Despite Rushton’s poor response with the orally administered β₁ block, ICI 118,551, β₂ specific blockers used topically on the eye have been reported to lower intraocular pressure potently in animal eyes. Furthermore we are not at all surprised that he found ICI 118,551 less potent than (+) propranolol. We have found that ICI 118,551 binds 10 times less potently to receptors in the ciliary processes than (+) propranolol (Trope GE, Clark B, paper in preparation). (Kd for ICI 118,551 = 5.5 x 10⁻⁸, Kd for (+) propranolol = 10⁻⁴.) Despite this finding we still feel that a trial of Topical ICI 118,551 on patients with glaucoma is probably indicated in view of this drugs β₁ specific blocking effects and its cardioprotective action. ³

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References

Angioid streaks in thalassaemia major

Sir, I read with interest the paper on angioid streaks in a case of thalassaemia major. ¹ I thought it appropriate to call to your attention a paper entitled ‘Laser treatment of choroidal neovascular membranes in angioid streaks.’ ²

Case 1 describes a 50-year-old man with a history of thalassaemia intermedia and haemochromatosis (as a result of his anaemia). Fundus examination revealed peripapillary angioid streaks in both eyes with dense disciform macular scarring in the left.

We noted in the case cited in our paper that haemosiderosis can occasionally accompany thalassaemia intermedia. Haemochromatosis also results in the clinical manifestation of iron overload in the tissues. The 2 haemolysis disorders combined could cause iron deposition on Bruch’s membrane and the resultant angioid streaks. Yet neither thalassaemia intermedia nor haemochromatosis effects a primary disturbance in the elastic tissue of the body. The brittle lamina basalis in this case may have occurred because of iron deposition, and thus Bruch’s membrane is probably quite similar to that in a patient with sickle cell disease.

We were interested to find that you also noted the similarity between the 2. We mentioned that the mechanism of the breaks in Bruch’s membrane in the eyes of patients with sickle cell disease is not primary elastic tissue degeneration either, since no elastic tissue defect occurs with this disease. It has been conjectured that the haemolysis of sickled red cells leads to iron deposition on Bruch’s membrane, which would lead to the brittleness of the lamina basalis.

We were pleased to find that your paper also supports the iron deposition theory with reference to development of angioid streaks. Congratulations on an excellent paper.

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References

Book reviews


The aim of the work, as explained in the editors’ preface, is to bring a new, dynamic approach to the study of ocphalimatic pathology, concentrating on disease mechanisms rather than on descriptive pathology, which is stressed in other books on the subject.

The work is in 2 volumes totalling 55 chapters, written by a host of experts, who are recognised authorities in the field of their contributions, including the 2 editors, who have taken an active part in the writing.

The size of the work allows only a brief survey here of the contents. The first chapter, appropriately, is on ultrastructure, and is in a subsection headed ‘Basic principles’; it is followed by a discussion on inflammation in general, incorporating recent and important advances. Infection of the eye is dealt with in 8 contributions, each one discussing a particular class of agents such as micro-organisms, viruses, etc. The techniques and methods described in this section often belong to disciplines other than histopathology. Principles of laboratory investigations in ocular infection are discussed and practical details are given, such as the selection of culture media. All aspects of trauma are covered in an extensive chapter, and another substantial contribution deals with development, normal and abnormal, featuring valuable tabulations of various defects. A section on glaucoma includes experts’ accounts of anterior and posterior segment changes in this condition and a discussion
Angioid streaks in thalassaemia major.

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