It is important to consider the origin of the astigmatism when planning cataract surgery in order to optimise the visual outcome. The greater component of astigmatism usually resides in the cornea,[6,7] and nomograms are readily available[8,9] for correction of corneal astigmatism via arcuate incisions that may be placed on meridian (steep) and be coincident or non-coincident with the meridian of the primary incision or side ports. However, it is essential that the astigmatism is fully evaluated with corneal topography, as a large difference between the cylindrical component of the manifest refraction and corneal astigmatism demonstrated on topography implies the presence of significant lenticular astigmatism. The magnitude of the lenticular astigmatism may be derived by vector analysis[10] from the total minus the corneal astigmatic components. Ninomiya *et al*[11] describe an elegant method for indirect evaluation of lenticular astigmatism using wavefront analysis, using traditional methods SIRC Koch and Holloday.

Patients may also present for cataract surgery with pre-existing conditions that place them at high risk for lenticular astigmatism. Tilted disc syndrome (TDS) is present in 1–3% of the population[12] and is readily diagnosed at a pre-operative examination. Gündüz *et al* found lenticular astigmatism to be significantly more common and of greater magnitude in patients with TDS than in controls matched by age and refractive error and suggested that tilting of the crystalline lens was responsible for induced astigmatism.

Whether the lenticular astigmatism is due to the morphology or position of the crystalline lens, it will be eliminated by cataract surgery. This must therefore always be considered when pre-operative assessment indicates the presence of astigmatism. The corneal and lenticular components must be clearly identified. This will also present a common cause of refractive surprise 'unmasking of corneal astigmatism' whereby pre-existing corneal astigmatism that is neutralised by opposite lenticular astigmatism suddenly emerges as a refractive astigmatic surprise. This will allow a bespoke surgical plan to be formulated in order to address it in an optimal manner and avoid leaving or even creating significant residual astigmatic error.

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Sir,

Intraoperative floppy-iris syndrome associated with chronic use of chlorpromazine

Intraoperative floppy-iris syndrome (IFIS) during phacoemulsification has been described in patients who are using α_1-adrenoceptor (AR)-blocking agents such as tamsulosin, terazosin, alfuzosin, and doxazosin.[1] Recently, labetolol and mianserin which are antihypertensive and antidepressant agents, respectively have also been reported to be associated with this syndrome.[2,3] We report a new case in which IFIS developed in a patient owing to chronic use of chlorpromazine.

A 48-year-old man with schizophrenia had bilateral nuclear cataract. Despite standard pharmacologic dilation (topical cyclopentolate 1%, tropicamide 1%, phenylephrine 10%) for fundus examination, the patient's pupils did not dilate well. He had no posterior synechia, pseudoexfoliation, history of miotic use, or diabetes mellitus. His medical history revealed that he has been using chlorpromazine 50–200 mg (average 100 mg) for 29 years for schizophrenia.

Right cataract operation was planned under general anaesthesia. A relatively small capsulorhexis was performed. During the phacoemulsification procedure, characteristics of IFIS developed: there was a flaccid iris stroma, which undulated, billowed, and prolapsed to the main and side incisions, and progressive miosis

occurred. Miosis did not respond to intracameral adrenaline irrigation. Although we performed nucleus emulsification successfully, posterior capsule rupture developed during cortical cleaning. Vitreous loss was not present. A foldable intraocular lens was implanted into the sulcus.

Despite well-documented adverse effects, and the advent of a new generation antipsychotic drugs, chlorpromazine remains one of the most commonly used and inexpensive treatments for people with schizophrenia.[4] It has antagonistic effects on α_1ARs, serotonin 5-HT_2 receptors, and dopamine D_1 and D_2 receptors. Its α_1AR-blocking activity is very prominent, and is responsible for some of the side effects including orthostatic hypotension, high-resting pulse rates, and impotence.[5]

We believe that the most likely cause for IFIS in this patient was chronic chlorpromazine use. α_1ARs predominate in sympathetically mediated iris dilator muscle contraction resulting in mydriasis. Long-term blockade of these receptors by chlorpromazine may prevent mydriasis and result in dilator muscle tone loss. We are not sure whether the occurrence of IFIS would be prevented or not if chlorpromazine had been stopped before surgery. Disuse atrophy may have developed in this patient because of long-term use of an α_1AR antagonist. Anyway, we suggest that discontinuation of chlorpromazine might be a wise course of action before cataract surgery to avoid the possibility of IFIS.

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Sir,

Vitreous and aqueous penetration of orally administered moxifloxacin in humans

We would like to comment on the above paper by Vedantham *et al*.[1] The authors state that 'the spectrum of coverage does not appropriately encompass the most common causative organisms in endophthalmitis, especially *Staphylococcus epidermidis*'. This is based on the assumption that the MIC_{90} of moxifloxacin for *Staph. epidermidis* is 2 μg/ml (Table 1).[2] This is contrary to other publications on MIC_{90} tables for moxifloxacin.[3–5] Published susceptibility tables for moxifloxacin include pathogens isolated from systemic infections and therefore are not representative of endophthalmitis pathogen susceptibilities. When these were examined, the great majority of *Staphylococci* were susceptible to moxifloxacin.[6,7] The authors also state that they decided to test moxifloxacin because of its low MICs against pathogens implicated in endophthalmitis (including Gram-positive bacteria), which is contrary to the MICs in Table 1 that they use for their analysis.

Of notice also are the wide variations of moxifloxacin concentrations in aqueous and vitreous samples in their series. These are contrary to other studies,[8–11] where moxifloxacin achieved very steady concentrations for at least 12 h after oral administration. Their serum concentrations also are variable and in a few cases extremely low, which is not in accordance with bibliography on serum moxifloxacin levels,[3–5] although the usual dose is the same as the one used by the authors (400 mg OD). The authors attribute (in part) low levels of moxifloxacin to the fact that only one dose of 400 mg was administered to 'simulate the clinical scenario'. In general, whenever intraocular penetration of a systemically administered antibiotic is to be determined, higher loading doses are used and in the case of moxifloxacin no hazardous side effects were noted previously.[8–11]

In conclusion, we mention that the results of this paper should be interpreted with caution and that there are numerous data suggesting that moxifloxacin may be a very useful systemic addition in endophthalmitis treatment.

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