Editorial: Pathogenesis of optic disc swelling

Swelling of the optic nerve head is a prominent ophthalmoscopic sign in a variety of ocular and systemic disorders including ischaemic optic neuropathy, raised intracranial pressure (papilloedema), accelerated systemic hypertension, and ocular hypotony. Recent experiments on primate models have led to a clearer understanding of the nature and pathogenesis of the disc changes in these disorders. Crucial to these advances have been investigations of the disturbed physiology of retinal ganglion-cell axons, which are the major constituent of the disc tissue.

The axonal swellings which develop adjacent to a ligation of an axon bundle result from interruption of the normal bidirectional transport of proteins and organelles within the axoplasm between the nerve-cell bodies and the synaptic terminals (Lubinska, 1964; Griffin et al., 1977). The organelle-packed swellings on the cell-body side of the ligature are due to physical obstruction of continuing rapid orthograd e transport (with a further contribution from obstructed slow orthograd e flow), while the swollen stumps on the synapse side of the ligature result from obstructed retrograd e flow. A similar transport block can also be produced by ischaemia, especially if this is sufficiently severe and prolonged to cause irreversible vacuolation and disintegration of a localised axon segment. In this issue Mr D. McLeod and colleagues (p. 591) present preliminary results which indicate that experimental occlusion of the posterior ciliary arterial supply to the optic nerve head causes infarction of the immediately retrolaminar myelinated optic nerve. The associated complete obstruction of rapid orthograd e axoplasmic transport at the lamina cribrosa (as shown by autoradiography) is thought to be the fundamental cause of the opaque prelaminar swelling that characterises acute ischaemic optic neuropathy. The disc swelling is equivalent to a 'cotton-wool spot' of the optic nerve head. Like most retinal cotton-wool spots, the disc swelling does not itself represent ischaemic axon damage but is an accumulation of axoplasmic debris adjacent to an infarct (McLeod, 1976).

The site of obstruction of orthograd e axoplasmic transport after posterior ciliary artery occlusion is the same as that found in experimental papilloedema due to implantation and inflation of intracranial balloons (Tso and Hayreh, 1977a). In papilloedema, however, the cause of the obstruction to rapid and slow axoplasmic transport at the lamina cribrosa is not so clearly defined. Hayreh (1976, 1977) believes that the obstruction is primarily mechanical and due to compression of the retrolaminar myelinated optic nerve by the raised pressure in the subarachnoid space, that is, equivalent to a partial ligation. Whatever the underlying mechanism, there is now good experimental evidence (Tso and Hayreh, 1977b) that the principal cause of the prelaminar swelling in papilloedema is distension of ganglion cell axons rather than intercellular or intraglial fluid accumulation as thought hitherto. Axonal distension is characteristic of even the earliest stages of disc swelling and is almost certainly a consequence, rather than the cause, of the axoplasmic transport block at the lamina. Thus, the swollen disc of papilloedema is truly a 'choked' disc in the axonal as well as the vascular sense. This concept is not incompatible with the preservation of vision in clinical papilloedema, since the transport block is incomplete and the axons are not disrupted and do not degenerate, at least in the early stages. The transport-block theory is also consistent with the observation that atrophic discs do not develop papilloedema, as in such cases there are no axons to swell.

Optic disc swelling with distended organelle packed axons also occurs in experimental systemic hypertension (Garner et al., 1975). Although no physiological studies were performed, an axoplasmic transport block is undoubtedly implicated owing to either posterior ciliary ischaemia or raised intracranial pressure.

Axoplasmic transport has been thoroughly investigated in disc swelling from ocular hypotony and also in ocular hypertension, the overall effect of the latter being cupping rather than swelling of the optic disc (Anderson and Hendrickson, 1974; Minckler et al., 1976; Minckler et al., 1977; Minckler et al., 1978). Again, an obstruction of slow and rapid orthograd e axoplasmic flow has been demonstrated at the lamina cribrosa together with a retrograd e transport block at the same site. From their detailed studies Minckler and colleagues conclude that the transport block is mechanical in nature, resulting from cramping of axons by the distorted glial-scleral columns of the lamina cribrosa.

There can be little doubt that the results of all these experiments have a direct bearing on optic disc swelling as seen clinically. An axoplasmic
transport block appears to be the 'final common pathway' (Tso and Hayreh, 1977b) through which many ocular and extraocular disorders produce swelling of the optic nerve head.

References


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