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CASE STUDY

Visual perception without awareness in a patient with posterior cortical atrophy: Impaired explicit but not implicit processing of global information

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Abstract

A patient with progressive posterior cortical atrophy (PCA) was examined on several tests of visual cognition. The patient displayed multiple visual cognitive deficits, which included problems identifying degraded stimuli, attending to two or more stimuli simultaneously, recognizing faces, tracing simple visual stimuli, matching simple shapes, and copying objects. The patient was also impaired in identifying visual targets contained at the global level within global–local stimuli (i.e., smaller letters that compose a larger letter). Although the patient denied any conscious awareness of the global form, he nevertheless displayed a normal pattern of global interference when asked to identify local level targets. Thus, the patient processed the global information despite not being consciously aware of such information. These results suggest that global–local processing can take place in the absence of awareness. Possible neurocognitive mechanisms explaining this dissociation are discussed. (*JINS*, 2002, *8*, 461–472.)

Keywords: Posterior cortical atrophy, Global–local processing, Consciousness, Balint's syndrome, Simultanagnosia

INTRODUCTION

Dementia has been defined as a loss of cognitive functioning in two or more areas of cognition, including memory, language, judgment, abstract reasoning, and visual cognition (American Psychiatric Association, 1994). The majority of dementia cases are due to Alzheimer's disease (AD), a progressive disorder characterized by neurofibrillary tangles and neuritic plaques in the medial temporal lobes and association cortices (Terry & Katzman, 1983; Terry et al., 1981). The specific deficits associated with AD are impairments in memory, naming, problem solving, and visual cognition (see Bondi et al., 1996). Although memory functions have been the primary focus of research in AD, it is now apparent that visual cognitive deficits can represent a profound area of impairment in these patients (Filoteo et al., 1994). The visual cognitive disorders observed in AD are often similar to those seen in patients with focal damage to the parietal lobes, in general, and the right parietal lobe, in particular (Filoteo et al., 1994; Mendez et al., 1990a, 1990b). For example, AD patients are often impaired on tests of visual construction, visuo-spatial judgments, and visual object recognition (Filoteo et al., 1994; Mendez et al., 1990a, 1990b). In most cases of AD, the primary sensory and motor areas are relatively spared as compared to other brain regions (Braak et al., 1989; Lewis et al., 1987), and as a result, most AD patients do not experience deficits in basic aspects of sensory or motor functioning.

Although a generalized cognitive decline occurs in most patients with AD, it is now clear that some patients develop progressive deterioration in a single cognitive domain prior to the development of more global cognitive impairment. For example, several case studies have been reported on patients with progressive aphasia (Galton et al., 2000; Luzatti

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& Poeck, 1991; Mendez & Zander, 1991; Mesulam, 1982; Morris et al., 1984; Petersen, 1998; Poeck & Luzatti, 1988; Snowden et al., 1992), alexia (Beversdorf & Heilman, 1998; Freedman et al., 1991), and apraxia (De Renzi, 1986; Dick et al., 1989; Fukui et al., 1996). Neuropathological studies of these patients have identified pathology in the expected brain regions, given the nature of the patients' neurocognitive deficits (e.g., left perisylvian involvement in cases of primary progressive aphasia; Mesulam, 1982). Further, histopathological studies have revealed not only the pathology observed in AD (i.e., neuritic plaques and neurofibrillary tangles), but also other histopathological changes including Pick bodies and Lewy bodies (Farina et al., 1996; Galton et al., 2000; Kirshner et al., 1987; Mesulam, 1982, 1987). These findings indicate that these focal progressive disorders can be due to pathology other than that which is typically associated with AD.

In addition to cases of primary aphasia, alexia, or apraxia, several cases have been reported on patients with progressive cognitive deterioration with the initial or primary cognitive disturbances in the area of visual cognition. This particular presentation of progressive cognitive change has been given a variety of labels, including the visual variant of AD, the occipital lobe variant of AD, or posterior cortical atrophy (PCA). Neuropathological studies of patients who initially presented with visual disturbances, and then later went on to develop other symptoms of dementia, have revealed primary involvement of the occipital lobes, occipital–parietal regions, and occipital–temporal regions (Berthier et al., 1991; Hof & Bouras, 1991; Hof et al., 1983, 1989, 1990; Morrison et al., 1991). Although most histopathological studies have identified AD pathology in these patients (i.e., neuritic plaques and neurofibrillary tangles), we use the term *PCA* to refer to this clinical presentation because other studies have found a variety of neuropathological processes in these patients (see Victoroff et al., 1994).

The primary deficit in patients with PCA is a profound impairment in perceiving visual information, although the nature of their deficits can vary. For example, some patients have been reported to have Balint's syndrome, which consists of simultanagnosia, deficits in reaching, optic ataxia, and gaze apraxia (Cogan, 1985; Coslett et al., 1995; Flekkoy, 1976; Graff-Radford et al., 1993; Mendez & Cherrier, 1998; Mendez et al., 1990). Other reports have indicated that these patients can exhibit Gerstmann's syndrome, which includes agraphia, finger agnosia, and right–left disorientation (Freedman et al., 1991; Mizuno et al., 1996). Mixtures of Balint's and Gerstmann's syndrome have also been reported in patients with PCA (Benson et al., 1988; De Renzi, 1986; Freedman et al., 1991; Pietrini et al., 1996; Wakai et al., 1994). Other deficits noted in patients with PCA have included alexia with and without agraphia, prosopagnosia, visual neglect, or extinction to double simultaneous stimulation (Ardila et al., 1997; Berthier et al., 1991; Cogan, 1985; Crystal et al., 1982; Freedman et al., 1991; Levine et al., 1993; Mendez & Cherrier, 1998; Neary & Snowden, 1987; Nissen et al., 1985). In some cases, patients can experience a single visual cognitive deficit for a number of years prior to the development of any other visual cognitive disturbances. Further, the profile of visual impairment in PCA patients can change as the disease progresses (Attig et al., 1993; Della Sala et al., 1996; Mendez & Cherrier, 1998; Ross et al., 1996).

Despite the variability of visual cognitive and associated deficits in patients with PCA, one finding in many of these patients has been a profound impairment in perceiving more than one object at a time. For example, these patients are often described as being relatively normal in identifying the elements of a visual scene, but are often impaired (or completely unable in some cases) in perceiving the overall scene. This deficit, which can be most accurately described as dorsal simultanagnosia (see Farah, 1990), will manifest behaviorally in several different ways, including impairments in identifying degraded stimuli, naming pictured objects that have been cut up and rearranged, or coherently describing a visual scene, to name a few. We suggest that simultanagnosia is a central deficit in many patients with PCA and could account for their general deficit in visual object perception.

A few recent reports have examined the possible neuropsychological underpinnings of PCA patients' visual impairments. Coslett et al. (1995), for example, examined 2 patients with presumed PCA. In their study, PCA patients were presented with global–local stimuli that consisted of small (local) letters (e.g., small 'S's) that were arranged to form larger (global) letters (e.g., a large 'A'; see Figure 1). Subjects were asked to detect a target that could appear at either the large or small level by pressing a key when the target appeared at either one of the levels and withholding a response if the letter appeared at neither level. The results indicated that both PCA patients were slower and less accurate in detecting the target when it appeared at the large, global level. In fact, one of their patients could not detect any of the large letters on a subsequent global–local task where the stimuli were presented for an unlimited time period. Because attention has often been likened to a "spotlight" or "beam," Coslett et al. (1995) interpreted these results as an indication that their patients suffered from a reduction in the size of their attentional "spotlight." Other investiga-

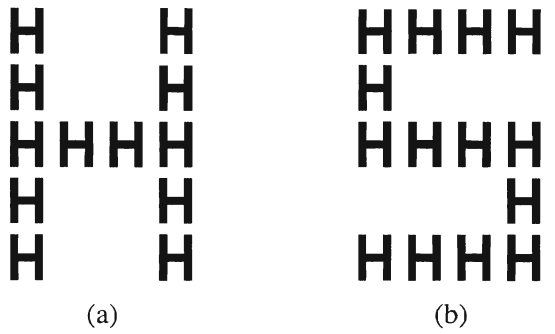


Fig. 1. Examples of global–local stimuli that are (a) congruent or (b) incongruent.

tors (Stark et al., 1997; Thaiss & De Bleser, 1992) have also suggested this explanation of the visual cognitive deficits in patients with PCA.

This interpretation of PCA patients' global processing impairment is consistent with other accounts of the visual cognitive deficits associated with simultanagnosia. For example, one of the earliest descriptions of a patient with simultanagnosia attributed such visual disturbances to an impairment in attention (Holmes & Horax, 1919). Attention often has been a difficult construct to define, but there is some agreement that this cognitive process serves to select information in our environment for further processing (see, e.g., Posner & DiGirolamo, 2000). This further processing is said to enable information to reach conscious awareness (Crick & Koch, 1990). Although there is a great deal of support for this conceptualization of attention, it is now clear that information that is unattended (or that does not reach consciousness) is nevertheless processed to some extent. For example, patients who have severe damage to the occipital lobes and display visual field defects can still display some residual visual abilities within their blind field (Marcel, 1998; Sanders et al., 1974). This "blindsight" phenomenon occurs in the face of the patients denying any awareness of the visual stimuli that is altering their behavior. Similar observations have been made in patients with simultanagnosia secondary to bilateral occipital–parietal dysfunction. For example, these patients can demonstrate normal facilitation in word identification when two semantically related words are presented simultaneously, despite their inability to see the two words at the same time (Coslett & Saffran, 1991). Such "implicit" processing in simultanagnosic patients also has been displayed using the Stroop task (Wojciulik & Kanwisher, 1998).

The purpose of the present study was to further report the visual cognitive abnormalities observed in an individual with PCA (patient M.H.).[1] We therefore report the visual cognitive deficits displayed by our patient on standard and nonstandard neuropsychological tests of visual cognition. Further, given that we feel that a fundamental deficit in many patients with PCA is in perceiving multiple objects at the same time (i.e., simultanagnosia), we examined our patient on a directed attention task of global–local processing. Note that the PCA patients in the study by Coslett et al. (1995) were tested on a divided global–local attention task in that the target could appear at either the global or the local level on consecutive trials. In the present study, our PCA patient was told at what level the target would appear and as such our patient did not have to divide or shift his attention between global and local levels (see Filoteo et al., 1992 for a description of this shifting phenomenon in AD patients). In addition, two types of stimuli were used in the present study: congruent stimuli, in which the global and local forms were the same (e.g., a large 'H' made up of smaller 'H's), and incongruent stimuli, in which the global and local forms were different (e.g., a larger 'H' made up of smaller 'S's). In his original study using these stimuli, Navon (1977) found that normal participants were slower to detect a target if the stimuli were incongruent as compared to congruent, regardless of the level to which their attention was directed.[2] This effect was said to be due to the irrelevant form interfering with the processing of the relevant, target form.

The use of congruent and incongruent global–local stimuli in the present study enabled us to examine whether our PCA patient would display normal interference effects, despite the fact that this patient denied being able to see the global figure at all. If the global processing deficits are due to a restricted attentional spotlight, as suggested by Coslett and colleagues, then our patient should demonstrate normal interference effects when his attention is directed to the local level. That is, if PCA results in a restricted attentional spotlight, then the global information should be processed somewhat normally at an implicit level despite the fact that the patient is impaired in explicitly processing global level targets.

METHODS

Case History

At the time of his participation in our study, M.H. was a 66-year-old, married male who had 12 years of formal education, denied any problems learning basic academic skills, and reported being an average student. The patient was employed by an oil company for approximately 30 years and retired in 1989. Prior to that, he was in the military for 9 years and obtained the rank of Sergeant First Class. M.H. was in relatively good health until 1989, when he began to experience problems with his vision, characterized as problems reading and difficulty driving. The patient also reported that around 1990 he started to have problems with his memory. His wife confirmed this report and stated that he continued to have memory problems that had become progressively worse. Neuro–ophthalmic examination of the patient revealed deficits in basic visual processes, including mildly constricted eye movements, impaired saccadic pursuits, and abnormal optokinetic nystagmus (particularly

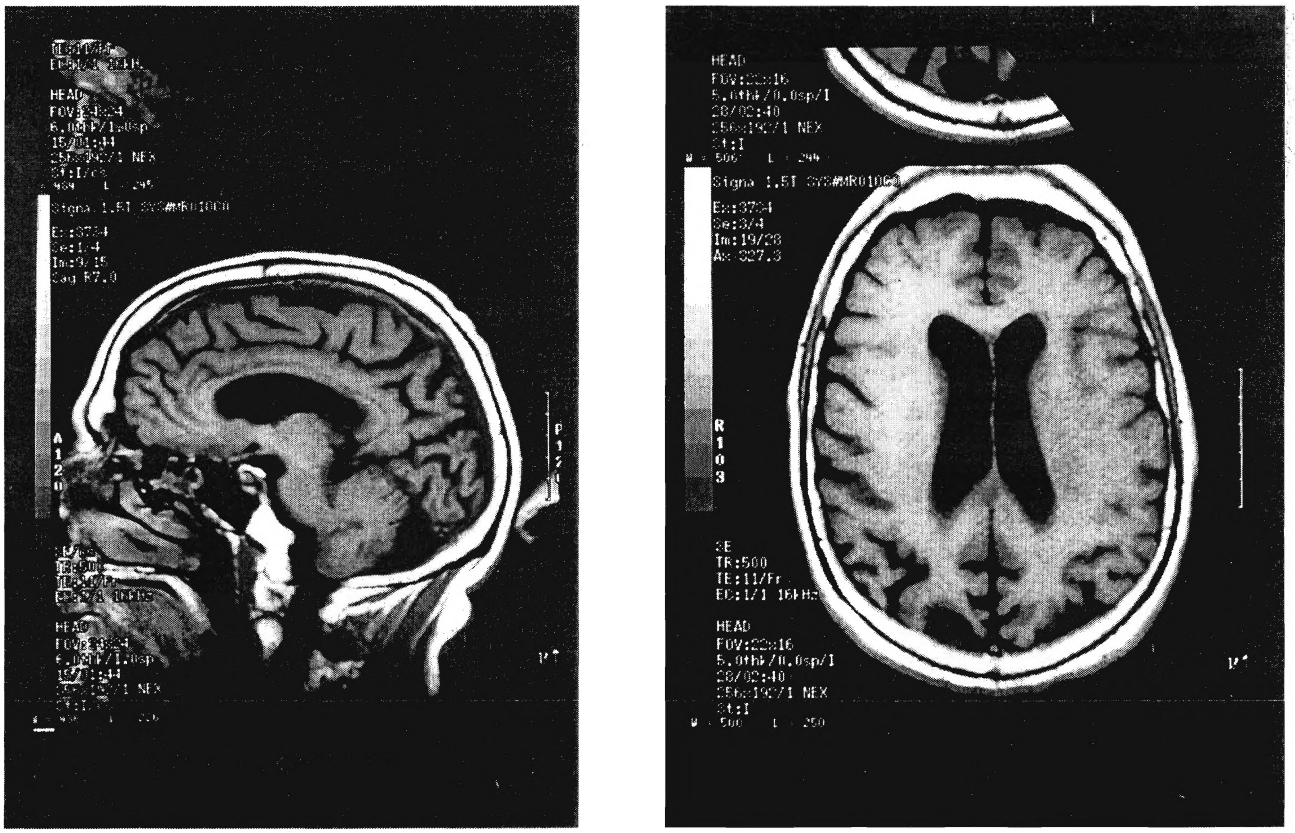
[1]It is important to note that although M.H. displayed global brain atrophy at the time of our evaluation (see Case History), he did display rather circumscribed atrophy of posterior cortices early in the course of his disease. Therefore, we chose to categorize M.H. as having PCA rather than categorizing him as having a visual variant of AD (which would be unwarranted given that we are uncertain of the nature of M.H.'s pathology) or as having a specific visual behavioral syndrome (e.g., Balint's syndrome; which would suggest that all patients with posterior degeneration have the same behavioral problems).

[2]Although interference was found in normal controls when attention was directed to either the global or the local level, Navon (1977) found greater interference when attention was directed to the local level than the global level. This led to Navon's proposal that global information takes precedence over local information. Based on more recent work, however, it appears that the global precedence effect can be altered by a number of factors, such as the size of the global and local features, the proximity of the local features, etc.

in the vertical plane). M.H.'s acuity was relatively intact (20/25 on the right and 20/30 on the left), and his optic nerves and maculae appeared to be normal. Tests of visual evoked potentials revealed delayed responses bilaterally with small wave forms. An MRI revealed significant global atrophy, although the posterior regions displayed greater volume loss relative to anterior regions (see Figure 2 as well as Footnote 1).

During our evaluation, M.H. was oriented to person and place, but had difficulty with the time of day and the exact date. He had severe problems naming past presidents spontaneously, but performed better when he was provided with phonemic cues (i.e., stating the first sound of the name). The patient was aware of current events, although he had to be prompted by the examiner. His speech was fluent, but he did have mild word finding problems during spontaneous conversation. His prorated Verbal IQ was 91 (Wechsler Adult Intelligence Scale–Revised; Wechsler, 1981). His nonverbal IQ could not be obtained because of his profound visual impairments (see below). M.H. displayed severe memory problems, consistent with his and his wife's reports, in that he was severely impaired in recalling word lists and short stories after 20- and 30-min delays. His language abilities were also impaired, but to a lesser extent. His phonemic verbal fluency (i.e., letter fluency) was within normal limits, whereas his semantic verbal fluency was mildly impaired. He performed poorly on a visual confrontation naming test because of his visual impairments, but he was able to name 52 out of 60 items from the task when verbal descriptions of the items were presented. He also was able to identify eight out of eight common objects (e.g., keys) tactually. These results suggested that his naming abilities were relatively intact. M.H.'s raw scores for these neuropsychological tests can be seen in Table 1. Overall, M.H. was very aware of his cognitive deficits and reported feeling frustrated about his visual impairments.

M.H.'s visual abilities were severely impaired. He had difficulty finding his way around the testing room. When greeted by the examiner with an outstretched hand, M.H. did not acknowledge the attempted handshake until his wife grabbed his hand and held it up. The patient displayed the three deficits characteristic of Balint's syndrome. First, he was unable to see more than one object at a time (simultanagnosia). This was demonstrated both by his inability to identify more than one object when presented simultaneously with other objects, and his impairment on clinical tests (see below). Second, he exhibited severe problems in redirecting his gaze once he fixated on a point in space



(a) (b)

Fig. 2. (a) Sagittal and (b) transverse MRI scans of patient M.H. displaying generalized cortical atrophy with greater posterior involvement.

(ocular apraxia). Third, he had severe difficulty in reaching for objects presented directly in front of him, despite his knowledge of the presence of these objects (optic ataxia). He was able to trace a line and a rectangle when presented separately (see Figures 3a–3b). However, he was unable to trace accurately a line that overlapped a rectangle and tended to not see the line and rectangle as separate objects (see Figure 3c). This latter finding was a further demonstration of his simultanagnosia.

M.H. was also severely impaired in copying a daisy (see Figure 4). When asked to count the number of black dots on a page (Dot Counting subtest from the Visual Object and Space Perception Battery or VOSPB; Warrington & James, 1991), he was correct on only 5 out of 10 items. On this test, he tended to both over and under count the number of dots on the page. M.H. was unable to identify any items on the Incomplete Letters subtest of the VOSPB. This task is somewhat like a global–local task in that the subject must integrate local features in order to see the overall global form. He was unable to describe pictured visual scenes as a whole, but tended to focus on only a single detail of the scene. M.H. was able to provide verbal descriptions of objects he was asked to imagine. For example, he provided a very detailed description about a banana that included comments about the texture, color, and shape. In contrast, M.H. was not very accurate in describing the layout of his house when asked to imagine how different rooms and pieces of furniture were related spatially. M.H. did not display any problems with right–left discrimination or praxis.

Table 1. Neuropsychological test results of patient M.H.

Neuropsychological test	Raw score	Impairment level
WAIS–R Subtests[1]		
Information	15	mild
Digit Span	11	mild
Vocabulary	50	wnl
Arithmetic	6	mild-to-moderate
Similarities	13	mild
CVLT[2]		
Trials 1–5 Total	28	moderate
Short Delay Free Recall	3	moderate
Long Delay Free Recall	1	severe
Discriminability	50%	severe
WMS–R[3]		
Logical Memory I	9	mild-to-moderate
Logical Memory II	0	moderate
Verbal Fluency[4]		
FAS	32	wnl
AFV	36	mild
Boston Naming Test[5]		
Spontaneous	52	N/A

[1]Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981). Impairment level is based on normative data from Heaton et al. (1991).
[2]California Verbal Learning Test (Delis et al., 1987). Impairment level is based on normative data from the CVLT standardization sample.
[3]Wechsler Memory Scale-Revised (Wechsler, 1987). Impairment level is based on normative data from the WMS–R standardization sample.
[4]Spreen & Benton (1969). *Neurosensory center comprehensive examination for aphasia (NCCEA)*. Victoria, BC, University of Victoria Neuropsychology Laboratory.
[5]Kaplan et al. (1978). An impairment level could not be determined because of the nonstandardized administration. See test for details.

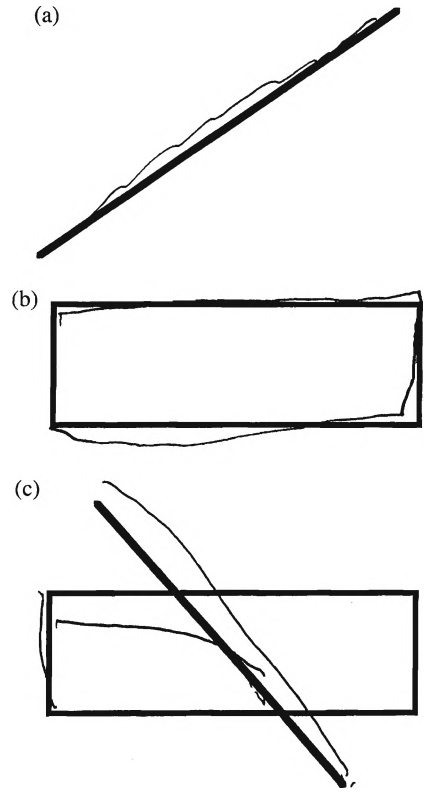


Fig. 3. M.H.'s tracing of (a) a single line, (b) a single rectangle, and (c) an overlapping line and rectangle. Note that when he traced the overlapping forms he did not appear to see the line and rectangle as separate objects.

Global-Local Tasks

Stimuli and apparatus

Global–local processing was first evaluated in a free-observation condition using pictured stimuli presented on a

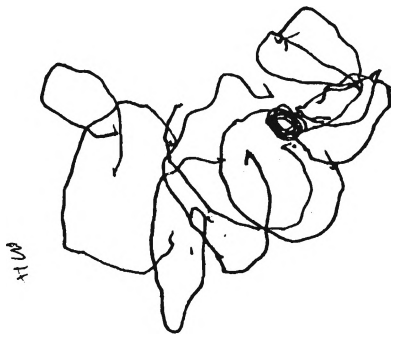


Fig. 4. M.H.'s drawing of a daisy.

piece of paper. These global and local forms consisted of the letters 'E' 'H,' and 'S.' All stimuli were incongruent in that the local form and the global form were never the same. The stimuli were black on a white piece of paper. The stimuli were in a block shape and were constructed in a 4 × 5 grid. The global stimuli were 10.5 cm in height and 7.5 cm in width. The local stimuli were 2.5 cm in height and 1 cm in width.

The stimuli for the computerized reaction time (RT) task consisted of four global–local figures: A large 'S' made up of smaller 'H's (incongruent), a large 'H' made up of smaller 'S's (incongruent), a large 'S' made up of smaller 'S's (congruent), and a large 'H' made up of smaller 'H's (congruent). The stimuli were block-shaped and were constructed in a 4 × 5 grid (see Figure 1). The global stimuli were 8.6 cm in height, 5.5 cm in width, and subtended about 12° of visual angle. The local stimuli were 1.3 cm in height, 0.8 cm in width, and subtended about 2° of visual angle. Stimuli were white on a black background and were presented on a color monitor using a personal computer. Responses were made on the computer keyboard and accuracy and RT data were recorded by the computer.

Procedure

M.H.'s global–local processing abilities were first evaluated by having him view global–local stimuli that were presented on a piece of paper. In the first few trials, he was asked to simply view the global–local stimuli and describe what he observed. On a second set of trials, he was told to attend to only the large part of the picture and describe what he observed, and in a third set of trials, he was told to attend only to the small part of the picture and describe what he observed. Ten trials were presented for each of these conditions.

We next evaluated M.H.'s global–local processing on a computerized version of the task. In this task, the patient was seated approximately 40 cm away from the computer screen, although he was allowed to move his head freely. Two conditions were presented: a global directed condition in which M.H. was told to attend to the "large" part of the picture (i.e., the global level), and a local directed condition in which he was told to attend to the "small" part of the picture (i.e., the local level). The global condition was presented first and was immediately followed by the local condition. Each trial consisted of the presentation of a global–local stimulus and was initiated by the examiner. Immediately following the initiation of a trial, a global–local stimulus appeared on the screen until a response was made or until 5 s had elapsed. M.H. was told that the target stimuli were letters and was asked to press one computer key if he observed an 'S' at the attended level (i.e., the global or the local level, depending on the condition) or another key if he observed an 'H' at the attended level. Each stimulus was presented 20 times and, as such, there were 40 congruent stimuli and 40 incongruent stimuli. The stimuli were presented in a predetermined random order.

RESULTS

Free Observation Global–Local Task

During the free-observation global–local task, in which M.H. was presented global–local stimuli on pieces of paper, he was unable to detect the global target under any condition. Specifically, when shown the stimuli and asked to simply report what he observed, M.H. reported seeing only the local form and did not report seeing the global form at all. In the global directed attention condition, he was unable to identify any of the forms on the 10 trials. In contrast, he correctly identified the form 9 of 10 times in the local directed condition, indicating that he was able to see and identify the local form fairly accurately.

Global–Local Reaction Time Task

As in the free-observation condition, M.H. was totally unable to identify the form in the global directed attention condition on our computerized task, so this condition was terminated after only 20 trials. Thus, it appeared that M.H. was completely unable to consciously report the form at the global level.

In contrast, his overall accuracy in the local directed condition was 64 out of 80 (80%). M.H.'s accuracy in identifying global level targets, however, depended on whether the stimulus was congruent or incongruent (see Table 2). Specifically, for congruent trials he was accurate on 36 out of 40 trials (90%), whereas for incongruent trials he was accurate on 28 out of 40 trials (70%). This difference in accuracy for congruent and incongruent stimuli was significant (Fisher's Exact Test = 5.00, $p < .05$). Thus, from an accuracy standpoint, M.H.'s ability to detect the local form was adversely affected by the incongruent global forms, despite the fact that he was unable to consciously report the global level form in the previous conditions.

This difference in perceiving congruent and incongruent stimuli was also observed in M.H.'s RT data. Prior to analyzing his RT data, we computed a 2 standard deviation cut-off for both the congruent and incongruent stimulus trials, and excluded any trial that fell outside of this range.

Table 2. Reaction time (ms) and accuracy rates (percent correct) for patient M.H. and normal controls (NC) for congruent and incongruent trials on the computerized global–local task. Numbers in parentheses are standard deviations.

	Congruent	Incongruent	Interference effect
M.H.			
RT	1508 (355)	1746 (495)	−238
Accuracy	90%	70%	20%
NC			
RT	552 (46)	577 (46)	−25
Accuracy	100%	100%	0%

This resulted in the exclusion of two congruent trials and one incongruent trial. The mean RT for the congruent trials was 1508 ms ($SD = 355$) and was 1746 ms ($SD = 495$) for the incongruent trials (see Table 2). These data were analyzed using an independent-sample T test, thus, each trial was treated as an independent observation. This T test indicated that M.H. was significantly slower to respond on incongruent trials than congruent trials [$t(59) = 2.2$, $p < .05$]. Thus, similar to his accuracy performance, it appeared that M.H.'s speed in responding to local level targets was slowed by incongruent global forms, in that he displayed a reliable interference effect.

In order to determine if M.H.'s interference effect was similar to other individuals his own age, we ran 4 normal control (NC) participants on the local directed attention condition. These NC participants were screened for any history of neurological or psychiatric conditions. Their average age was 63.5 years (range: 54–70 years). None of the NC participants erred in the task, so only RT data were analyzed. Their mean RTs for the congruent condition was 552 ms ($SD = 46$) and 577 ms ($SD = 46$) for the incongruent condition (see Table 2). This difference was statistically significant based on a paired-sample t test [$t(3) = 6.6$, $p < .01$]. Therefore, like patient M.H., the NC participants demonstrated a reliable interference effect.

Solid Stimuli

In order to help determine if M.H.'s deficits in consciously processing global level forms were due to the size of the stimuli, we presented him with single solid large letters that were the same dimensions as the global forms in the paper-version of the test, and single solid small letters that were the same dimensions as the local forms. The stimuli were either 'S's or 'H's. Ten small and 10 large stimuli were presented. M.H. was 100% accurate in identifying both the small and the large stimuli. His latency in identifying the large stimuli, however, was somewhat longer.

DISCUSSION

This study examined a patient with progressive visual cognitive disturbances that included impairments in object identification, visual matching, and visual construction. Tests of more basic visual functions indicated that his visual acuity was intact, but he did have problems with eye movements, saccadic pursuits, and optokinetics. MRI scans demonstrated that M.H. had substantial atrophy in the posterior cortical regions (see Figure 2). From a behavioral standpoint, M.H. displayed many of the same visual cognitive deficits as other patients with presumed PCA. Of primary interest to the present discussion was M.H.'s deficit in seeing more than one object at a time (i.e., his simultanagnosia). This impairment was displayed in a number of ways, including in his ability to describe a visual scene, count dots on a piece of paper, and identify degraded letters.

Consistent with M.H.'s simultanagnosia, the results of global–local tasks indicated that M.H. had a profound impairment in processing larger, global forms, whereas he was considerably more accurate in processing local targets. In fact, he was unable to identify any global forms under any of the conditions. Thus, M.H. had severe difficulty in consciously perceiving global information. Nevertheless, when his attention was directed to the local level on a computerized RT task, the nature of the global form affected his ability to identify the target at the local level in that he was significantly slower and less accurate when the stimuli were incongruent as compared to congruent. Thus, M.H. displayed a significant interference effect that was similar to (if not larger than) that exhibited by normal controls. These results suggest that M.H. perceived the global form, albeit at an implicit level.

The major question that arises is, what is the mechanism that enabled the significant global interference in M.H., but did not enable him to consciously perceive the global information? One possibility is that M.H.'s cortical atrophy resulted in a restriction in the spotlight of visual attention. This notion has been raised by Coslett et al. (1995) to explain their findings with PCA patients. Further, Stark et al. (1997) invoked the notion of a reduced spotlight of attention to explain the visual cognitive deficits observed in their patient N.J., who also displayed progressive visual impairments consistent with PCA. This explanation assumes that attention acts like a beam or zoom-lens (Eriksen & St. James, 1986; Posner, 1980) that can move over the visual field. Objects that fall within this beam are then selected for further processing. Based largely on their findings that patients with PCA are better at visually identifying smaller objects as compared to larger objects, Coslett et al. (1995) and Stark et al. (1997) have suggested that patients with PCA have a narrowed attentional spotlight. This explanation of PCA patients' deficits could also explain M.H.'s impairment in consciously perceiving the global form, in that a restricted attentional spotlight would enable M.H. to see only one (or perhaps two) local forms at any given time and would not permit him to consciously integrate the local forms into a gestalt. This could be the case especially if we are to assume that one of the primary roles of attention is to select information for further *conscious* processing. That is, if a patient had experienced a narrowed attentional beam, as opposed to simply a restriction in the visual field, then one would anticipate that such patients would have difficulty in consciously perceiving global level forms, but could still perhaps demonstrate some evidence of unconscious or nonattentive processing of such information.

Although this is a viable explanation of M.H.'s global–local processing results, there are some limitations to this interpretation. First, it did not appear that size *per se* could entirely explain the global–local findings, in that M.H. was able to identify larger, solid forms that were the same size as the global forms in the paper version of the global–local task. Although M.H. was somewhat slower to identify larger, solid figures as compared to smaller, solid figures (a find-

ing that supports a narrowed spotlight explanation), his complete inability to identify global level targets is clearly out of proportion to his simply being slower to identify larger, solid stimuli. Therefore, the absolute size of the stimulus could not be the sole explanation.[3] Further, M.H. was unable to identify the global form when the stimuli were presented to him without a time limit (i.e., during the free observation condition). If M.H.'s impairment were due simply to a reduced attentional spotlight, he should have been able to compensate by moving the focus of his attention around in the untimed condition, much the same way a patient with a visual field cut is able to compensate by moving his or her head around. Second, the spotlight metaphor of attention has not always been successful in explaining normal and abnormal global–local processing (see Robertson, 1996). For example, Filoteo et al. (2001) found that damage to the temporal–parietal area resulted in qualitatively different patterns of impairment on a global–local shifting task as compared to a spatial orienting task, where it is presumed that attention acts like a spotlight (Posner, 1980). Therefore, an explanation of M.H.'s performance based entirely on a narrowed spotlight of attention is somewhat debatable.

Another possible explanation for the pattern of M.H.'s findings is that a deficit in attentional disengagement accounted for his results. Farah (1990), for example, has argued that patients with dorsal simultanagnosia are impaired in perceiving more than two objects at once because they are unable to disengage their attention away from one object in order to see the other object. As Farah (1990) pointed out, such an explanation does not argue that there is a general reduction in visual attentional processes *per se*, but that normal attention becomes overly fixated on a single object, and the other objects are not perceived. Such an impairment could also account for M.H.'s global processing deficit in that it is possible that when viewing global–local stimuli, his attention becomes fixated on one local stimulus, and he does not integrate the other features because they are not overtly processed. Note that this explanation does not necessarily require a reduction in the attentional spotlight or an impairment in attentional binding. Instead, it is possible that M.H. could not overtly identify the global form because he never consciously attended the requisite local forms that composed the global form. Although this is an attractive explanation, it does not explain why M.H. had problems identifying visual objects, such as those on the visual confrontation naming task. The process of object identification is not typically thought of as requiring a disengage and shift of attention. As such, problems in the movement of attention should not impact simple object detection.

A third, and perhaps more plausible, explanation of M.H.'s explicit global processing impairment is that he was impaired in some form of attentional binding. This interpretation is related to the narrowed spotlight hypothesis, in that both explanations argue that attention serves to bind features together. The binding hypothesis is based largely on Treisman's feature integration model, in which the primary role of attention is to bind or "glue" features into objects (Treisman & Gelade, 1980). The notion of a deficit in binding information has been used to explain the visual cognitive deficits in other patients with focal lesions who display simultanagnosia (e.g., Friedman-Hill et al., 1995; Wojciulik & Kanwisher, 1998). For example, Wojciulik and Kanwisher (1998) evaluated R.M., a patient with simultanagnosia secondary to bilateral parietal lesions, on tests that examined the ability to integrate visual features. These investigators found that R.M. was unable to explicitly integrate the color of a word with the actual word itself, indicating that the patient had problems in binding two features of visual information. In contrast, however, R.M. displayed normal implicit binding of these visual features when tested under a Stroop-like condition. Specifically, R.M. was slower to identify the color of a word when it was incongruent with another word presented in the display, compared to when the color of the word was congruent with the other word in the display. These results indicated that R.M. implicitly bound the color of one word with the form of another word, despite the fact that this patient could not consciously report such binding. Based on these findings, Wojciulik and Kanwisher (1998) concluded that the parietal lobes are responsible for the explicit binding or integration of visual information. Such an interpretation could also account for our findings in patient M.H. That is, it could be that damage to posterior brain regions resulted in a deficit in explicit feature integration.[4] In the case of M.H., however, the deficit would be in binding local elements together rather than in binding two different features, such as color and shape. This interpretation of M.H.'s deficits would also help explain why he had problems in object identification on the visual confrontation naming task. A deficit in feature binding has been used to explain other patients' deficits in object identification (Riddoch & Humphreys, 1987; Shelton et al., 1994). Interestingly, a characteristic feature of the patients in these past studies was the inability to identify the overall form of an object but rather a predilection to focus on the details.

[3] It should be noted, however, that Stark et al. (1997) found that their patient with progressive visual disturbances displayed faster responding to smaller, solid letters as compared to larger, solid letters on a true RT task, and that this was somewhat opposite to the pattern observed in their controls. We continue to feel, however, that M.H.'s complete inability to consciously identify global forms as compared to his slowness in identifying larger forms is so greatly out of proportion, that it argues against a simple size account of the present findings. It is also important to note that Coslett et al. (1995) have distinguished between simultanagnosia, which is not impacted by the size of the visual stimulus, and "attentional restriction agnosia," which is affected by the size of the visual stimulus. If patient M.H. is not impacted by the size of the stimulus, as we suggest, his deficits are best described as a simultanagnosia.

[4] Interestingly, R.M. has also been reported to show normal global interference when asked to identify local level targets, despite the fact that this patient is impaired in explicitly identifying global targets (Egly & Robertson, reported in Rafal, 1997). This finding further suggests that some sort of binding deficit could account for the pattern of global–local processing displayed by patient M.H. in the present study.

Although the possibilities described above could account for M.H.'s inability to consciously perceive global level forms, the question that remains is what is the cognitive process that enabled M.H. to implicitly process global level information, but not consciously perceive such information. One possible explanation is based on the notion that some features can be processed as a whole prior to attention. These preattentive explanations of feature binding have been based primarily on studies with normal individuals. For example, previous studies have demonstrated that, under certain conditions, normal participants perceive features as unitized objects when attention is not directed at those features (see Baylis & Driver, 1992; Julesz, 1981; Moore & Egeth, 1997; Navon, 1990). These results suggest that some forms of perceptual integration can occur without attention. Preattentive processes have also been demonstrated in patients with unilateral neglect. Driver et al. (1992) found normal grouping of visual stimuli in the neglected hemifield in a patient with unilateral neglect, despite the fact that this patient denied having seen any information in that visual field. These results suggest that attention may not be necessary to integrate certain types of features, and, as such, preattentive processes could account for the relatively normal, implicit interference effects M.H. displayed with global–local stimuli. There is, however, considerable debate regarding the types of visual processes that can occur independent of attention (Lavie, 1997; Mack et al., 1992; Rock et al., 1992). Therefore, it is unknown at this time whether such preattentive processes could provide an entire account of M.H.'s normal implicit global processing.

As stated above, previous studies with bilateral parietal lesion patients found that these brain regions may be crucial for some sort of feature integration or binding (Wojciulik & Kanwisher, 1998). Therefore, it is possible that damage to these brain regions is responsible for M.H.'s impairment in the explicit processing of global level forms. Indeed, neuropathological studies by Hof and colleagues (Hof et al., 1989, 1990, 1991, 1993; Hof & Bouras, 1991) of patients with PCA and Balint's syndrome (which includes simultanagnosia) have indicated that the primary distribution of pathology in these patients is in occipital, occipital–parietal, and occipital–temporal regions. Given that the neuropathology associated with Balint's syndrome in focal-lesion patients appears to be bilaterally in the occipital–parietal junction (Coslett & Saffran, 1991; Pierrot-Deseillgny et al., 1986; Rafal, 1997), it is likely that the Balint's syndrome that appears in patients with PCA (such as patient M.H.) is due to a similar distribution of pathology.

The neuroanatomical basis of M.H.'s normal, implicit global processing is less clear. Other studies of neurological patients who demonstrate visual perception without awareness have suggested two possibilities. First, it is possible that different neuroanatomical regions mediate conscious and unconscious visual processes. For example, in cases of blindsight where patients with severe occipital lobe damage display residual visual abilities under implicit testing conditions, several investigators have suggested that other brain regions, such as subcortical visual pathways, may mediate the residual visual abilities (Cowey & Stoerig, 1991). In the case of M.H., it may be that brain regions not impacted by PCA are responsible for his normal implicit processing of global information. One potential candidate could be the occipital lobes, which have been implicated in certain binding processes (Grossberg et al., 1997; Sugita, 1999). Indeed, a recent functional activation study implicated the occipital lobes in the processing of global–local stimuli (Fink et al., 1997). However, given the extent of M.H.'s pathology at the time of our testing (see Figure 2), and the distribution of neuropathology found in other patients with PCA (which includes primary visual cortex), it is difficult to determine if the occipital lobes are intact enough to enable such processing. Another possible candidate brain region is the temporal–parietal junction. Lamb et al. (1989) found that patients with focal lesions of the temporal–parietal region did not display normal interference effects when the participants were required to direct their attention to the global or the local level. This finding suggests that these brain regions may be involved in the interference effect displayed by normal individuals, and in as much as M.H.'s pathology did not extend to this area, it is possible that this brain region was responsible for his normal implicit processing of the global information.

The second possibility in regard to the neuroanatomical basis of M.H.'s normal implicit processing of global information is that damage to a single brain region in M.H. resulted in an impairment in explicit global processing but not implicit processing. That is, one brain region (perhaps the occipital–parietal association cortex) is responsible for both implicit and explicit processing of global information, and the likely damage to this region in patient M.H. impacted one process (explicit global processing), but left the other process (implicit global processing) intact. Although it is difficult to determine if this is the case in patient M.H., other investigators have used this approach to explain other cases of implicit visual perception. For example, Campion et al. (1983) suggested the possibility that spared regions of the occipital lobes could account for the residual visual abilities in patients with blindsight. In addition, Farah et al. (1993) have presented evidence based on computational models that damage to a single neural system can result in a dissociation between implicit and explicit facial processing. Finally, a recent fMRI study by Rees et al. (2000) examined visual processes in a patient who displayed extinction to double simultaneous stimulation. These investigators found normal activation in visual cortex contralateral to the side of a stimulus that had not been consciously perceived, suggesting that the visual stimulus had been processed normally in that brain region despite the fact that the patient was unaware of the stimulus. Taken together, these studies suggest that it may not be the case that a nondamaged brain region mediated the normal, implicit global processing observed in patient M.H., but that damage to a single region resulted in this dissociation.

Although M.H. displayed what appears to be normal global interference, it is important to point out that not all patients with progressive visual disturbances have displayed this profile. Specifically, Stark et al. (1997) examined congruency effects in a patient with progressive visual disturbances using a global–local divided attention task. Their patient, N.J., was impaired in processing global level information and also displayed simultanagnosia. In their task (Experiment 5), participants were asked to identify at what level (global or local) a specific target appeared within a global–local stimulus. The stimuli were either congruent or incongruent, and were presented under a global bias condition (i.e., targets appeared mostly at the global level) or a local bias condition (i.e., targets appeared mostly at the local level). In the case of the congruent trials, the correct response was either global or local. The critical comparison for this present discussion was between congruent trials and incongruent trials when the target was at the local level under the local biased condition. These trials are most like the congruent and incongruent trials in the present study, and if their patient displayed a similar congruency effect as patient M.H., RTs should have been larger in the incongruent condition as compared to the congruent condition. Stark et al. (1997), however, did not observe this pattern, in that patient N.J. did not display any RT differences in the congruent and incongruent trials. Thus, their patient did not display the same pattern as M.H. in the present study.

There are a few differences between our study and that of Stark et al., however, that could account for this discrepancy. First, Stark and colleagues utilized a divided attention condition in which participants had to attend to both the global and the local level. Divided attention global–local tasks can invoke other attentional processes, such as requiring participants to shift attention across consecutive trials (see Filoteo et al., 2001; Robertson, 1996), and these additional processes can be disrupted following posterior lesions (Filoteo et al., 2001). In contrast, participants in our study were told to focus on only one level of the stimulus (the local level) and to report what target they saw at that level. These differences in divided *versus* directed attention could possibly account for the discrepant findings. A second possibility, however, is that the patient in the Stark et al. study had pathology that extended into the temporal–parietal regions. As stated earlier, damage to these regions has been known to eliminate the interference effects observed with incongruent global–local stimuli. Obviously, future studies need to examine these possibilities more closely.

In summary, patient M.H. displayed a profound impairment in the explicit processing of global information. In contrast, his implicit processing of global information appeared to be intact. The cognitive basis of his explicit global processing deficit may be due to an impairment in the conscious binding of features into objects or a deficit in disengaging attention away from individual local features, whereas his normal unconscious processing of global information may be due to preattentive processes. The neuropathological basis of M.H.'s explicit global processing deficit may be bilateral damage to the occipital–parietal regions. The neuroanatomical substrates of M.H.'s normal implicit global processing is less clear but could be due to normal processing in the occipital lobes or temporal–parietal regions.

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