Endonasal laser dacryocystorhinostomosis

Editor,—I read the article by Sadiq and others with great interest.1 The figures presented, though in accordance with the literature, may be misleading at first sight. Of the 21 patients with endonasal laser dacryocystorhinostomosis without intubation postoperatively, only 10 can be reported to be successful after 12 months. These are only 47.6% 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 upon (86 patients) only 63 (59.3%) were successful after 12 months instead of the 81% reported. The figures given by Sadiq and others are not 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 dacryocystorhinostomosis results in an earlier than arranged return. We are at present conducting long term observation of the technique and in the near future we hope to present the most suitable candidates for ELDCR.  

S A SADIQ  
Eye Clinic, Queen’s Medical Centre, Nottingham NG7 2ZU

Acute glaucoma, chronic glaucoma, and serotoninergic drugs

Editor,—We were interested to read Kirwan et al.’s2 report of acute angle closure glaucoma (AAGC) associated with the antidepressant paroxetine (Seroxat),3 as we have reported a similar case.4 In Kirwan’s report, AAGC occurred within 24 hours of the first dose, suggesting an anticholinergic effect. Our case became symptomatic some 2 weeks after daily dosage was commenced, leading us to postulate that the effect could have been mediated by serotoninergic pathways. This raises the possibility of a great number of ocular side effects with drugs of this class.

Paroxetine is an antidepressant of the selective serotonin reuptake inhibitor (SSRI) class. SSRI’s 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.5 The 5-HT receptors involved have not yet been fully elucidated, and their role in oculomotor physiology is a subject of ongoing research. In animal studies, serotoninergic stimulation may cause mydriasis, and can have an independent effect on IOP.6 Receptors for 5-HT have been demonstrated in the human eye.7 It is therefore possible that SSRI’s could have an effect on IOP in humans. Consequently, we should look for raised IOP and open angle glaucoma as a side effect of SSRI’s. 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.8 We have found 5-hour IOP levels, in response to open publication regarding the effect of 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 6 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

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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 dynamic nature 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 separate success rates from the analysis altogether, although it can easily be seen that if these dropout rates 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 44% for patients without and with intubation respectively. We do believe these figures to be lower than the true rates owing to the reasons stated above. In addition, we have found that in several cases requiring revision 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 not have increased our success rates, but we do not feel that selection of only low risk cases is a guarantee 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 we hope to present the most suitable candidates for ELDCR.

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CORRESPONDENCE

Medical School, University of Nijmegen, Nijmegen, The Netherlands
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patient population of 21 million (Dista personal communication).

at 2 months” (Dista Products Limited, personal communication).

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properties, reduces IOP in animals and humans

that serotonin receptors can cause changes

in IOP. However, the manufacturer’s

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in IOP. Experimental evidences demonstrated that topical application of serotonin increased

IOP in rabbit eyes and that

5-carboxamidotryptamine, a 5-HTla receptor agonist, is even more e
tective than 5-HT itself

in elevating IOP.3 These results confirm the

involvement of serotonin receptors in the regulations of IOP. Moreover, ketanserin, that ketanserina compound with serotenergic blocking properties, reduces IOP in animals and humans emphasises the role exerted by 5-HT on IOP.4 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)5 and that of POAG (approximately 1/1000 individuals over the age of 40 years), 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 a 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 Hg in a non-glaucomatous population, equivalent to the diurnal fluctuations of IOP found in normal subjects, 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, 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 compromised eyes have the need to include an ophthalmological examination in the protocol of depressed patients before starting SSRI administration.

CIRO COSTAGLIOLO

Eye Clinic, II University of Naples

LEONARDO MASTROPAQUA

Eye Clinic, Giuseppe University of Chieti


4 Osborne NN, Tobin AB. Serotonin accumulat-

ing cells in the iris-ciliary body and cornea at higher concen-


5 Barnett NL, Osborne NN. The presence of sero-

tonin (5-HT1) receptors negatively coupled to adenylate cyclase in rabbit and human iris-


6 Costagliola C, Mastropassqua L, Staeardo L, et al. Fluoxetine oral administration increases in-


TOM EKE

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SUAN CARR

Drug Information Centre, Leicester Royal Infirmary, Leicester LEI 2WF

Reply

Editor,—In 1991 Ahmad1 first reported the occurrence of glaucoma, probably an acute angle closure glaucoma (AAGC), in a 35 year old man as a result of fluoxetine administration, during the fifth week of therapy. More recently, also Barnett and Eke2 described two cases of AAGC associated with the administration of paroxetine, an antide-

pressant 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.3

Serotonin (5-HT1D) receptors on human 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.4 These results confirm the involvement of serotonin receptors in the regulations of IOP. Moreover, ketanserin, a compound with serotenergic blocking properties, reduces IOP in animals and humans emphasizes the role exerted by 5-HT on IOP.4 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)5 and that of POAG (approximately 1/1000 individuals over the age of 40 years), 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 a 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 Hg in a non-glaucomatous population, equivalent to the diurnal fluctuations of IOP found in normal subjects, 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, 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.

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Authors’ reply

Editor,—Eke et al’s letter raises several inter-
esting points. The rapid onset of acute angle closure glaucoma in our case implies an anticholinergic mechanism. On the contrary, this same variation, together with the diurnal fluctuations of IOP found in normal subjects, 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, 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 compromised eyes have the need to include an ophthalmological examination in the protocol of depressed patients before starting SSRI administration.

LUCA STEARDO

Department of Neurology, Federico II University of Naples, Via S Pansini, 5 80131 Napoli, Italy

1 Barnett NL, Osborne NN. Serotonin accumu-


2 Costagliola C, Iuliano G, Rinaldi M, et al. Effect of topical ketanserin administration on intra-


3 Costagliola C, Mastropassqua L, Staeardo L, et al. Fluoxetine oral administration increases in-


4 Osborne NN, Tobin AB. Serotonin accumula-


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tonin (5-HT1) receptors negatively coupled to adenylate cyclase in rabbit and human iris-


6 Costagliola C, Mastropassqua L, Staeardo L, et al. Fluoxetine oral administration increases in-

by suicide. 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 patients commenced on SSRIs. 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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2 Scriptcount 300 MAT to 26/8/94 [program]: Taylor Nelson Healthcare.

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 W1X 1HT. 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 may 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.


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 mostly 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 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 touched on 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.

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 at length to the right 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, cheap, and widely available.

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 Thyl-efors, 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@aol.com) 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 Inho, University Eye Clinic, Department of Ophthalmology III, Schleichstrasse 12, D-72076 Tuebingen, Germany. (Tel: +49-(0) 7071-292968; fax: +49-(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 Filatow Institute, 49/51 Boulevard Francais, Odessa, 270061, Ukraine. (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.)

Vth International Symposium on Graves’ Ophthalmopathy

The Vth International Symposium on Graves’ Ophthalmopathy 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 (ICIRMK) will be held at the Shangri-La Hotel, Singapore on 28–30 November 1998. Further details: Organising Secretariat, 3rd SNEC International Meeting and 11th ICIRMK, 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/)
