Increased sensitivity after repeated stimulation of residual spatial channels in blindsight

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Lesions of the occipital cortex result in areas of cortical blindness affecting the corresponding regions of the patient's visual field. The traditional view is that, aside from some spontaneous recovery in the first few months after the damage, when acute effects have subsided the areas of blindness are absolute and permanent. It has been found, however, that within such field defects some residual visual capacities may persist in the absence of acknowledged awareness by the subject (blindsight type 1) or impaired awareness (type 2). Neuronal pathways mediating blindsight have a specific and narrow spatial and temporal bandwidth. A group of cortically blind patients (n = 12) carried out a daily detection ''training'' task over a 3-month period, discriminating grating visual stimuli optimally configured for blindsight from homogeneous luminance-matched stimuli. No feedback was given during the training. Assessment of training was by psychophysical measurements carried out before and after training and included detection of a range of spatial frequencies (0.5–7 cycles per degree), contrast detection at 1 cycle per degree, clinical perimetry, and subjective estimates of visual field defect. The results show that repeated stimulation by appropriate visual stimuli can result in improvements in visual sensitivities in the very depths of the field defect.

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Damage affecting striate cortex and the optic radiation as a result of stroke, trauma, or surgery can result in an area of blindness affecting the visual fields of both eyes (1). Within a visual field defect, some specific residual visual capacities may be demonstrated, either with or without limited awareness reported by the subjects. These abilities have been termed "blindsight" (2–4). Stimulus attributes such as orientation, color, motion, illusory contours, and emotional expressions have been reported within the visual field defects of some cases (see refs. 5 and 6). It is often assumed that after a limited "spontaneous" recovery within the first few months following brain damage, the remaining field defect is permanent and stable. Over these short periods of time, patients often perform remarkably consistently (7). There is, however, some evidence that repeated stimulation of the borders of the field defect over extended periods may lead to significant changes in sensitivity. Among the earlier studies are those reported by Zihl and von Cramon (8) showing that repeated stimulation during a saccadic localization task within the field defect can lead to increased visual sensitivity. More recently, repeated practice detecting a small light target at the borders of the field defect has been shown to lead to increased sensitivity (9). A common problem in studies involving stimulation of the sighted/blind field border is that such measurements are sensitive to small gaze shifts that may in some cases contaminate the data (10, 11). Evidence of changes in sensitivity deep in the field defect are rare, although not unknown. For example, the well known patient GY showed increased motion sensitivity over two testing sessions separated by 2 years (12), perhaps because of extensive testing on various tasks in many laboratories over that interval.

There are many reports of residual visual processing, in single cases and small group studies, of cortically blind patients. Commonly, sensitivity for the detection of basic visual attributes within the field defect is attenuated by between 1–2 log units compared with that in the corresponding eccentricities in the sighted field. These include responses to orientation discrimination (3, 13), spectral sensitivity (14), and pupillary responses (15). However, variations in methodology and experimental design do not allow for generalization of findings across subjects. We have been interested in studying spatial and temporal processing in blindsight by using the same metrics for all subjects. A systematic approach of identifying the spatial and temporal properties of channels mediating responses in GY demonstrated the existence of a narrowly tuned channel with bandpass characteristics and peak sensitivity at 1 cycle per degree and a temporal peak sensitivity between 10 and 20 Hz (16). Recently, a study of 10 cortically blind patients demonstrated the existence of a similar spatial channel in 8 patients (17). Given the evidence for increased sensitivity at the blindfield borders, it is of great interest to investigate whether visual stimulation deep within the field defect, with stimuli designed to maximally excite the spatial channels of processing, can lead to increased sensitivity in the blind field. Should this outcome be the case, can the increased sensitivity also lead to some awareness of visual events? Here we report on a small group of blindsight patients (n = 12) who have carried out a psychophysical discrimination task daily (training program) for a period of 3 months. A range of subjective and objective measures of visual sensitivity before and after the training sessions show evidence of increased visual sensitivity.

Results

A group of 15 cortically blind patients were recruited for this study. Three patients were excluded: two for withdrawing from the study before completion and one who had a second stroke shortly before commencing the after-training tests (see *Methods* and Fig. 6, which is published as supporting information on the PNAS web site, for a full description of brain lesions, case histories, brain scans, and visual field defects of the patients).

Prior to the training program, patients' visual fields were assessed in two separate sessions by using a Humphrey automated perimetry analyzer (both 10–2 and 30–2 full thresholds). On the basis of visual field data, four separate field locations were identified in each subject: two locations as candidates for repeated stimulation during the training, one as a control area that was not stimulated, and one straddling the edge of the field defect. Presentations on the blindfield borderlines were included to keep the patients' attention on the task and to avoid boredom because of monotonous presen-

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Abbreviation: 2AFC, two-alternative forced-choice.

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tation of stimuli within the blind area. During four to six separate visits to the laboratory, a number of measurements were carried out before the training to obtain baseline measurements of visual sensitivity for each individual at one of the retinal locations chosen for the training and at the control area. These measurements included detection for all subjects of a range of contrasts with spatial frequency held constant (1 cycle per degree, $n = 12$) and detection of a range of spatial frequencies with contrast held constant (50%). For the latter condition, it was not possible to find a single fixed value of contrast that was suitable over the whole range of spatial frequencies for these clinical subjects, for whom protracted threshold determinations were not practicable, but it proved possible in seven subjects. In addition, visual field measurements using automated perimetry, as well as subjective field measurements, were recorded for all subjects. These procedures were repeated 3 months later to assess the effect of repeated stimulation in the intervening period.

Repeated Stimulation Training Task. A full description of this task is given in *Methods*. In brief, by using computer equipment specially designed and installed in their homes, the patients carried out discrimination of a vertical sinusoidal grating vs. a uniform field in a temporal two-alternative forced-choice (2AFC) task. The gratings were circular patches (1 cycle per degree, 6° in diameter) modulated temporally at 10 Hz, with space-averaged luminance that was the same as the background (37 cd/m^2). Initially, the grating contrast for all subjects was set at 90% (Michelson contrast), and the contrast for stimuli in all locations was kept constant during each session. At the end of each session, the contrast was automatically adjusted by the computer program to maintain performance at a constant level (see *Methods*). Each training session consisted of 50 trials at each of the three visual field locations (150 trials total). The two temporal intervals in each trial were signaled with audible beeps, and a separate beep signaled the end of the trial. By means of response keys, patients indicated in which of the two intervals the grating was presented. Also, by using commentary keys after each trial, patients reported (binary scale) whether they had any awareness of the stimulus presentation. As in previous studies (18), patients were asked to respond "no" only if they had no awareness whatsoever of the visual stimulus. No feedback was given to the patients as to whether they were correct after each trial, nor was feedback given after each session.

Fig. 1 shows a computer record of the performance during the training sessions of one patient for the three stimulus locations. At two locations deep within the field defect, performance remained at chance level (50%) for the first 25 consecutive sessions, and stimulus contrast remained constant at 90%. Over time, there was a gradual improvement, inasmuch as performance remained high even when the stimulus was presented at lower contrast levels (Fig. 1 *Middle*). Note that even when performance reached 90% after >80 training sessions, awareness was rarely reported, revealing blindsight type 1.

As expected, stimuli were always detected at the location straddling the sighted field (Fig. 1 *Bottom*). The computer record of the training sessions in Fig. 1 *Upper* for subject EB was perhaps the clearest in displaying an obvious improvement in performance with no change in the incidence of awareness reports. Another pattern, seen in six patients (identified as DL, JL, AM, CS, EBr, and SR), was a gradual reduction in contrast as induced implicitly by improved performance but with low or only moderate levels of awareness. Overall, the computer program succeeded in maintaining relatively constant measured performance levels. However, it would be difficult to characterize the individual subjects' patterns of response in the course of the training sessions or to predict the results of the psychophysical assessments. The latter are the results of importance.

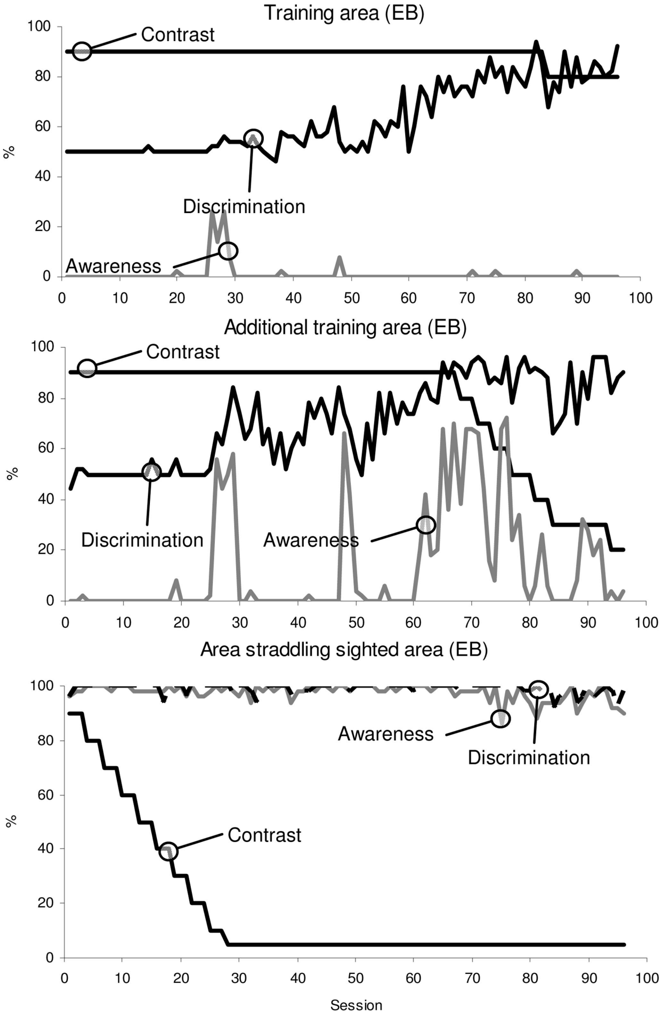

Fig. 1. Daily performance of one patient on a detection task. Results are shown for targets presented in the training area (*Top*), additional training area (*Middle*), and area straddling the sighted area (*Bottom*) in the blindfield.

Psychophysical Assessment 1: Detection of High-Contrast Gratings at Various Spatial Frequencies. Before the start of the training program, in a temporal 2AFC task, patients reported the interval in which a temporally modulated (10-Hz) circular patch of vertical gratings (6° diameter), at spatial frequencies in the range of 0.5–7 cycles per degree, was presented for a 2-s duration. The stimulus contrast was fixed at 50%. The other interval contained a uniform field with luminance equivalent to the space-averaged luminance of the grating patch (37 cd/m^2). Each trial also included a commentary-key response. The patients' visual fixations were monitored with a modified ASL 5000 pupillometer (Applied Science Laboratories, Bedford, MA). Results for the group are shown in Fig. 2. The data for both before and after training show the existence of a residual spatial channel with a lowpass characteristic similar to characteristics reported in ref. 17. The results and statistical analysis are presented for the seven subjects who qualified for this constant-contrast setting. Considering the discrimination data in the 2AFC task, a two-factor, within-subject, repeated-measure ANOVA (training × spatial frequency) showed a significant main effect of training [$F(1, 5) = 8.017$, $P = 0.019$, $\eta^2 = 0.616$] and spatial frequency [$F(5, 25) = 6.81$, $P < 0.001$, $\eta^2 = 0.577$] and of training × spatial frequency interaction [$F(5, 25) = 2.254$, $P = 0.04$, $\eta^2 = 0.311$]. Further post hoc paired-sample comparisons showed significant improvement after training for spatial frequencies of 0.5

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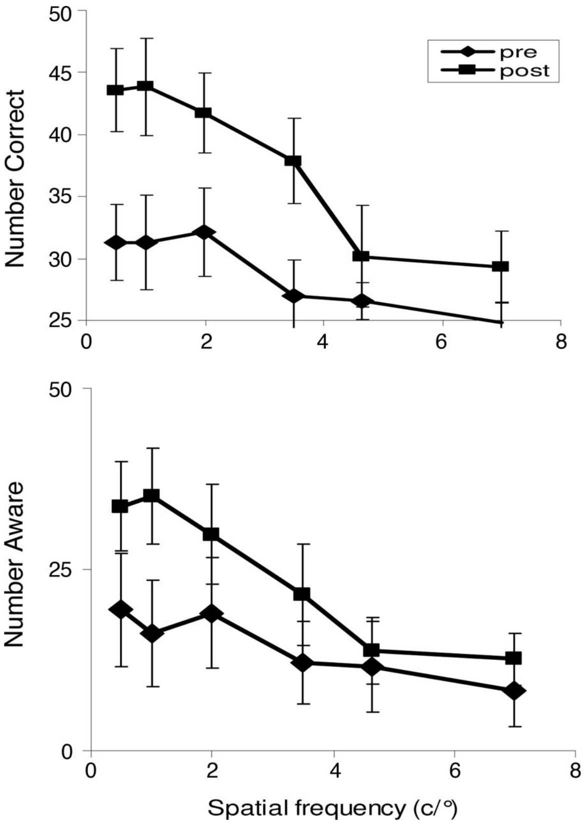

Fig. 2. Discrimination (*Upper*) and reported awareness (*Lower*) of cortically blind patients for stimuli presented within the blindfield, before and after taking part in the training task. Stimuli were circular spatial gratings (6° diameter) at a range of spatial frequencies and a fixed contrast of 50%.

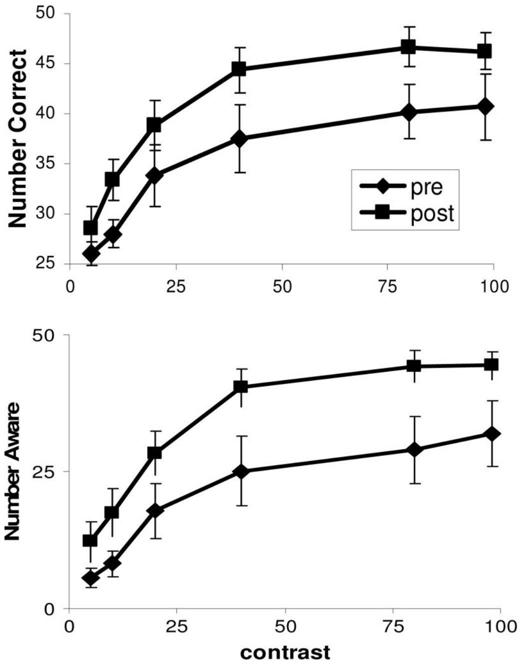

Fig. 3. Discrimination (*Upper*) and reported awareness (*Lower*) of cortically blind patients for stimuli presented within the blindfield, before (filled diamonds) and after (filled squares) taking part in the training task. Stimuli were circular spatial gratings of 1 cycle per degree (6° diameter) at a range of contrasts.

cycle per degree (t = 3.179, df = 6, $P < 0.01$), 1 cycle per degree (t = 2.376, df = 6, $P < 0.027$), 2 cycles per degree (t = 2.716, df = 6, $P < 0.017$), and 3.5 cycles per degree (t = 3.113, df = 6, $P < 0.011$). The improved performance did not reach significance at 4.7 and 7 cycles per degree.

Considering the awareness reported in the commentary-key paradigm, a two-factor repeated-measure ANOVA (training × spatial frequency) showed a significant main effect of training [$F(1, 5) = 3.963$, $P = 0.05$, $\eta^2 = 0.442$] and spatial frequency [$F(5, 25) = 10.205$, $P = 0.012$, $\eta^2 = 0.671$]; however, a training × spatial frequency interaction was not significant [$F(5, 25) = 2.965$, $P = 0.073$, $\eta^2 = 0.372$]. Further post hoc paired-sample comparisons revealed significant improvement after training for spatial frequencies of 0.5 cycle per degree (t = 2.120, df = 6, $P = 0.039$) and 1 cycle per degree (t = 2.556, df = 6, $P < 0.022$), but not for 2 cycles per degree (t = 1.408, df = 6, $P < 0.11$), 3.5 cycles per degree (t = 1.445, df = 6, $P < 0.2$), or higher frequencies.

Psychophysical Assessment 2: Detection of a Grating of 1 Cycle per Degree at a Range of Contrasts. Before and after the repeated-stimulation training sessions, we measured detection of the same stimulus used in those training sessions, namely, temporal modulation at 10 Hz for a grating of 1 cycle per degree (the peak sensitivity of the residual blindsight spatial channel), over a range of contrast levels. We also used the same temporal 2AFC and commentary-key paradigm as earlier.

Results for discrimination performance and reported awareness for all 12 patients are shown in Fig. 3. As expected, performance increased monotonically with increasing contrast. Considering the discrimination data in the 2AFC task, a two-factor repeated-measure ANOVA (training × spatial frequency) showed a significant main effect of training [$F(1, 10) = 11.697$, $P < 0.004$, $\eta^2 = 0.539$] and contrast [$F(5, 50) = 25.675$, $P < 0.000$, $\eta^2 = 0.720$], but no significant interaction between training × contrast [$F(5, 50) = 0.534$, $P = 0.74$, $\eta^2 = 0.051$]. Further post hoc paired-sample comparisons showed significant improvement after training for all contrasts except the lowest (5%): 5% contrast (t = 1.128, df = 11, $P = 0.142$), 10% contrast (t = 2.640, df = 11, $P < 0.013$), 20% contrast (t = 2.639, df = 10, $P < 0.013$), 40% contrast (t = 3.383, df = 10, $P < 0.004$), 80% contrast (t = 2.501, df = 10, $P < 0.016$), and 98% contrast (t = 1.827, df = 11, $P < 0.046$). [Because three midrange contrast levels (15, 50, and 90%) tested in the first-recruited patient, DL, were different than those for the other patients (20, 40, and 80%), these data points were omitted from analysis.]

Considering the awareness reported by using a commentary-key paradigm for the range of contrasts tested, a two-factor repeated-measure ANOVA (training × contrast) showed a significant main effect of training [$F(1, 10) = 22.215$, $P < 0.001$, $\eta^2 = 0.690$] and contrast [$F(5, 50) = 26.149$, $P = 0.000$, $\eta^2 = 0.723$]; however, training × spatial frequency interaction was not significant [$F(5, 50) = 0.679$, $P = 0.642$, $\eta^2 = 0.064$]. Further post hoc paired-sample comparisons showed significant improvement after training for all contrasts except the lowest (5%): 5% contrast (t = 1.555, df = 11, $P = 0.074$), 10% contrast (t = 2.433, df = 11, $P < 0.017$), 20% contrast (t = 4.448, df = 10, $P < 0.001$), 40% contrast (t = 3.748, df = 10, $P = 0.002$), 80% contrast (t = 3.274, df = 10, $P = 0.004$), and 98% contrast (t = 2.300, df = 11, $P = 0.021$).

Specificity of Sensitivity Improvement to the Stimulated Areas. Fig. 4 shows the discrimination and awareness reports for the same group of patients (n = 7) for grating stimuli at a range of spatial frequencies presented to a control location, before and after the training program. All of the stimulus parameters were the same as those reported for the training area (Fig. 2), except that none of the patients received systematic stimulation in the control area during training.

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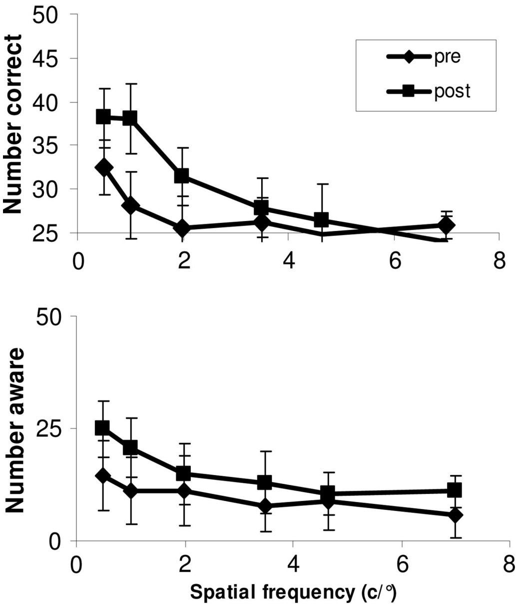

Fig. 4. Discrimination (*Upper*) and reported awareness (*Lower*) of cortically blind patients before (filled diamonds) and after (filled squares) training, for stimuli presented at a control location in the blindfield that was not stimulated during training. Stimuli were circular spatial gratings (6° diameter) at a range of spatial frequencies and a fixed contrast of 50%.

Considering the discrimination data in the 2AFC task, a three-factor repeated-measure ANOVA (locus × training × spatial frequency: $2 \times 2 \times 5$) showed no significant main effect of locus $[F(1, 5) = 3.216, P = 0.066, \eta^2 = 0.391]$ but a significant main effect of training $[F(1, 5) = 11.117, P = 0.011, \eta^2 = 0.690]$ and spatial frequency $[F(5, 25) = 9.936, P < 0.000, \eta^2 = 0.665]$. There were no significant interactions between locus × spatial frequency $[F(5, 25) = 1.694, P = 0.082, \eta^2 = 0.248]$, nor was there any significant three-way interaction [locus × training × spatial frequency; $F(5, 25) = 14.524, P = 0.158, \eta^2 = 0.2$], but there was significant interaction between training × spatial frequency $[F(5, 25) = 86.246, P = 0.006, \eta^2 = 0.425]$. Further post hoc paired-sample comparisons showed that, within the control area, there was significant improvement after training for spatial frequencies of 0.5 cycle per degree ($t = 2.022, df = 6, P = 0.045$), 1 cycle per degree ($t = 3.335, df = 6, P = 0.008$), and 2 cycles per degree ($t = 2.639, df = 6, P < 0.02$), but no significant change at 3.5 cycles per degree ($t = 0.737, df = 6, P > 0.23$) or above.

A similar analysis for awareness data indicated significant main effects of locus, training, spatial frequency, and locus × spatial frequency interactions. To explore this finding further, the control area awareness data were further analyzed by a two-factor repeated-measure ANOVA (training × spatial frequency). The results showed no significant main effect of training $[F(1, 5) = 2.774, P = 0.079, \eta^2 = 0.357]$ at the control area but a significant main effect of spatial frequency $[F(5, 25), F = 8.162, P = 0.000, \eta^2 = 0.620]$ and no significant training × spatial frequency interaction $[F(5, 25) = 0.906, P > 0.49, \eta^2 = 0.153]$. Further post hoc paired-sample comparisons also showed no significant improvement in reported awareness after training for any spatial frequencies (0.5 cycle per degree: $t = 1.101, df = 6, P > 0.15$; 1 cycle per degree: $t = 1.535, df = 6, P = 0.073$; 2 cycles per degree: $t = 0.398, df = 6, P = 0.302$; 3.5 cycles per degree: $t = 0.567, df = 6, P > 0.29$) or higher frequencies.

Our findings indicate that, in contrast to results for the training stimuli, in the control area there is no significant improvement in reported awareness at any of the spatial frequencies tested. The improvements in discrimination, although statistically significant, cover a narrower range of spatial frequencies than were found for the loci used in the training session.

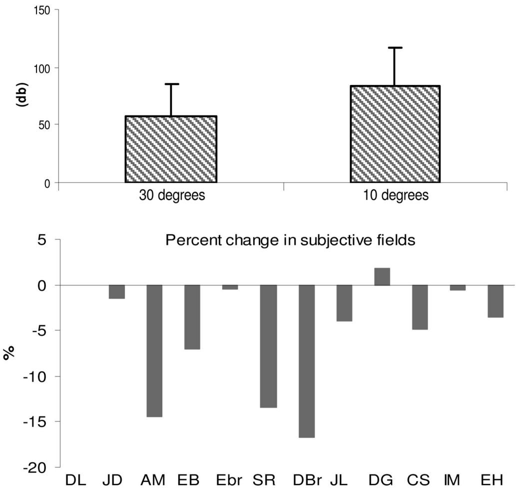

Fig. 5. Increase in visual field sensitivity in the central 30° and 10° of the affected hemifield after training, as measured by using a Humphrey visual field analyzer (*Upper*), and reduction in blindfield reported by using subjective fields (*Lower*).

Assessment of Visual Field Sensitivity by Clinical Perimetry. Visual field sensitivity in the central 10° and 30° fields in all patients was assessed by using a Humphrey automated visual field analyzer. Although the device and the programs used (full threshold and central 10–2 and 30–2 programs) have limitations in the assessment of residual processing in field defects in the absence of subjects' acknowledged awareness, their use was warranted because the device is currently the accepted clinical gold standard for defining the limits of the field defect and approximate thresholds of sensitivity. For each patient, visual fields were obtained over two separate sessions prior to the baseline measurements. These measurements were repeated twice 3 months later, after completion of the training program. To determine the extent of the field defect before choosing the training and control areas, we chose the higher sensitivity value recorded at each visual field test location. The change in sensitivity after training was determined as follows. For each patient at each visual field location, the average of two recording sessions before training was obtained. These values were summed over the entire affected hemifield to obtain an overall measure of visual sensitivity in each eye. The values for the two eyes were then averaged. Repeating this procedure for the data obtained in two recording sessions after training led to a value for visual sensitivity after training. Fig. 5 *Upper* shows the change in visual sensitivity (the difference between before- and after-training values) averaged across all patients. As indicated in Fig. 5 *Upper*, central 10° and 30° both showed increased sensitivity. The increased visual field sensitivities were significant (one-sample t test vs. value 0) for both central 10° ($t = 2.062, df = 10, P = 0.033$) and 30° ($t = 1.852, df = 11, P < 0.046$).

We also estimated each patient's subjective assessment of their field defect by asking them to draw, within a circle, their perception of their field defect. This technique has been used as a means of measuring the perceptual impact of a visual field defect on an individual (19). The difference in size of the affected area before and after training was calculated, and inferences could be made about any perceived changes. DL was the first case to participate in this study. Because we added the measurements of the subjective fields and central 10° visual fields after DL had started the study, data for DL are missing in Fig. 5. The subjective visual field loss averaged between the left and

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right eye in other cases indicated a significant shrinkage of the field defect ($t = 3.096$, $df = 10$, $P = 0.011$; two-tailed test). The apparent shrinkage of the field defect after training appeared to be similar in both eyes ($t = 2.110$, $df = 10$, $P = 0.061$).

Discussion

Since the demonstration of a retinotopic representation of the visual field in the striate cortex of brain-injured soldiers in two world wars, there has been much interest in the extent of the blindness and the properties of any residual vision within the field defect (20–22). The convergence of recent evidence from a variety of methodologies, including psychophysics, electroencephalography, functional brain imaging, and pupillometry, has established the presence of residual visual processing with altered or absent normal awareness by the patients, termed "blindsight." The existence of blindsight is not inherently surprising given that there are several retinal routes to the brain that can bypass the primary visual cortex (see refs. 5 and 23 for review).

Based on evidence from animal studies that changes in visual sensitivity can occur with training in monkeys with ablated striate cortex (24, 25), even showing that extensive retraining can lead to the recovery of visual shape discrimination (26), interest has developed in the possible beneficial rehabilitation of human patients and shrinkage of their field defects. To date, one major strategy has focused on reducing the size of the field defect by concentrating on training at the boundaries between the sighted and blind fields (8, 27). The approach of the present study was based on the assumption that if the narrow spatial and temporal bandwidths characteristic of blindsight are used for retraining, it is possible that repeated stimulation, even deep within the field defect, may lead to increases in sensitivity. In particular, we chose a spatial frequency grating of 1 cycle per degree, with temporal modulation at 10 Hz, as optimal parameters for exciting the neuronal mechanisms mediating blindsight. Patients were deliberately chosen whose field defects were measured beyond the period when changes in sensitivity can occur in association with subsidence of ischemic and other acute postlesion effects of the brain damage. The study subjects' lesions occurred on average 13 months before retraining and, in two cases, much earlier (CS at 37 years and EH at 4 years).

By changing the stimulus contrast automatically, performance levels of success were kept more or less constant during the 3 months of retraining. The result that emerged during the training regime itself was that patients were able to detect gratings at lower contrast levels at the end of the 3 months of training than at the beginning, with improvement occurring gradually or erratically, although in some cases steeply (e.g., as shown in Fig. 1). The use of the temporal 2AFC technique ensured that the ability to respond to lower contrast levels could not be attributed to a shift in subjective response criteria. Interestingly, improvement could occur even though reported awareness levels often remained low or absent during the retraining sessions. The after-training psychophysical assessments in the laboratory revealed an increase in reported awareness levels, along with improved performance levels, over a range of spatial frequencies tested and also for varying contrasts with spatial frequency fixed. Patients were given no feedback during the training sessions but were debriefed after completion of the study. Thus, perceptual learning in blindsight can occur over time as a result of repeated stimulation in the absence of feedback, revealing learning and plasticity within the neuronal mechanisms mediating blindsight.

Some improvement was also observed in psychophysical tests for the "nonstimulated" control loci, although there was no significant increase in awareness levels of the control loci, in contrast to the result for the tests in the retraining regions. However, the control regions were often necessarily spatially quite close to the retraining regions to ensure that both the control and training loci were matched for eccentricity and separation from the blind field borders, therefore spatial stimulus generalization might have occurred. A possible additional factor is one of attention. In the rehabilitation training, the subjects learn to attend to the blind field, which they typically often ignore in everyday interactions in favor of greater attention to their areas of normal vision. General improvements in the direction of visual attention may also have had a role in the increased sensitivity found for the nonstimulated area of the blind field, together with the use of stimuli especially configured for blindfield sensitivity.

Overall, the findings reported here indicate that repeated stimulation of cortically blind visual field defects with appropriate stimuli can lead to significant changes in visual sensitivity. It cannot be absolutely ruled out that the increased sensitivity might have occurred even without the training intervention, although it seems very unlikely, especially given the long postlesion intervals, the steepness of some of the changes, and the reduced effects in the control nonstimulated loci. To be certain, however, it would be necessary to embark on randomized trials with separate nonintervention groups, for which the present positive findings provide an essential prerequisite.

Methods

Participants. Patients were recruited from patients referred to the Acute Stroke Unit and the Ophthalmology, Neurology, and Neurosurgery departments of the Aberdeen Royal Infirmary (Aberdeen, U.K.). The main inclusion criterion was a homonymous visual field defect after damage to the occipital cortex or optic radiation as determined by computer tomography scans during admission. Most participants (10/12) also underwent MRI of the brain for more detailed assessment of their visual pathway lesion. All those with mobility problems, previous ocular surgery, or any retinopathy or cataract were excluded. None of the patients has significant cognitive deficits, as determined by examination by the clinical neuropsychologist at the hospital. Fig. 6 provides a summary of the lesions, brain scans, visual fields, and location of the training areas for the study subjects. Ethical approval was granted for this study by the Grampian Research Ethics Board, and the study was carried out in accordance with the Helsinki convention. The hypothesis tested in this study is that repeated stimulation improves the performance of patients on a number of psychophysical tasks. Given the hypothesis tested, the significance values given, unless stated otherwise, refer to one-tailed distributions.

DL is a 52-year-old male patient tested 30 months after stroke. The radiological report based on his MRI scans states ". . . there is an old infarct in the anteromedial aspect of the left occipital lobe. This involves the anterior aspect of the primary visual cortex and the underlying white matter. Elsewhere in the brain there are tiny white matter areas of increased signal on T2 weighted images, typical of ischemic change. These are noted in the periventricular white matter and in the left side of the mid brain."

JD is a 41-year-old female who was tested 12 months after stroke. The radiological report based on her MRI scans describes evidence of bilateral old occipital infarcts, larger on the right. "On the right the area of gliosis involves the posterior aspects of the primary visual cortex and adjacent posterior parietal lobe and measures 70 × 58 × 31 mm in maximum dimensions. The left sided infarct is smaller and more inferior."

AM is a 56-year-old female patient tested 15 months after stroke. The radiological report based on her MRI scans states ". . . there is an old infarct in the left occipital lobe involving the occipital pole and the cortex medially and laterally, immediately adjacent to this."

EB is a 54-year-old female patient tested 11 months after stroke. The radiological report based on her MRI scans states ". . . there is an old infarct in the left occipital lobe involving the occipital pole and underlying white matter but with some sparing of the primary visual cortex. There is no involvement of the lateral geniculate nucleus. However, there is evidence of more widespread cerebro

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vascular disease with small infarcts in the right internal capsule and corona radiata, right thalamus and pons."

EBr is a 59-year-old male patient tested 12 months after stroke. The radiological report based on his MRI scans states "There are multiple small white matter lesions returning increased signal on T2 weighted images, typical of small vessel disease. These are present in the frontal and parietal white matter, in the medial temporal lobes and in the mid brain and pons. In addition, there is a well defined ovoid lesion involving the right optic tract/optic radiation, typical of a small infarct. No thalamic abnormality or occipital cortical abnormality is demonstrated."

SR is a 55-year-old female patient tested 9 months after stroke. The radiological report based on his MRI scans states ". . . there is gliosis extending from the occipital horn of the right lateral ventricle to the primary visual cortex, typical of old infarction. No other intracranial abnormality. The thalamus is not involved."

DBr is a 60-year-old male patient tested 19 months after stroke. The radiological report based on his MRI scans states ". . . there are several areas of gliosis in the right middle cerebral arterial territory. The largest involves the right posterior parietal lobe with further areas of gliosis in the right anterior temporal lobe and subcortically in the right postfrontal region. All are typical of old infarction. There is no involvement of the thalamus or primary visual cortex."

JL is a 83-year-old male patient tested 12 months after stroke. The radiological report based on his MRI scans states ". . . there is gliosis in the left occipital cortex involving the primary visual cortex and underlying white matter, extending to the occipital horn of the left lateral ventricle. There is no involvement of the lateral geniculate nucleus. T2 weighted images demonstrate periventricular and mild deep white matter ischaemic change. There is also generalised cortical atrophy."

DG is a 69-year-old male patient tested 10 months after stroke. The radiological report based on his MRI scans states ". . . there is gliosis in the medial aspect of the left occipital lobe, involving the primary visual cortex. This extends anteriorly to involve the white matter medial to the trigone of the lateral ventricle but not as far as the lateral geniculate nucleus. There is minor ischaemic change in the right parietal white matter. No other intracranial abnormality is demonstrated."

CS is a 40-year-old female patient who suffered an ischemic occipital lesion after heart surgery at age 3. Details of a number of investigations on CS have been reported elsewhere (28). CS took part in this study 39 years postlesion. The radiological report based on her MRI scans states ". . . there is evidence of an old infarct in the medial aspect of the left occipital lobe. This is involving the primary visual cortex and the adjacent white matter and between the cortex and trigone of the left lateral ventricle, involving the terminal fibers of the optic radiation on that side."

IM is a 59-year-old male patient tested 4 months after stroke. The radiological report based on her computer tomography scans states ". . . small, focal low attenuation changes noted in periventricular white matter involving both frontal lobes and right lentiform nucleus in keeping with chronic ischaemic change. In addition there is a large wedge shaped low attenuation change in left parietooccipital region, typical of a recent infarct."

EH is a 52-year-old female patient who suffered occipital hemorrhage 47 months before testing. Because of possible metal implants, MRI was contraindicated. However, the computer tomography scans indicated the presence of a haemorrhage in the right posterior occipital lobe, leading to a left hemianopia.

Repeated Stimulation. Fig. 6 shows the location of the control area (black circles), training area (white circles), and two additional locations that were used during repeated stimulation (gray circles), one straddling the blind and sighted borders (gray circle with line). In a 2AFC paradigm, patients detected the presence of a circular (6° diameter) vertical grating patch at the spatial frequency of 1 cycle per degree, temporally modulated at 10 Hz, vs. a uniform patch. The grating had the same space-averaged luminance as the background (37 cd/m^2). There were 50 trials at each of three stimulated areas per session. A 2-min break was imposed at after 75 presentations, to ensure that the patients did not become fatigued. After each trial, the patient reported the interval containing the grating patch by pressing one of two response buttons. By pressing one of the two further response buttons, they also indicated whether they had any awareness of the stimulus presentation or no awareness (binary-scale, commentary-key paradigm). The stimulus presentations were self-paced, and the program paused if there were no responses. No feedback was given to the patients at any point during this study. The patients were debriefed after the completion of the study.

The stimulus contrasts were kept constant during a session. If, at any location, the performance was equal to or better than 42 of 50 correct in three consecutive sessions, then the contrast for stimuli at that location was reduced by 10%. If performance fell below 33 of 50 correct at any session, then the contrast for stimuli at that location was increased by 5% in the next session. This method ensured that performance remained high and relatively constant throughout the training session.

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