BOOK REVIEW

Dictionary of Visual Science. 4th Edn. By David Cline, Henry W Hofstetter, John R Griffin. Pp 820. US$57. Chilton Trade Book Publishing: Radnor, Pennsylvania, 1989.

The 4th edition of this book, first published almost 30 years ago, is dedicated to the memory of the late senior editor described as the motivator of this lexicographic work. The majority of the 68 contributors are optometrists or basic scientists, with a mere handful of clinicians. Not surprisingly, there is a certain bias towards optometric terminology, and indeed the strength of the book lies in its coverage of physical and geometrical optics, ophthalmic dispensing, and vision therapy. As stated in the preface, this edition extends the coverage of ocular genetics, pharmacology, familiar and unfamiliar acronyms, and abbreviations and syndromes with ocular manifestations.

On the whole the clinical listings are rather disappointing and somewhat limited, with inclusion of many obsolete terms and nomenclature. As intended, the definitions are generally succinct, and a phonetic respelling follows most entries. There are very few listings accompanied by their derivation, which is somewhat disappointing. Throughout the text there is a random selection of line diagrams depicting mostly optical principles and equipment with photographs of clinical disorders, many of which are of poor quality. Clinical terms can prove difficult to find, and eponymous titles are listed under disease, syndrome, or anatomical structure. There is a wealth of archaic clinical terms which offer a certain fascination and would be guaranteed to floor or possibly demolish an unsuspecting examination candidate. There is a brief appendix including ophthalmic clinical abbreviations, symbols, and useful optometric data. Although the dictionary has a number of shortcomings, it will nonetheless prove a useful reference manual for all workers in the field of visual science.

ROBERT J COOLING

LETTERS TO THE EDITOR

Carbamazepine, epilepsy, and optic nerve hypoplasia

SIR,—We report a case of bilateral optic nerve hypoplasia (ONH) in a child born to a woman taking carbamazepine (CBZ) during pregnancy. A mother of two healthy girls developed generalised seizures at the age of 27 years. She has remained on CBZ (1 g/day) monotherapy since then and has had no further seizures. During her third pregnancy (aged 29 years) she was admitted to hospital at 17 and 20 weeks for blood loss, which settled uneventfully. At 39 weeks she had a normal delivery of a healthy 4 kg girl.

Initial ophthalmic examination of the baby at nine weeks showed roving eye movements. Pendular nystagmus and a right convergent squint have subsequently developed. Examination under anaesthetic at five months revealed isolated extremely small hypoplastic optic nerves. A computed tomographic brain and orbital scan was unremarkable apart from abnormally slender optic nerves. At eight months visual function remains severely impaired. The baby is otherwise developmentally and physically normal.

Possible teratogenic effects of anticonvulsant drugs during pregnancy have been reported for more than 20 years. The fetal hydantoin syndrome has been primarily associated with phenytoin. It combines poor growth with mild mental retardation, a characteristic craniofacial appearance, and skeletal abnormalities, including digital hypoplasia. It is said to occur in 11% of at-risk infants. Incomplete forms are known to occur in another 31%. The number of isolated malformations described following fetal phenytoin exposure are protean and includes ONH in seven previously described cases.[1] The relative contributions of the epilepsy and anticonvulsants to the described abnormalities are disputed, though phenytoin has been shown to be teratogenic in experimental animals. Furthermore there is evidence that combinations of hydantoins and barbiturates may increase the risk to the fetus.

CBZ in high doses has been shown to reduce fetal body and brain weight in pregnant mice.[2] In 1973 Starreveld-Zimmerman *et al.*[3] observed no congenital malformations in 50 CBZ exposed babies and 22 malformations in 247 babies whose mothers had taken anticonvulsants other than CBZ, but Hiilesmaa *et al.*[4] subsequently described fetal head growth retardation in babies born to mothers taking CBZ.

While many cases of ONH are idiopathic, several substances have been implicated as causative. They include quinine, lysergic acid diethylamide (LSD), phencyclidine, alcohol, and phenytoin.[5] There is also an increased incidence of ONH in infants born to diabetic mothers.[5]

Numerous studies now suggest that susceptibility of the fetus to teratogenic effects of hydantoins depends on the fetal genotype. Inherited defects in phenytoin arene oxide detoxification may contribute. As CBZ and phenytoin are structurally different, it is not possible to postulate a similar pathogenic mechanism for ONH development in the former.

To our knowledge, the occurrence of isolated ONH in association with CBZ monotherapy is unique. Could they be causally related, or is this a chance occurrence?

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Intravitreal therapy with ganciclovir for posterior pole cytomegalovirus retinitis in AIDS patients

SIR,—Several authors have reported the use of intravitreal ganciclovir therapy for patients with cytomegalovirus retinitis (CMV).[1-5] The associated complications due to this therapy are substantial and include subconjunctival haemorrhage, laceration of the lens by the needle during injection, retinal detachments secondary to iatrogenic tears, vitreous haemorrhage, and endophthalmitis. Despite these shortcomings some authors have been encouraged by its ability to improve CMV retinitis.[1 3 5]

We have treated eight eyes in five patients with severe involvement of the posterior pole (optic nerve-macula region) who also had severe neutropenia which precluded their continued treatment with systemic ganciclovir at normal levels. Treatment consisted of a series of three intravitreal 200 μg injections, which was administered through the pars plana region of the eye under retrobulbar anaesthesia every five days during a 15-day period. A 30 gauge short needle on a tuberculin syringe was used. Thereafter the injection was administered once per week until the appropriate intravenous induction (5 mg/kg intravenously twice a day) or maintenance (5 mg/kg intravenously four times a day) dosage could be resumed. We found that this therapy was useful only as an adjunct to the systemic dosage. By itself the injections were not able to improve clinically either the retinitis or the visual acuity. However, the retinitis ceased to advance and the acuity stabilised when intravitreal ganciclovir was given concomitantly with the systemic ganciclovir. Retrobulbar anaesthesia should be used to ensure that the patient does not experience pain during the injection. Sudden movement of the patient during the injection may lead to complications noted by several authors.[4 5]

We feel that intravitreal ganciclovir by itself will not improve CMV retinitis of the posterior pole. It must be administered with systemic ganciclovir, even in a reduced dosage, in order to at best stabilise the retinitis.

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