Accuracy in strabismus surgery

Depending on the type of strabismus, the surgical goal may vary between accurate alignment, undercorrection, or overcorrection.1 The amount of under or overcorrection compatible with a good anatomical and functional result is fairly specific.1,2 The long term sensory and motor result depends on a number of factors.3 Most importantly the eyes need to be placed in alignment if the potential for fusion is to be best realised. When indicated, achieving accurate alignment with strabismus surgery is critical to success.

The prospective multicentre study of the accuracy of surgery for horizontal strabismus that appears in this issue of the journal (p 10) sets out to examine the current level of accuracy representative of the strabismus practice patterns in the United Kingdom. The definition of accuracy is expressed with reference to a preoperative surgical goal and is perhaps a better measure than the usual 'within 5–10 prism dioptries of straight.' This definition should be well received. Using this definition (within 10 prism dioptries of the surgical goal), success was obtained in over 90% of cases. Despite the short term follow up, this might appear to be a good result, and a standard against which other surgeons might compare their own results. However, does the evidence in this paper justify the conclusion that there is no difference in the outcome of surgery performed by a specialist in strabismus surgery and a general ophthalmic surgeon?

There are a number of reasons why this conclusion should be accepted with caution. It is true that Student's $t$ test used to compare accuracy of alignment achieved by specialists with non-specialists showed no difference. However, to be convinced that there is no difference one must consider the power of the study to detect a difference.

Were there sufficient numbers in each group studied? For example, considering grade 1 alignment in the esotrope group, there was an inaccuracy rate of 36% among specialists. Therefore, to detect a 20% difference between specialists and non-specialists would require approximately 925 patients in each group.4 To detect even a 50% difference would require at least 133 in each arm of the study.

If we consider grade 1 and 2 together the rate of inaccurate alignment among specialists is 8% and therefore the number of patients needed to detect a 20% inaccuracy would be about 4300 in each group. The logistics of such a study are clearly daunting, and would require a much larger database than may be available to the limited number of centres that contributed to this study.

Although there are strong reasons not to accept the conclusions of the authors, their paper is important in that it draws attention to the question of the accuracy of strabismus surgery performed by general ophthalmologists in comparison with specialists. This is not only important in terms of audit but also in terms of deciding what should be core surgery for the general ophthalmologist and is applicable to other surgical subspecialty disciplines.

It must be a concern of ophthalmologists and the professional colleges of ophthalmology to define what is core surgery for the general ophthalmologist and what cases should most appropriately be referred initially to those whose subspecialty training fits them to manage the more complicated cases.

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4 Javitt JC. When does the failure to find a difference mean that there is none? Arch Ophthalmol 1989; 107: 1034–40.

Latanoprost – a promising new glaucoma drug

Our knowledge of the role of endogenous prostaglandins and other eicosanoids belonging to the families of autacoids in ocular inflammation has progressed significantly over the years. A possible therapeutic role for prostaglandins in the reduction of intraocular pressure as suggested by Bito on the basis of animal experiments1 has greatly interested ocular pharmacologists.

In the last decade prostaglandin F2α (PGF2α) and various esters of PGF2α have been shown to reduce the intraocular pressure significantly in normal volunteers and patients with glaucoma or ocular hypertension when applied topically.2–10 However, PGF2α and its simple esters cause significant ocular side effects, such as conjunctival hyperaemia, which preclude their routine clinical use. This problem may now have been circumvented.

Recent studies11–18 with a new group of phenyl substituted prostaglandin F2α analogues: 13, 14-dihydro-17-phenyl-18, 19, 20-trinor-prostaglandin F2α-isopropylester (latanoprost, PhXA41) and its epimeric mixture PhXA34 have established their potent ocular hypotensive effect in patients with primary open angle glaucoma or ocular hypertension and the side effects were found to be minimal – that is, mild conjunctival hyperaemia in some patients on short term treatment.

In this issue of the British Journal of Ophthalmology Alm and colleagues report (p 12) the results of a well designed long term multicentre trial with a two dose regimen of latanoprost plus timolol in patients whose glaucoma was not adequately controlled by timolol alone. Addition of latanoprost 0-006% given either once or twice for 3 months resulted in marked and sustained reduction of intraocular pressure by about 30–33% throughout the treatment period without any significant side effects. Mild to moderate conjunctival hyperaemia was noticed in about a third of the patients and once a day administration of latanoprost was found to be as effective as latanoprost given twice a day. Another recent study19 with latanoprost has also shown similar levels of sustained intraocular pressure reduction with once a day administration of latanoprost 0-006% which may prove to be a significant factor in improving compliance
by patients on long term medical therapy for glaucoma.

Though the exact mechanism of the ocular hypotensive effect of latanoprost is still not clear, a recent study by Toris et al.\(^4\) indicates latanoprost decreases intraocular pressure in humans by increasing uveoscleral outflow without significantly affecting other variables of aqueous humour dynamics. Logically, such a mode of action would make the drug's effects additive to those of drugs which are aqueous humour suppressants. The present study of Alm et al. has established conclusively that this is indeed the case.

Can latanoprost prove to be a significant advance in the treatment of glaucoma? The published data would suggest that it is likely to be a prospective candidate for such a consideration. However, many important questions still remain unanswered. What are the long term effects of a chronic increase in the outflow of aqueous humour through the uveoscleral pathway on the eye? Is there any significant effect of the drug on ocular blood flow especially chorioretal circulation? Conjunctival hyperaemia with the use of latanoprost would suggest a possible effect on episcleral venous pressure. Could it also increase iridociliary vascular and/or epithelial permeability? The role of the receptors involved is yet to be established. A multicentre long term trial with latanoprost in a larger number of patients would provide answers to many important questions and also should be able to establish its efficacy and safety for long term clinical use. An ideal drug for glaucoma should be able to control the intraocular pressure significantly for longer periods with minimal side effects and also preserve the visual function primarily by improving the chorioretal circulation. Can these conditions be fulfilled by latanoprost and possibly other analogues of prostaglandins? There is still some way to go before such a drug is available. However, latanoprost has the potential to become a useful ocular hypotensive drug for many patients.

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