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## Editorial: Retinal vein occlusion and glaucoma

For a long time there has been a tradition among ophthalmologists that occlusion of the central retinal vein (CRVO) is more common in cases of primary glaucoma, even of a mild degree, than in the rest of the population. However, such cherished ideas do not always bear close examination.

Verhoeff<sup>1</sup> found CRVO in eight of 39 cases of glaucoma and 'endovasculitis at the level of the lamina cribrosa' in all the others. But these were cases of secondary glaucoma, and it was not until Foster Moore's important monograph in 1924<sup>2</sup> that primary glaucoma became implicated. It has to be remembered as well that many of Verhoeff's cases, even if they had originally been of primary glaucoma, were in any case terminal in that they came to enucleation, and most of us would accept central vein occlusion as a not unexpected finding in an eye with 'absolute glaucoma'.

Foster Moore's paper, which incidentally is extremely difficult to winkle out from most of the major ophthalmological libraries in London, is quite modest in its claims. He studied 31 cases of branch vein occlusion (BVO) and 31 of CRVO. His chief conclusions were that the principal cause of occlusion was arteriosclerosis associated with hypertension and this was more marked in BVO and CRVO. The

second conclusion, which is the one that has received the most attention, was that there was a possible link between primary glaucoma of 'an insidious type' and CRVO. He based this conclusion on the fact that well marked cupping of the optic disc was seen in only one of 31 cases of BVO but 13 of 31 cases of CRVO. There is really not much more evidence than this, but from this fairly shaky start the tradition has originated.

Salzmann<sup>3</sup> in 1933, in another painstaking monograph including a wealth of beautiful clinical and histological drawings, thought that the glaucoma might lead to venous occlusion by causing collapse of the veins. This is quite distinct from the type of glaucoma which results from rubeosis iridis due to retinal ischaemia,<sup>4</sup> though it was pointed out in 1955<sup>5</sup> that in some cases primary glaucoma could precede the CRVO-neovascular glaucoma sequence. In the latter paper the author described a number of illustrative cases and followed much the same lines of reasoning as Foster Moore, and pointed out the existence of primary glaucoma in the other eye. In three of the cases pathological specimens became available, but all they showed were changes typical of neovascular glaucoma. It has to be admitted that the paper, which was written by the author of this

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editorial, contained no data on the general medical condition of the patients.

The study by Cole, Dodson, and Hendeles in this issue makes it clear that we have to review currently held convictions about the relationship between pre-existing glaucoma and CRVO. These authors have been able to show by a thorough examination of 43 patients with glaucoma and 24 with ocular hypertension presenting with a retinal vein occlusion, and a similar examination of a matched group without glaucoma or ocular hypertension, that the prevalence of the 'other' risk factors for retinal vein occlusions were remarkably similar in the two groups.

It should be emphasised at this point that Cole, Dodson, and Hendeles are not writing solely about unequivocal CRVO but are also including patients with shunt vessels on the disc as assumed CRVO. In addition, cases of branch and hemisphere occlusion (BVO and HVO) are included. They admit in their introduction that the association between CRVO and chronic simple glaucoma is more strongly supported in the literature than BVO, though they state that HVO has been reported as 'unquestionably related'.

I must admit that I have never held the view that BVO was significantly associated, so it comes as no surprise that the risk factors for these cases were the same whether they were in the primary glaucoma group or not. But the fact that the same applied to the CRVO cases certainly came as a surprise. One is perhaps just a little uneasy about the ready acceptance of old bypass vessels as proving prior CRVO, but, even if we were to assume that this was incorrect, nevertheless it does not seem likely that it would have materially altered the conclusions, especially as there

were only six such cases out of the total of 43 'CRVOs'.

After an exhaustive discussion in which consideration is given to every conceivable aspect of the relationship between IOP, blood flow, plasma lipids, blood pressure, treatment for blood pressure, glaucoma and treatment for glaucoma, and optic disc conformation and much more the authors come to the modest conclusion that glaucoma or ocular hypertension may have a less prominent aetiological role in venous occlusion than has been widely believed. I have to say that I believe they are possibly being too modest and their findings may well be confirmed by further studies of this nature to have more or less demolished the primary glaucoma—venous occlusion aetiological relationship which so many of us have accepted for so long, without ensuring that there was adequate evidence. If only I had used a sphygmomanometer as well as a tonometer back in 1955!

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## Editorial: Postoperative eye padding

There was a time when an ophthalmic ward would be full of patients wearing not only one eye pad but two. At Moorfields they were fixed on by means of a brassiere-like contraption, but I have no doubt in other institutions there may have been other methods,

such as swathes of crepe bandage skilfully wrapped round the patient's head or even, in one surgeon's hands for certain patients, a plaster-of-Paris cast.<sup>1</sup> One of my earliest memories as a junior house-surgeon (the title of 'resident' was not in use in those