VISUAL PROCESSING DEFICITS AS ASSESSED BY SPATIAL FREQUENCY CONTRAST SENSITIVITY AND BACKWARD MASKING IN NORMAL AGEING AND ALZHEIMER’S DISEASE

by G. SCHLOTTERER, M. MOSCOVITCH and D. CRAPPER-MCLACHLAN

(From the Department of Psychology, Erindale College, University of Toronto, Mississauga, Ontario, and the Toronto General Hospital, Toronto, Ontario, Canada)

SUMMARY

Visual functions of patients with senile dementia of the Alzheimer type were compared with those of young people and age-matched controls. Visual acuity and spatial frequency contrast sensitivity did not differ significantly between Alzheimer patients and normal elderly subjects, although both were impaired in comparison with young subjects. Alzheimer patients required more time than ageing controls to identify letters and were susceptible to the interfering effects of a backward pattern mask on letter recognition over a longer interval. The spatial extent over which the pattern mask was effective, as well as the time interval over which a homogeneous mask interfered with letter recognition, were equivalent in normal old people and Alzheimer patients. In all the masking tasks, young people performed better than the old. It is suggested that Alzheimer’s disease affects later central visual functions more than early relatively peripheral ones.

INTRODUCTION

Recent interest in the behavioural deficits accompanying Alzheimer’s disease has focused almost exclusively on memory, language, and other cognitive functions (see Katzman et al., 1978; Schwartz et al., 1979; Corkin, 1981; Bayles, 1982). To our knowledge, there has not been a single published study on sensory and perceptual function in Alzheimer’s disease. This is all the more surprising since Alzheimer patients or their families often complain spontaneously of perceptual problems and the neuropathology associated with Alzheimer’s disease affects structures (such as secondary or association cortex) believed to mediate higher-order perceptual functions. This paper is the first to document the effects of Alzheimer’s disease on some visual functions.

1 Present address: Department of Psychology, Cornwall General Hospital, Cornwall, Ontario, Canada.
Reprint requests to Dr M. Moscovitch, Psychology Department, Erindale College, University of Toronto, Mississauga, Ontario, Canada L5L 1C6.
Brain atrophy, senile plaques, and neurofibrillary degeneration appear to be the histopathological changes most relevant to the dementia of the Alzheimer type (Tomlinson et al., 1970). Brain atrophy is a result of both neuron loss and reduction in neurocytoplasmic volume, but does not necessarily correlate with the degree of intellectual impairment observed in an individual (Blessed et al., 1968; Tomlinson et al., 1968). Both senile plaques and neurofibrillary degeneration, however, correlate with the presence of dementia in ageing individuals. Recent work with animals (Crapper, 1973; Crapper and Dalton, 1973a, b; Crapper and Tomko, 1975) and in man (Ball, 1976, 1977) suggests that neurofibrillary degeneration is a particularly important correlate of intellectual decline in Alzheimer's disease, although the exact functional significance of this characteristic histopathological change is unknown.

The effects of these degenerative processes on perceptual functions have not been investigated. In Alzheimer's disease, neurofibrillary degeneration occurs throughout most of the cerebral cortex and limbic structures. These changes occur most frequently in the large pyramidal shaped cells of the hippocampus and of layers III and V of the association areas of cerebral cortex (McMenemey, 1963; Tomlinson et al., 1968, 1970), but are absent in the geniculostriate system up to and including area 17 (primary visual cortex) (Schenk, 1955; Crapper, 1974, 1976). In contrast, senile plaques are more uniformly distributed throughout the visual cortex (Crapper et al., 1975). This pattern of degeneration suggests that some visual functions, such as early low-level processes associated with the geniculostriate system might be spared in Alzheimer's disease, and others, such as those later higher-order processes mediated by structures central to the striate cortex, might be impaired in Alzheimer's disease. The major purpose of this paper, therefore, is to document some aspects of visual perception in Alzheimer patients and, if possible, to relate our findings to the degenerative processes associated with the disease.

Of the many tests at our disposal, we first chose to look at visual acuity, spatial frequency contrast sensitivity, and susceptibility to visual masking. For reasons that will become apparent, we considered that these tests would illustrate the range of visual functions that are spared and impaired in Alzheimer's disease.

**Experiment 1. Acuity and Contrast**

Testing acuity by traditional methods seemed a logical first step. We chose to examine spatial frequency contrast sensitivity because it provides a different test of spatial resolution in that it can specify which part of the spatial frequency domain is most severely affected by the disease. Spatial frequency is defined as the number of alternating pairs of black and white regions (sinusoidal gratings) per degree of visual angle. It is now generally accepted that visual input is processed along various spatial frequency channels of unequal sensitivity. Under ideal conditions humans can distinguish gratings of spatial frequencies between 0.5 to 50 c/deg from a uniform grey stimulus, the greatest sensitivity occurring at 2 to 4 c/deg (Cornsweet, 1970; Campbell, 1974; Lennie, 1980).
It is also widely believed, but not universally held, that low spatial frequencies are processed by a system whose characteristics are distinct from that involved in processing high spatial frequencies and that each system subserves different perceptual functions (Lennie, 1980; Graham, 1981). This dissociation suggests that neural damage could selectively affect one system but not the other. There have been a number of reports of loss of sensitivity restricted only to some spatial frequencies in patients with various disorders of the visual system but with normal or near normal Snellen acuity (Bodis-Wollner, 1972, 1977; Bodis-Wollner et al., 1978; Regan et al., 1977, 1981; Zimmermann et al., 1979). For example, Regan et al. (1977, 1981) found that patients with multiple sclerosis had a loss of sensitivity for low and/or intermediate spatial frequencies, whereas Bodis-Wollner (1972) found that patients with striate cortex lesions had reduced sensitivity primarily to high spatial frequencies. In Alzheimer patients the striate cortex is relatively spared from the degenerative effects of the disease, but the prestriate and posterior temporal and parietal lobes are not. It would be particularly instructive, both theoretically and practically, to know whether spatial frequency contrast sensitivity is affected by diffuse damage to these extrastriate structures and, if it is, whether all channels are affected equally.

Subjects

Three subject groups were examined: (1) a young control group consisting of 11 individuals aged 20 to 29 yrs (mean 25 yrs; (2) a control group of 11 nondementing persons aged 53 to 77 yrs (mean 63), who were either fully employed or were still active in the community; and (3) a group of 10 individuals with the clinical diagnosis of Alzheimer’s disease aged 53 to 75 yrs (mean 64). Verbal IQ of the patients at the time of testing ranged from 57 to 128 with an average of 80.4. Performance IQ, except for one patient who was untestable, ranged from 55 to 111, with an average of 75.6. The full scale IQs for the testable patients ranged from 50 to 121, with an average of 87.1. The clinical diagnosis of Alzheimer’s disease in this study was based on a careful process of elimination. Relatively slow intellectual decline, highlighted by impairment of memory functions, in the presence of marked cerebral atrophy, evidenced on either a pneumoencephalogram or CT brain scan, were critical diagnostic indices. In addition there was no evidence of space-occupying lesions, nor of rapid deterioration of motor function or basic personality traits. In a separate study, D. Crapper-McLachlan (unpublished observations) found that of patients diagnosed as Alzheimer’s disease by these criteria, 86 per cent proved to have the characteristic neuropathology of this disease at autopsy.

From the original group of 10 individuals with Alzheimer’s disease, 6 were capable of reliable performance on the spatial frequency contrast threshold procedure. The failure of the remaining 4 subjects to perform reliably was due more to their inability to comprehend and follow instructions than to their poor visual function. Nonetheless, the average IQ of these patients was similar to the group average. All subjects were successfully tested on the visual masking procedures (see below). Aside from the neurological disorder of Alzheimer’s disease, all subjects were in good health and had a corrected visual acuity of 20/40 (Snellen) or better. Informed consent was obtained from all the subjects or members of their family.

Procedure

The effects of normal ageing and Alzheimer dementia on primary visual function were tested by measurements of visual acuity and spatial frequency contrast sensitivity.
Visual acuity. A Bausch and Lomb Master Ortho-Rater was used to measure visual acuity. The acuity scores were maintained in Ortho-Rater format for analysis (Ortho-Rater 10 = 20/20, Snellen; Ortho-Rater 5 = 20/40, Snellen).

Spatial frequency contrast sensitivity. Contrast is defined as the ratio of the difference between the regions of highest and lowest luminance and the sum of these luminance intensities ($C = I_{max} - I_{min}/I_{max} + I_{min}$). The amount of contrast required to detect the presence of a particular spatial frequency grating defines the contrast threshold for that spatial frequency. In this study, contrast thresholds were determined for six different spatial frequencies, 0.5, 1, 2, 4, 8, and 12 c/deg. The ‘sine-wave’ gratings were produced using methods similar to those described by Campbell and Green (1965). Two Heath Company signal generators were used to drive the Y and Z axes of a Tektronix 502A oscilloscope. After setting the time base to sweep the width of the screen 200 times per second, a 1000 line raster was generated by applying a 200 kHz triangular waveform signal to the Y-axis. Modulation of the Z-axis was accomplished by applying a sine-wave signal from the second signal generator to the CRT input. Luminance was linearly related to Z-axis voltage. Spatial frequency of the resulting grating was manipulated by varying the frequency output of the Z-axis signal generator. The intensity control at the front of the oscilloscope was used to adjust the luminance of the raster. Z-axis modulation varied the intensity of the raster equally above and below this baseline, therefore maintaining the mean luminance of the gratings at a level equal to the unmodulated raster. The amplitude of the signal generator output driving the Z-axis was measured on a digital voltmeter. Directly (2.54 cm) in front of the CRT face was a 30 cm square field of white bristol board. There was a 5 cm square opening in the centre of this white field forming a visual angle of 4° through which the subject viewed the sine-wave gratings binocularly from a distance of 73 cm. For all gratings, the overall mean luminance level was maintained at 5 cd m$^{-2}$. The surround field was matched both in colour and luminance to the central raster by chromatic lighting with filtered, variable intensity incandescent light sources. The target gratings were presented intermittently for a duration of 1 s alternating with a 1 s presentation of a uniform background.

The subject was seated comfortably in a darkened room, illuminated only by the light sources used to maintain the luminances of the surround field. The subject’s head was positioned securely by a Bausch and Lomb chin rest and head restrainer. After familiarizing the subject with each of the spatial frequency gratings at moderate contrast levels, contrast was set to zero and the detection trials were begun. The six spatial frequencies were presented in pseudorandom order a total of five times. Once a grating was chosen by the experimenter, the subject was instructed to begin attending to the target field. Contrast was then increased by a constant 0.003 contrast units every 2 s (1 cycle of alternation between grating and background) until the subject reported detection of a grating. As a check of response reliability, the subject was asked to report the relative size of the detected grating (‘wide’, ‘medium’ or ‘fine’). If this report was not accurate, the trial would be repeated later in the session. The criterion for accurate report of the spatial frequency required the subject to assign the following spatial frequency gratings to the designated category: 0.5, 1, 2 (‘wide’); 2, 4, 8 (‘medium’); 4, 8, 12 (‘fine’). Ten erroneous reports excluded the subject from the study. The contrast sensitivity for each spatial frequency was defined as the inverse of contrast threshold averaged over the five trials. A plot of contrast sensitivity vs spatial frequency generates a spatial-frequency contrast-sensitivity function.

Results and Comment

The young control group had a mean acuity of 10.6 ± 1.4 (Ortho-Rater). The normal ageing and Alzheimer’s disease groups were similar to each other with acuities of 7.8 ± 1.5 and 8.3 ± 1.2, respectively. As expected, the two older groups had slightly but significantly ($P < 0.05$) poorer visual acuity than the younger group.

The spatial frequency contrast functions for each group were identical in form
(fig. 1), yielding a significant main effect for spatial frequency ($F(5,125) = 18.9$, $P < 0.01$). However, the young control group had significantly greater sensitivity (ANOVA, $F(2,23) = 19.5$, $P < 0.01$) at all spatial frequencies except 2 c/deg as indicated by post hoc Newman-Keuls tests. No significant differences were found between the ageing control and Alzheimer groups. Ageing leads to reduced optical transmission and increased light scatter due to changes in the lens, humours, and pupil size (Said and Weale, 1959; Birren, 1960; Ruddock, 1965; Corso, 1971), as well as loss of granule cells in the primary visual cortex (Brody, 1955, 1976) and may account for the observed decrease in sensitivity in the elderly (Bodis-Wollner, 1972, 1977). These processes, however, seem not to be accelerated by Alzheimer's disease. The results are consistent with neurohistological studies (Schenk, 1955; Tomlinson et al., 1968, 1970) and radiological evidence from PET scans (Benson et al., 1981) that the striate cortex is not more severely affected in Alzheimer’s disease than in normal old people. Lastly, the results suggest that damage to extrastriate cortical visual areas do not noticeably influence spatial frequency contrast sensitivity.

It should be noted that our results are somewhat at variance with those of Sekuler and Hutman (Sekuler and Hutman, 1980; Sekuler et al., 1980; Hutman and Sekuler, 1980) who found that old people had reduced sensitivity only at low and intermediate spatial frequencies but were normal at frequencies of 16 c/deg, and presumably higher. This is surprising given the increased light scatter in the ageing eye which would most affect the high spatial frequencies. Be that as it may, the source of the discrepancy between our study and theirs is difficult to ascertain.
because the studies differed in so many ways. First, the average age of their subjects was ten years older than ours; secondly, their stimuli flickered at 0.33 and 6 Hz whereas ours flickered at 1 Hz; thirdly, they measured threshold by approaching it from above and below whereas we approached it only from below; fourthly, the overall luminance of their grating was higher than ours by a factor of 10.

It is most likely that one of the last two differences was critical. Old people may adopt a different criterion from younger individuals in deciding when a grating is visible and these criterion differences may exert a greater influence on threshold detection when threshold is approached from only one direction. Against the idea that criterion differences account for the discrepancy between our study and those of Sekuler and Hutman is the fact that Alzheimer patients, who are demented and often behave inappropriately, nonetheless behaved similarly to old people on this test. If criterion differences were at work, we would expect significant differences between the old and the Alzheimer patients, but no such differences were found.

The most likely explanation is that overall luminance differences of the gratings were the source of the discrepancy. It is possible that at low luminance, differences between old and young people would emerge at high spatial frequencies. Because our study was completed (Schlotterer, 1977) and reported (Schlotterer et al., 1978) before that of Sekuler and Hutman, we were in no position to replicate their findings. We plan, however, to investigate these ideas in future work. We are encouraged that subsequent work by Sekuler and his colleagues (Owsley, 1982; Sekuler and Owsley, 1982) has yielded results more consistent with our own and with the known optical properties of the aged eye, namely, that sensitivity to high spatial frequencies is decreased.

Whatever the source of the discrepancy, the finding of primary interest is that the Alzheimer patients who were sufficiently alert to complete the task were no worse than their age-matched controls on tests of spatial frequency contrast sensitivity. One reason for this may be that spatial frequency analysis is a relatively low-level visual function that is dependent primarily on early stages of visual processing. If this is true, other functions that are similarly dependent on early stages of processing should also be intact in Alzheimer patients, whereas those functions that are dependent on later higher-order stages should be impaired. The second experiment was designed to explore this possibility.

EXPERIMENT 2. BACKWARD VISUAL MASKING BY SUPERIMPOSITION OF A HOMOGENEOUS AND A PATTERNED STIMULUS

Visual masking occurs when a stimulus interferes with the perception of a temporally contiguous target. In the typical backward masking paradigm, the onset of a briefly presented target precedes the onset of the interfering mask. Target perception may be perfect without the mask but it can drop to zero at optimal temporal separation between the mask and target. As the interval between the onset of the target and the onset of the mask increases (stimulus onset asynchrony or
SOA), the target gradually escapes the effects of the mask until perception is once again perfect. This interval is known as the critical SOA.

Backward visual masking can involve both peripheral and central processes (Turvey, 1973; Breitmeyer and Ganz, 1976; Michaels and Turvey, 1979; Felsten and Wasserman, 1980). If the mask is a homogeneous stimulus without contours, it interferes with the target if it is presented to the same eye as the target. Because a homogeneous stimulus is not an effective mask when it is presented to the eye opposite to the target, it is presumed to act peripherally, prior to the point at which inputs from the two eyes converge. This convergence first occurs in the striate cortex (Hubel and Wiesel, 1968), but is found more extensively in the pre-striate cortex and beyond (Zeki, 1978). Because the geniculo-striate system up to and including the striate cortex is relatively spared in Alzheimer patients, we expect them to show normal masking functions when a homogeneous mask is used.

If the mask is patterned such that it has contours similar to those found in the target, then the mask is effective even when it is presented to the eye opposite to the target. This dichoptic interaction suggests that backward masking with patterns is also a central phenomenon that occurs at higher-order stages of processing where inputs from the two eyes interact. If the critical interaction for backward pattern masking occurs beyond the striate cortex, then Alzheimer patients should react abnormally to backward pattern masks. The results of our study confirmed our predictions regarding the performance of Alzheimer patients on tests of backward masking with both a homogeneous and a pattern mask.

Subjects

Ten young controls whose average age was 25 yrs, 11 ageing controls whose average age was 64.1 yrs and 10 patients with Alzheimer’s disease whose average age was 64.0 yrs, served as subjects in the experiment.

Procedure

The targets were letters and the masks were either a flash of light or a meaningless pattern. A Pandora’s Box 3-channel tachistoscope was used to present visual stimuli to both eyes at an optical distance of 1 m. The target consisted of 9 symmetrical letters of the alphabet (A, H, O, T, U, V, W, X, Y) constructed from 1.25 cm Franklin-Gothic style Quik-Stik lettering and subtending a visual angle of 0.7 to 1.0 deg square depending on the letter. Each target was positioned centrally in a 5 cm (2.9 deg) square field which overlapped with the apparent location of the target field. The patterned mask consisted of an overlapping Franklin-Gothic letter ‘I’ and segments of the letter ‘T’ cross-bars that subtended a visual angle of 1.2 deg square within a 2.9 deg square blank field. All masks were exposed for 30 ms at a constant luminance of 170 cd m⁻². For the purpose of light adaptation and target localization, the 2.9 deg square blank fixation field was provided. This field was illuminated whenever the target or masking fields were not present. In the centre of this dimly illuminated (4 cd m⁻²) field was located a small black point (1 mm diameter) which corresponded to the apparent centre of the target stimuli. Five minutes of adaptation at the reduced luminance of the fixation field preceded actual testing. During this period the subject was completely familiarized with the target stimuli and testing procedures. The interval between the onset of the target and the onset of the masking stimulus is defined as the stimulus-onset asynchrony (SOA). The SOA at which the target escapes the effects of the mask defines optimal performance.
After an adaptation period, the subject was asked to identify each letter as it was presented in the centre of the fixation field. The nine target stimuli were randomly chosen for each trial. The first stimulus was presented beginning with a target duration of 1 ms. If the subject responded correctly, a new target was chosen and presented at the same target duration. Following each error, the target stimulus was changed and 1 ms was added to the target duration. This procedure continued until the subject correctly identified four targets in a row. The target duration at which this was accomplished is defined as the target-duration threshold.

All masking procedures were carried out with the time between the termination of the target and the onset of mask (interstimulus interval, ISI) equal to zero ms. An initial value of target duration was chosen for each subject which was equal to the target-duration threshold. Randomly chosen letters were then presented at this duration and were followed immediately by a 30 ms mask. If the subject failed to report the target correctly, the duration was increased in 2 ms steps. If the subject succeeded in identifying four targets in a row correctly, the existing value of the target duration was noted. Because the ISI was equal to zero, this critical value of target duration was equivalent to SOA for that mask. This value was determined three times for each mask with the average of the last two values being used in the final analysis.

**Results**

The minimum time required to identify a constant luminance target (target duration threshold) varied for each of the three groups. The youngest group required 4.3 ± 1.0 ms which differed significantly from the ageing control group who required 8.1 ± 5.1 ms (F(1,19) = 5.18, P < 0.05), which differed significantly from that of Alzheimer patients who required 16.0 ± 10.2 ms (F(1,19) = 5.20, P < 0.05). The critical SOA derived for both the flash and pattern masks is represented in fig. 2. The ageing controls differed significantly from the young both in the homogeneous flash mask condition (F(1,19) = 21.1, P < 0.01) and in the pattern mask condition (F(1,19) = 18.8, P = 0.01). The Alzheimer patients differed from ageing controls only in the pattern mask condition (F(1,19) = 18.53, P < 0.01), but not with homogeneous flash condition (F(1,19) = 1.23). When covariate adjustments for visual acuity were made for the young and ageing controls, the significant differences between them in the pattern and flash mask condition were removed. Since there were no significant differences in acuity between the ageing control and dementia groups, covariate adjustments for these groups cannot be made.

**Comment**

The results are clearly consistent with the predictions that Alzheimer patients would be impaired relative to age-matched controls only on the higher-level pattern masking task. On the peripheral masking task, Alzheimer patients were no worse than their age-matched controls, although both age groups had higher critical SOAs than the young control subjects. As we discussed, it is not clear whether the deficits in the elderly are due to a deterioration of the ocular medium (pupils, lenses, cornea, humours) or to the slow neural degeneration of the geniculostriate system. Whatever the cause, it is consistent with the notion that homogeneous masking is a peripheral phenomenon.
On backward masking with a homogeneous flash, Alzheimer patients were no worse than their age-matched controls although both groups required more processing time than the young to escape the effects of the mask. As with spatial frequency contrast sensitivity, these masking functions varied with acuity suggesting that relatively peripheral factors determined performance. It is difficult to decide, on the basis of currently available evidence, whether these factors are extraneural, such as age-related changes in the ocular media, or neural, such as cell loss in the primary projection pathways in the geniculostriate system that is found to the same degree in normal elderly people as in Alzheimer patients (see below).

On pattern masking, however, Alzheimer patients differed significantly from their age-matched controls, who in turn differed from the young. Although it is known that some peripheral factors also influence performance on the pattern masking task under monocular presentation, there is sufficient evidence to indicate that additional higher-order central factors are involved. Walsh (1982), for example, found that in the normal elderly, performances on the two masking tasks do not correlate well with each other. As a more direct test, Byrd and Moscovitch (cited in Moscovitch, 1982) restricted participants in their masking study to those individuals whose acuity was sufficiently good for them to detect words exposed for 2 ms at normal intensity and contrast. As expected, these elderly individuals who took only a little longer to

![Graph showing mean stimulus onset asymmetry](image)

**Fig. 2.** The effects of normal ageing and Alzheimer's disease on visual masking performance. Mean stimulus onset asynchrony in ms when interstimulus interval is zero is compared for young, ageing and Alzheimer groups for homogeneous and pattern masking conditions. The error bars represent standard deviations.
escape the effects of the peripheral mask required much more time than the young to escape the effects of the central pattern mask, but these results parallel those between normal elderly and Alzheimer patients in our study.

That performance on backward pattern masking can differ among individuals with equivalent visual acuity and homogeneous masking functions reinforces the view that pattern masking is a higher-order central phenomenon that is not completely determined by peripheral factors. From a functional point of view, backward masking with a homogeneous unpatterned stimulus affects target perception by interfering with the extraction of physical features of the target such as its brightness, contrast and contour. Changing the brightness and contrast of the target or mask will have a profound effect on homogeneous masking functions (see Turvey, 1973; Coltheart, 1980, for review). It is easy to see, therefore, why performance on the masking task will vary with acuity or with any alterations in the ocular media that produce changes in the physical quality of the stimulus. Backward pattern masking, on the other hand, is believed to operate by interrupting the action of a central processor that integrates these physical features into relational or categorical information. Consequently, once it occurs centrally, central pattern masking is relatively immune to changes in the physical features of the target. What is critical is the total time the central processor has to integrate these features into a relational percept. The results from our study suggest that the central processor, which slows down somewhat with age as others have observed (Birren, 1965), is slowed even further by the degenerative effects of Alzheimer's disease. As a result, it needs more time to complete its task and is, therefore, susceptible to the effects of the mask for longer intervals.

This framework can be used to explain the unexpected finding that target identification, even in the absence of any mask, paralleled the performance of our three groups in the pattern masking study. Clearly, to identify a letter, a central processor is needed to integrate physical features into a meaningful percept. Peripheral factors are responsible for the degraded or delayed input of these features to the central processor. In addition, the termination of the target itself produces a signal that may act as a pattern mask to interrupt the central processor (Breitmeyer and Ganz, 1976). Lastly, it is conceivable that the deterioration of central areas outside the visual system that are concerned with verbal processes may also have contributed to the increased threshold of letter identification in old people and Alzheimer patients.

EXPERIMENT 3. SPATIAL EXTENT OF BACKWARD PATTERN MASKING

The focus of the study thus far has been on temporal factors affecting performance on masking tasks. Spatial factors, however, are also important. It is known, for example, that for the pattern mask to be effective, its contours must lie in close spatial proximity to the target. If there is loss of resolving power with age, as our findings on acuity and contrast sensitivity suggest, the spatial extent over which
pattern masking is effective should increase. By determining whether this increase is retarded or accentuated by Alzheimer's neuropathology, information can be gained about its effects on the spatial organization of neural networks.

Subjects

Except for two Alzheimer patients who were not available, all the subjects who participated in the previous masking studies took part in the experiment.

Procedure

The target duration was set at the value at which each subject reached criterion performance of four consecutive correct identifications of the target with a flash mask at zero ISI. The pattern mask was then superimposed directly over the target stimuli and presented at zero ISI. After each identification error the patterned mask moved to the left by 0.125 deg. The lateral displacement necessary to return to criterion performance was thus determined. At this displacement the pattern mask would be no more effective than the flash mask.

Results and Comment

As predicted, for the pattern masking stimulus array to be no more effective than a featureless homogeneous flash, the ageing control group required an average displacement of 1.54 deg which was consistently greater than the 0.91 deg displacement required by the young (ANOVA, F(1,19) = 4.56, P < 0.05). This statistical significance is lost after adjustments for the covariate of visual acuity (ANOVA, F(1,19) = 0.27, P < 0.5). No differences were seen between normal ageing and Alzheimer's disease groups who required an average displacement of 1.25 deg (ANOVA, F(1,20), 0.54, P < 0.5).

In general it appears that increases in the spatial extent of the metaccontrast masking effect coincides with the decline in visual acuity which accompanies advancing age. No further increases are seen as a result of the neuropathology of Alzheimer's disease. If Alzheimer's neuropathology leads to functional neural loss, we might have predicted that the possibility of spatial interaction should have been reduced as functional neural networks shrink in size. This shrinkage might have overcome the increased spatial extent of interaction that occurs due to loss of resolving power with age. Although the spatial extent of metaccontrast is smaller in Alzheimer patients than in the elderly, the differences were not significant. These results thus indicate that the spatial organization of neural networks mediating pattern masking is not as grossly affected as temporal organization by Alzheimer neuropathology, at least in the relatively early stages of the disease at which our patients were tested. This is not surprising in that the results also suggest that relatively peripheral factors, such as those related to acuity, influence the degree of spatial displacement over which a pattern mask is effective.
DISCUSSION

Impaired visual functions in Alzheimer patients are of two types—those which are mild and result from changes that normally accompany old age and those which are severe and are exacerbated by the neuropathological processes of dementia. The first is correlated with loss of visual acuity and is of no greater severity than that found in intellectually normal aged individuals. Thus, visual acuity, spatial frequency contrast sensitivity, and susceptibility to homogeneous visual masking is equivalent in Alzheimer patients and age-matched controls, although both groups are impaired when compared with young individuals. The clear relationship between declining visual acuity and performance on the latter two tasks suggests that the deficits are relatively peripheral and associated with declining sensitivity to low-contrast, high spatial frequency targets in old age. This pattern of results is consistent with those most recently reported by Owsley (1982) and Sekuler and Owsley (1982). Much of the spatial luminosity information which is necessary for correct identification of sharp edged visual targets falls in the middle and upper spatial frequency range (Campbell et al., 1971; Hood, 1973; Graham, 1981). If detection of these frequencies is slightly impaired, as it appears to be in the normal ageing adult, then performance deficits would be expected on tasks, such as backward masking with a homogeneous flash, that directly manipulate apparent contrast.

The second type of impairment, which is associated with higher-order, central visual processes, seems to be independent of visual acuity. Alzheimer patients needed twice the duration as normal aged people to reach the threshold of letter identification, although both groups were matched for age and visual acuity. Similarly, Alzheimer patients were much more susceptible to the effect of backward masking with a pattern. Both these tasks impose limits on the time available for the formation of a patterned relational percept. The sudden presentation of a contour, as in pattern masking, or the termination of an existing contour, as in letter identification threshold, is believed to disrupt ongoing central visual information processes (Turvey, 1973; Breitmeyer and Ganz, 1976). These results strongly suggest that the speed of central visual processes that normally slow down with age is reduced even further in Alzheimer’s disease. Interestingly, only temporal aspects of backward masking are noticeably altered in the early stages of Alzheimer’s disease. The spatial extent over which the mask is effective is equivalent in Alzheimer patients and aged controls, although both groups are susceptible to the mask over a larger area than young controls. These results are reminiscent of Wilson’s (1967) finding that in areas of impaired vision, temporal summation is altered only by postgeniculate lesions, whereas spatial summation is affected equally by both pre- and postgeniculate lesions.

As we discussed earlier, it is difficult to determine the structural locus at which both types of impaired function arise. The high correlation between visual acuity and the impaired functions that Alzheimer patients share in type and severity with
normal old people suggest that they may result from changes in the physical ocular media that accompany old age. The loss of retinal illumination and resolving power as a result of changes in the lens, in pupil size, and of the composition of the ocular humours could lead to the age-related changes in contrast sensitivity and acuity. It is equally possible, however, that this impairment is associated with neural factors such as cell loss in the primary visual pathways or even with loss of efficiency of existing neurons. The latter could account for the slowing of visual processes with age even for low-level tasks such as are measured by backward masking with a homogeneous mask (Walsh, 1982) and by the persistence of visual sensations after the stimulus has physically disappeared (DiLollo et al., 1982; Kline et al., 1982). At the moment, it is difficult to decide between the two alternatives. In any event, the evidence does suggest that the source of the impairment is relatively peripheral in that it is not likely to affect structures central to the primary visual pathways. With regard to Alzheimer’s disease, it indicates that the degenerative processes spare these visual functions, at least in the early stages of the disease. Thus, although the primary visual cortex contains some Alzheimer’s histopathology, such as abundant senile plaques but sparse neurofibrillary degeneration, the visual functions traditionally attributed to this area remain relatively preserved. Consistent with this view is the finding that metabolic functions as measured by PET scan are normal in the primary visual cortex in Alzheimer patients (Benson et al., 1981).

In contrast, neurofibrillary degeneration is extensive and metabolic processes are abnormal in areas such as prestriate, posterior temporal and parietal cortex that are central to primary visual cortex. Functions related to form perception, such as patterned masking, that are thought to be mediated or dependent on input from these areas (Gross and Mishkin, 1977) are severely impaired even in the early stages of Alzheimer’s disease. Because the spatial extent of masking is severely limited, even in Alzheimer patients, it suggests that masking effects are mediated by areas that still retain fine retinotopic organization. This does not exclude the possibility that areas involved with more abstract verbal processes also contribute to letter identification. Their deterioration, which is known to occur in Alzheimer’s disease, can also contribute to deficits in letter identification in the absence of masking.

Although the results of our study are by no means conclusive, they attest to the viability of our working hypothesis that visual processes up to and including the striate cortex are normal in Alzheimer patients whereas those central to it are impaired. Encouraged by these results, we have recently begun to examine such functions as colour vision and stereoscopic depth perception which require intact extrastriate areas for normal performance (Zeki, 1973, 1978; Pearlman et al., 1979; Ratcliff and Cowey, 1981). Preliminary evidence indicates that stereoscopic depth perception (G. R. Schlotterer, unpublished observation) and colour vision (Moscovitch, 1982) are impaired in Alzheimer patients whereas brightness discrimination, which seems not to be as dependent on these areas, is relatively normal.

Whatever the ultimate fate of our working hypothesis, it is clear from our findings that perceptual processes merit further investigation in Alzheimer patients. Because
so much is already known about the neural organization of perceptual systems, such research may not only provide clues about the way Alzheimer pathology affects normal function but also about the relation between neural structure and complex perceptual processes in normal people.

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ADDENDUM

After this paper was accepted for publication, the authors were informed of a poster presentation at the Association for Research in Vision and Ophthalmology in 1983 by M. J. Nissen, S. Corkin, J. Growdon, S. Wray and J. Bauer entitled ‘Spatial contrast sensitivity in Alzheimer’s disease’. They reported that an unselected group of Alzheimer patients showed spatial frequency contrast sensitivity deficits at all frequencies between 0.5 and 8.0 c/deg. Because their testing procedure was similar to ours, it is not clear what caused the discrepancy between our results and theirs. One possibility is that all our patients had Snellen acuity equivalent to age-matched controls whereas theirs may not have. Since they do not report Snellen acuity in their poster, it is difficult to ascertain whether this is a critical variable. For the moment we still wish to conclude that most Alzheimer patients with normal Snellen acuity also have normal contrast sensitivity functions.