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Vision Research 40 (2000) 3435–3446

Vision Research

www.elsevier.com/locate/visres

A transient deficit of motion perception in human

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Received 2 February 2000; received in revised form 23 May 2000

Abstract

We studied the motion perception abilities in a young adult, SF, who had her right occipito-temporal cortices resected to treat epilepsy. Following resection, SF showed transient deficits of both first- and second-order motion perception that recovered to normal within weeks. Previous human studies have shown either first- or second *n* order motion deficits that have lasted months or years after cerebral damage. SF also showed a transient defect in processing of shape-from-motion with normal perception of shape from non-motion cues. Furthermore, she showed greatly increased reaction times for a mental rotation task, but not for a lexical decision task. The nature and quick recovery of the deficits in SF resembles the transient motion perception deficit observed in monkey following ibotenic acid lesions, and provides additional evidence that humans possess specialized cortical areas subserving similar motion perception functions.

Keywords: Human; Motion; Deficit

1. Introduction

A small region in the lateral visual association cortex in monkey is important for visual motion perception. This region, called the medial temporal area or MT, occupies the posterior wall of the superior temporal sulcus (Allman & Kaas, 1971; Dubner & Zeki, 1971), and has a large proportion of neurons that show directional and velocity selectivity (Maunsell & Van Essen, 1983; Albright, 1984). Ibotenic acid lesions of area MT in monkey produce a short lived deficit for motion perception in the contralateral visual hemifield while the ipsilateral visual hemifield remains unaffected (Newsome & Paré, 1988; Andersen & Siegel, 1988; Pasternak & Merigan, 1994). This selective perceptual deficit is transient and typically returns to prelesion levels within a few weeks.

In contrast, the handful of well documented human motion perception deficits have persisted for much longer. For instance, the motion perception deficit documented in patient LM has endured for more than a decade (Zihl, von Cramon, Mai, & Schmid, 1991). Similarly, the deficit in patient AF has endured for years (Vaina, Lemay, Bienfang, Choi, & Nakayama, 1990), the deficits studied by Plant and Nakayama (1993) persisted for at least several months, those studied by Braun, Petersen, Schonle, and Fahle (1998) recovered after 20 months, and Greenlee and Smith's (1997) deficits were studied an average of 3 years after surgery. This apparent incongruity in the neurophysiology of primate and human motion perception is compounded by the difficulty in localizing a possible human homolog for area MT. As human lesions are typically large and extend through much of a particular vascular territory including underlying white matter, the localization of a putative human MT homolog is still questionable. Recent functional brain imaging (Watson et al., 1993; Tootell et al., 1995; Orban et al., 1995) and anatomical evidence at autopsy (Tootell & Taylor, 1995) implicate a region in human located near the dorsolateral arm of the inferior temporal sulcus, several cm. anterior and dorsal to the occipital pole. This is the general area under investigation in this study.

A human homolog to simian MT should be involved in the processing of two different types of motion, first- and second-order, which differ in spatio-temporal lumi-

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0042-6989/00/$ - see front matter
PII: S0042-6989(00)00177-2

nance covariance (Albright, 1992; see Cavanagh & Mather, 1989 for a comprehensive review; and for cat see Zhou & Baker, 1993). First-order motion refers to a change in luminance over both space and time that would arise, for instance, when a dark object translates over a light background. The perception of first-order motion relies on neural mechanisms that correlate luminance displacements over time (spatiotemporal energy) (Watson & Ahumada, 1985; Van Santen & Sperling, 1984). In contrast, second-order motion is characterized by complex movement for which the simple first-order mechanism is insensitive. For instance, a second-order motion stimulus is created by applying an uncorrelated contrast transformation to successive portions of an otherwise static random dot pattern (Chubb & Sperling, 1988). That is, a second-order motion is created in a region filled with black and white random dots, by reversing the luminance of dots first at one side of the region and continuing this transformation successively across this region over time. Perception of second-order motion requires a more complex mechanism sensitive to change in contrast information, not only spatiotemporal changes in luminance information (Chubb & Sperling, 1989; Wilson, Ferrera, & Yo, 1992). Because physiological recordings in simian MT indicate some neurons are form-cue invariant and respond to both first- and second-order motion (Albright, 1992; O'Keefe & Movshon, 1998), a true human homolog to simian MT could be involved in processing both first- and second-order motion.

Another important aspect of motion perception is the ability to recover object contours and boundaries from motion. This so-called form-from-motion is very efficient compared to other cues such as color and luminance (Nawrot, Shannon, & Rizzo, 1996). Andersen and Siegel (1988) found that MT lesions produced a very large, but short lived, deficit in the detection of shearing boundary motion. Schiller (1993) found that MT lesions, along with V4 lesions, produced some deficit in the perception of shape-from-motion. Marcar, Xiao, Rainuel, Maes, and Orban (1995) found that MT neurons in macaque responded to kinetic boundaries, but that this processing was particular for the change in local motion at the boundary and did not code the actual orientation of the boundary. Instead, neurons in inferotemporal cortex show orientation selectivity for shape-from-motion as well as shape from luminance cues. Additionally, using functional imaging techniques, Orban et al. (1995) found a right hemisphere area, located near, but separate from, the putative location of the human MT homolog, that was selectively active during a motion-defined-form task.

Mental rotation could also place demands on the neural mechanisms responsible for the perception of motion. Mental rotation represents a type of visual motion task believed to rely on the manipulation or movement of an internal representation of a visual stimulus, not the perception of visual movement in a stimulus. In a standard mental rotation task (e.g. Shepard & Metzler, 1971) an observer presumably uses mental imagery to rotate a visual stimulus before it can be compared to a standard. This is a distinctly separate process from simply recognizing a rotated stimulus (Farah & Hammond, 1988). Furthermore, there is evidence for right hemisphere specialization from lesion studies in human (Ratcliff, 1979; Ditunno & Mann, 1989; see also Corballis, 1997). Using function imaging techniques, Papanicolaou et al. (1987) and Deutsch, Bourbon, Papanicolaou, and Eisenberg (1988) found increased right parietal cerebral blood flow in mental rotation tasks. Cohen et al. (1996) found that mental rotation activated, among other areas, a lateralized extrastriate region in areas 19 and 39 corresponding to a possible human homolog of area MT. Whether damage to this cortical region produces a deficit in the ability to perform the mental rotation task was studied in patient SF.

2. The subject

We investigated the neuroanatomical substrate of motion perception in human by studying the perceptual concomitants to acute topectomy of a small portion of the right lateral occipito-temporal cortex in a 19-year-old patient with medically intractable epilepsy.[1] Patient SFs seizure focus was localized to a small region that included the anterior occipital gyrus and the posterior sector of both inferior and middle temporal gyri. Microsurgical techniques were used to resect gray matter in this area of cortex. Blood vessels traversing the region were spared. The depth of resection was 5–6 mm and thus the underlying white matter was largely spared. Four intact cortical tissue specimens were processed histologically to assess pathological changes and to determine cytoarchitectural characteristics.[2] There were no surgical complications.

The cortical resection was limited to the posterior temporal region as indicated in Fig. 1. This resected

[1] SFs epilepsy began 3 years prior to surgery. Two weeks prior to the surgical resection, subdural surface EEG electrodes were placed over the cortical surface of the patients right temporal and occipital lobes. Recording and stimulating depth electrodes were implanted at this time. One depth electrode was positioned posterior to where specimen 3 was subsequently excised. SF gave informed consent for all research tests.

[2] Specimens were carefully removed to avoid compression and immediately immersed in cooled fixative (4% parafomaldehyde in 0.1 M phosphate buffer, pH 7.4). After 24 h, specimens were transferred to 30% sucrose buffer, sectioned at 50 μm on a freezing microtome, and processed for several histological markers (cell bodies, myelin, parvalbumen, AChE, and SMI 32).

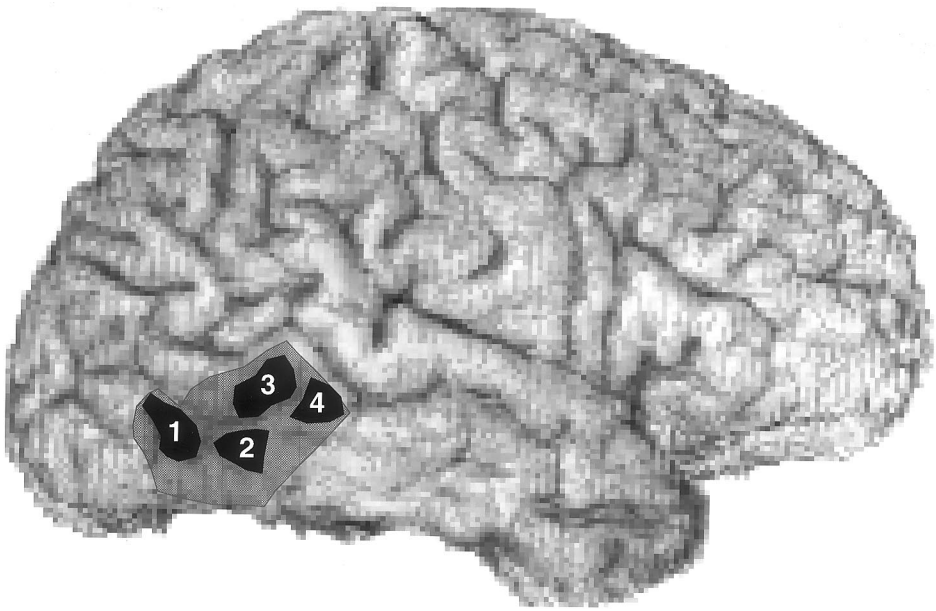

Fig. 1. Region of cortical resection. This lateral view of the right hemisphere of patient SF was generated by voxel reconstruction of magnetic resonance images using Brainvox (Damasio & Frank, 1992). Solid black regions denote the location from which the four specimens were removed. The region of the topectomy, shown in gray, extended ventrally over the inferior temporal gyrus and included cortex between the excised specimens. (Figure courtesy of H. Damasio, University of Iowa.)

area includes the region hypothesized to correspond with area MT on the basis of functional imaging (Watson et al., 1993). Although analysis of the resected area was constrained by surgical requirements for tissue removal,[3] histological characteristics of specimen 3 in particular were consistent with previous descriptions of mediotemporal cortex as shown in Fig. 2. As in macaque MT, neurons in layer 6, as well as in layers 3 and 5, stained positive for SMI 32 and there were regions of dense myelination.

In the days following the cortical resection, SF was alert and willing to participate in further psychophysical investigations. At post-operative day 4, her near and far visual acuity (20/20, Snellen) and contrast sensitivity (1.95, Pelli-Robson Chart, Clement Clarke Inc., Columbus, OH, see also Pelli, Robson, & Wilkins, 1988) were unchanged. Stereoacuity decreased slightly from 40 s arc to 50 s arc (Randot, Chicago, Stereo Optical). Eye movement recordings with a Skalar (Delft, The Netherlands) head mounted Limbus tracking system showed that SF could maintain stable fixation (Fig. 3a). Pursuit eye movements showed only slight reductions in gain with no catch-up saccades (Fig. 3b and 3c). Prior to the topectomy, SF's gain was 1.00 (SD = 0.05), with gain right = 0.99 (0.07) and gain left = 1.01 (0.02), comparable to the values of normal controls in Thurston, Leigh, Crawford, Thompson, and Kennard (1988). Following topectomy, SF's gain was 0.91 (0.07), with gain right = 0.90 (0.06) and gain left = 0.93 (0.08), values that are much closer to normal than the group of cortical lesions patients studied by Thurston et al. (1988). However, we used a target oscillation frequency (0.4 Hz) below that (up to 1.6 Hz) used by Lekwuwa and Barnes (1996) to detect pursuit gain deficits.

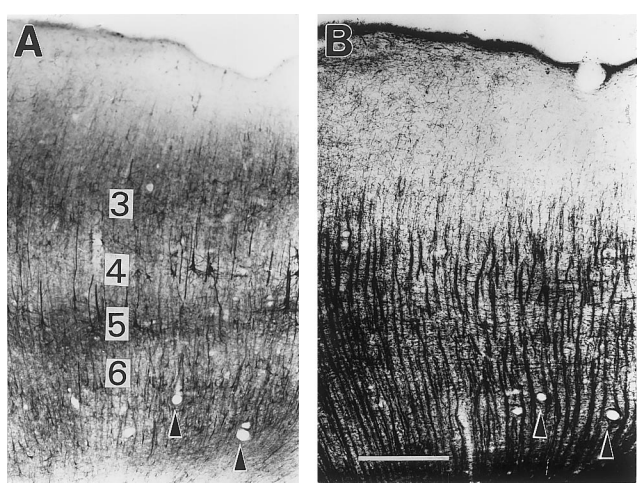

Fig. 2. Photomicrographs of cortical tissue taken from the ventrolateral surface, midway through specimen 3 (coronal sections). (A) SMI 32 shows a subpopulation of pyramidal neurons in layers 3, 5, and 6. The staining pattern in layer 6 has been described as characteristic of mediotemporal cortex in monkey (Hof & Morrison, 1995). (B) An adjacent section (arrowheads in A and B point to corresponding features) stained for myelin. Denser levels of myelin are particularly evident at left. Scale bar equals 500 μm.

[3] The small size of the individual specimens (≈ 0.5 cm) was not well suited to showing tangential gradients, although this has been a favored way of visualizing MT, which appears as a conspicuously myelin dense region. See Tootell and Taylor (1995) and references therein.

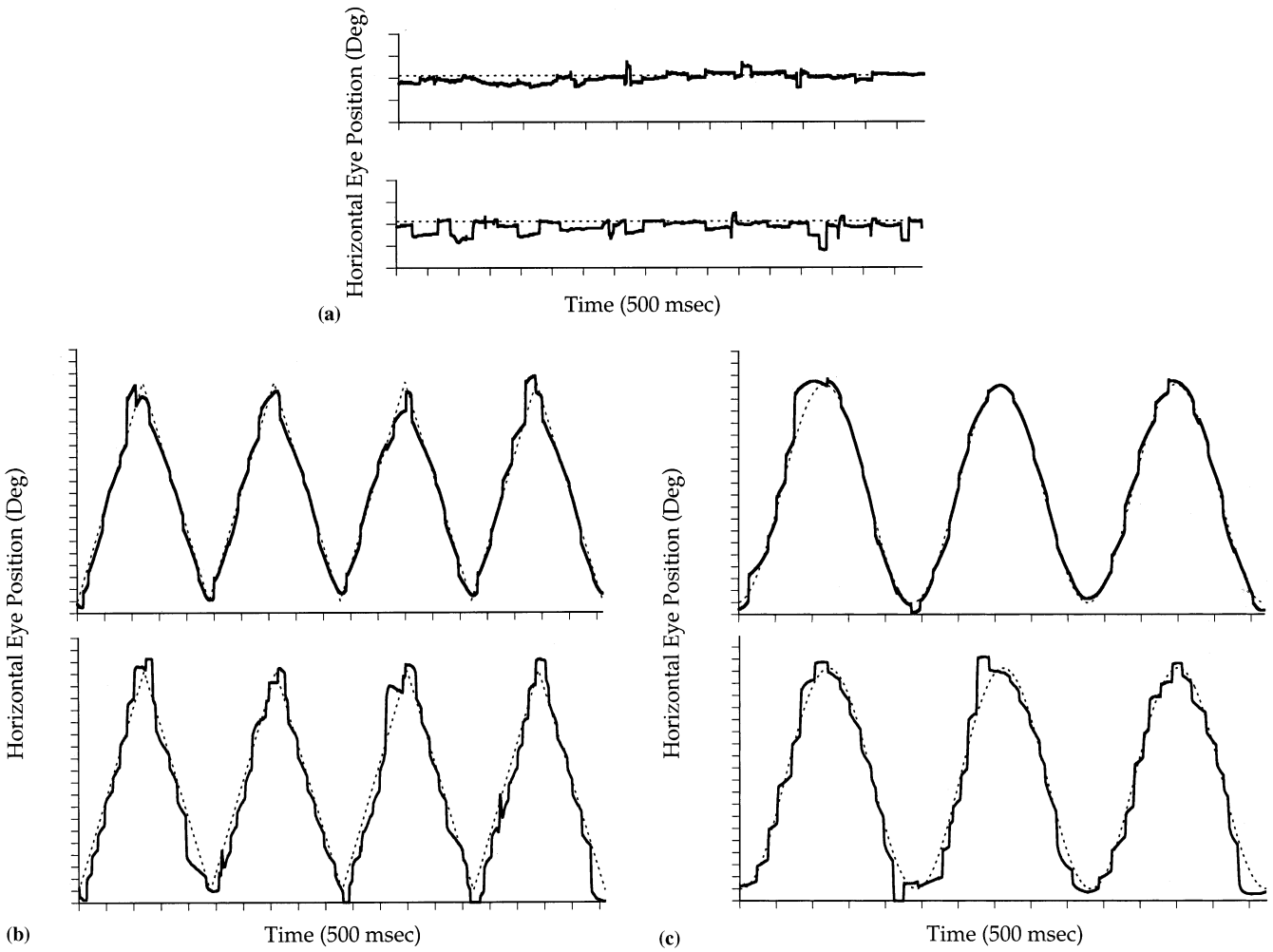

Fig. 3. Eye movement recordings were taken from SFs right eye both prior to surgery (top panel) and 4 days after surgery (bottom panel). The target in each condition was a small (26 min) dot, the movement of which is shown as a dashed line in each panel. Eye position is shown by the solid line. (a) Fixation. (b) Triangle waveform. (c) Sinusoidal waveform.

3. First and second order motion perception

Computer generated random dot cinematograms (RDC) were used to assess motion perception before and after surgical resection. These RDC stimuli resembled those used to assess acute motion perception deficits in primates (Newsome & Paré, 1988; Pasternak & Merigan, 1994). These RDCs depicted movement of a number of small black dots against the white background of the computer monitor. A portion of these dots (signal dots) moved uniformly in one of the cardinal directions while the remaining dots move in a set of random directions. When the proportion of signal dots is above an observers threshold value, the entire stimulus appears to flow in the direction of the signal dot movement (Williams & Sekuler, 1984). The observer integrates the local dot movements into a global motion percept. However, when the proportion of signal dots is below threshold, the stimulus appears to move randomly and no coherent global flow is perceived. The threshold value for perceiving the direction of the coherent flow is used to assess the integrity of underlying motion perception mechanisms. Because the observer is detecting a spatio-temporal bias dispersed throughout the entire set of dot movements, a high threshold value indicates a motion processing deficit. Normal, uncompromised motion perception requires about 12% signal with these stimuli for young observers (Nawrot & Rizzo, 1995), while, for instance, the well known motion blind patient LM required about 40% signal to perceive the direction of coherent motion in the identical tasks (Rizzo, Nawrot, & Zihl, 1995).

3.1. Methods

Both first- and second-order RDC motion stimuli were generated by a Macintosh computer and displayed on a monochrome monitor at the rate of 15 ms per

frame. RDCs comprised 150 small ($2 \times 2'$) black dots drawn within a 4° square region. Within this region, dots all moved at the speed of 11°/s, but the direction of dot movement varied. First, the cardinal direction of signal dot movement varied between stimuli. Second, within each stimulus, the direction that a particular dot moved was apparently random. Dots could switch between conveying signal and noise information, but over the entire stimulus the proportion of signal and noise dots was preserved along with the flat distribution of directions that noise dots moved.

Second-order RDC motion stimuli comprised a random two dimensional array of $4 \times 4'$ min black and white squares. Half of the squares in the 62×62 array were randomly assigned to white and the remaining squares were assigned to black. Second-order motion was generated by inverting the luminance of a proportion of squares within sequential four column (or row) intervals in each successive RDC frame. For example, downward motion at 100% signal was created by reversing the contrast of all 248 squares in rows 5–8 when creating the second RDC frame. All squares in rows 9–12 were reversed for the third RDC frame. This pattern continued through all RDC frames. The speed of this second-order motion was 17.8°/s. The proportion of signal was controlled by varying the number of squares to which the luminance inversion was applied. For instance 10% signal would correspond to a randomly selected set of 25 squares changing for each RDC frame, rather than all 248 squares in the 100% signal condition.

In these RDC stimuli signal proportion varied in a method of constant stimuli between 5 and 40% signal (5% intervals) for first-order motion and 1 and 35% (1 and 5% intervals) for second-order motion. SFs fixation was held on a small cross in the center of the screen and was monitored by the experimenter. Presentation durations were 195 m. SFs task was a four alternative forced choice of the direction of signal dot movement. SF initiated trials with a hand-held button. Stimuli were presented unpredictably at fixation or 5° into one of the four visual quadrants. SFs verbal responses were recorded into the computer by the experimenter. SF completed 480 trials for each stimulus type in each testing session.

3.2. Results

SF showed a significant increase in motion thresholds following surgery. With first-order motion stimuli, the motion processing deficit was limited to the contralateral hemifield (Fig. 4). The ipsilateral hemifield was unaffected and provided a useful comparison. With second-order motion stimuli, both visual hemifields showed a significant threshold increase (Fig. 5). The downward shifting of SFs acute psychometric functions (Figs. 4 and 5) indicated a change in absolute sensitivity to first- and second-order motion stimuli. There were no changes in slope and upper asymptote and this implies that factors such as increased noise in the system and increased observer variability, perhaps involving attention, could be discounted in this task. Moreover, elevated thresholds for both first- and second-order motion returned to normal values within 2 and 4 weeks, respectively. Most other aspects of SFs perceptual and cognitive performance remained unaffected. Pelli–Robson contrast sensitivity remained constant (1.95) throughout the testing period as did performance on static control tasks, such as judging the orientation of small, static arrows in a brief, noisy presentation.

4. Perception of shape from motion

One important role for motion perception is the detection of boundaries between objects, or image segmentation (Nakayama, 1985). That is, as an observer views a figure translating across a backgound, the movement of all the visible points on the figure will be common, but different from the motion of points in the background. The visual system appears well tuned to use these localized differences in movement for image segmentation (Regan & Hong, 1990). Nawrot, Shannon, & Rizzo (1996) showed that human observers displayed remarkable sensitivity to motion-defined-form when compared to the perception of form from color and luminance cues. Regan, Giaschi, Sharp, and Hong (1992) found that parietotemporal lesions produced a deficit specific to motion-defined form. Although these patients exhibited rather broad areas of damage, the most common region of overlap was white matter underlying the putative location of a human MT homolog. Expanding on this with functional imaging, Orban and colleagues (Orban et al., 1995; Dupont et al., 1997; Van Oostende, Sunaert, Van Hecke, Marchal, & Orban, 1997) have shown a specific cortical area, separate from human MT homolog, as being selectively active during a motion-defined-form task. This region, labeled kinetic occiptial (KO), is located in lateral occipital cortex a couple of centimeters caudal to the putative location of the human homolog of area MT. Considering these results and the extent of SFs overall lesion, we wondered if it were possible that SF would show a deficit on the perception of motion-defined form that spared the perception of form from other cues.

4.1. Methods

This experiment used portions of a 5AFC letter identification task previously described by Nawrot et al. (1996). The shapes of five different letter shapes were

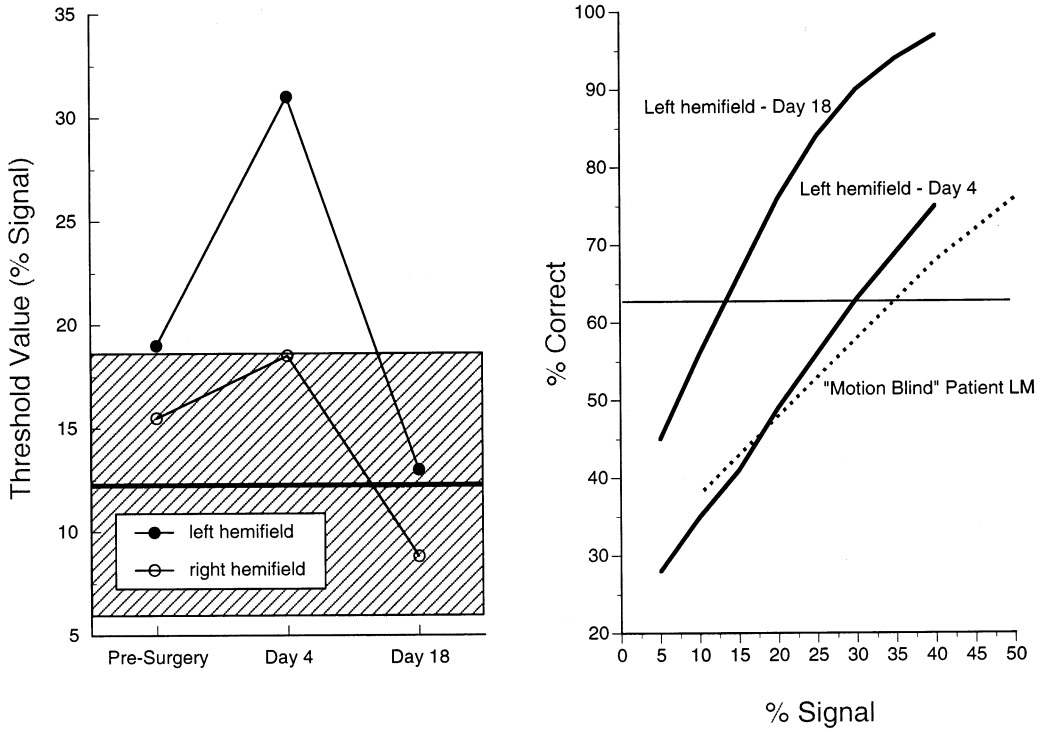

Fig. 4. First-order motion perception. (A) Shown are thresholds for first-order motion perception in the left and right visual hemifields at three successive testings. The thick horizontal line and hatched region show the mean threshold and ± 2 SD for normal controls ($n = 22$). In the contralateral field a deficit is seen at day 4, but normal performance returns by day 18. The pre-surgery testing was performed 8 days after the subdural and depth electrode placement. SFs pre-surgery performance, although normal, might have been affected by the prior depth electrode placement just posterior to specimen 3. However, this pre-resection surgery offers an excellent control for the general effects of surgery. (B) The acute threshold increase is the result of a downward shifting of the entire psychometric function. The lower two functions compare SFs abnormal performance in the left hemifield at day 4 with the performance of the motion blind patient LM on the identical task (Rizzo et al., 1995). Intersections of these functions with the horizontal line at 62.5% signal signify relative threshold values.

made visible against a background in a random-dot display in three different conditions: (i) dot movement within the letter shape against a static background; (ii) dot movement in different directions within the letter shape and the background; and (iii) differing dot densities within the letter shape and the background, a control condition for the two moving conditions.

The stimulus comprised 4000 randomly positioned 2×2 min arc white dots within an $8.3 \times 8.3°$ region of a darkened monitor face. Each stimulus presented one of five letter shapes (E, H, L, O, or T) in one of five different locations. To make the letter visible, a proportion of the dots within the letter were given: (i) 3.3°/s movement in one of four cardinal directions while background dots remained stationary; (ii) 3.3°/s movement in the direction opposite the direction of background dots; and (iii) an increase in the number of dots within the letter regions. In all three conditions it was possible to titrate the amount of signal denoting the letter region by varying the proportion of dots. At 100% signal: (i) all the dots within the letter region would move; (ii) all the dots would move in the direction opposite the background movement; or (iii) the number of dots would double thereby doubling the density of dots within the letter region.

To begin each trial SF fixated a small cross in the center of the screen. The experimenter triggered a 1 s presentation of the stimulus and then recorded SFs verbal response to computer with a keypress. Stimuli of varying signal proportions were presented in a method of constant stimuli. SF completed 125 trials for each condition at three intervals: pre-surgery; post-op day 5; and post-op day 18.

4.2. Results

For each condition a threshold was determined with probit analysis and these thresholds were then normalized to the data of ten normal observers (Nawrot et al., 1996). Fig. 6 shows SFs thresholds plotted in terms of standard deviations from the mean so that the three tests, having different means and SDs can be compared. For all three conditions SFs thresholds were within the normal range before surgery. However, immediately

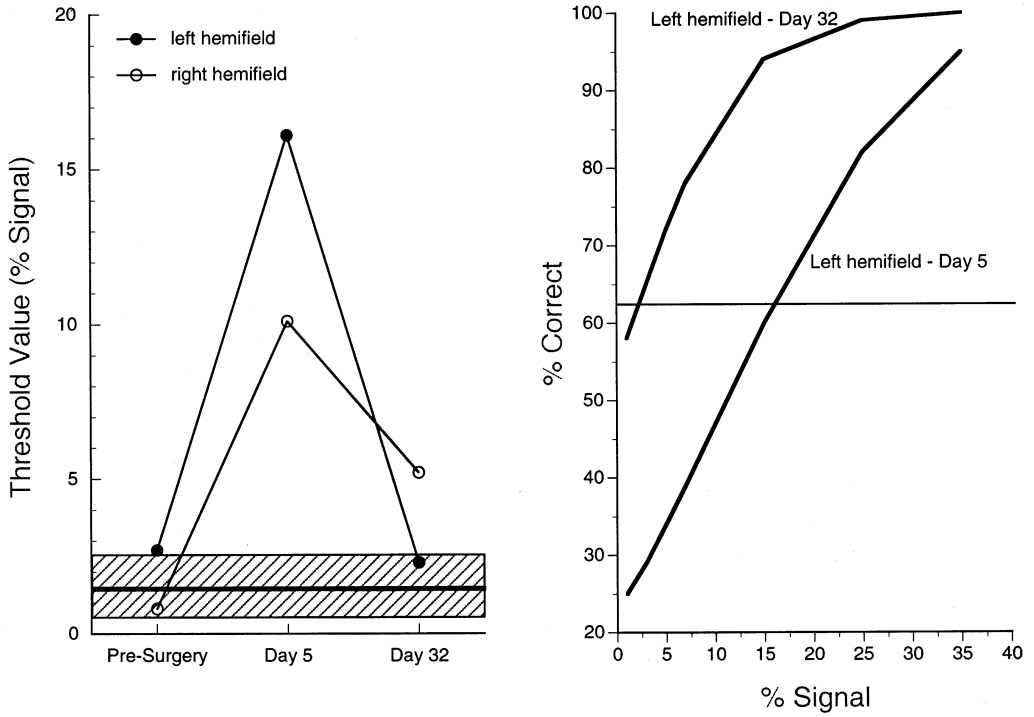

Fig. 5. Second-order motion perception. (A) Shown are thresholds for second-order motion perception in the left and right visual hemifields at three successive testings. The thick horizontal line and hatched region show the mean threshold and ± 2 SD for normal controls. A deficit is seen in both visual fields at day 5. Normal performance returned in the contralateral field by day 32. (B) Like first-order motion, the acute threshold increase is the result of a downward shifting of the entire psychometric function. The upper function shows SFs normal performance in the left hemifield at day 32 and the lower function shows SFs abnormal performance at day 5.

following the topectomy, thresholds for detecting shape from motion against a static background jumped to 7 SDs higher than the normal mean and then returned to normal by the 18th day. Similar transient deficits were not seen for either movement in opposite directions or for the dot density control condition.

An analysis of SFs errors in the shape-from-motion task on day 5 shows that SF made more errors on letters presented to the left hemifield ($\chi^2(2) = 14.11$, $P < 0.005$), twice as many errors as in the right hemifield. Remember that the left visual field was contralateral to the lesion and was the field in which the first-order motion deficit was predominant. This concordance underscores the importance of first-order motion perception in the shape-from-motion task, helps define the selectivity of this specific deficit, and suggests a bilateral representation of KO (Dupont et al., 1997) rather than a singular right hemisphere representation (Orban et al., 1995).

5. Mental rotation

Mental rotation is an aspect of motion processing related to mental imagery. Although relationships between reaction time and orientation of visual test figures are described (Shepard & Cooper, 1982), less is known about the underlying neural mechanisms. Ditunno and Mann (1989) found right parietal lesions

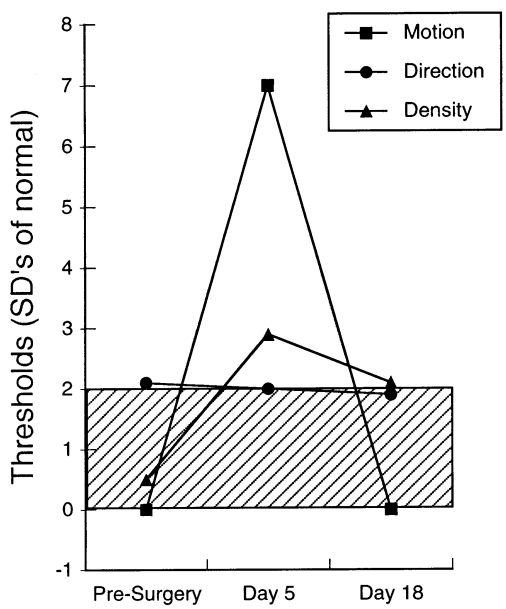

Fig. 6. Perception of shape-from-motion. As the three tasks have different normal thresholds, the vertical axis represents SFs threshold values in standard deviations from the normal mean for each task. SFs threshold for the perception of shape-from-motion (squares) shows a 7 SD increase 5 days after surgery and then improves to normal by 18 days after surgery.

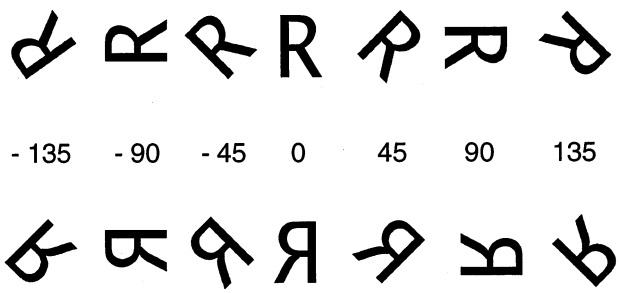

Fig. 7. The 14 possible stimuli for the mental rotation experiment are shown here as the various rotations and two registrations of the capital letter R.

produced longer reaction times, more errors, and differential accuracy over the angle of rotation when compared to both normal observers and observers with comparable left hemisphere lesions. Using functional imaging techniques, Papanicolaou et al. (1987) and Deutsch et al. (1988) found increased activation in right partietal regions with similar mental rotation tasks. In contrast however, Alivisatos and Petrides (1996) found only selective left inferior parietal activation for mental rotation after subtracting activity seen in a mirror image task, while Cohen et al. (1996) found no evidence of such lateralization in mental rotation. However, Cohen et al. found that Brodmanns areas 39 and 19, areas believed to contain the human homolog of area MT, was selectively activated in most subjects performing the mental rotation task. We wondered if the selective damage to these areas from SFs topectomy would produce a deficit in SFs ability to perform a mental rotation task along with other selective motion perception deficits that this lesion would produce. For comparison to a left hemisphere dominant language task (Gazzaniga, 1983; Faust, Kravetz, & Babkoff, 1993; Abernethy & Coney, 1996), changes in SFs mental rotation performance are also compared to changes in SFs performance in a lexical decision task. Such a language based task used the same 2AFC reaction time methodology but should remain unaffected by SFs topectomy and provide a useful comparison for the mental rotation task.

5.1. Method

The mental rotation task was modeled on Cooper and Shepard (1973). A single capital alphabetic character, R, was used without a priming cue.[4] The letter could be presented in any of seven orientations (rotated up to $\pm 135°$) in either forward or backward (mirror image) registration (Fig. 7). The subjects task was to respond as quickly and accurately as possible whether the letter was normal or backward registration.

The subject initiated each trial with a keypress after fixating a small dot in the center of the screen from a distance of 57 cm. One large ($3 \times 2°$), white letter was presented in the center of the dark background. The letter remained on the screen until the subject responded with one of two keypresses signaling whether the letter was presented forward or backward registration. The subjects reaction action time was measured to the nearest millisecond.[5] SF completed 240 trials at her own pace taking breaks as needed. Testing took place prior to topectomy, 5 days after surgery, and 18 days after surgery.

Like the mental rotation task, the lexical decision task used a simple unprimed presentation and a 2AFC reaction time response. For the lexical decision task the subject was presented with 40 words all four-letter nouns (e.g. shoe, fork, ball, tree) and 40 meaningless four-letter non-words (e.g. durg, rerk, limk, upos). Again the subject fixated a small dot in the center of a black screen. The words and non-words were presented as white letters 1.3° in height. Using the same two keypresses as the mental rotation task, subject signaled whether the letters were a word or non-word. A total of 80 trials were completed by SF, proceeding at her own pace, taking breaks as needed. The test was performed on the same days that the mental rotation test was performed.

5.2. Results

As Cooper and Shepard (1973) have shown that the mental rotation response function is symmetrical for clockwise and counterclockwise rotation, reactions times were averaged for each magnitude of rotation (Fig. 8). In pre-surgery testing, SFs mental rotation reaction times are indistinguishable from our normal controls and similar to the performance shown in Cooper and Shepard (1973), Fig. 2, condition *n*). Lexical decision reaction time is also identical to our normal controls. However, five days following topectomy, SFs mental rotation reaction times increased 380% (Fig. 8B). In contrast her lexical decision reaction times increased less than 100%. The magnitude and pattern of the reaction time changes suggests that mental rotation processing was differentially affected by the topec-

[4] A familiar asymmetric letter (R) was chosen over more complicated three-dimensional structures (Shepard & Metzler, 1971) to minimize the possibility that SF would find the task too difficult and be unable or unwilling to perform the task in the acute post-operative phase.

[5] There is potential error of up to several milliseconds in the computers ability to measure a keypress, but this error is very small compared to the duration of the mental rotation effect and the size of the effect measured in this study.

tomy.[6] At 5 days after surgery, the entire function is shifted vertically suggesting the forward/backward decision was affected. Furthermore, there was no difference in reaction time for forward and backward letters (means of 2337 and 2348 ms, respectively). Additionally, as estimated from the slope of the functions, the rate of mental rotation decreased from 409°/s in presurgery testing to 175°/s following acute topectomy. This indicates a slowing in SFs rate of mental rotation, independent of any generalized slowing in reaction time. Again, this effect was transient as SFs mental rotation reaction time function returned to normal by the 18th day (Fig. 8C) similar to the deficits in visual motion perception. It is important to remember that the presurgery testing was preceded by a craniotomy during which subdural electrodes were placed over the region subsequently removed during the topectomy. Aside from the topectomy during the second surgery, the procedures were remarkably similar and any generalized or systemic effects would likely be the same for both presurgery and the acute post-surgery testing.

6. Discussion

Our results clearly show that removal of this small region of right occipito-temporal cortex produced a specific and transient motion perception deficit in patient SF. First-order motion perception was affected in the contralateral visual hemifield. Second-order motion perception was impaired in both hemifields, as was perception of shape-from-motion (albeit more so in the contralateral visual field), and reaction time in a mental image rotation task increased almost 400%. These deficits were all short lived, and performance returned to normal within weeks.

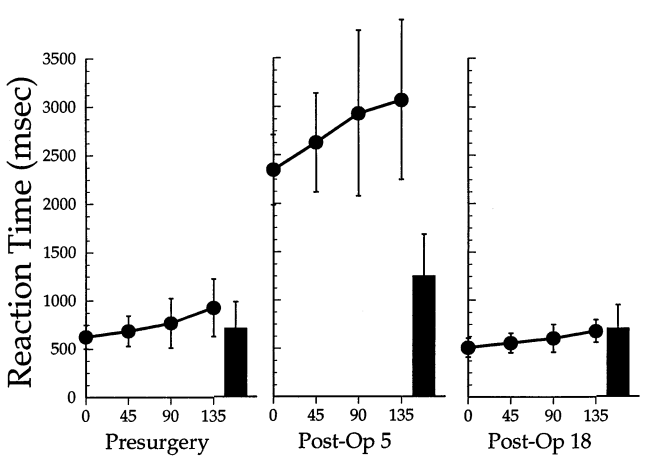

Fig. 8. (A) Reaction times on mental rotation (line and circles) and lexical decision (bar) tasks in the pre-surgery testing. Reaction time is shown on the vertical axis while degrees that the letter was rotated from vertical is shown on the horizontal axis. The error bars denote 1 SD. (B) Following the topectomy, SFs performance in the mental rotation task increased nearly 400% while her performance in the lexical decision task increased less than 100%. (C) SFs reaction time in the mental rotation task recovers to normal levels by the next testing at day 18.

[6] There was little change in SF's error rates: 4, 7, and 3%, respectively for mantal rotation, and 3, 11, and 9% for the lexical decision task.

The transient deficit in the perception of direction for first-order motion in SF is similar to the specific deficit reported in monkey by Newsome and Paré (1988), who reported recovery times of 1–3 weeks, depending on the size of the unilateral lesion in MT. While the specificity and timeline of recovery suggests that the topectomy in SF affected a human homolog of MT, this lesion produced other motion processing deficits.

The finding of a second-order motion processing deficit in the ipsilateral visual field in SF suggests that second-order motion depends on a mechanism with broad receptive fields that span the vertical midline. This mechanism may involve V2 (Wilson et al., 1992), the first area in the visual processing hierarchy with strong interhemispheric callosal connections. It appears that first-order motion processing relies on a projection from V1 to MT, and second-order motion upon the sequence of projections from V1 to V2 to MT. In monkey, the terminations in MT of axonal projections arising from V1 and from V2 have different morphological features (Rockland, 1995, 1989) that may provide different functional combinations for motion perception. A single lesion in MT could damage afferent connections from V1 and result in a unilateral first-order motion deficit, while damage to V2 afferents could result in a bilateral deficit in the perception of second order motion.

Previous studies of motion perception in patients with cerebral lesions provide differing accounts of the relationship between first- and second-order motion processing. Braun et al. (1998) found the motion perception deficits in two patients with lateral occipito-temporal lesions included both first-order and second-order motion, but the deficits were confined to the contralateral visual field. Similarly, Plant, Laxer, Barbaro, Schiffman, and Nakayama (1993) found increased thresholds for a second-order motion stimulus in the contralateral visual field but not the ipsilateral visual field. Greenlee and Smith (1997) found that superior temporal and lateral inferoparietal lesions produced threshold increases for both first- and second-order motion with little difference between the visual hemifields (their Figs. 7 and 6a), and inferred a strong overlap in the cortical processing of both motion types. Vaina, Makris, Kennedy, and Cowey (1998) reported that a unilateral medial occipital lobe lesion produced a selective deficit in the contralateral field for first-order motion tasks that spared the perception of second-order motion. Conversely, Vaina

and Cowey (1996) reported that a small unilateral left occipito-parietal lesion produced a selective deficit of second-order motion perception in the contralateral visual hemifield, but spared the perception of first-order motion. Together these findings support the notion of a separation of first- and second-order motion processing streams (Vaina, Cowey, & Kennedy, 1999) that reconverge at some point such as the human homolog of area MT. It is this later stage that appears to be damaged in the case of SF.

SF also showed a transient deficit in the perception of shape-from-motion which may have been the result of damage to the kinetic occipital (KO) region discovered by Orban et al. (1995). KO is a small region in lateral occipital cortex, neighboring, but distinct from, the putative homologs of areas MT, V3, and V3a. KO is selectively activated by boundaries defined by motion cues and is relatively inactive when subjects view uniform motion or when boundaries where defined by static luminance cues. Similar to what one would expect if this region were damaged, SF's right hemisphere lesion produced a deficit that was greatest when shape information was given by movement against a static background, while little change was seen when shape information came from motion in opposing directions and from a static cue to shape. Even more, SFs shape motion contral deficit was greatest in the lateral visual field a finding consistent with a bilateral representation (Dupont et al., 1997). However, as SFs deficits included the perception of first- and second-order motion it is difficult to assess the contributions of area KO, area MT, or connections between the two.

One final aspect of SF's motion processing deficit was a reduction in the speed at which she could perform a mental rotation task. Like SF's other deficits, this reduction in mental rotation speed recovered within a few weeks. Functional imaging studies have shown that diverse cortical areas are activated during mental rotation (Cohen et al., 1996; Alivisatos & Petrides, 1996). One location activated in three-fourths of Cohen et al.'s (1996) subjects is believed to be the putative location of a human homolog of area MT, that overlaps with the location of SF's lesion. However, it remains unclear what role this human MT homolog plays in mental rotation. It is certainly not of singular importance. Parietal lobe regions were most commonly activated by the mental rotation task in the Cohen et al. (1996) study, and right hemisphere parietal lobe lesions produce large deficits in mental rotation (Ditunno & Mann, 1989). Additionally, there are suggestions of motor planning processes in mental rotation (Wexler, Kosslyn, & Berthoz, 1998) and Cohen et al. (1996) found activation in premotor cortex but no activation in motor cortex with mental rotation tasks. Therefore the precise role of the human MT homolog in mental rotation remains unclear.

Acknowledgements

This work was supported by NIH PO 20 RR11817, NIH PO NS19632, the Hoover and Carver Foundations. Portions of this work were previously presented at the Association for Research in Vision and Ophthalmology, Ft. Lauderdale, FL, May, 1997 (Rizzo, & Nawrot, 1997). We thank Hanna Damasio for Fig. 1 and for comments on this work.

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