Lecture 2:
An Exemplar Neural Mechanism: The Brain's Visual Processing System

In this lecture I will put further flesh on the notion of a mechanism and the idea that adducing a mechanism can provide an explanation by examining one of the few cases in cognitive neuroscience in which the basic structure of the relevant mechanism is known, the visual processing system. Since my interest is not just in mechanisms as finished explanatory products, but in the process of developing an account of a mechanism, I will develop the analysis historically.

When first approached, visual processing both looks straightforward and utterly baffling. We are not aware of intermediate operations that the brain is performing. But this should not be surprising. Natural systems typically work very smoothly and fail to reveal their components. Even when one recognizes that the retinal receptors only respond to individual bits of information about the visual scene an animal is confronting, it might seem that the task is still relatively straight-forward. One only needs to specify some procedures or algorithms that will allow one to construct the visually recognized world from these inputs. Adopting such a perspective and recognizing the utility of vision for artificial intelligent agents, Marvin Minsky, a pioneer in the development of artificial intelligence, presented the challenge of developing a computer vision system to an undergraduate student as a summer project in 1966. Although Minsky's choice of a student to confront the task, Gerald Sussman, was inspired, he grossly underestimated the challenge. Fifteen years later David Marr (1982) offered a sketch of how develop a computer system that could accomplish visual tasks. Although his sketch was motivated by information about the brain, it turns out to correspond in only limited ways to the manner in which the brain actually performs the activity of seeing.

As noted in the previous chapter, a critical question in developing a mechanistic model is the level of organization at which it is situated. Since neurons are prominent architectural components of brains, one could, for example, try to develop an analysis in terms of individual neurons. For many purposes, focusing on individual is relevant. But for understanding the overall mechanisms of vision it is too low a level. The component operations that figure in visual processes occur at a higher level, involving population of neurons working together. Brodmann (1909/1994) differentiated areas in the brain in terms of their different cytoarchitectural properties, which he thought would likely correspond to function. More recent efforts at brain mapping have been able to employ tools beyond what Brodmann had available.

Focusing just on areas that seem to be responsive to visual stimuli (e.g., as measured by single-cell recording), David van Essen and his collaborators have identified 32 distinct regions on the cortex of the macaque monkey that are associated with vision. Together these constitute more than half the cortical surface of the macaque (Felleman & van Essen, 1991; van Essen & Gallant, 1994). Since the cortex is a highly convoluted surface, van Essen developed algorithms for developing flat maps that preserve topological relations, and these 32 cortical areas, plus the retina, lateral geniculate nucleus, superior colliculus, and pulvinar, are shown in Figure 1.

These 32 cortical regions are highly interconnected. Approximately one third of the possible pairwise connections between areas are in fact found in the macaque. The majority of the connections are reciprocal, but typically there are differences in forward, backwards (recurrent) and lateral connections that allow neuroanatomists to differentiate them. (As Brodmann established, cortex generally consists of six layers of cells, and forward projections generally originate in the middle layers whereas recurrent connections project from the lowest and highest layers and lateral connections project from all layers.) This enables researchers to arrange the different areas hierarchically. In addition, as we shall see, relying on techniques such as lesion analysis and single-cell recording, researchers have been able to identify the features of stimuli that generate activity in several of these areas, leading to suggestions as to the sorts of information they are processing. Figure 2, from van Essen and Gallant, arranges hierarchically those areas for which functional hypotheses as to their operation have been advance and uses icons to indicate these functions. Although many known structures have not yet been situated in this functional map, it clearly provides a sketch of a mechanism of visual processing.

I will return at the end to this overall characterization of the mechanism of visual processing. While still incomplete, this account is the product of a host of research efforts over the past two centuries, and reviewing them provides perspective on what it takes to develop an understanding of such a mechanism.

Getting Started: Localizing Visual Processing in Striate Cortex

A major first step in developing a mechanistic explanation is to identify the mechanism itself—the system that is taken to be responsible for the phenomenon of interest. By focusing on a given system as the mechanism one is not denying that it interacts with other systems. But one is maintaining that the central activities occur within it. In the case of visual processing, a variety of research in the late 19th and early 20th century focused on the occipital lobe, especially a portion of that appeared differently from the rest of cortex due to an unusual striation pattern that had first been observed by Francesco Gennari in the course of examining frozen sections of human brains. (Glickstein, 1988; Glickstein & Rizzolatti, 1984). Grafton Elliot Smith (Elliot Smith, 1907) named it the *area striata*; the area is now often referred to as the *striate cortex*. The area was also distinguished on cytoarchitectural grounds in a wide variety of species by Korbinian Brodmann (1909/1994), who assigned this area the number 17 since it was the 17th cortical area he had examined. (Much later the terms *primary visual cortex* and V1 also came to be applied to this area.)

As with many neural processes, the first indication of the mechanism for visual processing resulted from analysis of patients with lesions. Bartolomeo Panizza (1855) on the basis of a study of patients who experienced blindness after strokes damaged the occipital lobe, proposed the occipital lobe as the cortical center for vision. He corroborated these observations on several other species in which lesions to the occipital lobe produced blindness, but his publications were largely ignored, perhaps because they only appeared in local Italian journals. But reports soon started to accumulate more broadly of visual deficits accompanied by damage to the striate cortex (Henschen, 1893; Wilbrand, 1890) and of visual deficits in animals after

lesions to the area (Munk, 1881/1960; Schäfer, 1888b). These results from lesion studies were supported by neuroanatomical evidence produced by Pierre Gratiolet (1854) and Theodor Meynert (1870) which indicated that the optic tract, which first projected to an area of the thalamus known as the *lateral geniculate nucleus* (LGN), then projected on to the occipital lobe (Meynert traced the projections more specifically to the area surrounding the calcarine fissure). Subsequently Paul Flechsig (1896) identified the striated area in particular as the target of the projections from the LGN.

But not everyone agreed with these findings. In particular, David Ferrier, recognized by many as the leading neurologist of the time, rejected the occipital cortex and contended that the angular gyrus, an area in the parietal lobe, was the locus of visual processing (1876). (See Figure 3) He relied in part on lesion studies from which he reported that bilateral lesions to the angular gyrus resulted in blindness, whereas large lesions in the occipital lobe produced little impairment. But even more he relied on a technique of mild electrical stimulation, which yielded the result that that stimulation of the angular gyrus caused monkeys to move their eyes toward the opposite side. Ferrier (1881) later moderated his opposition to a occipital lobe locus, holding that both the angular gyrus and the occipital lobe figured in vision and that only lesions to both could produce complete and enduring blindness, but he continued to emphasize the angular gyrus.

Such disagreements over the locus of the mechanism responsible for a given phenomenon are quite common in science (Bechtel & Richardson, 1993). Noting them helps us recognize that mechanisms in nature do not come pre-labeled. In this case the neuroanatomical information plus the overwhelming amount of lesion results favoring the occipital lobe caused Ferrier's proposal to be rejected.[1] By the 1890s the striate cortex was identified as the locus of visual processing

But how does it do it? (Or, what does it do?) Decomposing the Mechanism

Identifying a candidate mechanism is only the first step in explaining a phenomenon. To explain how a mechanism works, one needs to decompose it into components and figure out what operaton those components perform. A major step in this direction was Salomen Henschen's (1893) study of lesion sites which produced vision deficits in humans. He showed that deficits in

[1]In retrospect, it appears that the reason Ferrier's lesions of the angular gyrus produced deficits in vision was that his incisions cut deeply and severed the nerve pathways from the thalamus to the occipital cortex (Finger, 1994). Moreover, his failure to eliminate vision with occipital lobe lesions was due to incomplete removal of the visual processing areas in the occipital lobe. But these shortcomings in his technique were only established much later and did not figure in settling the conflict. Moreover, one should not just infer that Ferrier misapplied the lesion techniques because he cut too deeply. Before this could be regarded as an error, difference between the underlying white matter and the grey matter had to be appreciated and standards for conducting lesions research developed. Standardized methods are often the outcome of such scientific controversies—they cannot be appealed to in settling the controversies.

different parts of the occipital lobe produced blindness in different parts of the visual field and proposed that the occipital lobe must be topologically organized so that different parts of the retina projected onto different areas of the visual cortex (leading him to refer to it as the *cortical retina*). The occipital lobe map that Henschen proposed lays the projections out in the reverse manner of what is now accepted. While it might seem surprising that someone could discover a topological structure, and yet get all the locations reversed, such developments are surprisingly common in the history of science. It is indicative of how difficult it is to extend beyond individual highly suggestive findings to generate a systematic account.

Discovering the correct topographical layout of striate cortex resulted from amassing much more data. Tatsuji Inouye was able to study 29 individuals who sustained highly focal damage to the occipital lobe during the Russo-Japanese war (as a result of new bullets introduced by the Russians, see (Glickstein, 1988), and with the additional data points was able to determine that the central part of the visual field projects to the rear of the occipital lobe and the peripheral parts to the front. A similar study by Gordon Holmes (1918) during World War I generated a even more detailed and accessible diagram of the topological projection of parts of the visual field onto the visual cortex (see Figure 4).

The discovery of topological maps revealed that the operation of the striate cortex was distributed among areas, each of which processed stimuli from a specific region of the visual field, but it could not reveal what this processing involved. For this a different technique was required. Since the nervous system was recognized to be an electrical system, what was required was some way to determine the local electrical activity in a brain region and relate this to the task the region was doing. Thus, many researchers were eagerly committed to discovering a way to record the electrical activity of an individual cell, a challenge that was finally met by Edgar Adrian in the 1920s (Finger, 2000).

The first application of single-cell recording to visual processing was to confirm the topological representation of the visual field on the striate cortex (Talbot & Marshall, 1941). But it was soon put to the task of determining what the striate cortex was doing with this information. One way to approach this question was to try to determine what features of visual stimuli elicited a response from cells in striate cortex. Steven Kuffler (1953) used to approach to determine that cells in the retina and the lateral geniculate nucleus responded to dark or light spots on the opposite background (see Figure 5, a and b). David Hubel and Thorsten Wiesel, two researchers in Kuffler's laboratory, attempted to replicate this research with cells in striate cortex, initially with no success. Eventually, and apparently serendipitously, they discovered that cells in striate cortex responded to bars, not spots, of light (Figure 5, c, d, e, and f).

Over the first ten years of their collaboration, Hubel and Wiesel probed the striate cortex of both cats (Hubel & Wiesel, 1962) and monkeys (Hubel & Wiesel, 1968) and discovered a rich organization of cells with different response patterns. What they termed *simple cells* had receptive fields with spatially distinct *on* and *off* areas along a line at a particular orientation (most typically, they had a long, narrow *on* areas sandwiched between two more extensive *off* areas). Whereas simple cells were sensitive to stimuli only at a given retinal location, what Hubel and Wiesel termed *complex cells* were responsive to bars of light at a particular

orientation anywhere within their receptive fields. Many complex cells were also sensitive to the direction of movement of bars within their receptive field. Complex cells are found primarily in layers 2 and 3 and 5 and 6.[2] In their papers from this period Hubel and Wiesel also distinguished *hypercomplex cells* which responded maximally only to bars extending just the width of their receptive field.[3]

Having identified three types of cells with different response properties, Hubel and Wiesel proposed a decomposition of processing within striate cortex, with one type of cell supplying information to other cells and each carrying out its own information processing. Thus, they proposed that several LGN cells with center-surround receptive fields might all send excitatory input to a single simple cell, causing it to fire when the spots of light detected by LGN cells fell along a line (Figure 6). Likewise, they proposed that complex cells received input from several simple cells and would respond when any of these simple cells were active. In terms of logic, the simple cells operated like an *and-gates* while the complex cells functioned as *or-gates*.

By inserting electrodes gradually and recording from cells at different depth in the cortex, Hubel and Wiesel also discovered two additional features of the organization of striate cortex. First, when they inserted an electrode at an angle of 30^{o} and recorded at successive locations, the preferred stimulus orientation for cells gradually changed. Over the first 18 locations (approximately 1 mm.) the preferred orientation varied through a full 180^{o}. As penetration continued, a point was reached (arrow) where the variation in preferred orientation suddenly reversed. Second, they discovered that complex cells in striate cortex generally received binocular input, although they tended to be more responsive to input from one eye than the other. If the electrode were inserted perpendicularly rather than at an angle, all the cells encountered would respond to the same orientation with the same eye dominance, leading Hubel and Wiesel to adopt Vernon Mountcastle's proposal of a columnar organization of cortex. They proposed that in one direction successive columns (each .5 mm wide) were dominated by alternate eyes (ocular dominance columns) while in the other direction successive columns were responsive to different orientations of the stimulus (Figure 7).

On the basis of their investigations, Hubel and Wiesel claimed to have discovered the primary function of striate cortex (but with a prophetic caveat to which we will return): "The elaboration of simple cortical fields from geniculate concentric fields, complex from simple, and hypercomplex from complex is probably the prime function of the striate cortex-unless there are

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[2]An important difference between the different layers is that they generally project to different brain areas: layers 2 and 3 project forward to other cortical areas, layer 5 projects backwards to the superior colliculus, pons, and pulvinar, and layer 6 backwards to the LGN.

[3]Hubel and Wiesel (1965) identified such cells only in areas 18 and 19 of the cat and assumed that these cells received their inputs from complex cells. Later, though, they found them in area 17 in both cat and monkey. After Dreher (1972) found cells in cats that were location specific like simple cells but whose response dropped off as the length of the stimulus exceeded an optimum length, they dropped the assumption that they received their inputs from complex cells.

still other as yet unidentified cells there" (Hubel and Wiesel, 1968, p. 239). From our perspective, what Hubel and Wiesel did was discover the structure and operation of a mechanism in striate cortex which took in information about dark and light spots of light detected by the retina and LGN and constructed representations of bars of light that specified both their location in the visual field and their motion. Discovery of the mechanism operative in striate cortex, though, posed a new puzzle. Detecting oriented bars of light in various parts of the visual field does not itself constitute vision. This focused a question for further research: where else is visual information processed, and what does each of these areas contribute? Accordingly, Hubel and Wiesel conclude their 1968 paper with the comment:

> Specialized as the cells of 17 are, compared with rods and cones, they must, nevertheless, still represent a very elementary stage in the handling of complex forms, occupied as they are with a relatively simple region-by-region analysis of retinal contours. How this information is used at later stages in the visual path is far from clear, and represents one of the most tantalizing problems for the future. (Hubel and Wiesel, 1968, p. 242).

Expanding the Mechanism by finding other visual areas

By showing that striate cortex only carried out part of the task of vision, Hubel and Wiesel demonstrated that the initial boundaries on the visual processing system were incorrect. The mechanism had to include more than striate cortex. But expanding the mechanism met confronted an obstacle. At the time Hubel and Wiesel were working, the legacy of early 20th century holism and anti-localizationism, especially the legacy of Karl Lashley, was still prominent. This tradition maintained that except for primary sensory processing areas, cortical areas did not individually perform different component operations. Rather, the cortex operated in a holistic fashion. This conclusion was in part motivated by the general failure to find loss of specific mental capacities with the destruction of cortical region. On the contrary, what seemed to matter was only how much cortex was destroyed with deficits corresponding in severity to the amount destroyed. Lashley termed this the *principle of mass action* and applied it in particular to the area immediately surrounding striate cortex, an area for which he coined the term *prestriate region*. He denied that prestriate cortex played a specifically visual function, insisting: "visual habits are dependent upon the striate cortex and upon no other part of the cerebral cortex" (Lashley, 1950).

For many researchers, one sign of the lack of differentiated function beyond striate cortex was the lack of evidence that these areas were topologically organized in the manner of striate cortex. The very lack of a topographical organization suggested that these areas operated holistically to integrate sensory information (thus, they were designated *association cortex*). Thus, one of the first indications of visual processing beyond striate cortex was Alan Cowey's (1964) discovery, using surface electrodes to record evoked responses, of a second topographically organized area in Brodmann's area 18 (which immediately adjoins area 17); this area came to be known as V2, with striate cortex being designated V1. Using single-cell recording, Hubel and Wiesel (1965) confirmed the topographical organization of this area and identified yet a third area, V3, in Brodmann's area 19. By tracing degeneration of fibers from discrete lesions in striate cortex to areas in surrounding cortex, Semir Zeki (1969) offered collaborative evidence for the existence of these additional areas. Zeki (1971) then extended this approach by creating lesions in V2 and

V3 and tracing degeneration forward into areas on the anterior bank of the lunate sulcus in which "the organized topographic projection, as anatomically determined, gradually breaks down" (p. 33).[4] Zeki labeled the areas into which he traced degeneration as V4 and V4a.[5]

The discovery of topological maps furthered the structural decomposition of the brain and indicated that these areas were part of the mechanism of visual processing. But they did not show directly what operations these areas performed. This required a more direct tool for analyzing function. As with V1, single cell recording played the major role. Zeki (1973) recorded from cells in V4 and found "in every case the units have been colour coded, responding vigorously to one wavelength and grudgingly, or not at all, to other wavelengths or to white light at different intensities" (p. 422). Using a similar procedure to that Hubel and Wiesel used in studying V1, Zeki recorded from successively encountered cells in a perpendicular penetration and found they responded to the same wavelength. Recording from successively encountered cells in an oblique penetration revealed that they responded to different wavelengths. Zeki interpreted this as evidence of a columnar organization dedicated to analyzing color. The next year Zeki (1974) reported on a study recording from cells on the posterior bank of the superior temporal sulcus, an area he would later label V5 and others would designate MT. He found that cells in this area responded primarily to movement, with some firing in response to movements in any direction, but with most being sensitive to the direction, and sometimes the shape of the moving stimulus. As with V5, he found evidence of a columnar organization of movement sensitive cells, with adjacent cells exhibiting slight changes in their preferred orientation. Soon after the topography of these areas was ascertained through single-cell recording, neuroanatomical staining studies revealed that the connections to area V4 were primarily from V2 (1978) and those to area V5 were from V1 (van Essen, Maunsell, & Bixby, 1981).

Zeki's discovery that cells in area V4 were particularly response to color provided a new framework for understanding late 19th century reports of patients with a specific deficit in seeing color, a condition known as achromatopsia.[6] At the time of Zeki's work there were no reports of

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[4]Zeki ends the paper with the following comment about projections to other brain areas: "How the prestriate cortex is organized in regions beyond (central to) V4 and V4a remains to be seen. It is perhaps sufficient to point out at present that the organisation of the prestriate areas would seem to be far more complicated than previously envisaged and that the simplistic wiring diagram from area 17 to area 18, from area 18 to area 19 and from area 19 to the so-called 'interior temporal' area will have to be abandoned. At any rate, we were not able in this study to find any projections to the 'inferior temporal' areas from areas 18 and 19 (V2 and V3)" (p. 34).

[5]During the same period John Allman and Jon Kaas, through single cell recording in squirrel monkeys, traced topographically organized visual areas not only into extrastriate regions but also into temporal and parietal cortexes.

[6]Verrey (1888) and MacKay and Dunlop (1899) had both found patients who could not see colors and connected the deficit with lesions in fusiform gyrus adjacent to the striate cortex. They had construed this as a second visual processing area, one devoted to color perception, but most 19th century researchers dismissed these claims in favor of the supposition of one cortical center for vision in the striate cortex, which might produce achromatopsia with mild lesions and

patients with deficits specifically in motion perception, but in 1983 Zihl, von Cramon, & Mai reported on a patient who, as a result of vascular damage, could not perceive motion. To the patient activities such as coffee being poured into a cup appeared as contiguous shapes, like a glacier. Thus, the designation of specific functions to these prestriate areas was supported by both single-cell recording and lesion research.

The discovery of visual processing areas surrounding V1 which analyzed distinct visual properties such as color and motion significantly advanced the functional decomposition of vision and enhanced the plausibility of identifying a visual processing mechanism in the brain. But these discoveries posed a major question: where is the information about edges, colors, and motion put to use to permit the recognition of objects and events in the world? To address this question researchers had to expand the quest for specialized visual processing areas into yet more anterior parts of the temporal and parietal lobes. The first suggestions\ that areas in the temporal lobe played a specific role in visual processing was actually a late 19th century study by Edward Schäfer (1888a) ostensibly devoted to showing that, contrary to Ferrier's claims, the temporal cortex was not the locus of an auditory center. In monkeys in which either the superior temporal gyrus or nearly all the temporal lobes were removed, Schäfer reports no detectable loss of hearing but describes a deficit in recognizing visually presented stimuli:

> the condition was marked by loss of intelligence and memory, so that the animals, although they received and responded to impressions from all the senses, appeared to understand very imperfectly the meaning of such impressions. This was not confined to any one sense, and was most evidence with visual impressions. For even objects most familiar to the animals were carefully examined, felt, smelt and tasted exactly as a monkey will examine an entirely strange object, but much more slowly and deliberately. And on again, after only a few minutes, coming across the same object, exactly the same process of examination would be renewed, as if no recollection of it remained (p. 375).

Little attention was paid to Schäfer's observations until after a study by Heinrich Klüver and Paul Bucy in the late 1930s in which removal of the temporal lobe in monkeys resulted in a condition they described as *psychic blindness* or *visual agnosia* in which "the ability to recognize and detect the meaning of objects on visual criteria alone seems to be lost although the animal exhibits no or at least no gross defects in the ability to discriminate visually" (Klüver, 1948; Klüver & Bucy, 1938). The effects of the lesions induced by Klüver and Bucy were referred to as a syndrome since the monkeys exhibited a variety of other behavioral changes, including loss of emotional responsiveness and increased sexual behavior. Pribram and Bragshaw (1953) addressed the question of whether these different deficits were due to a common process in the same brain area or to different processes in near-by areas. By demonstrating that different lesions in temporal cortex could generate one or another deficit they showed that the various deficits were due to interrupting different processes. In particular, they traced visual agnosia to lesions of the amygdala and adjacent cortex. Subsequently, Pribram

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full blindness with more serious lesions. One finding supporting this interpretation was that most cases of achromatopsia also manifested scotomas or areas of total blindness, suggesting that one lesion produced both effects.

collaborated with Mortimer Mishkin in localizing visual agnosia specifically to lesions in inferotemporal cortex (Mishkin & Pribram, 1954). Subsequently, through a complex set of lesions involving the striate cortex in one hemisphere and the inferotemporal cortex in the other and the sectioning of the forebrain commisures, Mishkin (1966), succeeded in separating striate and inferotemporal cortex, and demonstrated that the deficits in visual learning and memory result when inferotemporal cortex is cut off from earlier visual processing. He also demonstrated that TE and TEO, areas within inferotemporal cortex that von Bonin and Bailey (1951) had distinguished on cytoarchitectonic grounds, produced differential deficits, with TEO lesions producing greater deficits in single-pattern discrimination tasks and TE lesions generating greater deficits on learning to perform multiple discriminations in parallel.

Again, the lesion studies indicating separate processing areas were complemented by single cell recording studies that sought to determine what stimuli generated specific responses in inferotemporal cortex. Charles Gross, together with Carlos Eduardo Rocha-Miranda and David Bender (1972), found cells in the inferotemporal cortex of the macaque which responded most vigorously to shapes such as hands. (Like Hubel and Wiesel's, their discovery resulted from serendipity: after failing to find a light source that would drive a particular cell, they waved a hand in front of the stimulus screen and produced a vigorous response). Although nearly a decade passed before further research was published confirming different areas where individual cells were responsive to specific shapes,[7] in the 1990s there was an explosion of reports of specific areas in inferotemporal cortex responsive to different specific shapes (see (Tanaka, 1996), for a review).

A similar pattern of first lesion studies, then single-cell recording studies, emerged in research on the parietal cortex.[8] Ettlinger and Kahlsbeck (1962) analyzed deficits in monkeys with lesions in posterior parietal cortex and revealed deficits in visual orientation and reaching, indicating that these areas are involved in analysis of the location of objects in the visual field. In the early 1970s Hyvärinen and Poranan began recording from neurons in posterior parietal cortex, where they found cells which they interpreted as involved in visuospatial guidance of movement.

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[7]Gross (1998, pp. 199-200) reports on the slowness of response: "for more than a decade there were no published attempts to confirm or deny these and our other early basic results, such as that IT cells have large bilateral fields that include the fovea and are not visuotopically organized. And unlike Panizza, the discoverer of visual cortex in the nineteenth century, we did not publish in obscure journals or from an unknown institution. Perhaps because of the general skepticism, we did not ourselves publish a full account of a face-selective neuron until 1981."

[8]Both Ferrier and Yeo (1884) and their opponents Brown and Schäfer (1888) reported deficits from lesions to the angular gyrus in the posterior parietal cortex which fit the pattern of deficit in spatial localization identified in the 1960s. Ferrier and Yeo report that the lesioned monkey was "evidently able to see its food, but constantly missed laying hold of it" and Brown and Schäfer report that their monkey "would evidently see and run up to [a raisin], but then often fail to find it . . ." (both quotations from Gross, 1998, p.. 200 and 201). Based on studies of brain injuries in World War I veterans, Gordon Holmes (1918) identified deficits in spatial localization of objects that the veterans could easily identify visually.

> . . . when a sensory stimulus which interested the animal was placed in a specific location in space where it became the target of the monkey's gaze or manual reaching, tracking or manipulation. . . . Some cells were clearly related to eye movements whereas others appeared to discharge in response to visual sensory stimuli (Hyvarinen & Poranen, 1974); quoted in Gross, 1998, p. 203).

The link Hyvärinen and Poranan found between activity of parietal cells and eye movement suggested a motor function for parietal cortex cells, a suggestion that was further developed by Vernon Mountcastle and his colleagues, who identified parietal cells linked not just to eye movement and visual tracking of objects, but to arm and hand manipulation (Mountcastle, Lynch, Georgopoulos, Sakata, & Acuna, 1975). Mountcastle interpreted these cells as involving motor commands linked to selective attention. Other research, however, suggested that the posterior parietal cortex was primarily involved in visual analysis since some cells are responsive in the absence of any motor activity (Goldberg & Robinson, 1980). But importantly, Richard Andersen and his colleagues demonstrated that cells in posterior parietal cortex mapped stimuli in terms of spatial location, a feature to which temporal lobe cells are relatively unresponsive (Andersen, Essick, & Siegel, 1985).[9]

Determining how the components are organized

Research such as that related in the preceding sections revealed a large number of components in the visual processing system and linked these with different kinds of visual processing. These are critical elements in developing an understanding of a mechanism, but in a mechanism the components are organized so that their activities are coordinated with each other. The organization of a neural system is provided by the patterns of cellular connectivity through which neurons in different brain areas communicate with each other. However, these patterns of connectivity in the brain are extremely complex. On average each neuron is connected to a thousand other neurons. To understand how the neural system is organized, a somewhat coarser view is required. To a degree this was already emerging from the patterns of discovery. Starting with primary visual cortex, researches expanded their conception of the visual processing mechanism by moving anteriorly in the brain. Moreover, it became clear that the connections from extrastriate cortex seemed to split into those projecting downwards into the temporal lobe and upwards into the parietal lobe. And as we have seen, the projections into the temporal lobe seemed to involve identification of objects while the projections into parietal lobe seemed to involve spatial information. Focusing on subcortical processing in which lesions seemed to differentially affect location versus object recognition, Schneider (1967; 1969) and Trevarthen (1968) proposed a distinction between *what* and *where* processing. Leslie Ungerleider and Mishkin (1982 see also Mishkin, Ungerleider, & Macko, 1983) adopted this framework to provide a overall macro-level organizing principle for visual processing. According to their model, processing pathways from extrastriate cortex down into temporal lobes are involved in

[9]Subsequent research, which will be discussed in lecture 4, has confirmed a close relation between parietal cells and motor action and has investigated whether these cells are directly involved in planning action or in maintaining attention on visual stimuli (Batista, Buneo, Snyder, & Andersen, 1999; Snyder, Batista, & Andersen, 1997).

identification of objects while pathways from extrastriate areas up into parietal cortex figure in determination of location of objects and events in the world.

For Ungerleider and Mishkin, the separation of two pathways began in prestriate cortex. Other researchers soon proposed extending the scheme back into V1, LGN, and the retina, generating a model of two processing streams from the very earliest visual input. An important piece of evidence for projecting the two streams further back was a distinction between two different cell types in the retina and the LGN. Enroth-Cugell and Robson (1966) had differentiated two types of cells in the cat retina, which they named *X* and *Y* cells. X cells had small receptive fields (hence, they were sensitive to high spatial frequencies), medium conductance velocities, and responded as long as the stimulus was present. In contrast, Y cells had large receptive fields, rapid conductance velocities, and responded transiently. A similar distinction of retinal cell-types was advanced for primates. Pα (or P ganglion) cells correspond to the X cells in the cat while the Pβ (or M ganglion) cells correspond to the Y cells in cat. Research on old world monkeys revealed that this scheme is maintained in the LGN where the cells in the two inner layers have large cell bodies (the layers are thus known as *magnocellular* or *M layers*) while the cells in the outer four have small cell bodies (thus called *parvocellular* or *P layers*). The M layers of the LGN receive projections from the M ganglion cells, while the P layers receive input from the P ganglion cells (Dreher, Fukada, & Rodieck, 1976).

The discovery of two pathways before and after V1 raised a question of whether they were related. The early studies of Hubel and Wiesel and others had suggested that V1 had a homogenous cytoarchitecture; if this were the case, the two precortical pathways would converge in V1 and then two other pathways would diverge beyond V1. But, in accord with the caveat in the passage quoted above from Hubel and Wiesel, a new technique, involving the application of cytochrome oxidase stains (developed by Margaret Wong-Riley, (1979), revealed additional complexity in V1. Cytochrome oxidase is an enzyme critical to the oxidative metabolism of the cell; staining for it reveals areas of high metabolic activity. In layers 2 and 3 and 5 and 6 of V1 these showed up as 'blobs'[10] which indicated regions of particularly high metabolic activity. Recording separately from cells in the blob regions and in the interblob regions, Livingstone and Hubel (1984) found orientation selective cells only in the interblob regions, and wavelength sensitive cells in the blobs, indicating a separation of processing within V1. On the basis of this differentiation, Livingstone and Hubel proposed extending Ungerleider and Mishkin's two pathways to account for all visual processing from the retina on.

The integrating scheme of two processing streams receives support from the neuroanatomy. The M layers of the LGN project to layer 4B in V1, where there are no blobs, whereas the P layers of the LGN project, via layers 4A and 4Cb, to layers 2 and 3 of V1, where there are both blob and interblob regions. Cytochrome oxidase stain also revealed a differentiation in V2 of alternating thick and thin stripes with interstripe areas between them. The differentiation in V1 is

[10]Livingstone and Hubel introduced the term *blobs* to characterize their appearance, citing the *Oxford English Dictionary* for the term. These blobs are "oval, measure roughly 150 x 200 μm, and in the macaque monkey lie centered along ocular dominance columns, to which their long axes are parallel" (p. 310).

maintained, with the thick strip regions receiving their input from layer 4B, the thin strip regions from the blobs of layers 2 and 3, and the interstripe regions from the interblob regions in V1. From the differentiated areas in V1 and V2, processing largely separates into the *what* and *where* pathways originally distinguished by Mishkin and Ungerleider.

Like most integrating schemes, this one is subject to a variety of qualifications. van Essen and Gallant draw attention to the fact that the two streams are not entirely independent—there are neural connections between areas such as MT and V4, which appear in different streams, and processing in later parts of one stream continues even when its primary input is removed. Moreover, there is considerable interaction between the two pre-cortical streams so that processing in both cortical streams can continue even if the supposedly specific precortical input is removed. Furthermore, the characterization of the two streams as processing *what* and *where* information has been questioned. Milner and Goodale (1995) argue that the dorsal stream receives information about the identity of objects (revealed in the ability of individuals with temporal lobe lesions to grasp objects appropriately for their use) and propose that what is distinctive about it is that it is primarily concerned with coordinating information about visual stimuli for action. In their view, the ventral stream is principally involved in extracting information about visual stimuli required for higher cognitive processing. Even with such qualifications, though, the idea of two visual streams plays an important integratory role in theorizing about visual processes, providing for a relatively coherent and graspable account of how the brain processes visual information.

Conclusion

We have now seen how the mechanism for visual processing in the primate brain was discovered through identification of a host of brain areas that carry out different parts of the task of analyzing visual inputs. The discovery involved both structural and functional decomposition of the brain and the offering of proposals as to how the system is organized. What has been achieved so far, though, is clearly not a complete account of visual processing. To begin with, operations have not yet been assigned many of the areas differentiate by van Essen and Gallant in Figure 1. The manner in which they are connected to other areas and responsiveness of cells in these areas to visual stimuli indicate that they do perform operations in the visual processing mechanism which still must be discovered. Moreover, Figure 2 does not show how to build a system that performs visual processing. The account must be filled in with details of how cells in each area utilize their inputs, perform an operation, and provide outputs to other areas.

Although the account of visual processing developed so far is both incomplete and subject to revision in the face of new research, it constitutes a relatively well-worked out sketch of the mechanism that is well supported. From studying Figure 2 one develops an understanding of how processes in the brain make it possible for us to see the world. As such, it offers what Thomas Kuhn refers to as an exemplar—an example of successful research which provides a model to be emulated by other domains of cognitive neuroscience.

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