Book reviews


In 1956 Dr Hugh Davson produced a memorable monograph on The Physiology of the Ocular and Cerebrospinal Fluids, and by a fortunate concatenation of circumstances he has been able, 20 years later and under the auspices of the Fogarty International Centre, to organise a symposium on the same topics. This symposium, which was held in May 1976, brought together contributions from the many research workers who have intrepidly voyaged into regions which 20 years ago could be referred to as ‘backwaters of physiology’. The contributions in this volume show that the backwaters have now become navigable seaways within which many different interests are represented.

The volume contains 40 contributions, of which 18 deal directly with the eye. Most of these are reviews and reports of experimental rather than clinical studies, though the contribution on papilloedema from Cogan and Kuwabara is an obvious exception. Of the review papers those by Raviola (blood-ocular barriers), Tripathi (morphology of outflow systems), Bill (physiology of aqueous drainage), and Langham (pharmacology of aqueous outflow) are especially valuable, and the papers by Bito and Wallenstein on the transport of prostaglandins and by Eakins on the breakdown of the blood-aqueous barrier break new ground and provide excellent introductions to fresh fields of interest. The papers dealing with cerebrospinal fluid and the blood-brain barrier are no less valuable, and one must single out the reviews by Brightman (morphology of blood-brain interfaces), Oldendorf (blood-brain barrier), and Welch (hydrocephalus).

As is always the case with papers provided at a symposium, the quality of the contributions is not uniformly high. Some have been added in reply to others presented at the meeting and, while valuable in their content, will prove difficult reading for the uninitiated.

The editors might have attempted to make a rather more logical arrangement of their subject-matter; excellent as the contents are for the cognoscenti, they will prove hard going for the tyro. Nevertheless, I believe that this ‘Fogarty Symposium’ will be an invaluable source of useful information and ideas for many years to come.

DAVID F. COLE


This textbook has undergone an extensive revision in its fifth edition, and new chapters on carbonic anhydrase inhibitors and local anaesthetics have been added. The aim of the book is to provide, firstly, a quick reference to treatment, both medical and surgical, of specific disorders, and secondly to give the usage, dosage, and side-effects of the drugs used in ophthalmology. It is a useful reference book for an ophthalmic library, although the less experienced eye surgeon may at times be confused by the number of therapeutic possibilities which are suggested.

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Pathogenesis of optic disc swelling

TO THE EDITOR, British Journal of Ophthalmology

SIR, Your editorial on 'Pathogenesis of Optic Disc Swelling' (September 1978, pp. 579-580) makes the all too common mistake of equating swelling of the disc from ischaemia and similar causes, wherein there is undoubtedly blockage of axonal transport, with papilloedema from raised intracranial pressure (ICP), wherein at least in the early stages any such blockage is questionable and should be questioned. I am assured, by Tso himself at the 1976 Cambridge symposium, that blockage of axonal flow is not consistent with continuance of normal function. One feature of papilloedema from raised ICP is that normal vision usually continues without interference, apart from variable enlargement of the blind spot, for many weeks or months. I watched 1 case for 5 months who kept normal vision to the end and whose discs on ophthalmoscopy showed nothing suggestive of axonal transport blockage (Primrose, 1976). Prolonged or severe cases may indeed show some suggestive signs on and around the disc, and one would expect these to be cases with some interference with vision, e.g., peripheral constriction of the fields or obscurations, which are both probably from vascular insufficiency and a warning that further severe visual failure is imminent.

Blockage of axonal transport is all very well for the 'final common pathway' but it just won't do for the early stages of papilloedema. The site of such blockage is understandably similar to that found in experimental acute glaucoma and hypotony (Tso and Hayreh, 1977) and posterior ciliary artery occlusion (McLeod, 1976), but how can this be a cause or an early sign in papilloedema from raised ICP when vision is so conspicuously unaffected? These disturbances upset the hydrodynamic balance at a site susceptible for anatomical reasons to vascular insufficiency, and it is surely anoxia from this which causes the blockage. Hayreh's idea (Hayreh, 1977) seems to be that raised retrolaminar tissue pressure from the raised ICP causes some blockage of axonal transport which, from the nerve swelling at the tight hole at Bruch's level, leads to pressure on the thin-walled veins, thus explaining away the venous (and other) congestion so consistently found. A continuing CSF pressure of 300 to 400 mm H₂O (22 to 29 mmHg) seems to be sufficient to produce papilloedema, and a tissue pressure of that level does not damage other nervous tissue (Hayreh, 1977). It doesn't seem to me sufficient by itself to impair axonal flow.

One must seek other reasons for the particular susceptibility of the disc, and the vascular hypothesis, which your editorial ignores, offers a means of explaining both