Eye (2004) 18, 557–558

www.nature.com/eye

EDITORIAL

Amblyopia—Duke-Elder was right

R Gregson

Eye (2004) **18,** 557–558. doi:10.1038/sj.eye.6700672

In this issue, Hwang and Ahn[1] describe their success in treating amblyopia in patients we would normally regard as too old. This paper covers many recent publications that are changing our long-held notions about amblyopia.

Most current ophthalmologists' education in this area followed the neurophysiological experiments of the 1960s and 1970s begun by Hubel and Weisel, and for which they shared 50% of the Nobel Prize for medicine in 1981. These experiments in cats and monkeys suggested that the neural basis for amblyopia was a massive reduction in binocular neurons and a shift in the ocular dominance of neural activity towards the unaffected eye. Following the period of cell division and development of the visual cortex, thought to end at about 7–8 years in humans, the visual cortex became fixed and little further improvement could be made.

It is now clear, however, that some amblyopes can improve later in life. The best evidence for this comes from the work of Rahi *et al*,[2] who studied adults in the UK who had lost their good eye. In many, the vision in the amblyopic eye improved, but the chance of this happening was related to the degree of amblyopia:

Initial vision in the amblyopic eye:	6/12–6/18	6/18–6/60	<6/60
Percentage that improved to driving vision: (data from Rahi *et al*[2])	36%	16%	3.5%

It seems that not all amblyopes are the same, and/or many cases of amblyopia are not human versions of the cats and monkeys of the neurophysiological experiments, with a finite critical period. For example, Goodyear *et al*,[3] using functional MR imaging, found that in six early-onset amblyopes there was indeed a shift in the ocular dominance columns of the visual cortex, but in two late-onset amblyopes this was not true—the ocular dominance columns were of the same size. Davis *et al*[4] found that there were qualitative differences in visual evoked potentials and contrast sensitivity in the early- and late-onset amblyopes, indicating differing neural mechanisms in these two groups. Levartovsky *et al*[5] found that following treatment of amblyopia up to the age of 9 years, between 51 and 75% of patients deteriorated again.

To his eternal credit, Duke-Elder[6] foresaw this difference. Although he devoted barely a couple of pages of the gigantic *System of Ophthalmology*, his comments are pithy. He distinguished between 'amblyopia of arrest' in which normal vision was never allowed to develop in the amblyopic eye, and 'amblyopia of extinction' in which normal vision had developed but was then inhibited. The latter came on after the age of about 4 years and was easier to treat. It seems likely that 'amblyopia of arrest' is indeed the human version of the animal experiments, with a shift in the neural populations producing amblyopia that is difficult to treat—due for example to a unilateral congenital cataract. 'Amblyopia of extinction' is probably the condition that fills orthoptists' clinics—caused by anisometropia, squint, or the combination of the two.

This recent research suggests three things: firstly that we should be more aggressive in treating amblyopia in younger children (3 years and under). Secondly, it clearly is worth while treating older patients, despite the difficulties they have with compliance. Thirdly, amblyopia treatment should not cease at 7 years. Probably it should continue throughout life, but in order to do this, we must find a treatment that is acceptable to older children, teenagers, and adults.

References

1 Hwang J-M, Ahn JK. Efficacy of amblyopia therapy initiated after 9 years of age. *Eye* 2004; **18**: 571–574.

Department of Opthalmology
University Hospital
Queen's Medical Centre
NG72UH, UK

Correspondence:
R Gregson
Tel: +44 115 924 9924
Fax: +44 115 970 9749

2 Rahi J, Logan S, Timms C, Russelll-Eggitt I, Taylor D. Risk, causes and outcomes of visual impairment after loss of vision in the non-amblyopic eye: a population-based study. *Lancet* 2002; **360**(9333): 597–602.
3 Goodyear BG, Nicolle DA, Menon RS. High resolution fMRI of ocular dominance columns within the visual cortex of human amblyopes. *Strabismus* 2002; **10**: 129–136.
4 Davis AR, Sloper JJ, Neveu MM, Hogg CR, Morgan MJ Holder GE. Electrophysiological and psychophysical differences between early- and late-onset strabismic amblyopia. *Invest Ophthalmol Vis Sci* 2003; **44**(2): 610–617.
5 Levartovsky S, Oliver M, Gottesman N, Shimishoni M. Long-term effect of hypermetropic anisometropia on the visual acuity of treated amblyopic eyes. *Br J Ophthalmol* 1998; **82**(1): 55–58.
6 Duke-Elder S. *System of Ophthalmology* Vol VI, Henry Kimpton: London, 1973, p 298.