Editorial: Ischaemic optic neuropathy

The authors writing in this issue of the journal perform a considerable service in bringing together many of the known features and some, as yet unreported, characteristics of a not too uncommon cause of visual loss in the elderly— ischaemic papillopathy or, as Hayreh (p. 955) prefers to term it, anterior ischaemic optic neuropathy. The abbreviation AION adopted by Hayreh almost of necessity presupposes the existence of a posterior counterpart “PION” which might, perhaps, be applied to the post-traumatic optic atrophy associated with damage to the optic nerve within its bony canal.

The occurrence of ischaemic optic neuropathy has been recognized for many years although its true nature has been in dispute. Gowers in 1879 described many of the features of ischaemic neuropathy, including the characteristic altitudinal field defect, and thought that the early stages of the condition were inflammatory in nature. For this reason, he ascribed visual loss in severe anaemia or after haemorrhage to a neuritis, a concept which has persisted in names such as ischaemic papillitis until fairly recently.

Hayreh’s careful anatomical and experimental work relating to the pattern of blood supply to the optic nerve and posterior globe has, however, provided a rational explanation for the mechanisms underlying the development and behaviour of ischaemic optic neuropathy.

The aetiology of ischaemic optic neuropathy is most commonly arteritis or arteriosclerosis. Certainly, recognizable cardiovascular disease is common in patients with nonarteritic ischaemic neuropathy and their prognosis for life is poor. The role played by arteriosclerosis per se is difficult to establish, but careful investigation of the cardiovascular system and the intraocular pressure will often reveal some significant and perhaps treatable disorder which may be of direct aetiological significance.

The earlier reports on the value of systemic corticosteroid therapy in the initial stages of non-arteritic ischaemic optic neuropathy did not claim to be based on a controlled trial with random selection of patients and the work reported in this issue may be subject to the same criticism. Inevitably, the ethical problems of withholding treatment in a blinding disease when one has some evidence of its value makes the carrying out of a controlled trial difficult.

The importance of intraocular pressure in the aetiology of one type of ischaemic optic neuropathy is undoubtedly and generally recognized. It is probably true that most patients suffering from an acute attack of closed-angle glaucoma develop ischaemic optic neuropathy and many, its subsequent optic atrophy. It seems possible that the catastrophic loss of the remaining visual field occasionally encountered after surgery for advanced uncontrolled chronic simple glaucoma may also be due to ischaemic optic neuropathy.
precipitated by the effects of general anaesthesia on the blood pressure or by the effects of local anaesthesia on the circulation of the optic nerve head.

One feature still remains obscure. It is very rare indeed, except when there has been an acute rise in intraocular pressure or a calamitous fall in blood pressure, to identify with any certainty the triggering factor for an attack of acute ischaemic optic neuropathy, although many of the predisposing factors may be identified without difficulty.

Patients without arteritis who have developed ischaemic optic neuropathy occasionally suffer an attack in the other eye or may even have a further attack in the same eye. In the absence of knowledge of the triggering factors, no prophylactic measures of proven efficacy exist and the long-term management of these patients remains difficult.

Nevertheless it is apparent from the paper by Eagling, Sanders, and Miller (p. 990) that ischaemic optic neuropathy is not a diagnosis but merely a recognition of local anoxia of the optic nerve, the causes of which are both multiple and complex, requiring the facilities of a general hospital to elicit the basic aetiological factor or factors.

Reference