical miotics. An incidence of approximately 90% has been reported for this type of abnormality.

2. An unreactive paretic pupil that returns slowly to normal.

3. Irreversible pupil dilatation with iris atrophy.

This syndrome has also been reported when no dilating drops were used.2 In addition to the pupil and iris abnormalities, Urrets-Zavalia also described other features—iris ectropion, pigment dispersion, anterior subcapsular cataract and posterior synechiae. No early postoperative pressure rises were documented, although some had peripheral anterior synechiae and secondary glaucoma. Gasset also describes the glaucoma as a secondary phenomenon, commenting that it is not integral to the syndrome. In the series presented by Pouliquen et al, severe anterior uveitis, fibrinous exudate, and binocular posterior synechiae are described.8 An early postoperative intraocular pressure rise is documented in two of the three cases presented by Tuft and Buckley.9

We describe the case of a patient undergoing deep lamellar keratoplasty for keratoconus who developed a permanently dilated pupil with iris atrophy. In addition she had marked anterior uveitis and posterior synechiae similar to the cases presented by Pouliquen et al. Dilating drops were not used.

Case report

A woman with keratoconus underwent a left deep lamellar keratoplasty at the age of 28 because of unstable contact lens fit and progressive central corneal scarring. A 7.75 mm graft into a 7.5 mm diameter recipient DLK was performed under peribulbar anaesthesia with no evidence of inflammation. From rapidly developing intimal fibrosis with severely narrowed lumen (asterisk). The tissue was examined using the serial section technique in order to minimise the chance of a false negative result.

The histological appearances were those of a severely fibrosed artery devoid of any inflammatory changes and consistent with arteriosclerosis. The intima was considerably thickened, the internal elastic lamina was preserved, and the media had some degree of sclerosis. The intima was considerably thickened (b) and shows tissue proliferation. There are no signs of necrosis, inflammation, or thrombus.
Asymptomatic oculopalatal myoclonus: an unusual case

Oculopalatal myoclonus is characterised by rhythmic pendular vertical eye movements associated with synchronous contraction of the soft palate. It produces intractable oscillopsia, and is normally the result of brainstem haemorrhage. However, it does not usually become manifest until several months or even years later, with the longest recorded interval being 49 months. We present an unusual case of a patient who sustained a brainstem haemorrhage following trauma. Eight years later he was incidentally noted to have oculopalatal myoclonus, and surprisingly was asymptomatic.

Case report

A 61 year old man was admitted to the neurosurgery department following head trauma. A computed tomography (CT) scan showed subarachnoid bleeding. An magnetic resonance image (MRI) revealed left frontal and subarachnoid bleeding. An magnetic resonance imaging (MRI) scan showed subarachnoid bleeding. An MRI scan revealed bilateral cranial nerve palsies, and a small brainstem haemorrhage. Following the head injury he complained of double vision on downgaze. Examination revealed bilateral fourth cranial nerve palsies, which resolved spontaneously.

Nine months later the patient developed acute angle closure glaucoma in his right eye, which was unresponsive to medical therapy. He subsequently underwent a right trabeculectomy. Thereafter, his visual acuities were 6/18 in the right eye and 6/6 in the left eye, and he was reviewed annually at a glaucoma clinic.

While attending 8 years after his head injury, an audible click was heard emanating from the patient. He was unaware of this because of longstanding sensorineural deafness. However, on further questioning his wife stated that she had been aware of the clicking for several months. Examination of the soft palate revealed rhythmic contractions that were synchronised with the auditory clicking. There was a right unilateral vertical pendular nystagmus, although no nystagmus was noted in the left eye. A diagnosis of oculopalatal myoclonus was confirmed by the demonstration of hydrolytic enzymes.

Comment

Oculopalatal myoclonus is a rare condition normally resulting in intractable oscillopsia, thought to be caused by a lesion in the myoclonic triangle, which consists of the red nucleus, the ipsilateral inferior olive, and the contralateral dentate nucleus. To our knowledge, this is the first reported case of asymptomatic oculopalatal myoclonus. It also illustrates that the latency period may be longer than that previously described.

Bilateral exudative retinopathy as the initial manifestation of retinitis pigmentosa

A 12 year old male presented with a 3 week history of blurred vision in both eyes. There was no significant medical or family history. Visual acuities were 6/120 in the right eye and 6/15 in the left eye. Anterior segment examination was normal. The posterior segment of
in 1956 and exudative retinopathy was first described. The association between retinitis pigmentosa and Coats' disease had mostly resolved, the exudation was slightly less extensive and the visual acuity was unchanged.

Resolution of proliferative venous stasis retinopathy after carotid endarterectomy

Ocular ischaemic syndrome (OIS) may present as an asymmetrical retinopathy in diabetic patients. We report a case of asymmetric diabetic retinopathy with posterior segment neovascularisation due to OIS associated with critical isspilateral carotid stenosis where the neovascularisation resolved after carotid endarterectomy.

Case report

A 50 year old woman presented in May 1996 with left sided weakness. She had hypercholesterolaemia, hypertension, a family history of vascular disease, and was a smoker. She was found to be diabetic with peripheral retinal ischaemia and disc neovascularisation in the right eye, and minimal retinal ischaemia in the left eye (Fig 1). Her visual acuities were 6/12 in the right eye and 6/9 on the left. There was no anterior segment neovascularisation in either eye. Carotid Doppler and carotid angiography showed critical stenosis at the origin of the right internal carotid artery. The right middle cerebral artery branches were visualised as a result of retrograde flow through the ophthalmic artery. The left internal carotid artery was narrowed by 50% and there were no collaterals to the right hemisphere (Fig 2). Fluorescein angiography revealed a prolonged transit time with slow filling of choroidal and retinal vasculature, peripheral retinal capillary closure, and leakage from the disc neovascularisation.

One year later the optic disc neovascularisation and retinal ischaemia were unchanged with no iris neovascularisation. In April 1997 she underwent an uneventful right carotid endarterectomy. Two months later she developed clinically significant macular oedema in the right eye that was treated with focal argon laser photocoagulation.

Six months later the maculopathy had resolved and 14 months after surgery there was complete resolution of the optic disc neovascularisation. Three years after surgery the right eye had a visual acuity of 6/9, a near normal fluorescein angiogram transit time, minimal peripheral retinal ischaemia, and no posterior segment neovascularisation.

Figure 1 Presenting fundus photograph showing disc neovascularisation (A) with corresponding fluorescein angiogram showing leakage from these vessels (B). The neovascularisation has resolved 14 months after surgery (C) and is confirmed on fluorescein angiography (D).

Figure 2 [A] Fluorescein angiogram of the right eye showing retinal ischaemia with adjacent telangiectasia. [B] Scotopic electroretinogram showing isoelectric response in both eyes.

Both eyes showed extensive subretinal exudation, serous retinal detachment, and overlying retinal telangiectasia (Fig 1A). There was cystoid macular oedema with a lamellar macular hole in the left eye. The optic discs appeared normal. Additionally, mottled granularity of the retinal pigment epithelium (RPE) was noted in the mid-periphery of both retinas (Fig 1B). Upon further questioning, he admitted to night blindness.

A fluorescein angiogram confirmed retinal telangiectasia, serous retinal detachment, and macular oedema (Fig 2A). An electroretinogram (ERG) showed an isoelectric response under both scotopic and photopic conditions (Fig 2B). Visual field testing revealed marked constriction in both eyes. A diagnosis of retinal telangiectasia, exudative retinopathy, and retinitis pigmentosa was made and the areas of most severe telangiectasia were treated with retinal cryotherapy. One year later the retinal telangiectasia had mostly resolved, the exudation was slightly less extensive and the visual acuity was unchanged.

Comment

The association between retinitis pigmentosa and exudative retinopathy was first described in 1956 and has been termed a “Coats'-like RP.” Various studies have suggested that 1–4% of RP cases will show such a response. This entity differs from true Coats' disease in that Coats'-like RP often occurs bilaterally, has no sex predisposition, and shows diffuse pigmentary alterations in both fundi. The cause is unknown but it may represent a vasodilatory response to toxic products of photoreceptor/RPE degeneration. Our case is unusual in that almost all previous reported cases have occurred in the setting of long-standing RP. Our patient had never consulted an ophthalmologist despite being night blind and having markedly constricted visual fields. We recommend that ophthalmologists consider an underlying diagnosis of retinitis pigmentosa in any patient presenting a picture of bilateral exudative retinopathy.

Acknowledgments

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Ocular ischaemic syndrome (OIS) is characterised in the anterior segment by flare and initial hypotony, with later iris neovascularisation. Retinopathy with neovascular proliferation occurs in the fundus because of chronic hypoperfusion. The development of neovascularisation of iris neovascularisation can lead to permanent blindness. In the diabetic patient OIS is superimposed on any pre-existing diabetic retinopathy, and markedly asymmetric retinopathy should prompt a search for underlying ischaemia from carotid occlusive disease. Diabetic patients with marked proliferative changes require treatment with panretinal photocoagulation (PRP), which has been shown to reduce the risk of severe visual loss and neovascular glaucoma. However, there is no clear evidence for the benefit of PRP in patients with OIS. In one study only 36% of OIS patients with iris neovascularisation responded to PRP which may be due to uveal rather than retinal ischaemia. In the case presented the patient was not treated with immediate PRP but reviewed regularly. The disc new vessels did not progress in the year before carotid endarterectomy and there was no immediate threat to vision.

Carotid stenosis can result in changes in the ophthalmic artery blood flow ranging from reduced antegrade to reversal of flow. If there is inadequate crossflow in the circle of Willis from the contralateral internal carotid, reversal of flow occurs in the ophthalmic artery as a consequence of a collateral circulation from branches of the external carotid artery. Although some series show no correlation between direction of flow and the severity of OIS Kert et al in a study of 45 patients found that only reversal of flow was associated with structural changes of OIS. One similar case exists in the literature where neovascularisation resolved within several days of carotid endarterectomy (CEA). Other case reports also show that the retinopathy without neovascularisation can improve following surgery. However, the benefit of carotid endarterectomy in patients with ocular ischaemic syndrome is not quantified and it has never been shown to reverse neovascular glaucoma. The European Carotid Surgery Trial showed that the risk of ischaemic stroke in symptomatic patients with 70–99% carotid stenosis with medical treatment was only 20% over 3 years and CEA lowered this by 50%. Based on the results of this a risk factor score suggested that a cerebral rather than an ocular event had a greater risk for stroke on medical treatment and would therefore derive greater benefit from surgery.

In the absence of iris neovascularisation and severe peripheral retinal ischaemia the ocular changes in patients with OIS can be monitored closely for the development of iris neovascularisation but the retinal vascularisation may not require early treatment with PRP.

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Figure 2 Angiography showing narrowing of the right internal carotid artery (A, arrow) and angiography of the left side (B) revealing lack of crossflow to the right cerebral hemisphere allowing the development of collateral circulation via the ophthalmic artery.
that 20–30% of the 200,000 cases of exudative AMD that present to ophthalmologists each year in the United States would benefit from such photodynamic therapy.8 Southampton Eye Unit serves approximately 570,000 people as part of its main catchment area, corresponding to approximately 1% of the UK population, and produced only one person over the 2 month study period suitable for treatment with verteporfin by the strict criteria for its use.9 The period under study was before the awareness of photodynamic therapy was fully developed and represents an unselected group of patients having some degree of AMD. Subsequent studies might show a higher proportion of suitable patients as the criteria was extended to include occult, myopic, and idiopathic lesions.8

Even with the addition of verteporfin therapy to the ranks of the treatment modalities available, the vast majority of AMD patients are still considered un treatable if the treatment criteria are observed.1,2 Rehabilitation in the form of low vision aids, registration as partially sighted or blind, and the treatment of co-existing ocular disease remains the mainstay of help that the ophthalmologist can offer.4 However, the interest created and accepted value of verteporfin should not be underestimated as it represents a new and non-destructive approach to the problem (in contrast with laser photocoagulation) and the first of a novel treatment option likely to be joined by others in the not too distant future.

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**References**


A new technique for delivering sub-Tenon's anaesthesia in ophthalmic surgery

Sub-Tenon's local anaesthesia has become an accepted technique for anterior and posterior segment eye surgery.4 It is a safe, quick, and effective method of local anaesthesia. However, it requires a certain amount of skill for dissection into the sub-Tenon's space. This dissection can lead to bleeding and chemosis. We describe a modification of the current technique of sub-Tenon's anaesthesia which aims to simplify the method of local anaesthetic delivery, avoid bleeding, and chemosis whilst maintaining effective anaesthesia.

**Case report**

Fifty consecutive patients undergoing anterior or posterior segment surgery scheduled for local anaesthesia were recruited for this study.

For this procedure, a 22 gauge Venflon standard intravenous cannula was used. The conjunctiva was anasthetised with topical 0.5% proparacaine. A Barraquet speculum was inserted. The conjunctiva was grasped 5 mm from the limbus using toothed forceps. Under direct visualisation the tip of a 22 gauge Venflon was used to introduce the plastic cannula under the conjunctiva and Tenon's fascia (keeping the needle tip visible at all times) (Fig 1A). The plastic cannula was advanced over the needle, which was drawn back and removed (Fig 1B). Four millilitres of lignocaine 2% with 30 international units (IU)/ml of hylan G-F22 was then injected through the plastic cannula (Fig 1C).

All 50 patients had anterior segment surgery. Forty six were cataract operations with posterior chamber lens implant, and four were phacoablation cases. None experienced excessive discomfort on delivery of the block. All local anaesthetic blocks were performed by one operator and no complication which prevented surgery occurred. No patient who was scheduled for local anaesthesia was considered unsuitable for this technique.

All patients had effective anaesthesia and akinsia for the surgical procedure. None complained of pain. Top up of anaesthesia was not required in any case. Twenty eight patients had complete or partial ptosis.

Subconjunctival haemorrhage occurring more than one quadrant occurred in one patient, but this did not interfere with surgery. None had chemosis.

**Comment**

Sub-Tenon's local anaesthesia is a well established technique for ophthalmic surgery. Although the Venflon cannula does have a sharp needle, it is used simply as an introducer to place the blunt plastic cannula in the correct tissue plane. The needle tip is kept under direct visualisation at all times. Thus there is minimal risk of ocular perforation with this technique.

Venflon cannulas are used for intravenous delivery of drugs and fluid so are readily available, inexpensive, and disposable. Sub-Tenon's cannulas in current use are specialised cannulas and therefore more costly than intravenous cannulas.

We describe a modification of the current technique of sub-Tenon's anaesthesia which simplifies the method using an intravenous cannula. We predict that this method is easier to learn and that it maintains the efficacy of this type of anaesthesia without compromising safety.

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**References**


Figure 1 New technique for delivering sub-Tenon’s anaesthesia injection.
Dependence of differences in IOP measurements from CCT. Data of figure 2 of Feltgen \textit{et al}. were obtained by digitisation; 68 of the 73 data points could be identified. The four outliers shown by Feltgen \textit{et al} as open circles are omitted. These outliers would have made the use of the same scale in both diagrams more difficult. Thus $n = 64$. Same scale as in Figure 2.

![Figure 1](http://www.bjophthalmol.com)

**Figure 1** Dependence of differences in IOP measurements from CCT. Data of figure 2 of Feltgen \textit{et al} obtained by digitisation. All data points could be identified. The four outliers shown by Feltgen \textit{et al} as open circles are omitted. These outliers would have made the use of the same scale in both diagrams more difficult. Thus $n = 64$. Same scale as in Figure 2.

![Figure 2](http://www.bjophthalmol.com)

**Figure 2** Dependence of differences in IOP measurements from CCT. Data of figure 4 of Ehlers \textit{et al} obtained by digitisation. All data points could be identified. The four outliers shown by Feltgen \textit{et al} as open circles are omitted. These outliers would have made the use of the same scale in both diagrams more difficult. Thus $n = 29$. Same scale as in Figure 1.
that Ehlers had not solved these problems in his trial on human eyes. We are also missing any information about the catana size. Additionally, the device used resembles an “open system” where fluid could circulate through the anterior chamber and trabecular meshwork. This can generate a noticeable change in intraocular pressure.

Ehlers et al measured IOP in patients with an acute eye disease (glaucoma patients requiring surgery) and cataract patients. He changed the IOP of 10 to 30 mm Hg. This method is questionable especially in glaucoma patients, because an acute IOP change could also entail endothelial alterations which could alter CCT. Unfortunately, he didn’t measure the CCT after IOP change. We have no information about the influence of IOP alterations on CCT.

In summary, the above mentioned study gives a hint on the influence of CCT on IOP measurement, but does not provide any assumption. It is amazing that within the last 25 years nearly 50 published papers refer to the Ehlers study without checking the results by intracameral measurement themselves. All papers measuring CCT and applanatory IOP renouncing intracameral measurement described an increasing IOP with increasing CCT. We could also confirm this finding in our study (y’ is the applanatory IOP in mm Hg). Of course, it would be easiest to claim the cornea for this correlation. But it is also conceivable that eyes with thick corneas (for example, OHT) have a reduced ocular outflow facility and consequently elevated IOP—for instance, because of a “thick” trabecular meshwork.

With the present study we tried to find out if the above recommended correcting factors are clinically applicable at all. According to our findings they are not. We found quite variable and unpredictable differences between intraocular pressure and applanation measurement in an individual patient. Interestingly, the same results can be found in the Ehlers study. Therefore, we renew our warning to recalibrate the IOP depending on central corneal thickness.

**Authors’ reply**

In reply to the comments of Stodtmünder and colleagues on our recent paper, we won’t argue about the correlation between central corneal thickness (CCT) and intraocular pressure (IOP), but we mistrust the clinical application of correcting factors. Stodtmünder et al compare our study to that of Ehlers et al which is often cited to prove an influence of corneal thickness in applanation measurement.

In our paper simultaneous IOP measurement by applanation and intracameral tonometry was performed. Assuming a normal CCT of 520 µm, an IOP correction for every 10 µm change in corneal thickness is recommended. But in the Ehlers paper, there are some confusing arguments. Ehlers et al describe a very good correlation between direct and intracameral IOP measurement (correlation coefficient approximated 1). Unfortunately, they didn’t give the measured IOP values. In figure 2, the slopes of correlation lines at different CCT are presented for power factors (not for human eyes!). The increase of the slopes are less than 45°.

The equipment for intracameral measurement in an individual patient. Interestingly, the same results can be found in the Ehlers study. Therefore, we renew our warning to recalibrate the IOP depending on central corneal thickness.

**References**


**NOTICES**

**Onchocerciasis**

The latest issue of *Community Eye Health* (No 38) discusses onchocerciasis and the impact of interventions, with an editorial by Bjorn Thylefors, former director of the Programme for the Prevention of Blindness and Deafness, WHO. For further information please contact *Community Eye Health*, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL. (tel: +44 (0) 20 7608 6990/6591; fax: +44 (0) 7250 5207; email: eyeresource@ucl.ac.uk).

**Annual subscription £25. Free to workers in developing countries.**

**International Centre for Eye Health**

The International Centre for Eye Health has published a new edition of the *Standard List of Medical Equipment, Instruments and Supplies* (2001) for eye care services in developing countries. It is compiled by the Task Force of the International Agency for the Prevention of Blindness. Further details: Sue Stevens, International Centre for Eye Health, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0) 20 7608 6910; email: eyeresource@ucl.ac.uk).

**Leonhard Klein Award 2002**

To promote ophthalmic surgery the Leonhard Klein Foundation bestows the Leonhard Klein Award 2002 for innovative, scientific works in the field of development and application of microsurgical instruments, as well as for microsurgical operating techniques.

The award is endowed with 15,000€ and can be conferred to an individual person as well as to a group of researchers. The prize sum must be spent for research in the field of ophthalmic surgery.

Individual and third party applications are accepted. Five copies of the works must be submitted in either English or German. The deadline for applications is 31 March 2002. Applications should be sent to: Stifterverband für die Deutsche Wissenschaft e.V., Frau Dr Marlien Machet, Postfach 164460, D-45224 Essen, Germany.

**Second Sight**

Second Sight, a UK based charity whose aims are to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British ophthalmologists, is regularly sending volunteer surgeons to India. Details can be found at the charity website (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@yahoo.com).

**Specific Eye Conditions (SPECS)**

Specific Eye Conditions (SPECS) is a not for profit organisation which acts as an umbrella organisation for support groups for any condition or syndrome with an integral eye disorder. SPECS represents over fifty different organisations related to eye disorders ranging from conditions that are relatively common to very rare syndromes. We also include groups who offer support of a more general nature to visually impaired and blind people. Support groups meet regularly in the Boardroom at Moorfields Eye Hospital to offer support to each other, share experiences and explore new ways of working together. The web site www.eyeconditions.org.uk acts as a portal giving direct access to support groups own sites. The SPECS web page is a valuable resource for professionals and may also be of interest to people with a visual impairment or who are blind. For further details about SPECS contact: Kay Parkinson, SPECS Development Officer (tel: +44 (0)1803 524236; email: k@eyeconditions.org.uk; www.eyeconditions.org.uk).

**4th International Conference on the Adjuvant Therapy of Malignant Melanoma**

The 4th International Conference on the adjuvant therapy of malignant melanoma will
be held at The Royal College of Physicians, London on 15–16 March 2002. Further details: Conference Secretariat, CCI Ltd, 2 Palmerston Court, Palmerston Way, London SW8 4AJ, UK (tel: +44 (0) 20 7720 0600; fax: +44 (0) 20 7720 7177; email: melanoma@confcomm.co.uk; website: www.confcomm.co.uk/Melanoma).

**EUPO 2002 Course Retina**
A course on retina will be held on 15–17 March 2002 at Erlangen, Germany, where European professors will teach European residents. Further details: Priv Doz Dr Ulrich Schonherr, Friedrich-Alexander-University of Erlangen-Nuemberg, Department of Ophthalmology, Schwabachanlage 6 (Kopfklinikum), D-91054 Erlangen, Germany (tel: +49 9131 853 4379; fax: +49 9131 853 4332; email: ulrich-schoenherr@augen.imed.uni-erlangen.de).

**XXIXth International Congress of Ophthalmology**
The XXIXth International Congress of Ophthalmology will be held on 21–25 April 2002 in Sydney, Australia. Further details: Congress Secretariat, C/-ICMS Australia Pty Ltd, GPO Box 2609, Sydney, NSW 2001, Australia (tel: +61 2 9241 1478; fax: +61 2 9251 3552; email: ophthal@icmsaust.com.au; website: www.ophthalmology.aust.com).

**12th Meeting of the European Association for the Study of Diabetic Eye Complications (EASDEC)**
The 12th meeting of the EASDEC will be held on 24–26 May 2002 in Udine, Italy. The deadline for abstracts is 15 February 2002. Three travel grants for young members (less than 35 years of age at the time of the meeting) are available. For information on the travel grants, please contact Pr CD Agardh, President of EASDEC, Malmö University Hospital, SE-205 02 Malmö, Sweden (tel +46 40 33 10 16; fax: +46 40 33 73 66; email: carl-david.agardh@endo.mas.lu.se). Further details: NORD EST CONGRESSI, Via Aquilea, 21–33100 Undine, Italy (tel: +30 0432 21391; fax: +39 0432 50687; email: nordest.congressi@ud.nettuno.it).

**Singapore National Eye Centre 5th International Meeting**
The Singapore National Eye Centre 5th International Meeting will be held on 3–5 August 2002 in Singapore. Further details: Ms Amy Lim, Organising Secretariat, Singapore National Eye Centre, 11 Third Hospital Avenue, Singapore 168751 (tel: (65) 322 8374; fax: (65) 227 7290; email: Amy_Lim@snec.com.sg).

**International Society for Behçet’s Disease**
The 10th International Congress on Behçet’s Disease will be held in Berlin 27–29 June 2002. Further details: Professor Ch Zouboulis (email: zoubbere@zedat.fu-berlin.de).

**CORRECTION**
We regret that an error occurred in the mailbox letter published by Kenawy et al in the November 2001 issue of BJO (2001;85:1394–5). The name of one of the authors was incorrect and should have been Omar M Ayoub.
Dense Kayser-Fleischer ring in asymptomatic Wilson's disease (hepatolenticular degeneration)
Charlotte Anne Sullivan, A Chopdar and G A Shun-Shin

Br J Ophthalmol 2002 86: 114
doi: 10.1136/bjo.86.1.114

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