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Blindness to form from motion despite intact static form perception and motion detection

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Abstract

We studied the motion perception, including form and meaning generated by motion, in a hemianopic patient who also had visual perceptual impairments in her seeing hemifield as a result of a lesion in ventral extrastriate cortex. She was unable to recognise 2- or 3-dimensional forms, and even borders, generated by motion alone, failed to recognise mimed actions or the Johannson 'biological motion' display, and ceased to recognise people well-known to her when they moved. Her performance with static displays, although impaired, could not explain her inability to perceive shape or derive meaning from moving displays. Unlike a motion-blind patient, she can still see and describe the motion, with the exception of second-order motion, but not what it creates or represents.

Keywords: Motion perception; Visual agnosia; Shape from movement

1. Introduction

There have been sporadic accounts for decades of neurological patients whose cortical brain damage led to disorders of motion perception that seemed more severe than any accompanying impairments of their perception of static displays. But it was not until 1983 that Zihl et al. [33] described a patient, LM, whose disorder seemed both specific and severe and whose large bilateral brain lesion principally involved the ventrolateral region of the parietal lobes and posterolateral portion of the temporal lobes, embracing the lateral occipital gyrus and therefore the area now considered to be equivalent to the motion area (MT/V5) of the macaque monkey. Not even the demonstration that LM has some residual perception of very slow movement [8,33,34] nor the subsequent finding that she also has considerable problems with certain static displays, such as those involving the perception of form generated by texture [17] seriously threatened the view that area MT/V5 is indispensable for most if not all forms of motion perception.

Only recently has it became apparent that the cortical motion system is regionally and functionally subdivided within the extrastriate cortex, much as the extrastriate visual cortex can be subdivided into regions concerned with the analysis of wavelength, shape, orientation and so on. For example, functional neuroimaging experiments on normal subjects show that several cortical areas are activated during the performance of visual tasks involving motion [3,6,13] and that some of them may be more activated than their partners by certain kinds of visual displays [13,20]. As tellingly, neurological patients with more discrete lesions than those of LM can be more impaired at discriminating second-order motion than first-order motion [1,7,14,15,22,24] or vice versa [26]. Further, there is now evidence that the perception of biological motion and form-from-motion can be selectively preserved [21], even in a patient as densely motion-blind

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as LM [11]. Aware of these dissociations of symptoms and their implications for the functional organisation of the human visual motion system, we were particularly struck by the pattern of deficits shown by a patient (AL) who could not distinguish simple shape from motion despite seeing the motion itself with little difficulty and recognising static shapes. However, and unlike the motion blind patient LM, AL is not seriously disabled in attempting to deal with everyday moving scenes. In this paper we describe her performance on a number of tests of shape and motion perception, including biological motion.

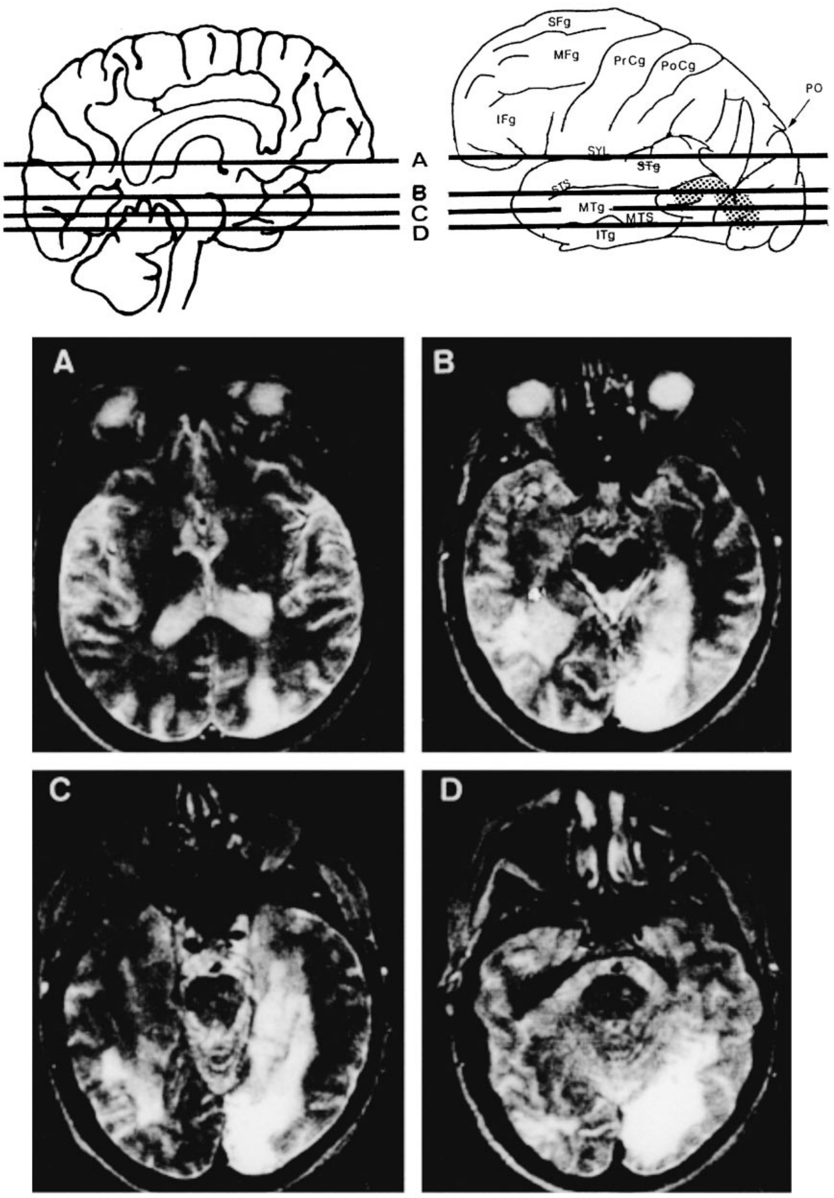

Fig. 1. A, B, C and D show four MR images through the brain of patient AL at the levels indicated on the medial (top left) and lateral (top right) views of one hemisphere. The scan was obtained one week after the large haemorrhage of her left hemisphere but three years after the much milder stroke caused by the very restricted lesion of her right hemisphere and whose consequences are the subject of this paper. The medial view, top left, is drawn from a near-midline parasaggital image but the temporal lobe, which is not visible so close to the midline, was added by hand. The lateral view is of a standard human brain at the same scale. The stippled area in the lateral view shows the projection onto the lateral surface of the largely white matter lesion in AL's right hemisphere. This small, and older lesion, is present on slices B,C and D and involves the cortex near the lateral surface only at the caudal end of the lesion (D). The later and much larger lesion, which led to a complete hemianopia and is not discussed in this paper, is present in all four images. Following radiographic convention, the right hemisphere is shown on the left, including the surface views at the top. Abbreviations: SFg, MFg, IFg, superior, middle and inferior frontal gyri respectively; STg, MTg, ITg, superior, middle and inferior temporal gyri; PrCg, PoCg, pre-and post central gyri; PO, parieto-occipital sulcus; SYL, Sylvian sulcus; STS, superior temporal sulcus; MTS, middle temporal sulcus.

2. Methods

2.1. Patient AL

Patient AL is a right-handed, retired female telephone operator. When aged 65 she suffered a large embolic left hemisphere stroke, for which she was admitted to a local rehabilitation hospital for cognitive assessment and retraining. Three years earlier she had suffered an infarction of mainly the white matter in the posterior temporal region of the right hemisphere (as revealed by computer axial tomography) and had made a good recovery. After the second episode the lesion localisation was made by MRI, and acuity, visual fields and eye movements were measured in neuro-ophthalmological consultations. AL underwent a more detailed evaluation of her perceptual abilities concerning form, colour, motion, binocular stereopsis and of her abilities to recognise objects and actions. Age-matched experimentally naive normal viewers served as control subjects. The patient and the control subjects provided written consent to testing after the procedures had been fully explained.

2.2. Lesion analysis

The lesions were demonstrated by magnetic resonance imaging (MRI). T1 and T2-weighted 6 mm thick axial images with a TR/TE of 571/16 and T2 2187/85 were obtained with a 0.3 T Fonar unit. Fig. 1 shows a large prominent hyper intense lesion in the medial inferior portion of the left temporal lobe, hippocampus and parahippocampal gyrus, probably also involving the amygdala. The lesion extends into the posterior parietal lobe and slightly into the medial temporal lobe, lateral portion of the thalamus and the posterior part of the internal capsule. Its anterior margin is just posterior to the anterior tip of the temporal horn of the lateral ventricle. Caudally the lesion extends below and medial to the temporal horn, trigone and occipital horn of the lateral ventricle, along the margin of the tentorium posterior to the interhemispheric fissure between the occipital lobes. The lesion explains why AL had a complete and permanent right-sided hemianopia, but with macular sparing, following her second stroke. The much smaller and earlier lesion can be seen in the right hemisphere in the posterior occipital and temporal regions lateral to, and below, the level of the occipital horn of the lateral ventricle. Although mostly a white matter lesion it involves parts of the fusiform and lingual gyri and extends to the cortex on the lateroventral aspect of the temporal lobe. It is presumably this lesion that is the chief cause of the perceptual disorders present after the second stroke and not noticed after the first stroke because of her normal vision in both hemifields at that time. Regrettably, patient AL was not subsequently available for a higher resolution structural MR scan that might have revealed whether her lesion involved particular occipito-temporal regions such as areas MT, MST, or the kinetic occipital region of Orban et al. [13].

2.3. Neuro-ophthalmological examination

The neuro-ophthalmological examination revealed no abnormality in either eye. Pupils were normal. Visual fields on Goldmann perimetry demonstrated loss of the right visual hemifield on both eyes, but with macular sparing. The left visual field was normal for her age. Acuity was 20/30 in both eyes without correction. Her ocular motility was normal, both for smooth pursuit and saccades to targets in the left visual field, although quantitative measures might have revealed deficiencies in pursuit gain or response latency. She saccaded in her right visual field, but due to her field loss the saccades to targets were grossly inaccurate. Her vestibular ocular response was normal.

2.4. General behaviour

The initial small right hemisphere infarction produced no discernible long term functional impairment; AL continued to drive a car, was independent in her home making activities, and led a fulfilling family and social life. She had no paresis, ataxia, or akinesia. On pin examination, she had a slight decrease in pin sensation on the left side. There was a moderate impairment of position sensation on her left side.

After the second stroke AL initially had a complete right hemianopia, prosopagnosia and visual object agnosia. During the first four weeks she was unable to recognise known people by their faces, reliably name or identify objects, or match or name colours. Initially her prosopagnosia was severe, and she was upset by her inability to recognise even close members of her family, except by their voice. She had no alexia or agraphia.

About two months after the second stroke AL regained reliable visual recognition of known people, and good colour matching on informal testing with coloured paper patches. However, she complained that when people moved she could no longer recognise them. For example, she could not recognise the faces of her own children and grandchildren as soon as they moved, even when they remained in her intact left hemifield. Nor could she recognise them by their gait. This paradoxical ability to recognise familiar people when they were still but not when they moved prompted our detailed examination of her perceptual abilities, especially visual motion perception. We also evaluated her object recognition and her ability to discriminate form and colour.

2.5. Neuropsychological evaluation

AL was tested formally four months after the second stroke. With subtests of the Wechsler Adult Intelligence Scale (WAIS-R), her verbal performance was average for her age and education (VIQ = 98), but she was severely impaired on the performance scale (PIQ = 68). She was unable to perform, or conceptualise how to achieve, 3D constructions (copying block design, putting together 3D puzzles) and copied simple 2D lines and contours laboriously. Her drawing from memory was similarly impaired (cube, square, house). Reading, writing and calculation were normal. Her verbal reasoning and attention were excellent.

3. Tests involving discrimination of static visual stimuli

3.1. Visual naming

Forty-two coloured pictures of common objects, animals, fruits and vegetables (cut from children's books) were shown one at a time to AL. Each picture was presented on a separate sheet of paper and AL was asked to provide the name or, if unable to name it, to provide any information helpful for identifying the picture. She was allowed as much time as she wished. Her performance on this task was revealing: She promptly recognised all the inanimate objects, even when the structure (e.g., roller-skate) or colour distribution (mittens with a complicated colour pattern) were complex. She was equally good at recognising fruits and vegetables. But she failed to identify any of the 11 pictures of animals. When shown the picture of an elephant she said 'animal, big' , but was unable either to name it or provide any information suggesting that she recognised it. She identified as animal all pictures of four-legged animals (e.g., dog, horse, pig, lion), but could not be more specific. When pressed to say why she thought it was an animal she said 'because it has legs'. The picture of a rabbit (without visible legs) she identified as 'vegetable' and that of a monkey as 'bald head man' and she could not identify a picture of a fish. These symptoms resemble those of patients with category-specific visual agnosia [30,31].

3.2. Matching silhouettes to coloured pictures

The test consisted of 12 coloured and textured pictures, six of inanimate objects and six of four-legged animals, and their matching silhouettes. AL was asked first to pick out the photograph that correctly matched a silhouette of an animate (kangaroo, giraffe, dog, cow, horse, pig) or an inanimate (roller-skates, truck, doll, train, car, boot) object, and then to name or describe the objects. The photographs of the objects, and therefore of the silhouettes, were all from a prototypical, front or side view. All 12 silhouettes were shown on one card and all 12 corresponding coloured photographs in a different arrangement on another card below it. AL promptly recognised 5/6 object silhouettes and then matched them correctly to the corresponding objects. However, she failed to recognise any of the animal silhouettes, categorised only one of them (a dog) as an animal, and failed to match any of them to their corresponding pictures.

3.3. Hue discrimination

Hue discrimination, as opposed to naming hues, was evaluated with the Farnsworth–Munsell 100-Hue Test. All 88 hues were presented, in four groups of 22. Each group of 22 was presented in random but predetermined order in a single row and AL was asked to rearrange them into an orderly progression of hues along the row between the predetermined anchor hues at each end. She also performed the task with the standard 22 grey chips, ranging in reflectance from low to high and of roughly equal difficulty to the hues for normal observers.

Fig. 2 shows that LA was moderately impaired at ordering hues but only in two regions of the colour circle, indicating that her impairment does not involve ordering *per se*. She was impaired in the orange–red region and even more so in the blue region. In contrast she made no errors in ordering the sequence of greys.

3.4. Shape discrimination

The stimuli and procedure were based on those devised by Efron [4]. AL fixated a small fixation mark 1° to the right of the stimulus which was therefore displayed always in her intact left hemifield. The target was a black square, $5^\circ \times 5^\circ$, and the distractors were oblongs of almost the same total area with the following dimensions: $5.25^\circ \times 4.77^\circ$, $4.6^\circ \times 5.5^\circ$, and $6.5^\circ \times 4^\circ$ (Fig. 3A). For each of these dimensions 10 trials were presented, each with a single stimulus (target or distractor) for 0.5 s. In a yes/no task AL was asked to report whether the shape was the square. Her overall score was 39/40, like that normal observers and she clearly did not suffer from apperceptive agnosia for simple shape, of the kind described by Efron [4] and by Warrington and James [32] and called pseudo-agnosia by the latter.

3.5. Shape detection

Warrington and Taylor [29], demonstrated that patients with apperceptive visual agnosia are notably impaired at detecting a simple fragmented figure superimposed on a static noisy background even though they can discriminate the full, not fragmented figure (Fig. 3B, top). AL was asked to detect the presence or absence of the stimulus (in the first 10 trials the stimulus was an X and in the second 10 an O). Her performance (Fig. 3B, bottom) was normal.

3.6. Global stereopsis

Binocular Stereopsis was measured with the Randot Stereotest (Stereo Optical Co., Inc.), which measures global stereoacuity with a series of stereograms of different disparities. The random dot stereograms are presented on polarised cards. With appropriate polarised glasses each eye views a different image. In 75% of the stereograms, a central figure (the letter E, or a disc, star, square, triangle, or circle) stands out in front of the surround if the viewer has normal stereopsis. In the other displays there was no disparity and hence no figure.

AL could see appropriately that something was present in the stereograms, in front of the surround, and could indicate its outline but not identify its shape. She correctly identified those displays where no shape was present. Her problem in recognising the shape in stereograms was therefore not caused by an inability to fuse the two disparate displays.

3.7. Position discrimination

We assessed AL's perception of the relative positions of objects in 2-D space using two tasks adapted from Warrington and Taylor [29]. If AL's performance on these tasks were impaired, this might explain some of her problems in recognising moving figures. In the first task, the display consisted of two squares positioned horizontally side-by-side and each subtending $6 \times 6°$, one with a small black dot presented exactly in the centre and the other with the black dot 'off centre'. In each trial the off-centre dot was in a different position within the square and its offset ranged between 0.5 to $1.0°$. In a two-alternative discrimination task, AL and the normal control subjects indicated which of the two squares had the dot in the centre (Fig. 3C, top). There were 40 trials in this test. AL scored 92.5% correct, which is within the range of the scores of the control subjects, whose mean percentage correct was 96.

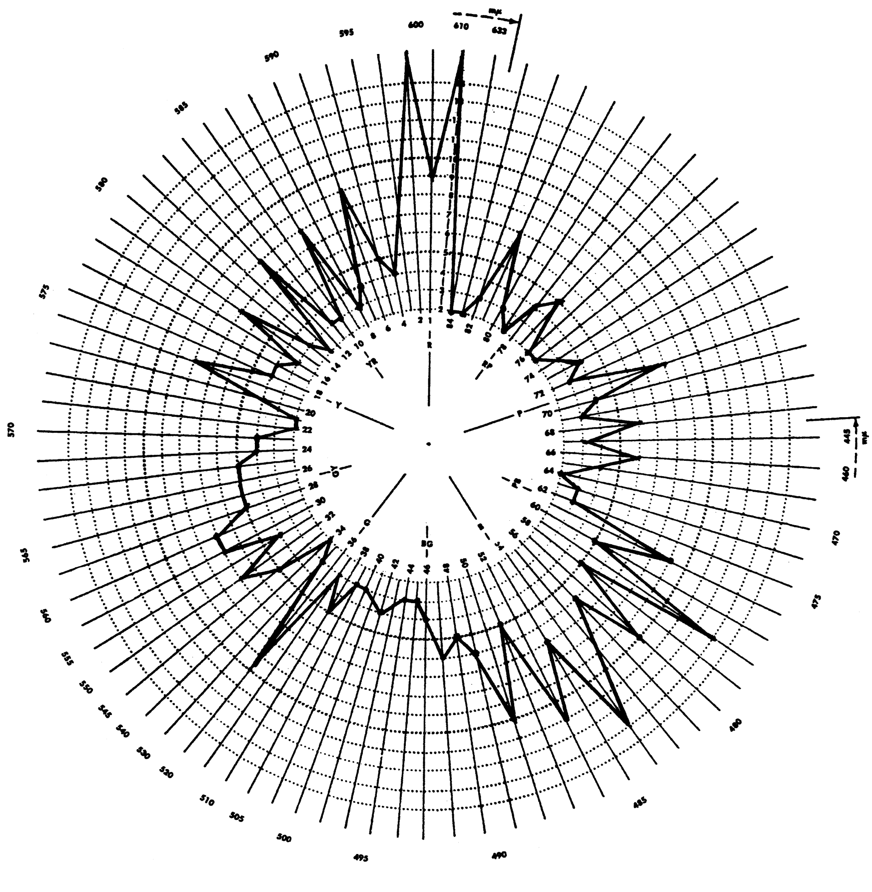

Fig. 2. Results of the Farnsworth-Munsell 100 Hue Test. Errors are plotted radially and positions on the circumference represent points along a continuum of hue. AL was moderately impaired, especially in the red (600–610 nm) and the blue (476–488 nm) range.

In the second task (Fig. 3D) we investigated AL's spatial perception with a number location test. The display consisted of two squares, each subtending 8 × 8°, placed one above the other. The top square contained randomly placed numbers (1–9) and the bottom square a single black dot corresponding to the position of one of the numbers. The position of the dot varied from trial to trial. In 30 trials AL and 15 normal control subjects were asked to identify the number corresponding to the position of the dot. AL's score of 80% correct responses was within the normal range.

4. Tests involving visual motion

We conducted several tests to determine the extent to which AL could use visual motion information to discriminate 2-D or 3-D forms. Experiment 1 addressed discrimination of simple forms on the basis of motion or flicker information, Experiment 2, shape discrimination based on speed difference, and Experiment 3, the perception of 3D structure from motion cues. The stimuli for Experiments 1–2 are described in detail by Vaina et al. [26] and those for Experiment 3 by Vaina et al. [21]. Bearing in mind AL's problem in recognising familiar people when they moved, Experiment 4, using the 'biological motion' demonstration from the original Johansson's movie [9], assessed her ability to perceive different patterns of biological motion generated from the trajectories of moving points. Finally, in Experiment 5, we examined AL's ability to recognise pantomimed actions on a video film and her ability to infer the appropriate actions from static drawings that portrayed objects before and

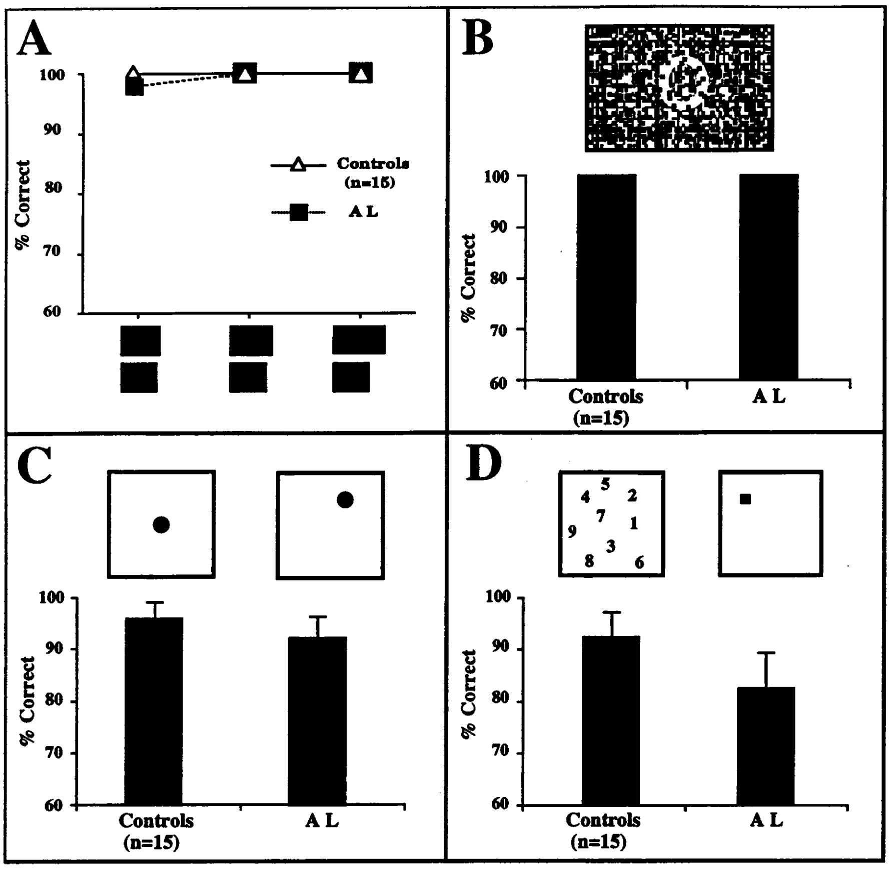

Fig. 3. A. Shape Discrimination Task. Percent correct performance of five normal controls and AL on shape discrimination for three ratios plotted as decreasing difficulty. Examples of the three shape ratios used are shown at the bottom. B. Shape Detection Task. The figure (top) shows an example of the fragmented shape (O) superimposed on a fragmented background. At the bottom, the x-axis show the results from seven age matched normal controls and AL. The y-axis portrays percent correct responses. C. Position Discrimination Task. The stimulus, shown at the top, consists of two adjacent horizontal squares, one with a black dot (5 mm) printed exactly in the centre and the other with a black dot just 'off' centre. Results, presented as percent correct from 15 age matched control subjects and AL are shown on the bottom. D. Spatial Localisation Task. The target is the black square in the right box. The observer's task is to choose the corresponding spatial location from among the numbers and letters in the left box. (In the actual test the two squares were arranged vertically one under the other.) The results show the percentage of correct responses of AL and 15 age matched normal control.

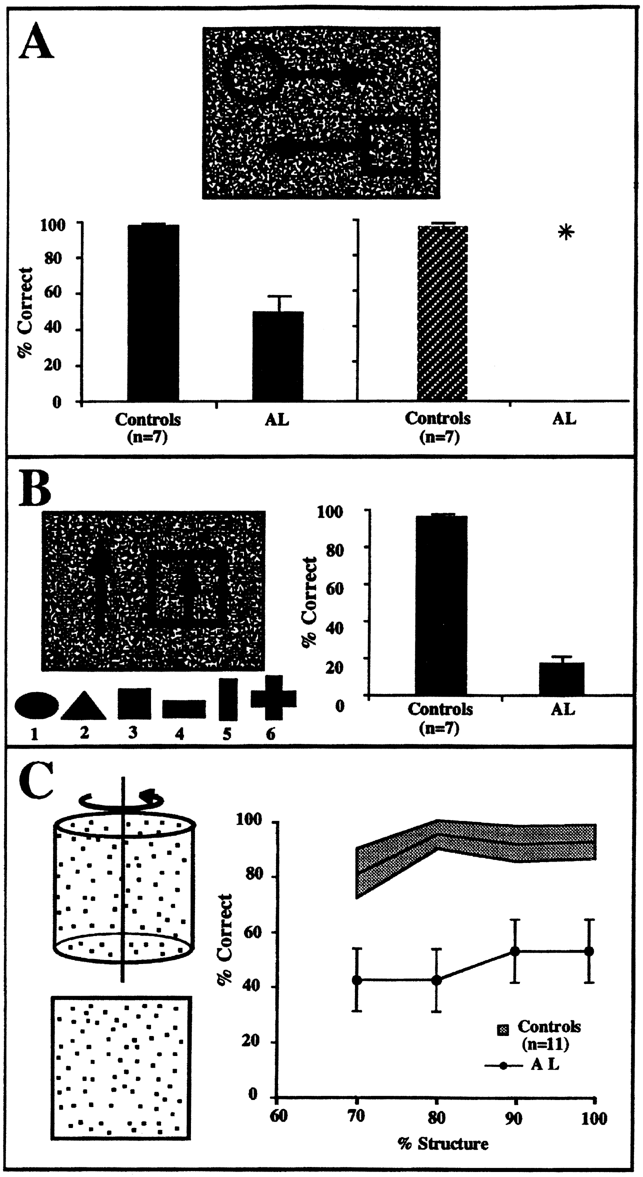

Fig. 4. A. 2-D Form From Motion in a Static Background Task. A schematic illustration of the display is shown on the left. The display consisted of a fine grained random dot pattern resulting from 50% black and 50% white dots. Two patches of contiguous dots were coherently translated across the static background at equal speeds. The observer had to say whether the two shapes were the same or different. There were two conditions in the test: (1) the outline of the shapes was defined by local difference in luminance from frame to frame; (2) the outline of the shapes was defined by twinkling borders, without any consistent difference in luminance. The graphs at the bottom show mean percent correct responses for seven control subjects and percentage correct responses for AL. On the left are the results from condition 1, and on the right the results from condition 2. The star symbol indicates that AL was blind to the form defined by twinkling borders. Range bar indicates standard deviation of the mean. B. 2-D Form from Differences of Velocity Task. On the left is a schematic illustration of the display. The background of random dots scrolls up or down and a central patch of contiguous dots, subtending $2^\circ \times 2^\circ$, moves in the same direction as the background but at a different speed. The shape of the central patch matches, at random, one of the six shapes illustrated beneath the display. On the right the bar graphs show mean percentage correct responses of the seven control subjects and the score for AL. Range bar shows standard deviation. C. 3-D Structure from Motion. Task. On the left is shown a schematic representation of the two dynamic random dot fields employed. On the top is shown the structured stimulus generated by the orthographic projection of a square shaped random dot velocity field onto a transparent cylinder which is rotated. On the bottom the velocity field represents the unstructured stimulus. The percentage of structure in the structured stimulus varied in 10% increments from 70–100. The graphs on the right show the performance of patient AL and that of 11 normal controls subjects. The shaded area represents the results from the normal controls, ± 1 SD.

after an action and her ability to select the possible implements for the inferred actions.

4.1. General methods and procedures

The stimulus displays, except in Experiments 4 and 5, were generated and presented, and responses collected and analysed, using a Macintosh IIcx computer with an extended 8-bit video card. The display appeared in the centre of a standard 13 inch Macintosh colour monitor with a resolution of 640 by 480-pixels, vertical retrace interrupt of 66.7 Hz, and P4 white phosphor. Random dots were used both to minimise familiar position cues and to isolate motion mechanisms [12]. In all experiments each pixel subtended 1.8 × 1.8 arc min at the viewing distance of 65 cm. The background in the display was black and the random dots were white. Where dense random-dot patterns were used, each pixel in the raster had a 50% probability of being light or dark. Presentation time was 2 s for each trial but the subject had unlimited time to respond. Subjects fixated a small mark near the centre of the display so that the entire display was clearly seen. As AL had macular sparing in her hemianopic field defect, she too could see the entire display.

All subjects were first familiarised with the tasks through examples and immediate feedback about performance. Experimental sessions followed, without providing any information about correctness of responses. The subject started each trial by pressing a response key. The room illumination was maintained at a low photopic level, and the observer was asked always to look at the centre of the screen.

The control group consisted of normal volunteers with no known ophthalmological, neurological or psychiatric disorders and of the same age group as AL. Several were spouses of other patients who have been tested in the laboratory and were therefore keen to take part in and understand the tests. None had previously participated in a psychophysical experiment. All subjects had corrected-to-normal vision and gave informed consent for the tests to be carried out.

4.2. Experiment 1. 2-D form from motion in a static background

The sensation of two simple 2-D moving forms was elicited by uniformly displacing two patches of random dots from one frame to the next in translational motion across a stationary random-dot display (Fig. 4A, top). The moving patches had one of the following shapes: square, circle, triangle, cross, or rectangle (oriented horizontally or vertically). The square, circle, and cross had roughly the same area, and the area of the rectangle was half that of the square. The entire display subtended $10^\circ \times 10^\circ$ and the two moving forms, each covering approximately $2.2^\circ \times 2.2^\circ$ of visual angle, moved at 3°/s. Only the dots delineating the outline of the forms were horizontally displaced from one frame to the next, creating the vivid impression of two forms moving smoothly in opposite directions across the static random dot background. Forty trials were given and in a yes-no task subjects were asked whether the two moving shapes were the same.

There were two conditions in this experiment: (1) the perception of the two moving forms resulted from the displacement of the shape borders by a constant horizontal shift between consecutive frames; (2) to eliminate local brightness cues in the translating borders, the luminance of these dots was randomly assigned to black or white from frame to frame, this provided the impression of two forms, defined by twinkling borders, moving in opposite directions across the screen.

4.2.1. Results

Fig. 4A, bottom, shows the performance on the 2D form-from-motion task of AL and of seven normal observers. The control subjects performed almost without error on discriminating form from simple translation, whereas AL was no better than expected from random responding (Fig. 4A-bottom left). Although she was able to detect the movement she reported not seeing any figures. In the second task, where the outlines were defined by moving twinkling borders (an example of second-order motion, see Discussion), the control subjects found the task easy (Fig. 4B-bottom right) but AL failed even to detect the movement. To determine whether AL's deficit on these tasks might be explained by poor acuity, she was asked to discriminate static contours of the same shapes as described above. She scored 100% correct.

4.3. Experiment 2. 2-D form from differences in speed

An example of the display is shown in Fig. 4B-left. The shape subtended roughly $2^\circ \times 2^\circ$ and was centrally positioned ($\pm 1^\circ$) within a $6^\circ \times 3.75^\circ$ rectangular aperture of a dense random dot pattern. The shapes were generated solely by differences in the velocity of a contiguous patch of dots (defining the shape) and the velocity of the random dot background. To identify the shapes the subject must therefore be able to detect that difference. Both the figure and the background were displaced in the same direction but at different velocities. The background dots moved at 3°/s. The velocity ratios between figure and background, with reference to the velocity of the background, were: 3/2, 2, 3, 2/3, and 1/2. Observers had to make a forced-choice judgement by indicating which of the six figures displayed at the bottom of the screen matched the moving shape. There were 60 trials and each shape was presented 10 times. Guessing would therefore yield 17% correct responses.

In case AL might fail this task because she was unable to perceive the differences in speed that generated the contours, we also measured her discrimination of relative speed of two simultaneously presented dynamic random-dot cinematograms. The display consisted of two such cinematograms shown in two elongated apertures each subtending 5° by 10°, one above the other near the centre of the screen. The distance between their centres was 6°. Each aperture contained 50 computer-generated dots. The distance a dot was displaced between successive frames, was uniform within an aperture and was assigned independently for each aperture. The speed of the dots in one aperture was always 3°/s. The initial speed of the dots in the other aperture — the test speed — was 6°/s and was varied by an adaptive staircase procedure. In any single trial each dot took a 2-D random-walk of constant step size defined by the speed. The direction in which any dot moved was randomly extracted from a 360 range and was independent of its previous direction and also the displacements of the other dots. The resolution of the Apple monitor constrained the direction sampling to every 45°. On half of the trials, at random, the test speed occupied the top half of the display. All the dots within an aperture moved with the same speed. The method of constant stimuli was used with speed ratios: 1.5, 2 and 3. Subjects had to indicate in which of the two apertures the dots appeared to move faster.

4.3.1. Results

The control subjects found the form discrimination task easy at all ratios (Fig. 4B, right), but AL scored no better than expected from random responding. She also complained that she could not even see the outline of the shape generated by motion, although she had no trouble in discriminating the shapes of the static matches beneath, and she was aware that things moved in the display. However, on the speed discrimination control task AL's performance was within the normal range, showing that her failure to perceive form from motion did not stem from any lower-level problem in detecting relative speed between adjacent regions.

4.4. Experiment 3. 3-D structure from motion

The human visual system can recover the 3-D structure of rotating objects from the changing 2-D retinal image. Even an object defined by a sparse pattern of dots on its imaginary surface suffices. Experiment 3 examined the ability of patient AL to perceive a rotating cylinder generated in this way. The stimulus, described before in detail [21] consisted of the parallel projection of points covering the transparent surface of a rotating cylinder. Two dynamic random dot cinematograms were presented simultaneously one below the other. One portrayed a vertical transparent revolving cylinder, the other a pattern of scrambled velocities (Fig. 4C, bottom) generated by spatially shuffling the velocity vectors present in the structured display (thereby destroying the local spatial relationship between velocity vectors.) Each cinematogram subtended $3 \times 3°$ and was composed of 128 dots with an average density of 14 dots/° and finite point life time fixed at 400 ms. At the end of its lifetime a dot disappeared and was replotted at a new random location within the display and it began a new trajectory. The angular velocity of the cylinder was 30°/s. Both the structured and the unstructured displays were generated by 50 frames, each displayed for 33 ms, which were repeated for the total duration of the display. The maximum distance travelled by a dot between two consecutive frames was 4.3 arc min.

Using the method of constant stimuli and a two-alternative forced-choice paradigm, the following percentage of structure were presented: 100, 90, 80, 70. The spatial positions (top vs bottom) of the structured and the unstructured fields were randomly assigned. There were 80 trials equally distributed among the four percentages of structure in the cylinder. AL and control subjects were asked to choose which of the two displays, top or bottom, appeared more like a rotating cylinder.

4.4.1. Results

Fig. 4C right shows that the control subjects performed at better than 90% correct in discriminating between the apparent rotating cylinder and the spatially unstructured field at every level of structure in the cylinder. On the other hand AL scored at chance level even for 100% structure. When asked what she saw, AL said 'moving dots' but could not perceive any difference between the two sets. She appeared to have lost all means of extracting 3D revolving structure from its corresponding correlated motion. We repeated the test at 100% structure contrasted to the unstructured display using sequential apertures. AL's performance remained at chance in this condition as well.

4.5. Experiment 4. Recognising biological motion

The term 'biological motion' was introduced by Johansson [9] to describe the percept of a moving figure generated by the pattern of luminous dots placed at the joints of a human subject who traversed a stage in darkness. By placing the light sources at the points of articulation, Johansson found that the motion of the dots alone enabled observers to perceive the activity (walking, bicycling etc) of the actor (Fig. 5A). Such patterns of biological motion have since been shown to be sufficient for the perception of specific actions, of gender [10] and of sign language [16], all computed by the observer from a pattern of dots whose organisation changes over space and time.

Scenes from a videotape of the original film by Johansson were used with patient AL and the control subjects. The following common actions were used: walking, climbing stairs, riding a bicycle, push-up, two men walking, shaking hands and hugging. When static, they gave no indication of the action from which they were taken. The subject was told beforehand that he would see a collection of dots moving in a certain direction and that he should first report the direction of motion and then say what it looked like.

All the control subjects almost instantly recognised that the patterns of moving luminous dots came from human subjects carrying out the actions listed above. In contrast, patient AL found the displays confusing and, even when told that they represented a person performing some action, she completely failed to see a person performing common actions. She perceived the movement of the dots and correctly identified their overall direction. She also reported that not all the dots moved in the same direction, but she experienced nothing that was more than the sum of the parts.

4.6. Experiment 5. Recognition of pantomimed actions

A videotape of 10 mimed actions (from the Pantomime Recognition Test described in Vaina et al. [23]) was presented on a large black and white TV monitor. All the pantomimes were presented as silhouettes, to eliminate any clues about those qualities of the movement which might be inferred from facial expressions, for example see Fig. 5B. Each pantomime was presented twice, in immediate sequence. There were two classes of mimed actions. In the first class (five items) were actions which do not have a unique functional relation with the object being used: throwing, pulling, plucking, breaking, and setting something down. The action is identified on the basis of specific features of the movement, such as its direction and speed, the position, shape and size of the opening of the hand or fin-

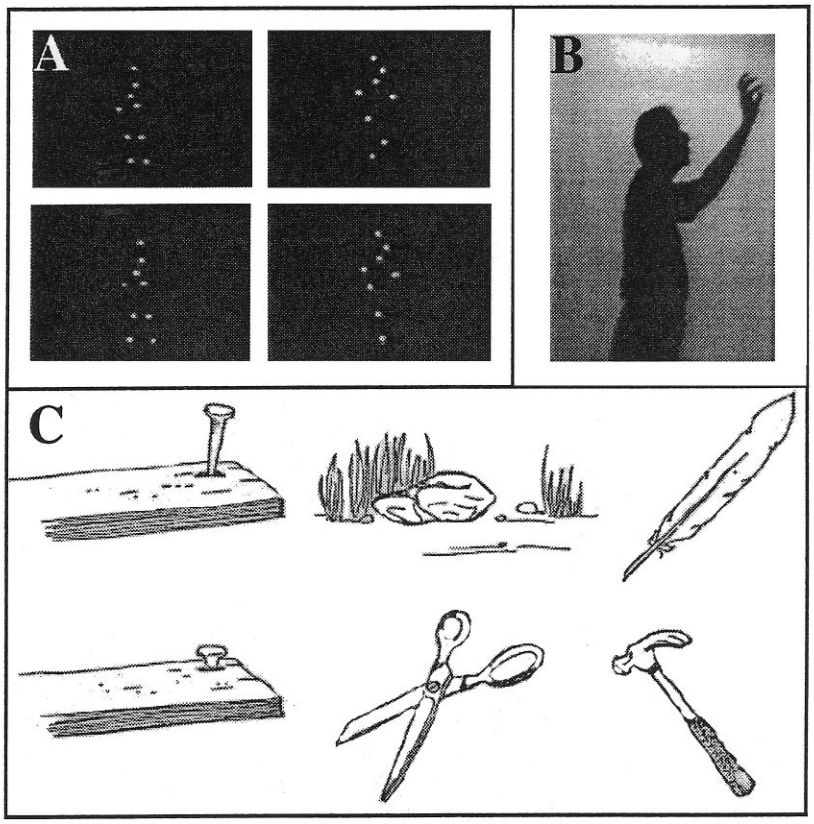

Fig. 5. A. Biological Motion Task. The figure illustrates a single frame from the videotape portraying a man walking (left top and bottom) and a man riding a bicycle. B. Recognition of pantomimed actions. The figure shows a single frame from the videotape presenting a silhouette of a man miming simple actions. C. Action Comprehension Task. The figure shows an example of the stimulus cards. On the left (top and bottom) is portrayed a nail, first barely entering the board and then driven through the board. Subjects had to identify which of the four objects presented could be used accomplish the change.

gers, and the location of the action with respect to the body. In the second category (five items) the action mimed the primary function for which an implement was designed, namely sawing, combing, phone-dialling, playing tennis, and shovelling. AL and two control subjects were asked to name the actions, or provide any information that would reveal knowledge of the action mimed.

The display was arranged so that AL could see the entire mimed action in her intact left hemifield. She was so impaired on this task, in spite of demonstrating that she saw the stimulus, that she could not identify any of the mimed actions in the first class. She reported, 'a man doing something, I don't know what he is doing, something with the hands'. In the second class of actions, she correctly identified two actions, namely 'combing' and 'phone-dialling'. She described shovelling as 'the man is pulling something towards him, and then away', and playing tennis as 'he is moving his arm'. The two age-matched controls correctly identified all the actions.

In order to determine whether Al's inability to recognise mimed actions was specific to actions involving motion, we presented a test of recognition of static portrayals of actions (Fig. 5C). The test consisted of 10 cards. Each card showed a before-and-after view of a completed action and, in the lower half of the card, four objects, of which one was the implement normally used for the portrayed action and one was an object that could be used as an improvised substitute for that implement. One of the remaining two was a semantically related but inappropriate object and one was unrelated. The target and the other objects were randomly distributed in four positions on the card. Subjects were first shown, by pointing, the difference between the views 'before' and 'after' the action (e.g., 'a nail barely inserted in a plank' and 'a nail fully driven into the plank'). Then they were asked to first choose the object most commonly used to accomplish the action (hammer), and second, another object that could also be used (rock).

AL correctly identified all the appropriate implements and all the possible substitutes, just like the control subjects.

5. Discussion

Patient AL complained that she was unable to recognise faces and people or objects while they were moving, although she could see how they moved and had little or no difficulty in recognising them when they were static. She and her family commented that she had no problem navigating in her surroundings, or seeing that something was approaching her and correctly appreciating the direction from which it was coming. It was this intriguing inability to recognise from motion even the most familiar things and people that prompted us to formally study her perceptual and visual cognitive abilities with respect to a broad range of aspects of form perception.

AL demonstrated normal performance on tasks of static 2D form and object matching discrimination and recognition, with two exceptions: matching and recognition of pictures of animals and the perception of 2-D form from global stereopsis. Unfortunately, we had no opportunity to assess her performance on tests of local stereopsis. In contrast, on all the visual motion tasks she performed at chance levels. In Experiment 1 she failed to recognise 2D-shapes defined by motion, although she correctly reported that there were two moving shapes in the display and identified their directions. In the same experiment, when the shapes were defined by twinkling borders she reported seeing nothing in the display. The main difference between the two conditions is that in the first, there was a consistent correspondence from frame to frame between the luminance of each dot, while in the second condition the twinkling borders removed this correspondence. The twinkling motion is detectable only by a second-order (non-fourier) mechanism. We and others have previously shown that the perception of second-order motion can be selectively impaired by cortical lesions [14,15,24] and here AL's performance indicates that lesions can even cause blindness to second-order motion or at least to one form of it. However, her performance on first-order (luminance contrast) displays was not examined psychophysically and we cannot be certain that her perception of first-order motion *per se* was unimpaired.

AL's performance was at chance on tasks of discrimination of 2D forms defined on the basis of speed difference (Experiment 2), or 3D form when solely motion cues were available (Experiment 3). Although AL could not utilise motion cues to extract sufficient information to recognise forms, she had no difficulties in recognising forms on the basis of luminance cues alone (Shape Detection and Shape Discrimination). Furthermore she was unable to use the pattern of motion for recognition, although she perceived motion and correctly commented on its direction. This is exemplified by her performance when presented either with motion stimuli defining a human figure performing simple actions (Experiment 4) or with mimed actions (Experiment 5). Her inability to perceive the Johannson illusion (Experiment 4) is especially informative because other patients with complete motion blindness [11] or severe impairment in discriminating form from motion [18], readily perceive the Johannson illusion. Interestingly, the two patients described by Schenk and Zihl [18] had dorsolateral parietal damage, much more dorsal than the damage in patient AL, and even the much more extensive lesion in the motion blind patient LM [11] barely overlaps with that of patient AL. AL's lesion is probably better described as involving chiefly the so-called ventral visual processing stream.

Although it had been suggested that the interpretation of mime is correlated with different aspects of language comprehension [2,5,19,27,28] the study by Vaina et al. [23] suggests that it does not make symbolic or verbal demands, but it clearly makes demands on mechanisms of visually based inference. The actions mimed are direct pictorial representations, not codified as symbols by any conventional system. Interpreting mime should not be confused with the interpretation of codified gestural symbols such as those of sign language, or of conventionalised, non iconic gestures. AL could recognise and herself mime conventionalised gestures such as beckon, salute, stop the traffic, wave good-bye, or blow a kiss. Her impairment was specific to recognising actions miming the use of implements. However, she had a perfect score on the Action Inference test, which required her first to identify the action performed on an object presented before and after the action, and then to choose the objects that have the appropriate attributes to enable her to carry out the action. This indicates that the semantic concept of actions was preserved; her deficits were specific to an inability to use visual motion information for recognition. Within this framework one could argue that she recognised conventional gestures because they can be well represented as static pictures, not unlike the concept of action in the Action Inference test. Her failure to recognise picture of animals may indicate that the pattern of movement of animals constitutes an intrinsic part of the concept of an animal. Although time did not allow us to carry out a more detailed formal analysis of visual motion perception capabilities of AL, the data we obtained strongly suggest that in contrast to motion-blind patient LM, whose motion deficits exemplify akinetopsia, where motion itself is not perceived, AL's deficits exemplify visual motion agnosia, where the motion itself is seen but what it normally generates is not.

It is instructive to compare AL's lesion with those of other patients who have conspicuous disorders of some aspect of motion perception. The best known of these is patient LM, who is often referred to as being motion blind. Her bilateral lesion certainly involves area MT/V5 on both sides, but many other extra-striate visual areas as well, particularly more dorsal to MT. However, LM does perceive the Johansson illusion [11] and is therefore less impaired than AL in this respect. The most plausible explanation is that AL has extensive damage to rostral ventral temporal cortex of her left hemisphere and that this, together with the white matter lesion in her right hemisphere, has disrupted even the remaining temporal cortex on the right. If cortex of the rostral and ventral temporal lobe is important for the processing of information about biological motion [25] AL's poor performance with the Johannson 'biological motion' display is not surprising. In further respects AL is different from LM. Whereas the latter does not experience the raw sensation of visual motion — with the exception of very slow motion — and must deal with a changing scene by responding to changes in the position of objects, AL does experience the qualia of motion. As a result she is much less visually disabled. Whether this is because her lesion involves mostly white matter and several of her extra-striate visual areas presumably continue to process motion information remains unclear. Perhaps AL lacks the functional connections between mechanisms that analyse motion and those in the temporal lobe that use the information to generate form from motion.

References

[1] Braun D, Petersen D, Schonle P, Fahle M. Deficits and recovery of first- and second-order motion perception in patients with unilateral cortical lesions. European Journal of Neuroscience 1998;10:2117–28.

[2] Duffy RJ, Duffy JR. Three studies of deficits in pantomimic expression and pantomimic recognition in aphasia. Journal of Speech and Hearing Research 1981;24:70–84.

[3] Dupont P, Orban GA, De Bruyn B, Verbruggen A, Mortelmans L. Many areas in the human brain respond to visual motion. Journal of Neurophysiology 1994;7:1420–4.

[4] Efron R. What is perception? In: Cohen RS, Wartofsky MW, editors. Boston studies in the philosophy of science, vol. 4. Dordrecht: Reidel D, 1968. p. 137–73.

[5] Gainotti M, Lemmo MA. Comprehension of symbolic gestures in aphasia. Brain and Language 1976;3:451–60.

[6] Gulyas B, Heywood CA, Popplewell DA, Roland P, Cowey A. Visual form discrimination from color or motion cues: functional anatomy by positron emission tomography. Proceedings of the National Academy of Science 1994;91:9965–9.

[7] Greenlee MW, Smith AT. Detection and discrimination of first- and second-order motion in patients with unilateral brain damage. Journal of Neuroscience 1997;17:804–18.

[8] Hess F, Baker CL, Zihl J. The motion-blind patient: low level spatial and temporal filters. Journal of Neuroscience 1989;9:1628–40.

[9] Johansson G. Visual perception of biological motion and a model for its analysis. Perception and Psychophysics 1973;14:201–11.

[10] Kozlowski LT, Cutting JE. Recognizing the sex of a walker from a dynamic point-light display. Perception and Psychophysics 1977;21:571–80.

[11] McLeod P, Dittrich W, Driver J, Perrett D, Zihl J. Preserved and impaired detection of structure from motion by a 'motion-blind' patient. Visual Cognition 1996;3(4):363–91.

[12] Nakayama K, Tyler CW. Psychophysical isolation of movement sensitivity by removal of familiar position cues. Vision Research 1981;21:427–33.

[13] Orban GA, Dupont P, De Bruyn B, Vogels R, Vandenberghe R, Mortelmans L. A motion area in human visual cortex.

Proceeding of the National Academy of Sciences USA 1995;92:993–7.

[14] Plant T, Laxer KD, Barbaro NM, Schiffman JS, Nakayama K. Impaired visual motion perception in the contralateral hemifield following unilateral posterior cerebral lesions. Brain 1993;116:1303–35.

[15] Plant GT, Nakayama K. The characteristics of residual motion perception in the hemifield contralateral to lateral occipital lesions in humans. Brain 1993;116:1337–53.

[16] Poizner H, Bellugi U, Lutes-Driscol V. Perception of American sign language in dynamic point-light displays. Journal of Experimental Psychology: Human Perception and Performance 1981;7:430–40.

[17] Rizzo M, Nawrot M, Zihl J. Motion and shape perception in cerebral akinetopsia. Brain 1995;118:1105–27.

[18] Schenk T, Zihl J. Visual motion perception after brain damage: II. Deficits in form-from-motion perception. Neuropsychologia 1997;35:1299–310.

[19] Seron X, van der Remitz A, van der Linden M. Pantomime interpretation in aphasia. Neuropsychologia 1979;17:661–8.

[20] Smith AT, Greenlee MW, Singh KD, Kraemer FM, Hennig J. The processing of first- and second-order motion in human visual cortex assessed by functional magnetic resonance imaging (fMRI). Journal of Neuroscience 1998;18:3816–30.

[21] Vaina LM, LeMay M, Bienfang DC, Choi AY, Nakayama K. Intact 'biological motion' and 'structure from motion' perception in a patient with impaired motion mechanisms. Visual Neuroscience 1990;5:353–71.

[22] Vaina LM, LeMay M, Gryzwacz NM. Deficits of non-fourier motion perception in a patient with normal performance on short-range motion tasks. Society of Neuroscience Abstracts 1993;19:1284.

[23] Vaina LM, Goodglass H, Daltroy H. Inference of object use from pantomimed actions by aphasics and patients with right hemisphere lesions. Synthese 1995;104:43–57.

[24] Vaina LM, Cowey A. Impairment of the perception of second order motion but not first order motion in a patient with unilateral focal brain damage. Proceedings of the Royal Society London B 1996;263:1225–32.

[25] Vaina LM, Belliveau JW, Burin des Roziers E, Zeffiro Z. The relationship between cortical activation and psychophysical performance in humans during fast learning of motion discrimination. Neuroimage 1997;5:S137.

[26] Vaina LM, Makris N, Kennedy D, Cowey A. The selective impairment of the perception of first-order motion by unilateral cortical brain damage. Visual Neuroscience 1998;15:333–48.

[27] Varney NR. Linguistic correlates of pantomime recognition in aphasic patients. Journal of Neurology, Neurosurgery, and Psychiatry 1978;43:71–5.

[28] Varney NR. Pantomime recognition defect in aphasia: implications for the concept of asymbolia. Brain and Language 1982;15:23–39.

[29] Warrington EK, Taylor AM. Contribution of the right parietal lobe to object recognition. Cortex 1973;9:152–64.

[30] Warrington EK, Shallice T. Category specific semantic impairments. Brain 1984;107:829–53.

[31] Warrington EK, McCarthy RA. Categories of knowledge: further fractionation and an attempted integration. Brain 1987;110:1273–96.

[32] Warrington EK, James M. Visual apperceptive agnosia: a clinico-anatomical study of three cases. Cortex 1988;24:13–32.

[33] Zihl J, von Cramon D, Mai N. Selective disturbance of movement vision after bilateral brain damage. Brain 1983;106:313–40.

[34] Zihl J, von Cramon D, Mai N, Schmid C. Disturbance of movement vision after bilateral posterior brain damage. Brain 1991;114:2235–52.