ophthalmodynamometry) and they then reflect the differing patterns of branching of the central retinal and posterior ciliary arteries.[2,6]

This alternative diagnosis circumvents the requirement for extensive investigation in a young patient like this, not least the futile search for a source of arterial embolism. It also obviates any need to postulate an underlying retinal microvasculopathy of systemic origin to account for the 'decompression retinopathy'.[1] Thus, hemisphere retinal vein obstruction predicts and explains the haemorrhagic consequences of a procedure (ie, paracentesis) that is ineffectual anyway in its aim of improving cilioretinal perfusion. I say this having made the same mistake myself 30 years ago.[7] Ocular hypertension as noted in this patient may have been a factor in determining the extent of the retinal haemorrhage developing after the sudden lowering of intraocular pressure. Moreover, it was probably pathogenic in respect of the original hemisphere venous obstruction.

Unfortunately, the loculated haemorrhage that developed at the fovea as a result of the paracentesis may well have had an adverse effect on this patient's eventual visual acuity. Spontaneous improvement in vision is otherwise the rule when only a sector of the perifoveal capillary net has had its perfusion compromised as part of a combined retinal venous and cilioretinal occlusion.[5] This highlights the importance of recognising the true nature of such occlusions, which represent a high proportion of the vascular accidents affecting the retina of young adults, have no association with serious systemic disease, and tend to resolve without intervention.[5,8]

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Sir,

Reply to Professor McLeod

We would like to thank Professor McLeod's interesting and stimulating comments regarding our recent case report concerning the formation of Roth spots and retinal haemorrhages after paracentesis for a presumed segmental retinal artery occlusion.[1] We agree with Professor McLeod that it is of fundamental importance to determine whether the initial event in our patient's disorder was arterial or venous in nature, as this could affect future prognosis and would have altered the acute management. Our patient presented within a few hours of a sudden and profound onset of a central scotoma, which reduced his visual acuity to hand movements. When he was examined, he was found to have an area of retinal thickening and pallor in the superior macular area. There were no retinal haemorrhages or venous changes noted. Based on the clinical history and the clinical findings, a presumed segmental retinal arterial occlusion was diagnosed and treated accordingly. The retinal venous changes and haemorrhages developed immediately after the paracentesis, suggesting that they were secondary to this intervention, perhaps due to an increase in the transmural pressure gradient across the affected retinal veins.

When the fundus fluorescein angiogram was performed, immediately after the paracentesis, the combination of arterial occlusion and masking haemorrhage prevented one determining if the occluded vessel was a branch of the central retinal artery or a true cilio-retinal artery. Subsequent FFA after resolution of the fundal changes and possible recanalisation of the

affected artery has shown that it fills at exactly the same time as the other retinal arteries, hence our labelling of the affected artery as a branch macular artery occlusion. In keeping with Professor McLeod's hypothesis on the involvement of cilio-retinal artery occlusion in retinal vein occlusion, this vessel, directly connected to the central retinal artery, would be unlikely to be affected secondarily by a venous occlusion due to its higher perfusion pressure.

Based on the temporal relationship between the clinical findings, we feel that an arterial occlusion was the initial event and feel justified in trying our best to treat the underlying event as we did and in our subsequent investigations.

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