Diabetic retinopathy; a new clue in the puzzle

The last sentence but one in the article by Rimmer, Fleming, and Kohner in this month’s issue reads, ‘The phenomenon of hypoxic viscosity could be important because it might present a new target for pharmacological attack.’ This had a familiar ring, and I managed after some thought to recall why. I had used a somewhat similar phrase myself when writing about the possible anoxic basis for ruberosis in neovascular glaucoma: ‘When pathology is understood, rational treatment may follow’. Unfortunately the word ‘may’ is especially important in the foregoing sentence. One of the most frustrating things in medicine is to understand the pathology and not be able to do anything about it so far as an individual patient is concerned. Many of the inherited diseases come into this category. Killing off affected fetuses may be one way of pre-empting the problem, but it doesn’t help a living patient. I see that I am wandering from my subject.

Does the paper from Rimmer and colleagues get us any further in our quest for control of diabetic retinopathy? As the paper reminds us, retinopathy is now the commonest cause of blindness in people of working age in the UK and USA. Present treatment is directed rather at the results of the retinopathy (neovascularisation and haemorrhage), as indeed is the treatment for ruberosis and sickle cell retinopathy.

It is of interest that it took the retinal hypoxic theory of neovascularisation, first proposed by Michaelson1 in 1948, more than 10 years to yield any fruit in the form of treatment. The first retinal ablations performed for the specific purpose of treating remote neovascularisation were not reported until 1961.1 One certainly hopes that, if other workers confirm the claim of Rimmer and colleagues to have established a working hypothesis to tie together their previous observation1 of slowing of the retinal blood flow in patients with retinopathy (by entoptic blue light studies), this will lead rapidly to advances in treatment of this damaging condition.

For a clinician such as myself to speculate on what might be possible in the realm of what I believe goes under the mysterious title of rheology would be unwise. Indeed the very writing of this editorial makes me feel vulnerable to the scorn of experts on the subject, and I would not have written it if the expert we had lined up to do it had not been unavoidably prevented from producing it at the last moment. Nevertheless even the ordinary clinician can see at once what a fantastic advance it might be were there to be developed a means of preventing the retinopathy rather than just trying to stave off its worst consequences, as at present. When one considers the numerous examples of the deleterious consequences of retarded blood flow which can be found in the various tissues of the body, from the simple example of varicose veins upwards, one fears that an answer to the problems possibly caused by retarded venous flow in the eye may be slow in coming. One may be left in the frustrating situation mentioned at the beginning of this editorial where the pathology is better understood but rational treatment has yet to be devised. This certainly would not detract from the merits of what might prove to be a genuine advance in the understanding of diabetic retinopathy on the part of the Hammersmith group.

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