AUTOMOBILE DRIVING PERFORMANCE OF BRAIN-INJURED PATIENTS WITH VISUAL FIELD DEFECTS[1]

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ABSTRACT Schulte T, Strasburger H, Müller-Oehring EM, Kasten E, Sabel BA: Automobile driving performance of brain-injured patients with visual field defects. *Am J Phys Med Rehabil* 1999;78:136–142

The purpose of this study was to examine whether patients with visual field defects resulting from cerebral injury are handicapped in their driving ability, because visual field loss as assessed in standard perimetry is often the basis for withdrawal of a person's driving license. Driving performance was tested on a driving simulator to obtain standardized results and for safety reasons. The visual field was assessed both with standard automated perimetry and computer-based, high-resolution, qualitative perimetry. We investigated nine patients with purely cerebral field defects (mostly homonymous binocular defects) who had no further neuropsychological or ophthalmological deficits. Their performance (driving speed, reaction time, and driving error rate) was compared with that of a control group of ten subjects. We found no differences in any of the tested parameters between the visually impaired subjects and the normal participants. This suggests that individuals with visual field defects, including those who suffer from homonymous hemianopia, may perform as adequately as normal individuals in realistic driving scenarios. The perimetrically assessed visual field may, thus, be of limited value for the prediction of driving safety, and we conclude that patients who have field defects should not summarily be denied a driving license.

KEY WORDS: Visual Field, Humans, Brain Injury, Perimetry, Traffic Safety, Driving Simulator

Traffic safety regulations in many countries require a driver to have a complete binocular visual field. Acquired loss of any part of the visual field may lead to withdrawal of the driver's license by traffic authorities. For most brain-damaged patients, it is important to know whether they are capable of safe driving to carry on their typical daily activities.

Objectives: Upon completion of this article, the reader should be able to (1) assess the role of visual field defects from cerebral injury in driving performance; (2) realize that partial blindness as diagnosed from static perimetry does not, per se, imply a driving security hazard; (3) consider that subjects can possibly compensate for their visual deficits in everyday tasks. Level: Advanced

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Unfortunately, there is little evidence as to whether visual field defects increase traffic risk.[1] Kasten et al.[2] found that only 57% of visually impaired patients had been informed by their physicians that their driving skills might be reduced as a consequence of their visual deficits. Such information would be particularly important because patients often fail to detect their own visual field loss. Of the surveyed patients, 65% still held a driver's license and one-third were still driving. There are few studies dealing with the question of driving skills after brain injury with subsequent visual field loss, and the empirical basis for denying one's driver's license on the basis of perimetrically evidenced field loss is weak.[3–8] Most patients are motivated to retain their mobility and, thus, exhibit a high interest for information concerning their driving skills.

The general increase of traffic density requires intensified attention and perceptiveness by drivers.[9] Accordingly, the German Ophthalmological Society (Deutsche Ophthalmologische Gesellschaft) demands an entirely intact visual field for uncompro-

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mised traffic safety. Measured monocularly, the intact human visual field covers a range of approximately 80° temporally, 60° nasally, 70° downward, and 60° upward. Perception of traffic events that are relevant to the driver, however, seems to take place only within the central 25° to 30° of visual angle from the point of fixation.[10] The central visual field, thus, plays the critical role in a person's driving ability, and one of the questions that arises is whether a small, central homonymous defect or a larger peripheral one can be relatively more harmful to a person's driving skills.

Brain damage affecting the visual system can be classified as being of prechiasmatic or postchiasmatic anatomical location, resulting in different kinds of monocular or binocular deficits. Homonymous field defects originate from lesions located behind the optic chiasm.[11] Among the most frequent causes of visual field loss are cerebral infarctions and hemorrhages of arteries supplying the posterior and middle parts of the brain.[12] Heteronymous field deficits, which lead to monocular scotoma, blindness of one eye, or other forms of heteronymous anopia, usually indicate lesions of the retina, optic nerve, or chiasm.

Basal perceptual deficits from such lesions are often insufficiently examined.[13] Many patients, therefore, remain uninformed as to the type, duration, and specific consequences of their perceptual disabilities and, thus, cannot adequately estimate the effects of their deficits on driving performance. Only by means of a comprehensive diagnosis and special treatment in rehabilitation centers can patients learn to deal appropriately with their visual deficits and, eventually, compensate for them.[14, 15]

The visual field is, by definition, the perceptual space available to a fixating eye. Central or paracentral binocular field defects may lead to reduced reception of information. As a consequence, identification of relevant traffic objects and situations that perceptually fall into the blind areas of the visual field escape the drivers' attention, and moments of danger remain unnoticed.[16] The normal chain of events (detailed detection of objects, processing of visual perceptual input, and immediate behavioral responses) becomes interrupted or remains incomplete.[17]

The question of how driving aptitude is affected in persons having visual field defects has received considerable attention. Hills and Burg[18] and Council and Allen[19] could not demonstrate that visual field defects had an influence on automobile accident rates. In both studies, however, the visual field was only examined along the horizontal meridian. Note that scotomata, which may be quite scattered over the retina, can be discovered only with the use of high-resolution perimetry. Because only low-resolution screening procedures were used in both studies, it is possible that smaller, yet central, field defects went unnoticed. A possible influence of visual field defects on automobile accident rates might, thus, have been overlooked.

Lövsund et al.[6] measured detection of stimuli with varying sizes that were presented in a driving simulator at 24 predefined positions at different eccentricities. Two groups of subjects were tested, one with normal sight and another with various field defects. Subjects in the latter group varied widely in their individual reaction times, most showing deficient detection capacity for stimuli presented within the defective areas. The results led the authors to conclude that compensation for field defects is possible in only a few cases. The patients in the study had a variety of visual disorders, including cerebral defects with both pre- and postchiasmatic lesion sites as well as retinal impairment from glaucoma. The highest driving accuracy was obtained in patients who had an active search pattern scanning stimuli presented in the blind area at very short time intervals. It is conceivable that there are different compensatory capacities and search patterns dependent on the underlying cause of visual deficit.

By using an interactive driving simulator, Szlyk et al.[1] examined the driving skills of six patients with visual field defects after brain damage (median age, 67 yr), three of whom were additionally diagnosed as having hemi-spatial neglect. The authors compared the patients' driving performance with that of seven age-matched and 31 younger, visually unimpaired subjects. The results showed significantly more crossings of traffic lane boundaries and an increased tendency to weave within lane boundaries by the visually impaired participants compared with the age-matched controls. The side of scotoma was found to be unrelated to the behavioral measures, which was discussed as functional symmetry in the primary visual cortex. Two of the three neglect patients had impaired eye and head movements, whereas all patients with pure hemianopia had eye movements similar to normal controls and an increased rate of "compensatory" head movements. However, when the performance of the elderly groups (i.e., both visually disabled and nondisabled subjects) was compared with that of the younger group of visually intact individuals, lower performance was found in all of the elderly subjects. The authors concluded that the patients differed in their driving ability from that of the control group but were able to compensate for their visual defects. Most deficits in the elderly subjects appeared to be related to their age rather than to scotoma-related impairments.

The goal of the present study was to assess how visual field loss from severe brain injury as assessed by detailed perimetry will affect these driving skills. To accomplish this in both a safe and standardized manner, we conducted the study with a driving simulator. Great emphasis was placed on precise mapping of each individual's visual field defects. Furthermore, we wanted to have a homogeneous

sample of pure brain injury-caused visual field impairments without further neuropsychological or ophthalmological deficits. The following questions were of particular interest:

- What differences in driving skills can be expected between visually impaired and normally sighted subjects as assessed by (1) driving errors (e.g., lane crossings and accidents); (2) the average driving speed; (3) their reaction time to a suddenly appearing moving object (i.e., a deer approaching the road)?
- Is there a relationship between the extent and location of central field defects and driving performance?
- Is such a relationship evident in those visually impaired individuals who still drive?
- Is driving performance related to the location of visual damage?
- Is there a link between onset or duration of visual impairment and driving performance?

METHODS

Subjects

We selected a group of subjects from a pool of patients who had participated in a previous investigation on the restitution of visual field defects resulting from brain damage.[20,21] Patients were in good physical condition. Nine patients had lesions of either their primary visual cortex ($n = 7$) or optic nerve ($n = 2$). Those suffering from postchiasmatic lesions, caused by accidents, infarctions, or tumors, had homonymous field defects, except for one patient who displayed an incomplete, peripheral, heteronymous, and crescent-like quadrant anopia. The median onset of the disability was 6 yr (range, 6 mo–15 yr). Field defects were binocular in eight patients, and one subject with an optic nerve lesion had a blind right but intact left eye. Demographic and lesion data of the subjects are summarized in Figure 1.

The patients were younger than 75 yr and had no other neuropsychological deficits as assessed in tests of attention (d2 concentration test, Brickenkamp, 1978), perceptual speed (Zahlen-Verbindungs test, Oswald and Roth, 1987), and dyslexia (Züricher Lesetest, Linder and Grissemann, 1981). All had intact foveal vision (macular-sparing) and no deficits in reading. The median age of the group was 45 (range, 29–74) yr. For the control group, we recruited ten subjects with unimpaired vision from the Magdeburg police department and three additional subjects from the surrounding community. Median age of the control group was 51 (range, 41–56) yr. Both groups underwent identical testing conditions. None of the subjects participating in our study had previous experience with a driving simulator. Throughout the study, data acquisition was conducted by the same person.

Measures

The driving simulator (L-Hybridbild-Fahrsimulator N6H; Reiner Foerst GmbH, Marienheidel Rodt, Germany) reproduced a Ford Scorpio with a 28-inch monitor substituted for the windshield. At a viewing distance of 70 cm, visual field size was 16° vertically and 21° horizontally. The simulator measured driving speed, reaction time to a suddenly appearing deer, and traffic violations. We used the *automatic transmission* simulator mode to minimize performance-influencing factors other than those caused by the visual field loss, among them, for instance, unfamiliarity with the handling of the car or interference from divided attention when changing gears. The patients were free to concentrate on the task of driving along a roadway without having to take their eyes off the screen. To first assess driving skills under optimal conditions, we had the simulation present a standard roadway under good weather conditions. Before running the actual test, each participant was familiarized with the simulator by presenting a practice trial over a distance of 2.6 km.

The subjects were then instructed to drive a 5.2-km roadway at an average speed of 100 km/hr and to pass slower cars while obeying traffic rules. Because driving requires the ability to recognize dangerous situations and to react immediately, the test included a deer suddenly crossing the road from right to left. A patient's reaction time for stepping on the brakes was measured.

To obtain detailed information on the sizes of the field defect, their borders, and the severity of functional loss, we used not only standard static luminance threshold perimetry on an Automated Tübinger Perimeter (TAP)[10] but also a qualitative high-resolution campimetry (i.e., perimetry on a cathode-ray-tube screen) for fine resolution in the central visual field.[21] On the TAP, we measured the central 30° field with the highest resolution of 191 test points provided to obtain good resolution in that part of the field that corresponds to the driving simulator screen (21° × 16°). The mean distance between stimuli was approximately 2°. We further measured the 80° radius with a medium resolution of 104 stimuli. Test stimuli on the TAP have higher spatial density toward the center of the visual field, the scaling based on the cortical magnification factor, to accommodate for the larger brain representation of central vision.[23] Stimuli are 0.2°-diameter-circular disks on a 10 cd/m^2 background, the luminance increment thresholds of which are measured. Subjects respond by pressing a button on detecting the stimulus. Measurements on the TAP were collected under monocular viewing conditions.

To obtain higher resolution in the central field, we used software (PeriMa) developed by Kasten et al.[21,22] in which above threshold (50 cd/m^2) circular disk stimuli are presented on the dark background of the cathode-ray-tube screen. At the standard

number of detected and missed stimuli, false alarms, and the mean reaction time for stimulus detection. Normative data exist for monocular and binocular viewing, and we can directly compare the binocular campimetric results with the driving simulator performance under normal binocular conditions.

RESULTS

Visual Impairment

In the campimetric visual field test (PeriMa), the subjects' mean total number of correctly identified stimuli was 386 (range, 278–498) for all brain-damaged patients, which represents 77% (range, 56–100%) of normal vision in the central ±25° × ±20° area. Mean reaction time was 206 (range, 190–290) ms. Only two patients, 45- and 74 yr old, had a prolonged reaction time in the simple stimulus detection task (290 ms, 260 ms) within the intact visual field (Table 1).

License Holding and Driving Practice

Seven patients still possessed their driver's licenses. Five of them still drove their cars, covering an average annual distance of 5,240 (range, 1,200–12,000) km. Three of the latter reported previous involvements in car accidents; only one of them, however, attributed it to visual impairment. Two patients had their driver's licenses suspended after the onset of visual impairment, one of them reclaimed it after official examination. All but one of the patients had been informed by their physicians about possible problems with their driving capability.

Driving Simulator Data from Visually Impaired and Normally Sighted Subjects

The nine visually impaired subjects drove at an average speed of 47.2 ± 6.8 (range, 37–56.2) km/h and had an average error/accident rate of 1 (range, 0–3). Mean reaction time to the suddenly appearing deer was 1.03 ± 0.15 (range, 0.48–1.56) s. The ten normally sighted subjects drove at an average speed of 51.5 ± 9.8 (range, 41–67.1) km/h along the 5.2-km roadway, showing a mean error/accident rate of 1.5 (range, 0–3). Mean reaction time to the suddenly appearing deer was 1.07 ± 0.32 (range, 0.65–1.51) s (Fig. 2).

Because of the small number of subjects, normality of the distribution and homogeneity of variance could not be tested. We, therefore, used the nonparametric Mann-Whitney *U* test to determine whether the level of driving performance was lower in the patient group. The *U* test showed no difference between the groups for any of the three performance variables obtained on the simulator. To check whether a possible performance difference was masked by the presence of the two visually disabled patients with intact binocular central field, the test was run again with these two patients taken out of the analysis. Also with this reduced sample ($n = 7$), the visually disabled did not differ in performance from the normally sighted subjects.

Correlations Between Simulator Performance and Visual Impairment

We further asked whether the extent of visual field loss is predictive of driving performance by correlating, within the patient group, PeriMa scores with simulator performance scores. There was a sizable but nonsignificant correlation between the extent of visual field loss (PeriMa) and simulator speed ($r = 0.57$; $P = 0.11$), with higher loss seemingly coupled to lower driving speed. The fact that significance was missed might be a consequence of the relatively small number of subjects for the statistics. At the same time, however, those with larger intact fields also have more practice in driving every day; because of the confounding of the two variables—current driving practice and field extent—it cannot be decided which of the two is critical. Time since onset of visual impairment did not show a significant correlation with any of the driving performance parameters.

We did, however, find an apparent relation between central field extent and simulator reaction time in the subgroup of patients with right-side

TABLE 1

Summary data for the qualitative campimetry in the central ±20° (horizontal) × ±15° (vertical) field

	Patients	Controls
No.	9	10
Age (in yr)	48	50
PeriMa	386/500 (77.2%)	100%

The computer program PeriMa[21,22] tests detection of high luminance (50 cd/m²), 15-foot circular test spots, presented in random sequence on 25 × 20 densely spaced positions on a dark (<1 cd/m²) background.

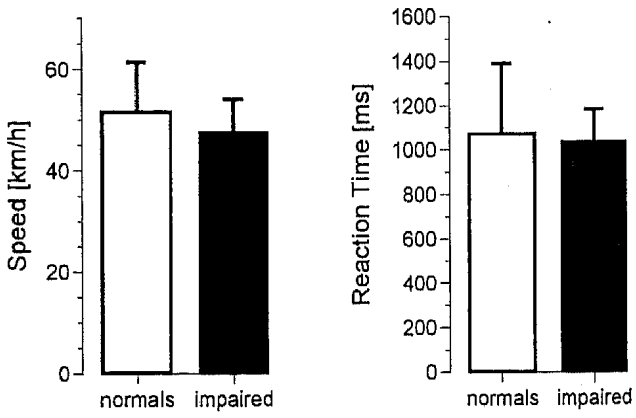

Figure 2. Simulator performance results, speed and reaction times, for the visually impaired and normal controls.

field defects (n = 4). The two persons with a complete right hemianopia showed faster reaction times (median, 0.72 ms) to the suddenly appearing deer than those two having only a scotoma or lower-right quadrant anopia (Md = 0.99 ms).

Performance Differences Among the Visually Impaired Subjects

To further differentiate within the group of visually impaired, we checked for possible effects of time since onset of visual impairment (recent v long term) and of driving practice and compared simulator performance between the groups. Again, no significant differences are evident. Concerning the effect of driving practice, we compared those who continued (n = 5) with those who had ceased driving (n = 4) and found a tendency for the latter to drive at lower speeds on the simulator (50 v 43 km/hr; z value = 1.715; P = 0.086). Furthermore, the individuals who still practiced driving (n = 5) had somewhat larger visual fields (86% v 66.2% of the central visual field intact; U test z value = 1.714; P = 0.086).

DISCUSSION

Contrary to our expectations, the findings showed no reliable difference in the performance of visually impaired and normally sighted subjects on a driving simulator. We had at least expected patients with visual field defects to drive more cautiously (i.e., at lower average speed) and to have longer reaction times in moments of danger. As a group, patients did not drive significantly slower than did age-matched controls (mostly police officers and, thus, experienced drivers), even though larger field losses seem to be coupled to more cautious driving. Most significantly, the reaction time in moments of danger and the accident rate were virtually identical between the visually impaired and the controls.

Thus on a practical level, our results indicate that suspension of driving privileges for persons having visual field impairments may be unwarranted on the basis of visual field loss alone. The results agree with previous research on the driving behavior of visually impaired subjects. Szlyk et al.[1] found performance deficits in driving to correlate more with age than with the presence or extent of visual field defects. Katz et al.[5] found brain-damaged patients who had passed a comprehensive driving assessment to be as fit to drive as their normal matched controls. Driving behavior, as measured by average speed and driving errors (lane boundary crossings), was on the whole equal between the patients and controls.

However, our patients had clearly defined central field defects from brain damage and, thus, did not receive conscious perceptual input from those blind areas. The question then is unsolved why such visual deficits did not result in more serious driving errors, that is, how are individuals with field damage able to compensate for their visual deficits?

One possibility is that saccadic eye movements allow sufficient functional exploration using healthy areas to counter potential problems resulting from blind areas, particularly when the defects are located centrally. Research on eye movement strategy in reading has shown that patients with hemianopic field defects can learn to shift their preferred retinal fixation locus to increase their useful visual field.[24] For the skill of driving, it is conceivable that patients learn specific compensatory eye movement strategies. Measuring strategies of saccadic eye movements and their influence on the drivers' available field of view while driving might shed light on the surprising driving skills of patients who have visual field defects resulting from circumscribed cerebral lesions of the primary visual system. Visual field defects, therefore, may have a smaller than expected effect on driving performance because elicited eye movements may be sufficient to overcome these deficits.

It seems unlikely, however, that eye movement strategy could also account for the surprisingly good reaction times to the deer appearing in the blind field. Regardless of the extent and location of blind areas within the right field, none of the patients failed to react quickly to the critical stimulus and there were no differences to normally sighted subjects. Patient group reaction times were not only similar to those of normal controls but were even slightly better in those who have larger field defects. An interesting possibility is that patients make use of a residual capacity of acting on moving stimuli. Rafal et al.,[25] for example, showed that distractor signals presented within the blind hemifield of hemianopic patients can inhibit saccadic movements toward stimuli in the intact part. From their findings, they concluded that orienting behavior in hemianopic humans may be mediated by brain structures other than the geniculocortical pathway, such as the retinotectal pathway of the oculomotor system. An explanation of the good reaction time is that the perception of moving stimuli may have remained intact in our patient sample. Perception of movement is localized in brain area V5. Ffytche et al.[26] reported on a patient whose brain area V1 (primary visual cortex) was damaged, whereas brain area V5 remained intact. This patient showed residual perceptual capacity for fast moving stimuli presented in his affected hemifield. Anatomically, the motion area receives direct input from the lateral geniculate nucleus via the pulvinar, thus circumventing brain area V1.[27] Presumably, there is also residual perception of moving objects in our patients, which suffices for normal driving. More generally, research on blind sight implies that conscious perception of a stimulus is *not* a necessary prerequisite for its reliable neural processing.[28,29] Furthermore, the work of Milner and Goodale[27] shows that the link between automated action, such

as responding to a sudden event, and visual input is neurally represented separately and independently from (conscious) perception.

CONCLUSION

Although there might be an effect of the extent of central field loss on driving capability within the patient group, this effect appears to be of minor importance, because the performances of the patient group and the control group were similar. Even though the number of participants in our study was low and it is mandatory to have more empirical data on the relationship between visual field defects and driving performance for a conclusive judgment, other studies support the view that there may be little direct connection between the two. The assessment of driving capability should, thus, certainly not be based on perimetric examinations alone, and patients having field defects should not summarily be denied a driving license. Furthermore, the behavior in situations of fast response might be mediated by brain structures other than those that underlie conscious detection as assessed in perimetry. Expert opinion to fairly judge fitness for driving should, thus, take into account compensatory behavior, such as saccadic eye movements, the perception of moving stimuli, or possibly, a direct demonstration of driving skill on a simulator.

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No.	age	Time since injury (in months)	Valid driver license	Driven distance per annum (km)	Visual field defects
1.	50	23	yes	0	homonymous scotoma in the lower right quadrant trauma
2.	74	89	yes	4000	small paracentral defects of the lower quadrants also left-eye defect of the optic nerve circulatory disturbance
3.	59	86	yes	1200	homonymous hemianopia right stroke
4.	41	184	no	0	homonymous hemianopia left hemiparesis left stroke
5.	59	27	yes	5000	homonymous scotoma in the upper left quadrant stroke
6.	44	175	yes	4000	heteronymous incomplete quadrantanopia tumor
7.	29	8	yes	0	incomplete homonymous inferior quadrantanopia injury of left occipital and right temporal lobe from shot wound
8.	34	32	yes	12000	lesion anterior to the optic chiasm-blindness of the right eye trauma
9.	45	77	no	0	homonymous hemianopia right trauma

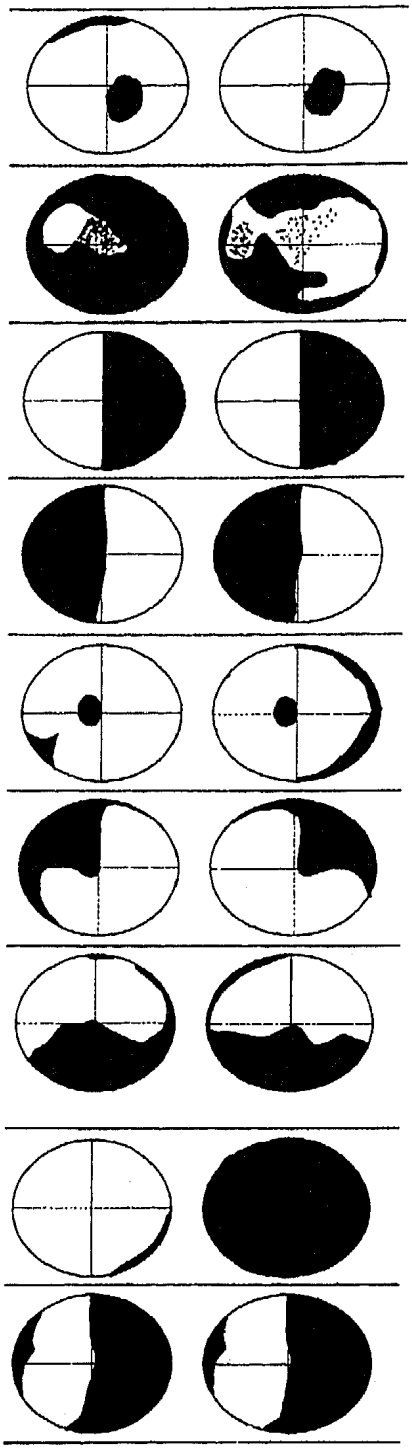

Figure 1. Demographics of patients having different types of visual field defects and data on their driving practice. Patients 2 and 8 suffered from an optic nerve lesion; the other patients have contralateral lesions posterior to the optic chiasm. Patients 6 and 8 have an intact binocular central visual field.

viewing distance of 30 cm, the 17-inch screen subtends 40° (horizontally) × 25° (vertically) of the visual angle. Stimuli are presented at 25 (horizontally) × 20 (vertically) positions, which gives a density of 1.6° horizontally and 1.25° vertically. Five hundred stimuli were presented in a random sequence within this raster, and the subjects responded by pressing a button on detection. Control of fixation was achieved by requiring the subjects to detect a change of color at the fixation point (a star of 4 mm diameter), yielding a fixation accuracy of approximately 1°. The data collected included the