Functional reinnervation in kitten visual cortex

MOST cells in the adult cat visual cortex, and even in the very young kitten can be activated by visual stimuli presented to either eye[1-3]. The retention and refinement of these normal binocular connections depends critically on simultaneous correlated visual experience through both eyes early in life. If a kitten is deprived of vision in one eye during a 'sensitive period', which stretches from about 3 weeks to 3 months, the normal pattern of functional connections is grossly disrupted; the deprived eye loses its influence and most cells can only be driven by stimuli through the experienced eye[4]. Blakemore and Van Sluyters[5] have recently shown that this situation can be partly or totally reversed by covering the previously experienced eye and opening the deprived eye, and allowing that eye a substantial period of visual experience. Thus one set of afferent terminals, robbed of almost all influence in the visual cortex, can re-establish working connections. We have now analysed the form and rate of this functional reinnervation.

In six kittens the lids of the right eye were sutured together at or before the time of natural eye opening (normally 6–10 d). Reverse suturing was performed at the age of 5 weeks: the lids of the right eye were opened and those of the left eye sutured shut. All precautions were taken to ensure that the animals never received simultaneous binocular vision. As a control, one kitten was monocularly deprived in the right eye for 5 weeks, and recordings taken immediately.

Animals were prepared for electrophysiology under barbiturate (Brietal) or steroid (Althesin) anaesthesia. They were then paralysed with Flaxedil and artificially respirated with 75–80% nitrous oxide[6]. The corneas were covered with contact lenses and 3 mm artificial pupils, and refractive errors corrected with supplementary lenses. Single units in the visual cortex were isolated with tungsten-in-glass microelectrodes[7], and their action potentials were conventionally amplified and displayed.

All penetrations were later shown histologically to be largely or wholly within area 17. The electrode was driven down the medial bank of the postlateral gyrus, obliquely crossing a large region of cortex. The first cells encountered in each penetration had receptive fields within 5° of the area centralis and all receptive fields were within 15°.

We studied the responses of neurones to flashing and moving patterns back projected on to a tangent screen 57 cm from the cat's eyes. Receptive fields were classified into one of five groups: orientation selective[1] (simple, complex or hypercomplex), pure direction selective, orientational bias, nonoriented or visually unresponsive[5]. Units with the brisk response, fibre waveform and clear concentric receptive field organisation of afferent fibres from the lateral geniculate nucleus were excluded from the analysis. For all cells that were excited by visual stimuli we assessed the relative responsiveness through the two eyes and classified them using Hubel and Wiesel's seven-point scale of ocular dominance[1]. Cells in groups 1 and 7 are solely monocularly driven, group 1 by the eye contralateral to the recording site, group 7 by the ipsilateral eye. Group 4 cells are equally influenced by the two eyes. Groups 3 and 5 are slightly more strongly driven by the contralateral and ipsilateral eyes respectively; groups 2 and 6 are strongly dominated by the contralateral and ipsilateral eyes respectively. All recordings were made from the right hemisphere, contralateral to the initially experienced left eye.

The ocular dominance histograms in Fig. 1a show the results for 27 cells from the control animal, monocularly

deprived in the ipsilateral eye until recording at 5 weeks of age, and for 189 cells from six kittens reverse sutured at 5 weeks and allowed various periods of time in which to use the previously deprived ipsilateral eye. As expected, the first histogram shows that 5 weeks of monocular deprivation silenced virtually all connections from the deprived ipsilateral eye: almost all cells are in group 1. But 3 d after reverse suturing many neurones could be driven by the initially deprived eye. After only 1 week the process was about half complete, and within 3 weeks there was almost no influence from the previously dominant eye. These data are presented graphically in Fig. 1b where the proportion of cells dominated by the initially deprived eye is plotted against the number of days after reverse suturing.

We observed striking changes in the functional architecture of the cortex during reversal of monocular deprivation. Cells were clustered into distinct regions dominated by one eye or the other—the 'ocular dominance columns' of Hubel and Wiesel[8]. We judged the dimensions of these columns by the distances between obvious switches in ocular dominance during each long penetration. The relative size of the columns seemed to change from animal to animal in an ordered manner. In kittens where the two eyes commanded roughly equal numbers of cortical cells, the columns for the two eyes were of about the same size; in the more
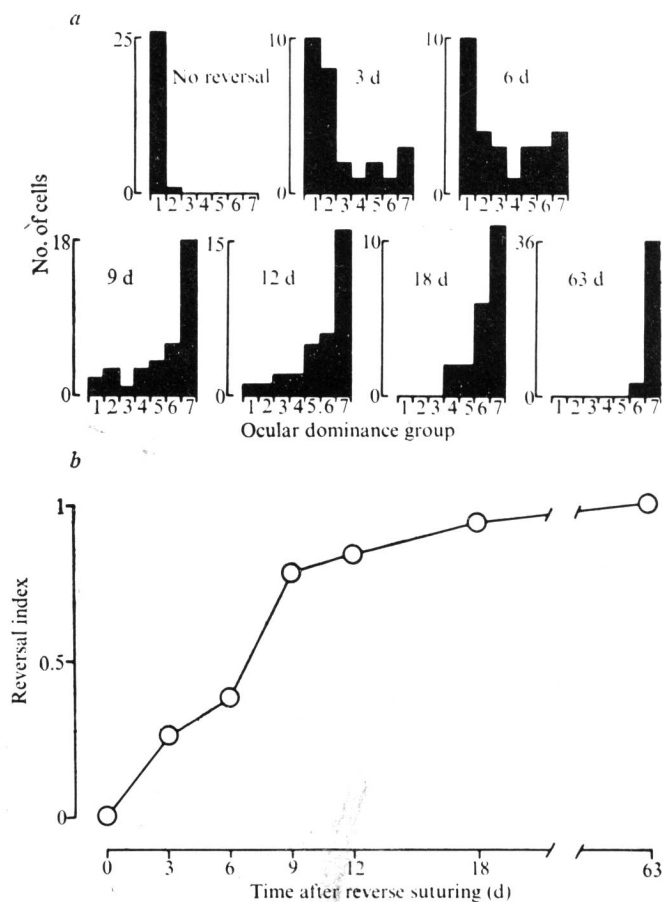

Fig. 1 *a*, The ocular dominance histograms of 216 visually responsive cells from the series of seven kittens recorded at varying times after reversal of lid suture. The ocular dominance classification is described in the text. *b*, The process of reversal is shown graphically, plotting reversal index against days after reverse suturing. The reversal index is calculated by dividing the number of cells dominated by the previously deprived eye (groups 5, 6 and 7) by the total number of cells.

extreme cases the regions devoted to the eye that, overall, dominated fewer cells were smaller and sparser.

Thus, after reverse suturing, areas dominated by the initially deprived eye appear, then expand and ultimately occupy the whole cortex. Such an orderly anatomical progression suggests that this physiological recapture may represent actual growth and reorganisation of afferent terminals, not merely the strengthening of existing but silent synapses. If so, it implies that the mammalian central nervous system, at least early in life, has some powers of rapid regeneration.

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