LEADING ARTICLE

THE LAZY EYE

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Amblyopia, or "lazy eye" in lay terms, is defined by Gunther K von Noorden[1] as "a unilateral or bilateral decrease of visual acuity caused by form vision deprivation, abnormal binocular interaction, or both, for which no organic cause can be detected by the physical examination of the eye and which, in appropriate cases, is reversible by therapeutic measures". Stated simply, it is "the condition in which the observer sees nothing and the patient very little" (von Graefe).

Amblyopia affects many aspects of visual function - monocular acuity, luminance detection, spatial localisation, optokinetic nystagmus and stereopsis[2]. The loss of visual function is most marked centrally while peripheral vision may remain unimpaired. Peripheral fusion is of paramount importance in maintaining good ocular alignment and allowing stereoscopic depth perception[3]. Eccentric fixation, anomalous retinal correspondence and interocular suppression can occur. Suppression (of the image of one eye) serves to avoid confusion - which arises from different images falling on corresponding retinal elements of both eyes, and diplopia - which results from the left and right eye images falling on non-corresponding elements of both retinae.

The prevalence of amblyopia is 2-5% in the general population[4]. The epidemiological study of amblyopia presents a challenge in that true figures for incidence may be elusive for a number of reasons. Firstly, the pick-up rate of amblyopia at its onset is lowered by the difficulty in diagnosis in the very young age group. Secondly, diagnosing amblyopia in the older age group gives no indication of when it actually began. Thirdly, no standard criteria for diagnosis exist. In most prevalence studies, an eye is classified amblyopic if the best corrected visual acuity is worse than 6/12 with no ophthalmoscopically detectable abnormality. Other criteria differentiate between the visual acuities of the two eyes (2 or more lines difference in Snellen acuity) or use different levels of visual acuity as the cut-off point[5].

Downing, in the Second World War, reported that over 3% of some 60,000 military inductees in the United States had vision of less than 6/12 without any ophthalmologically detectable defect commensurate with the decreased vision[6]. This study, done under wartime circumstances, could be potentially biased by malingering, improper examination techniques, or other artifacts. Helveston[7] studied military volunteers in 1962-63 following Downing's criteria and found the prevalence to be 1%. Possible explanations for the difference include more careful examination techniques with special efforts at detecting malingerers, prior treatment or pre-screening of Helveston's population, population differences between military volunteers in the early 1960s and draftees in World War II, and true temporal changes in prevalence[5]. A similar line of reasoning could apply to the study presented in this issue.

The classification of amblyopia is based on etiology. It may be 1) refractive-anisometropic, isoametropic (bilateral), meridional; 2) strabismic; or 3) of the vision deprivation type, in which congenital cataract is the most common cause. Poor visual acuity from refractive errors correctable with appropriate lenses is not amblyopia whereas uncorrected refractive errors during the sensitive period of life (vide infra) can give rise to amblyopia. In strabismic amblyopia, strabismus can lead to amblyopia, but so can amblyopia result in strabismus and at times, it is difficult to tell which came first.

Early development of the visual system is characterised by proliferation of connections amongst neurons, followed by selective elimination. The initial increased contact between cells probably triggers differentiation of the cells for special roles. This sequence of events begins without any complete specification. Postnatal development is necessary to refine the visual system such that experience is used to achieve one precisely tuned for the visual environment in which it must function. This mechanism of plasticity occurs during the "sensitive period" of life when changes in visual input can produce dramatic changes in cortical function and cytoarchitecture[8-10]. The sensitive period in man has been estimated by clinical data obtained from occlusion therapy. The human visual system is found to have increasing sensitivity from birth, reaching a peak around eighteen months of age. This then falls off rapidly to the thirtieth month but remains plastic up to 8 years of age[11], the estimated general upper limit of the human sensitive period.

Occlusion therapy has been practised for the past 200 years and remains till today the single most effective form of treatment for amblyopia. The principle is elegant in its simplicity - occluding the sound eye encourages use of the unsound eye and the plasticity of the visual system during the sensitive period allows for recovery of visual function of the unsound eye[12]. Appropriate corrective lenses should be prescribed at the same time as occlusion of the preferred eye. Periodic follow-up with maintenance therapy where needed is mandatory up to age 9 as amblyopia frequently recurs before then[3]. In amblyopes older than 8 years, some advocate a 3-month therapeutic trial of occlusion with favourable results obtained[13]. The visual prognosis worsens if there is no improvement within the first 3 months. If improvement is observed after this trial period, occlusion can be maintained to 10 years of age and, even up to 15 years in the refractive type of amblyopia. However, enforcement of occlusion therapy after the age of 9 should be tempered with a consideration of the social burden placed on the child. The management of amblyopia thus depends on the collaboration of patient, parent and physician[3].

At the primary health care level, screening with early detection of amblyopia offers the best possible hope for

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improving the visual prognosis of young children so affected. It is ideal that all children be checked with simple vision tests appropriate for their age. The problem remains of screening all, especially those of the pre-school age and, unless every child is brought to health care centres at regular intervals for vision testing, many amblyopes may be missed at an early age. That screening techniques for amblyopia have not been verified[14] and their sensitivity and specificity unknown necessarily compounds the problem. As vision screening for all at any age is impractical, one that is done for 4 years of age, before the child enters kindergarten, still allows some room for therapy of the detected amblyope. Selective screening of those children, at any age, with a family history of any visual disorder would definitely increase the detection rate.

As we understand amblyopia and its underlying mechanism, we begin to appreciate the value of early diagnosis and therapy. The monumental and momentous works of Hubel and Wiesel[8-10,15] that earned them the 1981 Nobel Prize continue to inspire visual physiologists to explore related avenues of research and in the process, uncover new ground. At this juncture, an interesting question comes to mind - is the length of the sensitive period determined by genes? Well, there is but one way to find out - let the search begin!

References

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