Hemianopic visual field defects elicit hemianopic scanning
Tant, M.L.M.; Cornelissen, F. W.; Kooijman, A.C.; Brouwer, W.H.

Published in:
Vision research : an international journal

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version
Publisher's PDF, also known as Version of record

Publication date:
2002

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):
Hemianopic visual field defects elicit hemianopic scanning

M.L.M. Tant a,e,*, F.W. Cornelissen b,e, A.C. Kooijman b,c,d,e, W.H. Brouwer a,e

a Department of Psychology, University of Groningen, Section Neuropsychology, University Hospital Groningen, Poortweg 4, 2de verdieping, P.O. Box 30.001, 9700 RB Groningen, Netherlands
b Laboratory of Experimental Ophthalmology (LEO), University of Groningen, P.O. Box 30.001, 9700 RB Groningen, Netherlands
c Department of Ophthalmology, University Hospital Groningen, P.O. Box 30.001, 9700 RB Groningen, Netherlands
d Visio, National Foundation for the Visually Impaired and Blind, P.O. Box 144, 9752 AC Haren, Netherlands
e School for Behavioral and Cognitive Neurosciences (BCN), University of Groningen, P.O. Box 145, 9700 AC Groningen, Netherlands

Received 31 July 2001; received in revised form 28 January 2002

Abstract

Previous explanations for the variability in success of compensating for homonymous hemianopia (HH) has been in terms of extent of the brain injury. In using on-line eye movement registrations, we simulated HH in 16 healthy subjects and compared their scanning performance on a dot counting task to their own “normal” condition and to real HH patients’ performance.

We evidenced clear parallels between simulated and real HH, suggesting that hemianopic scanning behaviour is primarily visually elicited, namely by the visual field defect, and not by the additional brain damage. We further observed age-related processes in compensating for the HH. © 2002 Elsevier Science Ltd. All rights reserved.

Keywords: Hemianopia; Brain; Visual; Simulation; Age dependence

1. Introduction

Homonymous hemianopia (HH) is a visual field defect (VFD) in which, for both eyes to the same extent, half of the visual field is blind. The HH can either be complete or incomplete, congruent or incongruent, and with or without macular sparing. This VFD results from unilateral post-chiasmal brain damage. Whether or not the HH is complete depends on the relative integrity of part of the visual stream or pathway. Macular sparing and congruency of the VFD are more frequently observed with posterior than with anterior lesions. Nearly 80% of patients with unilateral post-chiasmal brain damage acquire a homonymous VFD (Zihl, 1994). Common causes are cerebrovascular accident, traumatic brain injury and tumours (e.g. Kerkhoff, 1999; Zihl, 2000).

Visual field defects often lead to visually related complaints and dysfunctions. Patients complain for example about having a limited overview, bumping into obstacles or persons and experience their vision as being too “slow”. These disabilities are related to the degree of compensation for the visual field loss. For comprehensive reviews, we refer to Kerkhoff (1999) and Zihl (2000). Oculomotor compensation, this is adaptive visual scanning behaviour, can be assessed by recording eye movements (e.g. Zangemeister, Meienberg, Stark, & Hoyt, 1982; Zangemeister & Oechsner, 1996; Zihl, 1995, 1999, 2000).

A paradigm to objectively and quantitatively assess oculomotor compensational behaviour was introduced by Zihl (1995, 1999, 2000) and consisted of inspection of a dot pattern. The stimulus display consists of 20 randomly arranged dots projected onto a screen. Subjects are asked to fixate the centre of the screen, after which the dot pattern is presented and eye movements are recorded. Subjects subsequently scan the pattern and silently count the number of dots. Upon completion they report the number of dots. A relatively simple stimulus display was chosen to restrict visual scanning to the process of visual sampling without any further identification component.
(Zihl, 1999), or the primary involvement of other complex higher-order visual functions. Using this paradigm, it was found that in HH typical defective oculomotor scanning behaviour is characterised by longer scanning times and scan paths, higher number of fixations and re-fixations, and, at least in part, longer fixation durations and shorter saccadic amplitudes (e.g. Zihl, 1995, 1999, 2000). These findings are in concordance with other reports (e.g. Chedru, Leblanc, & Lhermitte, 1974; Ishiai, Furukawa, & Tsukagoshi, 1987; Kerkhoff, 1999; Meienberg, Zangemeister, Rosenberg, Hoyt, & Stark, 1981; Neetens, 1994; Zangemeister et al., 1982; Zangemeister & Oechsner, 1996).

In large, it was found that about 40% of the HH patients spontaneously compensate effectively for their VFD (Zihl, 1995, 1999, 2000) and that the subjective visual complaints by HH patients were substantiated by eye movement recordings during the dot counting task, confirming its practical relevance. Interestingly, it was concluded (Zihl, 1999, 2000) that the presence, time since, and severity of the VFDs could not sufficiently explain the observed scanning deficit and that additional factors are crucial for explaining the impaired oculomotor scanning. Zihl suggested that the extent of the brain injury is a crucial factor and that occipito-parietal and posterior thalamic brain injury may be responsible for inefficient compensation.

Nevertheless, there are some peculiar aspects in the results, which cast doubt on the provided explanation for the individual differences in the efficiency of compensation for HH. Firstly, it was noted by Zihl (1999) that the scanning (e.g. in terms of scanning time) was found to be impaired in this very simple visual sampling task. Hence, even in a task, in which complex higher-order (that is brain related) functions are not involved, a disability appears. This calls into question the crucial importance of the integrity of the brain for the visual disability. Zihl therefore cautions against the (mis)interpretation of the results in term of “unspecific” cognitive slowing, suggesting in our view, an interpretation in terms of mere visual slowing. He suggested that the “slowness of vision” may, at least in part, be explained by the use of hypometric saccades which are provoked by a homonymous VFD.

Secondly, it was found that the side of the VFD (and therefore the side of the brain lesion) was not a crucial factor. Zihl (1999) comments this observation to be surprising, because of the assumed specialisation of the right (posterior) hemisphere for visuo-spatial function, including the spatial guidance of eye movements. If predominantly higher-order visual (that is brain related) functions were involved, one would have expected left-sided HH patients (with right-sided brain damage) to perform worse, due to hemispheric specialisation and the inherently visuo-spatial nature of the dot counting task.

Both observations suggest that the deficit in visual exploration is perhaps not predominantly related to additional brain damage, but is merely a knock-on effect of the lower-order dysfunction, in this case the hemianopic visual field loss. In order to preclude the effects of brain damage, we simulated HH in healthy subjects and compared the visual exploration to their own ‘normal’ condition. By simulating the hemianopic visual field loss, we ‘create’ subjects with the lower-order visual dysfunction, but without higher-order dysfunctions caused by brain damage. The observed disabilities (if any) during simulation result from the visual limitation only and do not require a further explanation in terms of brain damage. If visual exploration deficits in real HH are predominantly provoked by the VFD, the visual exploration behaviour, displayed in simulated and real HH, should be comparable, including the variability in performance between individuals. Our primary research question hence concerns the influence of the pure visual component on hemianopic visual exploration during a dot counting task. We also included real HH patients in this study to compare the patterns of performance with simulated and real HH.

A secondary question concerns the explanation of individual differences in the efficiency of compensating for HH. Apart from differences in extent and site of brain injury in patients, in healthy subjects there are large individual differences in higher-order visual and cognitive abilities, for example differences in visual speed and other components of intelligence. Some of these abilities are suggested to be highly dependent on age, for example perceptual speed and spatial orientation (Schaie & Willis, 1993) and fluid intelligence (Rybash, Roodin, & Hoyer, 1995). As was also suggested by Szlyk and colleagues (Szlyk, Brigell, & Sciple, 1993), it is quite conceivable that such age-related abilities play an important role in the efficient compensation of HH. To investigate the effect of ageing on the efficiency of compensation, we included both younger and older adults in the study. It is predicted that the older subjects will have significantly more problems in coping with HH.

In summary of the research questions, we expect to find typical HH scanning performance in healthy subjects with a simulated complete congruent hemianopic VFD, since we hypothesise that HH scanning is primarily generated by the VFD and not by brain damage. To fully compare and characterise HH scanning performance, we will perform, in addition to general analysis, also directional, hemispace, and trend analysis (see further). Secondly, we expect to find the disabilities to be more pronounced in an older age group, since we assume that individual differences in perceptual and intellectual abilities, which tend to decrease with age, are important factors governing the compensation process.
2. Methods

2.1. Subjects

Sixteen healthy subjects participated in this study (seven males, nine females). Their mean age was 40 years (range 16–71). Two age groups were included: a younger group with a mean age of 21 years (range 16–23) and an older age group with a mean age of 60 years (range 46–71), each consisting of eight subjects. They showed no signs of cognitive decline (CST; De Graaf & Deelman, 1991), reported to be right-handed, and had normal or corrected-to-normal visual acuity. They declared to have no visually related complaints.

Twenty-nine patients were included (23 males, 6 females). They showed no evidence of cognitive decline (CST; De Graaf & Deelman, 1991 and MMSE; Folstein, Folstein, & McHugh, 1975), aphasia (SAN; Deelman, Liebrand, Koning-Haanstra, & van der Burg, 1987) or apraxia (De Renzi, Faglioni, & Sorgato, 1982). Neither of them showed severe unilateral visual hemi-neglect (UN) or visual agnosia. The selection procedure for UN is described elsewhere (Tant, Kuks, Kooijman, Cornelissen, & Brouwer, 2002). All patients had a binocular optimally corrected acuity of 0.8 or better and contrast sensitivity within normal ranges. Automated perimetry was performed using the Humphrey Field Analyzer (Full Field 246 screening program, age corrected, three-zone strategy). Forty-four patients had left-sided HH (10 incomplete, 4 complete). Their mean age was 54 years (range 29–76), the mean time since lesion was 32 months (range 6–157). Ten of these patients had macular sparing (mean 4°, range 2–10°). All were victims of stroke, except two patients, who were surgically operated for tumour. One patient, with left-sided HH, only had (right) monocular vision. Fifteen patients had right-sided HH (nine incomplete, six complete). Their mean age was 50 years (range 17–68), the mean time since lesion was 80 months (range 3–390). Eleven of these patients had macular sparing (mean 4°, range 3–8°). One patient was surgically operated for hydrocephalus, and one for tumour. Two patients suffered closed head injury. The remaining patients were victims of stroke.

Keeping the limitations of sensitivity and fixation control of the perimetric procedure in mind, we observed that the incompletenesses of the HHs consisted of a small wedge-shaped sparing near the midline in the upper or lower quadrant. None of patients showed VFDs which were strikingly incongruent.

2.2. Dot counting task and apparatus

Our dot counting task is based upon the work of Zihl (1995). We presented in total 29 patterns of dots. The screen dimensions were 36° and 27° horizontally and vertically respectively. The dot size was 1°. Dots were white (luminance 25 cd/m²) on a grey background (50% contrast). The viewing distance was 52 cm. Dot patterns were created by giving individual dots a random horizontal and vertical offset relative to a rectangular imaginary 4 × 5 grid. The random offsets were 2.0° relative to the grid position. As the grid positions were 6.0° apart, dots never overlapped. Dots were assigned randomly to any of the 20 possible grid positions. We presented five different patterns consisting of 19, 20 and 21 dots each (15 trials). For the trials with 21 dots, one extra dot was added to the (generated 20 dot) pattern, so that it did not overlap any other dot. These patterns were identical for all subjects. Additionally we presented patterns consisting of 5–17 dots (two-dot increment), which each were presented twice (14 trials). Their spatial distribution was randomly generated on each presentation. The 29 trials were presented in a random order.

During the experiment the eye movements were recorded using an EyeLink Gaze tracker (SensoMotoric Instruments GmbH, Teltow, Germany) which registers real-time gaze at 250 Hz. When simulating HH, a window, with the same properties as the background, continuously and completely blanked one side of the screen with reference to the current gaze position. This could either be left or right of fixation in order to simulate left- or right-sided HH respectively. The length of the entire system’s delay (from eye movement to screen update) was 20 ms. Prior to the experiment, the equipment was calibrated using a nine-point grid. The initial central fixation dot, prior to each trial, was also used for drift correction which may result from slips of the Eyelink’s headset. Small head-movements were allowed (and corrected for) during the experiment. This equipment allows for relatively normal free viewing conditions.

2.3. Procedure

The healthy subjects performed the task on two different occasions. On each occasion, they firstly performed the task in a non-simulation (that is normal) and subsequently in a simulation condition. During the task in a simulation condition, the side of the simulated homonymous hemianopia (sHH) was fixed. On the second occasion, the side of the sHH was changed for each subject. During simulation, half of the subjects had a macular sparing of 2.7° on both occasions. The patients performed the task once. Obviously, no simulation was imposed.
2.4. Statistical analysis

The oculomotor parameters are number and duration of the fixations, number and amplitude of the saccades, and the length of the scanpath, which is sum of the saccadic amplitudes. When both number of fixations and number of saccades are used in the same analysis, only the number of fixations are reported. As a saccade typically follows a fixation, both parameters are logically linked and hence the number of one or the other provides no additional information. We further report the absolute error in counting the dots (henceforth referred to as error) and the search time. Additionally, we perform directional and hemispace analysis. Since the healthy subjects suffered no actual brain damage, directions and side of the hemispace are defined with respect to the side of the VFD. “Ipsilateral” and “contralateral” hence refer to “in or towards” the affected and intact visual hemifield respectively. Directional analysis is performed on the amplitudes and number of saccades. Hemispace analysis is performed on the fixation parameters. The hemispace is defined in terms of the centre of the screen, which is also the start of exploration in each trial. To further characterise the scanning performance, we perform a trend analysis using the errors, search time, number of fixations, and length of the scanpath to investigate how the difficulty of the task (operationally defined by the number of dots in each pattern) influences performance in each subject group.

To analyse the data by the healthy subjects, we performed a MANOVA on a doubly repeated measures design. When significant multivariate effects were observed, the univariate effects were inspected. When necessary, we additionally performed simple-main-effects analysis, to untangle the interaction components. For this last type of analysis, only P-values will be reported. The design is graphically depicted in Fig. 1. We included the factors of age (young/old), macular sparing (yes/no), and sequence (A/B) as between-subjects factors. The sequence factor represents whether subjects were first imposed with a left- (A) or right-sided sHH (B). Within-subject factors were measurement (first or second occasion) and mode (normal/simulated HH). The measurement-, mode- and age- factors are interpretable as main-effects factors. Since the sparing factor has no relevance in the normal mode and can only exert effects during simulation, a main effect of sparing will arise as a mode × sparing interaction. Effects of the side of the sHH are inferred from simple-main-effect analysis on the measurement × mode × sequence interactions. Namely, a significant measurement × sequence interaction, within the simulation mode, reveals differential performances by left- and right-sided sHH subjects. When observed, further analysis and inspection of the means will then reveal the nature of the effects of the side of sHH and the consistency of the difference between left- and right-sided sHH in both sequences. Absence of this two-way interaction indicates no difference in left-versus right-sided sHH. Although statistically interactional, the effects of both sparing and side of the sHH will be reported as main effects.

The patient data conform to a repeated measures design with side of the HH as a between-subjects variable. We analysed the same parameters as for the healthy subjects.

3. Results

3.1. General analysis: multivariate

MANOVA failed to reveal significant multivariate main effects of measurement and age, suggesting that nor repetition of the experiment or age did have any overall influence on the data. There was a significant mode-effect ($F(7, 2) = 147$, $P < 0.007$), suggesting an overall effect of the simulation. We observed no effect of sparing, indicating that macular sparing did not lead to better performance in sHH. We found no measurement × mode interaction, confirming the absence of learning effects in both modes. Age, however, did interact significantly with mode ($F(7, 2) = 62$, $P < 0.016$). The age effect will be explored further. We observed a significant measurement × mode × sequence interaction ($F(7, 2) = 27$, $P < 0.036$), suggesting a possible influence of the side of the sHH. Simple-main-effect analyses will be performed to reveal the nature of these effects and interactions.

3.2. General analysis: univariate

In the simulation mode (compared to the same subjects in the normal mode), subjects took more time and made more errors in counting the dots. They fixated more and the mean fixation duration was longer. Also the scanpath was prolonged (Fig. 2). All parameters showed significant differences, except the saccadic amplitudes ($F$-range: 11–184, $P$-range: 0.011–0.0001).
In comparison to right-sided sHH, subjects with left-sided sHH made more errors \( F(1, 8) = 13, P < 0.007 \) and presented a longer search time \( F(1, 8) = 11, P < 0.01 \) (Table 1). Simple-main-effects analysis revealed that none of the parameters produced significant differences in normal modes. Significantly worse performance by left-sided HH was also observed in the patient group, but only for the errors \( F(1, 27) = 9, P < 0.005 \) (Table 1).

The effect of age was apparent in the search time \( F(1, 8) = 19, P < 0.002 \) and number of fixations \( F(1, 8) = 19, P < 0.006 \). The increase for both parameters in the simulation mode was greater for the older age group (Fig. 3). Exploratory, we plotted the search time per dot (this is relative search time) in function of the trial order, and observed that, although

![Fig. 2. Simulated HH (black) provokes longer search times, more errors, more fixations, longer fixation durations, and longer scanpaths than in normal conditions for the same subjects (white). Patient data (grey bars) for comparison. Error bars are 1 S.E.](image)

![Fig. 3. Subjects from the older age group had longer search times and made more fixations (and saccades) in the simulated conditions. N: Normal condition, sHH: simulated condition. Error bars are 1 S.E.](image)

<table>
<thead>
<tr>
<th>Side of the VFD</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Errors sHH</td>
<td>0.95 (0.11)</td>
<td>0.34 (0.11)</td>
</tr>
<tr>
<td>sHH</td>
<td>0.68 (0.10)</td>
<td>0.25 (0.10)</td>
</tr>
<tr>
<td>Search time (s)</td>
<td>13.2 (0.83)</td>
<td>10.2 (0.83)</td>
</tr>
<tr>
<td>HH</td>
<td>10.1 (0.67)</td>
<td>8.8 (0.65)</td>
</tr>
</tbody>
</table>

Left-sided sHH subjects take more time and make more errors than right-sided sHH. Left-sided HH patients make more errors than right-sided HH. The difference between left- and right-sided HH was not statistically significant for the search time. Standard errors between brackets.
Fig. 4. The age effects in sHH are especially evident in the beginning of the experiment. N: Normal condition, sHH: simulated condition, Y: Younger age group, O: Older age group, HH: patient group.

always present, the age effects are specially evident in the beginning of the task (Fig. 4). We also observed slight to moderate age effects in the patient population, as evidenced by the Pearson’s correlation of age with search time ($r(29) = 0.38$, $P < 0.05$) and number of fixations ($r(29) = 0.42$, $P < 0.05$).

3.3. Directional analysis

Multivariate directional analysis on the healthy subjects data, including the number and amplitude of the saccades, was significant ($F(2, 13) = 4, P < 0.05$) for the mode × direction interaction. The saccadic amplitudes in either direction did not differ in the normal mode, but there was a significant directional effect in the simulation mode ($F(1, 14) = 5, P < 0.05$). Namely, ipsilateral saccadic amplitudes were smaller than contralateral amplitudes (Table 2). There was no effect of the side of the sHH, indicating that the amplitudes of saccades into the blind hemifield are smaller than into the seeing hemifield, for both left- and right-sided sHH. This pattern of results was paralleled in the patient group. Multivariate analysis failed to reveal any effect of the side of the HH, but presented a significant directionality effect ($F(2, 26) = 11, P < 0.000$). Inspection of the univariate analysis showed the saccadic amplitudes to be smaller in ipsilateral than in contralateral direction ($F(1, 27) = 19, P < 0.000$) (Table 2).

3.4. Hemispace analysis

A multivariate hemispace analysis was performed on the number and durations of the fixations by the healthy subjects. A multivariate mode × field interaction was found ($F(2, 13) = 11, P < 0.002$).

Univariate analysis showed a significant effect of fixation duration ($F(1, 14) = 9, P < 0.010$). This effect was however not apparent in the simulation condition. In the normal mode, the durations proved to be longer when they occurred on the right side of the screen than on the left side (352 and 411 ms for left and right hemispace respectively, $P < 0.004$). Tentatively, in the simulation mode, inspection of the means would suggest ipsilateral fixation durations (577 ms) to be longer than contralateral ones (533 ms), but this difference proved not to be significant.

There was also a significant effect of hemispace on the number of fixations ($F(1, 14) = 8, P < 0.012$). In the normal mode, there were as many fixations in either left or right hemispace, but clearly more fixations in the ipsilateral hemispace in the simulation mode ($P < 0.009$) (Table 2). There was no interaction with the side of the HH, indicating that, in both left- and right-sided sHH, subjects fixated more on the same side of the screen as their VFD.

This pattern of results was paralleled in the patient group. We observed a multivariate effect of hemispace ($F(2, 26) = 55, P < 0.000$) and no effect of side of the HH. Both left- and right-sided HH patients fixated more in the ipsilateral hemispace ($F(1, 27) = 93, P < 0.000$) (Table 2).

3.5. Trend analysis

To assess the relative difficulty level of the patterns, induced by the number of constituent dots, we expressed the errors, search time, number of fixations, and length of the scanpath as relative measures in dividing them by the number of dots in the patterns. These parameters hence indicate the performance per dot. We then performed a trend analysis by way of polynomial contrasts, separately for the normal and simulation mode. If the dot counting task functionally is performed in the same manner in both groups (N and sHH), the same trends should appear. If different or additional trends appear, the number of dots assert a different influence on the

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Directional and hemispace analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>Saccadic amplitude (degree)</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>8.4 (0.59)</td>
</tr>
<tr>
<td>sHH</td>
<td>9.08 (0.96)</td>
</tr>
<tr>
<td>HH</td>
<td>7.00 (0.18)</td>
</tr>
<tr>
<td>Number of fixations</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>7.3 (0.39)</td>
</tr>
<tr>
<td>sHH</td>
<td>12.5 (1.2)</td>
</tr>
<tr>
<td>HH</td>
<td>15.7 (0.94)</td>
</tr>
</tbody>
</table>

Saccadic amplitudes are smaller in ipsilateral than in contralateral direction in simulated (sHH) and real hemianopia (HH). In the normal condition (N), there were no differences in amplitudes between saccades to the left or to the right (in table termed ipsilateral and contralateral respectively). In both HH groups, there are more fixations in ipsilateral than in contralateral hemispace. We observed no such difference in the normal condition. Standard errors between brackets.
performance, suggesting functionally different subcomponents or processes. The results are summarised in Table 3. For all but one parameter, at least one additional trend was present in the simulation mode, compared to the normal mode. For the length of the scanpath, only linear trends were present in both modes, but in simulation mode being far more distinct (as evidenced by the F-value being almost five times higher).

This overall pattern was observed to be similar for the patient data, except that occasionally also additional higher-order trends were present (Table 3). For illustrative purposes, we plot the search time per dot in function of the number of dots (Fig. 5). It can be observed that in sHH, there is relatively more time consumption for the patterns with less dots, as evidenced by the linear trends. We similarly observed relatively increasing number of fixations and the length of the scanpaths with decreasing number of dots. The reverse pattern was found for the errors (not in the figures).

4. Discussion

We did observe hemianopic scanning behaviour in healthy subjects without brain damage with an imposed HH. This suggests that hemianopic scanning behaviour is largely visually elicited, namely by the VFD. The parallels between simulated and real HH are evidenced by several findings. Firstly, we found elevated search times, errors, number and duration of fixations and length of scanpath (Fig. 1) in sHH compared to the normal condition. We did not observe a main effect of saccadic amplitude. These findings are in perfect concordance with previous findings reported by Zihl (1995, 1999, 2000) for real HH patients and confirmed by our own patient data (Fig. 1). In sHH, we also found in general longer fixation durations, which was not observed in our patient data (Fig. 1). Zihl (1999) reported the mean fixation duration to be longer in some (“impaired”) and shorter in other (“unimpaired”) HH patients. We did not create these subgroups, and hence, in Zihl’s view, are likely to have a “pooled” patient population in this respect. This could account for the total null-effect of fixation duration in our patient group (compared to the normal condition, Fig. 2). The finding that we observe fixation duration increase, fortified by the elevation of the other parameters, suggests that, in many respects, our sHH subjects resemble the “impaired” HH patients. The observation that, for most parameters, the performance in sHH is more deviant (from the normal condition) than in HH, is agreement with this suggestion. Alternatively, the HH patients did have (more) time to adapt to their VFD, while for the

Table 3

<table>
<thead>
<tr>
<th>Trends</th>
<th>Linear</th>
<th>Quadratic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Error</td>
<td>$F(1,15) = 13, P &lt; 0.002$</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>$F(1,15) = 19, P &lt; 0.001$</td>
<td>$F(1,15) = 5, P &lt; 0.046$</td>
</tr>
<tr>
<td></td>
<td>$F(1,27) = 38, P &lt; 0.000$</td>
<td>¥</td>
</tr>
<tr>
<td>Search time</td>
<td>N</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>$F(1,15) = 15, P &lt; 0.001$</td>
<td>$F(1,15) = 15, P &lt; 0.001$</td>
</tr>
<tr>
<td></td>
<td>$F(1,27) = 5, P &lt; 0.026$</td>
<td>ns</td>
</tr>
<tr>
<td>Number of fixations</td>
<td>N</td>
<td>F$(1,15) = 24, P &lt; 0.000$</td>
</tr>
<tr>
<td></td>
<td>$F(1,15) = 42, P &lt; 0.000$</td>
<td>$F(1,15) = 8, P &lt; 0.012$</td>
</tr>
<tr>
<td></td>
<td>ns</td>
<td>$F(1,27) = 7, P &lt; 0.015$</td>
</tr>
<tr>
<td>Length of scanpath</td>
<td>N</td>
<td>$F(1,15) = 11, P &lt; 0.004$</td>
</tr>
<tr>
<td></td>
<td>$F(1,15) = 50, P &lt; 0.001$</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>$F(1,27) = 56, P &lt; 0.000$</td>
<td>$F(1,27) = 29, P &lt; 0.000$</td>
</tr>
</tbody>
</table>
# indicates presence of higher-order trends. ns: not statistically significant.

Fig. 5. Trends for relative search time in function of number of dots. In the normal condition (N) no trend is present. In the simulated condition (sHH), a linear trend was found. For the patients (HH), both a linear and quadratic trend was present. Different trends suggest different functional components.
sHH subjects, the acquisition of the VFD was very recent.

The second parallel between real and simulated HH concerns the side of the VFD, which was previously reported not to affect any oculomotor parameter in real HH patients (Zihl, 1999). This was confirmed by our patient data. We did however find left-sided HH patients to count less accurately than right-sided HH patients (Table 1). Zihl did not observe this difference. In his paradigm, only one pattern (one trial) was presented and subjects made no errors. Since we presented 29 trials, we were more likely to observe errors, and hence our data are not optimally comparable in this respect. The absence of effects of the side of the VFD in oculomotor parameters and the worse error performance in left-sided HH was paralleled in sHH (Table 1). We did additionally find a longer search time in left-sided sHH. Hence, the results in sHH parallel the results in HH, in that the side of the VFD does not differentially influence oculomotor performance. Left-sided sHH subjects, however, tend to make more errors and need more time than subjects with right-sided sHH. This was also the case with respect to the errors for the HH patient group.

Thirdly, directional and hemispace analyses further confirm the same pattern of results in sHH and HH (Table 2) and are in concordance with previously reported findings. Differential hemifield distribution of the fixations has previously been reported (e.g. Chedru et al., 1974; Ishiai et al., 1987; Kerkhoff, 1999; Meienberg et al., 1981; Zangemeister & Oechsner, 1996; Zihl, 1995, 1999, 2000). Also in our data, both sHH and HH fixated more in the ipsilateral hemispace. Saccadic dysmetria and more specifically ipsilateral hypometric saccades are considered typical for hemianopic scanning (e.g. Chedru et al., 1974; Ishiai et al., 1987; Meienberg et al., 1981; Neetens, 1994; Zangemeister et al., 1982; Zangemeister & Oechsner, 1996; Zihl, 2000). Our data, both for sHH and HH, confirm saccadic amplitudes in ipsilateral direction to be smaller than in contralateral direction. As in previous studies (e.g. Zihl, 1995) no effects of the side of the VFD were found.

We were able to replicate all aspects known to be typical for hemianopic scanning behaviour in simulated HH. These healthy subjects did not suffer brain damage, but were imposed with a simulated homonymous hemianopic VFD. It follows that the typical HH scanning behaviour is largely due to the VFD (visually elicited) and not to concomitant brain damage. To further explore underlying components in the scanning behaviour, we performed the trend analysis in function of the number of dots (Table 3). We assume that functionally different components will result in different trends. In the normal condition, we observed linear relationships between the number of dots and the errors, number of fixations and length of the scanpath per dot. The search time per dot did not seem to be influenced by the number of dots. These same trends appeared in sHH, suggesting the same underlying mechanisms. However, in nearly all parameters, also other (higher-order) trends were observed, suggesting additional components. It is reasonable to assume that these additional trends are brought about by the simulated VFD, since this was the only difference with the normal condition. These additional trends are suggested to be visually elicited. However, the additional trend in HH, compared to sHH (e.g. quadratic in Fig. 5), suggest that still additional components are into play in real HH scanning. Although different subjects comprise the sHH and HH groups, and hence are not ideally comparable, this suggests that also brain damage functionally influences the scanning behaviour. More dots most likely summon more visuo-spatial, memory and organisational functions. Brain damage is likely to affect (some of) these functions, which are likely to interact reciprocally with adequate visual exploration and proper cerebral representation of space, hence resulting in the appearance of additional trends. Alternatively, the appearance of the additional trends could be statistically induced by generally better performance by the HH subjects (compared to sHH). As a result, HH subjects sooner perform at their maximal effectivity, inducing flattening of the
(relative) performance curve, which will appear as additional trends. With this statistical alternative in mind, we would like to indicate that the additional trends can at least be suggestive for the additional impact of brain damage on the HH scanning behaviour, but also that psychometrically fully comparable data is needed to support our suggestion.

In summary, we can conclude that HH scanning behaviour is largely visually elicited, namely by the VFD. We further suggest that subtle interplay of brain-related functions and the VFD complete real HH scanning.

Our interest in the effects of age were aroused by Szlyk et al. (1993) who suggested that age-related losses, when compounded by CVA-associated impairments, significantly influenced visuo-spatial driving related skills. Such an age-related loss could be fluid intelligence, defined as the ability to new-problem solving. Our healthy subjects were exposed to a new experience (sHH) for which adaptive behaviour was required. We found this compensation indeed to be worse for the search time in the older age group. This age effect remained when the log-transformed values were used, as suggested by Cornelissen and Kooijman (2000).

Clear differences were also observed for the number of fixations, in that the increase in the sHH condition was far greater in the older age group (Fig. 3). These findings were paralleled in the patient group. Hence, becoming (simulated) hemianopic seems more disabling for older subjects. It would follow that on second simulation, these effects would weaken, since it then is no longer a new situation. The absence of a learning effect seems to contradict this, but since on both occasions the side of the sHH was changed, it cannot be considered a valid test for our hypothesis. We therefore explored the compensation effects within the simulation conditions by trial order. The rationale is that the sHH is very new at the first trial, but less with increasing trials. If the older age group is less capable of new-problem solving, it should be most prominent during the first trials. This is exactly what we observed (Fig. 4). This pattern, although still very prominent, was slightly reduced on second occasion for the older age group (not in figure). For the younger age group, patterns on both occasions were identical (not in figure). We therefore conclude with Szlyk and colleagues that age-related processes are related to hemianopic compensation, but we add this to be the case even if the disabilities are merely visually elicited and hence are not specific for brain damaged subjects.

In conclusion, HH scanning behaviour, as assessed by eye movement recordings during a dot counting task, can largely be accounted for by the VFD. It follows that most typical HH oculomotor dysfunctions, as for example ipsilateral hemypetropic saccades, do not result from the brain damage but are visually elicited. Age-related processes, in this case worse compensation to these visually elicited disabilities, were apparent. The implication of this study is that at least some typical HH disabilities and complaints, as for example slowness of vision and prolongation of scanpaths, can no longer be merely associated to brain damage, as they also do appear in subjects with sHH. A further implication would be that these visually elicited impairments can be most pronounced during (seemingly) the simpler situations. This can have also ramifications both for rehabilitation and diagnosis. Firstly, these results suggest that, at least for some HH patients, more emphasis can be devoted to visual than to cognitive components in rehabilitation. Secondly, diagnosing higher-order visuo-spatial impairment can only occur in the light of concomitant lower-order visual impairment.

Acknowledgements

We would like to thank E.M. Havik for collecting the healthy subjects data. F.W. Cornelissen was supported by Visio.

References


