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Sensory and Non-Sensory Visual Disorders in Man and Monkey

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Sensory and non-sensory visual disorders in man and monkey

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The posterior third of the cerebral cortex in monkeys consists of a patchwork of visual areas in each of which there is a 'map' of the retina. The details of the 'map' vary considerably from one area to another and one notable variation concerns the optimal visual feature to which the cells respond. Orientation, disparity, colour and movement are emphasized in separate areas that appear to be concerned with sensory analysis. Their existence and the possibility that brain damage is occasionally restricted chiefly to one such area may explain the rare highly selective visual sensory impairments that can follow posterior cerebral damage in man. Other areas are notable for having little or no retinotopic representation. Here the cells may have huge receptive fields and complex trigger features. When such regions are removed, the animal's visual sensory abilities are intact but its recognition of patterns and objects is not. This condition resembles human visual agnosia.

Introduction

The controversy over the nature of the effects on visual perception of localized brain damage in man has simmered throughout this century. It is not the unimportant squabble that some believe it to be, for the consequences of damage can shed a powerful light on how the brain works in general terms, and as there has never been a shortage of ideas about the latter, the results of brain damage should actually help us to test hypotheses about brain function. One area of disagreement concerns the existence of highly specific disorders of some narrow aspect of visual processing, e.g. colour or movement or position. If dissociations of this kind do exist they indicate that some degree of parallel processing occurs. A second bone of contention concerns the status of what are sometimes called, for want of a better description, higher-order defects, like object agnosia or facial agnosia. Do they simply reflect a constellation of rather simple component defects, or is there something about the recognition of complex objects that can be seriously impaired while the discrimination of all the components of an object remains unscathed? Recent investigations of clinical patients have tipped the balance towards accepting the existence of both specific sensory impairments, and object agnosia that cannot be accounted for by a general elevation of sensory thresholds. Over the same period of time investigations on the visual cortex of monkeys have provided ample evidence for a type of brain organization that, if present in our brain, makes good sense of the puzzling effects of brain damage.

Specific sensory disorders

The clearest examples of a highly specific visual sensory loss concern colour, movement, position and depth. Cerebral achromatopsia is a condition associated with bilateral damage to the ventral aspect of the prestriate cortex (Meadows 1974). The patient performs poorly on tests of colour vision, e.g. naming, sorting and matching colours, and his world looks grey or

drained of vivid colour. Yet the basic trichromatic mechanism of the retino-cortical projection is intact as judged by increment threshold measurements for coloured targets on coloured backgrounds (Mollon *et al.* 1980). So information about wavelength may be coded without colour being seen, as if one outcome of a mechanism to discriminate among wavelengths has been detached from the otherwise intact processor. Specific disorders of the perception of movement are even rarer and their independence has often been challenged (Teuber 1960; Teuber *et al.* 1960) but the recent investigations by Zihl (1981, and personal communication) demonstrates the syndrome in almost pure form. After bilateral damage in the territory of the posterior cerebral artery the patient had gross impairment of movement detection, especially in the peripheral visual field, and little appreciation of its direction when the movement was detected. Perception of motion in depth was abolished. Continuously moving stimuli, like tea pouring from the pot, appeared to be frozen, like a glacier. Yet there was no field defect, and acuity, stereopsis, colour perception and the detection of stationary stimuli were normal.

Correctly registering the position of an object seems to be such a fundamental feature of vision and of the retinotopic arrangement of the visual pathways that it may seem unlikely that it could be impaired in relative isolation. Nevertheless, damage that includes area 7 of the parietal lobe may lead to the mislocation of very simple and readily detected visual targets (Holmes 1918; Cole *et al.* 1962; Ratcliff & Davies-Jones 1972). The effect is usually measured by asking the patient to point to the target, but as it is limited to the contralateral half-field after unilateral damage and is demonstrable with either hand it follows that it cannot be explained as a purely motor disorder. Furthermore it can be revealed simply by asking the patient to describe verbally the positions of targets in relation to his own body. Finally, disorders of stereoscopic depth perception that cannot be explained by common concomitant symptoms, such as field defects, have frequently been described. They are probably commoner than we suspect because the patient usually has no subjective or disabling disturbances of depth perception, perhaps because monocular depth cues suffice (Danta *et al.* 1978).

The importance to the present argument of the disorders just described is their dissociation from each other. Unfortunately the information about the precise localization of the brain damage responsible for the defects is scanty, which is why investigation of the visual cortex of monkeys has proved so valuable. Only 20 years ago there was still no demonstration of any physiologically defined visual area beyond the striate cortex, area 17, in primates. Now there are ten or more separate regions in which the visual field, or parts of it, are mapped or in which there is some other non-retinotopic form of visual representation. Figure 1 shows the position of these secondary visual areas in the owl monkey (see Allman (1977) for review) and the rhesus monkey (see Zeki (1978) for review). The posterior third of the cerebral cortex in these primates resembles a patchwork quilt in which the seams tend to represent the vertical or horizontal meridian of the retina. The first investigations of these secondary areas concentrated on defining their borders, their retinal topicity and their interconnections. More recently, attention has turned to what feature other than position most influences cells in different areas. Do the cells of these different areas have similar or even indistinguishable receptive field properties, which if true suggests that the key to their function must lie in their different outputs, or are they analysing and coding different aspects of the visual scene? What evidence we have suggests that although the differences are not always sharp it is possible to say that the emphasis varies among areas. For example, the retinal disparities that are coded in V1 are much smaller than those encountered in V2 for the same region of the retina (Poggio & Fischer 1977;

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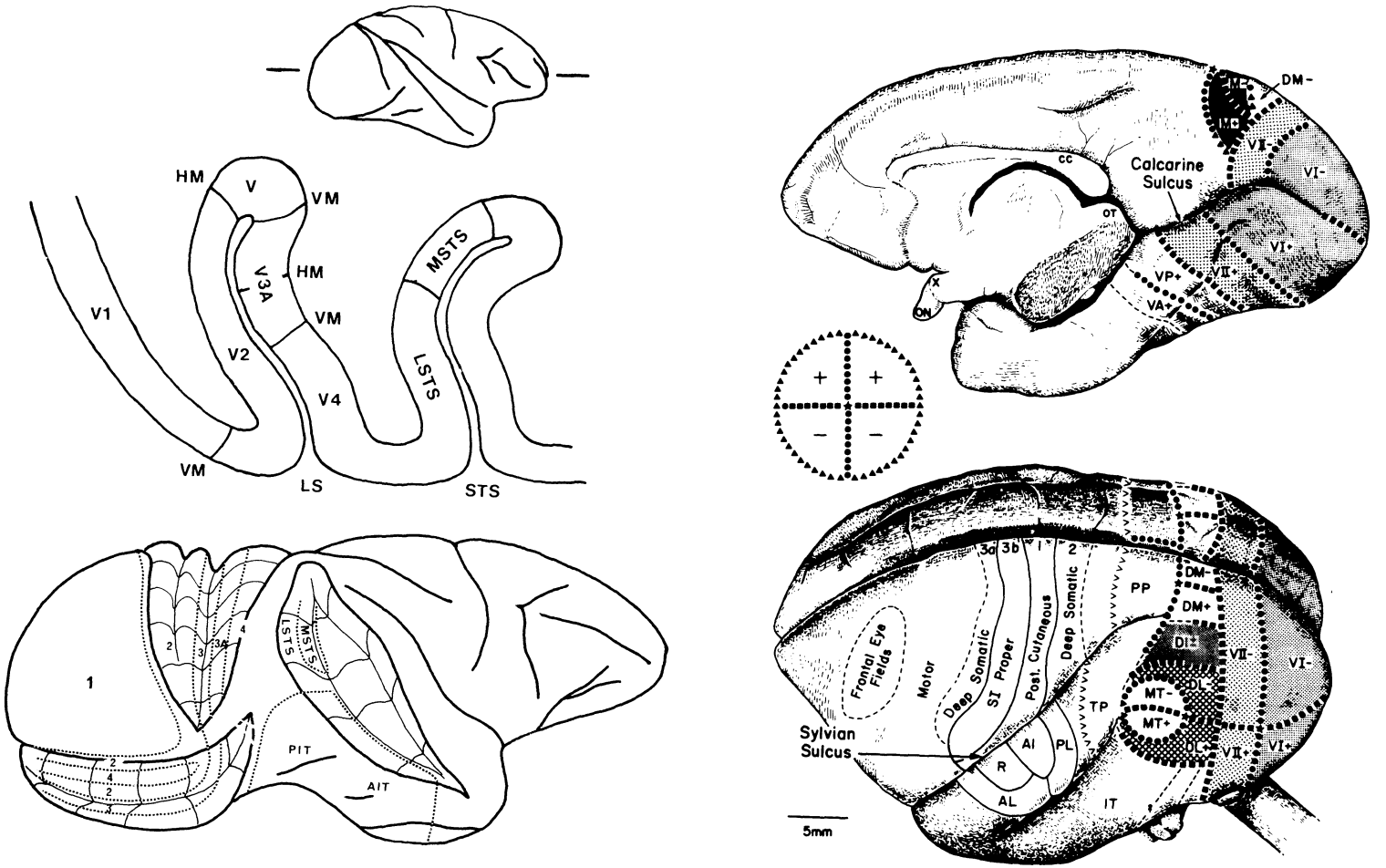

FIGURE 1. Position of various visual areas in the posterior cerebral cortex of the rhesus monkey (left) and owl monkey (right). In the rhesus monkey a horizontal section through the occipito-temporal region of the right hemisphere is shown, and below it is a schematic diagram of the gross positions of these areas in a brain where the lunate, inferior occipital and superior temporal sulci have been opened. In the owl monkey the medial view of the brain is shown above, with a dorso-lateral view below. The symbols for the visual field chart between them are also superimposed on the brain. The relevant abbreviations are discussed in the text. The drawing of the rhesus monkey brain is taken from Cowey (1979) and that of the owl monkey from Baker *et al.* (1981).

Fischer & Poggio 1979) and the proportion of cells coding disparity seems to be higher in V2. By contrast V4 and the lateral bank of the superior temporal sulcus (STS) are preoccupied with colour (Zeki 1977; 1980), and those in medial STS are primarily concerned with direction and velocity of movement (Zeki 1974). All these findings were made in rhesus monkeys, but a similar picture is emerging in the owl monkey, where cells in area MT (see figure 1) are much more fussy about direction of movement than those in areas DL, DM and M; cells in DM show the greatest orientation specificity to moving bars; and moving arrays of random dots are very effective for cells in MT while barely being detected by cells in DM and DL (Baker *et al.* 1981).

If this scheme of regional specialization is present in our brain, the specific defects in the perception of colour, movement, position and depth make sense and their rareness is not surprising. Strokes, tumours, blood clots and traumatic wounds do not respect functional boundaries, which is why the detailed investigation of a single patient with rare symptoms can tell us much more than group studies of patients with multiple defects. However, things that make sense are not always correct and the fact that there are many and varied visual areas in monkeys does not end the search to explain disordered perception of space, colour or movement in man. Can these disorders be reproduced in even purer forms in monkeys, where in theory the damage can be neatly confined to a particular visual area? Paradoxically our ignorance is greatest here, perhaps because the functional, as opposed to the cytoarchitectonic, maps are so recent. But there are several clear pointers. Wilkinson and I (in preparation) found that when the part of V2 concerned with central vision was removed there was an average fivefold increase in stereoacuity thresholds. There was no impairment in controls in which the splenium of the corpus callosum was severed, and there was a much smaller impairment in animals in which an even larger area of cortex was removed more anteriorly and from a different visual area. A different impairment, much more reminiscent of the visual disorientation reported in patients with parietal lobe injury, follows ablation of area 7 of the parietal lobes in monkeys. The animals have difficulty in responding to a particular location on the basis of its proximity to another visual cue, or 'landmark' (see Ungerleider & Mishkin (1982) for review). Yet they are as proficient as normal monkeys at discriminating among complex visual stimuli as long as the spatial relations among them are irrelebant to the solution of the problem. Finally Fries & Zeki (1980, and personal communication) have found that the selective ablation of V4, where colour cells predominate, elevates hue discrimination thresholds at short wavelengths without affecting orientation thresholds.

These parallels between perceptual performance in monkeys and human patients are important, but require much more extensive investigation before we can accept that the same syndrome, or its anatomical basis, is being described in both species. For example, if cortical achromatopsia in patients is caused by the destruction of a region comparable with V4 in rhesus monkeys, we might expect its removal in monkeys to do much more than elevate wavelength discrimination thresholds in the blue region of the spectrum. Of course, patients with achromatopsia may respond just like monkeys when their wavelength discrimination is tested in the same way, but the totally colour-blind patient studied by Mollon *et al.* (1980) could hardly do so. Furthermore, rhesus monkeys in which V4 proper (but not its extention into lateral STS) was removed were not impaired at discriminating between Munsell colour standards in the red–green range (Dean 1979) although patients with cortical achromatopsia are impaired with similar stimuli. An entirely different possibility is that V4 is concerned with colour

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constancy rather than wavelength discrimination, and that achromatopsia follows the destruction of a different region. Certainly the responses of many of the colour-coded cells in V4 display colour constancy, i.e. although a cell responds only to a particular narrow band of wavelengths when monochromatic lights are presented in the cell's classical receptive field, it responds to a particular *colour* (as judged by human observers) when complex chromatic displays illuminated by a variety of wavelengths of light are used. In this case the perceived colour is determined by the energy–wavelength relations among clusters of adjacent objects. Zeki (1980, 1981) reports that for many cells in V4 there is a close correspondence between the response of the cell and the colour seen by human observers, but that when only one uniform part of the complex display is presented the response of the cell, like that of the human observer, is again solely determined by wavelength. Although the effects on colour constancy of removing V4 in monkeys have yet to be studied, it is clear that cortical achromatopsia is more than defective colour constancy, which alone should not cause the disappearance of the subjective experience of colour.

The possible limitations of particular tests may also be seen in an experiment by Collin & Cowey (1980). They removed the movement area of the superior temporal sulcus in monkeys and found no change in the threshold for detecting the smallest movement of a luminous spot. This result does not demonstrate that the so-called movement area has nothing to do with seeing movement. Direction of movement, laterally or in depth, and an awareness of its velocity are important features that have not yet been examined.

I have discussed in detail elsewhere possible reasons for the plethora of visual areas in each of which a relatively simple attribute of the visual image is analysed by the cells (Cowey 1979, 1981). The chief argument stems from anatomical and physiological observations that the vast majority of neurons in the cortex are local circuit interneurons and that they are involved, often by inhibitory mechanisms, in fashioning the fine tuning properties of single neurons to such things as orientation, disparity, direction, velocity, wavelength and size. Attempting to do all these things in one area, where each neuron has to be interconnected to others involved in analysing the same attribute, creates great demands on specifying the correct local connections in development. Regional specialization on the other hand, means that columns of cells can simply be connected with all or most of their immediate neighbours who are all involved in the same job. The average length of the interneurons can also be kept shorter. The highly specific visual disorders that can follow localized posterior cerebral damage may therefore reflect a parcellation of the visual sensory areas that achieves maximum efficiency in the process of sensory analysis. It is gratifying that such a conclusion was reached on entirely different grounds by Marr (1976) in considering the computational problems involved in perception: 'Any large computation should be split up and implemented as a collection of small sub-parts that are as nearly independent of one another as the overall task allows. If a process is not designed in this way, a small change in one place will have consequences in many other places. This means that the process as a whole becomes extremely difficult to debug or to improve, whether by a human designer or in the course of natural evolution, because a small change to improve one part has to be accompanied by many simultaneous compensating changes elsewhere.'

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Non-sensory defects: visual agnosia

Once the relevant features of the visual scene have been selected and analysed by a visual cortex that is modular, there is no necessity for the information in different areas to converge on groups of cells for the information to be understood. Such convergence on to higher-order cells provides no information that is not already available in a widespread network of cells. Nevertheless, electrophysiological recordings in regions of the temporal outside the areas already discussed have revealed cells that respond best to complicated but narrow classes of stimuli such as faces, paws or other objects. What is their purpose and what would be the consequences of damaging them?

The analysis of the visual scene leads to a response, such as recognition, action, ideas or emotion. The response is often learned, varies according to the context of a stimulus and should be capable of repeated and rapid modification as the significance of a stimulus changes. In other words the same stimulus must be capable of producing a different pattern of neural activity according to its learned significance. Whatever the cellular events underlying learning, it may be much simpler to make them in the connections of a relatively small number of higher-order neurons with convergent input than in cells throughout all the visual areas. The consequences of damaging areas in which all the cells have these more complicated properties, derived from convergent input from visual sensory analysing areas, should be the intact analysis of components but a faulty recognition of their collective meaning, i.e. agnosia without sensory loss. Two possible clinical examples will be discussed, together with relevant work on monkeys.

Prosopagnosia is a defect in recognizing faces (for review see Damasio *et al.* 1982). In all cases in which there was a post-mortem examination the damage was bilateral in the region of the fusiform, lingual and para-hippocampal gyri, i.e. ventro-medial occipito-temporal cortex. The patient is unable to recognize faces by sight, even those of close relatives or his own in a mirror. It is commonly associated with achromatopsia and field defects, but even in conjunction these can hardly be responsible, for we can all recognize black and white photographs of faces with a small part of the visual field. Naming and describing parts of the face, e.g. mouth, eyes, ears, are normal. The recognition of objects is not always impaired, although in recognizing a particular face one may be making a different and more difficult kind of judgement than identifying an object, i.e. one is specifying the individual rather than the species. Nevertheless, recent electrophysiological recordings show that in rhesus monkeys there lies deep in the fundus of the temporal lobe a region where many of the neurons respond selectively to faces (Perrett *et al.* 1979, 1982; Rolls 1981) and in a manner that shows they are not simply reflecting the arousing or emotional effects of faces or the overt motor responses they provoke. Some of these cells respond to facial components such as eyes or mouth, but they are remarkably uninfluenced by changes in the orientation or size of a frontal face, although a rotation to profile reduces the responsiveness of some cells. The full significance of these findings needs further experiment, for example prosopagnosic patients have no trouble in knowing that a face is a face yet any one cell in the face area of the temporal lobe of the monkey seems to be signalling that any face rather than a particular face is present. One might therefore expect their removal to make it difficult to identify faces as a class. This problem may be resolvable on the reasonable assumption that small differences in the response of individual cells to different faces yields an unambiguous message about the identity of that face in a cooperative network of such cells. Certainly the existence of such an area in our brain would remove much of the incredulity that greets reports of facial agnosia in which elementary visual sensations are largely intact.

The second example of a recognition defect concerns object agnosia, which it must be said at once is not confined to three-dimensional objects. It is a disorder of recognition that is not secondary to language impairment, general intellectual deterioration or loss of elementary sensory function. As used here the term excludes patients who can mime the function of an object, thereby demonstrating that they recognize it despite being unable to name it, i.e. patients with nominal dysphasia or optic asphasia. The remaining patients, who have 'pure' visual agnosia, are rare but even so can be divided into two groups (see Rubens (1979) for review). The first have apperceptive visual agnosia. Along with their recognition defect they have great difficulty in copying a drawing, or matching a drawing or pattern to its twin in an array. The disorder may be so severe that the patient cannot discriminate between simple geometric figures. In fact there is a good deal to be said for describing the disorder as a defect in pattern discrimination. Associative visual agnosia differs from apperceptive in that the patient can copy and match drawings. Pattern discrimination seems to be intact, but is divorced from the meaning it normally provokes. As the various hypotheses put forward to explain visual agnosia are dealt with by Warrington in the next paper I shall concentrate here on experiments with monkeys that may reproduce the apperceptive–associative distinction and throw light on its anatomical and physiological bases.

It has been known for nearly 30 years that ablation of inferotemporal cortex, a region that includes most of the cortex labelled PIT and AIT in figure 1, severely impairs visual discrimination learning for patterns, objects and colours presented in a large variety of tasks (see Dean (1976) for review). Later investigations compared the effects of posterior or anterior ablation (PIT or AIT of figure 1) and found differences that were not simply quantitative (Iwai & Mishkin 1968; Cowey & Gross 1970; Gross *et al.* 1971). Discrimination learning and retention of geometric patterns was much more severely impaired in the PIT group, yet this group was indistinguishable from normal controls in discriminating between plain coloured cards that were just as difficult as the patterns for the controls. The impairment was therefore specific for shape rather than for any difficult visual task. It was also exacerbated in the PIT but not the AIT group when additional geometric features were added to patterns that the animals had previously succeeded in learning to discriminate. When simple visual objects were used instead of patterns and each of the several pairs to be discriminated was presented randomly in the same testing session in a concurrent learning paradigm, the AIT group was now more severely impaired and their disability was described as 'associative', as opposed to 'perceptive'. The parallel with apperceptive and associative visual agnosia will be obvious but, like others that have been drawn in this paper, needs much more detailed examination and testing. Of one thing we can be certain: like visual agnosia the defects following PIT or AIT ablation do not stem from simple sensory disorders. After large inferotemporal lesions involving both posterior and anterior regions, critical flicker fusion is normal (Symmes 1965), the visual fields are intact (Cowey & Weiskrantz 1967), there is no reduction of grating acuity (Weiskrantz & Cowey 1963), incremental brightness thresholds are unaltered (Ettlinger 1959), and the detection of brief flashes of light is undisturbed (Bender 1973). There are no published studies in which the effects on sensory thresholds of complete posterior or anterior inferotemporal lesions are measured and compared, but we have recently shown (A. Cowey, P. Dean & L. Weiskrantz, unpublished) that contrast sensitivity and contrast matching are not affected by either lesion. Like patients with visual agnosia, monkeys with inferotemporal lesions appear to possess the normal means of detecting all the components of the visual world. Furthermore, the brain damage is comparable in both patients and monkeys. Both Pötzl (1928)

and Nielson (1937), who reviewed neuropathological investigations of patients, conclude that the common locus is the latero-ventral occipito-temporal cortex, a description that is as appropriate for monkeys as for people.

It is more difficult to find informative anatomical and physiological information that illuminates the contrasting roles of the posterior and anterior regions of the temporal lobe. The region called PIT certainly receives projections from secondary visual areas that include V2, V3, V4 and the superior temporal sulcus (Kuypers *et al.* 1965) but the receptive field properties of its cells have been extensively examined only with respect to their size (Gross *et al.* 1972), which falls between those of the occipital lobe and the AIT region. However, much more is known about the receptive field properties of cells in middle and anterior inferotemporal cortex (Gross *et al.* 1972; Rolls 1981; Bruce *et al.* 1981; Gross & Mishkin 1977; Gross *et al.* 1977; Sato 1981). Of greatest interest for the present discussion are the great size and bilaterality of the receptive fields together with the relative invariance of the response when the optimal stimulus, which may be much more complex than a bar or grating, is changed in size, orientation or colour. This indifference to size, orientation and position of a pattern smacks of visual constancy and suggests that one explanation of associative visual agnosia is that the means of recognizing an object irrespective of the exact shape and size and position of its retinal image has been destroyed. Without it all exemplars of an object may appear unique and therefore different. Such a view is of course not very dissimilar, except in terminology and the evidence by which it was reached, from that of Ratcliff & Newcombe (1982), in which visual agnosia is described as a defect in the construction or storage of the object-centred 3D model representation that forms such a central role in Marr's computational theory of pattern recognition (Marr & Nishihara 1978*a*, *b*). Is there any other evidence to support it? I should like to consider two. The first is an experiment by Humphrey & Weiskrantz (1969) where it was found that monkeys with inferotemporal lesions were no longer able to discriminate between discs of different sizes irrespective of their distance. The pattern of errors suggested that they were unable to combine information about retinal image size and about distance, although they perceived each component. Their conclusion that size constancy had been disrupted has been criticized, correctly, on the grounds that the result can equally well be explained by slow relearning of a difficult task (Dean 1976). But there is no strong evidence that the original conclusion is wrong, and it should certainly be tested in other ways. A similar impairment on a size-constancy task after inferotemporal lesions in monkeys was reported by Ungerleider *et al.* (1977), but it was still unclear whether size constancy *per se* was abnormal.

The second experiment (Gross 1978) concerns the discrimination of mirror-image or rotated patterns. Gross (1978) and Gross & Mishkin (1977) point out that if inferotemporal cortex is necessary for recognizing the equivalence of patterns that differ only in orientation, its removal will, as already suggested, disrupt pattern and object recognition but might actually abolish the well known confusion between left–right mirror-image patterns, which will now be perceived as unique and therefore no longer confused. Gross found that although inferotemporal lesions impaired discrimination learning or retention when using different patterns, the animals were not significantly worse than controls when tested with patterns differing only in orientation by 180° or 90°. Furthermore, this result was not a trivial consequence of rotated patterns being easier for all animals to discriminate. If anything, the rotated identical patterns were more difficult than the dissimilar patterns for the normal animals. Why then were the animals with inferotemporal lesions not *better* than the controls when rotated patterns were used?

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The answer may lie in the other aspect of constancy, i.e. position, and its possible relation to the huge receptive fields of cells in inferotemporal cortex. Although freed from the confusion caused by regarding rotated identical patterns as the same thing, the animals will, on the hypothesis advanced, now regard an image as novel when it falls on disparate parts of the retina and no longer excites the same cells. This will impede discrimination learning.

What is being suggested in this analysis of the effects of inferotemporal ablation in monkeys is that perceptual categorization is impaired, as has also been suggested to explain some examples of impaired object recognition in patients (Warrington & Taylor 1978). In the absence of the mechanism to extract the class or category of an object, the resemblance between different examples of the same object is not appreciated. Everything looks unique, including the same stimuli from trial to trial in learning tasks as an animal looks at them from different distances and angles. It would be nice to end on this confident note were it not that Dean (1976), while arguing that only a categorization hypothesis can explain all the effects of inferior temporal damage in monkeys, proposes that the faulty categories are too *broad* rather than too narrow. His suggestion is certainly supported by the demonstration that generalization gradients to orientation and wavelength are much shallower in monkeys with inferotemporal lesions (Butter *et al.* 1965), i.e. the animals respond to new orientations and colours as if they closely resembled those to which they had been trained to respond. Furthermore, when orientation thresholds were measured by using a successive presentation method that requires the animal to indicate whether a square-wave grating was oriented at 45° or not, monkeys with inferotemporal lesions had significantly and permanently elevated thresholds, suggesting that their categorization of orientation was abnormally imprecise (Dean 1978). But as their categorization of wavelength was not imprecise (Dean 1979) it is difficult to explain why such animals find it difficult to learn both orientation and colour discriminations. The evidence that visual perceptual categorization is impaired in patients with visual object agnosia and in monkeys with damage to the inferior temporal lobe is strong, but there is no good evidence to decide whether the categories are too broad and few or too narrow and many. As Dean (1976) points out, 'It is an interesting reflection on the state of the field that for lack of evidence, hypotheses apparently so opposed can co-exist within it.'

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