Journal of Clinical and Experimental Neuropsychology
1998, Vol. 20, No. 1, pp. 118-134
1380-3395/98/2001-118$12.00

Speed of Elementary Visual Recognition Operations in Parkinson's Disease as Measured by the Mutual Masking Method*

Talis Bachmann[1], Toomas Asser[2], Mari Sarv[1], Pille Taba[2], Ene Lausvee[1], Endel Põder[1], Neeme Kahusk[1], and Tarmo Reitsnik[2]

[1] Laboratory of Cognitive Neuroscience, Tallinn University of Social and Educational Sciences, Tallinn, Estonia and [2] Department of Neurology & Neurosurgery, University of Tartu, Tartu, Estonia

ABSTRACT

Pairs of mutually different, spatially overlapping letters were exposed for recognition to groups of patients with Parkinson's disease (PD) and the age-matched control group. Stimulus onset asynchrony (SOA), medical treatment status (de novo vs. treated), and predominant symptoms (tremor vs. hypokinetic rigidity) were an other main variables. The highly significant main effects of SOA and health status demonstrated slowing of elementary visual recognition operations in Parkinson's disease; the results are based on the experimental method that requires neither fast manual responses nor tracking of the display events by saccadic eye movements. Significant interaction between the temporal order of stimulus exposure and health status showed that impairment due to PD was more pronounced for the first stimulus, including the de novo group. Qualitatively similar recognition functions in the binocular and dichoptic conditions showed that the typical pattern of results – prevalence of S2 over S1 at intermediate SOAs – cannot be attributed to retinal processes and should be originating from central processes. An earlier finding (Bachmann, 1994) that PD patients whose nonspecific thalamic nuclei were stimulated intracranially produced qualitatively unusual recognition functions that should have been the result of stimulation, rather than PD as such.

The list of the effects of Parkinson's disease (PD) on behavioural/psychological functions is both long and controversial. There is no doubt that executive functions and motor processes suffer in PD as a direct and/or indirect result of impairment in the dopaminergic system. However, the picture is less clear for relatively more pure cognitive and perceptual functions due to their being cofounded with measured motor and effector components in most experiments. Whereas a number of studies suggest that PD causes at least some impairment of purely cognitive functions, including perceptual processing at various levels, other studies either fail to find evidence of cognitive/perceptual impairment or report ambiguous findings, often qualifying measured cognitive impairments as conditional on the more concrete values of other variables. Let us list some examples from three groups of studies, beginning with more elementary levels of function.

1. Elementary sensory processes of temporal resolution. According to Artieda, Pastor, Lacruz, and Obeso (1992), two-pulse temporal discrimination thresholds may be elevated in Parkinsonian subjects with temporary improvement being effected by levodopa treatment. A related finding was reported earlier by Bodis-Wollner et al. (1987) who showed that sensitivity to flicker may be diminished in PD.

* The present research was conducted with the support from Estonian Science Foundation Grant No. 885.
Address correspondence to Talis Bachmann who is now at the Department of Psychology, University of Portsmouth, King Henry Building, Portsmouth PO1 2DY, Hampshire, UK. E-mail: bachmannt@port.ac.uk.
Accepted for publication: November 29, 1997.

2. Elementary processes of preconscious sensory detection. Not only conscious processes and explicitly reportable and intentionally executed responses may be impaired in PD. For example, automatic detection of stimulus change as revealed by mismatch negativity component of auditory ERPs may be impaired as well (Pekkonen, Jousmäki, Reinikainen, & Partanen, 1995).

3. Speeded responses to sensory-perceptual stimuli. Jordan, Sagar, and Cooper (1992) have found that cognitive components in reaction time (RT) may be slowed in PD, but are not sensitive to dopamine treatment. On the other hand, increase in average RT in PD may be caused not by steady slowing of perceptual operations, but by the increase in the number of occasional long-latency trials as a result of oscillation of attentive state – the main distribution of RTs in PD having been found to coincide with that of a normal group, except for asymmetrical edge at the long-latency range (Brown et al., 1993). The relatively more response-stage contribution to the effects of RT slowing can also be inferred from the fact that latencies of sensory ERP components have been found to be unimpaired even though overt response latencies were slowed in PD (Karayanidis, Andrews, Ward, & Michie, 1995). The benefit from pre-cueing in simple RT and choice RT tasks can be equal for PD and control groups, given a smaller number of alternatives with lower demands on higher cognitive levels of processing (Willingham, Korozhets, Treadwell, & Bennett, 1995).

In some studies prolongation of simple RT in PD has also been documented in conditions where stimuli and responses are predictable, with this impairment being susceptible to dopamine treatment (Henderson & Goodrich, 1993); these authors found that treatment was less demonstrable with simple RT than with choice RT. Deficits in performing concurrent choice reaction time tasks have been found to be dependent on the state of PD patients, which was interpreted as referring to the necessity of adequate dopaminergic transmission in concurrent processing of cognitive information (Malapani, Pillon, Dubois, & Agid, 1994).

4. Visuospatial information processing. Specific deficits such as impairment of facial recognition or visuoconstruction (Levin et al., 1991) or impairment of 3-D stereovision, figure-ground discrimination, and pattern perception (Flowers & Robertson, 1995) have been reported in PD. Similar cognitive impairments, for example, impairments in visual discrimination among 15 superimposed objects, and cognitive slowing that were found among a PD group have been found to be not treatable by levodopa (Pillon et al., 1989).

5. Divided attention. Accuracy of performing a dichotic attention test has been found unimpaired in PD regardless of overt response slowing (Karayanidis et al., 1995) and regardless of the findings by other researchers concerning impairments in set-shifting abilities (cf. below).

6. Top-down controlled attentional and/or productive cognitive processes and capacities. Attentional focusing (Bradshaw et al., 1993), word fluency (Flowers, Robertson, & Sheridan, 1995) and cognitive set-shifting ability (Raskin, Borod, & Tweedy, 1992; Van Spaendonck, Berger, Horstink, Borm, & Cools, 1995) have also been found to be impaired in PD.

Mixed findings have also been reported. For example, in experiments where PD subjects performed parallel and conjunction search tasks, no changes in the latency of the ERP component P300 were found, although the diminished ERP amplitude together with the finding of the specific site of the strongest effect was interpreted as indicative of parietal identification system involvement in PD dysfunctions (Weinstein, Troscianco, & Calvert, 1995). In attentional and movement programming tasks, strategic level operations have been reported to be impaired in PD (Jones et al., 1994). At the same time, in a study by Taylor, Saint-Cyr, and Lang (1987), cognitive processes at various stages were found not to be affected in PD if standard test methods were used.

7. Short-term memory scanning. Russ and Seger (1995) found that the slope of the memory scanning RT functions for words or pictures was unaffected in PD, showing the absence of slowing of elementary cognitive operations (lack of

bradyphrenia), although the intercept of the functions was increased, which is indicative of response system impairment. If motor components of the RT are disentangled from cognitive components of memory scanning, attentional orienting, or movement preparation, then RTs of a PD population were not different from those of a control group in the study by Rafal, Posner, Walker, and Friedrich (1984). Nevertheless, the decrease of memory scanning speed in PD can be found in other conditions. For example, when Wilson, Kaszniak, Klawans, and Garron (1980) used digits as stimuli and compared two age groups of PD subjects, then slowing of the scanning speed in comparison with the age-matched normal controls was found in the older group, but not in younger subjects.

8. Learning and working memory operations. Various findings can be listed here: impairment of procedural learning in PD (Jackson, Jackson, Harrison, Henderson, & Kennard, 1995); impairment of the speed of discrimination learning in PD, but without the concomitant deficit in the shift of the decision rule (Joosten, Coenders, & Eling, 1995); impairment of spatial working memory and cognitive planning whereby dopamine treatment restored the accuracy, but not the latency of performance (Owen et al., 1995).

In their recent meta-analytic review of the studies of the components of visual cognition in PD, Waterfall and Crowe (1995) point out that a number of methodological and theoretical faults characterize the respective research literature. Generalizing from 70 studies, they concluded that PD subjects tend to demonstrate deficits in higher-order attention and problem-solving tasks, but that evidence in favor of deficits in more basic visuoperceptual functions remains far from conclusive. Therefore, many observed deficits may be either a consequence of hidden lower level visual-cognitive deficits, a compromise in executive functioning, or both. Studies by Flowers and Robertson (e.g., 1995) add to this equivocity by showing that a mixture of bottom-up and top-down processes determine perceptual abnormalities in PD, depending on other factors such as severity of the stage of disease.

In analysing the body of research literature about the effects of PD on visual cognitive functions, we noticed that although there are several strongly established traditions of study, some obvious and theoretically important experimental approaches have not received sufficient attention. Whereas our main theoretical interest concerns the problem of possible impairments of the speed of elementary perceptual recognition operations in PD (especially without the need to involve manual reaction times as dependent measures) and, therefore, also relates to the problem of the hypothetical Parkinsonian bradyphrenia, we decided to concentrate our attention on finding out if there are any more or less definite data and conclusions with regard to this problem. If we regard the studies listed in items 1, 3, 6, and 7 above as closest to those capable of disclosing some basic information processing components and prerequisites of Parkinsonian bradyphrenia, it is not difficult to notice that it is premature to speak about definitive conclusions. Temporally distributed processes, processes with strong strategic and decision-making components, processes that are measured with manual reaction-time methods, and processes that take part within short-term memory or heavily rely upon complex memory processes are widely studied, whereas simple, predominantly bottom-up directed, single-act processes of visual recognition without the need to invoke fast manual responses are relatively neglected.

The studies that have been directed towards elementary processes of temporal resolution such as two-pulse integration or flicker fusion also lack the component of pattern or form recognition. In other words, the speed of the obvious, elementary perceptual recognition operations that could be an important aspect in the development of bradyphrenia in PD and that should be measured without heavy participation of higher-level cognitive processes have remained relatively unnoticed by researchers.

Those studies that have been directed towards the solution of the problem of bradyphrenia are (1) quite controversial, (2) deal mostly with the cognitive processes of top-down heritage and/or short-term memory, and (3) are based on methods that require manual responding by the subjects. In light of these specifications, the fundamental question remains as to whether one

can document impairment in bottom-up directed, perceptual-cognitive processes (including slowing of the elementary mental operations) in addition to motor- and planning-system impairments. Because a general behavioral outcome – say, slowing of perceptual recognition or discrimination performance – can be both the result of impairment in afferent and in efferent stages of information processing, then it would be useful to find experimental paradigms that are capable of disentangling these stages.

The basic strategy in studying the temporal aspect of information processing (including speed of processing) is based on the well-developed standard RT methods for studying the speed and temporal dynamics of perceptual processing in normal populations. However, this strategy seems to be inadequate for a Parkinsonian population due to the obvious basic central motor system impairment both at the executive and coordinating levels. Thus, methods capable of measuring the speed and time-course of perceptual-cognitive processing without the need to invoke fast manual or oculomotor responses and reaction time measurement are necessary.

Other desirable characteristics of such a method are (1) low demand on memory capacity (no more than two to three response alternatives of a highly overlearned nature per trial); (2) absence of image scanning by saccadic eye movements (see White et al., 1988, for hypometric saccades with diminished compensatory movements in PD); (3) the possibility to control the impact of possible retinal dopamine deficiency (see Flowers, Robertson, & Sheridan, 1987, on respective deficiency in PD); (4) good temporal resolution of the method and possibility to vary temporal variables parametrically. The method of mutual masking (Bachmann & Allik, 1976; Michaels & Turvey, 1979) satisfies these criteria well. Pairs of mutually different, but spatially overlapping visual stimuli are exposed successively, for a very short duration (e.g., 10 ms). Subjects are asked to recognize both stimuli at each trial. With gradually varying stimulus onset asynchrony (SOA) between the values of, say, 0 ms and 300 ms, recognition efficiency for the first stimuli in the pairs (S1) and for the second stimuli in the pairs (S2) can be plotted as a function of SOA. The values of SOA where the function of recognition of S1 approaches upper asymptote can be regarded as the indice of the speed of processing (the time value of the processing episode by which S1 can be successfully recognized without interference from the following S2).

The relative efficiencies of S1 and S2 at various SOA values are indicative of the perceptual system tendency to switch or not to switch processing operations from the previous input signals to the succeeding ones and/or of the tendency to process signals in parallel. Exposure of two small, spatially overlapping stimuli at the center of the visual field and within the brief time window helps to obviate the need to invoke purposeful eye movements.

Because both binocular (both stimuli exposed to both retinas) and dichoptic (S1 to one eye, S2 to the other eye) exposure regimes can be used, then retinal effects can be controlled. Only two required identity responses at each trial put no high demand on memory. Fast manual responses are eliminated from this procedure as well. Overlearned and relatively simple stimuli, such as letters, are common means to satisfy useful experimental requirements and to adapt automatized (computerized) experimental techniques that can be easily standardized between laboratories.

In Figure 1, typical results of mutual masking are seen in the conditions where the intensity of S1 exceeds that of S2 (function A: solid line with discs) or where respective intensities are more or less equal (function B: dashed line with triangles) (Bachmann, 1994). Intermediate SOAs are characterized by the dominance of S2 over Sl for perceptual processing regardless of the condition of their relative brightness. The values of SOA around 150 ms that are indicative of equalization of the values of S1 and S2 recognition functions close to upper asymptotic level can be regarded as an operational measure of the typical duration of the elementary visual recognition cycle.

If PD patients lag behind the normal control group in terms of the isoeffectiveness values of SOA, this outcome, if statistically significant, can be regarded as support for the regularity by

which perceptual recognition functions in PD are slowed. This constitutes our first and main hypothesis in this study. The second, additional hypothesis concerns the possibility to discriminate between the de novo PD group and a PD group that has had treatment. The third hypothesis is related to the tentative possibility to discriminate between two groups of PD patients differentiated according to their predominant symptoms – a tremor group and a hypokinetic rigidity group.

In an earlier study (Bachmann, 1994) we found mutual masking functions with hospitalized PD patients who were undergoing stereotactic treatment in the Department of Neurology and Neurosurgery of the Institute of Experimental Medicine of the Russian Academy of Medical Sciences (St. Petersburg). The patients participated in the tachistoscopic experiments immediately following the sessions of activating stimulation of the nonspecific thalamic nuclei via the implanted electrodes (Smirnov & Reznikova, 1985). Qualitatively unusual functions were found (Figure 2): PD patients tended to perceive Sl with unusual efficiency and PD patients did not produce typical predominance of S2 over S1 at intermediate SOA values in contrast with the normal control group and with the typical functions of other studies where the same experimental variables were used. ''Sticking to first stimulus'' could be the proper name of the effect.

Unfortunately, it cannot be said at present if this effect was a result of PD or a result of activating thalamic stimulation. If it was a result of PD, one could think along the lines of developing an early diagnostic test for the proneness to develop PD symptoms – unusual mutual masking functions could indicate the presence of potential risk factor(s). If it is a result of activating treatment via nonspecific thalamic nuclei, it gives additional converging evidence for the

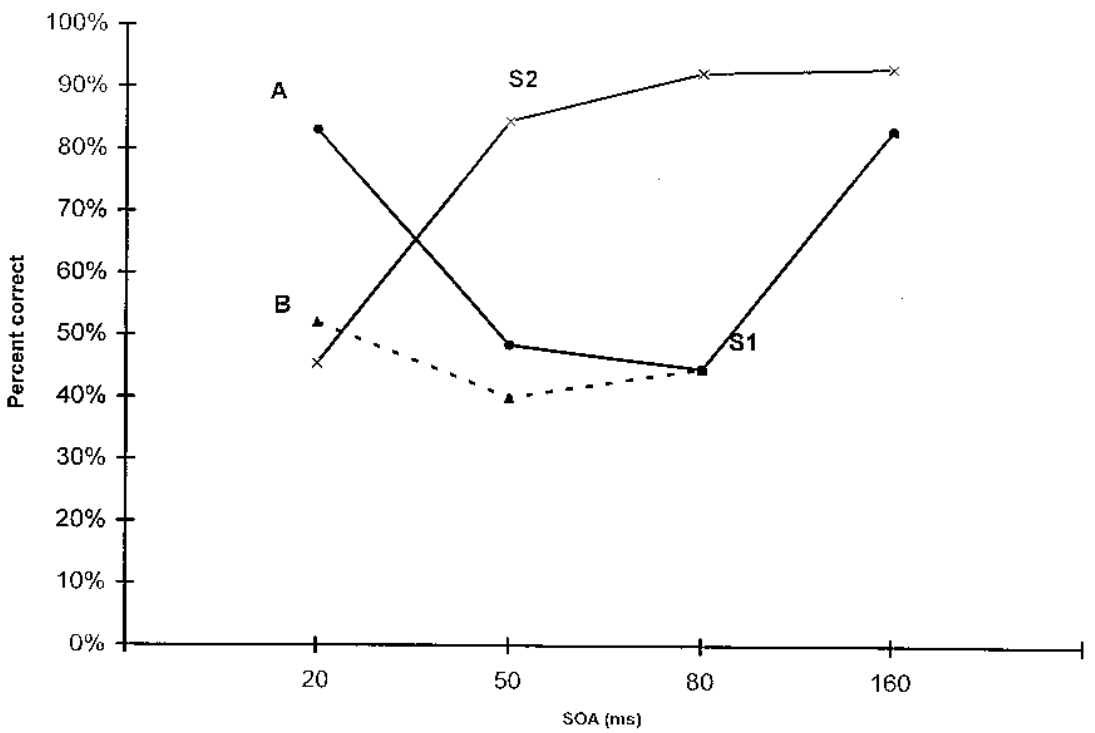


Fig. 1. Typical mutual masking functions (percent correct recognition as dependent on SOA) for the pairs of successively exposed, spatially overlapping visual stimuli, S1 and S2. A: the intensity of Sl exceeds that of S2 (solid line, discs). B: the intensities of S1 and S2 are compatible (dashed line, triangles).

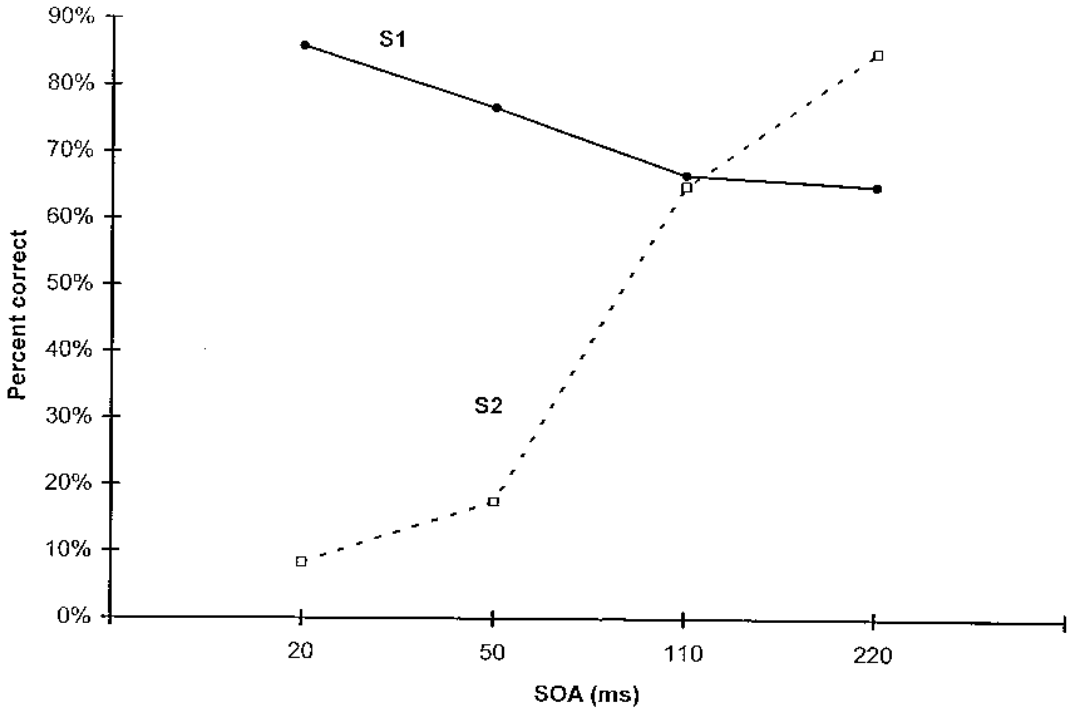


Fig. 2. Unusual mutual masking functions obtained with PD patients whose nonspecific thalamic nuclei were stimulated by chronically implanted microelectrodes for treatment purposes: S1 is strongly dominating over S2 and intermediate SOAs do not lead to S2 prevalence. (Adapted from Bachmann, 1994.)

perceptual retouch theory of visual masking (for details see Bachmann, 1984,1988,1994).

One of the objectives of the present study was to test these alternative predictions. Hence, our fourth specific hypothesis inquires if the PD group could display qualitatively different mutual masking functions as compared to the control group. This should reveal itself in statistically significant interaction between the factors of health status and SOA and/or first or second stimulus (Sl vs. S2).

EXPERIMENT 1

METHOD

Thirty-seven PD patients and 19 age-matched normal controls were employed as subjects in this study. The patients were recruited from among those who were subject to PD treatment in the Department of Neurology and Neurosurgery at the University of Tartu or the Neurology Department at the Mustamäe Hospital of Tallinn. Informed consent was obtained. Participants were assigned to experimental groups according to their treatment history ((1) de novo without dopaminergic medication – 21 subjects, 11 males and 10 females aged between 38 and 81 years; (2) medicated – 16 subjects, 8 males and 8 females aged between 51 and 82 years) and according to predominant symptoms ((1) hypokinetic rigidity – 15 subjects, 8 males and 7 females aged between 43 and 75 years; (2) tremor – 16 subjects, 8 males and 8 females aged between 44 and 82 years). Two PD subjects belonged to stage 3.0; 5 to 2.5; 11 to 2.0; 7 to 1.5; and 12 to stage 1.0 according to the Hoehn and Yahr scale. The Wilcoxon-Mann-Whitney U-test showed that the distribution of de novo and treated patients between different Hoehn and Yahr PD stages was statistically unbiased (sum of inversions was equal to 163, $p > 0.05$; scale averages were: 1.61 for de novo patients, and 1.81 for

treated patients). All subjects had normal or corrected to normal vision. All subjects participated in the mutual masking experiment. In addition PD patients completed the standard memory test from the Luria test battery in order to control the possible memory deficits. The subjects in the present study had unimpaired memory capacity in terms of reproducing the first two remembered stimuli from the list. This was necessary in order to ascertain if the subjects would be capable of performing the two-letter recognition without limitations from the immediate memory-related aspect of processing.

In the mutual masking experiment the standard IBM PC was used for stimulus exposure. Pairs of mutually different letters from the English alphabet were chosen at random for each exposure trial. Letters were exposed for 25 ms each. They appeared successively at the same, overlapping central area immediately above the fixation point. The following values of stimulus onset asynchronies (SOAs) were used, each value for an equal number of times: 25 ms, 40 ms, 55 ms, 95 ms, 165 ms, or 285 ms. Letters were light on dark background. The luminance of the background equalled 5.16 cd/m^2, the point luminance of the first letter (S1) equalled 64.2 cd/m^2 and that of the second letter (S2) 35.5 cd/m^2. The size of the letter was equal to 0.6 degrees of the visual angle along the vertical dimension. Each subject was given 40 exposures of the letter pairs for each of the SOA values, thus 240 exposures in toto. The task of the subjects consisted in recognition of both letters in a pair according to the forced response requirement. (subjects had to guess if unsure.)

Procedure

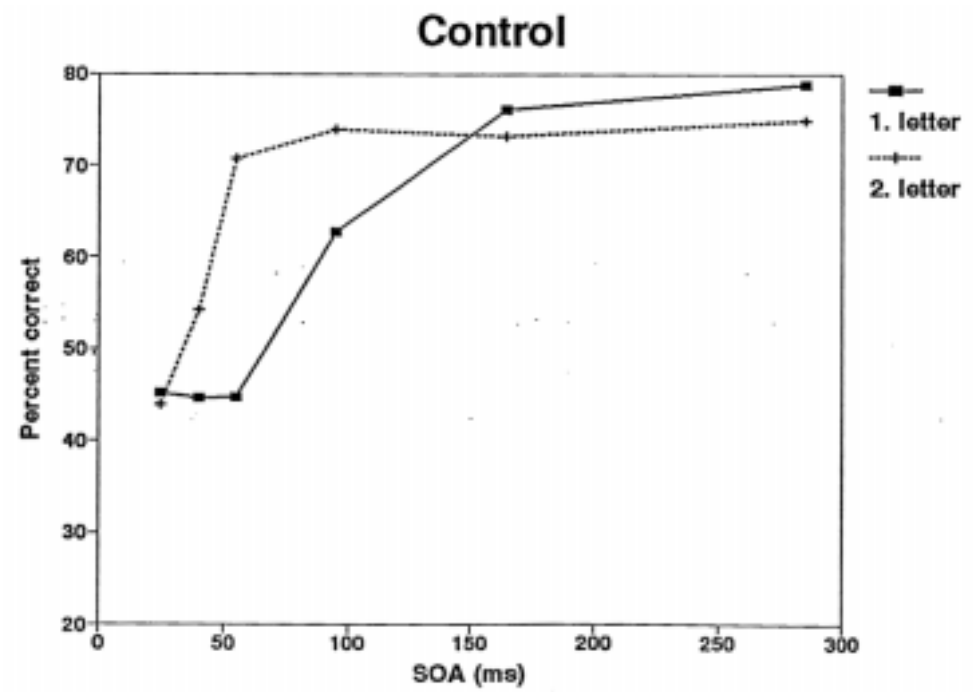
The subject was seated in front of the computer display and the task was explained to him/her. It was emphasized that responses are required for both of the letters in each pair and if unsure, the best guess(es) should be made. Each trial began with an oral warning, then a small lighted fixation dot appeared for 1000 ms at the center of the display, followed by the exposure of the stimuli immediately above the fixation point. Responses were recorded by the experimenter's assistant via the computer keys. Order of SOAs was randomized by the computer. Subjects were allowed periods of rest if necessary. Conditions of light adaption were not changed.

RESULTS AND DISCUSSION

As one can observe from Figure 3 both the control group (Figure 3 A) and the PD group (Figure 3 B) produced typical recognition functions for S1 vis-à-vis S2 – the clear advantage of S2 recognition at intermediate SOAs is evident. The interaction between SOA and temporal order of the stimuli (S1 vs. S2) was highly significant [F (5,443) = 10.270; $p < .001$]. Two types of general highly significant main effects were revealed by ANOVA. First, the effect of health status shows that the control group performed significantly better than did the PD groups [F (2,335) = 20.397; $p < .001$ for Sl recognition and F (2,335) = 11.033; $p < .001$ for S2 recognition]. Comparing Figure 3A and Figure 3B, it takes about 50 ms increase of SOA for the PD patients to reach the level of Sl recognition that is equal to that of the normal control group. If with SOA = 165 ms in the normal control group the efficiency of S1 recognition already exceeds that of S2 (76 and 74 %, respectively) then in PD groups S1 recognition level has not reached that of S2 (62 and 67 %, respectively). Second, the effect of the SOA shows that recognition efficiency increases with SOA both for Sl [F (5,335) = 45.828; $p < .001$] and for S2 [F (5,335) = 24.112; $p < .001$].

There was no significant interaction between health status and SOA neither for S 1 recognition [F (10,335) = 0.546; $p = 0.85$], nor for S2 recognition (F (10,335) = 0.162; $p = .99$), however. As one can see on Figure 4 (A,B), the effect of health status is additive across SOA values. This finding, together with the clear predominance of S2 recognition over S1 recognition at intermediate SOAs that is even stronger in the PD groups than in the control group (cf. Figure 3) invalidates our hypothesis about possible qualitative differences in mutual masking functions of PD patients (both treated and de novo) as compared to normal control. PD patients did not display abnormally high relative levels of S1 recognition in comparison with S2 recognition as was found by Bachmann (1994). At intermediate SOAs respective Sl and S2 recognition levels in the PD group in the present study were 35% versus 52% for SOA = 40 ms,

A.



B.

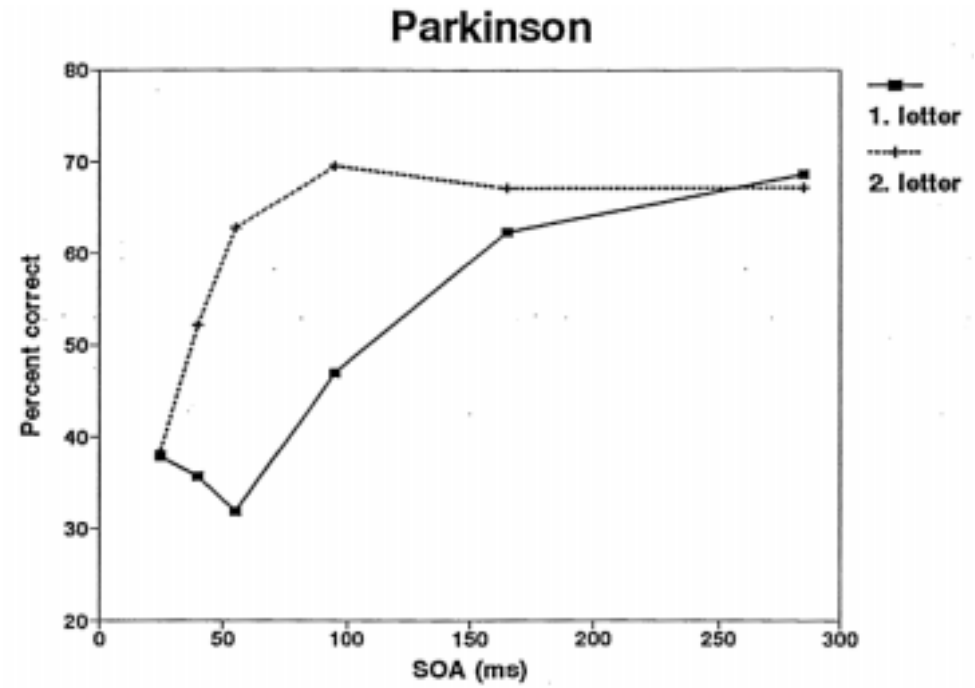
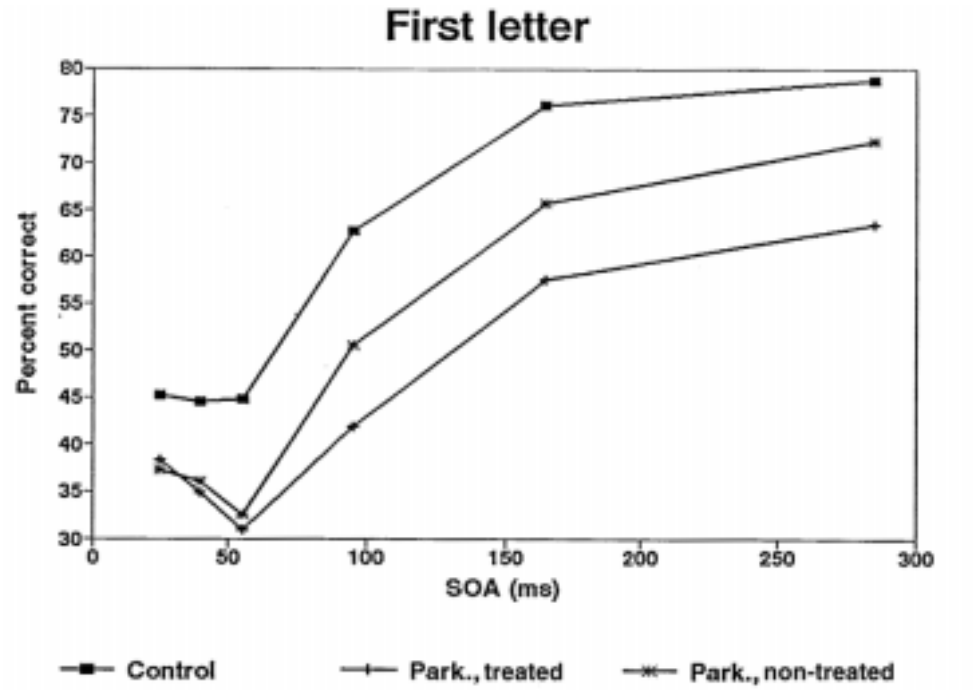


Fig. 3. Efficiency of recognition of first and second letters (solid line for S1 and dashed line for S2, rerspectively) in successively exposed pairs as a function of SOA for the control group (A) and for the Parkinson's disease group (B); Experiment 1.

A.



B.

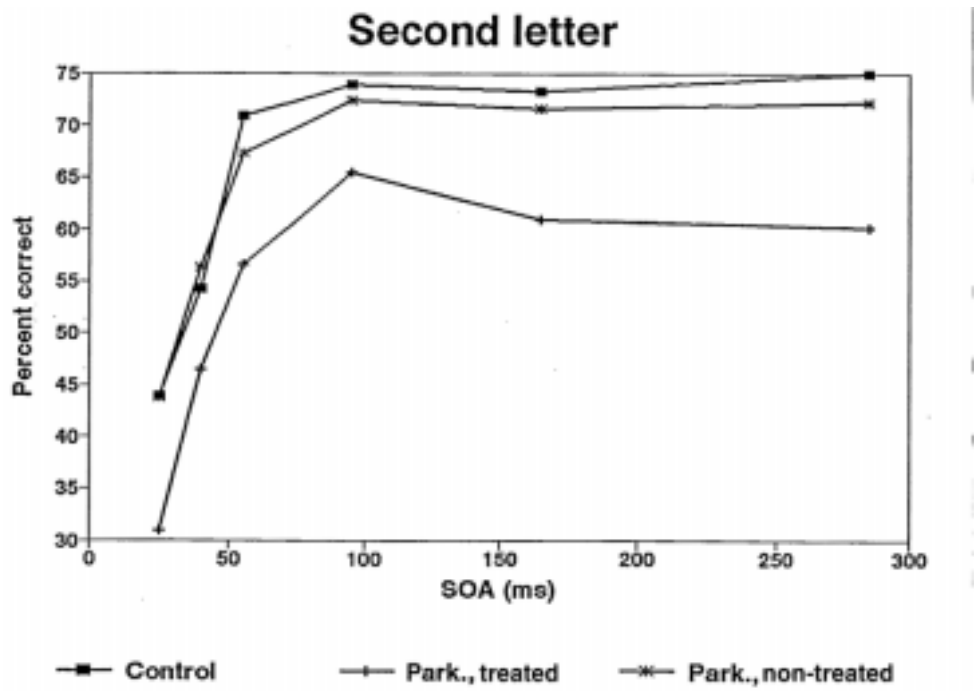


Fig. 4. A: Efficiency of recognition of S1 as a function of SOA and treatment status (de novo, treated, and normal control). B: Efficiency of recognition of S2 as a function of SOA and treatment status. Data from Experiment 1.

31% versus 62% for SOA = 55 ms, and 47% versus 69% for SOA = 95 ms. Thus that earlier effect should most probably have been the result of direct thalamic activation through implanted electrodes.

A significant interaction between the temporal position of a stimulus letter in a pair (S1 vs. S2) and health status was found between the control group and de novo group [F (l,479) = 5.136; $p < .02$]: the effect of PD was clearcut primarily in impairment of S1 recognition, but not in S2 recognition (compare respective functions on Figures 4A and B).

This again substantiates the thesis that what matters first of all is speed of recognition operations that can be executed on perceptual evidence of S1 before the succeeding, interfering letter (S2) arrives. We sought for the analoguous ANOVA interaction between the control group and the treated PD group but did not find a significant effect [F (1,419) = 0.448; $p = .50$], although the main effect – impairment of recognition in the treated PD group – was significant across all SOAs both for S1 recognition [F (1,209) = 36.430; $p < .001$] and for S2 recognition [F (1,209) = 18.175; $p < .001$]. If our PD group with treatment were equal to our de novo PD group in terms of average rating according to the Hoehn and Yahr scale (see the Method section), then we speculate that the baseline Hoehn and Yahr scale rating in the treated PD group may be actually higher, but is partly alleviated by treatment.

To test if this might be the reason why the treated group performed worse than the de novo group (especially in comparison with control group if S2 was to be recognized) should be a possible task in future studies where the experimental design with before-after medication scheme within the treated group should be used. At present we cannot assign this effect exclusively either to medication or to baseline stage of PD, however. What we can say is that PD has a detrimental effect on fast recognition regardless of medication status (cf impairment of S1 processing).

The switching to S2 processing is not impaired in our group of patients with relatively mild, nontreated PD (cf. equal level of S2 recognition functions in nontreated and control groups).

We found also a significant main effect of the Hoehn and Yahr ratings both for S1 recognition and S2 recognition [F (1,221) = 30.485; $p <$ .001, and F (1,221) = 41.250; $p < .001$, respectively]. There were significant negative correlations between Hoehn and Yahr ratings and recognition efficiency: the stronger the PD symptoms, the more impaired the recognition level (r = – 0.237; $p < .001$ for Sl, and r = –0.307; $p <$ .001 for S2). There were no significant interactions between the Hoehn and Yahr scale ratings and any other factors, however. The PD effects seen in these ratings were uniformly additive across SOA values and differently positioned stimuli. If one expects increased difficulties in shifting attention from S1 to S2 with the advancement of PD, then interaction between scale ratings and the position of stimuli could be found. Because this effect was absent, we conclude that either the stages of PD of our patients were not sufficiently high or this experimental paradigm is not a proper means to test the shift of attention that is related to variable mental set to simple characteristics of stimulation like temporal position of stimulus order. (It may also be that set-shifting effects in PD in principle can be found only with more complex or more abstract inter-categorical or with incompatible inter response-category experimental designs.)

We did not find significant differences between the performance of two different groups of predominant symptoms – tremor versus hypokinetic rigidity – in recognizing S2 [F (1,185) = 0.336; $p = .56$], but there was a significant main effect of symptom category if Sl had to be recognized: the hypokinetic rigidity group performed at a higher level than did the tremor group uniformly at all SOAs [F (1,185) = 4.221; $p < .04$]. Future studies should look for the possible causes and underlying mechanisms of this effect. There were no significant interactions between predominant symptoms and SOA neither with S1 recognition, nor with S2 recognition [F (5,185) = .042; $p < 0.99$, and F (5,185) = 0.009; $p = 1.0$, respectively].

There were no significant effects of medication type. ANOVA that contrasted Madopar and

Nakom treatment revealed F (1,191) = 2.321; p = 0.13. The Luria memory test results showed no significant main effects or interactions with other factors. The average memory test index was equal to 0.765.

In the present experiment both successive stimuli were exposed to the same retinas of the subjects, thus making it possible that interactions between competing processing operations can begin already at the peripheral sensory level. In the study by Bachmann (1994), a similar binocular exposure regimen was used, yet unusual functions without S2 prevalence were found.

It would be useful to see, whether we could obtain that type of unusual mutual masking function if we exclude retinal interactions between the stimuli. Another difference between our Experiment 1 and that of Bachmann (1994) was the number of stimulus alternatives. Bachmann (1994) used only five different geometric forms but the whole alphabet was employed in Experiment 1. Thus in addition to dichoptic regime we may want to decrease the number of stimulus alternatives as well. It is also known (e.g., Flowers, 1987), that in PD subjects retinal dopamine deficiency is a common finding.

Therefore it is not possible to ascribe the obtained recognition functions unambiguously to either peripheral or central levels of processing. Dichoptic tachistoscopic exposures would be necessary for this reason as well in order to see if the same qualitative picture would appear in the results if the interaction between the stimuli takes place only beginning with cortical levels of processing. By decreasing the number of response alternatives we also diminish the possible effects of capacity limitations for memory search. To test the possible impact of these factors and to seek other conditions for obtaining the potentially important, qualitatively unusual functions with S1 prevalence (possibility to develop diagnostic methods!) another experiment was completed.

EXPERIMENT 2

METHOD

12 PD patients (8 de novo, 4 treated) aged between 42 and 82 years were used as subjects. The basic rationale of the experiment was similar to that of Experiment 1, except the following differences. In this experiment stimuli were exposed by the means of five-channel tachistoscope (manufactured by TSU Experimental Construction Shop) dichoptically (one stimulus in a pair to one eye, the other stimulus of the same pair to the other eye). Sl was exposed for 30 ms with background intensity equal to about 3 cd/m^2, S2 was exposed for 15 ms. Stimuli consisted of dark capital letters – H, M, R, W, K, or B – on light ackground and were presented at the center of the visual field. The center was designated by the small fixation dot. The size of the stimulus letters was equal to 0.72 degrees of the visual angle along the vertical dimension. After the ready-signal in each trial fixation was exposed for 1000 ms, followed by the two successive, spatially overlapping stimuli. The SOA values used were 0 ms, 20 ms, 50 ms, 90 ms, 140 ms, or 260 ms.

Procedure

Each subject received 30 successively paired Sl and S2 exposures at each SOA value. SOA values were counterbalanced within and between subjects. Brief periods of rest were allowed when necessary, however without change in light adaptation. After each exposure subjects responded with reporting the identities of both letters they saw, guessing if unsure. Their responses were recorded with the aid of the computer by the experimenter's assistant.

RESULTS

As it can be seen from Figure 5, both groups of subjects – treated PD, de novo PD – have produced in dichoptic conditions mutual masking/recognition functions that are qualitatively typical to this type of interaction: prevalence of S2 at intermediate SOAs, and general increase of efficiency with increase in SOA. The different dynamics of Sl and S2 recognition is substantiated by the significant ANOVA interaction between SOA and ordinal position of a stimulus: F (5, 1074) = 15.97; p < 0.0001 for de novo PD patients; F (5,1074) = 12.57; p < 0.0001 for treated PD patients. (Due to the small number of

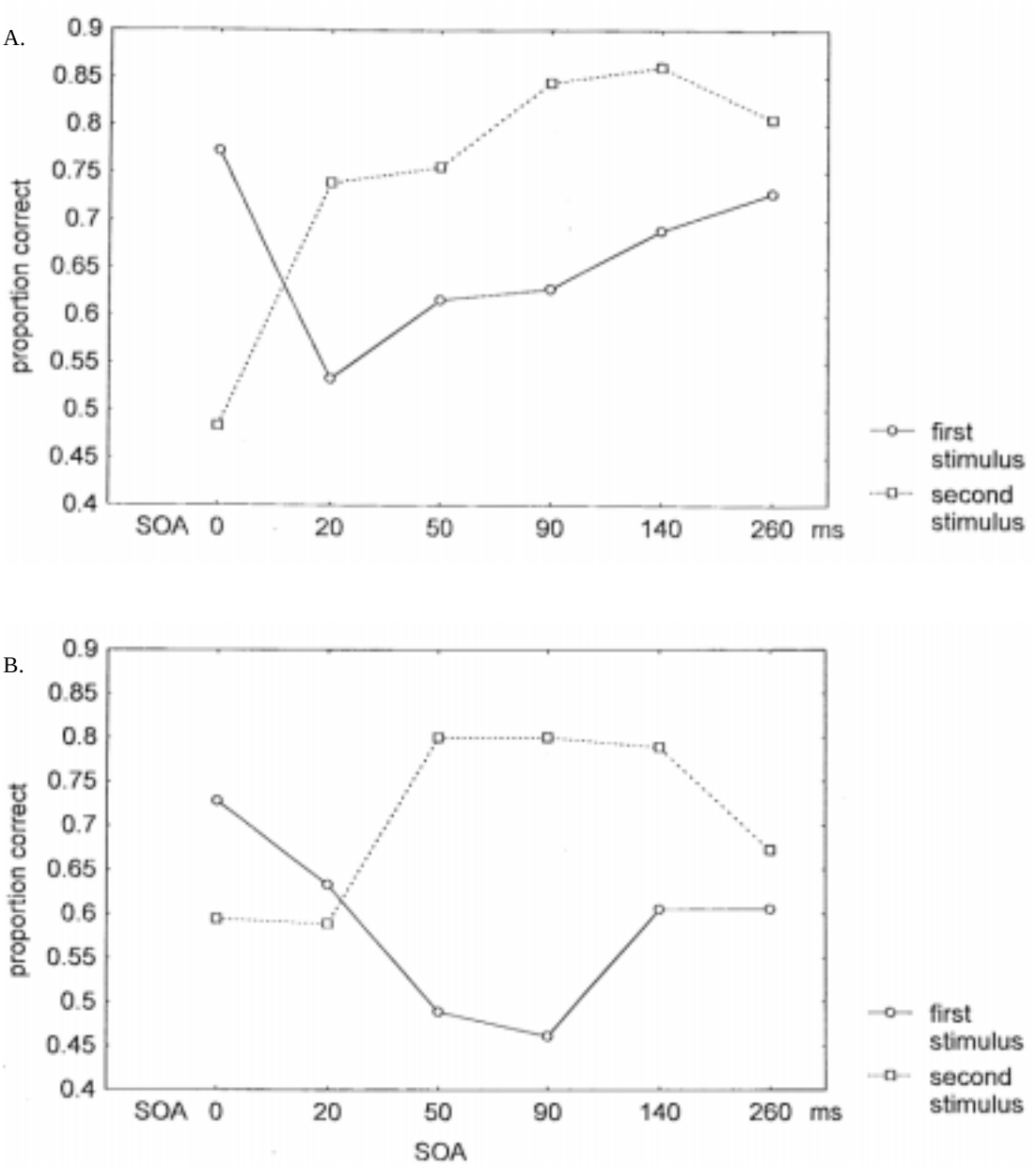


Fig. 5. Efficiency of recognition of S1 (solid line) and S2 (dashed line) as a function of SOA for de novo patients (A) and treated patients (B) in dichoptic exposure regime; Experiment 2.

subjects in this experiment we included all responses of respective subjects' groups to the ANOVA in contrast to Experiment l, where averages of different subjects were forming the entrance table for ANOVA.) Thus we can conclude that this qualitative picture is most probably of the central processing level origin and that unusual mutual masking functions with Sl

facilitation relative to S2 found in Bachmann (1994) were not the result of PD (either pharmacologically treated or not), but the result of intracranial electrical thalamic stimulation that immediately preceded the tachistoscopic experiment.

GENERAL DISCUSSION

The main feature of our present study consisted of the absence of the need to execute fast manual or ocular movements by the subjects in order to measure the speed of elementary perceptual-cognitive processes. Yet the results of the Experiment 1, by the virtue of juxtaposing Sl and S2 processing (as collated via parametrically varied SOA values and thus indirectly measuring the time course of processing), provide support for the hypothesis about slowing of elementary visual recognition processes in PD. In the context of this result the findings of Artieda et al. (1992) and Bodis-Wollner (1987) who reported the impairment of simple temporal resolution in PD seem to be hinting at the possible basis of our obtained perceptual slowing. If elementary processes of temporal integration in the PD subject's visual system are slowed, then also the perceptual evidence for recognition operations should be accumulating more slowly and it would take longer SOAs to reach at some critical levels of Sl recognition in comparison with normal subjects.

It seems to us, however, that – given impairments in spatiotemporal integration – it is still difficult to interpret why S2 recognition also takes longer in treated PD subjects. If it were simply for stronger forward masking as a result of longer temporal integration of the luminous energy from Sl that interferes with S2 processing, then we would obtain the interaction between health status and the temporal position of stimulus exactly in the opposite direction – relatively larger impairment of S2 recognition in PD. That was not the case (see the Results section of Experiment 1). These facts together with the clearly observable switch from Sl processing to S2 processing (see Figure 3 about the prevalence of S2 over S1 at intermediate SOAs in PD group and notice that this prevalence is not less expressed than in normal control) suggest that simple impairment of the pre-recognition processes of fast temporal integration of luminous energy need not be the only cause of the slowing. We may thus conclude that our results of Experiment l lend support to the standpoint that central, elementary perceptual-cognitive operations are impaired in PD and that medication may partly restore the distribution of resources between Sl and S2 in relative terms, but not in absolute terms. That these effects are not caused by general memory deficit can be derived from the fact that the results of Luria memory test were quite good and that only two responses of reporting highly overlearned stimuli were required from the subjects at each trial.

Various examples of decreased visuospatial information processing in PD have been presented (e.g., Pillon et al., 1989; Levin et al., 1991; Flowers and Robertson, 1995; see also Waterfall and Crowe, 1995). It would thus be possible to hypothesise that the effect of PD on recognition found in the present study could considerably or partly stem from certain general or specific spatial perceptual disabilities. If simple, overlearned stimuli are presented in invariant spatial position then it is highly improbable that spatial orientation deficiencies form the basis of the obtained effects. The general ability to perceive forms and execute recognition operations should not be impaired as well, because the functions at S2 recognition were not much different in PD as compared with normal controls.

Cummings and Huber (1992) proposed to divide visuospatial functioning in the context of Parkinsonian research into six categories: (1) visual sensory functions, (2) visual perceptual skills, (3) visuomotor abilities, (4) visuospatial attention, (5) visuospatial cognition, and (6) body-spatial orientation. Again, relying on unimpaired efficiency of S2 recognition, invariance of spatial position of the stimuli, and bottom-up nature of the processing task in our experiments none of the listed subcategories could be regarded as the site of the impairment that was found in this study. In the similar, but relatively more thorough list of 12 categories that was proposed by Waterfall and Crowe

(1995) the only item that is relevant in order to identify the visual cognitive function that seems to be impaired in our perceptual task is their item 5: perception and recognition of shapes (and faces). And even here we should introduce a constraint by specifying the function as that of fast or speeded shape recognition.

In a recent study, Russ and Seger (1995) found that in a memory-scanning experiment with words and pictures as stimuli, reaction time slopes (increments in RT per unit of increase in the size of the memory set) of PD subjects and controls do not differ. The authors conclude that although the speed of executive components may be decreased in PD (the intercept of the RT function indicating the generally slower responses in PD), the speed of cognitive operations need not: switching between the successively matched alternatives in memory-set proceeds equally fast in PD and in control groups. This variety of the lack of bradyphrenia somewhat contradicts our present results. We again draw attention to one important difference between their study and our study: while Russ and Seger used a task that had strong top-down component of processing and operations relevant to their theoretical interest took part in short-term memory, then in our task the principal processing route consisted in bottom-up direction. This fact, together with the observations by Weinstein, Troscianko, and Calvert (1995) who found that in PD-subjects diminished ERP components tend to be especially localized in parietal sites, suggests that the locus of slowing and/or impairment could be assigned to identification and perceptual classification system.

On the other hand, Wilson et al. (1980) have found that if PD subjects perform memory scanning with relatively more simple stimuli – digits – then in the older group of PD subjects the effect of slowing obtains. It could be that indications of bradyphrenia in relatively mild stages of PD can be better disclosed in conditions where extreme rapidity of processing is at hand either due to simpler perceptual material (as in Wilson et al., 1980) and/or due to experimental design that enforces speeded processing (as in our study, where arrival of S2 after Sl constrains the time-window of recognition-processing and perceptual shape formation).

The effects of predominant symptoms (tremor vs hypokinetic rigidity) cannot be discussed in a clearcut fashion. The small effect with S1 recognition may include a cofound from the medication status and/or Hoehn and Yahr scale evaluation. This should be a matter of respective follow-up studies. Lack of the effects of medication status (Nakom vs Madopar treatment) indirectly refers to cognitive sites of the obtained perceptual slowing. It should also be clear that standard levodopa medication that is targeted at alleviating dopaminergic deficiencies, although through somewhat different drugs, need not have significantly different effects on recognition processes unless we specifically control the temporal dynamics of the drug effects and especially test the effects of these respective time-patterns on recognition performance. (Another dimension of potential interest that was left out of the present study was the potential impact of the on-off dynamics of the parkinsonian states.)

In the present experiments mostly the PD subjects who belonged to the stages from the 1st to 2nd of PD (Hoehn and Yahr scale) were employed. Thus, if combined with the absence of substantial complications in the planning or executive components in the present experimental task, the potential effects of pharmacological treatment and predominant symptoms need not manifest themselves very strongly. On the other hand, several authors (e.g., Pillon et al., 1989) have shown that cognitive impairment in PD patients is not correlated with symptoms that are treated by levodopa. (Also, for example, careful inspection of the study by Daniels, Harding, & Anderson, 1994, reveals that reduced amplitude of the N2-P2 component of the ERP in response to luminous flash in PD subjects can be obtained with dopaminergic and/or anticholinergic treatment.) Accordingly we may hypothesise that some indirectly affected systems out of the basal ganglia might be involved.

Indirect support to this view comes from Bachmann (1994) where mutual masking functions change substantially if thalamic nonspecific nuclei of the patients are stimulated. The

phenomenon of ''sticking to the first stimulus'' found in that study may mean at least one of the two different scenarios, or both: (1) unusually fast and ''contrasted'' perceiving of Sl and respectively larger spending of the share of limited capacy attentional resources for the fast processing of S1; (2) inhibition of the attentional switching and/or novel stimulus detecting mechanisms or perturbations in the mental shift of set that usually would push the system from S1 to S2. The latter possibility is supported by the finding of Pekkonen et al. (1995) who showed that in PD the mismatch negativity – the early automatic, negative deflection of ERP in response to novel or changed stimulus, cf. Näätänen (1992) – is diminished in PD patients.

In our results this idea is supported by the fact that nontreated patients, whose baseline PD stage is most probably lower than in treated PD (whose average Hoehn and Yahr rating was now equal to that of the nontreated group) performed very well on S2 recognition (which is possible if efficient switch from Sl was made), but not so well on Sl. Larger values of negative correlation coefficients between recognition efficiency and the value of Hoehn and Yahr ratings were found for S1 recognition than for S2, which supports our statement. Whether our results are related to automatic or focal-attentional components of perceptual-cognitive processing would require some special studies, however.

Although slowing in elementary visual recognition operations in PD was found in Experiment 1, the effects are by no means very dramatic. The general pattern of results demonstrates that patients are quite good at performing the task of recognition of two consecutive, very brief visual stimuli that alternate within very short temporal window. The same regularity whereby the perceptual system tends to favor the following one from the two rapidly alternating inputs characterizes both the normal control group and the PD patients regardless of the treatment history and predominant symptoms of the latter group. The results of Experiment 2 show that this regularity is based on central processes and not on any masking artefacts of the retinal origin. With dichoptic exposures the factor of eye dominance would require much larger number of subjects in order to avoid the possible eye dominance artefact if we would like to repeat Experiment 1 in dichoptic conditions. As a matter of fact, since the factorial combination of (1) inputs to the right and left eye, and (2) temporal order of exposure was balanced in Experiment 2, then subjects with strong eye dominance should have suffered more from the dichoptic regimen than subjects with weak dominance factor. This question needs to be analysed in some special future research.

The not so dramatic, however highly significant from the statistical point of view, effect of perceptual slowing/impairment in PD together with the absence of any striking qualitative differences in the mutual masking functions of S1 vis-à-vis S2 of PD patients as compared to normal controls (although the results of Bachmann, 1994, hinted towards this possibility) leaves us with the conclusion that it would be premature and unjustified at present to expect the development of a diagnostic or classificatory method of PD based on the mutual masking paradigm or any other similar visual processing paradigms. On the other hand, given that patients who can be rated as having more advanced stages of PD will be studied and/or given the possibilities of more purposeful and systematic psychopharmacological intervention be involved, it seems justified to continue some basic science studies that are based on the time-course paradigms that abandon necessity to use manual reactions on the one hand and too simple temporal resolution methods on the other.

REFERENCES

Artieda, J., Pastor, M.A., Lacruz, F., & Obeso, J.A. (1992). Temporal discrimination is abnormal in Parkinson's disease. *Brain, 115,* 199-210.

Bachmann, T. (1984). The process of perceptual retouch: Nonspecific afferent activation dynamics in explaining visual masking. *Perception and Psychophysics, 35,* 69-84.

Bachmann, T. (1988). Time course of the subjective contrast enhancement for a second stimulus in successively paired above-threshold transient forms: Perceptual retouch instead of forward masking. *Vision Research, 28,* 1255-1261.

Bachmann, T. (1994). *Psychophysiology of visual masking: The fine structure of conscious experience.* Commack, NY: Nova Science.

Bachmann, T., & Allik, J. (1976). Integration and interruption in the masking of form by form. *Perception, 5,* 79-97.

Bodis-Wollner, I., Marx, M.S., Mitra, S., Bobak, P., Mylin, L., & Yahr, M. (1987). Visual dysfunction in Parkinson's disease: Loss in spatiotemporal contrast sensitivity. *Brain, 110,* 1675-1698.

Bradshaw, J.L., Waterfall, M.L., Phillips, J.G., Iansek, R., Mattingley, J.B., & Bradshaw, J.A. (1993). Re-orientation of attention in Parkinson's disease: An extension to the vibrotactile modality. *Neuropsychologia, 31,* 51-66.

Brown, V.J., Schwarz, U., Bowman, E.M., Fuhr, P., Robinson, D.L., & Hallett, M. (1993). Dopamine dependent reaction time deficits in patients with Parkinson's disease are task specific. *Neuropsychologia, 31,* 459-469.

Cummings, J.L., & Huber, S.J. (1992). Visuospatial abnormalities in Parkinson's disease. In S.J.Huber & J.L.Cummings (Eds.), *Parkinson's disease: Neurobehavioural aspects* (pp. 59-73). New York: Oxford University Press.

Daniels, R., Harding, G.F.A., & Anderson, S.J. (1994). Effect of dopamine and acetylcholine on the visual evoked potential. *International Journal of Psychophysiology, 16,* 251-261.

Flowers, K.A. (1987). Parkinsonism. In R.L. Gregory (Ed.), *The Oxford companion to the mind* (pp. 587-591). Oxford: Oxford University Press.

Flowers, K.A., & Robinson, C. (1995). Perceptual abnormalities in Parkinson's disease: Top-down or bottom-up processes? *Perception, 24,* 1201-1221.

Flowers, K.A., Robertson, C., & Sheridan, M.R. (1995). Some characteristics of word fluency in Parkinson's disease. *Journal of Neurolinguistics, 9,* 33-46.

Henderson, L., & Goodrich, S.J. (1993). Simple reaction time and predictive tracking in Parkinson's disease: Do they converge on a single, fixed impairment of preparation? *Journal of Motor Behavior, 25,* 89-96.

Jackson, G.M., Jackson, S.R., Harrison, J., Henderson, L., & Kennard, C. (1995). Serial reaction time learning and Parkinson's disease: Evidence for a procedural learning deficit. *Neuropsychologia, 33,* 577-593.

Jones, D.L., Bradshaw, J.L., Phillips, J.G., Iansek, R., Mattingley, J.B., & Bradshaw, J.A. (1994). Allocation of attention to programming of movement sequences in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology, 16,* 117-128.

Joosten, J.P.A., Coenders, C.J.H., & Eling, P.A.T.M. (1995). Shifting behavior: An analysis of response patterns of Parkinson patients in discrimination learning. *Brain and Cognition, 29,* 115-126.

Jordan, N., Sagar, H.J., & Cooper, J.A. (1992). Cognitive components of reaction time in Parkinson's disease. *Journal of Neurology and Psychiatry, 55,* 658-664.

Karayanidis, F., Andrews, S., Ward, P.B., & Michie, P.T. (1995). ERP indices of auditory selective attention in aging and Parkinson's disease. *Psychophysiology, 32,* 335-350.

Levin, B.E., Llabre, M.M., Reisman, S., Weiner, W.J., Sanches-Ramos, J., Singer, C., & Brown, M.C. (1991). Visuospatial impairment in Parkinson's disease. *Neurology, 41,* 365-369.

Malapani, C., Pillon, B., Dubois, B., & Agid, Y. (1994). Impaired simultaneous cognitive task performance in Parkinson's disease: A dopamine-related dysfunction. *Neurology, 44,* 319-326.

Michaels, C.F., & Turvey, M.T. (1979). Central sources of visual masking: Indexing structures supporting seeing at a single, brief glance. *Psychological Research, 41,* 1-61.

Näätänen, R. (1992). *Attention and brain function.* Hillsdale, NJ: Lawrence Erlbaum.

Owen, A.M., Sahakian, B.J., Hodges, J.R., Summers, B.A., Polkey, C.E., & Robbins, T.W. (1995). Dopamine-dependent frontostriatal planning deficits in early Parkinson's disease. *Neuropsychology, 9,* 126-140.

Pekkonen, E., Jousmäki, V., Reinikainen, K., & Partanen, J. (1995). Automatic auditory discrimination is impaired in Parkinson's disease. Paper submitted to *Electroencephalography and Clinical Neurophysiology EEG Journal, 95,* 47-52.

Pillon, B., Dubois, B., Cusimano, G., Bonnet, A.-M-, Lhermitte, F., & Agid, Y. (1989). Does cognitive impairment in Parkinson's disease result from non-dopaminergic lesions? *Journal of Neurology, Neurosurgery, and Psychiatry, 52,* 201-206.

Pillon, B., Dubois, B., Bonnet, A.-M., Esteguy, M., Guimaraes, J., Vigouret, J.-M., Lhermitte, F., & Agid, Y. (1989). Cognitive slowing in Parkinson's disease fails to respond to levodopa treatment: The 15-objects test. *Neurology, 39,* 762-767.

Rafal, R.D., Posner, M.I., Walker, J.A., & Friedrich, F.J. (1984). Cognition and the basal ganglia: Separating mental and motor components of performance in Parkinson's disease. *Brain, 107,* 1083-1094.

Raskin, S.A., Borod, J.C., & Tweedy, J.R. (1992). Set-shifting and spatial orientation in patients with Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology, 14,* 801-821.

Russ, M.O., & Seger, L. (1995). The effect of task complexity on reaction times in memory scanning and visual discrimination in Parkinson's disease. *Neuropsychologia, 33,* 561-575.

Smirnov, V.M., & Reznikova, T.N. (1985). Artificial stable functional relations as a method of research and treatment [in Russian]. *Vestnik Akademii Meditsinskikh Nauk SSSR, 9,*18-23.

Taylor, A.E., Saint-Cyr, J.A., & Lang, A.E. (1987). Parkinson's disease: Cognitive changes in relation to treatment response. *Brain, 110,* 35-51.

Van Spaendonck, K.P.M., Berger, H.J.C., Horstink, M.W.I.M., Borm, G.F., & Cools, A.R. (1995). Card sorting performance in Parkinson's disease: A comparison between acquisition and shifting performance. *Journal of Clinical and Experimental Neuropsychology, 17,* 918-925.

Waterfall, M.L., & Crowe, S.F. (1995). Meta-analytic comparison of the components of visual cognition in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology, 17,* 759-772.

Weinstein, A.M., Troscianko, T., & Calvert, J. (1995). *Impaired visual search mechanisms in Parkinson's disease: A psychophysical and event-related potentials study.* Paper presented at the 18th European Conference on Visual Perception, Tübingen, Germany, 21-25, August 1995. [Abstract in *Perception, 24, Supplement,* 113.]

White, O.B., Saint-Cyr, J.A., Tomlinson, R.D., & Sharpe, J.A. (1988). Ocular motor deficits in Parkinson's disease. III. Coordination of eye and head movements. *Brain, 111,* 115-129.

Willingham, D.B., Korozhets, W.J., Treadwell, J.R., & Bennett, J.P. (1995). Comparison of Huntington's and Parkinson's disease patients' use of advance information. *Neuropsychology, 9,* 39-46.

Wilson, R.S., Kaszniak, A.W., Klawans, H.L., & Garron, D.C. (1980). High speed memory scanning in Parkinsonism. *Cortex, 16,* 67-72.