LETTERS TO THE EDITOR

Orbital mass in a patient with leukaemia

EDITOR,—The detection of a mucosa associated lymphoid tissue lymphoma in the orbit should motivate the ophthalmologist to a comprehensive systemic evaluation, given a significant association of this tumour with extranodal disease. We present the case of an orbital mass in a patient with leukaemia. Following excision and histopathological studies of the tumour, a diagnosis of MALT lymphoma was made, which led to the prompt evaluation and detection of extensive multiorgan involvement, and life saving therapy.

CASE REPORT

A 60-year-old man with a history of chronic lymphocytic leukaemia presented with a slowly growing mass in the right inferior orbit over the past year. Examination revealed a smooth, non-tender mass near the inferior orbital rim, distinct from the globe. Computed tomography showed a homogeneous tumour with smooth contours and no bony erosion (Fig 1). An excisional biopsy by anterior orbitotomy was subsequently performed. Histopathology revealed marginal zone cells and diffuse infiltration by plasma cells and plasmacytoid lymphocytes in a characteristic nodular cellular pattern, consistent with MALT lymphoma (Fig 2). Immunohistopathology showed the cells to be 60% B cell, and 40% T cell in origin. The patient was therefore promptly referred for colonoscopy, which disclosed multiple tumours, all consistent with MALT lymphoma. Two months later, the patient developed several cutaneous lesions on his arms, which again were MALT-type lymphoma by biopsy. He subsequently began chemotherapy and orbital radiotherapy, and has been doing well.

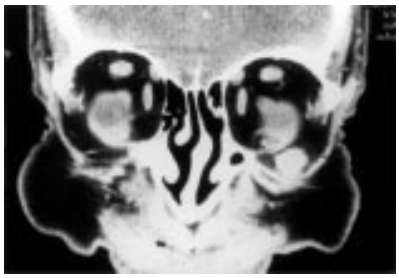

Figure 1 Coronal computed tomography scan demonstrating left periorbital mass.

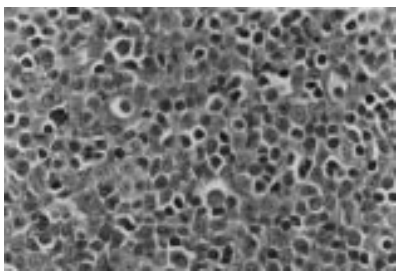

Figure 2 Photomicrograph of plasmacytoid and small lymphocytes characteristic of low grade B cell lymphoma of the MALT type. Haematoxylin and eosin, ×20.

COMMENT

The mucosa associated lymphoid tissue lymphomas comprise part of a group of low grade B cell lymphomas presenting in the gut and other glandular epithelial tissues. These lymphomas differ from usual non-Hodgkin's lymphomas in that they contain a variety of neoplastic B cells, rather than a monomorphous cell population. Histopathologically, these lymphomas contain small lymphocytes, plasma cells, and centrocyte-like cells, often in a reactive follicular pattern as shown here.[1] MALT lymphomas may occur in a variety of extranodal sites, including breast, skin, kidney, and prostate.[2] The common denominator of all these extranodal sites appears to be the glandular epithelium, for which these lymphomatous cells have a particular affinity.

Ocular adnexal MALT lymphomas are usually primary orbital tumours that typically carry a relatively small risk of mortality. They may, however, be associated with subsequent extraorbital disease involving other mucosal sites, as demonstrated here. The estimated time interval from presentation of an orbital mass to the development of an extraorbital lymphoma varies from 6 months to 5 years.[3 4] Furthermore, an estimated 25% of patients presenting with primary orbital lymphoma will develop extranodal disease during a 1 year follow up.[3] This relatively long interval requires careful long term evaluation including serology, bone marrow biopsy, as well as chest and abdominal computed tomographies.

The often salient presentations of orbital masses necessitates serious evaluation. The detection of an orbital mucosa associated lymphoid tissue lymphoma, in particular, may be associated with systemic mucosal disease, and should direct the physician to the appropriate systemic studies

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A previously unrecognised side effect of dapsone

EDITOR,—Dapsone (diaminophenyl sulphone) has been in use for many years for the treatment of ocular cicatricial pemphigoid (OCP) as an anti-inflammatory agent. It was found to be the most effective initial agent for active or acute OCP and a safer treatment in the elderly than steroids and immunosuppressants.[1 2] We report a previously unreported side effect of dapsone consisting of a taste disturbance and tingling sensation in the mouth and lips. This continued in the patient for about a year after commencing dapsone for acute pemphigoid and resolved following cessation of the medication.

CASE REPORT

A 70-year-old man presented to the eye casualty department with a 1 week history of sticky, red, and irritable eyes. He had no previous eye problems. His medical history included angina and myocardial infarction in 1984 and two cerebrovascular accidents (1986, 1987) with full recovery. He was taking frusemide 40 mg daily, isosorbide mononitrate 10 mg four times daily, Slow K 400 mg three times daily, aspirin 300 mg daily, and ranitidine150 mg daily. On examination, conjunctival inflammation and ulceration, symblepharons, mouth ulcers, and cutaneous blisters on the hands and face were noted. A diagnosis of acute bullous and mucous membrane pemphigoid was suspected and confirmed following dermatological consultation and skin biopsy. The patient was commenced on 80 mg daily of oral prednisolone with rapid resolution of the acute lesions. Dapsone was introduced 1 month later in a dose of 50 mg daily. The dosage of prednisolone was tapered gradually and dapsone was increased to 100 mg daily. Six weeks later the patient complained of a sickening sweet taste. The pemphigoid was completely quiet so dapsone was reduced to 50 mg daily. One year after commencing dapsone he was still in remission but complaining bitterly that everything tasted sweet with a bad taste in the mouth when getting up in morning and tingling of the face and lips. Dapsone was stopped and when he was reviewed 1 month later, all symptoms of altered taste sensation had disappeared. His eyes remained quiet using only Viscotears drops.

COMMENT

The main pathophysiological mechanism in pemphigoid is thought to be the formation of antibasement membrane antibodies which lead to subepithelial blistering, granulation tissue, and inflammatory infiltrate formation in the substantia propria. The infiltrates consist mainly of polymorphonuclear leucocytes (PMN) in the acute phase and also lymphocytes and plasma cells. Healing occurs by progressive fibrosis and shrinkage.[3 4] The exact mode of action of dapsone in OCP is unknown but it appears to inhibit the migration of PMN by inhibiting lysosomal enzyme activity, interfering with the leucocyte cytotoxic system or preventing the cells from responding to chemotactic stimuli.[5] A dose of 100 mg daily is usually required to control the disease in the acute phase while in the chronic phase a smaller maintenance dose of 50 mg daily or every alternate day is sufficient.[5] The drug is recirculated in the liver and excreted in saliva. Side effects include haemolysis, neutropenia, agranulocytosis,[6] methaemoglobinaemia, peripheral neuropathies, tremors, head-

aches, insomnia, anorexia, nausea and vomiting, ulcerative stomatitis, allergic dermatitis (Stevens–Johnson syndrome), depression, confusion, fatigue, raised erythrocyte sedimentation rate, pyrexia, rigors, hepatitis, and hypoalbuminaemia.[7] To our knowledge, altered taste sensation (sweet taste) is not a known side effect of dapsone. No other cases have been reported to the Committee on Safety of Medicines over a period of more than 30 years (Committee on Safety of Medicines, personal communication).

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A patient with long standing melanin laden macrophages in cerebrospinal fluid in Vogt–Koyanagi–Harada syndrome

EDITOR,—The cause of Vogt–Koyanagi–Harada syndrome (VKH) is suspected to be systemic immunological reactions in various organs containing melanocytes.[1–5] It has been suggested that the cell mediated immune process involving melanocytes plays an important role in the pathogenesis of VKH.[1–5] Supporting this idea, we previously reported the existence of melanin laden macrophages (MLMs) in the cerebrospinal fluid (CSF) of VKH patients.[6] In clinical practice, as in our present case, detecting MLMs in CSF provides useful information on the activity of the patient's systemic immunological reactions.

CASE REPORT

A 60 year old woman visited our hospital with blurred vision, tinnitus, and headache. Our first examination revealed that her best corrected visual acuity was 0.02 in the right eye and 0.01 in the left. Slit-lamp examination disclosed cellular infiltration in the anterior chamber and vitreous. Ophthalmoscopy showed serous retinal detachment with choroidal detachment in both eyes. Fluorescein angiography showed multifocal hyperfluorescent spots and diffuse subretinal pooling (Fig 1). CSF examination revealed pleocytosis (cell counts 273×10^6/l) and a large number of MLMs (Fig 2). Following the diagnosis of VKH, we started the patient on intravenous prednisolone succinate 200 mg daily, gradually tapering the dose. Three months after the initial administration of corticosteroid, visual acuity recovered and the main clinical manifestations almost disappeared. At that time cell counts in the CSF had decreased (cell count 13×10^6/l) to within normal range, but MLMs were still present.

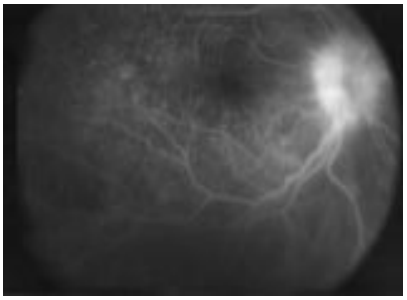

Figure 1 Fluorescein angiogram of the patient's right eye. Serous retinal detachment is noted at posterior pole with multiple punctate hyperfluorescence. Active leakage is also seen at the optic nerve head. Visibility is not good because of diffuse vitreous infiltration.

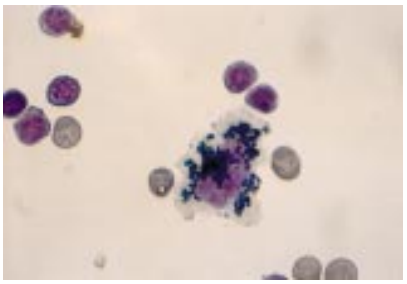

Figure 2 Morphological characteristics of a melanin laden macrophage (May–Grünwald staining, original magnification × 400) in CSF from the patient obtained 2 weeks after the initial corticosteroid therapy was started. Melanin pigments are identified as basophilic granules in the cytoplasm of a macrophage.

The dosage of corticosteroid was then decreased to 15 mg daily because no sign of recurrence was seen. After that, the patient developed fungal pneumonitis, which was successfully treated with antifungal agents for 3 weeks. Four months after the first attack, while MLMs did not disappear, she complained of blurred vision again. At that time her best corrected visual acuity decreased to 0.02 in the right eye and 0.01 in the left. Inflammation in the anterior chamber and serous retinal detachment recurred in both eyes.

We restarted the patient on corticosteroid therapy, using the same protocol as before. The main clinical manifestations disappeared in a month. The MLMs in the CSF finally disappeared 2 months after the second attack. Since then no recurrence has been noted even after corticosteroid therapy was reduced.

COMMENT

VKH is thought to be an autoimmune disease involving systemic melanocytes.[1 5] Pleocytosis in VKH is considered a sign of the focal immune response against melanocytes in meninges.[2 3] Although lymphocytes are predominantly observed in the CSF and uvea from patients with VKH,[2 3] a small number of macrophages are also detected.[4]

In our previous report,[6] we found melanin granules in the cytoplasm of macrophages in CFS obtained from VKH patients. In those patients MLMs were detected only in the early stage of the clinical course, and they disappeared after initial treatment. There was no recurrence of inflammation in those patients. In contrast, our present case showed the presence of MLMs for a long time, even though other clinical features, such as pleocytosis, had disappeared. It should be noted that there was a recurrence of VKH during the period when MLMs were detected in CSF and that no recurrence has been observed since the disappearance of MLMs in CSF. We suspect that the immune reaction against melanocytes is still present as long as MLMs are found, even though other clinical features were normal.

In clinical practice it is difficult to determine how to taper corticosteroid therapy to prevent recurrence. Detecting MLMs in a clinical course may indicate an immune reaction and also the prognosis of a patient.

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CORRESPONDENCE

Monitoring and evaluating cataract intervention in India

EDITOR,—I read the article by Limberg *et al* [1] with interest. I am reminded of 'Confusion of goals and perfection of means characterise the age' by Albert Einstein. It is a well tried attempt to infuse quality initiatives in a blindness control programme. However, it is evident that the Indian experience is no different from that of many other organisations in designing, monitoring, and evaluating process indicators.[2] I do have some serious concerns about possible (ill) use of the indicator sight restoration rate (SRR) as a variable be to included in a mathematical model for assessing the impact of interventional strategies. A study under the aegis of reputed agencies assumes tremendous prestige and conclusions become a weighty verdict. The potential to provide serious misinformation leading to decisions in slowing down the programme is of interest.

(1) It is difficult to understand the rationale of operating in one eye only in a bilaterally blind person. Probably the authors chose to ignore that by denying the operation would make one permanently blind due to complications of hypermature cataract.

(2) I sincerely feel that the indicator SRR is not suitable to monitor qualitative aspects of the programme. It would be evident that the overall sight restoration is not alarmingly low when the first two groups (6/6–6/18 and 6/18–6/60) are taken into consideration. It would have been better if the groups were considered individually rather than treating them as separate groups preoperatively and combining them at the postoperative stage (Table 6). In this way SRR improves dramatically. Suitably modified instruments like ADVS and SF-36[3] are appropriate to measure vision related quality.

(3) There are 3000 non-operating ophthalmologists without any surgical facilities. No measures have been suggested to include them in the programme. The target of 700/OS/year seems unattainable with the actual figure at 440/OS/year. Any increase above the present actual numbers might dilute the quality of cataract surgery ('Focus presently is on achieving the targets than focusing on prevention of blindness'). It is well established that any targets, let alone increasing the present levels of targets, are detrimental to the programme objectives altogether.

(4) 1.32 to 2.1 million cases (at 440 cases/surgeon/year to 700 cases/surgeon/year respectively by 3000 non-operating ophthalmic surgeons) can be operated by induction of 3000 non-operating surgeons, with available resources like staff, facilities, and supplies of material, which the authors claim are going unused and with the number of cases increasing through demand generation and case finding. Their suggestion to encourage ophthalmologists to work in areas of low cataract surgery utilisation is wishful thinking. In a health sector where primary health centres remain unmanned for years, the suggestion is impracticable. A model with paradigm shift towards optimum utilisation of available resources efficiently and effectively is needed. The 'Arvind eye hospital model'[4] with suitable modifications to suit the different geographic locations is one of the alternatives.

(5) The suggestion of selecting better cases is like 'improving the indicator rather than the programme objectives and performance'. This is in contravention of the objectives given in the document prepared by DGHS, Government of India.[5] An effectiveness indicator is valid only when it truly serves as a measure of goal achievement.[6] By careful selection of 'proper' cases, there is a possibility of denying a chance of restoring whatever vision possible with cataract surgery. Hence, selection criteria are not in tune with the doctrine of equity and justice inherent in any national health programmes. The policy document of the World Bank assisted cataract blindness control project (1.4.7[5]) laying down quality related guidelines is not a justification to link funding of cataract surgery to NGOs and private surgeons with sight restoration rate.

(6) In programme implementation, in developing countries with limited resources, the question of equity and justice matter more than quality which is being experimented on with limited success in some countries. No doubt effectiveness is as important as efficiency. But when it comes to quantifying effectiveness on arbitrary rates, caution should be exercised before proper survey research methodology is adopted.

(7) The projected population figure of 200 million in +50 year population by 2011 is higher by 20 million as 5 year groups were not considered individually in the calculation. Ideally, 1 year grouping is preferred to project future population, as artificially high projected population figures necessitate huge resource allocations in planning the programmes (at present both 1 year break up and 5 year break up figures are available[7]). This would be unrealistic in a country with limited resources.

I hope the readers and policy makers bear in mind the above observations while considering diverse aspects of the National Programme for Control of Blindness.

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Reply

EDITOR,—I am grateful to Dr Seshubabu for the opportunity to expand a bit more on some of the operational concepts that might not have been explained well enough in the context of the article.[1]

(1) From a public health point of view, the main problem of blindness due to cataract is the disability. This can be corrected by a successful operation of one eye. Operating on the second eye may increase quality of sight, but does not reduce blindness. Ideally, both eyes should be operated, but since resources are limited, the challenge is to spread out the same resources among a larger number of individuals and to restore sight in as many people as possible. The author of the letter himself states that the questions of equity and justice matter more than quality in developing countries with limited resources. In India, it is estimated that half of all people blind from cataract die without ever being operated.

(2) The use of the indicator sight restoration rate (SRR) has been termed 'unsuitable'. In blindness control programmes, the most crucial question to assess the impact is: how many blind people had their sight restored as a result of the intervention? Most programmes cannot answer that question. The total number of cataract operations performed is known, but that reflects the workload, not the impact. The SRR is an attempt to measure the percentage of all cataract operations performed that resulted in changing a blind person into a sighted person. It measures the quality, efficiency, of the intervention programme. It is not designed to measure quality of vision in the individual. The SRR has to be used in combination with the other variables to monitor changes over a period of time, not as a one time assessment based on one observation only. Surgeries on cataract patients with VA better than 6/60, to prevent them getting blind, are captured in the other indicators used, such as the cataract surgical rate.

(3) In India, two groups of non-operating ophthalmologists exist. One group are general duty medical officers—doctors posted in a position of a general doctor. Specialists accept such posts since there is a surplus of specialists and a shortage of generalists in the government health services. The other group are ophthalmologists in the private sector who prefer to do medical ophthalmology only, or who are unable to invest in a hospital set up. It is a well accepted strategy to involve these ophthalmologists. However, the experience with refresher courses for such non-operating ophthalmologists so far has been poor. The norm of 700 operations per year per eye surgeon is set by the Ministry of Health.[2] Many surgeons in the government, NGO, and private sector achieve this already. In all these situations, efficient management is a key factor.

(4) A scheme of subsidies, to assist NGOs and private eye surgeons to establish eye clinics in underserved areas, has already been introduced and many applications were received.

(5) Besides the demographic changes, case selection is the key to understanding why cataract blindness is increasing despite more operations being performed. Whether we like it or not, there is already a lot of 'case selection' in eye care, and certainly not only in India. With the shift to high technology during the past decade, patient charges have increased and ophthalmologists have been targeting the wealthier urban population in order to pay back their investments. At the same time, basic cataract surgical services in the rural areas are reduced. Surgical camps, mainly practising ICCE + aphakic spectacles, are replaced by screening camps, where cataract cases are diagnosed and subsequently transported to base hospitals. These screening camps do not reach the more remote rural areas, since that would increase the transport costs too much. So these are held repeatedly

in accessible places only. This is visible in the pattern of surgeries: 5 years ago 75% of the surgeries was in first eyes and 25% in second eyes; at present, 40% are in first eyes and 60% in second eyes. What we see is an increased coverage of accessible areas and an erosion of services in more remote areas. What the authors suggest is a programme of active case finding of bilateral cataract blind people all over India, including the remote places. The number of bilateral cataract blind people in India is increasing rapidly,[2] mainly in the rural areas where eye care facilities are limited. Active case finding of patients who were operated earlier, and whose addresses are available, may assume less priority.

(6) See (1).

(7) Prevalence of blindness data are available for 5 year age groups only, therefore I used the population projections for 5 year age groups.

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Effect of latanoprost on intraocular pressure in patients with glaucoma on maximal tolerated medical treatment

EDITOR,—Latanoprost 0.005% is a prostaglandin analogue that causes reduction of the intraocular pressure (IOP) by increasing the uveoscleral outflow of aqueous humour through the ciliary muscle region to the suprachoroidal space and the episcleral veins.[1 2] Initial studies on latanoprost demonstrated reductions of 20% to 40% in the IOP of normotensive volunteers, patients with ocular hypertension, and glaucoma patients.[3–10] The efficacy of latanoprost 0.005% in lowering the intraocular pressure (IOP) in different glaucoma populations, especially when added to anti-glaucoma medications already being used, however, is not well established. The effectiveness of latanoprost in lowering the IOP in glaucomatous eyes with uncontrolled IOP that were already receiving maximal tolerated medical treatment was evaluated.

All patients who used latanoprost as compassionate treatment at Wills Eye Hospital/Jefferson Medical College in 1995 and 1996 and had at least 1 month of follow up were evaluated (n = 24). In patients treated with latanoprost in both eyes, only the right eye was chosen for this study. Criteria for success were defined before data collection. Complete success was defined as a reduction of the IOP >20%; relative success was defined as any reduction of the IOP. Kaplan–Meier analysis and Student's *t* test were used for data analysis.

Twenty four patients were evaluated with a mean age of 77.9 (SD 9.3) years. There were 14 (58.3%) females and 10 (41.7%) males, most of them being (n=19, 79.1%) white. The most frequent diagnosis (n=17, 70.8%) was primary open angle glaucoma (POAG). Most patients (n=18, 75.0%) were using two or more anti-glaucoma medications. Baseline IOP (21.4 (SD 5.6) mm Hg) was reduced after 1 and 3 months to 16.5 (3.8) mm Hg ($p<0.001$) and 16.4 (3.7) mm Hg ($p<0.001$), respectively. The probabilities of complete and relative success at 3 months' follow up were 26.7% and 87.0%, respectively (Figs 1 and 2). Latanoprost was discontinued in three patients (12.5%) because of problems that appeared after the treatment was started—bronchitis, throat irritation, and myocardial infarction. The first two cases may have been related to local irritation from the medication.

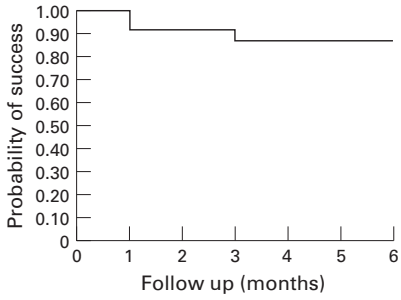

Figure 1 Life table analysis. Probability of relative success (any lowering of IOP after addition of latanoprost).

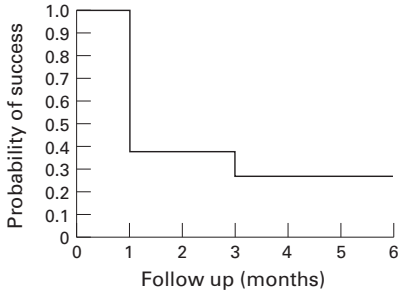

Figure 2 Life table analysis. Probability of complete success (reduction of IOP by >20% after addition of latanoprost).

This report suggests that the use of latanoprost slightly lowers the IOP in most glaucomatous patients already receiving maximal tolerated medical treatment. However, the reduction reaches a target level of > 20% less than the baseline IOP in fewer cases, approximately 1/4 of patients.

Each author states that s/he has no proprietary interest in the development or marketing of this or a competing drug.

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Current management of corneal abrasions: evidence based practice?

EDITOR,—Corneal abrasions are often painful, sometimes disabling but usually self limiting. They form a common presenting problem in general and ophthalmic casualties.[1 2] However, there is no scientifically proved, universally accepted best method of treating this condition. Here in Plymouth, we attempted to document the various methods used in the management of corneal abrasions (including iatrogenic cases) nationally. Therefore, a questionnaire postal survey of all the ophthalmic units in the UK (England, Wales, Scotland, and Northern Ireland) was carried out during February and March 1997. In total, out of 162 questionnaires sent out, 134 were received, representing a response rate of 83%. Only 22% of the respondents have an established departmental policy for the treatment of corneal abrasions. In three quarters of the non-policy holding majority, patient management decisions are made by doctors alone. Patients are most commonly treated with an immediate dose of topical antibiotic or antibiotic and cycloplegic followed by a course of topical antibiotic. Padding and patient follow up is practised some of the time by most units and all of the time by the remaining minority. There is no statistically significant difference ($p>0.05$) between the policy holders and non-policy holders in their management regimes. The traditional trio of topical antibiotic, cycloplegic, and padding is still the mainstay of corneal abrasion treatment among units nationwide. However, there is a lack of reproducible scientific evidence to support this treatment.[3 4] Larger randomised trials looking at the efficacy of the different treatment options are needed. Based on the outcome of future research, national practice protocols may be formulated and put into practice. This could reduce wasteful expenditure on ineffective treatments and make patient review more selective thus reducing costs to patient and provider alike. Furthermore, with clear policies in place, the management of corneal abrasions can be restructured so that the nursing staff and perhaps general

practitioners play an increasingly active role in the diagnosis, treatment, and follow up of patients.

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OBITUARY

Roy H Steinberg MD, PHDRoy H Steinberg

Roy H Steinberg died peacefully on 26 July 1997 at his home after a four year battle with multiple myeloma. The ophthalmic community has lost a good friend and ally. He was one of a breed of visual scientists who was prepared to spend the time, and had the patience, to communicate intelligibly with clinicians. These qualities, in someone who was pre-eminent in science and who was without prejudice, are uncommon. Roy had the desire to bring the advances in science into the clinical forum, which was helped by being medically qualified.

This motivation is exemplified by his latest work with Matt LaVail. Initially, it was shown that a variety of growth factors slowed retinal photoreceptor cell loss in the RCS rat. Subsequently, they demonstrated retinal rescue in light damage, and in rodents with different genetically determined retinal dystrophies. The ultimate goal is the development of a form of treatment for inherited retinal degeneration in humans. Of all the forms of potential therapy for these disorders, the use of growth factors seems to be the easiest to incorporate into clinical practice. During this work Roy always appreciated the sense of urgency that existed in the patient community, and yet was scrupulous in the maintenance of scientific discipline. His determination is illustrated by his attendance at the laboratory until a few days before his death.

Roy was brought up in New York and went to college in New York and Michigan, before going to medical school at New York Medical College. Following an internship at Massachusetts Memorial Hospital, he acquired a formal training in research with Herbert Jasper at the Montreal Neurological Institute. We should all be grateful that he decided to specialise in the visual system. He continued his research during military service at the Naval Aerospace Medical Institute in Pensacola, Florida, and was subsequently appointed to the University of California, San Francisco where he spent the remainder of his working life.

Roy won the highest respect and reputation for his scientific work, gaining numerous accolades including the Friedenwald award in 1987. Most recently, he and Matt LaVail received jointly the Moran prize from the University of Utah, which was presented at a very touching ceremony at Roy's house shortly before his death. Roy had many outside interests, including gardening, and had a passion for sport, following San Francisco baseball closely. He even tried to fathom out rugby during one of his many visits to the UK. Recently, he attended the Oxford Congress where his ability to communicate with clinicians was well demonstrated.

Roy will be remembered for his scientific achievements, which may influence the practice of clinical ophthalmology in the foreseeable future, but most of all as a generous and totally honest colleague. Roy will be sorely missed by his friends and coworkers, who enjoyed walks with him in Golden Gate Park, and drank phenomenal quantities of strong coffee on the sidewalks of San Francisco.

ALAN C BIRD

BOOK REVIEWS

If you wish to order, or require further information regarding the titles reviewed here, please write or telephone the BMJ Bookshop, PO Box 295, London WX1H 9TE. Tel: 0171 383 6244. Fax: 0171 383 6662. Books are supplied post free in the UK and for British Forces Posted Overseas addresses. Overseas customers should add 15% for postage and packing. Payment can be made by cheque in sterling drawn on a UK bank, or by credit card (MasterCard, VISA, or American Express) stating card number, expiry data, and your full name. (The price and availability are occasionally subject to revision by the Publishers.

Biopsy Pathology of the Eye and Ocular Adnexa. Ed Narsing A Rao. Pp 402. £85. London: Chapman & Hall Medical, 1996. ISBN 0 412 56720 2.

This multiauthored book has been written to support pathologists and ophthalmologists who are engaged in diagnostic routine ophthalmic pathology and are dealing mainly with biopsy specimens. The chapters are based on the anatomical regions of the eye and at the end of each there is valuable advice on the ways in which specimens can be examined macroscopically, processed, and dealt with at the histological level. Details of fixation, orientation, and plane of section are stressed and there is also a helpful description of the artefacts which are encountered in examination of histological preparations. Modern technology—for example, electron microscopy, in situ hybridisation, polymerase chain reaction, etc, is outlined in chapters 1 and 2, but these techniques are not included in the subsequent text. Each chapter describes normal anatomy before the pathology is considered and this is very useful for the relatively inexperienced person. The systematic chapters describe the common lesions encountered and provide a compact description accompanied by black and white photomicrographs. Useful tables summarise disorders such as the corneal stromal dystrophies and immunohistochemistry of orbital neoplasms.

As the authors stress, the nature of the material submitted to a laboratory is changing with clinical practice so that the descriptions of the pathological features of epiretinal membranes, subretinal membranes, vitreous samples, and intraocular lenses are of great value.

There are inevitable weaknesses in a multiauthored textbook and in this book there is unnecessary repetition. For example, chapter 8, which describes the pathology of the lens, reiterates text in previous chapters. The final chapter describes methods of dealing with an enucleated eye and rather surprisingly, at this stage, the reader is faced with histopathological descriptions of the common intraocular tumours of uvea and retina which could have been included in the earlier chapters.

Another criticism is that the arrangement of the subdivisions within chapters is unconventional and in several chapters neoplasia precedes degenerative and inflammatory diseases, so that the reader is forced to rely on the index. This is particularly a problem in the section on uveal tumours, which is not subdivided by headings.

Minor criticisms are that some of the illustrations are of less than desirable quality and the lettering is insufficiently bold for clear identification. Also the bibliography is based primarily on the American literature and the majority of the references were published more than 5 years ago.

None the less, this bench book will be extremely valuable to any diagnostic pathologist who has a limited background and requires a clear, succinct account of the abnormalities he is likely to encounter.

WILLIAM R LEE

Ophthalmic Photography—a Textbook of Retinal Photography, Angiography, and Electronic Imaging. By Patrick J Saine, Marshall E Tyler. Pp 334. £87.50. Oxford: Butterworth-Heinemann, 1996. ISBN 0 7506 9793 8.

This textbook contains a great deal of information on ophthalmic photography, from conventional fundus photography and film processing to fluorescein angiography and digital imaging technology.

After an initial short history of ophthalmic photography and a brief description of the evolution of fluorescein angiography, the authors launch into a thorough description of the practicalities of performing fundus photography. The photographic routine is described in some detail, from seating patients correctly and dilating their pupils, to aligning the camera, obtaining the required view of the fundus, and ensuring correct focus.

Chapter 3 describes stereo photography as performed with both dedicated stereo and conventional monocular fundus cameras. The next two chapters are concerned with fluorescein angiography and include descriptions of the dye itself, as well as taking the reader step by step through the angiography procedure. Anterior segment angiography is also described briefly.

Chapter 6 contains a thorough description of the processing and printing of fluorescein angiograms, from designing the darkroom and processing the negatives to procedures for producing contact sheets and enlargements.

Practical advice on the choice of suitable films, developers, and development times is also included.

The field of electronic imaging is evolving rapidly and the current state of the art is described in chapter 7. This is mainly centred around the use of digital fundus cameras and their benefits or otherwise when compared with conventional photographic fundus cameras. The issues to consider when purchasing a digital system are discussed in some detail. These include minimum practical image resolution, potential cost, and time savings over photographic development, as well as ensuring that the images can be conveniently viewed, printed, and archived. Although the examples of commercially available cameras considered in the text are certainly up to date, these issues are discussed in a way that can be detached from the state of the art of current hardware and software. The hazards associated with the ease with which digital images may be manipulated and enhanced are also discussed. A brief description of indocyanine green angiography is given and the chapter concludes with little more than a passing reference to scanning laser ophthalmoscopy.

Chapters 8 and 9 discuss the issues involved in maximising the information content of fundus photographs and include descriptions of ocular anatomy and the appearance of common retinal and choroidal abnormalities in these images.

Ophthalmic photography is an important and highly skilled profession and this is very much a practical guide to the techniques involved in obtaining consistently informative, high quality images. It is clearly aimed squarely at the retinal photographer, containing detailed descriptions of the techniques of both fundus photography and fluorescein angiography whether performed using conventional photographic or digital imaging techniques. It is well written, amply illustrated, and contains a wealth of detailed practical information.

JOHN HIPWELL

NOTICES

10th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 10th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 11–13 December 1997 at the Johns Hopkins Medical Institutions, Baltimore, Maryland, USA. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical Education, Turner 20/720 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage: http://ww2.med.jhu.edu.cme)

20th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 20th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 5–10 February 1998 at the Hyatt Regency Cerromar Beach Hotel, Dorado, Puerto Rico. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical Education, Turner 20/720 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage: http://ww2.med.jhu.edu.cme)

2nd International Glaucoma Symposium (IGS)

The 2nd International Glaucoma Symposium will be held on 15–20 March 1998 in Jerusalem, Israel. Further details: The 2nd IGS Sectretariat, PO Box 50006, Tel Aviv 61500, Israel. (Tel: +972-3-514-0000; fax: +972-3-517-5674; email: glaucoma@kenes.com)

15th Annual Wilmer Institute's Current Concepts in Ophthalmology

The 15th Annual Wilmer Institute's Current Concepts in Ophthalmology will be held on 15–20 March 1998 at Manor Vail Lodge, Vail, Colorado. Further details: Program Coordinator, Johns Hopkins Medical Institutions, Office of Continuing Medical Education, Turner 20/720 Rutland Avenue, Baltimore, MD 21205, USA. (Tel: 410 955-2959; fax: 410 955-0807; email: cmenet@som.adm.jhu.edu; homepage:http://ww2.med.jhu.edu.cme)

Globe 98—International Telecommunication Live-Surgery Event

Globe 98, the International Telecommunication Live-Surgery Event will be held on 27–28 March 1998 in Innsbruck, Austria. Further details: International Telecommunication Live-Surgery Network (ILSN), Fürstenweg 165, A-6020 Innsbruck, Austria. (Tel: 0043-512-286688 or 0043-512-581860; fax: 0043-512-264838; email: ilsn@net4you.co.at; homepage:http://www.carrier.co.at/ilsn/)

11th Annual Meeting of German Ophthalmic Surgeons

The 11th Annual Meeting of German Ophthalmic Surgeons will be held on 28–31 May 1998 in the Meistersingerhalle, Nürnberg, Germany. Further details: Organisation Nürnberg GmbH, Wielandstrasse 6, D-90419 Nürnberg, Germany. (Tel: +49-911-393160; fax: +49-911-331204.)

9th British Association of Day Surgery Annual Scientific Meeting and Exhibition

The 9th British Association of Day Surgery Annual Scientific Meeting and Exhibition will take place at the Harrogate International Centre on 4–6 June 1998. Further details: Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, W Yorks HD7 3AP. (Tel: 01484 854575; fax: 01484 854576; email info@kitecomms.co.uk)

XVIIIth International Congress of Ophthalmology

The XXVIIIth International Congress of Ophthalmology will be held in Amsterdam on 21–26 June 1998. Further details: Eurocongres Conference Management, Jan van Goyenkade 11, 1075 HP Amsterdam, Netherlands. (Tel: +31-20-6793411; fax: +31-20-6737306; internet http://www.solution.nl/ico-98/)

First Combined International Symposium on Ocular Immunology and Inflammation

The First Combined International Symposium on Ocular Immunology and Inflammation will be held in Amsterdam on 27 June–1 July 1998. The meeting is sponsored by the International Ocular Immunology and Inflammation Society, the International Uveitis Study Group, and the Immunology and Immunopathology of the Eye Organisation. Further details: Professor Aize Kijlstra, The Netherlands Ophthalmic Research Institute, PO Box 12141, 1100 AC Amsterdam, Netherlands (email: a.kijlstra@amc.uva.nl)

2nd International Conference on Ocular Infections

The 2nd International Conference on Ocular Infections will be held on 22–26 August 1998 in Munich, Germany. Further details: Professor J Frucht-Pery, 2nd International Conference on Ocular Infections, PO Box 50006, Tel Aviv, 61500, Israel. (Tel: 972 3 5140000; fax: 972 3 5175674 or 5140077; email: ocular@kenes.com)

First International Conference on the Optic Nerve

The First International Conference on the Optic Nerve to be held in Tel Aviv, Israel on 30 August–4 September 1998 has been cancelled due to lack of financial support.

ICOP 98

The next International Conference in Ophthalmic Photography (ICOP) will be held on 19–21 September 1998. Further details: Mrs Gillian Bennerson, Senior Ophthalmic Photographer, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX. (Tel: 0117-928-4677.)