Diabetes and retinal function

The article in this issue entitled ‘Localised blood-retinal barrier leakage and retinal light sensitivity in diabetic retinopathy’ provides food for thought. The authors, Bek and Lund-Andersen, studied the retinal sensitivity to light of small areas of retina where diabetic damage had been positively identified by fluorescein angiography. To their surprise they were unable to demonstrate failure of retinal function even where severe damage, giving rise to gross breakdown of the blood-retinal barrier, could be shown. Aggregations of exudates produced scotomata but only where there was enough to mask the underlying retina.

The authors’ main conclusion, on the basis of the techniques used, is that breakdown of the blood-retinal barrier is an earlier event than disturbance of neurosensory function. But it is the techniques used of which one has to take careful note before assuming that the diabetic process does not affect neural function via the vascular system but via some other subtle, as yet undiscovered biochemical mechanism. For the question has to be asked: in a sensory organ how can aberrations in performance due to true abnormalities of function be accurately assessed? From our experiences with correlating optic nerve and disc damage in glaucoma with visual fields it is obvious that deficit in sensory ‘performance’ tends to lag far behind clinical and even histopathological evidence of nerve damage. Linking optic nerve changes with visual field changes is not very different from attempting the same exercise with the retina itself. While accepting the authors’ conclusions subject to the proviso they wisely insist upon, one suspects that the real problem may lie not so much in a mysterious absence of abnormality where abnormality would be expected but rather in an inability to demonstrate abnormality by the means at our disposal. One has to accept the fact that the eye’s sensory performance is so fantastically sensitive that it can appear to be relatively unscathed even in the face of substantial damage. I well remember a dramatic demonstration of retinal sensitivity by Rushton at a lecture he gave some years ago in University College. He dropped a piece of blackboard chalk on the desk and announced that the energy liberated by the impact would have been enough to stimulate (adequately) all the cones of all the men who had ever lived and all the women too.

A glance through the literature on diabetic retinopathy reveals that, for the last few years at least, virtually every paper has been concerned with the vascular aspects or at least their immediate sequelae. In attempting to glean some sort of evidence from the literature as to the likelihood of there being mechanisms involved in diabetic retinal malfunction independent of vascular changes one is impressed by the small number of papers to be found. Most of the papers in this category have tended to be on rather high-powered experimental pathology. There have, for example, been several studies on experimental diabetes produced by injection of streptozotocin into rats. In one of these a reduction in thickness in the outer nuclear layer of the retina was induced by exposure to light,1 and in another2 the effects of aldose reductase inhibition of the retina was studied. Improvement in electrophysiological test results was found, but it was thought that this might have been associated with changes in the thickness of the capillary basement membrane. However, MacGregor et al3 found evidence in experimentally diabetic rabbits that diabetes affects the metabolism of retinal structures independently of vascular disease. There are papers on colour vision in diabetes,4 contrast sensitivity,5 macular recovery time (nyctometry), and visually evoked responses (VER),6 though in the last paper the degree of effect on the VER seemed to depend on the amount of vascular damage. But apart from these virtually every other paper is about anomalies in the vascular system, ranging from the most minute and esoteric details of the biochemistry of the capillary wall to the heroic surgery needed in advanced cases of proliferative retinopathy.

In summary, therefore, the more you look through the literature, the more unconvinced you are of anything other than vascular abnormalities causing visual damage in diabetic retinopathy. One intriguing point appeared in a paper by Bresnick and coworkers.7 It appears that the ‘capillary free zone’ round the fovea is larger in diabetics than in controls. A corollary to this is that areas of capillary closure do not give rise to fluorescein leakage. Can one therefore postulate that there might be areas of relative underperfusion in diabetics which could give rise to visual deficit without showing fluorescein leakage? The snap with this theory, however, is that not only did Bek and Lund-Andersen not find areas of visual deficit in zones of leakage, but they did not find them in other zones either.

REDMOND SMITH