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Editorial

Video laser ophthalmoscopy in diabetes

If the histopathological study of diabetic retinopathy is a relatively new activity following on the improved life expectancy of diabetics after the discovery of insulin, then the study of the vascular dynamics of the retina is even newer.

It has been known for many years that the capillary-free zone round retinal arterioles in diabetics tends to be enlarged, but the early knowledge was based on static post-mortem histological material where the retina was injected with Indian ink and mounted flat on glass.¹ Several ingenious attempts to demonstrate the dynamics of flow in the small vessels of the retina have been described, including a study of the entoptic impression of the paramacular circulation² and laser Doppler velocimetry,³ but neither of these methods gave anything approaching the graphic demonstration of the retinal capillary flow afforded by the combination of fluorescence angiography and the scanning laser ophthalmoscope as described by Arend and colleagues in the present issue. It appears that the resolution of the system is so good that two different densities of fluorescence are detectable within the blood column, the less fluorescent probably corresponding to rouleaux of red blood cells and the more fluorescent to plasma. Assuming that this is right, and there seems little reason to doubt it, one is then in a position to make direct measurements of the velocity of the rouleaux. It is also possible for measurements of the foveal avascular zone (FAZ) and the perifoveal intercapillary areas (PIA) (in effect the density of the capillary bed) to be estimated by this technique.

Over the years the ordinary ophthalmologist has been somewhat baffled by the complexity of some of the attempts to explain what is going on in diabetic retinopathy – the role of pericytes, the reason for thickening of the basement membrane, the origin of microaneurysms, the part played by variations in viscosity, irregularities in vascular calibre, arterial blood pressure, and so on. The impression gained by reading the early histopathological work was of a gradual accumulation of foci of occlusion of pre-venous capillaries with consequent backing up of the flow in the 'upstream' areas of the bed leading to slow flow, leakage of lipids through the capillary walls, and microaneurysm formation, the ischaemia associated with this gradual process of vascular disorganisation leading ultimately to neovascularisation.

Apart from the fact that the reason for the capillary occlusions was not obvious the rest of the scenario was reasonably convincing, so it was a great delight to read in the

current paper that the authors agree that the 'development of diabetic retinopathy is, at least in part, due to progressive capillary occlusion and decreasing capillary perfusion.'

In the blue light entoptic study of Rimmer *et al*² the principal finding was a tendency to slowing of velocity of the blood in the perifoveal capillaries both in comparison with controls and over a time interval of several years; in other words the slowing appeared to be progressive. Several possible reasons for the phenomenon were discussed in that paper, including alterations in retinal metabolic rate, a decrease in red cell deformability, and an increase in red cell aggregation, but none of those factors were measured at that time.

The current study confirms the reduction of the blood flow velocity but offers a further interesting piece of information. If the mean capillary velocity were to be reduced in comparison with normal velocity because of abnormalities in the vessels themselves, owing to variations in calibre in particular, one would have thought that the diabetics would be likely to show wider variations in velocity from site to site in the retina as compared with normal persons. However, this was not the case; the coefficient of variation of the velocity showed no significant differences between diabetic and normal subjects.

This would suggest that, whatever the cause of the slowing, it ought to be detectable in the blood itself rather than the capillary bed. Whether the sensitivity of this 'smart' system of looking at the circulating blood can be increased sufficiently to determine differences in the characteristics of the flowing blood itself, such as the size and shape of the rouleaux, the way they roll and tumble, and so on, will be a fascinating future prospect.

It is of interest that the present authors found reduction of velocity even in diabetics with little or no retinopathy, and there did not seem a strong correlation between greater slowing and greater retinopathy. This was in contrast to the rather dramatic increase of the PIA and FAZ with severer grades of retinopathy.

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- 1 Ashton NA. Injection of the retinal vascular system in the enucleated eye in diabetic retinopathy. *Br J Ophthalmol* 1950; 34: 38–41.
- 2 Rimmer T, Fallon TJ, Kohner EM. Long-term follow-up of retinal blood flow in diabetes using the blue light entoptic phenomenon. *Br J Ophthalmol* 1989; 73: 1–5.
- 3 Grunwald JE, Riva CE, Sinclair SH, Brucker AJ, Petrig BL. Laser Doppler velocimetry study of retinal circulation in diabetes mellitus. *Arch Ophthalmol* 1987; 104: 991–6.