would appreciate it if the authors could address this issue.

NEIL R MILLER
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Reply
EDITOR,—We thank Dr Miller for his interest and commentary about our paper on pleomorphic adenomas of the lacrimal gland. We are unable to answer the question of whether the incidence of pleomorphic adenomas in comparison to other tumours into surrounding tissues is less if the whole of the tumour is removed immediately after biopsy or after an interval of several days. Theoretically, biopsy with frozen section can reduce contact between normal tissue and the biopsy site. Biopsy will be trans-septal, however, whereas excision of lacrimal pleomorphic adenomas should use a lateral orbitotomy approach, so that there is potential for seeding of tumour cells during the surgical intervention. Furthermore, the histopathological differential diagnosis of lacrimal gland lesions is more difficult with frozen section material than with appropriately stained paraffin sections.

Thankfully the majority of pleomorphic adenomas can be correctly diagnosed using the clinical and radiological criteria outlined in our paper. In these cases there is no need for a biopsy and the tumour can be removed with an intact capsule.

Our recommendation against biopsy of pleomorphic adenomas is based upon the landmark clinicopathological review of Font and Gamel, a view strengthened by the high rate of recurrence in a large series of Chinese patients,1 many of whom were biopsied. With the current follow up intervals, our paper is unable to either confirm, or refute, this issue.

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Caution for lower lid entropion
EDITOR,—We read with interest the article by El-Kasaby.1 It is important to recognise the basic pathophysiology of involutional entropion and the surgical approach must correct the anatomical involutional changes. These changes cause the preseptal orbicularis muscle to override the pretarsal muscle. Lack of pretarsal orbicularis muscle to override the preseptal muscle. Lack of pretarsal orbicularis muscle may cause more traction on the lid margin. Lack of pretarsal orbicularis muscle may cause more traction on the lid margin.

There is no doubt that a systematic approach to the evaluation of the major contributing factors in entropion is to be commended. Nevertheless, this needs a degree of experience as does the surgical tackling of the problem. The procedure described is simple, effective, and can be done by any surgeon who has no access to specialist oculoplastic training.

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This condition was previously termed spastic entropion but the spastic component is secondary to ocular irritation by internuming lashes. The author makes the mistake of trying to treat the effect rather than the cause of the condition and states that scars interrupt the continuity of the pretarsal part of the orbicularis muscle, thus eliminating the spasm which contributes to the entropion. While this may be partly true in the very short term, one has only to look at the orbicularis function of patients who have had a total orbicularis myectomy for blepharoplasty to appreciate that three burns would have a negligible effect on the function of the orbicularis muscle.

The author documents one recurrence of entropion in 50 procedures but the means of lowing this is not stated. This is best evaluated by asking the patient to squeeze shut the eyelids and assessing the lids on opening.

In the series of photographs presented, there is clearly hyperpigmentation at the sites of the burns and this should be remembered as a side effect. El-Kasaby recommends this procedure for patients who are 'infirm and bedridden'. Most entropion surgery is performed using local anaesthesia and takes no longer than 15 to 20 minutes and addresses the underlying pathophysiology.

Modern oculoplastic surgeons such as Collin in Europe and Anderson in the United States have greatly advanced our understanding of the anatomy and pathophysiology of normal and abnormal eyelid and orbital structures. They have both stressed the systematic approach to evaluation of oculoplastic problems and defined specific operations to address the underlying pathology. Oculoplastic surgeons would do well to follow the examples set by these surgeons. While the author is to be congratulated for confirming the findings of Ziegler in 1990, the above factors must be borne in mind by the oculoplastic surgeon.

BHUVENDRA C PATEL
PATRICK FLAHARTY
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Reply
EDITOR,—I thank Drs Patel and Flaharty for their interest in my article and for their comments. One should bear in mind that there are many specialist oculoplastic surgeons in the United States, but in the United Kingdom correction of lower lid entropion is usually performed by general ophthalmologists and the job is mainly left to juniors without much experience.

There is no doubt that a systematic approach to the evaluation of the major contributing factors in entropion is to be commended. Nevertheless, this needs a degree of experience as does the surgical tackling of the problem. The procedure described is simple, effective, and can be done by any surgeon who has no access to specialist oculoplastic training.

Drs Patel and Flaharty believe that caution of the anterior lamella only corrects preseptal orbicularis override. I do not share their evaluation. They chose to ignore the effect of contraction of three vertical linear scars which was mentioned in the article. Also, relieving the spasm of the orbicularis, although it may not be a long term effect, stops the vicious circle of spasm, ocular irritation, leading to more spasm. Involutional entropion is a multifactorial problem as pointed out and to correct one factor, such as orbicularis override, may often be sufficient.

Regarding the method of assessment of entropion postoperatively, the article is critical. The article states that three burns would have a negligible effect on the function of the orbicularis muscle. While this may be partly true in the very short term, one has only to look at the orbicularis function of patients who have had a total orbicularis myectomy for blepharoplasty to appreciate that three burns would have a negligible effect on the function of the orbicularis muscle.

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5-Fluourouracil and ocular toxicity
EDITOR,—We would like to draw your attention to an unusual side effect of the chemotherapeutic agent 5-fluourouracil. A 48-year-old man had been taking 5-fluourouracil orally, once weekly for 1 year following large bowel resection for colorectal cancer with hepatic metastases. He remained systemically well, but after starting the tablets he developed eye problems. Initially he became intolerant of his hard contact lenses, which he had worn without problems for 20 years. At first this was attributed to the antiemetic, which was thought to be causing dry eyes. The antiemetic was stopped but the eye problem became worse. He developed recurrent episodes of pain in one or both eyes, which woke him from sleep in the morning and persisted for 2 to 3 days. During these episodes he was severely incapacitated by photophobia. His eyes were slightly pink during the attacks but did not water continuously.

When first examined his visual acuities were 6/5 in the right eye and 6/6 in the left eye with spectacles. Examination was difficult owing to extreme light sensitivity. The lower lid margins showed keratinisation but his corneas looked normal. The 5-fluourouracil was stopped and after 2 weeks his symptoms had almost completely resolved. His conjunctiva and cornea could now be examined readily and were normal.

Ocular surface toxicity with 5-fluourouracil, giving rise to symptoms of photophobia and irritation, has been previously recorded.1,2 We think that this case is of particular interest because the patient’s symptoms mimicked those of the recurrent corneal erosion syndrome.3 However, examination of the corneas revealed no evidence of such a lesion. The length of time the symptoms took to resolve after stopping the drug is consistent with previous reports in which symptoms from acute surface toxicity resolve within 2–3 weeks after discontinuing treatment.4