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Editorial

How safe are intraocular lenses?

The general idea of the monthly editorial is to highlight a paper or papers. The choice of a paper on which to comment is not based on merit nor is it made as the result of putting the titles in a hat and drawing one out. The choice is rather a purely idiosyncratic one, the urge to comment on something that interests one personally, the desire to emphasise something which it is believed is not widely enough known, or the challenge of trying to understand something outside one's own special interests, and hence delving into literature not usually consulted.

This month's editorial is about the paper by Furuse and colleagues, which interested me, not because it reported world shattering discoveries but strangely enough because it discovered nothing. This is not supposed to be a disparaging remark; the paper is carefully researched and well presented but its results are negative. The negativity, however, refers to the stated object of the investigation, which was to discover if the corneal endothelium of diabetics fared worse than that of non-diabetics after intraocular lens (IOL) implantation. The investigation was successfully completed, and the result was that diabetes appeared to make no difference to the fate of the endothelium.

One might be tempted to conclude that no further information was likely to be gleaned from the paper, categorising it in the diabetic file as a negative finding. Such a dismissal would, however, be a mistake. Hidden in the paper is some information which in years to come could prove to be of importance. Those of us who saw the early results of IOLs were at first uneasy about the unacceptable number of cases of bullous keratopathy which occurred, leading some distinguished surgeons to abandon the operation and others not even to start it. We also know how, following the discovery of the vital role of the endothelium, bullous keratopathy became much less common, so that now the average surgeon and certainly the average patient normally take little notice of the possibility. We should ask ourselves if this is wise. Could it be that we are building up to a massive epidemic of bullous keratopathy in the future? Do we know enough about the natural history of the corneal endothelium to be confident that all is well and that we are safe to carry on implanting IOLs in all and sundry without a care in the world?

The present paper does not set out to evaluate the long-term prospects for endothelial survival after IOL implantation but only to compare diabetics with non-diabetics. Although the authors are encouraging about the probability of diabetes not being a risk factor for endothelial cell loss, their findings are not completely reassuring in this regard.

There does appear to be a tendency for the diabetics to do a little worse, though the results do not achieve statistical significance. However, it must be pointed out that the numbers in the diabetic populations studied are rather small, arousing the suspicion that with larger numbers the results might not have been so reassuring. But this is pure speculation, and it would not be fair to question the present conclusions, which are justifiably made.

Leaving aside the question of the diabetics for the moment, we may consider the incidental finding that neither the diabetics nor the normal persons showed continued cell loss. This certainly gives grounds for a certain degree of optimism for the prospects of the world's huge and ever growing army of pseudophakes.

The authors point out that there have been mixed opinions on the chances of the endothelium of pseudophakes giving trouble in the distant future, some authors being optimistic but others pessimistic. Some of the reported studies are on the natural history of the endothelium, without pseudophakia, and even these do not altogether agree. For example, Laule and others¹ calculated that at the rate of endothelial cell loss found by them there would be no cells remaining by the age of 100 years, a daunting prospect for aspiring centenarians. But Wilson and Roper-Hall in 1982² found that, though there was a decline in the endothelial cell count up to the age of 50 years, there was no appreciable difference thereafter. The same authors also found³ that approximately 17% of endothelial cells were lost after uncomplicated intracapsular cataract extraction with or without an IOL, but (not surprisingly) endothelial damage was far more serious if prolonged endothelial touch occurred. (The authors noted that the then little tried substance sodium hyaluronate showed promise.)

Substantial improvements in technique have been introduced in the last few years, not least the rapidly increasing use of sodium hyaluronate and other barrier substances, and it is becoming increasingly likely that the fears felt by many at the beginning of the decade that an epidemic of bullous keratopathy was on the way will prove to be unfounded. Certainly the present paper adds to the weight of evidence against the chances of such an epidemic.

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- 1 Laule A, Cable MK, Hoffman CE, Hanna C. Endothelial cell population changes of human cornea during life. *Arch Ophthalmol* 1978; **96**: 2031-5.
- 2 Wilson RS, Roper-Hall MJ. Effect of age on the endothelial cell count in the normal eye. *Br J Ophthalmol* 1982; **66**: 513-5.
- 3 Roper-Hall MJ, Wilson RS. Reduction in endothelial cell density following cataract extraction and intraocular lens implantation. *Br J Ophthalmol* 1982; **66**: 516-7.