# What Processing Is Impaired in Apperceptive Agnosia? Evidence from Normal Subjects

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## Abstract

■ Visual agnosia is a neuropsychological syndrome characterized by a failure of object identification. Apperceptive agnosia, an object identification deficit caused by damage to early perceptual processes, has been explained by appealing to both damaged early sensory processes and to damaged preattentive grouping processes. Which of these two explanations best accounts for the behavior of these patients? We present results from two experiments designed to distinguish rival theoretical accounts of apperceptive agnosia. In our studies, we attempted to simulate apperceptive agnosia in neurologically intact subjects. Sensory-deficit accounts of the syndrome predict that degrading visual processing would make normal subjects perform like patients; grouping-deficit accounts predict that removing perceptual organization cues from visual displays would make normal subjects perform like patients. We were able to simulate the behavior of an apperceptive agnosic patient by removing perceptual organization cues, consistent with a grouping-deficit account of this syndrome. The implications for understanding both apperceptive agnosia and normal visual functioning are discussed. ■

## INTRODUCTION

Among the problems that the visual system must solve, object representation and identification are perhaps the most difficult. Attempts to understand object representation processes have come from several disciplines, including computational vision (e.g., Marr, 1982), cognitive psychology (e.g., Biederman, 1987; Tarr, 1995), and neuroscience (e.g., Tanaka, 1996; Tanaka, Saito, Fukada, & Moriya, 1991). Neuropsychological studies of the visual agnosias, disorders in object recognition, also have contributed to the understanding of object representation processes. One significant contribution of neuropsychological accounts of object processing has been the identification of multiple systems/processes that are important for object representation and recognition.

Most neuropsychological approaches to object representation have followed the theorizing of Lissauer (1890/1988), one of the earliest theorists of the agnosias. Lissauer suggested that there were two ways in which object recognition could be impaired following brain damage. One form of damage was to the object representations themselves, which would prevent recognition because of lost visual memory representations. The second form of damage was to earlier perceptual processing, processing Lissauer termed "apperception." Damage to the process of apperception would prevent recognition because of poor visual input to the otherwise intact object representations. These two forms of damage correspond to two types of visual agnosia that were suggested by Lissauer. Associative agnosics, Lissauer suggested, appear to have the first type of damage; they are typically characterized by an inability to recognize objects, despite having intact early-level perceptual representations. The quality of these perceptual processes typically is assessed with a copying task; associative agnosics often *can* copy visually presented objects, although they are unable to recognize those same objects. Apperceptive agnosics, in contrast, appear to have the second type of damage; they have damaged early-level perceptual processes. Unlike associative agnosics, apperceptive agnosics cannot copy visually presented objects, suggesting damage to lower-level processing. Although Lissauer's original scheme is limited in that it cannot account for all of the recent agnosic patients reported (see Farah, 1990; Grailet, Seron, Bruyer, Coyette, & Frederix, 1990; Humphreys & Riddoch, 1987; Riddoch & Humphreys, 1987), the two forms of agnosia suggested by Lissauer remain in current neuropsychological theories of object processing.

More recent theorists have explored what cognitive processes are impaired in both associative and apperceptive agnosia. Associative agnosia has been explained by postulating a disconnection between intact visual processes and verbal processes (e.g., Geschwind, 1965), as well as by postulating damage to internally stored visual memories or templates (e.g., Mesulam, 1985). Apperceptive agnosia has been explained as being caused by


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viewing the world through a "peppery mask" caused by multiple scotomas (e.g., Campion, 1987) and as a loss of perceptual grouping processes (e.g., Farah, 1990).

Within the two broadly defined forms of visual agnosia, apperceptive agnosia is particularly interesting because multiple hypotheses have arisen to explain it despite a great deal of homogeneity among these neuropsychological patients. Although the multiple accounts of associative agnosia might easily be explained by the different lesion locations and etiologies in these patients, apperceptive agnosics, when the syndrome is narrowly defined (see Farah, 1990), exhibit many similarities in both etiology and locus of damage: Most cases that have been reported have been the result of an anoxic episode, which leads to diffuse damage over posterior cortices, most notably the occipital lobes (see Adler, 1944; Benson & Greenberg, 1969; Campion & Latto, 1985; Efron, 1968; Milner et al., 1991; Vecera & Behrmann, 1997).[1]

Given this apparent homogeneity of apperceptive agnosia, a single cognitive function may be disrupted in most, if not all, of these patients. But which cognitive function? Is the syndrome better explained by suggesting it is caused by perception through a peppery mask (Campion, 1987; Campion & Latto, 1985), or is it better explained as a disruption of perceptual grouping processes (e.g., Farah, 1990; Gelb & Goldstein, 1918/1967)? To address this issue, we attempted to simulate apperceptive agnosia in neurologically normal subjects to identify the affected cognitive functions and to distinguish the various accounts of the syndrome (see Farah, Monheit, & Wallace, 1991; Miyake, Carpenter, & Just, 1994, for similar simulation approaches to neuropsychological deficits). Because the study of apperceptive agnosic patients has led to multiple explanations of the syndrome, evidence from a converging methodology may be required to overcome the limitations of this previous research. In what follows, we first discuss the rival accounts of apperceptive agnosia; we then present two experiments aimed at distinguishing the various accounts.

## Sensory-Deficit Account

One of the earliest explanations of the visual agnosias (both apperceptive and associative) was the sensory-deficit account. Several investigators argued that the agnosias are nothing more than degraded low-level visual processing; also, the deficit may be further compounded by impairments to general mental functioning. Bay (1953) held this position, noting that "to my knowledge of the literature, there does not exist a single case of agnosia without elementary sensory disorders and without mental deterioration" (p. 534). Bender and Feldman (1972) also held this position, and to support the role of impaired sensory processes in agnosia they examined all of their hospital's neurological case records appearing within a 20-year period. None of the agnosic cases they reviewed were free of either low-level sensory deficits or of general mental dysfunction, consistent with Bay's (1953) analysis of visual agnosia.

The terminology used with the sensory-deficit account is somewhat vague and lacks a cognitive mechanism that could have been damaged. This approach could be updated by appealing to spatial frequency channels (see Ginsburg, 1986; Regan, 1982); the visual agnosias could potentially be explained as a selective loss of high spatial frequency channels, which would result in a loss of perception for fine detail. Indeed, some patients with lesions to temporal lobe visual areas have difficulty performing spatial frequency discriminations (Greenlee, Rischewski, Mergner, & Seeger, 1993), and apperceptive agnosics have been found to have abnormal contrast sensitivity functions (Campion & Latto, 1985).

Although the sensory-deficit account might explain some of the neuropsychological data, this explanation of the visual agnosias is problematic because it has failed to explain distinctions within the syndrome of visual agnosia. Most importantly, a general sensory-deficit account does not explain the differences between apperceptive and associative agnosia. Failing to account for the different types of agnosia is problematic because apperceptive and associative agnosics show very different patterns of behavior, which would seem to preclude a single explanation of both types of agnosia. Other explanations have been put forward specifically for apperceptive agnosia, thereby overcoming this difficulty with the earlier sensory-based accounts.

## The Peppery Mask Account

Given the failure of the sensory-deficit account to explain differences between associative and apperceptive agnosia, more specific accounts of the agnosias were needed. One such specific account is the peppery mask account of apperceptive agnosia. Based on a patient with apperceptive agnosia, patient RC, Campion and colleagues (Campion, 1987; Campion & Latto, 1985) argued that such patients may have multiple infarcts of early visual cortices, and these infarcts cause the visual field to be covered ("peppered") with multiple scotomas of varying size and spatial distribution. The result of these scotomas is that perception is degraded as if the patient was looking through a peppery mask—that is, a mask that degrades visual processing with the presence of random visual noise.

In order to test the peppery mask hypothesis, a detailed perimetric study of patient RC's visual field was conducted; in this study, RC's visual field was divided into 800 sectors, each being 1° of visual angle (Campion & Latto, 1985). Flashes of light were presented in each of the sectors, and RC was asked to rate the perceived brightness of each flash. The results were consistent with

the peppery mask hypothesis in that RC had impaired light sensitivity throughout much of his visual field. Further, this impairment was not systematic; instead, the degraded light sensitivity varied across the visual field, as if it had been peppered with light perception impairments that differed in both position and severity.

In addition to testing patient RC, Campion and Latto (1985) tested the peppery mask account in a normal subject. This subject's contrast sensitivity function (CSF) was computed under different masking conditions. The grating stimulus was either not masked or masked with either a low or a high spatial frequency mask. Campion and Latto (1985) found that in both of the masking conditions (low and high spatial frequency), the subject's CSF looked similar to patient RC's CSF; in both there was a general flattening of the function (i.e., the CSF was less curved in the masking conditions relative to the no-mask condition). These results suggest that some aspects of apperceptive agnosia might be explained by, and simulated with, a peppery mask.

### The Grouping-Deficit Account

Although the peppery mask account seems to explain some attributes of apperceptive agnosia, this account is not without its problems. Probably the biggest hurdle for this account of apperceptive agnosia is that is offers no straightforward explanation of the perceptual grouping disorders that appear in these patients (Gelb & Goldstein, 1918/1967; Landis, Graves, Benson, & Heben, 1982; Milner et al., 1991; Vecera & Behrmann, 1997; see Farah, 1990, for further discussion). Thus, a rival hypothesis for apperceptive agnosia is the grouping-deficit account, which states that patients with apperceptive agnosia have impairments in preattentive perceptual grouping processes, including those processes outlined by the gestalt psychologists (e.g., Wertheimer, 1923/1958), and figure-ground organization (e.g., Rubin, 1915/1958).

Brain (1941) originally discussed visual agnosia in terms of perceptual grouping, and more recently, Farah (1990) specifically has asserted that apperceptive agnosia appears to be due to damaged grouping processes. Farah pointed out that these patients have profound difficulties in organizing local feature elements (e.g., edges and line segments) into larger, more coherent "wholes," and it is unclear how such a disruption could occur from the application of a peppery mask to the visual field. A more serious difficulty for the peppery mask account arises in that the type of degradation caused by such a mask would seem to increase the reliance upon global, or wholistic, information. Because this is exactly the type of information that apperceptive agnosics *cannot* use, the peppery mask account does not seem to fully explain the processing deficits in apperceptive agnosic patients.

One limitation with the grouping-deficit account is that there is little data from apperceptive agnosic patients directly examining lower-level grouping processes. In addition, the work that has previously tested perceptual grouping in these patients (see Milner et al., 1991; Vecera & Behrmann, 1997) was not aimed at distinguishing the grouping-deficit account from the peppery mask account. Thus, the grouping deficits observed in these patients could be caused by either perception through a peppery mask (caused by multiple scotomas) or by damage to the grouping processes themselves.

### A Simulation Approach

Which of these accounts of apperceptive agnosia better explains the deficits observed in apperceptive agnosic patients? The work with patients has led to an impasse: Two very different accounts of the syndrome appear to explain the disorders observed in these patients. Distinguishing the peppery mask and grouping-deficit accounts may require converging evidence from another methodology, such as from attempting to simulate the disorder in normal subjects. The behavior of other neuropsychological patients has been simulated in several studies, leading to a better understanding of the behavior, and the syndrome, simulated.

Such a simulation approach was taken by Farah and colleagues (Farah et al., 1991), who asked if the neglect of a contralesional stimulus could be caused by a failure in perceiving this stimulus. In order to test this, Farah et al. simulated neglect in normal subjects by placing drafting stock over half of a computer monitor; the covered region of the monitor was visible, yet stimuli appeared degraded. When normal subjects were shown stimuli in the intact field, there were no differences between perceiving the presence of the stimulus and identifying the stimulus; when stimuli were shown in the degraded ("neglected") field, subjects behaved like neglect patients, in that perceiving the presence was much easier than was identification. These results suggest that the dissociation between perception and identification in neglect patients may simply be caused by different thresholds for these two different tasks. These results were beneficial to the interpretation of the neglect syndrome because they demonstrate that one does not need to appeal to conscious awareness in order to explain some results from neglect patients. This study also demonstrated that simulating a neuropsychological disorder in normal subjects can contribute to an understanding of that disorder.

We have taken a similar approach in the present study to distinguish the various accounts of apperceptive agnosia. We had normal subjects perform a spatial cuing task that measures two separate visual processes, spatial attention processes and lower-level grouping processes (see Egly, Driver, & Rafal, 1994; Vecera, 1994). A recent case study of a patient with apperceptive agnosia, patient JW, used this paradigm and found a dissociation between these processes; spatial attention was intact in

patient JW and the earlier visual processes were impaired (Vecera & Behrmann, 1997). Although these results suggested a grouping impairment in JW, the Vecera and Behrmann study did not directly test between the peppery mask and grouping-deficit accounts, necessitating the present study. To simulate apperceptive agnosia, we combined this spatial cuing task with (1) manipulations in which displays were viewed through a peppery mask and (2) manipulations that would impair the low-level grouping of the displays.

The task, depicted in Figure 1, required subjects to detect the onset of a spatially cued target. The cue, which precedes the target, can be predictive of the target's location (a valid cue) or unpredictive of the target's location (an invalid cue). In this paradigm, as with most studies of spatial attention, subjects' reaction times (RTs) are faster to detect validly cued targets than invalidly cued targets (Egly et al., 1994; Vecera, 1994; see Posner & Cohen, 1984; Posner, Snyder, & Davidson, 1980, for more general cuing results). In addition, there are lower-level grouping effects that modulate spatial attention in this task. There are two types of invalidly cued targets, those that appear in the cued rectangle and those that appear in the uncued rectangle (see Figure 1c). In these trials, subjects are faster to detect invalidly cued targets appearing in the cued rectangle than those appearing in the uncued rectangle, *even though these targets are the same spatial distance from the previously cued location.* This grouping effect is thought to be due to preattentive grouping processes influencing the allocation of spatial attention (Vecera, 1994).

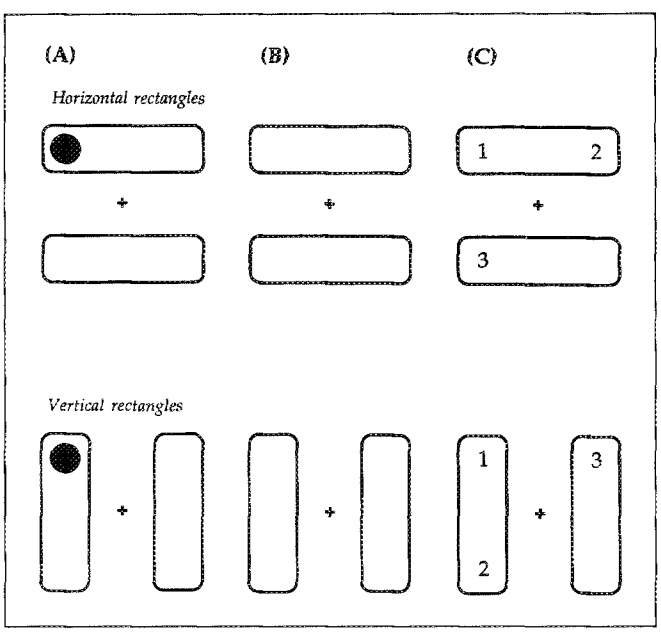


**Figure 1.** The structure of the task; fully intact rectangles appear for illustration only. (a) A cue first appears at the end of one of the rectangles. (b) The interstimulus interval. (c) A target then appears in one of three possible locations. A validly cued target appears at the cued corner and is depicted in Location 1. An invalidly cued target appearing in the cued rectangle is depicted in Location 2, and an invalidly cued target appearing in the uncued rectangle is depicted in Location 3.

As noted previously, patient JW, who has apperceptive agnosia, showed a dissociation between the two processes measured with this task, spatial attention processes and low-level grouping processes. JW showed normal spatial orienting in that he was faster to detect validly cued targets relative to invalidly cued targets. By contrast, the grouping component was abolished in JW: He was equally fast to detect invalidly cued targets appearing in the cued rectangle and invalidly cued targets appearing in the uncued rectangle (Vecera & Behrmann, 1997). Any successful simulation of apperceptive agnosia using neurologically normal subjects would need to account for both of these findings.

We conducted two experiments in order to test the peppery mask and grouping deficit accounts of apperceptive agnosia. (Note that elsewhere we have discussed these studies in a broader context; see Vecera & Gilds, 1997.) In Experiment 1 we tested the peppery mask account. Subjects performed the task while viewing unobscured displays (e.g., Figure 1) or while viewing displays that were covered with a peppery mask similar to that shown in Figure 2. If the peppery mask account of apperceptive agnosia is correct, the normal subjects' data should look similar to JW's data when the displays are viewed through the mask; when the displays are unmasked, the normal pattern of results should manifest themselves. In Experiment 2 we tested the grouping-deficit account. In this study, subjects performed the spatial cuing task while viewing displays that had nonaccidental properties deleted. Nonaccidental properties are properties that aid in the organization of the visual

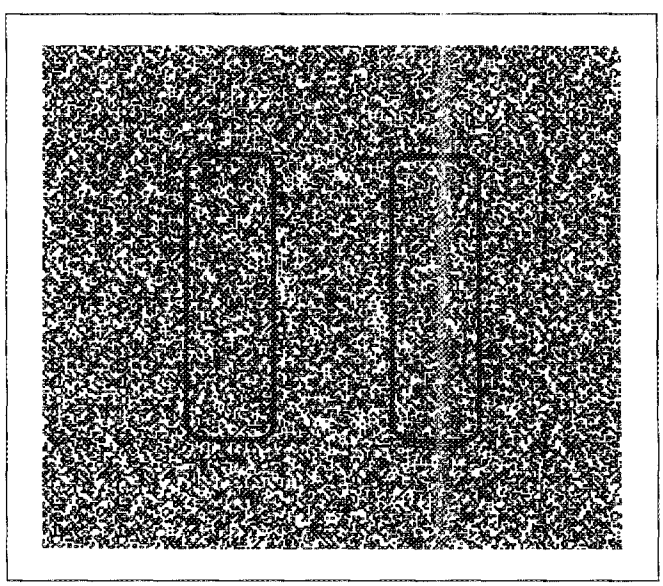

**Figure 2.** An example of the masked displays used in Experiment 1. The peppery mask is based upon published examples (e.g., Campion, 1987).

field (Lowe, 1985, 1987); removing these properties should impair low-level grouping processes. If the grouping-deficit account of apperceptive agnosia is correct, the normal subjects' data should look similar to JW's when the displays have had nonaccidental properties removed; when the displays contain these nonaccidental properties, the normal pattern of results should be obtained.

## EXPERIMENT 1

The peppery mask account of apperceptive agnosia was tested in Experiment 1. Subjects performed Egly et al.'s (1994) spatial cuing task while viewing clear displays or while viewing displays that had been covered with a peppery mask. We created our mask by examining published versions of Campion's mask (e.g., Campion, 1987; Campion & Latto, 1985); using image processing software, we generated a field of pixels that were randomly distributed about the display. This peppery field of pixels was then printed and converted to an overhead transparency; this transparency was placed in front of the computer monitor during the masked trials. This procedure was used because superimposing the mask on the stimuli in the computer's image file resulted in slow display times, making the masked displays appear slower than the clear displays.

We anticipated that the results from the clear displays would replicate previous results from neurologically intact subjects using this task (Egly et al., 1994; Vecera, 1994). Specifically, we should see faster RTs to validly cued targets than to invalidly cued targets. We should also see faster RTs to invalidly cued targets that appear in the cued rectangle relative to those that appear in the uncued rectangle. The results from the masked displays will provide the critical test of Campion's account of apperceptive agnosia. If a peppery mask is the correct mechanism for explaining apperceptive agnosia, our normal subjects' data should look similar to that of an apperceptive agnosic patient who was tested on this task (patient JW; Vecera & Behrmann, 1997). For the peppery mask account to be correct, two results should be obtained with the masked displays: (1) RTs to validly cued targets should be faster than RTs to invalidly cued targets (i.e., there should be a normal validity effect), and (2) RTs to invalidly cued targets appearing in the cued rectangle should not differ statistically from RTs to invalidly cued targets appearing in the uncued rectangle (i.e., there should be an absence of a grouping effect). The apperceptive agnosic patient tested by Vecera and Behrmann (1997) showed both of these effects.

### Results

To prevent subjects from responding to the cue and not to the target, 20% of the trials were "catch trials" in which no target appeared. Subjects were required to withhold their responses to these trials. Subjects who responded to more than 10% of the catch trials were excluded from the analyses because of these high false alarm rates. Two of the original 22 subjects tested were excluded from the analyses because of high false alarm rates on catch trials in which no target was presented (a mean 17% false alarms). For the remaining 20 subjects, any reaction times RTs that were over 1000 msec or less than 100 msec were excluded; this trimming eliminated less than 1.9% of the data. For each subject, the median RT for each condition was calculated, and these median RTs were then analyzed with a within-subject analysis of variance (ANOVA).

Mean RTs for all conditions appear in Figure 3. The results from the clear (nonmasked) displays provide a replication of previous results from normal subjects. Valid versus invalid trials were first analyzed with a

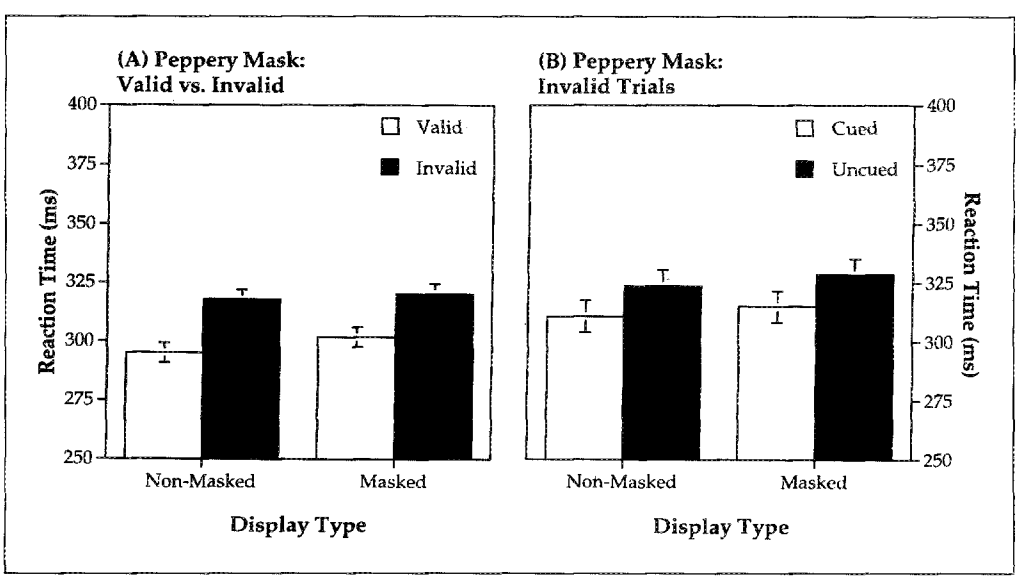


**Figure 3.** Results from Experiment 1. (a) Valid versus invalid trials. Subjects are faster to detect validly cued targets relative to invalidly cued targets, irrespective of the masking manipulation. (b) The invalid trials. Subjects are faster to detect invalidly cued targets appearing in the cued rectangle than those appearing in the uncued rectangle. This effect holds for both masked and nonmasked displays, a result that does not replicate the results observed in apperceptive agnosia. (Error bars are within-subject 95% confidence intervals; see Loftus & Masson, 1994.)

two-factor ANOVA, with factors being display quality (clear versus masked) and cue type (valid versus invalid). This analysis revealed a main effect of cue type, $F(1, 19) = 24.29, p < 0.0001$, with RTs to validly cued targets being faster than RTs to invalidly cued targets (298.91 msec versus 318.96 msec, respectively). There was neither a main effect for display quality, $F < 1$, nor an interaction between cue type and display quality, $F < 1$. Planned comparisons showed that the validity effect was significant for both the clear displays, $t(19) = 8.08, p < 0.0001$, and for the masked displays, $t(19) = 6.56, p < 0.0001$. These results appear in Figure 3a, and they replicate the standard spatial cuing effects observed in normal subjects (Egly et al., 1994; Vecera, 1994) and in patient JW, who has apperceptive agnosia (Vecera & Behrmann, 1997).

Next, the invalid trials, shown in Figure 3b, were analyzed with a two-factor ANOVA, with conditions being display quality (clear versus masked) and target location (target in the cued rectangle versus target in the uncued rectangle). The main effect for target location was statistically reliable, $F(1, 19) = 37.42, p < 0.0001$, with RTs to targets in the cued rectangle being faster than targets appearing in the uncued rectangle (312.85 msec versus 326.19 msec, respectively). The main effect for display quality was not significant, $F < 1$, suggesting that RTs were similar for the masked and clear displays (317.24 msec for clear displays and 321.80 msec for masked displays). These two factors did not interact with one another, $F < 1$. Planned comparisons revealed that RTs to targets appearing in the cued rectangle were faster than RTs to targets appearing in the uncued rectangle for clear displays, $t(19) = 2.87, p < 0.01$ and for masked displays, $t(19) = 3.01, p < 0.008$. Both display types produced the normal effect on invalid trials that has been observed in previous studies (Egly et al., 1994; Vecera, 1994).

### Discussion

There are several findings of theoretical importance from Experiment 1. Recall that a successful simulation of apperceptive agnosia would need to find two particular results in this task: (1) a normal validity effect and (2) a lack of a low-level grouping effect. A reliable validity effect was obtained for both the masked displays and the clear displays. In the clear displays, this result replicates previous findings with this task (Egly et al., 1994; Vecera, 1994). In the masked displays, the validity effect replicates JW's validity effect, suggesting that apperceptive agnosia may indeed be the result of perception through a peppery mask. The peppery mask only simulated JW's results in the valid versus invalid comparison; the peppery mask did not fully simulate JW's behavior. This conclusion arises from the comparison between the two types of invalidly cued targets. In the clear displays, our normal subjects again replicated previous findings. However, this grouping effect was also replicated in the masked displays, a result problematic for the peppery mask account of apperceptive agnosia. If apperceptive agnosia were due to perception through a peppery mask, the normal subjects, when viewing masked displays, should have shown results similar to those from JW. JW showed no differences between invalidly cued targets appearing in the cued rectangle and those appearing in the uncued rectangle. The invalid trials from our normal subjects looked quite different from JW's data; the present results show reliably faster RTs to invalid trials appearing in the cued rectangle than to those appearing in the uncued rectangle.

Based on these results, we concluded that the peppery mask account does not fully explain the constellation of results observed when an apperceptive agnosic patient performs this spatial cuing task. Of course, there may be other explanations of our results. One might argue that our peppery mask was not peppery enough to simulate apperceptive agnosia. This critique seems warranted given our failure to find a statistically significant difference between the masked displays and the clear displays. To address this shortcoming, we performed an additional analysis. We first computed a "masking coefficient" for each subject. This coefficient was simply the RT difference between the masked and clear displays for the validly cued targets. A large masking coefficient would suggest that the mask was quite effective for a subject (i.e., effectively impaired processing), and a smaller masking coefficient would suggest that the mask was not as effective (i.e., not as impairing) for a subject. Next, using simple linear regression, this masking coefficient was then used to predict the size of the grouping effect for the masked displays. If our failure to simulate JW's grouping effect was due to a weak masking manipulation, the masking coefficient should be negatively related to the grouping effect (masked displays only). That is, as the masking coefficient increases (indicating a greater masking effect), the grouping effect should decrease, suggesting that if we had used a more peppery mask, we could have simulated the absence of a grouping effect in apperceptive agnosia. As shown in Figure 4, the results from this regression failed to find a statistically reliable relationship between the masking coefficient and the grouping effect, $r = -0.143$, $t(19) < 1$. Although this correlation was slightly negative, the masking coefficient only accounted for 2.1% of the variability in the grouping effect, $R^2 = 0.021$. Our failure to simulate the grouping effect in apperceptive agnosia does not appear to be caused by the particular peppery mask that we employed in this study.

Another argument concerning the results from Experiment 1 is that our mask did not mask low-level vision in the same way as the masking that occurs in apperceptive agnosia.[2] These neuropsychological patients may indeed best be described with the peppery mask account, but with a mask that degrades some spatial frequency chan-

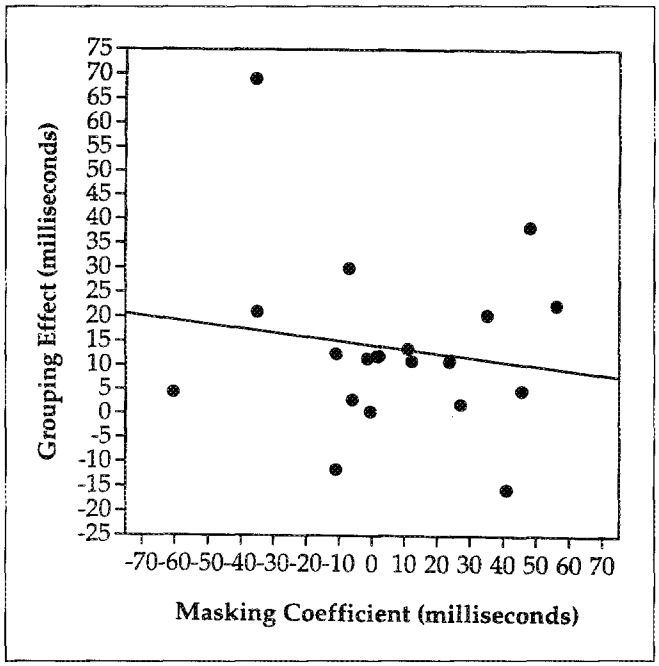


**Figure 4.** Regression analysis from Experiment 1. The masking coefficient does not reliably predict the size of the grouping effect. See text for additional discussion.

nels more than others. Low spatial frequencies are known to contribute to some types of organizational processing, including apparent motion (Ramachandran, Ginsburg, & Anstis, 1983; see Ginsburg, 1986). Might an appeal to spatial frequency channels explain the grouping problems that are observed in our study, as well as in apperceptive agnosia? It could be the case that the peppery mask in the patients has little, if any, low spatial frequency information because the channels that carry this information have been damaged. The loss of these channels may result in grouping impairments, although the grouping processes themselves are intact. This analysis is consistent with some observations of apperceptive agnosic patients, who show flattened contrast sensitivity functions (Campion, 1987; Campion & Latto, 1985). Although we used published versions of a peppery mask (Campion, 1987; Campion & Latto, 1985) as the basis of our mask, slight differences could have prevented us from replicating the lack of a grouping effect observed in patient JW.

Two points argue against this possibility. First, although some have argued for the importance of spatial frequency channels in perceptual organization (e.g., Ginsburg, 1986; Watt, 1988), filtering different spatial frequencies out of an image does not appear to impair grouping (Jáñez, 1984; Palmer, 1992). Thus, an appeal to spatial frequency channels does not appear to fully explain grouping phenomena. In terms of our studies, it seems unlikely that spatial frequency information can fully explain the results from apperceptive agnosic patients such as JW. Second, we have collected data indicating that spatial frequency information does not abolish the grouping effect in this spatial cuing task. We first high-pass filtered our original peppery mask.[3] This procedure removed low spatial frequencies, leaving only the higher frequencies; if apperceptive agnosia is due to a loss of low spatial frequency information, then the results from the new mask should better simulate JW's performance.

Despite using this new mask, the results from three neurologically normal subjects were qualitatively similar to the results from Experiment 1: In the masked condition, a validity effect was again obtained (295.68-msec valid trials versus 327.14-msec invalid trials), and a reliable grouping effect was also found (321.89 msec in cued shape versus 332.40 msec in uncued shape). Thus, we conclude that spatial frequency information is not the sole determinant of perceptual grouping effects in either normal observers (Jáñez, 1984; Palmer, 1992) or apperceptive agnosic patients. To further address this issue, we next high-pass filtered the actual displays used in Experiment 1 (Figure 1).[4] These filtered displays were occluded with the original peppery mask. If the spatial frequency analysis is correct, the results should simulate those of patient JW. Again, however, the results were qualitatively similar to those from Experiment 1. Neither of these spatial frequency manipulations abolished the grouping effect in this task, which fails to simulate the behavior observed in apperceptive agnosia.

Having compromised the peppery mask account of apperceptive agnosia, we tested the grouping-deficit account of apperceptive agnosia in Experiment 2. The logic is identical to that used in Experiment 1. We employed a manipulation that would remove perceptual grouping cues and tested neurologically intact subjects with these displays. If the grouping account explains the behavior of apperceptive agnosic patients, then we should observe a reliable validity effect, but we should *not* see a grouping effect in neurologically intact subjects.

## EXPERIMENT 2

As in Experiment 1, we degraded the displays used in the spatial cuing paradigm. The degradation was designed to remove salient grouping cues from the displays; such degradation can be achieved by removing *nonaccidental properties* from the displays. Nonaccidental properties are heuristics such as cotermination and parallelism that can be used to organize or group the visual field (see Lowe, 1985, 1987). These properties, when present in the visual image, are likely to be true of the external world; that is, these properties are unlikely to be due to chance or to an accidental viewing position (hence the name *nonaccidental properties*). Nonaccidental properties have been useful in implementing perceptual grouping in computer vision systems (Lowe, 1985, 1987). These properties also seem important in normal human vision; removal of nonacci-

dental properties hinders object recognition (Biederman, 1987), possibly due to inadequate visual input to object representations.

In the present displays, 50% of the pixels were removed, and this removal was performed in one of two locations on the rectangles as shown in Figure 5. In the Corners Only condition, pixels were removed from the midsegments of the rectangles (Figure 5a), and in the Midsegments Only condition, pixels were removed from the corners of the rectangles (Figure 5b). The Midsegments Only condition corresponds to a removal of one of the nonaccidental properties present in these displays—the cotermination of the line segments in the rectangles. Cotermination cues are present in the Corners Only displays.

Given the results from Experiment 1, we anticipated that both types of degradation would result in a validity effect—validly cued targets should be detected faster than invalidly cued targets. The critical test of the grouping-deficit account comes from the responses to the invalidly cued targets. The grouping-deficit account would predict that differences should exist between the two types of degradation. Specifically, removal of nonaccidental properties should hinder performance much more than removal of other features; in the present displays, removal of the corners would hinder performance more than removal of the midsegments. With respect to the invalid trials in the present task, removal of the corners (Midsegments Only condition) would impair grouping, thereby eliminating differences between invalidly cued targets appearing in the cued rectangle and those appearing in the uncued rectangle. Such results would replicate the results from patient JW. However, removal of the midsegments (Corners Only condition) would leave intact those grouping cues (i.e., cotermination cues) needed to establish the rectangles as separate perceptual groups, thereby allowing for the normal differences between the two types of invalidly cued targets—invalid targets appearing in the cued rectangle would be detected more quickly than those appearing in the uncued rectangle.

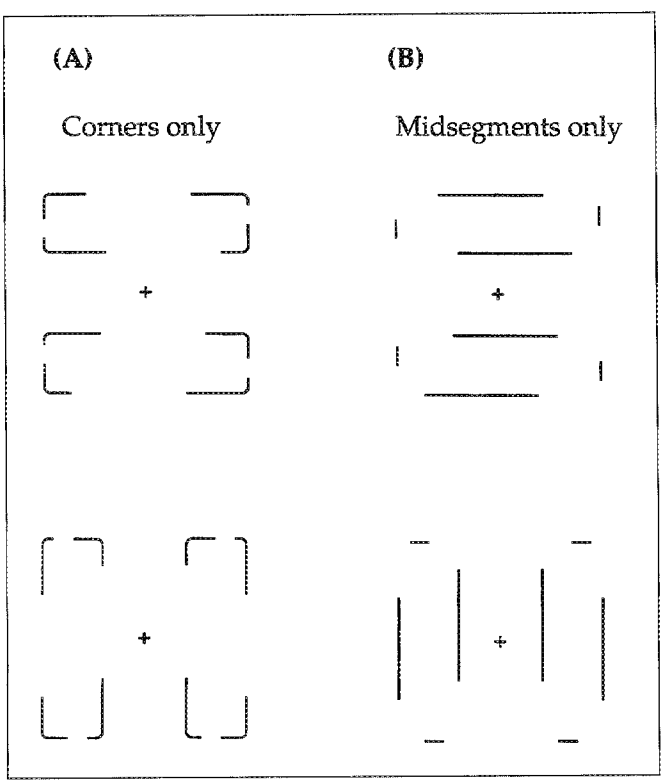


**Figure 5.** The displays used in Experiment 2. (a) Corners Only condition. (b) Midsegments Only condition. Exactly 50% of the pixels were removed in each condition, and the conditions were complementary in that those pixels that were present in the Corners Only condition were exactly those removed in the Midsegments Only condition and vice versa.

## Results

Four of the 24 subjects tested were excluded from the analyses because of high false alarm rates on catch trials in which no target was presented (a mean 19.34% false alarms). For the remaining 20 subjects, any RTs that were over 1000 msec or less than 100 msec were excluded; this trimming eliminated less than 1.2% of the data. For each subject, the median RT for each condition was calculated, and these median RTs were then analyzed with a within-subject ANOVA.

Mean RTs for all conditions appear in Figure 6. Valid versus invalid trials were first analyzed with a two-factor ANOVA, with factors being display type (Corners Only versus Midsegments Only) and cue type (valid versus invalid). This analysis revealed a main effect of cue type, $F(1, 19) = 65.34, p < 0.0001$, with RTs to validly cued targets being faster than RTs to invalidly cued targets (306.26 msec versus 341.94 msec, respectively). There was neither a main effect for display type, $F < 1$, nor an interaction, $F < 1$. Planned comparisons showed that the validity effect was significant for both the Corners Only condition, $t(19) = 8.00, p < 0.0001$, and for the Midsegments Only condition, $t(19) = 8.56, p < 0.0001$. These results appear in Figure 6a and replicate the standard spatial cuing effects observed in normal subjects (Egly et al., 1994; Vecera, 1994) and in the patient JW, who has apperceptive agnosia (Vecera & Behrmann, 1997).

Next, the invalid trials, graphed in Figure 6b, were analyzed with a two-factor ANOVA, with conditions being display type (Corners Only versus Midsegments Only) and target location (invalid target in the cued rectangle versus invalid target in the uncued rectangle). The main effect for display type was not significant, $F < 1$, suggesting that RTs were similar for the Corners Only displays and the Midsegments Only displays. There was a marginal effect for target location, $F(1, 19) = 3.85, p < 0.065$, with RTs to targets in the cued rectangle being slightly faster than targets appearing in the uncued rectangle (338.74 msec versus 344.86 msec, respectively).

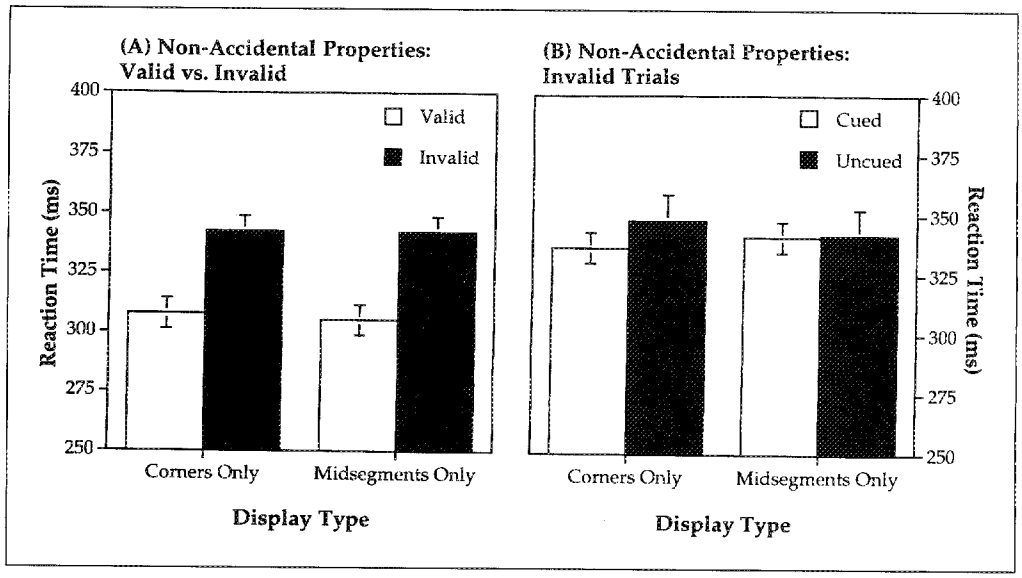


**Figure 6.** Mean RTs from the experiment. (a) Valid trials versus invalid trials; subjects were faster to detect validly cued targets relative to invalidly cued targets in both display types. (b) Invalid trials; the normal pattern on invalid trials (faster RTs to targets appearing in the cued rectangle relative to those appearing in the uncued rectangle) was reliable in the Corners Only displays but not in the Midsegments Only displays, consistent with a grouping-deficit account of apperceptive agnosia. (Error bars are within-subject 95% confidence intervals.)

Most importantly, this marginal main effect was subsumed by a statistically reliable interaction between display type and target location, $F(1, 19) = 5.64, p < 0.03$.

To further explore this interaction, planned comparisons were computed between RTs to targets appearing in the cued rectangle and RTs to targets appearing in the uncued rectangle. This difference was statistically reliable in the Corners Only displays, $t(19) = 4.26, p < 0.002$, but not in the Midsegments Only displays, $t < 1$. The Corners Only displays produced the normal effect on invalid trials similar to that observed in previous studies (Egly et al., 1994; Vecera, 1994), whereas the Midsegments Only displays did not exhibit this effect.

### Discussion

There were two important results of the present study. First, a reliable validity effect was found: Subjects detected validly cued targets faster than invalidly cued targets, and this effect was not influenced by the type of degradation (corner deletion or midsegment deletion). Second, and theoretically more important, there were statistically reliable differences between the two types of degradation for the invalid trials. As predicted by a grouping account of apperceptive agnosia, in the Midsegments Only condition there was no grouping effect; that is, there were no differences between attending to an invalidly cued target appearing in the cued rectangle and one appearing in the uncued rectangle. This result differs from that observed in normal subjects (Egly et al., 1994; Vecera, 1994) and is exactly what was observed in patient JW. In addition to this finding, the Corners Only condition exhibited the normal pattern for the invalid trials. Subjects were faster to detect invalidly cued targets appearing in the cued rectangle relative to those appearing in the uncued rectangle. Thus, it was not the degradation itself that impaired performance on the invalid trials but rather the features that were removed. Removal of a nonaccidental property, cotermination, removed an important grouping cue, thereby impairing performance.

## GENERAL DISCUSSION

In two experiments, we have tested between two accounts of apperceptive agnosia, a neuropsychological syndrome in which patients cannot recognize objects due to impaired lower-level perceptual processes. In Experiment 1 we covered displays with a peppery mask; such a mask, presumably caused by a peppering of the visual field with scotomas of various sizes, has been proposed as the basis of apperceptive agnosia (Campion, 1987; Campion & Latto, 1985). If such masking is the consequence of the neural damage underlying apperceptive agnosia, then normal subjects, when looking through such a mask, should simulate the behavior of apperceptive agnosic patients. However, our masking manipulation did not alter the pattern of results from the results in the clear (unmasked) displays. Reaction times were slower in the masked condition (although not statistically slower), suggesting that the mask had an additive effect with all of the conditions in this task. Under additive-factors logic (Sternberg, 1969), such an effect would indicate that the peppery mask influenced a stage of processing that was separate from both those processes that were the basis of the validity effect (presumably the spatial attention stage) and those processes that were the basis of the grouping effect (presumably a perceptual organization stage). Although there are limitations to additive-factors theory (e.g., McClelland, 1979), this analysis suggests that a peppery mask may influence an early level of visual processing that occurs prior to perceptual organization. The masking manipulation does

not appear to influence the same processes influenced in apperceptive agnosia, thereby weakening the peppery mask account of this visual disorder.

The results of Experiment 2 stand in contrast to those in Experiment 1. Using normal subjects, we successfully simulated the responses of an apperceptive agnosic patient by deleting nonaccidental properties from our displays. When we deleted the corners of our displays (corresponding to a removal of the nonaccidental property of cotermination), normal subjects still exhibited a validity effect, but they no longer exhibited a grouping effect; this replicates results from patient JW, an apperceptive agnosic patient who was recently tested with this same paradigm. Because nonaccidental properties act as grouping cues or heuristics that organize the visual field (Lowe, 1985, 1987), removal of these properties abolishes grouping cues that can guide spatial attention. This analysis suggests that the deficit in apperceptive agnosia is due to impaired perceptual grouping. It was not the removal of features per se that determined our results; when the same amount of visual information was deleted from the midsegments of the displays, the results replicated previous results from normal subjects. Deletion of midsegment data does not remove nonaccidental properties, thus leaving intact information necessary for perceptual grouping.

Before discussing the implications of a grouping-deficit account of apperceptive agnosia, we should first discuss the plausibility of the peppery mask account. Is this merely a straw man account of apperceptive agnosia? We think not for several reasons. First, without rigorous experimental tests, such as those we have attempted, it is entirely possible that the grouping deficits observed in apperceptive agnosia are secondary to damage that could be simulated with a peppery mask. Thus, one cannot a priori rule out a peppery mask account. Second, apperceptive agnosics do have damage to early visual cortices, which may result in scotomas that partially occlude the visual field and provide a mechanism for a peppery mask. Third, Campion and Latto (1985) were able to simulate some aspects of apperceptive agnosia, such as a flattened CSF, when stimuli were viewed through a mask. These points provide reasonable evidence for the peppery mask account and do not permit this explanation to be swept away without closer examination.

Despite the arguments that can be made for the peppery mask account, the grouping-deficit account of apperceptive agnosia seems to better explain many of the results observed in this patient population. For example, in normal subjects, removal of grouping cues, such as nonaccidental properties, impairs object recognition (Biederman, 1987), and apperceptive agnosic patients have difficulties recognizing objects. These patients also have difficulties with perceptual organization itself (Vecera & Behrmann, 1997), consistent with the loss of organizational processes. Finally, here we demonstrated that removal of grouping cues (again, nonaccidental properties) impairs some aspects of attentional allocation but leaves other aspects intact. Similar results have been obtained with an apperceptive agnosic patient (Vecera & Behrmann, 1997).

Although the grouping-deficit account can explain many of the deficits in apperceptive agnosia, we do not wish to argue that nonaccidental properties are the basis of all grouping deficits observed in these patients. These patients have difficulties with a variety of tasks, including shape matching (Efron, 1968). Can a loss of perceptual grouping processes explain these other deficits as well? We think so, although the deletion of nonaccidental properties may not be the best manipulation for simulating all deficits in apperceptive agnosia. It is possible that other grouping heuristics exist in addition to nonaccidental properties. If some tasks, such as figure-ground organization (see Rubin, 1915/1958), relied on these other heuristics, deletion of nonaccidental properties might not be the best manipulation for simulating the shape-matching deficits in normal subjects. We would not argue that all grouping effects are the result of nonaccidental properties (see Rubin, 1915/1958; Wertheimer, 1923/1958, for examples of other properties that are used for perceptual grouping). In addition, grouping processes may not be separate from other visual processes, such as feature representation (e.g., both may occur in primary visual cortex; see Hummel & Biederman, 1992; Mozer, Zemel, Behrmann, & Williams, 1992; Sajda & Finkel, 1995, for models consistent with this). Thus, other forms of damage, such as damage to feature representations, may also impair grouping processes in apperceptive agnosia. This could be simulated by adding and deleting visual features in displays.

Another aspect of the present studies that warrants discussion is the generality of the results from patient JW. We have chosen to simulate the results of one apperceptive agnosic patient. This is potentially problematic: What if JW was somehow atypical of these patients? To the extent that JW is representative of apperceptive agnosic patients, we expect our results to generalize to other patients with this syndrome. At the present time, we cannot determine if all apperceptive agnosics show the same pattern of results in the spatial cuing task. This is an empirical issue and deserves to be tested with other apperceptive agnosic patients, although the rarity of this syndrome will make this difficult. But, given that JW shows many of the same deficits as other apperceptive agnosic patients (impairments in object recognition, shape matching, perceptual organization; intact color perception and visual fields; see Vecera & Behrmann, 1997), there is no a priori reason to argue that he is radically different from the other patients.

The present results not only have implications for the understanding of apperceptive agnosia but also for understanding normal visual processes. The role that nonaccidental properties play in object representation and

recognition has been studied extensively (e.g., Biederman, 1987; Lowe, 1985, 1987), with the main finding being that object recognition is difficult, if not impossible, when nonaccidental properties have been removed. The present results extend the role of these properties; it is clear that nonaccidental properties also can have an influence on spatial attention processes. Spatial attention must obey the perceptual groups established by nonaccidental properties (and other grouping heuristics). When these properties are selectively removed, as in corner deletion, the visual system no longer represents perceptual groups, yet spatial attention can operate in the absence of grouping cues.

The fact that spatial attention can operate in the absence of grouping cues is important because it places constraints on models of visual processing. Much research with normal subjects has demonstrated that earlier, preattentive grouping processes can influence later spatial attention processes (e.g., Baylis & Driver, 1995; Egly et al., 1994; Kramer & Jacobson, 1991; Vecera, 1994), which suggests that spatial attention follows a full preattentive analysis of the visual field. This sequential view in which grouping precedes spatial attention seems logically necessary, because how could spatial attention be influenced by grouping cues before these cues had been computed? The present results suggest that such a strict bottom-up view cannot be entirely correct. Disrupting the grouping processes by removing nonaccidental properties does not impair spatial attention; instead, spatial attention is able to operate independently of grouping processes. Neuropsychological evidence is consistent with this conclusion (see Vecera & Behrmann, 1997). A sequential view seemingly would predict that damage to the earlier grouping processes would impair the inputs to spatial attention, thereby causing impairments in spatial attention itself (see Vecera & Gilds, 1997, for further discussion). Thus, these results not only concern apperceptive agnosia, but they also provide a better understanding of the normal interrelations between early preattentive grouping processes and later attentional processes. Spatial attention can be influenced by perceptual organization processes, but such organizational processes are not a necessary precursor for spatial selection of visual inputs.

## METHOD: EXPERIMENT 1

### Subjects

Subjects were 22 University of Utah undergraduates. All reported having normal or corrected-to-normal vision.

### Stimuli

Stimuli were two rectangles that were oriented either horizontally or vertically within the display. Each individual rectangle (see Figure 1) was black and drawn on a white background. Displays were viewed from a distance of approximately 65 cm. The fixation point measured 0.6 by 0.6 cm (0.59 by 0.59° of visual angle). Each of the rectangles measured 9.0 by 2.7 cm (8.76 by 2.64°). The lines that composed the rectangles were three pixels wide. The cue consisted of a black circle, measuring 1.7 by 1.7 cm (1.66 by 1.66°), that was centered at the end of one of the rectangles (Figure 1b). The target was an asterisk measuring 1.2 by 1.2 cm (1.18 by 1.18°), which, like the cue, was presented at the end of one of the rectangles. The centers of the rectangles were 2.75 cm from the center of fixation (2.69° of visual angle). The rectangles were equidistant from each other, and this distance measured 6.1 cm (5.96°) from the center of the target in each corner.

The peppery mask was created by generating a random pixel field using the spray can in the NIH Image software (see Note 3 for additional information on this package). The pixel density was adjusted until the mask met two constraints. First, it must appear similar to the published examples from Campion and colleagues (Campion, 1987; Campion & Latto, 1985), and second, the displays must remain partially visible when occluded with the mask. "Partially visible" was defined as being visible enough for subjects to perform the task (i.e., note the presence of the cue and detect the target). The pixel field that met these constraints was then copied to an overhead transparency. This transparency was then placed in front of the computer monitor for the masked blocks. The transparency was 21.59 cm tall by 27.94 cm wide and masked the entire monitor.

### Procedure

All stimuli were presented via a Macintosh Performa 6214CD computer. Subjects sat approximately 65 cm from the monitor. Subjects participated in nine blocks of 80 trials each; the first block was used only as practice. Subjects were instructed not to make eye movements during the task, and they were allowed to rest between blocks.

An individual trial began with a 1000-msec fixation display that contained the fixation point and the two stimuli groups. Following this display, the cue was presented for 100 msec. The fixation display was then presented for another 200 msec. Finally, the target appeared and remained until the subject responded by pressing the spacebar on a standard keyboard. Half of the subjects responded with the left hand and half with the right hand.

The trials were distributed as follows. Four of the eight experimental blocks were nonmasked (clear) displays, and the other four blocks were masked by placing a peppery mask over the computer monitor. Half of the subjects saw the clear displays first, and half saw the masked displays first. In addition, the practice block was divided such that half of the trials were masked and half

were nonmasked so that subjects would receive practice on both types of trials. Half of the time the rectangles were presented horizontally (i.e., above and below fixation) and half of the time they were presented vertically (i.e., to the left and right of fixation). This factor was collapsed across trials because previous studies using this paradigm (e.g., Egly et al., 1994; Vecera, 1994) have found no effect for horizontal versus vertical presentation.

Within each block of trials, 20% were catch trials in which no target appeared. Subjects were to withhold their responses on these trials, and on these trials the fixation display followed the cue for 2000 msec. If subjects responded on these trials, a visual error message would appear on the screen for 500 msec before the start of next trial. Of the remaining trials in which a target was presented, 75% of the time the cue was valid and 25% of the time it was invalid. In half of the invalid trials the target appeared in the cued rectangle and in the other half the target appeared in the uncued rectangle.

## METHOD: EXPERIMENT 2

### Subjects

Subjects were 24 University of Utah undergraduates. All reported having normal or corrected-to-normal vision.

### Stimuli

The stimuli were similar to those used in Experiment 1, except for the removal of nonaccidental properties. Both the Corners Only and the Midsegments Only conditions had the same number of pixels. Beginning with the intact rectangles used in Experiment 1, exactly 50% of the pixels were removed to create the present displays. The Corners Intact stimuli are the exact opposite of the Midsegments Intact stimuli so that, if placed on top of one another, they would form one complete rectangle with no overlap. Examples of these stimuli appear in Figure 5.

The stimuli used in Experiment 2 subtended the same visual angles as those in Experiment 1. The only difference was the removal of some of the image features, and this procedure is discussed in the text.

### Procedure

The procedure was identical to that in Experiment 1, with the following exception. The Corners Only and Midsegments Only conditions were intermixed within a block. These two conditions appeared randomly, preventing subjects from predicting whether the next trial would contain corners or midsegments. The distribution of trials was as in Experiment 1.

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### Notes

1. Warrington (1982, 1985) uses slightly different terminology. In her theoretical account of the visual agnosias, apperceptive agnosia is a deficit in perceptual categorization that follows right-hemisphere lesions. Patients described as apperceptive agnosics in most accounts, including the present paper, are referred to as "pseudo-agnosics" by Warrington. We do not discuss these right-hemisphere patients because the locus of damage and behavioral deficits are quite different from the apperceptive agnosic patients of most researchers.
2. Thanks to Bill Banks for discussing this possibility.
3. High-pass filtering removes low spatial frequency information from images. We used the high-pass filter macro in the NIH Image (http://rsb.info.nih.gov/nih-image/) software package. The filter size was 20% of the total image size with a transition of 0 pixels.
4. These displays were filtered with a standard high-pass filter. Filtering was again performed using the NIH Image software package; the images were filtered four times with the 3 × 3 "sharpening" filter that comes standard with this software package.

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