Eye (1991) **5**, 27–30

Cortical Development and Visual Function

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Summary
Hypotheses are advanced regarding some of the processes underlying the development of an accurate retinotopic map in striate cortex, and the development of cortical magnification. In the first case it is suggested that competitive pruning of afferent synapses may be important in the increase in grating acuity seen in infancy, and that this process may be disrupted in anisometropic amblyopia. In the second it is argued that the development of vernier acuity in infancy may reflect changes in cortical magnification, and that this may be due to increasing functional independence of the columnar units in striate cortex.

Visual function develops rapidly in the last trimester of pregnancy and in infancy. During this time there is an improvement in the sensitivity of those visual functions such as grating acuity, which are present in the newborn, together with the development of new functions, such as stereopsis and orientation discrimination. How does cortical development in infancy contribute to this? In the mature primate visual system the striate cortex 'V1' is the only cortical area known to receive an input directly from the lateral geniculate nucleus (LGN) of the thalamus. V1 is organised as a retinotopic map of the visual field with adjacent points on the retina projecting to adjacent points in V1. The representation of the visual field in V1 is magnified compared with that in the retina. That is to say; the area of V1 representing a specific area of the visual field is larger than the corresponding area of retina and this is particularly so for the macular area. In this paper two developmental processes in visual cortex which are possibly involved in the development of retinotopy and cortical magnification are discussed, and suggestions made as to how they might influence measurable aspects of visual function.

Development of Retinotopy in V1

Afferent axons from the LGN initially make widespread excitatory connections in V1. During development these are circumscribed to give a more ordered retinotopic arrangement.[1] In the immature situation where each afferent makes widespread cortical connections, each point in V1 will receive projections from comparatively widespread areas of retina and the image of the retina on the cortex will be subject to 'neural blurring'. With maturation, not only is the arrangement of cortical afferents more ordered, but they are reduced in number and spread less widely. Two interrelated processes appear to be involved: the spatial ordering of afferents from the LGN and the 'pruning' of their synaptic terminations.

Synapses whose activity is associated with depolarisation of the target neurone membrane are stabilised and their transmission efficiency increased. Such synapses tend to be those which are active in synchrony with others at the target cell, and are thus statistically likely to come from adjacent retinal points.[2] In contrast those synapses which are active during target neurone inactivity may be

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reduced in efficiency.[3] Accurate cortical retinotopy may therefore depend on patterns of activity in developing cortical afferents.[2]

Synaptic pruning is known to occur in the cortex in infancy, but it is not known if the principles governing cortical synaptic pruning are the same as in subcortical structures, such as the tectum, where it has been most studied. In the tectum afferents compete for 'synaptic space' on target neurones during a critical period of development. Synapses whose activity is asynchronous with respect to target neurone depolarisation are at a comparative disadvantage and are lost. In experimental situations where any competitive advantage between afferents is removed, such as rearing under stroboscopic light, which effectively synchronises activity over the whole retinal area, or blocking axonal conduction with tetrodotoxin, then pruning still takes place, but in a random manner. In the optic tectum, this leads to the persistence of aberrant projections, which produce disorder in the normal retinotopic arrangement.[4]

Grating acuity develops rapidly in infancy. Part of this increase can be attributed to foveal maturation,[5] part to the role of inhibitory processes in developing the mature 'centre-surround' type of receptive field in LGN and cortex, and, possibly, part to cortical influences on the LGN, which may influence receptive field size.[6] It seems probable that pruning of cortical afferents is also involved. There is, as yet, only indirect evidence for this. Suturing the lids of one eye during development may increase the cortical 'territory' occupied by afferents from the normal eye at the expense of afferents from the sutured eye,[7] indicating that competitive advantage does play a part in the development of the map of the visual field in V1. If, as in tectum, reduction of the possible competitive advantage between afferents from one eye leads to the persistence of aberrant connections in V1, then this would produce a disordered representation of the retina on V1. The effects of such disorder on grating acuity should be the same as blurring of the image, however positional acuity would be affected independently of the blurring, as positional information from the retina would no longer be accurately represented on V1. A persistently defocussed image on one retina, as in anisometropic amblyopia, might be expected to synchronise activity and thus reduce competition between afferents from that eye and lead to a spatially disordered map in V1. Watt and Hess[8] have shown that positional representation in the amblyopic eye in anisometropic amblyopia is indeed spatially disordered and cannot be mimicked by blurring. It must be stressed that the proposed connection between anisometropia and disordered synaptic 'pruning' is highly speculative.

Development of Cortical Magnification

Mature cortex has a columnar organisation, with columns of functionally linked cells extending from the cortical surface to subcortical white matter. The basic processing unit in V1, corresponding to an individual 'point' on the cortical map of the retina, is thought to be the 'hypercolumn', which can be subdivided into two 'ocular dominance' columns each of which responds preferentially to input from one eye and can be further subdivided into a number of 'mini-columns', each responding to objects at a particular orientation.

The cortical magnification factor is defined as the extent of striate cortex corresponding to a degree of arc in visual space and is related to the density of retinal ganglion cells.[9] It is greatest in the area of cortex representing the fovea and diminishes with eccentricity. Grating acuity appears to correspond to retinal ganglion cell density, and in common with it, is lower in the area of the visual field subtended by the temporal retina, as compared to that subtended by the nasal retina. If one cortical point is considered to correspond to a unit of cortical area, and one retinal point to a retinal ganglion cell, then for some visual functions, such as contrast sensitivity and vernier acuity, performance appears to depend more on the number of cortical 'points' involved than the number of retinal 'points'.[10] Cortical magnification may therefore be partly independent of retinal ganglion cell densities.[11] Fahle and Schmid[12] have suggested that vernier acuity depends more on positional information present at a cortical level than at a retinal level. I wish to suggest

that the development of vernier acuity may provide an index of the development of cortical magnification. Vernier acuity in infancy is initially lower than grating acuity, but increases faster[13] to be approximately 1.5 octaves greater at nine months[14] suggesting that different developmental processes are involved. Vernier acuity reflects the accuracy with which the position of a point on the retina can be determined. I suggest that, whereas the increase in grating acuity in infancy is dependent on changes in the receptive fields of cortical neurones, as discussed above, the increase in positional accuracy may depend on changes in the number of independently active hypercolumns subtending a given area of the visual field.

The receptive fields of adjacent hypercolumns are unlikely to be identical even though there may be considerable overlap. Thus, although the positional accuracy of any one cortical column is limited by its receptive field size, more accurate information on position in the visual field could be obtained by integrating the responses from an array of neighbouring columns with overlapping receptive fields, each of which returns slightly different positional information.[15] This is only true if the columns are active independently. If activity in one column spreads to and activates neighbouring columns, then positional accuracy would be even less than that provided by the receptive field of an isolated column, as is the situation in early infancy.[14] Effective cortical magnification could therefore be lowered by a reduction in the number or density of cortical hypercolumns, or by a spread of activity between columns possibly due to a reduction in the electrical isolation provided by myelin and glial cells or due to a reduction in inter-columnar inhibition.

Columnar organisation of cortex is present from the beginnings of cortical development, although it is not certain how developing columns are related to mature hypercolumns. When neurones migrate from the ventricular zone to the cortical plate, they do so in ontogenetic columns, developing neuronal interconnections and passing through waiting thalamic afferent terminations en route.[16] During the last trimester and in infancy the complexity of neuronal dendrites and interconnections increases rapidly in V1[17] and the width of each column increases.[18] Since the number of cortical neurones is thought to be fixed by the end of the second trimester[16] later cortical growth probably reflects columnar widening due to neuronal differentiation with axonal and dendritic development, glial cell development, and myelination, rather than an increase in the number of functional columns. I suggest, tentatively, that increasing columnar width together with the development of inhibition between and within columns, results in the functional independence of adjacent columns and that this may underly the apparent increase in cortical magnification suggested by increasing vernier acuity in infancy.

Intrauterine growth retardation (IUGR) is known to impair cortical development, with particular effects on myelination[19] and dendritic development. Most IUGR in humans takes place after the second trimester and therefore probably after neuronal numbers are determined. Vernier acuity is significantly reduced in IUGR infants at nine months of age compared to normal controls, although grating acuity is normal.[14] Vernier thresholds in the IUGR infants were significantly correlated with head size. This study suggests that factors that might impair inter-columnar independence affect vernier acuity but not grating acuity, implying an effect on cortical magnification.

The relations between cortical developmental processes and measurable visual functions remain speculative. Increasing knowledge of the development of the cerebral cortex, sensory electrophysiology and psychophysics in the fetus and infant should allow us to design experiments aimed at elucidating such relations.

Keywords: Cortex, Development, Vision.

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