Selective deficit of visual size perception: two cases of hemimicropsia

Laurent Cohen, Françoise Gray, Christian Meyrignac, Stanislas Dehaene, Jean-Denis Degos

Abstract

Hemimicropsia is a rare disorder of visual perception characterised by an apparent reduction of the size of objects when presented in one hemifield. We report two cases of hemimicropsia resulting from focal brain lesions. The first patient was an art teacher and could accurately depict his abnormal visual perception. He subsequently died and his brain was examined post mortem. In the second patient, micropsia was assessed by a quantified size comparison task. The size of a given object is normally perceived as constant across any spatial position. Hemimicropsia may thus be considered a limited violation of the size constancy principle. Behavioural and anatomical data are discussed in relation to the neural basis of visual object perception in humans.

(*J Neurol Neurosurg Psychiatry* 1994;57:73–78)

Service de Neurologie, Hôpital de la Salpêtrière, Paris, France
L Cohen

Service de Neuropathologie, Hôpital Henri Mondor, Créteil, France
F Gray

Département de Neurosciences, CHU Henri Mondor, Créteil, France
C Meyrignac
J-D Degos

Laboratoire de Sciences Cognitives et Psycholinguistique, INSERM and CNRS, Paris, France
S Dehaene

Address correspondence to Dr L Cohen, Service de Neurologie, Hôpital de la Salpêtrière, 47 Boulevard de l'Hôpital, 75651 Paris CEDEX 13, France.

Received 27 November 1992 and in revised form 8 February 1993. Accepted 15 February 1993

It is now widely acknowledged that different types of visual information are processed in the brain along anatomically distinct pathways.[1] Neuropsychology has long provided data suggestive of this organisation, through the description of deficits selectively affecting the perception of—for example, colour or movement.[2,3] Building upon these initial clinical data, animal studies and brain functional imaging in humans have recently allowed a more systematic and detailed study of these processing modules.[4,5] There is, however, only scant evidence about the neural basis of size constancy, one major property of the normal visual system.[6] The size of a given object is perceived as constant, whatever its location and distance. A correction process allows for the location and the size of the retinal projection in the course of object perception.

Dysmetropsia (also called dysmegalopsia), is a disorder of visual perception characterised by an apparent modification of the size of perceived objects. Objects appear either shrunk (micropsia) or enlarged (macropsia), relative to their normal size. An overview of published reports shows that micropsia and macropsia result from similar causes,[7–9] but micropsia occurs much more frequently.

Monocular micropsia can result from retinal oedema causing a dislocation of the receptor cells.[10] Exceptionally, lesions affecting other parts of the extracerebral visual pathways, such as the chiasmatic tumor reported by Bender and Savitsky,[11] can cause micropsia. Micropsia of neurological origin is most frequently reported as a manifestation of temporal lobe seizures.[12,13] It then affects either the entire visual field, or the object that the patient fixates at the moment of the seizure. It is accompanied by a broad variety of temporal epileptic symptoms.[7,8] More rarely, micropsia can be part of purely visual seizures. It then affects only one half of the visual field, and is accompanied by other cerebral visual disturbances, such as metamorphopsia or dyschromatopsia.[14,15] Apart from epileptic phenomena, transient micropsia can also result from migraine,[16] or from the action of mescaline and other hallucinogenic drugs.

Permanent dysmetropsia following focal cerebral lesions is rare and affects lateral homonymous segments of the visual field.[17,18] It often may be overlooked, because of severe associated visual impairments,[19,20] or because of the mildness of the functional disability.[7] The only probable case of permanent dysmetropsia with satisfactory localisation data was recently reported by Ebata *et al*,[21] with an unexpected retrosplenial lesion. We describe two cases of pure hemimicropsia following posterior cerebral damage.

Case 1

The patient was a 50-year-old right-handed man with no history of psychiatric disorders, working as an art teacher. Since the age of about 40, he had suffered occasional attacks of ophthalmic migraine. His mother and one brother had similar ophthalmic migraine. At the age of 44, he had suffered a myocardial infarct. On his way to a routine cardiological consultation, the patient experienced sudden left homonymous hemianopia. At the same time, he noticed that he could not recognise the face of a friend who was with him at that moment. He was able to find his way to the hospital. He did not recognise his usual cardiologist, nor the other members of the medical staff. One hour later, right-sided throbbing headache began, and lasted for about two hours. The patient was admitted to the hospital. Two days later, left hemianopia had almost completely disappeared but the prosopagnosia was still severe. The patient could not identify the members of his family visually, although he recognised them readily upon hearing their voices. In addition, the patient had some difficulty analysing complex

Figure 1 Sample drawings of symmetrical objects by the first patient. The left half is consistently larger than the right half.

visual scenes. He would describe isolated parts of the scene, mostly picking out the region on the right handside, but had difficulties perceiving the visual field as a coherent whole, a behaviour suggestive of simultanagnosia with slight left spatial neglect.[22] Finally, he complained that he had special difficulty perceiving depth, proportions, and symmetry. There was no sensory or motor deficit, aphasia, alexia, or apraxia.

One week later, prosopagnosia and simultanagnosia had receded. Visual field was normal on Goldmann perimetry. The patient complained, however, that objects falling in his left visual field appeared somewhat shrunk and compressed. He felt it particularly difficult to appreciate the symmetry of pictures. When drawing, he spontaneously tended to compensate for his perceptual asymmetry by drawing the left half of objects slightly larger than the right half (fig 1). He was also presented with truly symmetrical patterns, which he perceived as smaller on the left than on the right. When asked to correct them so as to make them look symmetrical, he either expanded the left part of the pattern, or reduced its right part (fig 2). In a sample of six spontaneous or corrected drawings of symmetrical objects, linear measures in the left half were on the average 16% larger than the corresponding measures in the right half. The patient did not mention any anomaly of colour or movement perception, which were not further explored.

Ten days after onset, CT showed a hypodense area in the right occipital region. A diagnosis of migrainous stroke was proposed.[23] The patient died 27 months later because of disseminated pancreatic carcinoma, and a pathological study of the brain was performed.

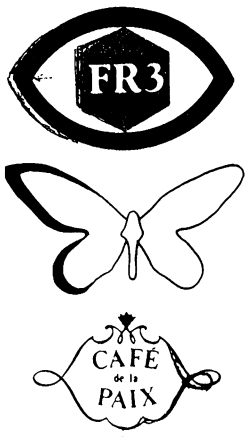

Figure 2 Examples of actually symmetrical patterns corrected by the first patient so as to make them look symmetrical to him.

POSTMORTEM EXAMINATION

Postmortem examination revealed an old, haemorrhagic, watershed infarct in the right cerebral hemisphere, at the boundary between the territories of the middle and posterior cerebral arteries. The most obvious part of the lesion destroyed the inferior occipital gyrus and the inferior part of the middle occipital gyrus (fig 3). This region corresponds to the lower part of the lateral aspect of areas 18 and 19. More anteriorly, the infarct successively affected the depth of the inferior temporal sulcus, part of the middle temporal gyrus, and the depth of the superior temporal sulcus. The occipitotemporal gyrus and the angular gyrus were spared. The cervical and intracranial arteries were normal. This completely haemorrhagic watershed infarct was interpreted as a consequence of transient arterial vasospasm having occurred simultaneously in the carotid and vertebrobasilar systems.

Case 2

The patient was a 60-year-old right-handed woman, working as a secretary, with no history of previous neurological or psychiatric disorders. She underwent surgery for a deviated nasal septum. On the fourth day following surgery, she presented with progressively increasing visual disorders. Firstly, she complained that objects and faces would disappear from her view although she could see part of them. She could see her daughter's earring, but not her daughter's face, or a hook in the wall between two windows, but not the windows themselves. Having got up from her bed, she turned back but could not see the bed any more. Secondly, she would miss objects she tried to reach, such as a glass of water. She was unable to follow a moving target visually. In addition to these complex visual impairments, she was confused, disoriented in time and space, and had anterograde amnesia. The condition worsened until the second day. Then confusion, disorientation and amnesia receded rapidly.

One week later, simultanagnosia was still present: the patient could not visually grasp the whole of an object, although she perceived isolated details. Visual field was grossly normal on confrontation. The ability to reach for targets in the right visual hemifield was severely impaired, indicating unilateral optic ataxia. The patient spontaneously reported that people's left eye (the one she saw on her right) seemed to be smaller and lower than their right one. Her difficulties in dressing and eating apparently resulted from her impaired ability to reach rather than from ideomotor apraxia. There was no prosopagnosia and colours were perceived normally. There was no sensory motor deficit; oral language comprehension and production were normal. The patient could also read correctly, except that, when arriving at the end of a line, she had great difficulty finding the beginning of the next one. CT showed bilateral occipitoparietal hypodense areas suggestive of bilateral cerebral infarction. Visually evoked potentials showed normal P_{100} waves for the two hemifields of each eye.

Two years after the stroke, the patient only complained of minor sequelae. Firstly, because of residual optic ataxia, she was slightly inaccurate when reaching with either

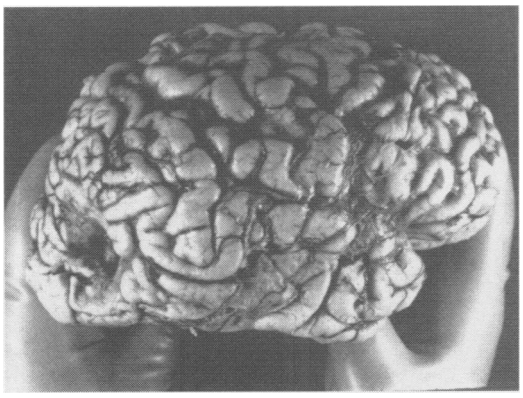

Figure 3A Lateral aspect of the first patient's right hemisphere, showing the occipital lesion, with a more anterior temporal extension.

hand for objects falling in her right visual field. She could prevent misreaching by careful visual fixation of the object she was aiming at. Secondly, the right half of symmetrical objects, such as faces, pairs of hands or telephone sets, seemed consistently smaller than their left half. The patient did not mention any anomaly of colour or movement perception, which were not further explored. There was a partial right inferior homonymous fascicular deficit on Goldman perimetry, sparing the central 15° of the visual field. Automated static perimetry was normal along the horizontal meridian.

MRI showed a limited left hemispheric lesion affecting the lower part of areas 18 and 19 and the underlying white matter (fig 4). On the right, there was a small spot of high signal intensity in the white matter posterior to the lateral ventricle.

Contrary to the first patient, the present patient was unable to represent her distorted visual perception graphically. To evaluate her micropsia objectively, we submitted her to a controlled size comparison task. On each trial, a pair of horizontally aligned circles was presented on a computer screen, and the patient had to decide which circle was larger. The patient was seated in front of the screen, at a distance of about 55 cm. She was moderately long sighted and was wearing her usual glasses during testing. The mean diameter of the two circles was 24 mm, and their centres were 72 mm apart. The largest circle was on the left in half the trials, and on the right in the other half. The target circles were preceded by a central fixation cross, and were flashed for 150 ms to avoid ocular movements. The patient was asked to press a key with her left hand if the circle on the left was larger, and to press another key with her right hand if the circle on the right was larger. She was informed that the two circles were never identical, and that she had to respond as rapidly and as accurately as possible. In a first set of trials, the difference between the diameters of the two circles was 5%, 10%, 15% or 20% of the mean diameter. Each of the eight possible targets was presented a total of 24 times in random order. A second set included trials with identical circles and trials with diameter differences of 5% and 10%. Still, the patient was instructed that the two circles were never identical. Each of the five possible targets was presented a total of 30 times in random order.

On the first set of trials, the overall pattern of responses displayed a normal distance effect: the more similar the two circles, the higher the number of errors (Kendall's $\tau = -0{\cdot}801$, $p = 0{\cdot}0023$). Performance was far better than chance on all trials (all χ^2 (1 df) $>10{\cdot}6$), except when the diameter of the right circle was larger by 5% (fig 5). In this latter condition, the patient did not respond better than chance ($\chi^2(1\text{ df}) = 0{\cdot}667$, $p = 0{\cdot}414$). This high error rate could not simply result from the difficulty to discriminate two circles whose diameter difference was only 5%, as performance was far better than chance when the left circle was larger by the same amount ($\chi^2(1\text{ df}) = 13{\cdot}5$, $p < 0{\cdot}001$). The performance was significantly better when the left circle was 5% larger, than when the right circle was 5% larger ($\chi^2(1\text{ df}) = 5{\cdot}17$, $p = 0{\cdot}023$). The results of the second set replicated those of the first one. As fig 5 shows, responses were far better than chance in all conditions with different circles (all $\chi^2(1\text{ df}) >8{\cdot}5$), except when the right circle was larger by 5% ($\chi^2(1\text{ df}) = 2{\cdot}13$, $p = 0{\cdot}144$). The performance was significantly better when the left circle was 5% larger than when the right circle was 5% larger ($\chi^2(1\text{ df}) = 9{\cdot}77$, $p < 0{\cdot}002$). The existence of a perceptive bias is therefore clearly demonstrated. When the right circle was slightly larger than the left circle, the patient perceived the two circles as identical, and randomly chose the left or the right key.

It should be noted that when the circles were identical, the patient showed no bias towards responding that the right one was

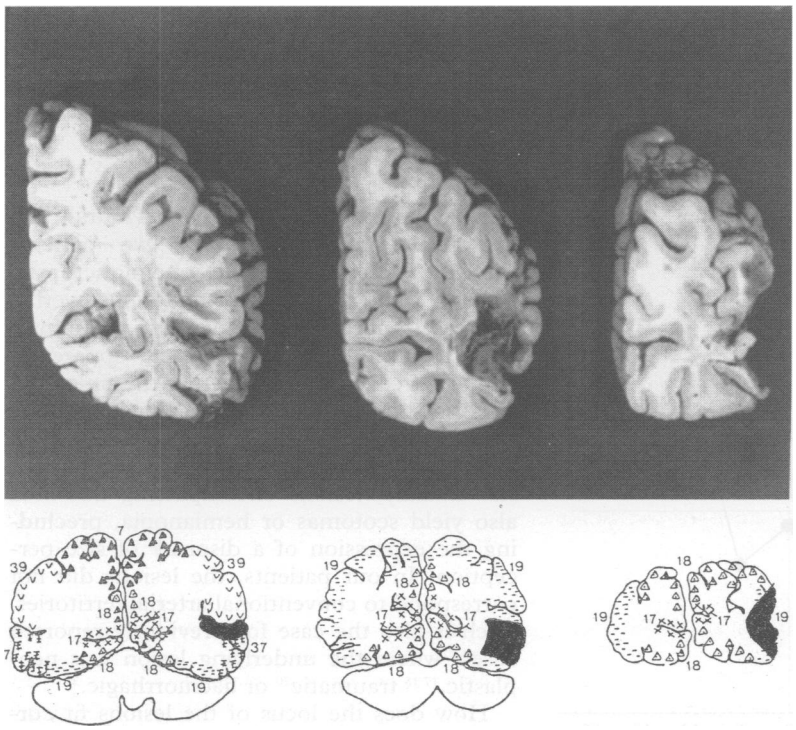

Figure 3B (Top) Frontal sections of the first patient's right occipital lobe and (bottom) corresponding reconstruction using templates from Damasio and Damasio.[33]

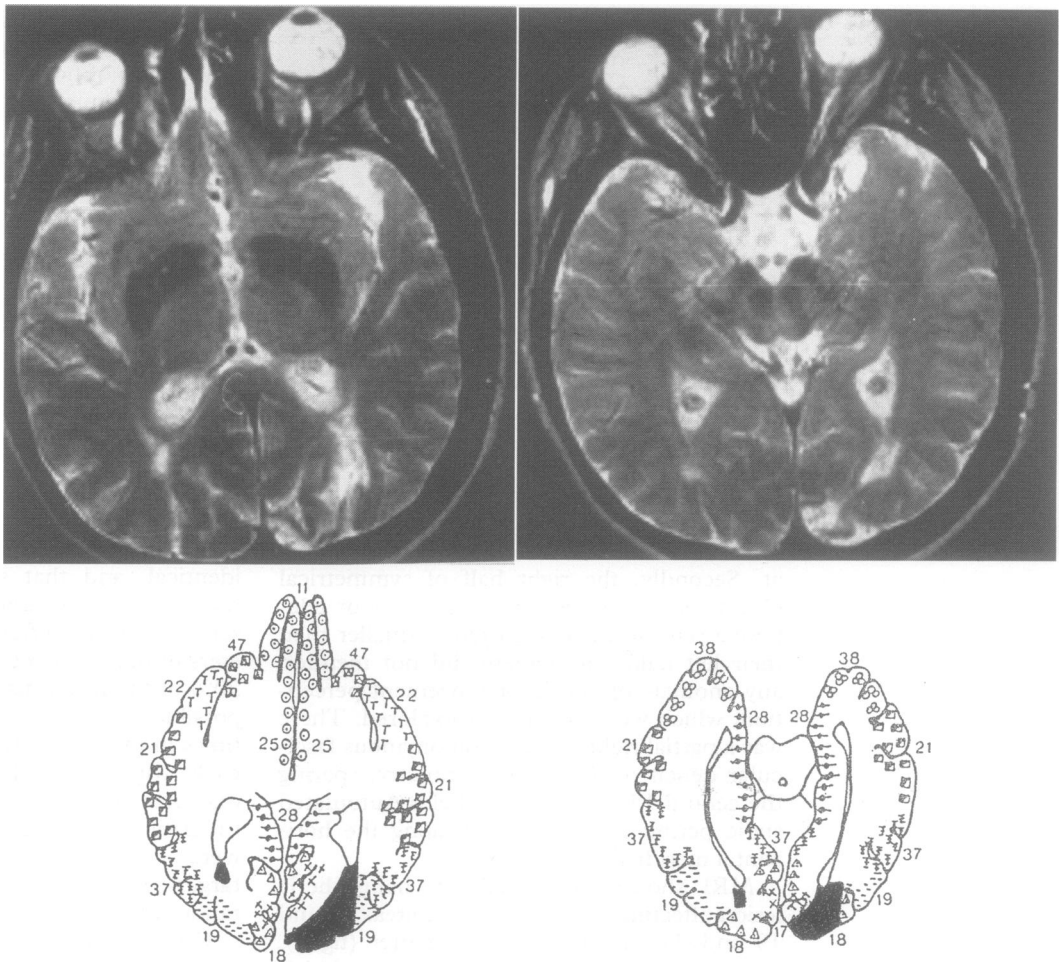

Figure 4 (Top) MRI transversal sections of the second patient's brain, and (bottom) corresponding reconstruction using templates from Damasio and Damasio[33]. The lesioned left hemisphere is represented on the right side.

smaller (χ^2(1 df) = 0·13, p = 0·715). This fact is apparently at odds with the spontaneous illusory perception of symmetrical objects as asymmetrical. It can be speculated that the actual amplitude of the bias is some proportion situated between 0% and 5%. Pairs of identical circles, as well as pairs with the right circle larger by 5%, would then be perceived by the patient as very slightly asymmetrical (about 2·5%). As stimuli were flashed very briefly, the patient could not reach a decision threshold for such small perceptual differences, and therefore responded randomly. By contrast, the spontaneous illusion of asymmetry occurred under natural conditions with unlimited viewing time.[24]

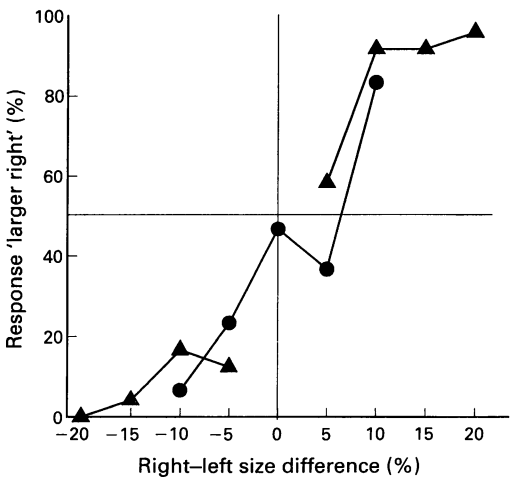

Figure 5 The second patient's performance on the size comparison task (triangles: first set; circles: second set). When the right circle was 5% larger than the left one, she equally often responded 'larger right' and 'larger left', probably perceiving the two circles as identical.

Discussion

We have presented clinical, behavioural, and anatomical data concerning two patients showing isolated stable hemimicropsia. Figures 3 and 4 show that the lesions overlap in areas 18 and 19 and underlying white matter. In both cases, the deficit apparently resulted from a lesion affecting the unimodal visual association cortex. Vascular lesions are rarely restricted to this cortical region, which could explain the infrequency of hemimicropsia. Usually, lesions encompassing this area also yield scotomas or hemianopia, precluding the expression of a disorder of size perception. In our patients, the lesions did not correspond to conventional arterial territories. Such is also the case for previously reported cases, where the underlying lesion was neoplastic,[17 18] traumatic[20] or haemorrhagic.[21]

How does the locus of the lesions fit current concepts about the organisation of the visual system? A parallel can be drawn

between size perception and colour perception, although the latter domain is better understood at present. The spectral composition of the light reflected by an object differs according to the spectrum of the ambient light. The brain, however, takes into account the characteristics of the ambient light and computes a constant subjective colour over wide illumination changes.[25] Similarly, the size of the retinal projection of a given object depends on its distance and angular position relative to the eye. Object perception requires that the image be corrected for these parameters, to compute a constant representation of the actual size of the object. In this respect, the property of size constancy is largely comparable to colour constancy. It may appear natural that the ventral occipitotemporal visual pathway, which is responsible for object vision, including probably colour constancy,[2] should also mediate perception of size.[26 27] Accordingly, Hart, Lesser and Lee[28] have reported a patient who, under electrical stimulation in the posterior part of the left middle temporal gyrus, could not make verbal size judgments—for example, "Is a tree bigger than an ant?". The authors suggest that this very selective deficit resulted from a disconnection between superior temporal language areas and inferior temporal regions responsible for the processing of visual size. There is further experimental evidence from animal studies pointing to the involvement of the occipitotemporal pathway in the perceptual equivalence of objects across translations of retinal position,[29] and across size modifications.[30 31] More specifically, cells in area V4 have been shown to be tuned to the length or to the width of visual stimuli within large receptive fields,[4] and removal of this area results in impaired size discrimination.[32]

In our patients, as in other cases of permanent dysmetropsia,[17 18 21] the anomaly was restricted to homonymous segments of the visual field. The micropsia affected the left hemifield in the first patient, and the right hemifield in the second patient. These observations suggest that each hemisphere is in charge of size processing only for contralateral stimuli. In primates, the size of the receptive field increases along the pathway from V1 to V4 and from V4 to inferior temporal cortex. Up to area V4, neurons show excitatory responses only to contralateral stimuli. By contrast, inferior temporal neurons also respond to stimuli presented in the ipsilateral field.[25] It is therefore plausible that hemimicropsia should result from lesions affecting the posterior part of the ventral visual pathway. This idea is compatible with the lesions we have described, which overlap in the lower part of the lateral aspect of areas 18 and 19. More precise correlations with animal data can only be tentative.

The two cases we have reported suggest that size perception may be dissociated from other aspects of visual perception such as colour or movement. Part of the ventral visual association cortex probably computes a representation of size that is constant across variations of distance and position. In both patients the relative size of objects within one hemifield was seemingly normal, indicating only limited impairment of the size correction processes. We may suggest that lesions affecting this functional module only resulted in a mistuning affecting the size correction process for stimuli within one hemifield. This impairment would therefore yield interhemispheric discrepancy in size perception.

1 Livingstone M. Segregation of form, color, movement, and depth processing in the visual system: Anatomy, physiology, art, and illusion. In: Cohen B, Bodis-Wollner L, eds. *Vision and the Brain*. New York: Raven Press, 1990:119–38.
2 Zeki S. A century of cerebral achromatopsia. *Brain* 1990;**113**:1721–77.
3 Zihl J, Von Cramon D, Mai N, Schmid CH. Disturbance of movement vision after bilateral posterior brain damage. *Brain* 1991;**114**:2235–52.
4 Desimone R, Schein SJ. Visual properties of neurons in area V4 of the macaque: sensitivity to stimulus form. *J Neurophysiol* 1987;**57**:835–68.
5 Haxby JV, Grady CL, Ungerleider LG, Horwitz B. Mapping the functional neuroanatomy of the intact human brain with brain work imaging. *Neuropsychologia* 1991;**29**:539–55.
6 Sedgwick HA. Space perception. In: Boff KR, Kaufman L, Thomas JP, eds. *Handbook of perception and human performance, Vol 1*. New York: John Wiley & Sons, 1986:21/1–21/57.
7 Mouren P, Tatossian A. Les illusions visuo-spatiales: Étude clinique. *Encéphale* 1963;**52**:439–80.
8 Müller C. Mikropsie und makropsie. *Monatschr Psychiat Neurol* 1956; Suppl.**98**:1–60.
9 Miller NR. Central disorders of visual integration. In: *Walsh and Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams & Wilkins, 1982:153–71.
10 Frisén L. Visual acuity and visual field tests: Psychophysical versus pathophysiological objectives. In: Kennard C, Clifford Rose I, eds. *Physiological aspects of neuro-ophthalmology*. London: Chapman & Hall, 1991:3–26.
11 Bender MB, Savitsky N. Micropsia and teleopsia limited to the temporal fields of vision. *Arch Ophthalmol* 1943;**29**:904–8.
12 Mullan S, Penfield W. Illusions of comparative interpretation and emotion. *Arch Neurol Psychiat* 1959;**81**:269–84.
13 Gloning I, Gloning K, Hoff H. *Neuropsychological symptoms and syndromes in lesions of the occipital lobe and the adjacent areas*. Paris: Gauthier-Villars, 1968.
14 Teuber HL, Battersby WS, Bender MB. *Visual field defects after penetrating missile wounds of the brain*. Cambridge Harvard University Press, 1960.
15 Mooney AJ, Carey P, Ryan M, Bofin P. Parasagittal parieto-occipital meningioma with visual hallucinations. *Am J Ophthalmol* 1965;**59**:197–205.
16 Wilson SAK. Dysmetropsia and its pathogenesis. *Trans Ophthalmol Soc UK* 1916;**36**:412–44.
17 Brégeat P, Klein M, Thiébaut F, Bouniol. Hémi-macropsie homonyme droite et tumeur occipitale gauche. *Rev Oto-Neuro-Ophtalmol* 1947;**19**:238–40.
18 Thiébaut F, Matavulj N. Hémi-micropsie relative homonyme droite en quadrant inférieur. *Rev Oto-Neuro-Ophtalmol* 1949;**21**:245–6.
19 Bender MB, Teuber HL. Spatial organization of visual perception following injury to the brain. *Arch Neurol Psychiat* 1947;**58**:721–39.
20 Bender MB, Teuber HL. Spatial organization of visual perception following injury to the brain. *Arch Neurol Psychiat* 1948;**59**:39–62.
21 Ebata S, Ogawa M, Tanaka Y, Mizuno Y, Yoshida M. Apparent reduction in the size of one side of the face associated with a small retrosplenial haemorrhage. *J Neurol Neurosurg Psychiatry* 1991;**54**:68–70.
22 Damasio AR. Disorders of complex visual processing: agnosias, achromatopsia, Balint's syndrome, and related difficulties of orientation and construction. In: MM Mesulam, ed. *Principles of behavioral neurology*. Philadelphia: F A Davis, 1985:259–88.
23 Rothrock JF, Walicke P, Swenson MR, Lyden PD, Logan WR. Migrainous stroke. *Arch Neurol* 1988;**45**:63–7.
24 Carlson VR. Instructions and perceptual constancy judgments. In: Epstein W, ed. *Stability and constancy in visual perception: Mechanisms and processes*. New York: Wiley-Interscience, 1977:217–54.
25 Desimone R, Ungerleider LG. Neural mechanisms of visual processing in monkeys. In: Boller F, Graftman J, eds. *Handbook of neuropsychology, Vol. 2*. Amsterdam: Elsevier, 1989:267–99.
26 Mishkin M, Ungerleider LG, Macko KA. Object vision and spatial vision: Two cortical pathways. *Trends Neurosci* 1983;**6**:414–7.
27 Plaut DC, Farah MJ. Visual object representation: Interpreting neurophysiological data within a computational framework. *J Cogn Neurosci* 1990;**2**:320–43.
28 Hart J, Lesser RP, Gordon B. Selective interference with

representation of size in the human by direct cortical electrical stimulation. *J Cogn Neurosci* 1992;**4**:337–44.
29 Gross CG, Mishkin M. The neural basis of stimulus equivalence across retinal translation. In: Harnad S, Doty R, Jaynes J, Goldberg L, Krauthamer G, eds. *Lateralization in the nervous system*. New York: Academic Press, 1977:109–22.
30 Ungerleider LG, Ganz L, Pribram KH. Size constancy in rhesus monkeys: Effects of pulvinar, prestriate, and inferotemporal lesions. *Exp Brain Res* 1969;**27**:251–69.
31 Weiskrantz L, Saunders RC. Impairments of visual object transforms in monkeys. *Brain* 1984;**107**:1033–72.
32 Schiller PH, Lee K. The role of the primate extrastriate area V4 in vision. *Science* 1991;**251**:1251–3.
33 Damasio H, Damasio AR. *Lesion analysis in neuropsychology*. Oxford: Oxford University Press, 1989.