Spontaneous venous pulsations should be monitored during glaucoma therapy

It is well established that lowering intraocular pressure slows or halts progression of glaucoma. None of the less, changes in intraocular pressure also affect the pressure gradient across the lamina cribrosa. Normal intraocular pressures combined with low intraocular pressures produce the same pressure differential across the laminae as elevated intraocular pressures in conjunction with normal intracranial pressures. Failing to factor in both intraocular and intracranial pressures may create an inappropriate distinction in the pathogenesis of glaucomas currently labelled as normal tension from those where elevated pressures are present. This may be secondary to mechanical deformation and destruction of lamina cribrosa causing axonal degeneration, or conceivably to pressure related ischaemic events. A goal of therapy in either situation should be equalisation of prelaminar intraocular pressure with opposing retrolaminar tissue and cerebrospinal fluid pressure.

Spontaneous venous pulsations occur when intraocular pressure during systole exceeds central retinal venous pressure, which, in turn, reflects intracranial pressure transmitted through the perineural subarachnoid space and retrolaminar tissue pressure. Lowering intraocular pressure until venous pulsations cease minimises the pressure gradient between mean intraocular and retrolaminar pressure, and allows a clinical estimation of intracranial pressure. Keeping momentary fluctuations in mind, such end points may guide intraocular pressure lowering strategies to help avoid overtreatment or undertreatment of individuals with glaucoma. Therapies that lower intraocular pressure without also lowering intracranial pressure (such as topical rather than oral carbonic anhydrase inhibitors) should be favoured. The question of whether intracranial pressure raising strategies should be considered may arise in select cases. Finally, a consistent lack of spontaneous venous pulsations in patients with ocular hypertension, especially when venous pulsations can be elicited by digital pressure on the globe, may indicate sufficient counterbalancing intraocular pressure to render intraocular pressure reduction unnecessary.

References

Acanthamoeba keratitis in Ghana

Suppurative keratitis due to Acanthamoeba spp is most commonly associated with poor contact lens hygiene. However, recently there have been reports of keratitis caused by Acanthamoeba spp in the tropics in non-contact lens wearers. We report one such case of Acanthamoeba keratitis in Ghana, west Africa.

Case report
A 25 year old male driver (from Accra) reported to a hospital in Bawku, northern Ghana. He presented with symptoms of chronic corneal ulceration (duration of symptoms 31 days) and visual acuity in his affected eye was reduced to perception of hand movements only at presentation. The patient did not recall experiencing any trauma to his eye before symptoms. The patient had already taken antibiotics before primary presentation at the hospital but did not bring them with him to clinic and therefore it is not known which they were.

On examination there was a central corneal ulcer (greatest diameter 8 mm) involving more than 50% of the corneal epithelium and more than two thirds infiltrate. The ulcer was stained with Gram stain smear. Morphologically consistent with the appearance of Acanthamoeba were seen (Fig 1) in the stained smear.

At the time of presentation the patient was treated empirically with chloramphenicol, gentamicin, and econazole. After 7 days on this regimen the ulcer had not reduced in size; none of the patients was a candidate for contact lens wearers. However, 49% of the patients reported experiences of trauma to the affected eye.

The first cases of Acanthamoeba keratitis in non-contact lens wearers in Africa were reported in Mali by Resnikoff et al. This group subsequently reported a further 22 cases between 1990–5. However, there have been no other reports, from Africa, of non-trauma related Acanthamoeba ulcers.

The case presented is the first report of a corneal ulcer caused by Acanthamoeba spp in Ghana and highlights the need to consider Acanthamoeba spp as a potential causative agent of chronic keratitis in patients presenting at rural hospitals in the tropics. In addition, the usefulness of simple stains such as lactophenol cotton blue and Gram stain to visualise Acanthamoeba spp is demonstrated as previously described in the literature.

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References
Need for measurement of porphyrins in teardrops in patients with congenital erythropoietic porphyria

Congenital erythropoietic porphyria (CEP; MIM#237630) is an extremely rare disorder inherited as an autosomal recessive trait. The cause of this disease is the deficient activity of uroporphyrinogen III synthase (UROS; EC 4.2.1.75). Since a cloning of UROS (Genbank NM000375), efforts have been made to clarify underlying mutations that cause CEP. To date, more than 20 mutations of UROS have been described. Identification of UROS mutations at the molecular level is important for genetic counselling and prenatal diagnosis of affected families.

Clinically, CEP is characterised by severe cutaneous photosensitivity, chronic haemolysis, and massive porphyrinuria resulting from the accumulation in the bone marrow, peripheral blood, and other organs of large amounts of predominantly type I porphyrins, which are not substrates for haem synthesis. Red urine may be observed from infancy, and the teeth become stained red. Haemolytic anaemia, an additional complication, may be helped by splenectomy. Besides such classic manifestations ocular involvement, including scleral changes, has been reported in patients with CEP. Furthermore, we recently reported the evidence of the accumulation of porphyrins in teardrops in a Japanese patient with CEP who showed scleral changes. To confirm whether the accumulation of porphyrins is a common feature and is a direct cause of ocular involvement, we analysed and confirmed the presence of porphyrins in teardrops in an additional three Japanese patients with CEP.

Case reports

We analysed three Japanese patients with CEP, all of whom were diagnosed by their typical clinical manifestations and by the elevation of porphyrins (uroporphyrin I and coproporphyrin I) in urine (Table 1). For case 2, we performed sequence analysis of UROS and identified a T to C transition of nucleotide 745 that predicted a serine to proline substitution (Fig 1), which predicted a premature stop codon (Q249X). All patients were observed to have sclerotic necrosis of the limbus and pigmentation of eyelids (Fig 1), but in case 4 (KT), these changes were relatively mild. No patients showed visual disturbance and other ocular changes, such as pigmentation of eye lids and cornea.

Analysis of teardrop porphyrins was performed in three patients, after obtaining informed consent. In normal controls, no porphyrin isomer was observed, whereas in all patients remarkable elevation of porphyrins, especially of uroporphyrin I and coproporphyrin I, were observed. Interestingly, in case 4, the accumulation of porphyrins was relatively mild compared with other patients.

Comment

Sclerotic changes at the body surface lesions in CEP are mainly caused by the accumulation of porphyrins. In some patients, remarkable elevation of porphyrins, especially of uroporphyrin I and coproporphyrin I, were observed. Interestingly, in case 4, the accumulation of porphyrins was relatively mild compared with other patients.

Table 1 Tear drop analysis of porphyrins in Japanese patients with CEP.

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uroporphyrin</td>
<td>1302.6</td>
<td>1235.3</td>
<td>1345.9</td>
</tr>
<tr>
<td>Heptaporphyrin</td>
<td>49.1</td>
<td>302.5</td>
<td>141.5</td>
</tr>
<tr>
<td>Hexaporphyrin</td>
<td>27.4</td>
<td>0</td>
<td>35.8</td>
</tr>
<tr>
<td>Pentaporphyrin</td>
<td>24.6</td>
<td>67.9</td>
<td>109.5</td>
</tr>
<tr>
<td>Coproporphyrin I</td>
<td>108.1</td>
<td>348.8</td>
<td>285.9</td>
</tr>
<tr>
<td>Coproporphyrin II</td>
<td>11.2</td>
<td>32.5</td>
<td>28.6</td>
</tr>
<tr>
<td>Protoporphyrin</td>
<td>1363.3</td>
<td>59.9</td>
<td>59.2</td>
</tr>
</tbody>
</table>

Figure 1 Scleral involvement shown in case 2.

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References


Uveitis initiating an autoimmune reaction resulting in Goodpasture’s syndrome in a Chinese man

Goodpasture’s syndrome is an autoimmune disease caused by IgG directed against the alpha-3 chain of type IV collagen found in basement membrane. It causes pulmonary haemorrhage and renal failure. The antibody has been demonstrated in the basement membranes in the eye. However, ophthalmological complications in Goodpasture’s syndrome are rare. As with other autoimmune diseases, Goodpasture’s syndrome patients have a strong susceptibility based on a genetic background. Much evidence supports the concept that cross reactivity with exogenous epitopes or insult to the basement membrane can initiate the process of autoimmunity resulting in Goodpasture’s syndrome. We present a case of a Chinese man in whom we believe uveitis precipitated the autoimmune reaction causing Goodpasture’s syndrome.

Case report

A 77 year old Chinese man from Hong Kong presented to the eye casualty department...
with a 1 month history of a black patch in the centre of his visual field in the left eye. Before this he noticed floaters and a “black cloud” in the left eye. He did not complain of pain or photophobia.

Three months earlier he had an episode of acute anterior uveitis, which resolved with dexamethasone and cyclopleptonate drops. His vision at that episode was 6/24 in the right eye and 6/12 in the left eye. Six months earlier he had developed septicemia secondary to a urinary tract infection treated with a course of intravenous flucloxacillin and fusidic acid and he made a good recovery.

Two months prior to his admission, he came to the United Kingdom and his general practitioner noted that his vision was reduced in the left eye. He noticed floaters and a “black cloud” in his central visual field in the left eye and started to have problems with his dizziness and nausea. There was no temperature or vomiting.

A blood test revealed that he was in acute renal failure with sodium of 123, potassium 6.9, urea 56.9, and creatinine 1681. ESr was 120, FBC 8.0, WCC 10.2, and platelets of 610. Liver function tests were normal apart from albumin of 21. He underwent haemodialysis and a renal biopsy showed Goodpasture’s disease (Fig 2). His glomerular basement membrane antibodies (ELISA confirmed by western blot) were also positive with a level of 77% (0–15 reference range), as were his PEG immune complexes. ANA, ANCA, and anti-DNA were all negative. He was given high dose steroids and had a course of plasma exchange (three 3 litre volume exchanges with FFP/PPF replacement).

His eyes continued to improve during the following month and the inflammation settled. At last review his visual acuity was 6/9 right eye and 6/12 left eye. Unfortunately, he developed haemoptysis and overwhelming septicemia and died 2 months later. A post mortem was declined.

Comment

In 1919 Ernest Goodpasture described a patient with haemoptysis, anaemia, and proteinuria and a post mortem showing pulmonary alveolar haemorrhage and proliferative glomerulonephritis. Goodpasture’s disease is a rare autoimmune organ specific disease in which IgG antibodies are directed against the NCl domain of the alpha-3 chain of type IV collagen leading to pulmonary haemorrhage, glomerulonephritis, and renal failure. Pasture’s syndrome, however, describes Goodpasture’s disease with either haemoptysis or crescentic glomerulonephritis, or both. Clearly, this patient had the complete syndrome. Thirty per cent of patients are ANCA positive and lung or kidney biopsy shows linear immunofluorescent staining for IgG along the basement membrane. The death rate has drastically improved from 75% to 25% with the advent of plasmapheresis combined with immunosuppressants. However, it is rare to escape irreversible dialysis dependency unless the disease is detected and anti-GBM antibody levels are reduced before oliguria has ensued.

Immunofluorescent and immunohistochemistry studies show that the deposition of the antibasement membrane antibodies can also occur in the eye in the basement membrane of the choroidal vessels, ciliary body, lens capsule, and Bruch’s membrane. The pathogenic significance of this is still unknown. Furthermore, there have not been any studies confirming the actual presence of type IV collagen or of the alpha-3 epitope in the eye, although it is widely assumed that they are both present.

Documented ophthalmological abnormalities in Goodpasture’s include retinal haemorrhages and exudates and serous retinal detachment. It is possible that these signs may reflect hypertension or ANCA positivity rather than direct damage from antiglomerular basement membrane antibodies possibly because of the small percentage of alpha-3 chain in its NCl domain. If the disease is so florid in the kidneys and the lung, one would expect a disease of similar severity in the eye if the antibasement membrane antibodies were pathogenic in the eye.

This man had a granulomatous panuveitis with choroidal infiltrates. These ophthalmic features have not previously been described in Goodpasture’s syndrome. The serum from his initial admission with uveitis in February, 2 months after his episode of septicemia, was normal apart from raised ESR and CRP levels and his antiglomerular basement membrane antibodies were 9%, which is within normal limits. Four weeks later in March, the titre of antibodies had risen to a grossly abnormal level of 77%. The temporal relation of developing antiglomerular basement membrane antibodies and disease means it is likely that these complexes occurred in response to his septicemia and produced a reactive uveitis. As type IV collagen is also found in the choroidal plexus, the uveitis may have exposed type IV collagen to the immune system causing anti-GBM antibody production which results in cross reactivity with the glomerular basement membrane in the kidney to produce Goodpasture’s syndrome.

As with other autoimmune diseases, Goodpasture’s syndrome patients have a strong susceptibility based on a genetic background. Much evidence supports the concept that insult to the basement membrane or cross reactivity with exogenous
Fungal keratitis is a rare complication of pho-
toablative laser surgery for myopia.

Fusarium solani

**References**


**Fusarium solani keratitis following LASIK for myopia**

Fungal keratitis is a rare complication of pho-
toablative laser surgery for myopia.

Fusarium solani

**Case report**

A 45 year old woman noted blur and discom-
fort of the right eye 3 days after bilateral LASIK procedures. The visual acuity was 6/9–3 unaided and there was a 1.5 mm diameter infiltrate beneath the flap (Fig 1A). The left eye was unaffected with an unaided visual acuity of 6/6. Cultures were not taken but her topical antibiotic was changed from chloramphenicol to ofloxacin, and dexam-
ethasone 0.1% four times daily was contin-
ued. After a further 4 days the vision had reduced to 6/34 and there was ulecreration through the flap over the infiltrate; treatment was changed to hourly gentamycin 1.5%, cefuroxime 5%, and econazole 1%, and dexam-
ethasone was continued to reduce the risk of further flap melt. A culture from the ulcer-
ated corneal surface over the infiltrate was negative. There was continued deterioration and treatment was therefore stopped for 24 hours before the flap was lifted and cultures taken for routine bacteriology, acid fast bacilli (Mycobacterium), and fungi. No organism was identified. Because of the uncertain diagnosis a loading oral dose of fluconazole 200 mg was given followed by 100 mg daily, and a 7 day course of intensive topical vancomycin (5%) and amikacin (2.5%) was started with oral clarithromycin 500 mg twice daily. Confocal microscopy of the edge of the lesion demon-
strated filamentary structures in the deep stroma (Fig 1B), but a subsequent tissue biopsy of the flap and the deeper stroma was again negative. Despite intensive treatment with topical amphotericin (0.15%) and na-
tamycin (5%), and courses of oral itraconazole (400 mg daily) and voriconazole (400 mg daily) there was continued deterioration and the cornea perforated (Fig 1C). On the 32nd day following LASIK a 9 mm penetrating cor-
neal graft was performed and the anterior chamber was irrigated with amphotericin (5 µg in 0.1 ml). Dexamethasone 0.1% four times daily was continued after surgery to treat severe intraocular inflammation. Histology of the excised corneal button showed filamen-
tary fungal elements anterior to Descemet’s membrane but no evidence of hyphae extend-
ing to the margin of the excised tissue. *Fusarium solani* was subsequently grown from the corneal button and the isolate was reported sensitive in vitro to amphotericin (1.0 µg/ml), partially sensitive to clotrimazole (8 µg/ml), econazole (4 mg/ml), and miconazole (8 µg/ml) and resistant to itraconazole (>16 µg/ml) and fluconazole (>16 µg/ml). Despite continued topical and oral antifungal treatment, two further penetrating grafts were necessary to remove recurrences at the graft interface. Following the last graft topical ster-
od was supplemented with topical cyclosporin A 2% four times daily for 4 weeks, after which fluoromethalone 0.1% four times daily was started to control inflammation and prevent vascularisation. All topical antifungal treat-
ment was stopped after 3 months. The final vision at 8 months was reduced to 6/60 as a result of irregular corneal astigmatism, cata-
ract, and graft oedema.

**Comment**

Fungal keratitis is rare in the United Kingdom but accounts for 17–37% of microbial keratitis isolates in warmer countries. To date all reported cases of fungal keratitis after LASIK have been due to filamentary organisms. The diagnosis may be difficult to confirm as cultures and biopsies of the anterior stroma can be negative because of the tendency of filamentary fungi to proliferate in the poste-
rior corneal stroma. A delay in identification of the causative agent often contributes to the poor outcome. Of the four previous reported cases of presumed flap interface infection, excisional keratoplasty was required in three, and perforation and vascularisation of the corneal occurred in one. The only case with a medical cure had a surface infection of a persistent epithelial defect.

The origin of this infection is uncertain. The patient had visited Florida 2 months previ-
ously, where *Fusarium* sp is the most common isolate from cases of fungal keratitis. On the day of treatment the ambient temperature in London was 80°F, but subsequent air culture plates from the treatment area and from the air conditioning system were negative. Be-
cause of a reluctance to disturb the LASIK flap there is a temptation to treat infiltrates empirically with broad spectrum antibiotics and steroid, rather than lifting the flap to culture the lesion directly. Unfortunately, in this case, two cultures were negative, and the diagnosis of a filamentary fungal infection was suggested by confocal microscopy. A fur-
ther negative biopsy then delayed a decision for surgical intervention. Corticosteroid may

**Figure 1** (A) Infiltrate beneath the flap (arrow) on the third day following LASIK. (B) Confocal image showing branching filamentary structures (arrows) in the deep stroma adjacent to the area of infiltrate. (C) Advanced abscess before 9 mm penetrating keratoplasty 32 days after LASIK.
increase the virulence of pathogenic fungi and it is not known whether these fungi or trichothecenes are soil contaminants.

Acknowledgement
Mr John Dart provided helpful advice with the management of this case.

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References
Comment

Notwithstanding their metastatic potential, carcinoids are compatible with long term survival (as attested by our patient’s case). To our knowledge, the 43 year interval between diagnosis of the patient’s primary tumour and recognition of uveal metastasis is the longest interval reported to date. Low immunoreactivity to Ki-67 and non-reactivity to p53 may possibly explain the indolent course of this tumour and its metastasis many years after the resection of the original tumour. Ki-67 is a DNA binding nuclear protein that is expressed in proliferating cells but not in quiescent cells. It is regarded as a fractional measure of tissue proliferation and overall prognosis.

We thank Dr James D Sanchez, medical oncologist, with long term survival. Bronchial carcinoid led to the clinical impression of uveal melanoma. Consequently, this case suggests that carcinoid tumours may metastasise as much as four decades after removal of the primary tumour. The prognosis for metastatic uveal tumour is generally poor, unless early recognition and treatment can be achieved.

Although bilateral ocular involvement has been reported, this is the first case of recurrent uveal carcinoid metastasis. The patient’s metastatic lesion like those reported by Fabbro et al exhibited no staining for the Ki-67 and negative staining for the p53 tumour suppressor gene, is the most commonly mutated gene in uveal carcinoids, however, are compatible with long term survival. Tumour’s histological trabecular pattern and negative serotonin staining favour the bronchus as its site of origin. Although bilateral ocular involvement suggests the diagnosis of a metastatic process, the 43 year long interval since resection of the bronchial carcinoid led to the clinical impression of uveal melanoma. Consequently, this case suggests that carcinoid tumours can metastasise as much as four decades after removal of the primary tumour. The prognosis for metastatic uveal tumours is generally poor, unless early recognition and treatment can be achieved.

Conflicts of interest: None.

Acknowledgements

We thank Dr James D Sanchez, medical oncologist, Southwest Cancer Clinic, Las Vegas Nevada. Supported in part by grant no EY03040 from the National Institutes of Health and the Research to Prevent Blindness Inc, New York, NY, USA.

References


Figure 1  (A) Left eye, February 2001: clinical aspects of the first examination: blepharitis marginalis, thickened, knobby lid margin, and a rarefaction of the eyelashes. Descemetocoele without corneal perforation or intraocular inflammation. (B) Right eye, May 2001: conjunctival injection, intraocular inflammation, anterior and posterior synchiae, and ulceration of the cornea.
Visual acuity was 0.02 in the right eye and 0.7 in the left eye. The right eye showed 3+ conjunctival injection, 3+ anterior chamber cells, and ulceration of the cornea. A corneal perforation at the peripheral 6 o'clock position was closed by an incarceration of the iris. The anterior chamber was flat.

After intensive topical and intravenous antibiotic therapy and insertion of punctum plugs infection and inflammation slowly reduced. Visual acuity did not improve. The patient was discharged and was again controlled by her ophthalmologist.

**Comment**
We report a 74 year old woman patient with recurrent bilateral corneal ulceration and uniconjunctival injection probably associated with keratosis follicularis.

Keratosis follicularis (Darier-White disease) is caused by mutations in the ATP2A2 gene, which encodes the Ca\(^{2+}\) ATPase 2 isoform. This defect results in disturbed cell to cell adhesion and differentiation of the epithelium.\(^1\) Histopathological hallmarks of the disease are focal suprabasal clefting due to acantholysis, and subsequent dyskeratotic round epidermal cells (“corps ronds”) with overlying columns of parakeratosis.

In our patient the recurrent corneal ulcerations with subsequent perforation could be explained by the participation of the ectodermal ocular tissues in keratosis follicularis: the lacrimal gland, the eyelids, and the corneal epithelium. A combination with Sjögren’s syndrome, dry eyes, oral mucosal lesions, and involvement of the salivary gland is known from the literature.\(^2\) A histological examination of the eyelid specimen showed a characteristic epidermal involvement, but no involvement of the specific subepithelial glands of the lid margin. It was presumed that the knob-like margin could provoke symptoms and sequelae similar to seborrhoeic blepharitis.\(^3\) Recurrent localised or widespread cutaneous viral infections and secondary bacterial overgrowth is also common. Bacteria and fungi can colonise the keratotic debris.\(^4\)

In our patient keratosis follicularis probably resulted in dry eye syndrome with reduced corneal protection and microbiological colonisation of the skin and ocular surface. Impaired functionality of desmosomes of the corneal epithelium caused the recurrent corneal ulcerations with perforation. Therefore it can be concluded that the increased risk of oculocutaneous complications in keratosis follicularis may include severe corneal infection. Beside the long term dermatological medical care an intensive ophthalmic therapy with lubricants and antibiotics may help to avoid this ocular complication.

**References**


**MAILBOX**

**Uveal melanoma: Finland v India**

We have read with great interest the article by Eskelinen and Kivelä.\(^4\) This study provides information as to how uveal melanoma can be diagnosed and treated early if fundus examination is done by an ophthalmologist and also emphasises the importance of indirect ophthalmoscopy should be done even in asymptomatic patients having an ophthalmic check up. We feel that indirect ophthalmoscopic evaluation of the dilated fundus is essential for identification of malignant uveal melanoma. This method of examination offers the best chance of detecting early retinal pathology and also allows follow up of progressive benign lesions suspected of potential malignancy.

In the Finnish population Eskelinen and Kivelä have found 184 uveal melanoma cases in 5 years. Interestingly, in a study done by us on the Asian Indian population at the ocular oncology department of a tertiary eye care institute,\(^5\) we found only 73 cases of uveal melanoma in 12 years (0.02% incidence). The mean age of presentation in the Finnish population was found to be 60 years (range 14–87) and in the British 59.7 years.\(^6\) However, the mean age at presentation of uveal melanoma in the Asian Indian population was found to be 46.1 (range 13–75) years in the 71 cases studied by us. Similar low mean age of presentation (43.7 years) has been reported earlier in the Chinese population.\(^7\)

In addition, there may be a difference between the Finnish and Asian Indian population in the mean basal diameter of the uveal melanoma at the time of presentation. In the Asian Indian population, the mean basal diameter of the tumour was 12.44 (SD 4.41) mm compared to 11.3 and 6.4 mm in the Finnish population and 11.6 mm and 4.9 mm in the British population. We feel that the Asian Indians present at an earlier age with large tumours in comparison with the Western population.\(^8\) This comparison re-emphasises the racial differences in presentation of uveal melanoma. Whether this has any bearing on the prognosis and the implication of early onset melanoma in pigmented populations remains to be seen.

**References**


A perspective on bovine pericardium for orbital implants

We have read with avid interest the article by Gupta et al on the use of bovine pericardium as a wrapping material for hydroxyapatite orbital implants.\(^1\) We are highly impressed by their results as none of the patients had implant extrusion. This is really commendable since implant extrusion rates have ranged from 9% to 21% in previous studies,\(^2\) and it indicates meticulous technique and follow up. We would like to clarify a few pertinent issues.

Although bovine pericardium is generally considered non-reactive, it has been reported to produce severe inflammation in cases of cardiac use.\(^3\) Another area of major concern with cardiovascular use of bovine pericardium has been the development of calcification seen in both laboratory studies and cardiac patients.\(^4\) It is still uncertain what impact such a calcification would have on an orbital implant and assessment of its effect would certainly require studies with a larger number of patients and longer follow up. However, it has been suggested that this is correlated with its motility.\(^5\) We suggest that the possibility of misinterpretation of imaging findings in cases of orbital recurrence of tumours should not be overlooked.

Another crucial area of concern is the risk of infection with xenografts, which cannot be totally eliminated even by highly stringent screening and processing procedures. We are referring to the group of bovine spongiform encephalopathies including Creutzfeldt-Jakob disease (CJD) and its variant found in the United Kingdom (vCJD).

Therefore, the quest for a comparable synthetic wrapping material and better implants which do not require wrapping continues, and bovine pericardium should be considered, bearing in mind its above mentioned shortcomings.

**References**

TTT for occult CNV: check the power!

Transpupillary thermotherapy (TTT) was originally introduced for the treatment of small choroidal melanomas. Although the precise mechanism of action for this treatment is unknown yet, the reported data appear to be beneficial compared to the natural course of the disease.

Currently, the following treatment parameters are recommended to treat occult choroidal neovascularisation (CNV) with TTT:
- one exposure, 60 second exposure time, 800 mW power for the 3 mm spot, 530 mW for the 2 mm spot, and 320 mW for the 1.2 mm spot.
- The output is automatically adjusted via the Goldmann fundus lens to the power per field is calculated to 247 mW/mm.

In order to ensure that the applied power is not inadvertently lower than 800 mW for the 3 mm spot (or the power equivalent for other spot sizes), the treating physician would not notice he has to compensate for the lower power by adjusting the instrument's power level.

We have used a diode laser (Iridex Corporation, Mountain View, CA, USA) with the recommended fiberoptic adapter since 1997 for the treatment of choroidal melanomas and haemangiomas without any apparent problems.

Lacking any possible explanation other than that the treatment method didn’t work, we finally asked a technician to check the laser system and immediately he noticed visible defects at the adapter of the fiberoptic resulting in a measurable reduction of power output from the 800 mW on the display to 560 mW at the end of exposure. Thus, if the applied power would be inadvertently lower than 800 mW at the end of exposure, this remains unexplained.

The panel of judges made a unanimous decision to award the $25,000 prize in recognition of Sight Savers’ support for the provision of vital eye care services in some of the poorest communities around the world over the last 50 years.

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 on the charity website (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@yahoo.com).

**Sight Savers International Honoured by American Medical Association Award**

Sight Savers International, the UK’s leading charity tackling blindness in the developing world, is the first non-US organisation to receive this prestigious award.

The charity works with partner organisations in poor and under served communities to develop and support healthcare programmes that prevent and cure blindness.

**Technology for Vision 2000**

The latest issue of *Community Eye Health* (No 42) focuses on technology and training, with an editorial by Catherine Cross, chairperson of the International Agency for the Prevention of Blindness (IAPB). For further information please contact: Journal of Community Eye Health, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; fax: +44 (0)20 7230 5207; email: eyeresource@ucl.ac.uk; website: www.jcem.co.uk). Annual subscription (4 issues) UK£23, US$40. 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 Medicines*, *Equipment, Instruments and Optical 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).
BEAVRS Meeting
The next BEAVRS meeting will be held in the Dalmahoy Hotel near Edinburgh on 31 October to 1 November 2002. Further details: Susan Campbell, Medical Secretary, Gartnavel General Hospital (email: susan.j.campbell.wg@northglasgow.scot.nhs.uk).

Cornea 2002—Celebrating 50 Years of Eyebanking
The Cornea 2002 meeting will be held in Le Meridien Hotel, London, Gatwick on 14–15 November 2002. Subjects to be covered will include eye banking, penetrating and lamellar keratoplasty, stem cell restoration, keratoprosthesis, advanced keratoplasty techniques, paediatric cornea, keratorefractive surgery, and intraocular refractive surgery. Spaces are limited and a beneficial package rate is available prior to 30 September 2002. Further details: CORNEA 2002 organiser at the Corneo Plastic Unit, The Queen Victoria Hospital, Holtye Road, East Grinstead, West Sussex, RH19 3DZ, UK (tel: 01342 410 210 ext 560; fax: 01342 317 181; email: Cornea2002@hotmail.com).

Introductory Course in Osteo-odonto-keratoprosthesis (OOKP)
The University of Brighton Postgraduate Medical School is holding an introductory course in osteo-odontokeratoprosthesis (OOKP) 20–21 November 2002 in the New Seminar Room, Sussex House, Brighton & Sussex University Hospitals Trust in Brighton. The course will comprise of a variety of lectures with live surgery, two way audio and video links (Stage 1 and Stage 2 OOKP surgery), and examination of patients. Further details: Mrs Erica Strange, University of Brighton, Postgraduate Medical School, Falmer Campus, Brighton, East Sussex BN1 9PH, UK (tel: +44 (0)1273 644 005; fax: +44 (0)1273 644 002; email: e.strange@brighton.ac.uk).

23rd Annual Conference and Dinner Glaucoma Society (UK & EIRE)
The 23rd Annual Conference and Dinner of the Glaucoma Society will be held on Thursday 21 November 2002, 8.30am to 5.00pm at the Central Conference Centre, London. The Annual Dinner is from 6.30pm to 10.00pm at The Royal College of Surgeons, London. Conference charges: £60 members; £80 non-members. Price entitles delegates to refreshments, lunch, abstract book, programme, and annual dinner. (Maximum number of places 250—apply now to secure your place). Further details: Janet Flowers, Administrator, 29 Quarry Hill, Grays, Essex, RM17 5BT, UK (tel/fax: 01375 383172; email: glausoc@ukeire.freeserve.co.uk).