A rational approach to the patient with floppy/lax eyelids

It has now been 15 years since Culbertson and Ostler first described 'the floppy eyelid syndrome'.1 Their initial report described obese, middle aged men who presented with chronic symptoms of ocular surface irritation and discharge associated with nocturnal eversion of rubbery, floppy, easily reversible, upper eyelids. The patients had a predilection for either unilateral symptoms or symptoms mainly affecting the eye on the side the patient would preferentially sleep, typically in a face down position on the pillow. Since the time of the original description of this syndrome, much has been published to expand our recognition and understanding of it, and many thought provoking questions have been raised with regard to the relation between generalised eyelid laxity and the effect on the ocular surface.

Van den Bosch and Lemij correctly demonstrate to us, in this issue of the journal, that a variety of patients present with chronic ocular surface irritation symptoms associated with lax eyelids, who do not have the classic floppy eyelid syndrome. While very definite subtypes of 'the lax eyelid syndrome' exist, the authors' use of a more broad term has merit. There are no doubt a variety of aetiologies, both mechanical and otherwise, to account for the chronic symptoms with which these patients suffer. The classic Culbertson and Ostler patients with floppy eyelid syndrome suffered from nocturnal eyelid eversion with chronic mechanical irritation and papillary tarsal conjunctival reaction. Even with an apparent, obvious, chronic, mechanical injury to the tarsal conjunctival surface, the mechanism of corneal and epithelial irritation has not been fully elucidated. The authors of the present study speculate that a sufficient decrease in the appositional force between the eyelids and ocular surface lead to a chronic abnormal tear film interface with inadequate wetting of the conjunctival surface and cornea as one of the possible mechanisms of chronic injury.

Recently, Culbertson and Tseng have postulated a variety of mechanisms of injury including low nocturnal oxygen perfusion, combined with pressure induced ischaemia in the tarsal plate, and possible reperfusion damage to the tarsus secondary to free radical release and chronic polymorphonuclear leucocyte reaction. These multiple hypotheses, as to the mechanism of injury, remain to be proved.

An especially interesting subgroup of disorders related to lax eyelids is the 'eyelid imbrication syndrome' as recently described by Karesh and associates.1 In this syndrome, a horizontally lengthened, lax upper eyelid is observed to override or 'imbricate' over the lower eyelid margin. This results in chronic irritation of the upper lid margin tarsal conjunctiva because of chronic rubbing against the lower lid margin lashes. The tarsal plate in patients with eyelid imbrication has much better 'tone' than the extreme rubbery loss of tarsal tone seen in patients with classic floppy lid syndrome. These patients present with symptoms of chronic conjunctival discharge, excess tearing, photophobia, and generalised chronic ocular surface irritation refractory to medical management.

In one lone sentence in their discussion, Van den Bosch and Lemij state, 'In our experience, many elderly patients have a similar eyelid laxity, without any symptoms'. I would emphasise this statement to the readership in that most elderly patients with lax eyelids are asymptomatic. This is important to realise, lest we declare a surgical 'open season' on anyone with loose eyelids. It is the subset of patients with chronic ocular surface irritation for which a rational approach to management is necessary. From the standpoint of a logical evaluation and treatment protocol, I would first emphasise to the readership not to overlook the basics, or indeed the importance of medical management. The most important, basic medical approach to these patients is to discontinue the use of all ophthalmic medicines so as to best evaluate the true natural presentation of their problem.

As demonstrated in the present and previous reports,1 the well chosen patient with chronic ocular irritation associated with lax eyelids, will show excellent symptomatic relief following surgical eyelid tightening, particularly patients refractory to medical management only.

The standard upper lid tightening procedure has been the excision of a full thickness upper lid pentagonal taken from the middle/lateral third of the upper eyelid. The surgeon should determine, in each individual case, the amount of upper lid resection necessary to stabilise the problem without causing any adverse surgical side effects. The 'usual' amount of upper lid resection necessary is a pentagonal of 10–12 mm in width.

In the report in this journal, the upper lid (and lower lid) tightening effect was often achieved by reinserting the lateral canthal ligament to its fixation point at the lateral orbital tubercle. This technique would rely on a suture refixation of the lateral canthal angle to correct the existing lateral canthal dehiscence, and to tighten the upper and lower eyelids. Under many situations, excision of the actual upper and lower lid margin, at the lateral canthus, may provide an equally secure result.

A satisfactory surgical outcome may also be complicated by simultaneous ptosis surgery. In many circumstances, it may be more prudent to perform the ptosis surgery as a second procedure since the upper lid ptosis may improve, resolve, or
both following treatment of the chronic conjunctivitis and irritation associated with eyelid laxity.

Even under optimal situations, the quantitation of ptosis repair surgery is challenging. In patients with lax eyelids, chronic conjunctivitis, and chronic irritation the quantitation of the ptosis repair surgery can be made very inaccurate. Finally, in certain patients with severe upper eyelid laxity and severe upper eyelid ptosis, it is important to note the position of the floppy upper eyelid tarsal plate.

The present report is a thought provoking presentation of patients with chronic ocular irritation associated with eyelid laxity. Just as so called classic floppy eyelid syndrome and more recently eyelid imbrication may represent specific subtypes of patients’ problems associated with eyelid laxity, it is important to recognise the different subtypes of chronic ocular irritation secondary to eyelid laxity.


The minified Goldmann applanation tonometer

For over a century we have witnessed much ingenious exploitation of physical principles in the development of tonometry. Maklakoff is credited with inventing the first practical tonometer.1 Fick’s refinement in 1888 laid the mathematical foundation that led to Hans Goldmann’s low displacement applanation tonometer,2 which remains an enduring standard. Nevertheless, the physical properties of the cornea are major factors affecting the accuracy of tonometry, the post-keratoplasty eye presenting unique difficulty. The paper on the minified Goldmann applanation tonometer (MGAT) in this issue of the journal addresses the problem of an application area, limited by sutures and the graft interface. The authors have produced an elegant study, worthy of the Goldmann tradition, and offer a simple solution: ‘... for that smaller parking space, use a smaller car.’

Glaucoma is one of the commonest reasons, after retraction, for graft failure, with one study reporting that up to one third of such failures are associated with increased intraocular pressure.3 Until the work of Irvine and Kaufman it was virtually impossible to measure accurately intraocular pressure in patients with corneal disease.4 Intraocular pressure measurement is not only important in protecting graft and optic nerve, but also in maintaining wound integrity and in detecting hypotony. Interestingly, an eloquent review by Kaufman implies that 25 years ago, in cases of corneal disease, the only way for most clinicians to tell if corneal oedema was secondary to raised intraocular pressure was by the response to treatment with Diamox.5

The Schiotz tonometer, which can double intraocular pressure on indentation, is not only unreliable, but grossly misleading. The point of application of tonometry is so simple to use, permits continuous recording and, importantly, provides an objective reading, but has a tendency to read high at low intraocular pressure and low at high intraocular pressure. There is reason to doubt that this instrument functions as an applanation tonometer, that it follows the Imbert-Fick law, and that it can be used in any position without a gravity correction.6 Neither has the Tono-Pen lived up to its early promise. Portable, compact, easy to calibrate and usable in varied positions, its disposable tip lowers infection risk and its digital readout minimises user bias. By virtue of its small contact diameter (1.5 mm) Rootman et al recommended its use for irregular corneas.7 However, Mincekler et al found significant discrepancy attributable to corneal disorder.8 More damningly, Geyer et al found that the Tono-Pen consistently overestimated intraocular pressure in normal and post-keratoplasty eyes, and did so in an unpredictable manner that was not amenable to mathematical correction.9

The standard Goldmann tonometer is versatile, robust, accurate, and relatively inexpensive; in my experience it tends to age better than its more sophisticated electronic alternatives. By the simple expedient of reducing tip diameter from 7 mm to 4 mm, Menage et al have extended the applications of Goldmann’s tonometer. The weight and location of plastic lost by minification are such that calibration characteristics are unchanged and the MGAT provides measurements that are virtually identical to the standard Goldmann tonometer.10

The MGAT is not easy to use. The perceived mires are reduced to two overlapping quarter circles, the alignment of which can present difficulty for the initiate. None the less, this is a useful addition to our diagnostic armoury, and results show that it is at least as accurate as its alternatives. One has the impression, scanning the authors’ data, that results would be even more compelling (in favour of the MGAT) had they employed a concordance rather than correlation approach in their statistical analysis. The need to de-epithelialise the cornea in some cases was a necessary expedient given the frequency of measurement and does not detract from the value of this study. This study, however, does not solve the problem of intraocular pressure measurement in the presence of epithelial oedema (akin to anaplaating ice cream) and all methods of tonometry remain subject to error in eyes with significant corneal scarring and irregularity.

Suitable for applications other than the post-keratoplasty eye, the MGAT will be useful for infants, patients with tarsorrhaphy, congenital or acquired lid abnormality, blepharospasm, cicatrising disease, nyctagmus, etc. Laboratory workers should also welcome this tool for a variety of animals with narrow palpebral apertures.

The MGAT solves one of the problems of tonometry in graft patients, that of limited space for application. Considerably less expensive, readily accessible to most units and potentially more versatile than its more complex alternatives, it also has a large number of clinical applications. However, the ultimate goal of precise, non-invasive, continuous measurement for patients remains elusive. Perhaps, with improving technology, the next decade will present us with a telemetric solution.


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Br J Ophthalmol 1994 78: 663-664
doi: 10.1136/bjo.78.9.663

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