

Ischaemia in vein occlusions

R. MAPSTONE

Department of Ophthalmology, University of Liverpool, and St. Paul's Eye Hospital, Liverpool

Paton, Rubinstein, and Smith (1964), Hayreh (1964), and Rabinowicz, Litman, and Michaelson (1968) suggest that the pathology in central vein occlusion is primarily an arterial ischaemia. If this in fact made a significant contribution to aetiology, then, in the presence of a unilateral vein occlusion, it would be reasonable to expect an ipsilateral ocular ischaemia. The purpose of this paper is to investigate the incidence and site of relative ocular ischaemia using a combined thermometric and angiographic approach.

Material and methods

Corneal and periorbital skin temperatures of 44 patients with vein occlusions (21 central and 23 branch veins) were measured with a bolometer (Mapstone, 1968a) over a period of up to 2 years from their first attendance at hospital.

In addition anterior segment angiograms were obtained from each patient using a Zeiss fundus camera and flash unit. 3 ml. 25 per cent. fluorescein were injected into an antecubital vein and photographs were taken at 5-second intervals for 90 seconds and again at 5- and 10-minute intervals.

Results

SURFACE TEMPERATURE

Of the 21 patients with central vein occlusions, all had a normal R-L temperature difference for medial forehead skin, whereas two had abnormal corneal temperature differences (normal = $\pm 0.4^\circ\text{C}$. for cornea and $\pm 0.6^\circ\text{C}$. for skin: Mapstone 1968c). Both of these had a rubeotic glaucoma with hyphaema. In one the latter was constant and the ipsilateral corneal temperature constantly greater as a result of the inflammation produced. In the other the cornea was significantly colder in the absence of hyphaema, but when blood was present it became hot (Fig. 1).

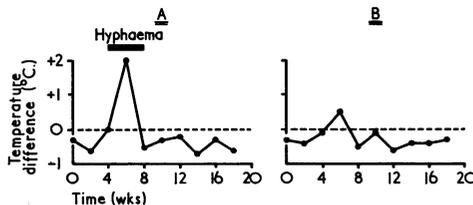


FIG. 1 *Temperature differences of patient with rubeotic glaucoma. Corneal differences are abnormally negative in the absence of hyphaema, the presence of blood producing an inflammatory reaction. Medial forehead differences are normal.*

Of the 23 patients with branch vein occlusions, three had an ipsilateral cold cornea (Fig. 2, opposite), and one had initially the thermal pattern of a carotid stenosis which subsequently became normal (Fig. 3, opposite).

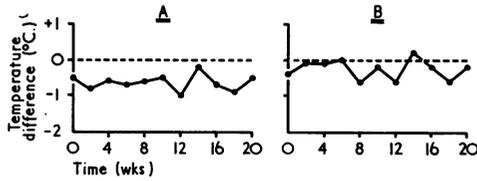


FIG. 2 Temperature differences of patient with an ischaemic anterior segment. Corneal temperature differences are abnormally negative, medial forehead differences normal

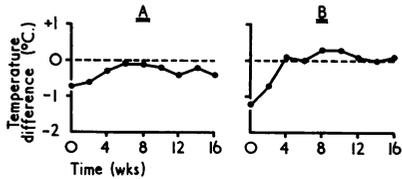


FIG. 3 Temperature differences of patient with thermal pattern of carotid stenosis, i.e. abnormally negative corneal and medial forehead temperature differences. This pattern was present for the first 2 weeks only.

ANTERIOR SEGMENT ANGIOGRAPHY

In eight of the 44 patients neovascularization was seen by slit-lamp examination. Fig. 4 illustrates the angiogram of one: (a) shows the fluorescein leak at 25 seconds and (b) the R-L difference of the same patient 5 minutes after injection. Five of the eight patients had rubeotic glaucoma and all had central vein occlusions. One had an ischaemic anterior segment (Fig. 1).

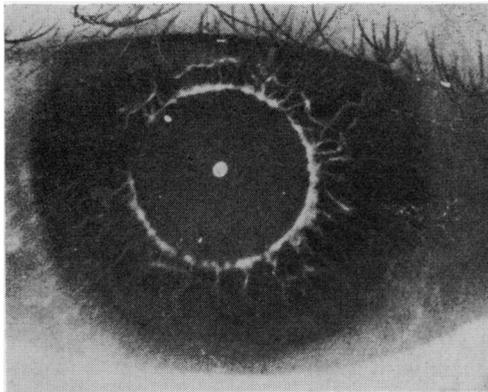


FIG. 4a Anterior segment angiogram of patient with early rubeosis iridis 25 seconds after fluorescein injection

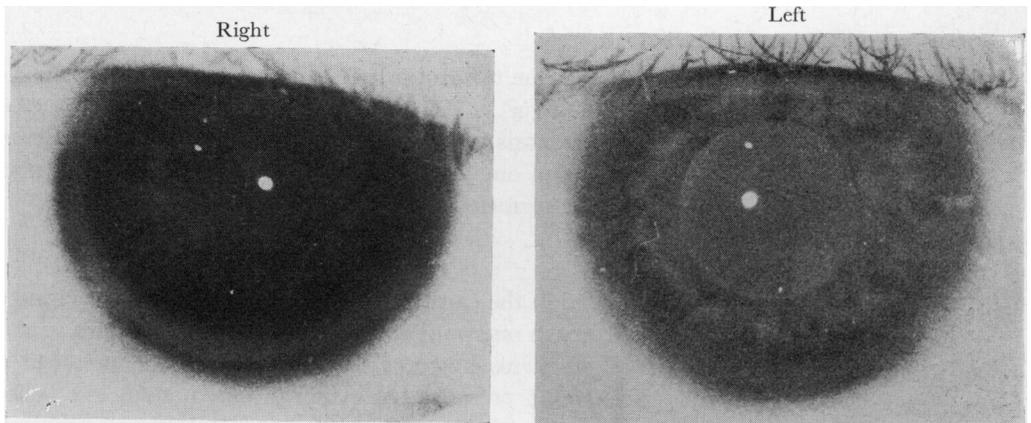


FIG. 4b Same patient as (a), showing R-L difference 5 minutes after injection.

Of the remaining 36 patients, although no neovascularization was visible, three had abnormal angiograms (in that present experience of approximately 150 angiograms indicates no fluorescein leak in the normal iris apart from the delineation of vascular patterns, whereas these patients had an abnormal blush at the pupillary margin: Fig. 5). None had an ischaemic anterior segment, but all three had central vein occlusions.

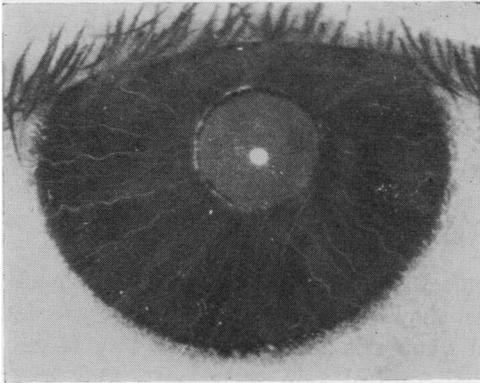


FIG. 5 *Angiogram of patient with normal slit-lamp appearances, showing fluorescein "blush" at pupillary margin 45 seconds after fluorescein injection*

Discussion

The probable site of a unilateral stenosis producing ischaemia can be inferred from surface temperature measurement, for the following reasons:

- (1) A lesion of the carotid vascular tree at any site before the origin of the ciliary arteries will produce a cold cornea and patch of medial forehead skin.
- (2) A cold cornea with normal skin temperatures is the result of a unilateral diminished ciliary artery supply or stenosis/destruction of vessels within the anterior uvea.
- (3) A lesion of the central retinal artery or its branches will leave the surface thermal patterns unaffected.

There are fallacies in this method, *e.g.* the development of R-L anastomoses, but the conclusions are probably valid in 70 to 80 per cent. of patients with stenosing carotid lesions, and in a higher percentage still with anterior uveal ischaemia (Wood, 1964, 1965; Austin and Sajid, 1966; Mapstone, 1968a, b, c, d, e).

Incidence of ischaemia

The incidence of ischaemia due to carotid lesions in this group of patients is very different from that found by Smith (1954). Using an ophthalmodynamometer, he found presumptive evidence of carotid stenosis in 30 per cent. of a group of 73 patients. Of the 44 patients in the present group, one had transient evidence of a carotid lesion (Fig. 3) and four of an isolated anterior segment ischaemia.

Site of ischaemia

The ischaemia was situated in the carotid system proximal to the branching of the ciliaries from the ophthalmic artery in one patient (Fig. 3), and in the ciliaries or anterior uvea in four. Surface thermal patterns, however, cannot indicate the presence or absence of posterior segment ischaemia; presumptive evidence can nonetheless be obtained from anterior segment angiograms and the presence of anterior uveal neovascularization: in the five patients mentioned above with ischaemic anterior segments, one had rubeosis iridis,

seven further patients had rubeosis, abnormal angiograms (Fig. 4), and normal thermal patterns, and three had an iris blush (and no diabetes—Cobb, 1968) but again normal thermal patterns. In eleven patients with neovascularization, therefore, one only had an anterior segment ischaemia, whilst in five with ischaemia one had new vessels. The inference is that anterior segment ischaemia and neovascularization are not causally related but rather that—as suggested by Smith (1954, 1955)—a vasoactive metabolite is released by the ischaemic retina and its anterior diffusion causes the vascular changes. Here, however, the retinal ischaemia is a consequence, and not a cause, of the vein occlusion.

In only five patients, therefore, is there evidence of relative extraretinal arterial ischaemia, and it is concluded that if ischaemia is a major causal factor of vein occlusions it must reside within the central retinal artery or its branches.

Summary

44 patients with vein occlusions were investigated using a combined angiographic and thermometric technique.

Five only had evidence of an extraretinal arterial ischaemia.

If ischaemia and vein occlusion are related, the arterial pathology lies within the central retinal artery or its branches.

I should like to thank the surgeons of St. Paul's Eye Hospital for access to their patients.

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