Visual Field Defects for Vergence Eye Movements and For Stereomotion Perception

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An objective visual field can be mapped in terms of stimulus-induced eye movement. The authors used the scleral coil technique to record vergence and conjugate eye movements while stimulating different visual field locations with a $3 \times 3$ deg target whose image vergence was oscillated. For each of three subjects tested there was a visual field location where vergence eye movements were much weaker than in a control location of equal retinal eccentricity. On the other hand, conjugate eye movements driven from these two locations by lateral motion were similar. Field defects for ocular vergence coincided with regions in which oscillating retinal disparity failed to produce a sensation of motion in depth, although visual responses to static disparity were normal, and psychophysical thresholds for lateral motion showed no defect with either binocular or monocular viewing. It was concluded, therefore, that the perceptual stereomotion scotomata were not due to a monocular loss, but to a defective binocular interaction between motion signals from the left and right eyes, and that this defective interaction was specific for opposed rather than parallel motion in the two eyes. Furthermore, the visual loss was specific for motion rather than for position. The correlation between the field defects for ocular vergence and stereomotion perception leads the authors to suggest that the same defect in binocular interaction is responsible for both the eye movement and sensory abnormalities. Two candidate hypotheses are proposed: one is framed in terms of a single population, and the other in terms of two populations of cortical neurons. Invest Ophthalmol Vis Sci 27:806-819, 1986

The concept that part of the visual field can be blind while other parts are sighted is familiar, because every human eye is blind at the optic disc. Acting as one’s own experimental subject, it is easy to convince oneself that the blind spot is absolutely blind in the sense of providing no visual sensation when illuminated. A less familiar concept is that of a local region of the visual field within which sensitivity to one specific visual submodality is depressed or totally absent, while sensitivities to all other visual submodalities are comparatively normal.

The so-called “stereomotion scotoma” provides a putative example of a field loss that is specific to one visual submodality. Stereomotion* is the sensation of motion in depth induced by stimulating the left and right eyes with a target that moves in horizontally opposed directions on the two retinae so that binocular disparity continuously changes.2-5 This sensation can be strong and compellingly realistic, but in the visual fields of many subjects there are areas called stereomotion scotomata where disparity oscillations fail to produce a sensation of motion in depth. A stereomotion-blind area of this kind may be only a degree or two in diameter, or it may extend over a quadrant or even over most of the field.1 Even subjects with perimetrically normal fields and no known neuro-ophthalmological history commonly have stereomotion scotomata.1,6 Some stereomotion scotomata exist for crossed disparities, but not for uncrossed disparities, or vice versa. In other words, the same retinal areas may be stereomotion blind or not depending on the disparity, so that the defect presumably originates after the stage at which the two eyes interact.1 As to the question of specificity, Richards and Regan1 reported that discriminations of “far” versus “near” can be achieved in field regions blind to stereomotion.

motion induced by changing disparity is abolished by closing one eye, and is abolished or much reduced by removing reference marks from the visual field,3,4 but neither of these maneuvers much affects the production of motion in depth sensation by changing size.3 In addition, the dynamics of the two processes are different.11 This article is restricted to motion in depth sensation produced by changing disparity, and this we call “stereomotion”.

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* It is necessary to distinguish between the production of motion in depth sensation by changing-disparity stimulation and by looming (i.e. changing-size) stimulation. Motion in depth sensation produced by changing disparity is abolished by closing one eye, and is abolished or much reduced by removing reference marks from the visual field.
motion blindness seems to be specific for continuous oscillatory motion: some impression of motion perception can be regained by stroboscopically illuminating the target so that it abruptly jumps from one disparity to another.1

Stereomotion scotomata include at least five kinds of abnormality. Rather than seeing sustained depth oscillations within a stereomotion scotoma, subjects report one or more of the following: (1) motion in depth is seen during the first few oscillations, but rapidly dies away; (2) diplopia occurs, even for low oscillation amplitudes, a kind of stereomotion loss that is equivalent to an abnormally small Panum’s fusional area for the perceptual and eye movement systems.

Previous studies of visual fields for stereomotion have been restricted to perception1,6: eye movements have been treated as an unwanted complication, and investigators have tried to avoid their possible effects by providing cues for convergence and by using non-nous lines to monitor vergence. Here we extend previous studies by measuring eye movements induced by stimulating different locations in the visual field. This can be regarded as a first step towards providing an objective description of the visual field as seen through the neural machinery that controls eye movements rather than through the mechanisms of perception. Our chief aim was to find whether or not stimulus-induced vergence and lateral eye movements are abnormal when driven from areas of the field that are behaviorally blind to oscillatory stereomotion and, in particular, whether visual fields for vergence eye movements show scotomata corresponding to the perceptual scotomata. This bears on the question whether the local defects in stereoscopic information processing is or is not shared by the perceptual and eye movement systems.

In brief, we find that there is a close correspondence between visual fields for stereomotion perception and stimulus-induced eye movements. In field areas “blind” to oscillatory stereomotion where lateral motion is seen normally, changing disparity stimulation induces only weak vergence eye movements, but stimulus-induced conjugate eye movements are normal.

Materials and Methods

Psychophysical Measurements

Informed consent was obtained from each of the five subjects after the nature of the procedures had been explained fully. Visual fields for stereomotion were measured with a technique based on one described previously.1 The image of a bar subtending $1 \times 0.2$ deg was back-projected through a red filter onto a screen via a mirror mounted on a galvanometer (General Scanning; Watertown, MA, model G300PD). The bar’s luminance was 14 cd/m². An identical arrangement imaged a second, independently movable, $1 \times 0.2$ deg bar onto the screen. This bar was green and also of 14 cd/m². Subjects wore a red filter in front of the right eye and a green filter in front of the left eye. In binocular vision a single fused target T was seen, so that stereoscopic stimulation could be achieved (Fig. 1A, B). The direction of motion in depth was determined as follows: when the red image on the screen moved to the left and the green image on the screen moved to the right, then the fused target T appeared to move in depth towards the head (Fig. 1A, B). Instantaneous position in depth was determined as illustrated in Figure 1: with the red image located on the screen to the left of the green image, the retinal disparity ($\alpha$) was said to be crossed, and the binocularly fused target T appeared to be in front of the screen. With the red image on the screen located to the right of the green image on the screen, retinal disparity ($\alpha$) was said to be uncrossed, and the binocularly fused target T appeared to be behind the screen (Fig. 1B). The plane of the screen was defined by projecting a white $30 \times 30$ deg Julesz pattern with an 18-min arc pixel size that had a 10-deg diameter circular hole at the center. The bar was located at the center of this hole.

First, visual fields were mapped in detail by moving the subject’s point of fixation rather than by moving the stimulus bar. The subject fixated on the image of a small white arrow subtending $1 \times 0.1$ deg whose position was controlled by the subject. Ocular convergence was assessed from time to time by means of nonious lines. Starting at an eccentricity of about 30 deg,
the subject slowly moved the fixation mark along a meridian towards the central bar. On request, the subject stated whether or not the bar appeared to be oscillating in depth, and the experimenter noted these responses on a field plot. Fortunately, this procedure for plotting stereomotion fields is straightforward because, as previously noted, the transition from a clear motion-in-depth sensation to no sensation is usually abrupt. Nevertheless, in order to assess repeatability, regions of stereomotion-blindness and of equivocal sensation were explored several times along different directions. In all, 16 half-meridia at 22.5-deg intervals were explored in each of the five subjects for both crossed and uncrossed disparities, and about 100–200 points tested for each field. The test bar’s disparity oscillation amplitude was held constant at 0–30-min arc and the frequency was 0.5 Hz.

These extensive 60-deg diameter fields were then used to select a stereoblind and a control location for detailed psychophysical and eye movement study. Stereomotion thresholds were measured for crossed disparities at a 0.5 Hz oscillation frequency by requiring the subject to adjust a potentiometer until depth oscillations were just visible. Each data point was the mean of ten settings. Lateral motion thresholds were measured by requiring the subject to adjust a knob until lateral motion oscillations were just visible. Ten settings were required for each point. Binocular lateral motion thresholds were measured at a constant disparity of 10-min arc crossed.

Thresholds for static disparity were obtained by two-alternative forced choice using the method of constant stimuli. This technique was preferred to the method of adjustment, because peripheral thresholds for disparity are considerably more difficult to set reliably than are peripheral thresholds for motion. Two 1.0 × 0.2 deg bars were presented stereoscopically. Each had a luminance of 14 cd/m^2. One bar was fixed in the plane of the screen. The disparity of the variable bar was randomly set to one of seven values. The subject’s task was to judge whether the adjustable bar was nearer or further away than the fixed bar. A psychometric curve based on 70 trials was obtained, and the disparity threshold obtained conventionally by Probit analysis.

**Eye Movement Recording**

Eye movements were recorded by the scleral coil method. Coils were placed in each eye, and eye position signals fed to a mini computer. Recordings made over a 32-sec period were stored on disc and could be analyzed off-line by averaging and by fast Fourier transform. Vergence eye movements were obtained by subtracting signals from the left and right eyes. The instrumental noise level was about 1 min arc, and frequency response was d.c. -100 Hz. Involuntary eye movements were induced by a stereoscopically presented random dot pattern subtending 3 × 3 deg with a pixel size of 6-min arc. Pixels were black or white with a probability of 50%. The left and right eyes’ half-images oscillated at 0.5 Hz through 1 deg peak-to-peak. When these oscillations were in antiphase, image vergence oscillated through 2 deg peak-to-peak and produced ocular vergence motion; when the half images were in phase, the resulting 1 deg peak-to-peak oscillation produced conjugate eye movements. Induced eye movements were small when stationary reference marks were present on the screen but were sufficiently
large to measure when reference marks were absent. Therefore, recordings were made with fixation marks removed. The following procedure ensured that the subject’s gaze did not wander appreciably during the 32-sec recording period. Before each recording, nonious lines were presented at the fixation point, while the subject gazed at the lines and confirmed that they appeared to be vertically aligned. Recording was then started, and the nonious lines were switched off. The eye position records were used to check that the initially correct fixation and convergence was maintained throughout the recording. In total, only one or two recordings were rejected on this account; subjects did not find it difficult to hold a steady angle of gaze. Note that subjects were not instructed to track the eccentrically presented target. Quite the contrary: they were instructed to maintain their initial convergence and direction of gaze. The stimulus target was located in one of two locations; either in a stereomotion-blind region of the field or in a stereomotion-sighted region at the same eccentricity. The two visual field locations from which eye movements were induced were as follows: 37.5 deg to the horizontal, 6 deg from the fovea in the left upper and right lower fields (subject 1); horizontal, 3 deg to left and right of the fovea (subject 2); 45 deg to the horizontal, 6 deg from the fovea in the left upper and right lower fields (subject 3). Psychophysical thresholds were measured along these same meridia.

Results

Visual Fields For Motion in Depth

The fields shown in Figures 2–5 are each based on up to 200 test locations, but for clarity we do not attempt to depict graded differences in stereomotion sensitivity within the stereomotion-sensitive areas, but rather to map areas of gross stereomotion blindness.

Figure 2A, B shows stereomotion fields for subject 1. The upper panel is for crossed disparity stimulation (Fig. 1A) for which the binocularly fused bar always appeared to be in front of the screen, and the lower panel is for uncrossed disparity stimulation for which the bar always appeared to be more distant than the screen. White areas signify that motion in depth was visible, while motion in depth was not perceived in the black areas. In the dotted areas of the visual field motion in depth was unstable, visible at some times but not at others. The two outlined black areas at about 16-deg eccentricity along the horizontal meridian are the two blind spots where, of course, stereoscopic perception was not possible. The chief feature of subject 1’s fields was the extensive regions of stereomotion blindness in the left and upper regions. Stereomotion perception was almost entirely restricted to the right lower field. The boundary between clearly visible stereomotion and no perceived stereomotion was particularly abrupt near the fovea in the left upper field; the transition occurred when fixation was moved by no more than 1 deg.

The most conspicuous feature of subject 2’s fields was a curious horseshoe-shaped stereomotion scotoma located 3 deg left of the fovea and approximately 2 deg wide (Fig. 3A). The scotoma did not terminate at the vertical meridian through the fovea, but extended approximately 0.5 deg into the right visual field. The edges

Fig. 4. Visual fields for stereomotion. Subject 3. Details as in Figure 2.

Fig. 5. Visual fields for stereomotion. Subject 5. Details as in Figure 2.
of the scotoma were remarkably sharp and reproducible from day to day. The transition from clearly perceived motion to stereomotion blindness certainly occurred over a distance of less than 0.5–0.75 deg (Figs. 6 and 7 show this in more detail). The horseshoe scotoma was evident only in the crossed disparity field. In the corresponding region of the uncrossed disparity field (Fig. 3B) only a small stereomotion scotoma was to be found, subtending approximately 2 deg × 1 deg.

The chief feature of subject 3's fields was a stereomotion scotoma of moderate size extending across more than one quadrant of the field (Fig. 4A, B). Although the boundary between clearly seen stereomo-
tion and stereomotion blindness was abrupt in several places, in other places regions of unstable stereomotion perceptions were interposed (dotted in Fig. 4). A small island of stereomotion blindness located about 7 deg above the fovea and subtending about 2 deg \(\times\) 5 deg was evident in the crossed disparity field (Fig. 4A); a small island of stereomotion perception subtending about 2 \(\times\) 2 deg could reliably be found within an extensive stereomotion scotoma in the uncrossed disparity field (Fig. 4B).

Our fourth subject’s visual fields included stereomotion scotomata, but these were too small and too peripheral to be studied by eye movement recording, so these fields are not shown.

Our fifth subject’s fields are included for completeness because of the rather unusual feature that they are full, almost complete and show no areas of stereomotion blindness (subject 5, Fig. 5A, B).

Thresholds For Motion in Depth and Thresholds For Lateral Motion Within and Near a Stereomotion Scotoma

Having mapped visual field areas out to 30-deg eccentricity where stereomotion was not seen, we then turned to the question of specificity, and selected for intensive study one stereomotion-blind and one control region in the fields of subjects 1, 2, and 3. Figure 6 plots absolute motion thresholds for three subjects. Subject 1’s stereomotion field showed large stereomotion scotoma in the left field extending to within about 3 deg of the fovea. Figure 6A is an oblique cut through the stereonormal area of the field into the stereomotion-blind area (see insert in Fig. 6A). The continuous line (open circles) plots stereomotion thresholds from a rightward eccentricity of 8 deg through the fovea to a leftward eccentricity of 8 deg. Threshold was lowest in the fovea. In the normal (right) field, log threshold rose progressively with eccentricity. In the left field a sharp-edged stereomotion-scotoma was superimposed on this gentle slope. The black area signifies a zone of absolute blindness for oscillatory stereomotion. Lateral motion thresholds presented a totally different picture (filled symbols). The stereomotion scotoma had no correlate in lateral motion sensitivity for the left eye (filled circles), nor for the right eye (filled squares), nor for binocular viewing of lateral motion (stars).

Findings were similar for subject 2. This subject’s stereomotion fields included a sharp-edged, horseshoe-shaped stereomotion scotoma (Fig. 3). Fig. 6B is a horizontal cut through this scotoma. The foveal stereomotion threshold was about \(\pm 0.015\) deg (\(\pm 0.9\)-min arc), comparable with previous reports.\(^2\,\,^3\) In the control (right) field, log threshold rose roughly linearly with eccentricity, reaching about \(\pm 0.1\) deg at an eccentricity of 8 deg. In the left field a sharp-edged region of absolute stereomotion blindness was superimposed on the uniform slope. Within the scotoma no sustained sensation of motion in depth could be elicited, even with the strongest stimuli used of \(\pm 0.46\) deg disparity oscillation amplitude. Figure 6B also shows that neither binocular nor monocular lateral motion thresholds gave any sign of this total failure of stereomotion perception. The scotoma could largely be attributed to a narrowing of Panum’s fusional area in that local area of the visual field: in the control zone at 3-deg rightward eccentricity, binocular fusion broke down at \(\pm 0.41\)-deg oscillation amplitude, that is at about six times the local stereomotion threshold, but within the scotoma, at 3-deg leftward eccentricity, fusion broke down at only \(\pm 0.14\) deg oscillation amplitude, so that Panum’s area was about three times narrower than in the control field. However, although this fusional restriction largely accounts for the scotoma, it is probably not a complete explanation because the fusional limit of \(\pm 0.14\) deg was still about twice the control stereomotion threshold, and no sustained motion in depth was seen in the scotoma even when oscillation amplitude was just below fusional breakdown (though a weak, rapidly decaying impression was visible for the first two or three oscillations).

Subject 3 provided an example of a stereomotion scotoma in which sensitivity loss was not total. The test oscillation that we used to map the fields of Figures 2–5 revealed a substantial stereomotion scotoma in his left visual field, and within this scotoma no sustained motion in depth was seen so that we classed the area as stereomotion-blind; the test oscillation was not strong enough to detect any residual stereomotion sensitivity. Figure 6C, however, shows that this scotoma was an elevation of stereomotion threshold rather than the absolute blindness to stereomotion experienced by subjects 1 and 2. (The black area in Fig. 6C has less vertical extent than in Fig. 6A, B to signify relative rather than absolute loss.) We might compare the Figure 6 scotoma to the relative scotoma of clinical perimetry, whereas the scotomata of Figure 6A, B are analogous to the absolute scotoma of clinical perimetry. Lateral motion thresholds (filled symbols) did not appreciably rise with eccentricity out to 8 deg within either the stereomotion-blind or the control areas. On the other hand, stereomotion thresholds were asymmetric, being up to two times higher in the scotoma than in the control area.

The stereomotion-blind and control areas of the visual field are quantitatively compared in Figure 7. Abscissae in Figure 7 are eccentricity along the directions of the cuts shown as inserts in Figure 6. Ordinates plot the ratios between thresholds in the left and right fields.
at eccentricities of 1 deg, 2 deg, etc. Figure 7 shows that, for all three subjects, lateral motion thresholds were accurately symmetric in left and right visual fields, but that the symmetry of stereomotion thresholds was sharply distorted in the stereomotion-blind areas. The value of using the subject’s own visual field as a reference is underlined in Figure 7C where the nature of the abovementioned “relative” stereomotion scotoma is clearly evident.

Visual Responses to Static Disparity in Stereomotion-Blind and Control Regions of the Visual Field

Subject 2 first used a 0.5 Hz disparity oscillation to confirm that a location 3 deg left of the fovea was indeed stereomotion-blind, whereas a location 3 deg right of the fovea was stereomotion-sighted. His next task was to make many judgments of whether a variable bar was nearer or further than a foveated comparison bar located in the screen’s depth plane (see Methods). Subject 2’s psychometric function was symmetrical, and centered within 5 min arc of zero disparity, whether the variable bar was located in the stereomotion-blind area or the control area of the visual field. Static disparity thresholds were approximately similar in the stereomotion-blind and control areas (7.0 and 5.0 min arc respectively). Similarly, subject 3’s psychometric functions were symmetrical, centered within 1-min arc of zero disparity, and apparent depth progressively grew with disparity. Disparity thresholds were similar in the stereomotion-blind and control areas (1.7 and 2.6 min arc respectively) and, in view of the substantial 6-deg eccentricity, low. Subject 1’s results were a little more complex. In the stereomotion-blind area, progressive increases of crossed disparity brought the bar progressively nearer, and the apparent depth between the variable and reference bars could be strikingly large. For uncrossed disparities, however, the situation was quite different. The largest uncrossed disparity produced only a weak and unreliable impression of depth, and the binocularly fused bar frequently disappeared abruptly. Visibility was never retained for more than 1 or 2 sec, but could be recovered by moving the point of gaze by a degree or so. It was as though Troxler’s effect were much stronger for uncrossed than for crossed disparities. Data for crossed disparities alone were analyzed: threshold was 2.6 min arc at the 6-deg eccentric location. In the control region of the visual field, the psychometric function was symmetrical, centered within 6 min arc of zero disparity and apparent depth progressively changed with disparity for both uncrossed and crossed disparities as reported by the other two subjects. Threshold was 1.9-min arc.

For subject 1, responses to static disparity were also estimated by placing both bars about 1 deg apart in the stereomotion-blind area. As the variable bar’s crossed disparity was progressively increased, its apparent distance in front of the reference bar grew progressively larger. The crossed disparity half of the psychometric function was of regular shape. Uncrossed disparities, however, produced no impression of depth, and the bar frequently disappeared abruptly. Nevertheless, although the bar seemed to remain in the fixation plane when disparities were uncrossed, uncrossed and crossed disparities could be distinguished using the artifactual cue that the bar made frequent abrupt disappearances and looked blurred when its disparity was uncrossed, but not when its disparity was crossed. Because the uncrossed disparity part of the psychometric function was aberrant we computed disparity threshold for uncrossed disparities only: this was 4.0 min arc compared with 4.7 min arc when the same technique was used to estimate threshold in the control area. The essence of these findings is summarized in Tables 1–3.
Eye Movements Induced by Changing-Disparity Stimulation in Stereomotion-Blind and Control Areas of the Visual Field

Figure 8A, B, C compares eye movements induced by changing-disparity stimulation in stereomotion-blind and stereomotion-normal areas of the visual field. Stimulation sites were located at the same eccentricity on opposite sides of the fovea in order to unconfound stereomotion abnormality from the normal effect of eccentricity. Eye movements caused by foveal stimulation are included to document the effect of eccentricity. Recordings are shown for subjects 1, 2, and 3, whose corresponding psychophysical data are given in Figures 2–4 and 7.

The chief finding of Figure 8 is that, for all three subjects, stimulus-induced vergence eye movements were considerably smaller in the stereomotion-blind area than in the control area. The correspondence between perception and visual fields was evident in detail. For example, stereomotion blindness for subjects 1 and 2 was more profound than for subject 3; correspondingly, vergence visual fields were more asymmetric in the abnormal and normal areas for subjects 1 and 2 than for subject 3. Furthermore, all three subjects confirmed a previous report\(^1\) that, although there was no sustained impression of motion in depth within the stereomotion-blind areas of the visual field, there was often an initial impression of depth motion that persisted for only two or three stimulus oscillations, and a weak, brief-lived sensation of motion was experienced intermittently thereafter: the vergence eye movement records of Figure 8 show just those features of some initial response followed by prolonged inactive periods interspersed with briefly sustained weak vergence motion that seldom reached the average level attained in the control area of the visual field. Table 4 lists quantitative data for the amplitude and phase of vergence and individual eye movements. Table 4 shows that both eyes responded more weakly to changing disparity stimulation in the stereomotion-blind than in the control region. This finding held for all three subjects.

For subjects 2 and 3, the phase lag of vergence movements was similar in abnormal and control regions, but phase lag was greater in the abnormal region for subject 1. Phase lags for subject 3 had the curious feature that the right eye’s lag was greater than the left eye’s; this subject’s right eye was dominant. Subject 3 was predominantly left-handed, as was subject 2.

Eye Movements Induced by Lateral Stimulation in Stereomotion-Blind and Control Areas of the Visual Field

Figure 9A, B compares eye movements induced by lateral-motion stimulation in the same stereomotion-blind and control areas as in Figure 8. Records are shown for subject 1 only, but findings were similar for the other two subjects. Conjugate (ie, versional) eye movements evoked by lateral motion stimulation were equally strong in the stereomotion-blind and control regions. Therefore, the absence of vergence eye movements was not due to an absence of motion sensitivity per se.

Discussion

Visual Fields For Stereomotion and Stereomotion Scotomata

Much as the classical visual field for a just-visible object is mapped by contours of equal detection threshold (called isopters),\(^8\) so the visual field for just-visible lateral motion\(^10\) for motion in depth can be mapped by plotting lines that join sites of constant motion threshold within the visual field. Visual thresholds for oscillatory stereomotion rise progressively with eccentricity so that, for any given disparity oscillation amplitude, motion in depth can be seen only up to some specific retinal eccentricity. All five subjects had more extensive lower than upper fields. Superimposed on the steady gradient of sensitivity are areas of the visual field that are “blind” to oscillatory stereomotion. Four of our five subjects had such stereomotion scotomata. Their locations and sizes were idiosyncratic.\(^6\)

We confirm a previous report\(^1\) that stereomotion scotomata range in size from a degree or two in diameter (Figs. 2A–B) through intermediate sizes (Fig. 4) to about half the area of the total stereomotion field (Fig. 2B). Conversely, areas of stereomotion sightedness range in size from tiny islands of about 1 × 1 deg (Fig. 4B) up to virtually the entire stereomotion field\(^1\) (Fig. 5B). The boundaries of a stereomotion scotoma can be precipitous: the transition from normal sensitivity to absolute blindness for stereomotion can occur over a distance of no more than 0.5–0.75 deg (Fig. 7B).

The chief features of subject 1’s stereomotion fields have remained unchanged over a period of over 14 yr: the stereomotion fields shown in Figures 1 and 2 of Ref 1 were recorded in 1971, and the fields reported here in Figure 2 were recorded in 1984.

It is not known whether stereomotion scotomata are important in everyday vision: few relevant quantitative studies have been reported. There has been some concern that a vehicle’s direction of motion in depth might be misjudged if it passed through a stereomotion scotoma, with potentially dangerous consequences both in traffic and when aircraft execute ground maneuvers in airports. However, it seems possible that the changing-size system takes over from the stereomotion system in stereomotion-blind areas of the visual field,\(^10\) though whether this leaves a residual deficit is yet to
be determined. (The dynamics of the changing-size system and the stereomotion system are quite different. For example, small, rapidly moving objects stimulate the stereomotion system more strongly than the changing-size system, so that behavioral responses to such objects might be impaired when the object passes...
through a stereomotion scotoma.\(^\text{11}\) Attempts have been made to find whether a pilot's landing performance is affected by the loss of binocular vision by occluding one eye while landing a jet aircraft,\(^\text{12-14}\) but the results are difficult to interpret\(^\text{11}\) because subjects whose sensitivity to changing-size is high will be comparatively unaffected by losing binocular vision, whereas subjects who are insensitive to changing-size will experience a severe loss of motion-in-depth information when one eye is occluded. A more academic problem, debated for many years, is why some strongly right-handed individuals adopt a left-handed stance in cricket and baseball. One possible reason may be that an individual with a left-field stereomotion scotoma would present the scotoma to the ball if a right-handed stance were adopted; the stereomotion-sighted hemifield can be presented to the ball only by adopting a left-handed or backhand stance. Figure 2 shows that

**Fig. 8.** Vergence eye movements (continuous lines) produced by stimulating a control area of the visual field (upper panel), the fovea (center panel), and a stereomotion blind area (bottom panel) with disparity oscillations of 2-deg peak-to-peak and frequency 0.5 Hz. The dotted lines show the stimulus positions. Records shown are 32 sec long. A, B, and C are respectively for subjects 1, 2, and 3.

### Table 4. Eye movement data

<table>
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<th>Subject</th>
<th>Abnormal area</th>
<th>Control area</th>
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<tbody>
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<td></td>
<td>Left eye</td>
<td>Right eye</td>
</tr>
<tr>
<td></td>
<td>amplitude</td>
<td>phase</td>
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<tr>
<td>2</td>
<td>0.17 (0.04)</td>
<td>-75</td>
</tr>
<tr>
<td>1</td>
<td>0.16 (0.04)</td>
<td>-111</td>
</tr>
<tr>
<td>3</td>
<td>0.22 (0.07)</td>
<td>-112</td>
</tr>
</tbody>
</table>

Mean amplitudes (in deg) and phase lags (in deg) of eye movements induced by stimulating abnormal and control regions of the visual field with oscillating disparity and oscillating lateral position. Stimulus oscillation amplitudes were ±1 deg and ±0.5 deg for disparity and lateral motion respectively. Sinusoidal oscillation frequency was 0.5 Hz in both cases. Results are shown for three subjects. Standard deviations are bracketed.
right-handed subject 1 has an extensive left-field stereomotion scotoma: it may not be coincidental that the subject is a left-handed cricket batsman.

Specificity of Stereomotion Blindness

A pervasive idea in vision research is that some abstract features of the retinal image are processed rather independently of one another. One kind of evidence for this idea is the existence of specific forms of blindness in which sensitivity to all other visual submodalities are unaffected. When stereomotion blindness is considered in this context, the degree of specificity is crucial. Here we consider the following three tests for specificity: Does the stereomotion-blind area retain (1) normal monocular sensitivity to motion; (2) normal binocular sensitivity to lateral motion; (3) normal stereoscopic sensitivity to position in depth? Although the general question of specificity has been discussed, this has been largely a qualitative level: relevant quantitative data have not previously been published.

Consider first subject 2's horseshoe-shaped stereomotion scotoma (Fig. 3A). Figures 6B and 7B compare stereomotion and lateral motion thresholds measured along a cut through the scotoma. As we pass from the control area of the field into the scotoma, stereomotion thresholds (continuous line in Fig. 6B) rise steeply, and we enter a zone of total stereomotion blindness (black area). On the other hand, monocular motion thresholds behave quite differently: they show no sign of the scotoma (Fig. 6B, dotted lines, filled circles and squares).
At points located 3 deg left and right of the fovea, monocular motion thresholds differ by only 20% for the right eye and by 50% for the left eye, whereas stereomotion thresholds differ by indefinitely more than 600% (see Results). The same point holds for binocular lateral motion thresholds: these were only 15% higher at 3 deg left of the fovea than at 3 deg right. The distinction between stereomotion and binocular lateral motion thresholds is emphasized in Figure 7 where threshold ratios are plotted for the stereomotion-blind and control areas. It should be noted that neither the left nor the right eye alone could possibly distinguish between the two cases: the only difference was that the phase difference between the two eyes was 0 deg in one case, and 180 deg in the other case. A similar dissociation between stereomotion and lateral motion thresholds was observed for subject 1 (Figs. 6A and 7A), and subject 3 (Figs. 6C and 7C).

Our finding that the three stereomotion scotomata of Figure 7 are not due to reduced monocular sensitivity to motion in either eye alone points to an abnormal binocular interaction as the cause of the stereomotion scotomata; further evidence that the site of processing abnormality is after binocular convergence is provided by the finding that different stereomotion scotomata can be found in the crossed and uncrossed fields (see also Figs. 2 and 3, and Ref 1). The abnormal interaction is specific to motion signals produced by opposed motion on the two retinae, since binocular lateral motion thresholds were unimpaired.

Having concluded that our subjects were blind to motion in depth but saw lateral motion normally, we now turn to the question whether visual sensitivity to position in depth was relatively spared, while sensitivity to motion in depth was selectively lost. Visual sensitivities to stereomotion and to static depth are summarized in Tables 1–3. Our chief conclusion is that it is possible to selectively lose the ability to see motion in depth, while retaining normal sensitivity, not only to lateral motion but also to relative position in depth; the visual fields of all three subjects contained restricted areas of such selective blindness to stereomotion. Less surprisingly, it is also possible to lose sensitivity both to position and to motion in depth (Table 1, subject 1).

Subject 2’s total loss of stereomotion perception was accompanied by a local fusional defect; diplopia occurred at an oscillation amplitude of ±0.41 deg in the control region, but at only ±0.14 min arc in the abnormal region but subject 2’s blindness cannot, however, be entirely attributed to fusional failure, because diplopia in the stereomotion-blind area did not occur until about twice the control stereomotion threshold.

Subject 1 reported that, in the abnormal area of the visual field, static disparity stimulation did not instantaneously generate depth sensation; rather the impression of depth built up over 1–2 sec. If the neural mechanism mediating static depth perception were the only stereoscopic mechanism, then this evident sluggishness would be expected to result in only weak responses to changing disparity.

Possible Physiological Bases For Vergence Field Defects

Figures 8 and 9 show that it is possible to record and measure eye movements driven by a small (2 × 2 deg) target located in different regions of the visual field. This finding raises the possibility of a new form of objective perimetry in which the “isopters” are contours of equal eye movement amplitude rather than being contours of equal subjective sensitivity. Eye movement perimetry might prove to be of both basic and clinical interest, in that such fields might provide complementary information to behavioral fields, since the neural pathways and mechanisms that govern eye movements are not entirely the same as those that determine perception.

Figure 8 shows that, for each of our three subjects, there is a zone in the visual field in which vergence eye movements induced by oscillating disparity are considerably weaker than in a control region of the visual field. Three possible explanations for the attenuation of vergence eye movements are as follows: (1) The amplitude of left and right eye movements are normal, but their relative phases are abnormal, thus causing partial cancellation; (2) The amplitude of one eye is attenuated, but not the other; (3) The amplitudes of left and right eye movements are both attenuated, but their relative phases are normal. Table 4 shows that our eye movement data rejects the first two possibilities and are consistent with the third above. This conclusion supports the idea that vergence and conjugate eye movements are controlled by neural mechanisms that are, to a substantial extent, independent (though, perhaps, not completely independent). In this context, the field defects of Figure 8 seem to reflect a specific abnormality of the vergence mechanism: although the left and right eyes’ responses were individually weak when the abnormal zone was stimulated by oscillating disparity, strong conjugate eye movements were elicited when the same field location was stimulated with oscillating lateral motion, and these strong conjugate eye movements were similar to the strong conjugate eye movements driven from the control region of the visual field (Fig. 9).

The full implications of these findings for eye movement control remain to be established, but we can say now that if stimulus-induced vergence eye movements are important in helping the eyes accurately converge
Possible Physiological Bases For Stereomotion Blindness

Two dichotomies are evident in our psychophysical findings: (1) motion in depth versus position in depth; (2) crossed versus uncrossed disparities (Tables 1–3).

First we consider the dichotomy between motion and position. Evidently, it is possible to lose visual sensitivity to both static and changing disparity (Table 1, uncrossed disparities), and it is also possible to lose sensitivity to changing disparity while retaining normal sensitivity to static disparity (Tables 1 and 2 crossed disparities and Table 3 crossed and uncrossed disparities). We consider the following two hypotheses:

Single-population hypothesis: One possible explanation for the above findings is that the visual system contains only one population of stereoscopic neural elements, all of which are d.c.-coupled and mediate static depth perception, but within the population there is a range of upper cutoff frequencies. Stereomotion scotoma would be caused by a selective loss of widebandwidth elements, leaving only the elements that respond from d.c. to a fraction of a Hz.

Two-population hypothesis: We have alternatively suggested that there are two stereoscopic mechanisms, one for relative position in depth and one for stereomotion; the first can be regarded as d.c. coupled, the second as a.c. coupled.22 Evidence that favors this second hypothesis above the first is provided by a report that selectivity to static depth can be lost while stereomotion sensitivity is spared1; in the present study, however, we found no scotoma of this kind. It is pertinent that some neurons in cat visual cortex have been reported to show binocular interactions that result in their being sharply tuned to the direction of motion in depth,27–29 if these two kinds of neuron exist in man, the motion-in-depth neurons might be absent or abnormal while the position-in-depth neurons were normal in the stereomotion-blind areas of the binocular visual field.

Now we turn to the dichotomy between crossed and uncrossed disparities evident in Tables 1–3. We can immediately reject the suggestion that this dichotomy might be an artifact of inaccurate convergence: our eye movement recordings show that convergence was maintained sufficiently accurately to eliminate this possibility. Some years ago, Richards suggested that crossed disparities and uncrossed disparities are processed by different neural mechanisms, and that depth discriminations are mediated by antagonism between these two mechanisms.30,31 In support of this idea, Poggio and Fischer32 have found four types of depth-sensitive neuron in the visual cortex of behaving rhesus monkey. Two of these classes are “near neurons” that respond well to stimuli in front of the fixation plane and are suppressed by those behind it, and “far neurons” whose behavior is opposite to that of near neurons. In this context, the loss of static depth sensitivity to uncrossed but not crossed disparities in Table 1 can be seen as the functional absence of the “uncrossed disparity pool”30,31 or the “far neurons”,32 a loss that is restricted to only part of the visual field. Thus, a stereomotion sensitivity loss for crossed but not for uncrossed disparities can be understood in terms of the “single population” hypothesis if, in the affected part of the visual field, “near neurons” had suffered a reduction in the upper cutoff temporal frequency, and in terms of the “two population” hypothesis if motion-in-depth neurons24–26 are segregated into near and far populations.

It is striking that for all three subjects whose eye movements were recorded, the field defect for vergence eye movements coincided with a field defect for stereomotion sensation. A second parallel between eye movements and perception is in the selectivity of the abnormality: both conjugate eye movements and lateral motion sensation were normal within the vergence/stereomotion field defect. These parallels lead us to suggest that the same abnormal binocular interaction between opposed-motion signals may underlie both vergence and sensory defects; candidate hypotheses for both defects are the “single population” and “two population” ones outlined above.

Key words: vision, eye movements, visual field, motion, stereopsis, figure-ground, retinal flow, image understanding

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