CORRESPONDENCE

Endonasal laser dacryocystorhinostomy

EDITOR,—I read the article by Sadiq and others with great interest.¹ The figures presented, though in accordance with the literature,²⁻⁴ may be misleading at first sight. Of the 21 patients with endonasal laser dacryocystorhinostomy without intubation postoperatively, only 10 can be reported to be successful after 12 months. These are only 47.62% of the total number of patients who underwent surgery without stenting instead of the 91% reported. In the group of 65 patients with intubation postoperatively 41 (63.08%) were successful after 12 months instead of the 70% reported. Of the total number operated (86 patients) only 63 (59.3%) were successful after 12 months instead of the 81% reported. The figures presented by Sadiq and others are doubtless in accordance with the literature, because success rates and reported figures are heterogeneous. The only common feature of all studies is that failure of laser assisted dacryocystorhinostomy as a result of fibromembraneous scarring of the lacrimal system occurs in a rather early postoperative phase.

The main problem of all reviewed studies⁵⁻¹² is the enormous heterogeneity in patient selection criteria, indications for laser assisted dacryocystorhinostomy, approach, equipment, methods, statistics, and follow up criteria. Obviously the results will be different in an idiopathic, punctual postsaccal stenosis in a child compared with a chronic inflammatory process due to degenerative changes of the lids and the lacrimal apparatus with superinfection in an old patient with rosacea and an extended fibrotic scar of the lacrimal system.

Thus, randomised and controlled prospective trials with a well defined standardised protocol are needed. We must define patient inclusion and exclusion criteria. Indications to specified treatment and surgical methods must be determined. It is not helpful to compare the treatment of different patients with different diseases treated with different surgical methods. We must define what particular method is best to treat what particular disease. There is no doubt, that the external dacryocystorhinostomy (Toti)^{13,14} as well as the endonasal approach (West)^{15,16} are the most frequently used with the highest success rates.¹⁷ The indications when to use a Toti or a West procedure are well established among ophthalmologists and ENT surgeons.¹⁸ There is no doubt that laser assisted dacryocystorhinostomy will find its place in lacrimal system occlusion surgery; the question is what method is best to use for what pathology in which patient at what price?

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Reply

EDITOR,—We thank Dr von Arx for his appraisal of our report. Retrospective analyses are prone to a great number of problems, one of these being the dropout rate of patients attending for continued review. This may be for a variety of reasons including death, relocation, or a desire not to return to the hospital for appointments which may not be deemed useful by the patient (usually due to a lack of symptoms rather than a return to the preoperative state—the latter of these usually results in earlier than arranged return). We (along with most clinicians) feel it better to remove such patients from the analysis altogether, although it can easily be seen that if these dropouts are regarded as a success or a failure, then this will either enhance or reduce the results respectively. Ignoring the dropouts, our results show 1 year success rates of 56% and 64% for patients without and with intubation respectively. We do believe these figures to be lower than the true rates owing to the reasons already stated. In addition, we have found that in several cases requiring revisionary conventional DCR, failure has occurred because of proximal pathology in the presence of a patent distal fistula created with the laser.

Our study involved consecutive patients undergoing endonasal laser DCR (ELDCR) and we have listed various risk factors which reduce the success rate of the procedure. Separating patients with and without these risk factors, we can achieve 1 year respective success rates of 56% versus 69% after intubation and 50% versus 66% without intubation (paper submitted). Case selection as suggested by Arx would no doubt have increased

our success rates, but we do not feel that selection of only low risk cases is a good measure of the usefulness of a procedure and would give artificially better results. Randomised controlled trials are required to assess techniques which may be of equal efficacy, and as we have never offered ELDCR as having the same success rates as conventional surgery, we do not think that this type of study would be appropriate. Indeed, the technique was initially commenced to provide treatment alternatives for patients who were medically unfit to undergo the demands of a major operation such as conventional external approach DCR. We are at present conducting long term observation of the technique and in the near future would hope to advise regarding the most suitable candidates for ELDCR.

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Acute glaucoma, chronic glaucoma, and serotonergic drugs

EDITOR,—We were interested to read Kirwan *et al.*'s report of acute angle closure glaucoma (AACG) associated with the antidepressant paroxetine (Seroxat),¹ as we have reported a similar case.² In Kirwan's report, AACG occurred within 24 hours of the first dose, suggesting an anticholinergic mechanism. Our case became symptomatic some 2 weeks after daily dosage was commenced, leading us to postulate that the effect could have been mediated by serotonergic pathways. This raises the possibility of other important ocular side effects with drugs of this class.

Paroxetine is an antidepressant of the selective serotonin reuptake inhibitor (SSRI) class. SSRIs act by inducing a gradual rise in postsynaptic levels of serotonin (5-hydroxytryptamine, 5-HT) via desensitisation of the feedback systems which control the rate limiting enzyme in 5-HT synthesis.³ The 5-HT receptors involved have not yet been fully elucidated, and their role in ocular physiology is a subject of ongoing research. In animal studies, serotonergic stimulation may cause mydriasis, and can have an independent effect in raising the intraocular pressure (IOP).⁴ Receptors for 5-HT have been demonstrated in the human eye.⁵

It is therefore possible that SSRIs could have an effect on IOP in humans. Consequently, we should look for raised IOP and open angle glaucoma as a side effect of SSRIs. A recent study has shown a significant short term rise in IOP after a single oral dose of the SSRI fluoxetine (Prozac). Twenty depressed patients were given either fluoxetine 20 mg or placebo in a randomised crossover blinded study, and fluoxetine was associated with a mean IOP elevation of over 4 mm Hg, lasting 6-8 hours.⁶ We have been unable to find any publication regarding the effect on IOP in the longer term, but we have recently become aware of previously unpublished data which partly address this issue.

In premarketing and subsequent clinical trials of fluoxetine, 585 adult patients of a wide age range have been assessed. Ophthalmological examinations were made at baseline, and again after a treatment period which ranged between less than 1 month and 24 months. "Five out of the 585 patients had a change in intraocular pressure... One had a decrease in IOP which was determined to be clinically insignificant [29 year old female, dosage 20 mg/day, examined at 1 month]. Four patients

experienced an elevation: in one patient the elevation was considered a normal diurnal variation [74 year old male, dosage 80 mg/day, examined at 2 months], in a second patient the elevation was felt to be related to angle closure [59 year old male, dosage 80 mg/day, examined at 1 month], the third patient acknowledged cocaine use prior to the examination and this was considered the probable explanation [37 year old male, dosage 80 mg/day, examined at 12 months], and the fourth patient had a 1 mm Hg rise with no probable extenuating circumstances [26 year old female, dosage 80mg/day, examined at 2 months]" (Dista Products Limited, personal communication).

Voluntary reporting of suspected adverse events with fluoxetine has identified a total of 63 cases of "glaucoma" in an estimated patient population of 21 million (Dista Products Limited, personal communication). The manufacturers of paroxetine are aware of four cases of AACG, six of "glaucoma (unspecified)", and one of raised IOP, in a UK patient population of over one million (Smith-Kline Beecham Pharmaceuticals, personal communication).

These data indicate that our understanding of the effect of SSRIs on IOP is still unclear. The demonstration of a short term IOP rise after a single fluoxetine dose⁶ implies that chronic dosage could lead to a sustained elevation of IOP. However, the manufacturer's own data suggest that this is not the case, in that less than 1% of patients showed any IOP change after treatment. The low incidence of reported glaucoma with SSRIs does not exclude a real effect: many clinicians may not suspect a particular drug to be a contributory factor when diagnosing a particular condition, especially if a causal relation has not been suggested in the literature. This is particularly true of open angle glaucoma, which is common and usually idiopathic, and of AACG, which is rarer but occurs in anatomically predisposed eyes.

We feel that this area merits further study and clarification, particularly regarding the effect of long term SSRI administration on IOP. In the meantime, we would encourage colleagues to report cases of glaucoma or raised IOP which may be associated with SSRIs to the Committee on Safety of Medicines.

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Reply

EDITOR,—In 1991 Ahmad¹ first reported the occurrence of glaucoma, probably an acute angle closure glaucoma (AACG), in a 35 year old man as a result of fluoxetine administration, during the fifth week of therapy. More recently, also Kirwan *et al.*² and Eke *et al.*³ described two cases of AACG associated with the administration of paroxetine, an antidepressant which acts as a selective inhibitor of the serotonin neuronal uptake (SSRI). In 1996 we described the effect of fluoxetine oral administration on intraocular pressure (IOP) in 20 consecutive depressed patients and demonstrated that after a single dose of 20 mg IOP was significantly increased.⁴

Serotonin (5-HT) is present in mammalian iris ciliary body and cornea at higher concentration than in non-mammalian species.⁵ While a transmitter role for serotonin in the retina has been established, conflicting data exist in the literature on whether or not activation of serotonin receptors can cause changes in IOP. Experimental evidences demonstrated that topical application of serotonin increased IOP in rabbit eyes, and that 5-carboxamidotryptamine, a 5-HT₁ receptor agonist, is even more effective than 5-HT itself in elevating IOP.⁶ These results confirm the involvement of serotonin receptors in the regulation of IOP, and the fact that ketanserin, a compound with serotonergic blocking properties, reduces IOP in animals and humans emphasises the role exerted by 5-HT on IOP.⁷

To date, no long term studies regarding the effect of SSRI on IOP have been published. We know the prevalence of primary open angle glaucoma (POAG) (conservatively estimated to be approximately 1/200 of the general population over the age of 40 years)⁸ and that of AACG (approximately 1/1000 individuals over the age of 40 years),⁸ while the incidence of new cases within the population treated with SSRI has not yet been evaluated, with the exception of the manufacturer's own data, which do not show a significant occurrence of IOP changes induced by SSRI treatment (Dista Products Limited, personal communication; SmithKline Beecham Pharmaceuticals, personal communication). POAG is an asymptomatic disease, until significant visual field loss occurs, and a single IOP reading does not necessarily exclude or confirm the diagnosis. The rise in IOP after a single dose of SSRI was about 4 mm Hg⁴ in a non-glaucomatous population, equivalent to the diurnal fluctuations of IOP found in normal subjects,⁹ and probably is without long term effect on the ganglion cell integrity. On the contrary, this same variation, together with the small changes in pupil size reported by other investigators,¹⁰ in the so called "predisposed eyes" (that is, patients with positive family history of glaucoma and/or patients with physiological risk factors—relatively anterior location of the iris-lens diaphragm, shallow anterior chamber, and narrow entrance to the chamber angle) is able to modify the hydrodynamic homeostasis of the eye, leading to the acute angle closure.

These considerations suggest the need to include an ophthalmological examination in the protocol of depressed patients before starting SSRI administration.

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Authors' reply

EDITOR,—Eke *et al.*'s letter raises several interesting points. The rapid onset of acute angle closure glaucoma in our case implies an anticholinergic mechanism. The patient in Eke's report may simply have had an anticholinergic response to commencement of the higher dose 3 days before. However, the serotonergic mode of action with this agent does raise the issue of both a mydriatic effect and an effect in eyes with open angles. As with any recently introduced class of drug, it is important for clinicians to monitor closely possible adverse effects.

So far the data reported from the manufacturers reveal a minimal number of cases of raised intraocular pressure, not different from the probable incidence of primary open angle glaucoma in the general population. However, a small study with careful monitoring of intraocular pressure demonstrated a small but significant elevation of IOP over 12 hours.¹ Subjects were, however, young adults and the study was over a short period. We have little idea what happens over the longer term. For example, tricyclic antidepressants have profound anticholinergic side effects on commencement of treatment to which patients physiologically adapt to the point that sudden withdrawal leads to a rebound cholinergic syndrome. The effect in older patients, those with anatomically predisposed narrow angles, or indeed glaucoma patients may be very different. If this class of agents does increase intraocular pressure by 4 mm Hg in a sustained fashion this could potentially have clinical implications in population terms for glaucoma risk. In the year ending August 1994, British general practitioners wrote 13 million prescriptions for antidepressants.² A significant proportion of these were SSRIs.

Should the possibility of this class of agent causing an elevation of intraocular pressure modify their current use? In general the answer should be no. Depression is common; the estimated lifetime prevalence of "major depression" in women is between 4.9% and 8.7% and in men, 2.3–4.4% with even higher figures applying to the less severe end of the spectrum.³ Depression is a severe, disabling disease. It has a high mortality. It is estimated that severe depressive illness increases the risk of suicide by a factor of 30 such that 15% die

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by suicide.⁴ Alternative treatments with other antidepressants are generally less well tolerated, especially in the elderly. Despite the possibility of raised intraocular pressure the risk/benefit ratio will almost always favour treating depression with the optimum agent.

Until the real effect of SSRIs on IOP has been ascertained it is difficult to make suggestions on management and further evidence on this subject is required. However, it would seem prudent to closely monitor glaucoma patients who have recently commenced treatment with an SSRI. Given limited ophthalmic resources, until we know more about the long term effects of these drugs on IOP it does not seem reasonable to recommend ophthalmological screening of all patients commenced on SSRIs.¹ Like Eke and Carr, we encourage colleagues to report cases of glaucoma or raised IOP that may be due to therapy with SSRIs. Additionally, we would stress the importance of communication between disciplines so that no doctor is unaware of prescribed medication.

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BOOK REVIEWS

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Eye Diseases in Hot Climates. By John Sandford-Smith. Pp 352. £45. Oxford: Butterworth-Heinemann, 1997. ISBN 0-7506-2544-9.

This is the 3rd edition of John Sandford-Smith's excellent companion volume to his *Eye Surgery in Hot Climates*. Its purpose is to be a practical guide for the hard pressed health worker, doctor, nurse, or medical assistant. The book is written bearing in mind that for

many readers English will not be their first language and so the text is kept simple and lucid.

It is written against a background of 38 million people in the world who are blind, the majority living in hot developing countries. The introductory chapter is an excellent overview of the problem touching on the contributory causes, particularly poverty and lack of education and all that follows on from that. Approaches to prevention and treatment are discussed.

The remainder of the book deals with eye disease in a systematic way starting with chapters on basic anatomy, physiology, history taking, and eye examination. The common eye diseases in hot climates are mainly the same as in more temperate parts but the thrust of the book is the approach to these diseases in situations where there are limited resources in expertise, diagnostic equipment, and treatment. There are clear descriptions of how to perform basic examinations with limited cheap equipment and treatments recommended include the cheaper alternatives as well as the most modern medications. I particularly liked the advice on how to make up fortified antibiotic drops for corneal ulcers for example. As expected there are several good chapters on those diseases more specifically associated with tropical countries such as trachoma, xerophthalmia, onchocerciasis, and leprosy.

A minor criticism is that the whole of ophthalmology is tackled and many rarer conditions just get a mention. Perhaps it would have been better to leave them out altogether and concentrate on the common conditions that are done so well. Nevertheless, this is a very valuable book for those working in developing countries where many workers find it indispensable. I will certainly continue to recommend it to students going overseas on electives for eye projects.

F D GREEN

Thyroid eye disease. 3rd ed. By Devron H Char. Pp 293. £70. Oxford: Butterworth-Heinemann, 1997. ISBN 0-7506-9893-4.

Thyroid associated ophthalmopathy continues to vex endocrinologists, ophthalmologists, and immunologists. At one of the earliest meetings of the Thyroid Club (now the British Thyroid Association) the debate on this subject was so intense that the secretary urged that it continue until no one was left, and the meeting closed with some difficulty at 10.00pm. Forty years later controversy still rages about the pathogenesis, the relation to the thyroid condition, and the best way to treat both the orbit and the thyroid in these patients.

This book, remarkably, is written by a single author and encompasses all of these areas. Devron Char is professor of ophthalmology and radiation oncology at the University of California at San Francisco. As the book is in its third edition already within 10 years, this demonstrates the changes that have been made in our understanding and provides a continuously revised text which gives a vast array of information. The shortcoming of this approach is that it is very difficult for anyone to be an expert these days in all areas that touch on ophthalmopathy. It is particularly in the areas of arcane immunology that the book is perhaps weakest and in future editions it might be worth commandeering the services of an immunologist to write specifically on this topic even if this leads to loss of uniform

style. Another difficulty is that the text, although divided into sections, might benefit from further breakdown under subheadings. This is particularly apparent in chapter 6 which covers the pathogenesis and pathophysiology of thyroid ophthalmopathy. All of the immunological studies are grouped together under a single heading with no subdivision into genetics, antibodies, T cell involvement, and so on and this makes a difficult subject for the non-specialist even more taxing. There also appears to be some confusion over the role of free T4 testing in chapter 3 (Systemic diagnostic tests for thyroid ophthalmopathy and euthyroid ophthalmopathy). Free T4 assays are now simple, reliable, and cheap.

These, however, are relatively minor drawbacks compared with the overall worth of the book. The layout is very good and the pictures are excellent. The references are generally up to date as far as 1995 and there are one or two from 1996. Moreover, the references are extensive and give a detailed overview of even the earliest history of work on this disease. The second half of the book which details management is superb giving an overview of medical management (including radiation therapy) and surgical approaches. The author shows what can go wrong as well as what can go right and the personal account given makes compelling reading. Anyone who deals with thyroid associated ophthalmopathy will learn from this book. Perhaps by the next, or next but one, edition some of the questions which still remain will have been answered.

A P WEETMAN

NOTICES

Avoidable blindness

The latest issue of the *Community Eye Health* (no 25) discusses the elimination of avoidable blindness. With an editorial by Bjorn Thylfors, the director of the WHO Programme for the Prevention of Blindness and Deafness, the issue covers treatment of cataract in regions of India and the role of patient counsellors in increasing the uptake of cataract surgery and IOLs. 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) 171 608 6910; fax: (+44) 171 250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Residents' Foreign Exchange Programme

Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, Secretary of the Foreign Exchange Committee, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.

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)

Vth Tuebingen Angiography Course on AMD

The Vth Tuebingen Angiography Course on AMD with stereoscopic angiography wet-lab will be held on 26–27 August 1998 at the auditorium, University Dental Clinic and University Eye Clinic, Tuebingen, Germany. Further details: Dr W Inhoffen, University Eye Clinic, Department of Ophthalmology III, Schleibstrasse 12, D-72076 Tuebingen, Germany. (Tel: +49-(0) 7071-292968; fax: +40-(0) 7071-293746; email: ingrid.kreissig@uni-tuebingen.de)

XVI Tuebingen Detachment Course

The XVI Tuebingen Detachment Course in retinal and vitreous surgery will be held 4–5 September 1998 in Odessa, Ukraine. Further details: Professor I M Logai, Director, The Filatov Institute, 49/51 Boulevard Francais, Odessa, 270061, Ukraine. (Tel: +38-0482-22 20 35; fax: +38-0482-68 48 51.)

International Agency for the Prevention of Blindness (IAPB)

The International Agency for the Prevention of Blindness (IAPB) will hold its next general

assembly in Beijing, China on 5–10 September 1998. Further details: Gullapalli N Rao, Secretary General, IAPB Secretariat, LV Prasad Eye Institute, LV Prasad Marg, Banjara Hills, Hyderabad 500 034, India. (Tel: 091-40-215389; fax: 091-40-248267; email: IAPB@lvpeye.stph.net)

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.)

IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES)

The IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES) will be held in Vittel, France on 9–11 October 1998. Further details: Mrs Nicole Charron, Director, Palais des Congrès, Av Bouloumie, BP 57, 8802 Vittel, France. (Tel: +33 329 08 18 30; fax: +33 329 08 6601.)

VIth International Symposium on Graves' Ophthalmology

The VIth International Symposium on Graves' Ophthalmology will be held on 27–28 November 1998 in Amsterdam. Further details: Amsterdam Thyroid Club, Department of Endocrinology, F5-171, Academisch Medisch Centrum, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands.

Singapore National Eye Centre

The 3rd SNEC international meeting and 11th international meeting on cataract, implant, microsurgery and refractive keratoplasty (ICIMRK) will be held at the Shangri-La Hotel, Singapore on 28–30 November 1998. Further details: Organising Secretariat, 3rd SNEC International Meeting and 11th ICIMRK, Singapore National Eye Centre Pte Ltd, 11 Third Hospital Avenue, Singapore 168751. (Tel: (65) 2277-255; fax: (65) 2277-290/1)

Ophthalmic technologies

The 9th Ophthalmic Technology Conference will be held on 23–24 January 1999 during the International SPIE symposium on biomedical optics. Further information: The SPIE Organisation, PO Box, Bellingham, WA 98227-0010, USA. (Fax: (+1) 360-647-1445; email: www.spie.org/info/pw)

Laser eye injuries

A conference on the epidemiology, prevention, diagnosis, and therapy of laser eye injuries will be held in San Jose, California on 25–26 January 1999 during the International SPIE symposium on biomedical optics. Further information: The SPIE Organisation, PO Box, Bellingham, WA 98227-0010, USA. (Fax: (+1) 360-647-1445; email: www.spie.org/info/pw)

XII Congress European Society of Ophthalmology

The XII Congress European Society of Ophthalmology will be held in Stockholm, Sweden on 27 June–1 July 1999. Further details: Congress (Sweden) AB, PO Box 5819, S-114 86 Stockholm, Sweden. (Tel: +46 8 459 66 00; fax: +46 8 661 91 25; email: soe@congrex.se; http://www.congrex.com/soe/)

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The web site is at a preliminary stage and there are plans to develop it into a more sophisticated site. Suggestions from visitors about features they would like to see are welcomed. They can be left via the opening page of the BMJ Publishing Group site or, alternatively, via the journal page, through "about this site".