would appreciate it if the authors could address this issue.

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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 presence of staining of tumour cells or fibres in the 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 excision.

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. Laxity of the lower lid retractors allows the lower border of the tarsal plate to rotate outwards and the loss of stiffness of the tarsal plate allows it to bend. Furthermore, involutional changes affecting the canthal tendons and tarsus lead to a horizontal laxity of the lid which is further aggravated by the relative enophthalmos caused by atrophy of the orbital fat. Caution of the anterior lamella only corrects one of these four factors—namely, the orbicularis override, and the same result can be achieved by bedside transverse sutures.

This condition was previously termed spastic entropion but the spastic component is secondary to ocular irritation by interumin 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 blepharospasm 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 assessment 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 aetiology.

Modern oculoplastic surgeons such as Collin in Europe and Anderson in the United States have greatly advanced our understanding of the anatomy and pathogenesis of normal and abnormal eyelid and orbital structures. They have both stressed the systematic approach to evaluation of oculoplastic problems and designed specific operations to address the underlying pathology. Ophthalmologists 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.

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5-Fluorouracil and ocular toxicity

EDITOR.—We would like to draw your attention to an unusual side effect of the chemotherapeutic agent 5-fluorouracil. A 48-year-old man had been taking 5-fluorouracil orally, once weekly for 1 year following large bowel resection for colorectal cancer with hepatic metastases. He remained systematically well, but after starting the tablets he developed eye problems. Initially he became intolerant of his contact lenses, which he had worn without problems for 20 years. At this point he was attributed to the antiemetin, 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 at night, and he was wearing his glasses all day. Over the weekend he became intolerant of his contact lenses and could not wear his glasses without a blinding pain. He was referred to us because the symptoms were so severe.

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-fluorouracil 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-fluorouracil, 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.1 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.3 4

5-Fluorouracil is a pyrimidine analogue and a DNA and RNA synthesis inhibitor. It is a synthetic antimetabolite and is protracted to the interphase of the cell cycle and to the synthesis of DNA and RNA. The therapeutic use of 5-fluorouracil is limited by its toxicity, which is high when used systemically and tolerable when used topically. The main toxicity of 5-fluorouracil is related to its ability to inhibit folate metabolism in the gastrointestinal tract. It is well known that 5-fluorouracil is associated with oral and pharyngeal toxicity.5–7 Oral and pharyngeal toxicity can be severe and life threatening and is worsened by the concomitant use of folate supplements.5–7 The treatment of ocular toxicity with 5-fluorouracil is limited to symptomatic therapy.8