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## Albinism and anatomy

The average clinician going about his or her daily work may not have the time or the inclination to search for rational explanations for many phenomena the exact mechanisms of which are not apparent but which tend to be taken for granted. Examples of the sort of thing I mean would be ocular dominance, retinal rivalry, stereopsis, diplopia and confusion, and why convergent squints seem to cause amblyopia more often than divergent squints.

I have now discovered another riddle, and I am ashamed to have to admit that I did not know of its existence until I read the article in this month's issue by Russell-Eggitt and colleagues. It appears that albinos have an anomalous chiasmal crossing arrangement, something I expect every ophthalmologist knows except me. Having heard of this anatomical oddity for the first time, I was prompted to wonder what lay behind it. At first sight it appears to be just an associated condition without any obvious causal relationship. This starts to seem unlikely, however, when we learn that all albinos have the anomaly; surely it must be very closely related to the albinotic state.

Digressing for a moment, may I remind readers of the discoveries of the last few years concerning the laying down of the visual pathways during postnatal development? The general principle which has been established is that the development of the intracerebral visual pathways depends to some extent on the input of appropriate stimuli. We have been told, for example, how the anatomy of the lateral geniculate body can vary according to whether or not occlusion has been applied to one eye in experimental animals, and the same sort of thing has been suggested in the occipital cortex.

The situation in albinism, as explained and illustrated in the current article, is that in the central 20° of the field in one eye, retina stimulated by light coming from the opposite side, which would normally be expected to project to the ipsilateral cortex, does not do so but projects back to the opposite side. (I prefer to talk about the 'opposite side' rather than the 'nasal

side' because it has more meaning in the context of what comes next.) I found this revelation profoundly disturbing for the following reason. I have always thought (and taught) that everything we 'relate to' (to use a bit of slightly unpalatable but I hope in this case appropriate jargon) on one side of our world is processed on the other side of our brain. This shows itself in many ways, not least in a variety of forms when hemispheric dysfunction occurs for one reason or another. The simplest example is in the unawareness of loss in many patients with homonymous hemianopia. It is not that they cannot see on one side, but that one side from a visual point of view has ceased to exist; they cannot even think about it.

This strict concept of 'side' implies a changeover point where right gives way to left at the centre. There is probably a small area at the centre which the brain recognises as centre and where the concept of one or other side does not operate. Such is probably true of the most basic of bodily functions, eating, excreting, and reproducing. We can probably add central vision as the fourth 'centralised' function. It is possible that, whereas the right and left sides are mediated by the opposite sides of the brain, central functions are bilaterally represented (sparing of the macula might be an example of this). Perhaps therefore the peculiar chiasmal arrangements of the albino are not so difficult to understand after all. Because their central vision is poor from an early age they may have developed a much less concentrated version of the 'centre'. Putting it another way, we may say they may have an extended central zone where right and left are not perceived as such but are dissolved into a large (20°) blurred centre. If this were to be the case I should expect a wide central area to be bilaterally represented in the cortex, and this might account for the anomalous decussation found in albinos. It would be interesting to have the views of an expert on this speculative explanation.

REDMOND SMITH

## Cataract surgery

There seems little doubt of the success of modern cataract surgery. The widespread acceptance of extracapsular surgery and the routine placement of a posterior chamber intraocular

lens is encouraged by many reports and papers recording final acuity levels of 6/12 or better for over 80% of patients. For many individual surgeons this figure rises to over 90%.

We can now use such figures with confidence when discussing likely outcomes of projected procedures with our patients and, hopefully, with a clear conscience.

It may therefore come as a surprise to learn how much of our vaunted success is dependent on factors over which we have little influence but take for granted. In Britain at least our patients are still reasonably compliant. What the doctor says tends to go. It does not occur to us that patients will fail to attend for postoperative follow-up appointments, fail to use the prescribed drops, or decline to be tested for or wear necessary spectacles.

In this issue a paper by Dr Al Faran reports the results of modern cataract surgery undertaken at the King Khaled Eye Hospital in Riyadh. The study was made because the staff at the hospital considered that their results were not matching those achieved elsewhere. They report, for instance, final acuities of 6/12 (20/40) or better in 37% of patients, with commensurate figures for those seeing less well. The major responsible factor is the patients' failure to attend for refraction or to wear a spectacle correction. In many parts of the Islamic world and elsewhere the wearing of glasses is equated with blindness and can constitute a serious social stigma. The recorded acuities above are mostly uncorrected and are probably no worse than those achieved in many Western 'centres of excellence', where, as in Riyadh, many contributing surgeons are at various stages of their surgical training. Imperfect techniques and inexperience are bound to be associated with a higher rate of operative misadventure and a wider margin for error with preoperative biometry.

We all have our own favourite parameters for assessing the results of anterior segment surgical procedures. Two valuable ones are the postoperative incidence of retinal detachment and bullous oedema of the cornea. Al Faran reports a detachment rate of 1% and no cases of persistent corneal oedema. Admittedly the follow-up time (mean of nine months only) is far too short to be dogmatic, but one sees nothing here to suggest that the surgeons involved have cause for reproof. A 6.4% rate of vitreous loss following capsular rupture is what one might expect from a surgical team including those in training, while transient postoperative oedema in as few as 4.5% has to be regarded as excellent and

unlikely to be followed by significant numbers suffering late decompensation.

Al Faran's problems are related to factors beyond his control. The high incidence of endemic sight-impairing disease, particularly trachoma and climatic droplet keratopathy (25% in this study), are gradually being overcome with improvements in community health. In my experience droplet keratopathy is more prevalent in the poorer strata of Middle Eastern society and often superimposed on pre-existing corneal scarring, due either to trachoma or smallpox. The elimination of these conditions should therefore be followed by a similar fall in the incidence of droplet keratopathy.

The problems concerned with the refusal of large sections of a community to wear glasses where these are indicated are likely to respond only to the influence of effective universal education, and in Saudi Arabia will take one or two generations. The same applies to the reported sex differences in the patients of this study. The older age groups contain fewer females despite the higher incidence of cataract in Saudi women. For the age groups under 50 the sexes are equally represented, which hopefully indicates already some modification of traditional social behaviour. In time, therefore, Al Faran or his successors will be able to serve a population who will not only be more prepared to report their difficulties but also more inclined to keep their postoperative appointments!

Overseas students in London have frequently complained (to me) that sophisticated surgical techniques which have so dramatically altered patients' expectations have no relevance to the developing world. Poorer conditions called for inferior methods – intracapsular extraction and the Graefe knife. Dr Al Faran has demonstrated how wrong this is. How much worse off his patients would have been had they not had the benefit of excellent modern management. Other surgeons of whom we hear, working under even worse constraints, both physical and cultural, struggle to follow the precepts of their teachers and to maintain the highest possible standards. They must be given every possible encouragement to persevere.

ARTHUR STEELE

## Histogenesis of retinoblastoma

More than a century of controversy has surrounded the cell of origin of retinoblastoma. At the outset Virchow described the tumour as a glioma in the belief that it arose from the glial cells of the retina.<sup>1</sup> Subsequently, in a report of a single case, Flexner was the first to describe the rosettes which may be present in retinoblastomas and to designate this tumour a neuroepithelioma.<sup>2</sup> Later still Wintersteiner described rosettes in a series of cases and substituted the term neuroepithelioma for glioma, whether or not rosetting was present.<sup>3</sup> Both authors regarded rosettes as an attempt to form photoreceptors, leading Verhoeff to suggest the description 'retinoblastoma' in order to indicate the origin of all histological variants of the tumour from embryonic retinal cells and to parallel the name 'neuroblastoma.'

The term retinoblastoma was adopted by the American Ophthalmological Society in 1926.<sup>4</sup> In the same year Bailey and Cushing produced a classification of brain tumours based on histogenesis.<sup>5</sup> The medullary epithelium lining the

embryonic neural tube differentiates into three groups of cells: the neuroblastic series which gives rise to neurons, the spongioblastic series which forms the glia, and the medulloblastic series of cells which are primitive and undifferentiated and which may lead to either glia or neurons. Using gold and silver stains they classified each tumour according to the type of cell predominating. Several attempts were made to apply the same principles to retinoblastoma.<sup>6-10</sup> Using silver impregnated preparations, Muñoz-Urra identified spongioblasts as well as astroblasts and astrocytes in the histogenesis of retinoblastoma.<sup>6</sup>

Parkhill and Benedict could not demonstrate any cell processes or fibrils indicative either of glia or of neurons using special stains and regarded the cells they saw as primitive and undifferentiated.<sup>7</sup> They postulated that the tumour was derived by dedifferentiation of normal astrocytes or Müller cells rather than from primitive precursors. They argued that the rosettes in retinoblastoma represented an attempt to



## Cataract surgery.

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