
Sir, Thank you for allowing me the opportunity to reply to Dr Simons’s letter. Perhaps I may answer, one by one, the points he raises.

Firstly, we cannot say that those in our sample who presented before 3½, almost inevitably with a cosmetically noticeable squint, would necessarily have done so in other socioeconomic conditions. The particular set-up we have in that part of our district could have predisposed us to the findings we reported.

Secondly, I accept that our ‘false negative’ rate was derived from the screening methods we used. 26% of those ultimately found to have abnormal vision had a ‘false normal’ visual acuity and/or cover test when screened at 3½. Likewise, 20% of those ultimately found to have abnormal vision, had a ‘normal’ refraction at 3½ and would have been ‘missed’ by this test.

The question of ‘false positives’ is equally important, because they cause increased and unnecessary parental concern, work and financial expense. In our series 56–7% of those who recorded normal or uncertain vision at 3½, and 51–2% of those who had an abnormal refraction (using our criteria) at 3½, actually had normal vision without any treatment. The majority of the ‘false abnormal’ refractions were hypermetropia without astigmatism or anisometropia. I have since realised that, if we had chosen only astigmatism and/or anisometropia as the criterion for abnormal refraction, the percentage of false positives using refraction would have been reduced to 29–5%. If we had combined this with a cover test, I calculate we would have identified 85% of those with abnormal vision (excluding myopia). It might be possible to simultaneously test for an abnormal refraction and squnt using the more modern developments of photo refraction. Nevertheless, some false positives and false negatives are both inevitable whatever method is used.

Thirdly, Dr Simons justifiably questions our method for stereoacuity testing. Maybe guessing and inadequate cooperation should not have been a problem, but they were. We found interpretation of the response to be too unreliable for use as a one-off, non-repeated screening test in the population sample we screened. This does not mean to say that others would also find this so.

I will admit to being slightly ‘partisan’ against testing for stereoacuity, but no more than I am against any other ‘subjective’ screening test. This is because in most cases defective visual acuity is associated with an abnormal refraction and/or a squint, and therefore I think that an objective test should be more accurate than a subjective one, particularly in preschool children. Having said that, we have not recommended screening by refraction—we merely advised against screening at 3½ by any method.

Finally, our views on the results of occlusion in these children are questioned again. I do not think that improvement to 6/6 is necessary before one can conclude that occlusion has been valuable. However, I wonder whether one or two lines improvement in the range 6/18 to 6/9 is worthwhile, and I do believe that the standard treatment of amblyopia should be questioned.

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Reference

Adverse reactions to guanethidine eye drops

Sir, I write to express my regret that guanethidine 5% with adrenaline 1% eyedrops have been voluntarily withdrawn by the company responsible for their manufacture.

I have for some years been concerned about the rare effects of antiglaucoma medication and other topical eye therapy on the conjunctiva and cornea.12 There is no question that these adverse effects are rare, unpredictable, and not normally dose or time related.

All topical therapy, in common with other medicines, carries both benefit and risk, and it is the job of the prescriber to calculate this risk/benefit ratio for any patient receiving treatment. I understand that at least 1000 patients whose glaucoma was currently controlled with guanethidine 5% plus adrenaline 1% have now been deprived of useful therapy. I believe that it would have been more appropriate for the company to insert a warning in the package advising that the combination had been associated with conjunctival cicatrization and advising that prescribers should monitor the outer eyes of patients receiving drops. In addition to depriving a large number of patients of useful treatment the decision to withdraw the drops also creates an unreal impression of safety. Any topical therapy can produce an unaccepted adverse reaction, and all prescribers will need to continue to watch for signs of these adverse effects, which are not limited to guanethidine and may even be associated with common constituents such as preservatives.

Responsibility for monitoring adverse effects as well as benefits will always be the prime duties of a prescribing doctor.

PETER WRIGHT
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References

Book reviews


This book was first published in 1978 and the revised edition includes chapters by a number of authorities to augment the
This book classic occurring Noorden's Burian-Von in view of the Motility. mined biochemical despite the seeing patients with the rarer mologists ocular there copper of prenatal treated and contents metabolism, disorders of purine and metabolism, disorders of aminoacid metabolism, organic acidemias, disorders of purine and pyrimidine metabolism, disorders of carbohydrate metabolism, erythrocyte enzyme deficiencies and haemolytic anaemias, disorders of porphyrin and haem, disorders of steroid metabolism, disorders of lipoprotein metabolism, disorders of thyroid hormones, and disorders of copper metabolism. In view of this very extensive list of contents it is not surprising that the clinical, genetic, pathological, and biochemical features of each disorder are treated in a very succinct manner, often with emphasis on prenatal diagnosis and genetic counselling. Most of the ocular manifestations of each disease are mentioned, albeit briefly. It is disappointing that, for a book published in 1985, there appear to be relatively few references after 1980, despite the enormous advances in molecular genetics over the past few years. This is a useful (relatively) small reference book which should be available to all ophthalmologists seeing patients with the rarer genetically determined biochemical disorders. It will have to be used with care in view of the considerable advances in this field occurring almost daily. BARRIE JAY


This book first appeared in 1974 and immediately became a classic text on its subject. Sadly, Hermann Burian died in the year of the first edition, but Professor Von Noorden has soldiered on valiantly with the two subsequent editions, of which this is the latest.

The book is divided into four sections—on the sensori-motor physiology of the eyes, the neuromuscular anomalies of the eyes, clinical characteristics of these anomalies, and finally principles of therapy. It is very difficult to fault the book. It has always been the best balanced and most impartial source of data where other authors have a tendency to ride hobby horses. The reader is particularly directed to the sections on exotropia, microtropia, and the surgical treatment of congenital esotropia. In addition each chapter has an enormous number of useful references with a full list at the end of each chapter. I had a few disagreements: in chapter 24 the original Harada-Ito procedure to correct exyelotropia is correctly described, but there is no mention of the Fells modification, which is the technique used by the majority of squint surgeons. Although the technique of botulinum toxin treatment of strabismus originated by Alan Scott is relatively new, it has been practised with considerable success since 1979 by an increasing number of investigators, and it would be hard to discover that from the 13 lines in which the technique is described.

However, apart from these minor points this is a splendidly comprehensive book which must be in every eye department library and which can be warmly recommended to any young ophthalmologist in training or who requires to read for the fellowship examination. J P LEE

Notes

Finance offered

David Cole Travel Fellowship

The David Cole Travel fellowship, instituted by Merck Sharp and Dohme in memory of Professor David Cole, will assist a visit to a hospital or research centre during the academic year starting 1 October 1987. The award will be equivalent to £2000. The purpose of the award is to enable the successful applicant to gain experience and knowledge in pursuit of a specific project related to glaucoma.

Glaucoma Group Research Grant

The Glaucoma Group Research Grant, sponsored by International Glaucoma Association, will be available for a research project clinically orientated towards glaucoma for 1987. The award will be equivalent to £2000. The Grant may be used towards salary or project expenses or for buying equipment.

Both these awards are available to medical graduates and non-medical scientists resident in the United Kingdom or the Irish Republic. They may be held concurrently with other awards. Further details and application forms from Dr S Nagasubramanian, Secretary Glaucoma Group, Glaucoma Unit, Moorfields Eye Hospital, High Holborn, London WC1V 7AN. The closing date for applications is 19 June 1987. The successful candidate will be informed by August 1987.