# Visual training of cerebral blindness patients gradually enlarges the visual field

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# Visual training of cerebral blindness patients gradually enlarges the visual field

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## ABSTRACT

**Background:** Multiple studies on recovery of hemianopsia after cerebrovascular accident report visual-field enlargements after stimulation of the visual-field border area. These enlargements are made evident by the difference between pre- and post-training measurements of the visual field. Until now, it was not known how the visual-field enlargement develops.

**Aim:** To study how the enlargement develops as a function of time.

**Methods:** 11 subjects were trained by stimulating the border area of their visual-field defect using a Goldmann perimeter. The visual-field border location was assessed using dynamic Goldmann perimetry before, after and during training (after each 10th training session). To monitor eye fixation, a video-based eye-tracker was used during each complete perimetry session.

**Results:** It was found that visual-field enlargement during training is actually a gradual shift of the visual-field border, which was independent of the type of stimulus-set used during training. The shift could be observed while eye fixation was accurate.

**Conclusion:** Visual-detection training leads to a decrease in detection thresholds in the affected visual-field areas and to visual-field enlargement. Training effects can be generalised to important daily-life activities like reading.

Recovery from cerebral blindness has been investigated for more than three decades, during which time three types of remediation have emerged: compensation, substitution and restoration. The last type targets an enlargement of the visual-field through stimulation of the border area between the affected and the unaffected visual field. Several studies show that this type of training can lead to enlargement of the visual field.[1–17] However, these findings could not be replicated in other studies.[18–23] Also, most studies appear to have poor internal validity or lack a methodological standard.[24 25] Therefore, the merits of the training are not generally accepted. It has been suggested that increasingly larger eye movements towards the visual-field defect cause the enlargements,[19 25] but Kasten *et al* and Mueller *et al* state that the visual-field enlargements they observed are not correlated with eye movements.[6 17] Because of this ongoing discussion, we paid special interest to eye fixations during stimulus presentations. Many restoration studies have subjects perform training at home.[2–6 9 10] We wished to gain an insight into visual performance development during training, so we trained subjects on a Goldmann perimeter so that performance could be directly monitored.[1 12] Also, restoration training uses stimulus locations that are adapted to improvement in performance of the subjects.[3] In our studies, we used the same technique. However, this adaptation may be the cause of the visual-field border shift. Therefore, subjects trained with stimuli on adaptive locations are compared with subjects trained with stimuli on fixed locations. To assess the generalisation of training effects to important daily-life functions, we studied reading performance before and after training.

## METHODS

### Training

Subjects were trained monocularly for both eyes consecutively with repeated trials of stimulus detection threshold measurements. The background luminance was 31.5 asb (≈10 cd/m$^2$). The luminance of the white, circular stimulus (Goldmann IV, diameter: 1°) was set at 12.5 asb (IV-1a ≈4 cd/m$^2$) and was increased stepwise with a 0.1 log unit change up to 1000 asb (IV-4e ≈318 cd/m$^2$). Subjects responded after stimulus detection during central fixation. Nine subjects were presented with a wide-ranging set of stimuli on fixed locations, and two subjects were presented with stimuli on locations periodically adapted to a growing visual field. Fixation was monitored visually so that small changes in eye position could be detected. However, when a subject starts a session with a parafoveal fixation, it can be detected only if the deviation from the fixation point is large enough. Therefore, in addition, the blind spot is probed on several occasions during each training session to check for fixation. The method is described in more detail in Bergsma and van der Wildt.[1]

### Perimetry

The training effect was measured monocularly with dynamic Goldmann perimetry before the first training session and after every 10th session. Goldmann dynamic perimetry is prone to low interexaminer reliability. To maximise the reliability, the examiner repeated measurements at each meridian three times and closely followed the perimetry instructions laid out by Frisen.[29] Eye positions were measured using an Eyelink II eye-tracker (sampling rate 100 Hz, spatial resolution 3 min of arc). A customised chin- and headrest stabilised the subject's head and the Eyelink headset. The Eyelink system was calibrated prior to each separate monocular measurement. With normal calibration, the development of a pseudofovea will not be detected because all stimuli, including calibration stimuli, will be fixated with that developing parafovea. However, because the blind spot has a fixed location on the retina, a

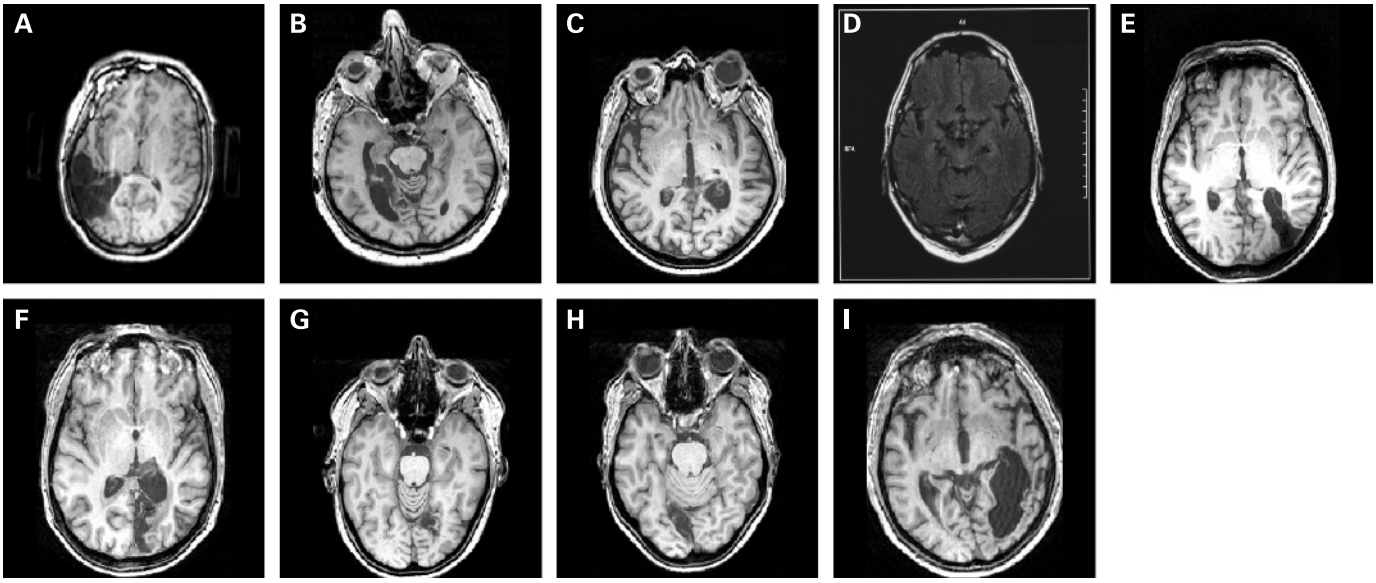


**Figure 1** See table 1 for details.

developing pseudofovea causing a fixation shift will immediately be detected as a shift of the blind spot. Therefore, the blind spot is mapped at the start of all Eyelink measurements with the Goldmann III-4e stimulus (diameter 0.25°). (The border of the blind spot is measured at eight points: up, down, left, right and in between. In the graphs, an oval is fitted to these eight points.) Also, during perimetry the blind spot is probed (Heijl–Krakau method) at random intervals for intermediate assessment of fixation. With blindspot probing, deviations from the fixation point up to 2° may remain undetected by the examiner. However, we also observed that most of our subjects could only restrict eye movements during fixation within a range of 2°, especially in the direction of a peripherally presented stimulus. Therefore, fixation is considered to be correct if it does not deviate more than 2° from the fixation point. The stimulus detection eccentricity during correct fixation is used to reconstruct the visual field. Because eye positions and the exact moments of stimulus presentations and responses were recorded during the whole perimetry session, we were afterwards able to discard measurements in which eye movements larger than 2° were made in the direction of the presented stimulus. These 2° are not a measurement error or Eyelink inaccuracy but a criterion that we have set to distinguish between correct and incorrect fixations. Of course, visual-field enlargements up to 2° are therefore not considered to represent the training effect. The 2° limit also allows for blind-spot probing, because a stimulus would still fall within the blind spot, even if the eyes deviate $\leqslant 2°$ from the fixation point. In the Results section (''Eye-position analysis''), an example of an included measurement ($\leqslant 2°$) and a discarded measurement ($>2°$) is given.

### Reading

To answer the question about whether improved stimulus detection leads to a transfer of training effects to daily-life activities, we studied reading performance before and after training in seven subjects. They silently read two standardised texts with 15 lines (152 words) and 18 lines (168 words) of Arial 14 pt text, respectively. Both texts were presented consecutively before and after training (minimal 3 months in between). Only three subjects remembered the topic of one or both texts after training: PK and GL remembered the topic ''lightning'' but nothing else about the story. IW remembered ''Lightning'' and ''New Zealand,'' but nothing else. The other four subjects could not replicate any topic. Eye movements were measured using the Eyelink II headset and a chinrest stabilising the subject's head 50 cm away from the texts. Dependent variables were reading time (words/min) and average number of saccades and regressions (reading errors).

## SUBJECTS

Eleven persons (six males, five females) were trained for 40 daily 1 h sessions. Subjects were volunteers with a visual-field defect after postchiasmal brain damage and without visual neglect. The average subject age was 59.8 (SD 9.2) years; the average lesion age was 3.0 years. One subject (MS) had a lesion age of less than 1 year. In this case, some spontaneous recovery cannot be ruled out. All others had lesion ages ranging from 1 to 8.5 years. Table 1 and fig 1 describe the subjects in terms of age, sex, cerebrovascular accident-type, visual-field defect, central field sparing before and after training, and time since lesion. For all subjects, except FL and WV, anatomical T1-weighted MRI scans are added to show the lesions. Scans are shown in neurological convention (left = left, right = right) and lesions as dark areas. For subject PK, the scan is a T2-weighted scan shown in radiological convention (L = R, R = L); the lesion is shown as a light area.

## RESULTS

### Visual fields

Figure 2 shows the monocular pre-, mid- and post-training visual-field borders of two subjects that were presented with adaptive stimulus sets during training (results are comparable for both eyes). The stimulus locations in these sets are periodically adapted to improved stimulus detection performance during training.

Figure 3 shows the monocular pre-, mid- and post-training visual-field borders of nine subjects that were presented with fixed stimulus sets during training (results are comparable for both eyes). All subjects have hemianopias, either complete (PK, WD, PV) or incomplete (MS, EG, GL, IW). Subjects MK and IT

**Table 1** Description of subject sample

| Subject | Age (years) | Sex | Cerebrovascular accident | Visual-field defect | Pretraining central field border | Post-training central field border | Time since lesion (years) | Anatomy |
|---|---|---|---|---|---|---|---|---|
| FL | 67 | M | Infarct right occipital | Hemi-R | 2° | 17° | 3 | NA |
| WV | 52 | M | Infarct right occipital | Hemi-R | 5° | 11° | 4 | NA |
| MK | 55 | F | Haemorrhage left temporal/parietal | Quadr-LR | 5° | 8° | 9 | See fig 1A |
| MS | 69 | M | Infarct left parietal/occipital | Quadr-UR | 2° | 9° | 0.5 | See fig 1B |
| EG | 67 | F | Infarct right temporal/occipital | (Incomplete) hemi-L | >10° | >10° | 1 | See fig 1C |
| PK | 73 | M | Infarct right occipital | Hemi-L | 5° | 9° | 1 | See fig 1D |
| GL | 59 | F | Haemorrhage right parietal/occipital | Quadr-LL | >10° | >10° | 1 | See fig 1E |
| WD | 54 | M | Infarct right temporal/occipital | Hemi-L | <1° | 1° | 3.5 | See fig 1F |
| IT | 49 | F | Infarct right temporal/occipital | Quadr-UL | <1° | <1° | 1 | See fig 1G |
| IW | 46 | F | Infarct left occipital | Hemi-R | <1° | 4° | 1 | See fig 1H |
| PV | 67 | M | Haemorrhage right parietal/temporal/occipital | Hemi-L | 2° | 9° | 8.5 | See fig 1I |

Hemi, hemianopsia; L, left; Quadr, quadrantanopsia; R, right; U, upper.

had a paracentral scotoma, surrounded by a relative field defect area (grey area in fig 3). We did not study the training effect on the relative field defect. As can be seen in figs 2, 3, both types of stimulus sets yield gradual visual border shifts.

### Stimulus detection in fixed stimulus sets

Figure 4 shows the "temporal development" of the detection thresholds on all stimulus locations during all sessions for subjects PK, PV and WD (complete hemianopias). The curves are graphically sorted based on the distance of the stimulus from the vertical visual midline. With the Goldmann perimeter, 1000 apostilbs (≈318 cd/m$^2$) is the maximum stimulus luminance that can be presented. Therefore, detection of this stimulus is plotted at "1000." Non-detection of a presented stimulus is given an arbitrarily high value ("4000") so it can be distinguished from detection at maximum luminance ("1000"). A curve dropping below "4000" indicates the moment that a former undetected stimulus is detected for the first time. This generally seems to depend on the distance of the stimulus from the vertical midline: the more remote from the midline (and the original field border) a stimulus is presented, the more training sessions are needed before that stimulus can be detected. This spatial progression of stimulus detection corresponds to the visual border shifts of PK and PV shown in fig 3, whereas WD showed no training effect which corresponds to the many horizontal curves at value "4000' in fig. 4 (bottom), representing undetected stimuli. Also, stimulus detection appears to take place at still lower thresholds as training continues. In fact, after training, many stimuli are detected at normal thresholds compared with the unaffected visual hemifield.

For subjects MK, MS, EG, GL, IT and IW, stimulus detection thresholds cannot be sorted in a meaningful way as shown in fig 4, due to the irregular visual-field border shape. Therefore, fig 5 shows a chart of the fixed set of stimulus locations for all 40 sessions and label stimuli with three detection moments. Before training, only stimuli near the unaffected visual field are detected (marked in fig 5 with white circles). Halfway through the training, more peripheral stimuli are detected (grey circle locations). After training, stimuli are detected on black circle locations. Crosses denote locations where stimuli remained undetected. This spatial progression of stimulus detection corresponds to the changes in visual-field size that we observed after dynamic perimetry: the visual fields "grow" gradually (see also figs 2, 3). This corresponds to the gradual visual border

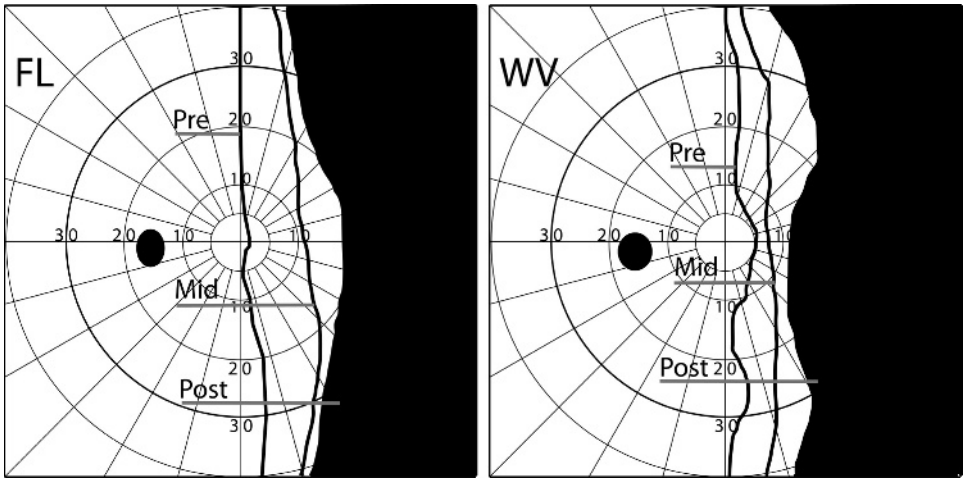


**Figure 2** Monocular pre-, mid- and post-training absolute visual-field borders of two subjects presented with adaptive stimulus sets during training (results are comparable for both eyes). The stimulus locations in these sets are periodically adapted to improved stimulus detection performance during training. In the black areas, no response is given to moving presented stimuli of maximum luminance.

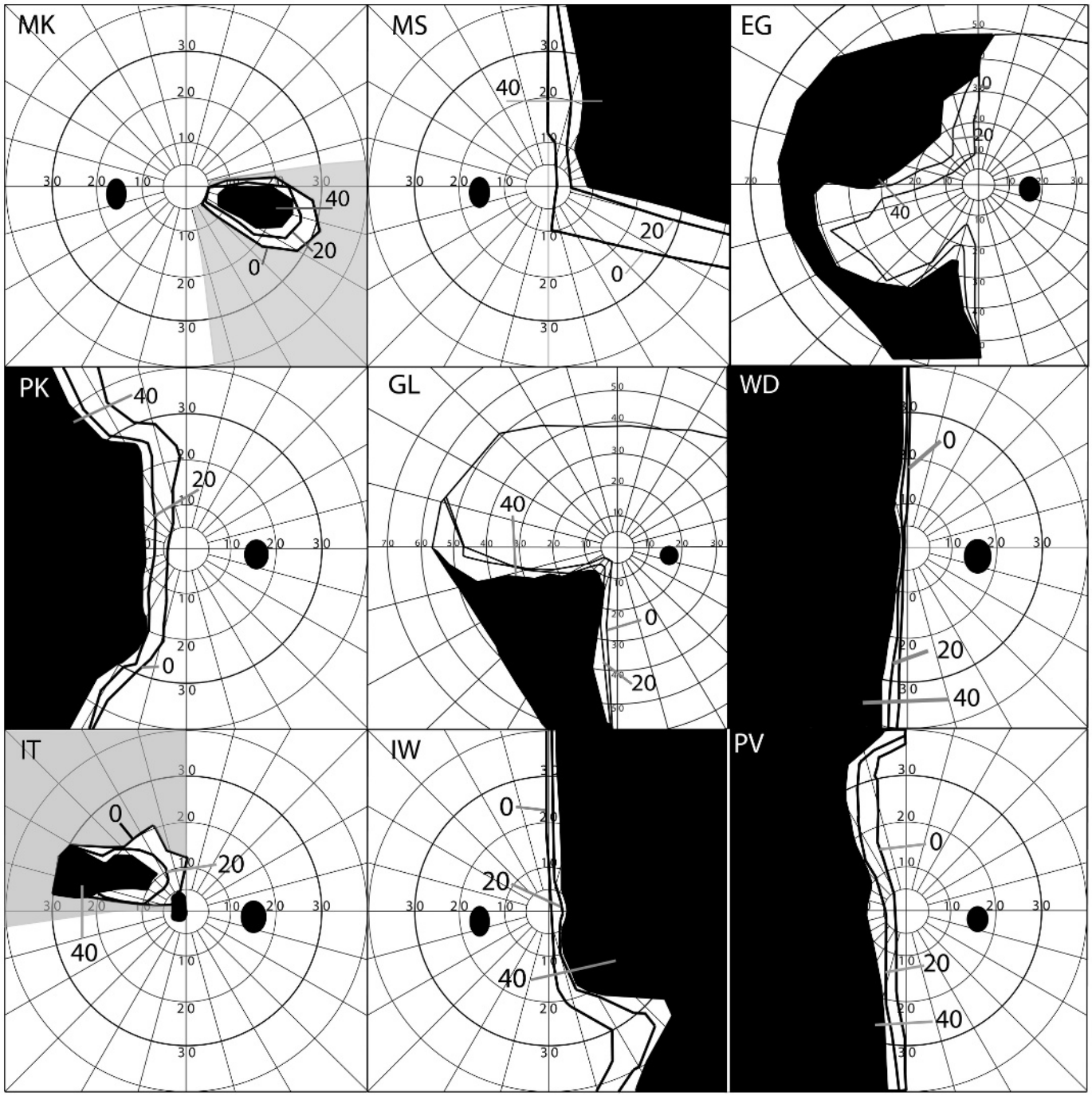


**Figure 3** Monocular pre-, mid- and post-training absolute visual-field borders of nine subjects presented with fixed stimulus sets (results are comparable for both eyes). The stimulus locations in these sets remained unchanged during the training. In the black areas, no response is given to moving presented stimuli of maximum luminance. Grey areas: areas of relative field in the quadrant—anopias of MK and IT.

shift. For comparison, the visual-field borders that are measured on the same moments (0, 20, 40 sessions) are also shown in fig 5.

### Eye-position analysis

During perimetry, we measured the eye positions continuously. Afterwards we considered eye positions during the period between stimulus presentation and the first detection, because this is the relevant time frame. Figure 6 shows two time-frame examples taken from subject PK.

Figure 6A shows a correct eye fixation. As can be seen, during the whole period of stimulus presentation the eye position does not deviate more than 2° from the fixation point. This can also be seen in fig 6B, where the same recording is plotted in an X–Y graph. In this particular registration, a stimulus was detected on a location on the 180° meridian and at an eccentricity of 11°. Figure 6C shows an example of a discarded perimetry measurement: after 1 s of stimulus presentation the eyes are turned towards the affected visual field and the presented stimulus, only to return to the central fixation point just before the detection response is given. At the moment of response, the stimulus was located on the 135° meridian and an eccentricity of 18° (approximately 13° from the central vertical midline). Based

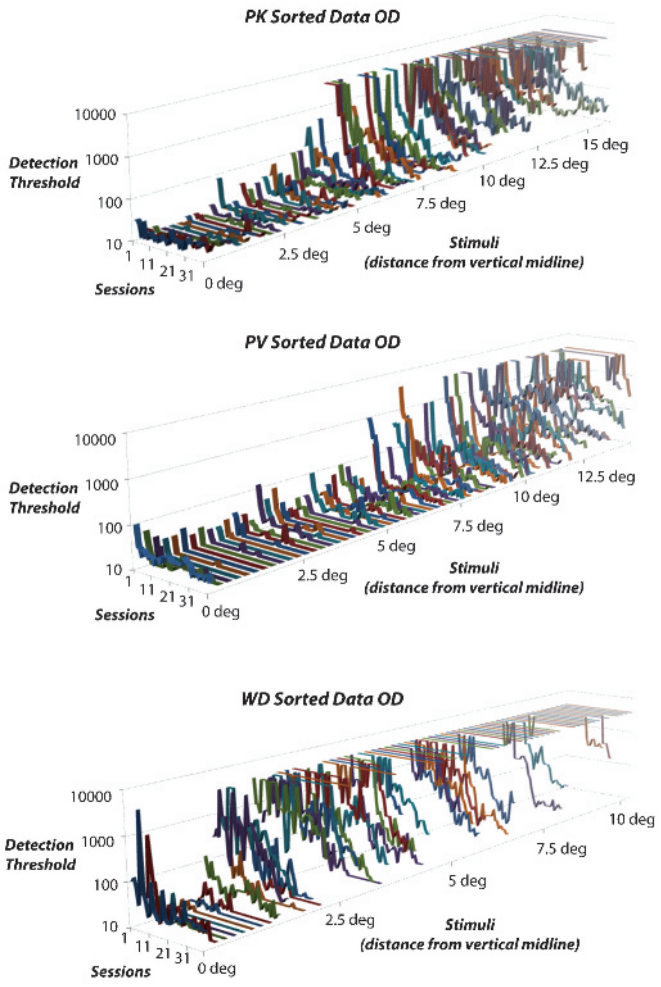


**Figure 4** History of the luminance detection thresholds of each stimulus location during the training period. Detection threshold curves are sorted by increasing distance from the central, vertical axis of the visual field. The value "1000" means stimulus detection at 1000 asb. The value "4000" was assigned when a stimulus was presented but not detected. (A) subject PK; (B) subject PV; (C) subject WD.

on trials with correct fixations only, we found visual-field enlargements varying from 4° (IW) to 13° (EG).

### Behavioural data

We have tested reading performance in seven subjects before and after training. (No data for MK (aphasia-related problems), MS (attentional reading problems), WV and FL (no Eyelink recordings)). The results are shown in table 2. The dependent variables are "reading speed" (words per minute) and "reading errors" (average number of regressions per line). We also looked at "average number of saccades and return saccades per line." The column on the right shows the pre–post training difference in reading speed. For each subject, reading speed was calculated for each line of text before and after training. Then, per subject, the difference between pre- and post-training reading speed means was tested using a paired-samples t test. Four subjects showed a significant improvement (*$p<0.05$; **$p<0.005$).

## DISCUSSION

In this study, we present data showing that visual-detection training can decrease detection thresholds in the trained visual-field area. This can also be the case in areas where there was no pretraining response on stimuli and a positive post-training response on stimuli at the same location (fig 4). In many cases, thresholds reach values comparable with normal. In this study, we compared the trained visual fields of two subjects, who were presented with a set of stimuli where the locations are near the visual-field border and are adapted to the subjects' performance, with the trained visual fields of nine subjects, who were presented with a wide-ranging stimulus set with fixed locations. We observed a gradual shift of the visual-field border, independent of the type of stimulus-set used (figs 2, 3). An eventual field enlargement can therefore not be caused by stimulus locations that are adapted to improved performance during training.

The gradually moving visual-field border keeps the same general shape. This could indicate that a pseudofovea developed progressively during training. We assessed the blind spot location at the start of all continuous Eyelink registrations during the perimetry sessions. A shift in foveal fixation would be manifested as a shift in the blind spot in the same direction. We did not observe a blind-spot shift during the several visual-field examinations. A progressively developed pseudofovea can therefore be ruled out as a cause for the visual border shift. The fact that the border shifts occur gradually may indicate that a stimulus needs to be in the vicinity of an originally unaffected or of a trained visual-field area in order to become detectable. This is supported by the fact that we did not succeed in training an "island" of stimulus detection within a blind field.

Eye movements can also be ruled out because we were able to limit our analysis to perimetry measurements that were made with correct fixations only, which were used to construct the visual-field border. During correct fixation it is possible that small eye movements (up to 2°) are made in the stimulus presentation period, so it could be argued that amplitudes of eye movements smaller than 2° should be extracted from the found perimetry values. We do not subtract the amplitude of these small eye movements from the found eccentricity of stimulus detection, because we do not know whether these small eye movements also actually led to stimulus detection. Sometimes such a deviation is made during the early part of presentation, when the stimulus is still located in the far periphery. Actual stimulus detection then takes place when the stimulus is closer to the centre and while fixation is actually central. Even if we were to subtract these amplitudes ($\leqslant 2°$) from the found detection eccentricities, we would still find a training effect in many subjects, but it may be underestimated. A recent study by Roth *et al* suggests that stimulation of the blind field does not improve attention and eye movements towards targets in the blind field.[23] This could mean that training in the shape of border stimulation would generate microsaccades that remain undetected by the blindspot probing. However, these micro-saccades are far too small to be the cause of the field enlargements of subjects MS, EG, PK, GL, IT and PV

Because the lesions of subjects MK, PV and WD are 9.0, 8.5 and 3.5 years old, spontaneous recovery is completely ruled out in these subjects. For the other subjects, spontaneous recovery seems unlikely (lesion ages of 6 months to 1 year). This is supported by the fact that in all subjects, the visual field at the beginning of training had not changed compared with the visual field during intake. We therefore conclude that the training accounts for the found effects.

But how does the visual training lead to visual-field enlargement? At the moment, this remains speculative. Any form of visual processing indicates that there are neurons present that serve visual functioning; relative field defects are

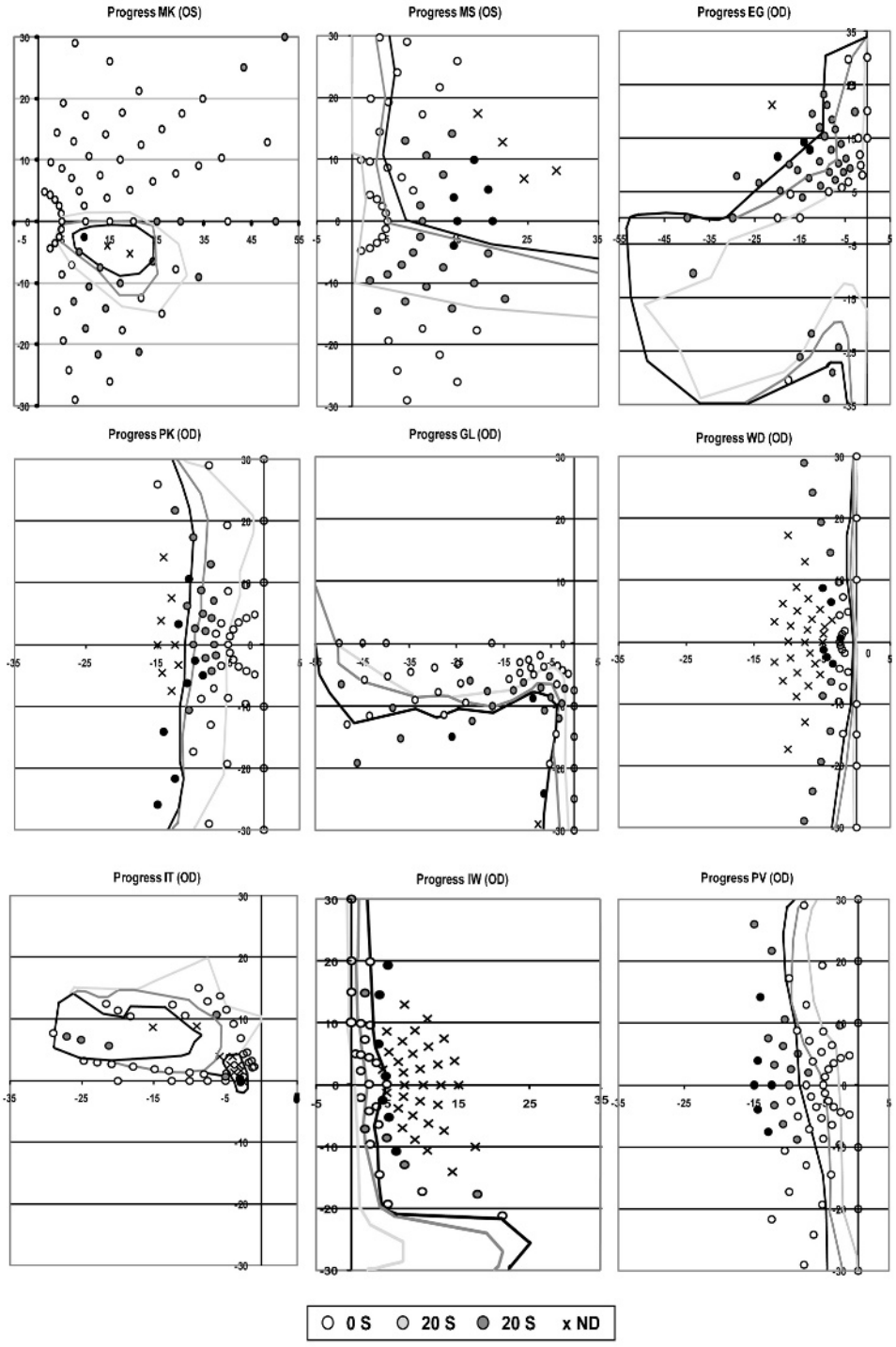


**Figure 5** Locations of stimulus detection at the start of training (white circles), after 20 training sessions (grey circles) and after 40 training sessions (black circles) of all subjects. Xs denote stimuli that remained undetected after all 40 sessions of training. The curves show the visual-field borders measured with dynamic perimetry at the start of training (light grey curves), after 20 sessions (dark grey curves) and after 40 sessions (black curves).

likely to be the consequence of residual or reduced neuronal activity after a subtotal lesion. Training, consisting of visual stimulation of the borderzone, may lead to increased sensitivity of spared but previously "silent" neurons. This means that less light energy is needed for detection, hence leading to decreased detection thresholds. The increased neuronal sensitivity can reach a critical level, needed for conscious vision. But there also may be so little residual neuronal activity in a certain brain area that stimulation of the related visual field does not lead to conscious vision. Conventional perimetry will thus underestimate that visual field as a "blind" area. If training enhances the activity of these neurons, this could lead to conscious detection of stimuli during perimetry: the visual field has "grown." The question then becomes: how does training lead to increased neuronal activity, that is decreased detection thresholds? This may be due to attentional effects, associated with training, on neuronal excitability. Zihl and von Cramon have shown how attention modulates light-difference thresholds in the visual field,[14] and Poggel *et al* describe how directed visual attention decreases detection thresholds in the visual-field area that is attended to.[26] Büchel and Friston reported that directing attention to visual motion led to increased connectivity and thereby increased neuronal activity.[28] Marshall *et al* observed in their imaging study that "(restitution) training appears to

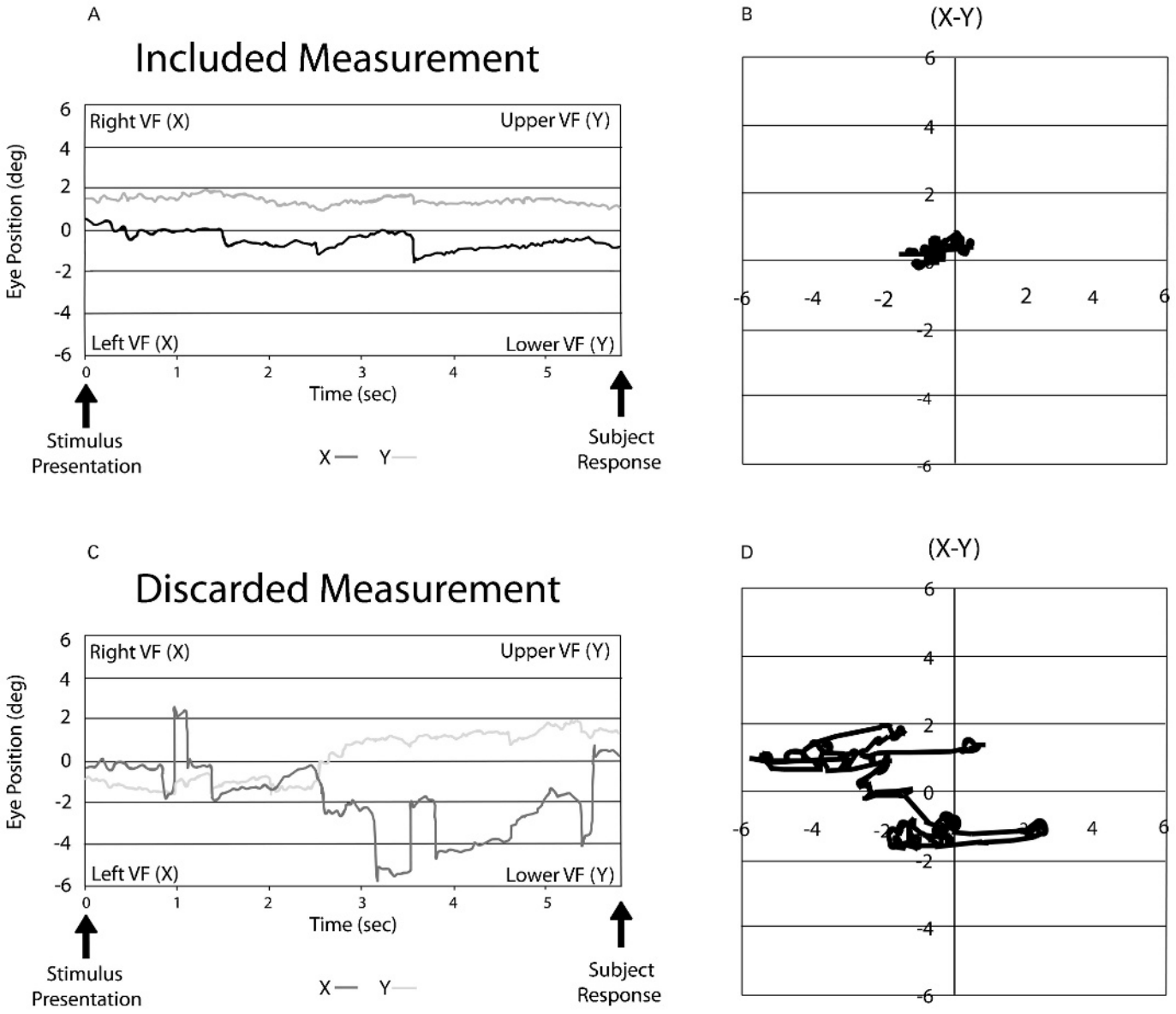


**Figure 6** Examples of eye position as a function of time. (A) Correct fixation during stimulus presentation and detection (eye movements $\leqslant 2^{\circ}$). Black curve: position in the x direction (up = right hemifield; down = left hemifield). Grey curve: position in the y direction (up = upper hemifield; down = lower hemifield). The arrows in the lower part of the graph represent start of stimulus presentation (black) and the detection response of the subject (grey). (B) Same eye position registration as in (A) shown as X–Y registration (bird's eye view). (C) Incorrect fixation during stimulus presentation and stimulus detection (eye movements $>2^{\circ}$). (D) Same eye position registration as in (C) shown as X–Y registration (bird's eye view). VF, visual field.

induce an alteration in brain activity associated with a shift of attention from the nontrained seeing field to the trained borderzone."[27] In our case, subjects cannot predict where a stimulus will appear, so they direct attention in the direction of the affected visual-field area. The training may teach subjects to successfully pay attention to visual input of which they were unaware before that moment. The subject may then become aware of this visual input, which brings us back to the fact that there must be spared neurons that can account for that visual input. In the case of the subjects with an old lesion age, this spared capacity is apparently absent for WD, and present for MK and PV. This may explain why some subjects show no recovery: a lesioned brain area may contain many or few spared neurons. If the number of spared neurons is too small, directed attention will not increase neuronal activity beyond the critical level that is needed for conscious perception. Of course, when there are enough spared neurons, stimuli must be sufficiently salient in order to ensure that subjects can attend to them. Just as different test stimuli may lead to differences in field testing, different training stimuli may lead to a different training outcome, as may be the case in the study by Roth *et al*.[23] Reading performance is improved significantly after training in four of seven subjects, which suggests that the training effects were also associated with an increase in reading performance. IW and WD show no improvement in reading performance, which is in agreement with the absence of visual-field enlargement (fig 3). The subjects with visual-field enlargement improved in total reading time (17–55% faster) and made fewer

**Table 2** Reading performance before and after training

| Subject | Pretraining read pro blems | Average per line pretraining | Average per line post-training | Pre–post difference | Reading speed pretraining (words/min) | Reading speed post-training (words/min) | Pre–post difference (words/min) |
|---|---|---|---|---|---|---|---|
| EG | 1 | Av sac 8.5 | Av sac 7.2 | Av sac −1.3 | 259 | 304 | +45* |
| | | Av reg 1.1 | Av reg 0.6 | Av reg −0.5 | | | |
| | | Av ret 1.6 | Av ret 1.3 | Av ret −0.3 | | | |
| PK | 1, 3 | Av sac 9.0 | Av sac 7.8 | Av sac −1.2 | 282 | 281 | −1 |
| | | Av reg 2.3 | Av reg 1.7 | Av reg −0.6 | | | |
| | | Av ret 1.4 | Av ret 1.2 | Av ret −0.2 | | | |
| GL | 1, 2 | Av sac 6.0 | Av sac 5.2 | Av sac −0.8 | 217 | 276 | +59** |
| | | Av reg 1.4 | Av reg 1.0 | Av reg −0.4 | | | |
| | | Av ret 2.6 | Av ret 2.5 | Av ret −0.1 | | | |
| WD | 1, 3 | Av sac 6.5 | Av sac 6.4 | Av sac −0.1 | 393 | 392 | −1 |
| | | Av reg 0.4 | Av reg 0.4 | Av reg 0 | | | |
| | | Av ret 2.9 | Av ret 2.7 | Av ret −0.2 | | | |
| IT | 1, 2 | Av sac13.1 | Av sac10.9 | Av sac −1.2 | 193 | 235 | +42** |
| | | Av reg 2.5 | Av reg 1.6 | Av reg −0.9 | | | |
| | | Av ret 1.9 | Av ret 1.7 | Av ret −0.2 | | | |
| IW | 1, 2 | Av sac12.2 | Av sac11.7 | Av sac −0.5 | 154 | 158 | +4 |
| | | Av reg 1.2 | Av reg 1.1 | Av reg −0.1 | | | |
| | | Av ret 1.9 | Av ret 1.8 | Av ret −0.1 | | | |
| PV | 1 | Av sac17.7 | Av sac13.5 | Av sac −4.2 | 150 | 233 | +83** |
| | | Av reg 6.0 | Av reg 3.7 | Av reg −3.2 | | | |
| | | Av ret 2.4 | Av ret 2.4 | Av ret 0 | | | |
| Mean | – | Av sac10.4 | Av sac 9.0 | Av sac −1.4 | 235 | 268 | +33** |
| | | Av reg 2.1 | Av reg 1.4 | Av reg −0.7 | | | |
| | | Av ret 2.1 | Av ret 1.9 | Av ret −0.2 | | | |

*$p<0.05$.
**$p<0.005$ (using t test).
1, missing parts of words; 2, reading slow, letter-by-letter; 3, trouble finding start of next line. AvReg, average number of regressions made per line read; AvRet, average number of return saccades after each line; AvSac, average number of saccades made per line read.

saccades and regressions, except for PK who did not read faster, although he showed an enlarged field and fewer saccades and regressions. Although a small scotoma remained present in the parafoveal region of IT, there was a substantial field enlargement, which may explain IT's improved reading. Subjects reported diminishing reading problems (EG, GL, IT and PV). PK, IW and WD did not experience any improvement in reading. An enlarged central visual field makes slightly larger saccades possible, so that fewer saccades are needed to read a line of text. This leads to faster reading. Apparently, faster reading may also improve comprehension of the read text, which is suggested by the reduced number of regressions in the same subjects. This result was also found by Zihl and von Cramon.[16]

Finally, subjects EG and GL started training with visual-field defects that did not affect the central 10° left of fixation, which is one of the crucial areas for reading speed. However, the visual fields and their enlargements, shown in fig 3, concern absolute defects only. Visual-field enlargement means that there is a visual-field area in which there was no detection of stimuli before training, and there is detection after training, in other words a change in an area with an absolute defect. A relative defect means that detection is present already. Enlargement cannot be shown with relative defects, and so we do not show them. Relative defects can improve, however. It means that the depth of the defect can decrease after training, because detection thresholds are lowered by the training (fig 4). This allows, for example, for improved peripheral acuity.[1] It is very likely that at all or most of the seemingly "unaffected" visual-field areas in the affected hemifields in fig 3 actually are relative field defects and that detection thresholds are lowered by the training in these areas. This may account for the improved reading performance in subjects EG and GL. The peripheral expansion may also allow for larger and thus a smaller number of saccades to the beginning of the sentence ("return saccades," "AvRet" in table 2), which EG clearly showed after training. GL only slightly decreased this number of return saccades.

GL remembered the main topic of one of the stories, which may have caused the reading speed to increase for that story but not for the other. EG did not report remembering any topic, so here the reduced number of return saccades may have contributed to the faster reading.

## CONCLUSIONS

Visual-stimulus detection training can result in visual-field enlargement that is manifested as a gradual shift of the visual-field border towards the visual-field defect during training. This gradual enlargement can also be observed when a stimulus set with fixed locations is used. We did not find any evidence for a slowly developing pseudofovea during training, or eye movements during perimetry, that can account for the training effect. Directing visual attention towards the visual-field defect may cause subthreshold stimuli to develop into suprathreshold stimuli and as such may be responsible for the training effect found. Training effects can be generalised to a daily-life activity such as reading.

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