Reprinted from Nature, Vol. 343, No. 6253, pp. 16-17, 4th January, 1990

NEUROBIOLOGY

The club-sandwich mystery

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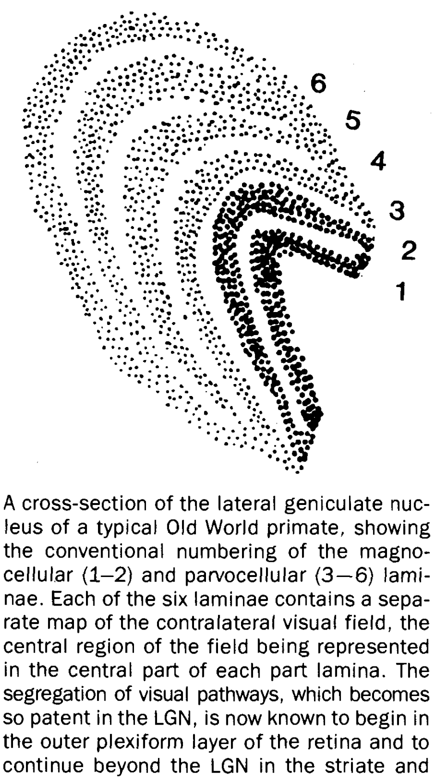
"THE whole organization of the human lateral geniculate nucleus (LGN) cries aloud that something is being segregated. The question before us here is simply: *what*?" It was with this question that the late Gordon Walls opened his engaging monograph on the LGN, the knee-shaped nucleus of the mammalian brain that lies in the visual pathway between the retina and the striate cortex[1]. A paper by Schiller, Logothetis and Charles on page 68 of this issue[2] offers an answer to Walls's question.

Medical students are still told that the LGN is a mere relay station. But to the neurobiologist the LGN is provocative in its exquisite organization. In Old World primates and in man, the lateral geniculate consists of six layers, each eye providing the inputs to three layers (see figure). The two layers that lie on the inside of the geniculate 'knee' consist of large cells and hence are known as the 'magnocellular laminae'; their main retinal inputs are from the axons of the large cells that Perry and Cowey[3] called Pα cells. The remaining four layers consist of small cells and so are known as the 'parvocellular laminae'; their main retinal inputs are from the class of cells known as Pβ cells. If we number the six laminae from the inside of the knee outwards, then layers 1, 4 and 6 of each geniculate are drawn from the contralateral eye and layers 2, 3 and 5 from the ipsilateral. Each lamina forms a map of the contralateral half of the visual field, and, what is most remarkable, the six maps are in precise alignment[4]. Gordon Walls[1] likened the lateral geniculate to a club sandwich: the toothpick piercing the sandwich corresponds to a single direction in visual space.

Why is the lateral geniculate stratified? How is the task of analysing the visual world distributed among the laminae (and their cortical projections)? Why does the developing visual system go to such trouble to align the six maps in the LGN? And why indeed does the LGN exist? These must be interdependent questions. But it is the second of them that has been most explicitly addressed, and three types of evidence have been offered.

First, electrophysiological recordings have been made from the primate LGN while various stimuli were presented to the eye. When the stimulus varies only slowly in space (that is, it is of low spatial frequency), parvocellular units typically exhibit chromatic selectivity, being excited by one part of the visible spectrum and inhibited by another part. Because the opposing receptor inputs to such cells are arranged in concentric areas of excitation and inhibition, and because these 'receptive fields' are small, parvocellular units are also sensitive to stimuli of high spatial frequency, whatever the stimulus wavelength. By contrast, the magnocellular units tend to respond maximally at lower spatial frequencies, exhibit little chromatic selectivity and are often more transient in their response[5-7].

One might think that the functions of the different LGN layers would be readily clear from such studies of single cells. But in fact there has been fierce controversy about the role of the parvocellular system in the analysis of spatial patterns. One extreme view was adopted by Shapley and Perry[5], who pointed out that magnocellular units are much more sensitive to spatial contrast than are parvocellular units and thus may be responsible for the spatial analysis of low-contrast stimuli even at relatively high spatial frequencies. This account, in which colour vision becomes the primary office of the parvocellular system, is rather implausible — there are about 7.5 times as many parvocellular units as magnocellular[8]; the magnocellular system does not sample the retina with the resolution needed to account for psychophysical performance[6]; and there is a well-developed parvocellular system in dichromatic platyrrhine monkeys. (For the detailed arguments see refs 5 – 7 and 9.)



A cross-section of the lateral geniculate nucleus of a typical Old World primate, showing the conventional numbering of the magnocellular (1–2) and parvocellular (3–6) laminae. Each of the six laminae contains a separate map of the contralateral visual field, the central region of the field being represented in the central part of each part lamina. The segregation of visual pathways, which becomes so patent in the LGN, is now known to begin in the outer plexiform layer of the retina and to continue beyond the LGN in the striate and prestriate cortex.

Why has the electrophysiological approach been so inconclusive? One reason is that we cannot properly compare the discriminatory performance of individual cells in layers 1 and 2 with the performance of individual cells of the parvocellular layers. If the magnocellular system is the more primitive or less encephalized system ("protopathic" in Henry Head's terms), then it is likely to have carried out much of its spatial integration before the LGN, whereas the "epicritic" parvocellular system is more encephalized and postpones its spatial integration to the cortex. So it is unjust and artificial to use a cell-for-cell comparison to judge the two sets of signals as they hurry through the LGN.

A second approach has been psychophysical: here the experimenter presents, to a human observer, stimuli that are thought to excite only one of the two channels. Particular favourites have been 'equiluminant' stimuli (stimuli that vary only in colour and not in luminance), which are held to silence all but the parvocellular pathway. Several perceptual functions (such as stereopsis, movement discrimination and figure–ground differentiation) are impaired when the stimuli are equiluminant, whereas others survive[7]. It is concluded that the signals of the colour-selective laminae are unable to sustain those perceptual functions that fail at equiluminance, but are able to sustain those that survive. But these arguments depend fatally on a reification of the psychophysical construct of 'luminance'[10].

Consider first a perceptual function that does survive when the stimulus is a red figure on an equiluminant blue ground. At the edge between figure and ground the long-wave cones must detect a spatial transient of one sign and the middle-wave cones must detect a transient of the opposite sign. We are asked to believe that the two transients will exactly cancel each other out in all the cells of the magnocellular pathway, a pathway that seems especially designed to detect transients. This is contrary to biological plausibility[10] and to empirical evidence[11]. Consider now a perceptual function that *is* impaired at equiluminance. We need not conclude that this task is primarily the charge of the magnocellular system. For equiluminance presents a potentially disruptive stimulus to the parvocellular system: whereas long-wave on-centre units of the Pβ type normally give the same sign of response to spatial transients as do middle-wave on-centre units, this yoking is lost at 'equiluminance', when the two types of on-centre unit (and the two types of off-centre unit) will give responses of contradictory sign[12].

The third approach is to make selective lesions in the LGN. Merigan and Eskin[13] tested monkeys after systemic administration of a neurotoxin, acrylamide monomer, which damages the small-cell system but seems to spare other pathways: the monkeys were still well able to detect high temporal frequencies or low spatial frequencies, but were badly impaired at low temporal frequencies and high spatial frequencies. More recently, the same laboratory has reported the consequence of making a local LGN lesion by injecting ibotenic acid into the magnocellular layers: the impairment was then apparent only at high temporal frequencies and low spatial frequency[14].

It is lesion experiments of this type that are now reported by Schiller, Logothetis and Charles. They have trained their monkeys to deflect their gaze to the part of the visual field that contains a target stimulus and have then made local lesions in the LGN. Their answer to Walls's question is one that would have been conventional in the mid-1970s, before the recent controversies and before the mischief of equiluminance was abroad. The parvocellular laminae appear to carry the signals that mainly sustain perception of colour, texture, shape and fine stereopsis, whereas the magnocellular signals sustain the detection of movement and flicker. □

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Printed in Great Britain by Turnergraphic Limited, Basingstoke, Hampshire