

Correspondence

Chronic ocular ischaemia

SIR, I was very much interested to read the paper by Sturrock and Mueller on 'Chronic ocular ischaemia'.¹ The authors,¹ commenting on the fundus changes seen in *one of their seven cases*, stated: 'The chance finding of blot haemorrhages distributed in the midperiphery of the retina in case 2 associated with a congested appearance of the veins which showed sludging probably indicates ischaemia of more gradual onset. This appearance, which has been misleadingly called venous stasis retinopathy,² must be distinguished from that due to central retinal vein thrombosis.³ Regrettably this same term, venous stasis retinopathy, was subsequently applied to certain cases of central retinal vein thrombosis,⁴ thereby compounding the confusion.'

These comments, unfortunately, raise an extremely important controversy which requires clarification. I would preface my comments by pointing out that over the past 10 years we have been prospectively conducting long-term studies on various types of ocular vascular disorders, and have collected data so far on about 450 patients with central retinal vein occlusion (CRVO) and about 200 patients with carotid artery disease (CAD). Based on our experimental and prospective clinical studies on CRVO I divided CRVO into ischaemic and non-ischaemic varieties.^{4,7} The non-ischaemic CRVO represents simply a stasis of retinal venous circulation and I designated it 'venous stasis retinopathy' (VSR). When I used the term 'VSR' I was fully aware that Kearns and Hollenhorst² had used the same term for what they felt was a specific type of retinopathy associated only with severe CAD. Kearns and Hollenhorst² postulated that 'VSR' was due to retinal hypoxia, secondary to CAD. Our detailed studies have indicated that the vast majority of the 'VSR' cases described by Kearns and Hollenhorst² and others were in fact cases of mild non-ischaemic CRVO; this was the basis of my using the term 'VSR' to describe non-ischaemic CRVO. I discussed this subject recently at length elsewhere.^{6,7} Kearns,³ to rebut my views on the subject, recently discussed in detail the differential diagnosis between these two types of retinopathy and claimed that his 'VSR' is very different from 'CRVO.' He stated, for example, that fluctuating blurred vision, a specific pattern of distribution of retinal haemorrhages, absence of optic disc oedema, and ocular hypotony are typical of 'VSR' due to CAD. Our detailed prospective long-term studies on about 350 cases with non-ischaemic CRVO, clearly revealed that these features *are seen* in non-ischaemic CRVO, with fluctuating blurred vision and ocular hypotony being very common findings.^{6,8}

In contrast to our studies on CRVO there is no systematic study on the natural history or other aspects of 'VSR' attributed to CAD²; all the reports making that claim are anecdotal, mostly based on a single examination or extremely short follow-up. Experimental carotid artery occlusion also failed to produce retinal changes typically seen in 'VSR'.^{9,10} Generalised severe atherosclerosis in carotid arteries produces CAD, in the coronary arteries it produces myocardial infarction, and in the central retinal artery it contributes to

secondary CRVO.⁶ Since most patients with CAD suffer from myocardial infarction,⁶ this in no way means that CAD and myocardial infarction are cause and effect; the same logic applies to CAD and VSR (mild non-ischaemic CRVO). Evidently all three conditions are simply independent manifestations of severe generalised atherosclerosis. Thus it can be concluded that CAD and VSR are *not* cause and effect but are two independent manifestations of severe generalised atherosclerosis.

While I question the validity of the concept and use of the term 'VSR' for the retinal changes being erroneously attributed to CAD, I have seen, in some patients with the latter disease, low retinal artery pressure, irregular calibre, and stasis of circulation in the major retinal vessels, and occasionally neovascularisation on the optic disc; however, *this fundus picture is very different from that seen in VSR*. I fully agree with Kearns³ that a more appropriate term is 'chronic ischaemic retinopathy' for the retinal changes secondary to severe CAD, and that their original term 'VSR' to describe the retinopathy 'was unfortunate' and misleading. Unfortunately, most of the cases reported as 'chronic ischaemic retinopathy' by Kearns and his colleagues and other authors are in fact mild non-ischaemic CRVO, and only a very small proportion of their cases represent true 'chronic ischaemic retinopathy.' Thus they have lumped non-ischaemic CRVO and 'chronic ischaemic retinopathy' under the title of 'VSR'—the two are very different types of retinopathy. Hence the present confusion.

The field of medicine is littered with myths, originally drawn from simple clinical impressions, anecdotal reports, personal biases, defective experiments, erroneous interpretations, and temporary lapses of famous physicians. These myths come to be regarded as 'established facts.' Any attempt to weed out myths is met with severe resistance and even ridicule. The original concept of VSR, put forward by Kearns and Hollenhorst² on simple clinical impression and little scientific evidence, falls into such a category. When I first advocated the use of the term 'VSR' for non-ischaemic CRVO, I had no intention of creating 'confusion'; on the contrary I wanted to eradicate the erroneous concept and give a strictly descriptive term for non-ischaemic CRVO. I still feel the term 'VSR' best reflects the clinicopathological features of non-ischaemic CRVO.

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SIR, Professor Hayreh attempts to circumvent the confusion resulting from the use of the term venous stasis retinopathy to describe two different retinal vascular disorders by claiming that most published cases of 'VSR' are actually VSR—i.e., that Kearns, Hollenhorst, and others misdiagnosed cases of central retinal vein occlusion as being ischaemic in origin due to carotid artery disease. Paradoxically, Professor Hayreh is prepared to challenge, wholesale, diagnoses made by experienced ophthalmologists without, I presume, being able to examine personally the patients in question, yet he decries 'personal biases' and 'erroneous interpretations' with which 'the field of medicine is littered.'

Professor Hayreh implies that he was the first to distinguish between the ischaemic and non-ischaemic varieties of central retinal vein occlusion. However, his classification is based on an unsubstantiated belief that the ischaemic-type central retinal vein occlusion is precipitated by blockage of the central retinal artery with arterial circulation becoming re-established 'within a few hours or days.'^{1,2} It was Laatikainen and Kohner³ who pointed out that in some patients central retinal vein occlusion causes extensive retinal capillary closure associated with a high risk of developing thrombotic glaucoma. It is this consecutive retinal ischaemia, also seen after retinal branch vein occlusion, to which the term ischaemic-type central retinal vein occlusion refers and not a hypothetical, transient interruption of the arterial blood supply to the retina.

Use of the words 'stasis' and 'haemorrhagic' to distinguish the two main forms of central retinal vein occlusion, both of which are characterised by slow perfusion and varying degrees of haemorrhage, is illogical. More importantly, the use of the two categories venous stasis retinopathy and haemorrhagic retinopathy may mislead the unwary into thinking that central retinal vein occlusions can be neatly subdivided into two types, whereas of course the retinal capillary response following central vein occlusion covers a spectrum with many patients showing well preserved capillary perfusion, some showing patchy non-perfusion and a minority developing very extensive capillary closure.⁴ Some patients with initially good capillary perfusion or only small areas of non-perfusion following central retinal vein occlusion may later develop progressively larger areas of retinal ischaemia, leading to an increased risk of rubeosis. In such cases it is easier to think of progressive retinal ischaemia rather than having to change the diagnosis from venous stasis retinopathy to haemorrhagic retinopathy.

In conclusion, the terms venous stasis retinopathy and haemorrhagic retinopathy should be abandoned.

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Safety glasses only for boys?

SIR, Belated congratulations to V Tommila and A Tarkkanen on their paper on incidence of loss of vision in the healthy eye in amblyopia¹ Their findings are most important and significant. I would like to know what the sex incidence was for the 23 patients in their group as a whole and especially the sex incidence for those 12 patients who lost their eye as a result of trauma.

From Vereecken and Brabant's work² one may suspect that trauma is almost always in males. If so, one would not have to insist that the girls with permanent amblyopia wear safety glasses—only the boys. That is, the probability of a permanently amblyopic female losing her good eye due to an accident might drop to perhaps 1:10000, while the probability for a boy would approach 1:300 (i.e., 1.75/500).
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SIR, We are grateful indeed for the letter of Dr Romano. As a reply to his question about the sex incidence of our series we can state that there were 17 males and six females. Of the females two were children aged 9 and 12 years. We agree with Dr Romano about the importance of suggesting safety glasses for patients with permanent amblyopia.

Although the probability of a permanently amblyopic female losing her good eye is low, we insist that the girls should also wear safety glasses because their good eye may be a target of flying objects, such as stones thrown by the boys.

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