Diabetic retinopathy and cataract surgery

Two papers in the current issue, one from Pollack, Dotan, and Oliver of Israel and the other from Cunliffe and colleagues of Cambridge, deal with the effects of cataract surgery on diabetic retinopathy and its complications. All ophthalmologists are aware that there are possible extra hazards when cataract extraction has to be done in the presence of diabetic retinopathy, but in view of the fact that about 10% of cataract patients have diabetes, and that most of those whose diabetes is of fairly long duration are also likely to have retinopathy, it is surprising that in the ordinary course of events serious postoperative complications of the sort one might fear seem to happen so seldom. Galloping rubeosis and/or accelerated retinopathy ought to be almost everyday occurrences in busy cataract programmes, yet they do not appear to be.

There is no doubt that the postoperative visual prognosis of diabetics with retinopathy is worse than of those without, but that is only what one would expect. Perhaps a more important consideration is whether the retinopathy is itself exacerbated by the patient being rendered aphakic. If microvascular ischaemia is the primary event in diabetic retinopathy, with neo-vascularisation of retina and eventually iris to follow, what would be the most likely effect of aphakia? One would have thought that the freer movement of fluids between anterior and posterior segments, with a general tendency towards the anterior part of the eye, would shift the neovascularising stimulus (whatever it is) forwards: Rubeosis ought to be more likely and retinal neo-vascularisation less likely. There is indeed some evidence that the risk of rubeosis is increased by extraction, but the evidence for a relative improvement in retinal neovascularisation does not seem to be forthcoming, and indeed one of the papers in the present issue indicates the exact opposite. It is even suggested that the increase of neovascularisation which is said to occur might be directly due to the absence of the lens, which is thought normally to contribute a component to the anti side of the pro-anti neovascularisation balance thought to exist in the normal eye. That there is evidence for an antineovascularising factor in the lens is not disputed (the matter is referenced in the paper by Pollack et al), but it is only one component in what is probably a complex interplay of factors and seems unlikely to play a major role in view of the large numbers of diabetic cataracts which are extracted without trouble.

One would not be surprised if factors tending to discourage vascular endothelial cell proliferation were not to be found in most normally avascular tissues of the body as part of their own defences against vascularisation, and so far as the eye is concerned this would mean the lens, the vitreous, and the cornea. Antineovascularising substances have been suggested in the first two but not I believe in the third. It is of some interest, however, that some of the original work involved in the search for neovascularising substances was carried out in corneal pockets, and one wonders whether, if the cornea itself is not such a passive vehicle for the test tissues and substances as researchers assumed, the results of such experiments may have been modified by the substrate in which they were performed.

Kissun and Garner, for example, failed to induce neovascularisation in experimental corneal tunnels by implants of extracts of ischaemic kitten retina. They themselves pointed out that 'local factors, such as may well be involved in using the cornea as a substrate,' might be better avoided by some other form of testing being devised. So far as laboratory work is concerned, tissue culture experiments with media conditioned by various tissue extracts and other substances would appear to be less susceptible to confounding influences than in-vivo experiments on the cornea, and we await with interest further work on this fundamental aspect of ocular pathology. Publication of the recent Cambridge symposium, which dealt with some of the issues involved, may throw some more light on the matter.

Both papers in this issue offer some consolation for clinicians, since they provide evidence, one intentionally and the other involuntarily, that if a cataract extraction does have to be done for a patient with retinopathy the implantation of an intraocular lens is unlikely to constitute an additional hazard.

REDMOND SMITH

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R Smith

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