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CEREBRAL AKINETOPSIA (VISUAL MOTION BLINDNESS)

A REVIEW

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SUMMARY

Cerebral akinetopsia is a syndrome in which a patient loses specifically the ability to perceive visual motion following cortical lesions outside the striate cortex. There has been only one good case of akinetopsia in the published literature. Yet that case was immediately accepted by the neurological world. In this, cerebral akinetopsia differs markedly from cerebral achromatopsia, the evidence for which was strongly contested for the better part of a century (Zeki, 1990). This article complements the one on cerebral achromatopsia, traces the history of akinetopsia and enquires into why it was so much more readily acceptable than achromatopsia.

INTRODUCTION

In 1983, the world of neurology witnessed two surprises. The first was the publication of a paper by Zihl *et al.* describing a patient who became defective in her ability to see objects in motion following a bilateral cerebral vascular lesion in cortex outside the striate area. Apart from a mild anomic aphasia, the defect was specific for visual motion perception and was not accompanied by a scotoma. It was the first clinical description of cerebral motion blindness, a syndrome which will be referred to as *akinetopsia* in this review, to bring it into line terminologically with the much more common achromatopsia, or cerebral colour blindness. The second surprise was that, although a single case study, it was immediately accepted by the neurological and, more generally, by the neurobiological world, without a murmur of dissent. This was in marked contrast to the more turbulent history of achromatopsia, the evidence for which was strongly challenged. Here there was no MacKay (1888) to complain that 'the cases are very few in number', no Henschen (1910) to claim that his 'cortical retina' (the striate cortex) is also a retina for '(movement) impressions', no Critchley (1965) to write of '. . . a mere handful of instances of alleged (motion) agnosia, most of which are unconvincing'. Compared with the many, and conceptually seemingly powerful, objections raised against the notion that cerebral achromatopsia results from lesions in a visual centre outside the striate cortex (*see* Zeki, 1990, for a review), this acquiescence is surprising; it makes one want to learn more about the early literature regarding disturbances of visual motion and contrast it with the literature on achromatopsia.

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Fortunately, that literature is very brief compared with the literature on achromatopsia. It has made no impression at all in the world of neurology.

EARLY HISTORY

Akinetopsia can be defined as a defect in the perception of visual motion. Like achromatopsia, it may occur in conjunction with other defects or manifest itself as a remarkably specific syndrome, as in the patient of Zihl *et al.* (1983). One of the earliest clinical papers describing akinetopsia was that of Pötzl and Redlich (1911). Following bilateral injury to the occipital lobes, their patient could neither detect moving objects nor fixate them when they approached her quickly. The akinetopsia was linked to a 'defect of peripheral vision with retention of light perception and colour vision (and with) evidence of mental blindness and disturbance of spatial orientation'. In addition, the patient suffered from disturbances of orientation and of recognition as well as the inability to localize acoustic stimuli. There was no attempt to localize the lesion, or discuss its extent. The paper is not an important one in the clinical literature and the akinetopsia described there, as well as in the later paper of Goldstein and Gelb (1918), can be attributed to a more general disturbance following lesions which were not verified by postmortem examination and the extent of which therefore remains unknown. This indeed was the interpretation given by Jung (1949) who re-examined the patient of Goldstein and Gelb in 1942 and found that the patient was not deficient in the perception of shapes and movements. In the absence of postmortem material, Jung concluded that it was more likely that the patient had suffered from a chiasmal defect rather than a parieto-occipital lesion, as Goldstein and Gelb had believed. Jung stressed 'The importance of elaborated investigations in single cases of brain injury', a cautionary remark not dissimilar to that delivered much earlier by MacKay (1888) with respect to achromatopsia. MacKay had urged ' . . . the desirability of investigating cases of hemianopsia with more thoroughness and precision than is usually shown', an exhortation which did nothing to prevent him from accepting the syndrome of achromatopsia 11 years later, on the basis of a single patient whose 'light sense' could not be studied 'for want of proper means in the patient's house'! (MacKay and Dunlop, 1899).

Just as achromatopsia has its obverse, so does akinetopsia. The obverse of achromatopsia is the selective sparing of colour vision in cases of acute carbon monoxide poisoning, first reported by Wechsler (1933). The first suggestion that visual motion may be selectively spared was made by Riddoch in 1917. He had been studying patients blinded by gunshot wounds during the Great War. He had found that, in several, the extent of the scotoma was not the same when he had plotted the scotomatous field with stationary and with moving objects, the scotomatous field being commonly smaller when plotted with moving stimuli. The patients had difficulty in describing the characteristics of the moving objects, 'But they are quite sure that neither shape nor colour can be attributed to it, and that it can be detected in a field which is entirely blind to stationary objects'. Moreover, he had found not only 'That the recovery of vision for movement preceded that for the object' but also 'That the amount of recovery was greater for movement than for the object' and began in the peripheral field. These findings had led him to conclude that 'Movement may be recognized as a special visual perception',

separate and in addition to the perceptions ' . . . of light, of form, and of colour'. Riddoch's evidence for a dissociation of motion vision was thus positive, in the sense that motion vision was relatively spared, while the remaining visual capacities were severely compromised. Here there are three similarities between the literature on colour vision and motion vision disturbances following cortical lesions. The first is that the literature shows that both can be selectively damaged but also selectively spared, or at least affected much less than the other submodalities of vision. The second is that the literature of both conditions leaves obscure the neural mechanisms responsible for the selective sparing; and the third is that neither literature describing the sparing of these attributes has made the slightest impression in the neurological world.

No sooner had Riddoch published his paper than his evidence was quickly dismissed by Holmes (1918) and, like achromatopsia, 'vanished' (Damasio, 1985) from the literature. Holmes wrote 'Riddoch's statement that 'the recovery of the appreciation of movements begins in the periphery of the field and extends towards central vision' is certainly incorrect', since 'In all my cases . . . the blindness was total . . . neither the presence nor the movement of any object of reasonable size could be recognized'. This would seem to leave out of account Holmes' (1918) own Case 11 in the same paper. This patient had been studied 3 months after injury, when it was found that 'there was a considerable return of vision in the periphery . . . but he was generally conscious only of the movement of the white test object, and saw it only 'as through a mist' and as a 'dirty grey colour'. Holmes conceded that there was an element of truth in Riddoch's work since 'I can . . . confirm his statements that the presence of a moving object may be recognized in which it is not perceived when stationary and in which its shape cannot be appreciated'. But there was an explanation for this, since 'I have always found that the acuity of vision in these (scotomatous) areas is considerably diminished . . . Further, colour vision is invariably affected in these areas'. Just as he thought that there was no dissociation of colour vision in striate cortex, so he explained somewhat impatiently ' . . . that the condition described by Riddoch should not be spoken of as a dissociation of the elements of visual sensation' since '. . . occipital lesions do not produce true dissociations of function with intact retinal sensibility'. With this, he despatched both Riddoch and the meagre evidence for a dissociation of function in the visual cortex.

Riddoch had tried to ascertain the position of the injuries in the brain as far as possible. He had relied mainly on x-ray examination and on notes taken during the operation. This was the best that could be done at the time. But these did not reveal the total area of the brain involved. Riddoch wrote 'At best only approximations can be made. Apart from the attempt to estimate how much brain substance has been destroyed . . . the presence or absence of sepsis has to be taken into account. Moreover, vascular lesions are never absent'. With these cautionary remarks he proceeded nevertheless to account for his findings in terms of the striate cortex alone, except where there was no dissociation of object and movement vision, i.e., where the extent of the scotoma for the two was the same. This led him to suggest ' . . . tentatively, that coincidence of the two fields and the absence of any signs of recovery are due to the injury being more extensively subcortical', even though there is some evidence to suggest that some of the wounds may have included the prestriate visual cortex. At that time, this was no cause for concern, since under the spell of Holmes and of Henschen, the striate cortex alone was regarded as the visual perceptive centre (*see* Zeki, 1990, for review). Today, the residual vision

consisting of motion perception could be accounted for by subcortical mechanisms although this is not certain (*see below*).

Riddoch was thus cautious in his interpretation, explaining the relative sparing of motion vision in terms of the striate cortex alone. This was not greatly dissimilar to the way in which Marie and Chattelin (1914–1915) and Monbrun (1939), among others, had tried to account for achromatopsia, though they had tried to make out that the absence of colour vision could be accounted for by its greater sensitivity to, and slower recovery from, cortical onslaught. Here the history of akinetopsia differs from that of achromatopsia. Although Verrey (1888) had tried to account for achromatopsia in terms of lesions in the fusiform and lingual gyri, he had nevertheless considered these two gyri to be part of the primary visual receptive cortex. When it became clear, through the work of Henschen and others, that the lingual and fusiform gyri were outside the primary visual (striate) cortex, the resistance to the notion of a colour centre outside the striate cortex began to mount (*see* Zeki, 1990). But, except for implicating subcortical centres, nowhere did Riddoch implicate cortical areas outside the striate area. Perhaps because of this conservative explanation, perhaps because he had positive evidence (presence of movement perception in an otherwise scotomatous field rather than its total absence in an otherwise nonscotomatous field, as in achromatopsia) and perhaps because he had studied many different subjects rather than single cases, neither his evidence nor his interpretation were challenged after the initial, and apparently successful, dismissal by Holmes (1918). Instead they suffered an even worse fate. They were completely forgotten. It would be difficult to find a reference to this work in most standard books of neuro-ophthalmology published before 1983, even those which dismiss the evidence for cerebral achromatopsia. It was only many years after Riddoch's paper was published that the notion of movement dissociation was to be dismissed again, although without any reference to Riddoch's work (*see below*).

Two years after Riddoch's paper was published, Best (1919) speculated that the perception of movement and of space may be the function of areas outside the striate cortex. But he had no evidence, either clinical or experimental. Moreover, his ideas on the organization of the visual cortex were themselves a little bizarre. He believed that the sole function of the striate cortex was the fusion of the images from the two eyes. This theory was not very different from that of Ewens (1893), which Henschen (1894) was to dismiss as 'a theory in the air'. So the subject remained until Poppelreuter (1917, 1923) speculated that there must be a separation of functions in the striate cortex (calcarina). He considered that the 'sensation of movement, or change' is separately localizable, and localized, within the striate cortex. But he did not specify whether the separate submodalities were segregated according to layer, as Wilbrand (1884) and Halpern and Hoff (1929) had speculated for form, colour and the light sense (though neither had considered movement to be a submodality of vision) or whether they were localized in subareas within the primary visual cortex, as Verrey (1888) had mistakenly believed. Lacking the evidence, Poppelreuter's speculations were soon to be relegated to oblivion as well. A view of the striate cortex as the unique visual perceptive area seems to have emerged as a consensus, with Monbrun (1939) writing that '*À l'heure actuelle, tous les auteurs sont ralliés à la théorie du centre* [*visuel*] *cortical unique*'. When Holmes (1945), in his Ferrier Lecture, spoke of the striate cortex as 'a merely perceptive centre', adding that 'The perception of colour also depends on (it) . . . there

is no evidence that this is subserved by any other part of the brain', he saw no reason to make a special point of emphasizing the same for motion. Riddoch's work had been truly forgotten and the speculations of Best and Poppelreuter had not been taken seriously by anyone. Only the evidence for a dissociation of colour and for a colour centre outside the striate cortex needed to be dismissed, so Holmes believed in 1945.

Just as Teuber *et al.* (1960) had believed that there is no dissociation of colour perception following cerebral lesions, so Teuber (1960) was sceptical of the dissociation of motion. In 1960, he wrote, 'It is commonly thought that cerebral lesions implicating central visual pathways tend to produce greater impairment for pattern than for motion perception. The evidence for such a statement, however, is unconvincing. It is not unexpected to find areas in defective visual fields where targets are perceived when they are in motion but not when they are stationary. The movement takes the target over a wider angular extent in the field and thus produces more stimulation. Moreover, the movement prevents the abnormally rapid fading in some (though not all) defective visual fields. Actual measurements . . . demonstrate that motion perception is impaired *pari passu* with defects in the forming of contours'. His views were unchanged over a decade later, following a detailed examination of patients with longstanding visual field defects following missile wounds to the brain, most of them sustained during the Korean War. Using both kinetic and static perimetry, Koerner and Teuber (1973) explained that a ' . . . rather surprising feature of our present findings was the thorough going association of symptoms that we encountered: once the sensitive technique of static perimetry was taken as a baseline, regional losses as defined by kinetic perimetry (and other techniques) turned out to be essentially redundant sources of information on the status of these visual fields', leading them to the conclusion that 'There was no evidence that one could dissociate detection of moving and stationary targets.' This statement was not greatly different from the earlier statement of Teuber *et al.* (1960) that 'There is thus no evidence for a genuine dissociation . . . of color and form vision'. Riddoch would not have had the satisfaction of seeing his work thus dismissed. Neither his name nor his work are referred to in this paper.

THE SPECIALIZED MOTION AREA (AREA V5) OF THE VISUAL CORTEX

At about the time that Koerner and Teuber (1973) dismissed the notion of a dissociation of motion vision from other kinds of vision, a report of a visual area lying outside the striate cortex of the macaque monkey, and apparently specialized for visual motion, was published (Zeki, 1974), a preliminary account having been published earlier (Dubner and Zeki, 1971). The area in question, area V5, lies in the posterior bank of the superior temporal sulcus and receives a direct and highly convergent input from the striate cortex, V1 (Cragg, 1969; Zeki, 1969, 1971). All cells in it are motion selective, and the overwhelming majority are directionally selective; most are not orientation selective and none is wavelength selective (Zeki, 1974). So distinctive is the area in its motion selectivity, that it was first referred to as the motion area, and subsequently called V5. It was the first visual area to provide strong evidence suggesting that the visual cortex of the primate brain is functionally specialized, with different areas processing different attributes of the visual scene, findings which were to lead to a theory of functional specialization in the visual cortex (Zeki, 1978). Within the context of this theory, it

became possible to entertain the notion that lesions in specific visual areas outside the striate cortex may lead to specific visual defects, rather than a global scotoma. Since its discovery, area V5 has been the subject of numerous physiological studies, all of which are in agreement about its specialization for motion (e.g., Gattass and Gross, 1981; Albright, 1984; Movshon *et al.*, 1985). An apparently similar area in the New World owl monkey was also identified in mapping studies by Allman and Kaas in 1971. Its physiological properties were later characterized and most cells in it were also found to be directionally selective (Zeki, 1980*a*; Baker *et al.*, 1981). The homologous area in the owl monkey was called MT, and the term is commonly also used as an alternative to V5 in the Old World monkey, although I much prefer to use the term V5 when referring to the macaque monkey and to man (*see below*) and the term MT when referring to the owl monkey.

It is obvious then that by the time that Zihl *et al.* (1983) had published their single case study of akinetopsia, a considerable amount of experimental evidence had accumulated to show that there is a specialization for motion perception in the visual cortex and that the specialized area lay well outside the striate cortex. This is very different from the history of achromatopsia. Several patients with acquired achromatopsia resulting from lesions outside the striate cortex had been described before the demonstration of functional specialization in the prestriate visual cortex. There were conceptual difficulties in accepting that evidence, and hence it was dismissed (*see* Zeki, 1990, for a review). With akinetopsia, the one remarkable case came after the demonstration of functional specialization in the prestriate cortex. By 1983 all conceptual difficulties relating to the representation of a particular visual submodality in a specialized area, lying outside the striate cortex, had been removed. There seemed little reason to doubt that an akinetopsic syndrome could result from a specific lesion in a specific, motion selective, visual area. Had there been several descriptions of akinetopsia before the evidence for a functional specialization in the macaque monkey prestriate cortex had accumulated, it seems likely that the causative factor for the syndrome would have been subject to the same acrimonious debate as with achromatopsia (*see* Zeki, 1990).

THE POSITION OF AREA V5 IN MAN

The lesion in the akinetopsic patient of Zihl *et al.* (1983) was bilateral but more extensive on the left side. It included the posterior portion of the middle temporal gyrus, the retrorolandic area and the temporoparietal and occipital white matter. It also involved the subcortical white matter and the lateral occipital gyri. But it did not involve the striate or calcarine cortex. As defined recently by the technique of positron emission tomography (Cunningham *et al.*, 1990; Zeki *et al.*, 1991), area V5 occupies the temporo-parieto-occipital pit, at the boundary of areas 19 and 37, a cortical region compromised in the patient of Zihl *et al.* Nevertheless, the lesion in this patient was quite extensive and it was likely to have involved cortical tissue other than V5, in addition to white matter. Given the specificity of the visual disturbance, it seems likely that the damaged cortex outside V5 is also motion-related visual cortex. In the macaque monkey, area V5 is surrounded by satellite areas (Zeki, 1980*b*; Maunsell and Van Essen, 1983; Desimone and Ungerleider, 1986; Tanaka *et al.*, 1986; Komatsu and Wurtz, 1988). These are all involved with processing of motion-related information but in ways which differ from the role of V5. They have not yet been delineated in man as separate areas

but are likely to be in the future, with the refinement of noninvasive techniques for localization. This is especially so since distinct defects related to tracking of visual stimuli have been identified in patients with lesions in cortex, in one of whom the defect was associated with akinetopsia (Thurston *et al.*, 1988). But the lesions in this latter study were relatively large. This makes it difficult to relate specific cortical loci to specific visual motion-related functions at the present time.

THE AKINETOPSIC SYNDROME IN MONKEYS

Recent evidence has shown that specific defects in motion perception can result from specific lesions in area V5 of the macaque monkey (Newsome *et al.*, 1985; Newsome and Paré, 1988). In particular, after ibotenic acid injections into area V5, monkeys are impaired in their ability to detect motion, there being a large elevation in motion detection thresholds while contrast detection thresholds are unaffected. The syndrome is short lived, however, the monkeys recovering their normal detection thresholds after a few days. The reason for this is not known but presents a contrast to the more pronounced and long-lasting akinetopsia described by Zihl *et al.* (1983). It is certain that the lesions in these controlled experiments were small compared with the relatively large lesions in the patient of Zihl *et al.* and that of Thurston *et al.* (1988). It is therefore possible that should a lesion in a monkey involve both V5 and its satellite areas, a more pronounced experimental akinetopsia would result.

THE MOTION PATHWAYS OF THE CEREBRAL CORTEX

On the face of it, the sparing of motion perception in Riddoch's 5 patients is surprising, given the fact that all had suffered from gunshot wounds, in 2 of whom the point of entry and exit of the projectile could be determined and in all of whom considerable damage to the occipital cortex and the optic radiations can be inferred from the presence and extent of the scotomas. In macaque monkey striate cortex, the directionally selective cells responsible for motion detection are situated in layer 4B and, to a lesser extent, in upper layer 6. They receive input predominantly from the magnocellular layers of the lateral geniculate nucleus, through layer 4C. The directionally selective cells in both layers project to area V5 (Lund *et al.*, 1975; Shipp and Zeki, 1989*a*). In both layers, cells projecting to V5 are separated from each other by cells projecting elsewhere, thus suggesting a degree of functional segregation for motion-selective cells (*see* fig. 1). Layer 4B also projects to V3 and to a subcompartment of area V2, the thick stripes (Shipp and Zeki, 1985; Livingstone and Hubel, 1987). In V2, directionally selective cells are concentrated in the thick stripes. (DeYoe and Van Essen, 1985; Shipp and Zeki, 1985). The thick stripes of V2 project in turn to both V3 and V5 (DeYoe and Van Essen, 1985; Shipp and Zeki, 1985, 1989*a*). These projections are quite different from those of layers 2 and 3 of V1, or of the thin stripes and interstripes of V2 (for review, *see* Zeki and Shipp, 1988).

It is conceivable that with superficial wounds affecting layers 2 and 3 of V1 and sparing layer 4 as well as the other layers, both colour and form vision would be compromised, while motion vision would be selectively spared. In 3 of Riddoch's 5 patients with selective sparing of motion, no exit point for the projectile could be found, and it is possible that the wounds were relatively superficial, though the extent of the scotomas in each

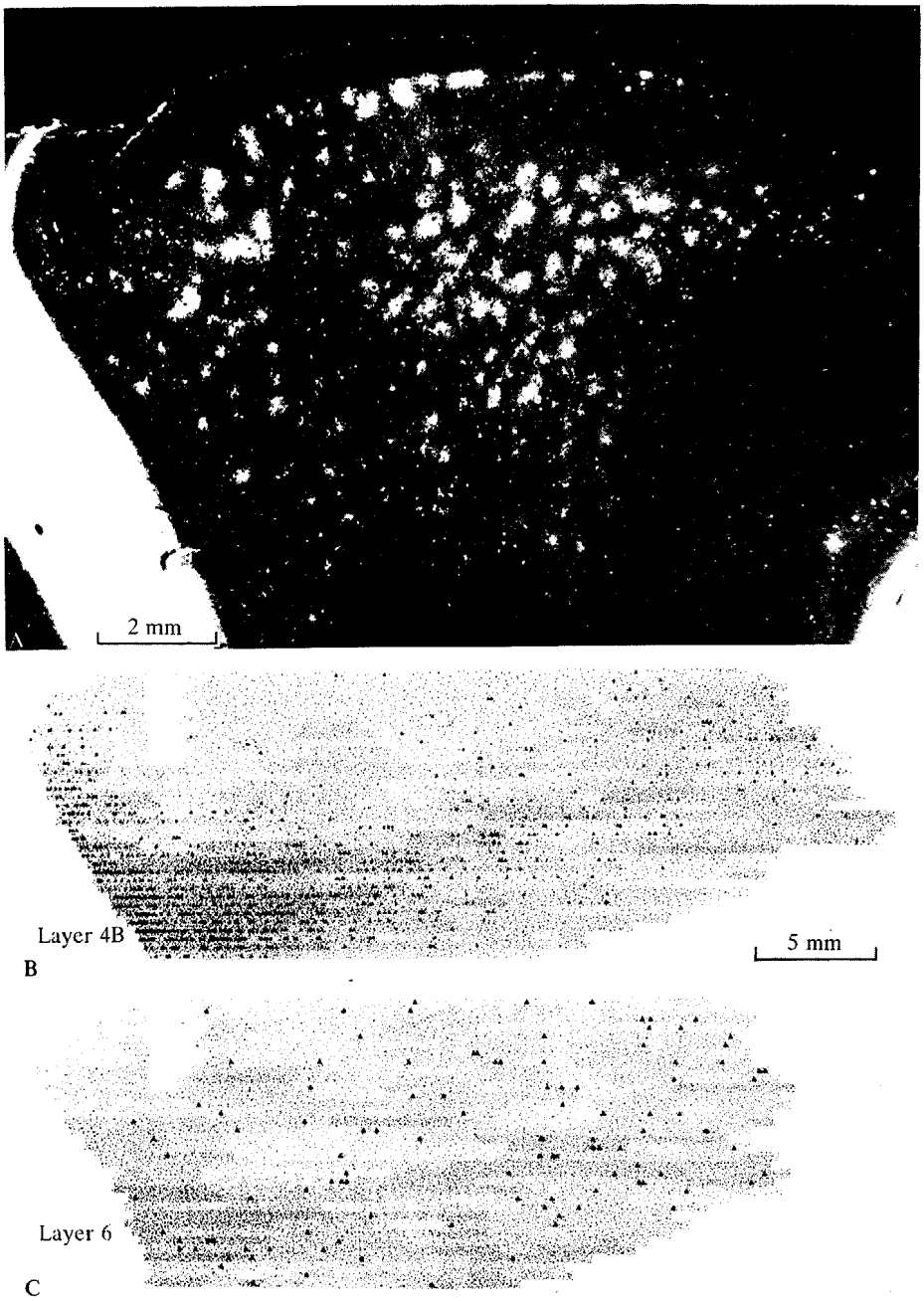

FIG. 1. A, reconstruction showing the tangential distribution of wheat-germ agglutinin horseradish peroxidase label in layer 4B of V1 following an injection of the same substance into area V5. Note the pronounced clustering of the label; B, composite computer-aided reconstruction of the distribution of labelled cells and fibres in layer 4B of V1; C, a similar reconstruction for layer 6. The reconstructions were prepared from many sections derived from the same brain. Black triangles show the distribution of cells; stippling the distribution of the return (re-entrant) fibres. Note the more widespread distribution of the return fibres. From Shipp and Zeki (1989*a*) with permission.

argues for a greater involvement. The problem is not quite so simple, however, for there is another, possibly dynamic, form system based on area V3, which also receives its projections from layer 4B (Zeki and Shipp, 1988). If layer 4B were selectively spared, at the very least some rudimentary form vision, based on the 4B to V3 system, would have been expected. Yet none of Riddoch's 5 cases had any ability to perceive forms. A more likely explanation may be that subcortical mechanisms may have been involved, although there is no evidence that this is so. In present day terms, the lesions are inadequately charted in Riddoch's patients and it is therefore difficult to account in satisfactory terms for the selective sparing of motion in these patients. There is no documented case of akinetopsia being the result of the specific involvement of the entire motion pathway from layer 4B to V5. In this, akinetopsia stands in contrast to achromatopsia where in some conditions at least, for example acute carbon monoxide poisoning or arterial insufficiency, a selective sparing or involvement of the specialized colour pathways has been suspected (*see* Zeki, 1990). It is also worth noting that the back projection from V5 to layer 4B is much more widespread than the forward, prograde, projection and encompasses not only the territory of cells in layer 4B projecting to V5 but also the territory of cells projecting to other destinations, among them V3 (figs 2, 3). Similarly, the back projections from area V5 to area V2 are also more widespread than the forward projections and, though densest in the region of the thick stripes from which V5 receives its V2 input, also include the territory of the thin stripes and the interstripes, subdivisions of V2 which do not project to V5 (Zeki and Shipp, 1988; Shipp and Zeki, 1989*a*, *b*). Given these prominent back projections, some secondary disturbances, beyond akinetopsia, might be expected after V5 lesions, but these are likely to be more subtle and are likely to have escaped notice.

It is therefore possible that the dissociation of motion vision observed by Riddoch cannot be accounted for in terms of the sparing of the relevant mechanisms within striate cortex; it may have been the consequence of intact subcortical mechanisms, or possibly the manifestation of a phenomenon analagous to residual vision or blindsight (Pöppel *et al.*, 1973; Perenin and Jeannerod, 1978; Weiskrantz, 1986; Blythe *et al.*, 1987). Present indications are that there is a small projection from the lateral geniculate nucleus to the prestriate cortex (Fries, 1981; Yukie and Iwai, 1981). Stoerig and Cowey (1989) have shown that blindsight patients can undertake wavelength descriminations, presumably through the direct pathway to V4 from the lateral geniculate nucleus. It is possible that a similar condition accounts for the phenomenon described by Riddoch. Holmes and Teuber may thus have been quite right to emphasize that the dissociation of motion vision is not the consequence of a sparing of the relevant mechanisms in the striate cortex itself. It is the insistence of both authors that visual submodalities are not dissociated in the cortex and that the striate cortex alone is responsible for visual perception that renders their conclusions untenable.

AKINETOPSIA AS A FAILURE OF A CENTRAL SYNTHETIC MECHANISM IN THE VISUAL CORTEX

Akinetopsia shares a common feature with both achromatopsia and prosopagnosia in being a condition in which the visual input to the specialized areas is intact, or largely so, while the relevant central area, being deranged, is unable to use the information reaching the cortex. In achromatopsic and prosopagnosic patients, as well as in the

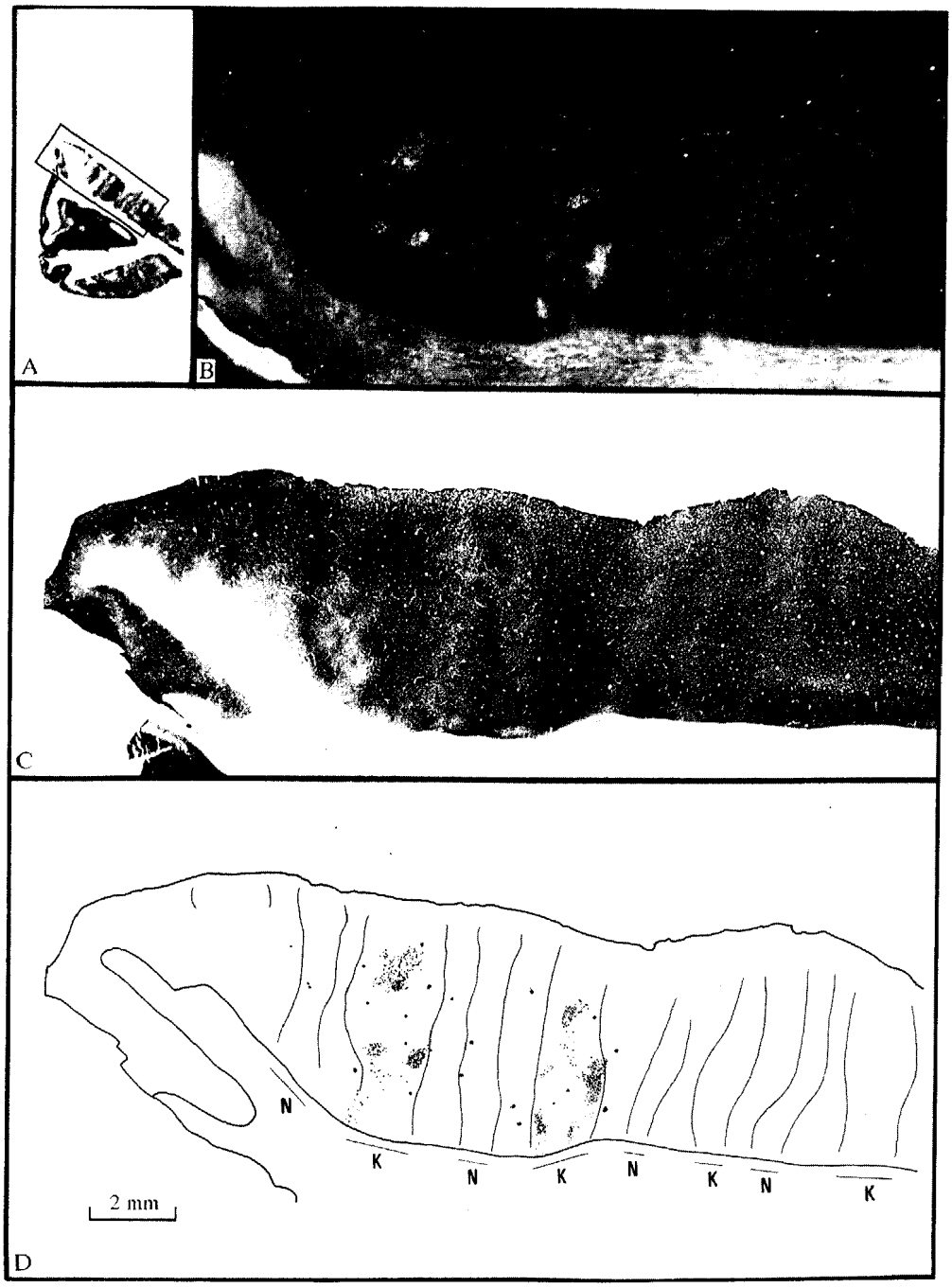

FIG. 2. Tangential sections through part of area V2 in the occipital operculum of the macaque monkey (A). B, dark-field photomicrograph to show the distribution of the label in V2 following an injection of horseradish peroxidase (HRP) into V5. C, contiguous section, stained for cytochrome oxidase activity to reveal the pattern of thick and thin stripes within V2. D, reconstruction made from B and C to show that the HRP label is confined to the territory of the thick cytochrome oxidase stripes. N = thin stripe; K = thick stripe. From Shipp and Zeki 1989*b*, with permission.

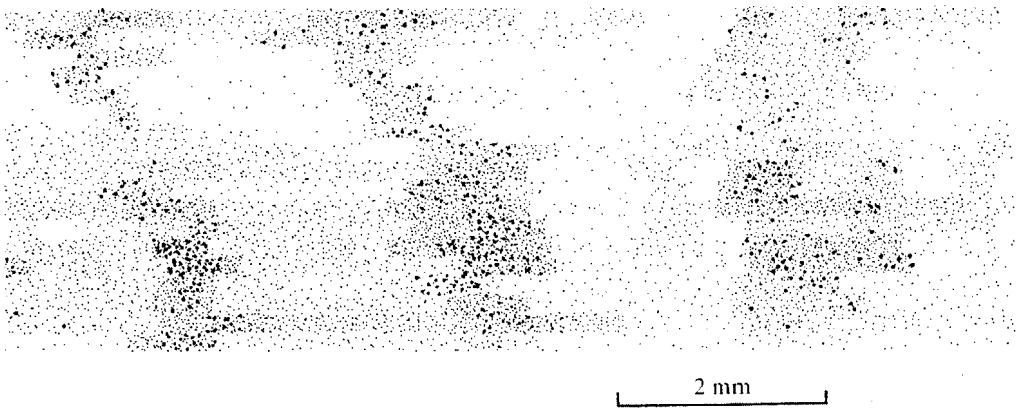

FIG. 3. Computer-aided reconstruction of the distribution of labelled cells and return fibres in tangential sections through V2, following an injection of label into V5. Conventions as in fig. 1. From Shipp and Zeki 1989*b*, with permission.

akinetopsia patient, retinal mechanisms have been found to be intact and in the latter patient even the visual evoked potentials recorded from the occipital scalp were within the normal range (Mollon *et al.*, 1980; Zihl *et al.*, 1983). Detailed psychophysical investigation of Zihl's patient has suggested strongly that all visual mechanisms up to the striate cortex are intact (Hess *et al.*, 1989), pointing to a more central failure. In all these conditions, the evidence for a failure in the central synthetic mechanism is both experimental and clinical.

In colour vision, experimental evidence suggests that the specialized cells of area V1 do not code for the colour of the stimulus, but for the wavelength (Zeki, 1983). Thus a long-wave selective cell in V1, for example, will respond to a surface of any colour provided it is reflecting a sufficient amount of long-wave light, in contrast to some cells in V4 whose responses correlate with the human perception of colours and are, within a wide range, independent of the precise wavelength-energy composition of the light reflected from the surfaces in their receptive fields. In other words, the cells of V1 are responding to component wavelengths while those of V4 are responding to the colour. It is for this reason, among others, that I have conceived of colour as being constructed by the cortex rather than being merely analysed by it (Zeki, 1984). The ingenious work of Movshon *et al.* (1985) has shown similarly that the directionally selective cells of layer 4B of V1 respond to the component motions and are unable to detect the global direction of motion of an object, which is the function of V5. In a similar way, Pallis (1955) and others have shown that a prosopagnosic patient can see all or many details of a face, such as the nose, eyes and ears, but cannot combine the features to construct a face.

CONCLUSIONS

In summary, the literature on achromatopsia contrasts with that of akinetopsia. The description of akinetopsia came at a time when the neurological world was ready to accept

it, precisely because the experimental evidence made it reasonable. Had the syndrome been described before the experimental evidence for a functional specialization in the prestriate visual cortex, it would have probably been accepted, accounted for in terms of the striate cortex and then forgotten about, just as with achromatopsia. Its reverse, the presence of motion perception in blind fields, was challenged at the very beginning and then fell into oblivion. Similarly, the presence of colour vision in patients who are severely incapacitated visually was never challenged and was also relegated to oblivion. Had the causative factor for akinetopsia been attributed to a lesion outside the striate cortex, it would probably have been dismissed, just like achromatopsia and, like it, would have 'vanished' (Damasio, 1985) from the clinical literature. The remarkable contrast in the fate of these two syndromes, akinetopsia and achromatopsia, stimulates much speculation concerning the nature of the evidence that makes neurological syndromes, both those which have been described and those which await description, acceptable.

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