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Is the Motion System Relatively Spared in Amblyopia? Evidence from Cortical Evoked Responses

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Visual evoked potentials (VEPs) produced by pattern reversal were compared with those elicited by onset of motion in 37 amblyopic children (20 with anisometropic amblyopia, seven with strabismic amblyopia and 10 with both anisometropia and strabismus). The amplitudes and peak latencies of the main P_1 peak in the pattern-reversal VEP and of the motion-specific N_2 peak in the motion-onset VEP through the amblyopic eye were compared with those through the normal fellow eye. Regardless of the type of amblyopia, the amplitude of the pattern-reversal VEP for full-field stimulation was significantly smaller and its latency significantly longer through the amblyopic eye ($P < 0.001$). In contrast, neither the amplitudes nor the latencies of the N_2 motion-onset VEPs differed significantly between amblyopic and non-amblyopic eyes. For pattern-reversal VEPs through the amblyopic eyes, the extent to which amplitude was reduced and latency prolonged correlated well with the reduction of visual acuity, whereas the amplitudes and latencies of motion-onset VEPs did not vary with visual acuity. Even for stimuli restricted to the central visual field (5 or 2 deg diameter) or to the peripheral field (excluding the central 5 deg), motion-onset responses were indistinguishable through the two eyes, while pattern-reversal responses always differed significantly in amplitude. These results suggest that the source of motion-onset VEPs (probably an extrastriate motion-sensitive area) is less affected in amblyopia than that of pattern-reversal VEPs (probably the striate cortex). The motion pathway, presumably deriving mainly from the magnocellular layers of the lateral geniculate nucleus, may be relatively spared in amblyopia.

Visual evoked potentials Pattern reversal Motion onset Magnocellular pathway Strabismus Amblyopia Anisometropia Motion system Human

INTRODUCTION

Sudden reversal of the contrast of a pattern (counter-phase modulation) elicits a characteristic visual evoked potential (VEP), dominated by a positive component (P_1) with a peak latency of 100 msec or so, localized to a dipole source in the striate cortex (Maier, Dagnelie, Spekreijse & van Dijk, 1987). Spekreijse, Dagnelie, Maier and Regan (1985), who considered that the typical response to the onset of motion was a positive peak with a latency of about 120 msec, suggested that the pattern-reversal VEP might be a mixture of responses to the onset and offset of motion associated with the abrupt displacement of a pattern. However, Kuba and Kubová (1992), who studied responses evoked by moving stimuli of various velocities, argued that the P_1 peak is specifically related to pattern offset. This component occurs mainly for stimuli of high temporal frequency (the multiple of velocity and spatial frequency), causing blurring of pattern at the beginning of motion, and/or when the duration of movement is long and the inter-stimulus interval short (causing adaptation to the motion itself).

The main element of the VEP associated with the onset of steady linear motion appears to be a later negative component (N_2) with a peak latency of about 160–200 msec (Yokoyama, Matsunaga, Yonekura & Shinzato, 1979; Göpfert, Müller, Markwardt & Schlykowa, 1983; Kuba & Kubová, 1992; Bach & Ullrich, 1994; Kubová, Kuba, Spekreijse & Blakemore, 1995). A similar negative component, though usually of smaller amplitude, is often also seen in VEPs for pattern reversal.

The deficit in visual acuity that characterizes developmental amblyopia is associated with a reduction in amplitude and an increase in latency of pattern-reversal

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VEPs through the amblyopic eye (e.g. Arden & Barnard, 1979; Wanger & Persson, 1980; Mayeles & Mulholand, 1980; Sokol 1983, 1986; Levi & Manny, 1986; Odom, 1991). Thus the neural generator of pattern-reversal VEPs may be the substrate of visual acuity. It is generally thought that the detection of fine spatial detail (as well as chromatic vision) depends primarily on signals from so-called P cells in the parvocellular layers of the LGN, which, like the P retinal ganglion cells that provide their input, have relatively small receptive field centres, are usually chromatically coded and have low contrast sensitivity. It is conceivable, then, that neuronal mechanisms at some point in the parvocellular pathway are compromised in amblyopia.

On the other hand, the perception of image motion is more likely to depend on signals from M cells, in the magnocellular layers of the LGN, which have slightly larger receptive field centres, with higher sensitivity to contrast, and are not obviously chromatically selective (see DeYoe & Van Essen, 1988; Lennie, Trevarthen, Van Essen & Wässle; 1990; Zeki, 1990). In macaques, the magnocellular system projects, principally via layer 4b of the striate cortex, to subdivisions of area V2 and to area MT (or V5) in the superior temporal sulcus (Maunsell & Newsome, 1987; Newsome & Paré, 1988; Livingstone & Hubel, 1988). While both M and P systems undoubtedly contribute to both the dorsal and the ventral cortical processing streams (Merigan & Maunsell, 1993), it is generally accepted that areas of the dorsal stream, feeding into the parietal cortex, are selectively concerned with the analysis of image motion and are dominated by the M system (Maunsell & Newsome, 1987; Newsome & Paré, 1988).

Functional imaging of the human brain suggests that a region anterior and lateral to the calcarine sulcus, which may be homologous to area MT or V5 in the macaque cortex, is specifically activated by moving stimuli (Mora, Carman & Allman, 1989; Corbetta, Miezin, Dobmeyer, Shulman & Petersen, 1991; Zeki, Watson, Lueck, Friston, Kennard & Frackowiak, 1991; Watson, Myers, Frackowiak, Hajnal, Woods, Mazziotta, Shipp & Zeki, 1993). Motion-related VEP signals, as well as the small N_2 component often seen for pattern reversal, probably originate from this extrastriate motion area (Probst, Plendl, Paulus, Wist & Scherg, 1993; Spekreijse, Gilhuijs, Kubová & Van Dijk, in preparation).

Now there is evidence that some aspects of the perception of motion are relatively less impaired in amblyopia than the perception of fine spatial detail (e.g. Hess & Anderson, 1993; Hess, Howell & Kitchin, 1978; Hess, France & Tulanay-Keesey, 1981; Levi, Klein & Aitsebaomo, 1984; Rentschler, Hilz & Brettel, 1981). In a preliminary study, Kubová and Kuba (1992) reported that motion-onset VEPs did not differ between the amblyopic and normal fellow eyes of five adult amblyopes. Here we have extended those experiments by comparing pattern-reversal and motion-onset VEPs for defined classes of amblyopic children, correlating the results with the deficit in visual acuity and examining responses from different parts of the visual field, in an attempt to see whether the neural mechanism associated with the processing of motion is indeed relatively spared in amblyopia.

METHODS

Subjects

In the main experiments, VEPs for full-field stimulation were recorded from 30 amblyopic children (nine girls and 21 boys) of 6–14 yr of age. Snellen acuity was measured at 4 m (with refractive errors corrected) using conventional Landolt C charts (NAS–NRC Report, 1980). The acuity of the amblyopic eyes ranged from 20/50 to 20/200, while that of the fellow eye was always 20/20 (or slightly better).

Children of this relatively late age were chosen because evoked responses are generally more variable in younger children. In 14 cases the amblyopia was associated with anisometropia, in six with strabismus (always esotropic) and in 10 with both strabismus and anisometropia. All the children had a history of occlusion or CAM therapy (Campbell, Hess, Watson & Banks, 1978; Peregrin, Sverák, Kuba, Vít & Juran, 1987), which had been only partially successful in restoring visual acuity. In 18 children fixation of the amblyopic eye was central but unstable, while in 12 cases it was consistently parafoveal or peripheral.

In a further seven amblyopic children (four boys, three girls from 7 to 12 yr old; six anisometropic, one strabismic), responses were studied not only with full-field stimulation but also with stimuli restricted to the peripheral visual field and to the central 5 and 2 deg.

Recording and analysis

All recordings were performed in a sound-attenuated, electromagnetically shielded chamber with a background luminance of 1 cd/m^2. The subject was seated in a comfortable dental chair with a neck support to reduce muscle artefacts. A dark fixation point of 15 min arc diameter was placed in the centre of the stimulus field: the subjects were instructed not to follow the moving or reversing pattern with their eyes (and the absence of obvious tracking eye movements was verified occasionally by means of electro-oculography). Stimulation was always monocular, with optimal refraction: the other eye was patched. All measurements for each individual subject (left eye and right eye; pattern reversal and motion onset) were always completed in a single recording session without changing electrode placements.

For the bulk of the experiments, involving full-field stimulation, performed on 30 amblyopic children, the patterned stimuli (square-wave, black and white checkerboards with an element size of 35 min arc mean luminance 15 cd/m^2 and contrast 0.9) were back-projected via a mirror on to a 20 deg diameter circular field. Mirror movement was produced by an optical scanner (General Scanning Inc., U.S.A.) controlled by square-wave or ramp signals.

For pattern-reversal VEPs we used a reversal rate of 1 Hz (2 reversals/sec) and we carefully adjusted the amplitude of displacement to be equal to the width of a single check. The frequency response of the scanner was such that the nominal square-wave displacement was completed in 2 msec. Just as with pattern reversal generated by television techniques, the whole array appeared either to flicker or to undergo stepwise displacement in any one of the four principal directions.

For motion-onset VEPs, the checks moved horizontally rightwards at a velocity of 6 deg/sec for 200 msec periods, with interstimulus intervals of 1 sec duration, during which the pattern was stationary. This regime was selected to minimize motion adaptation but to keep the sessions to a tolerable length.

VEPs were recorded in the bipolar lead O_Z–C_Z and in three unipolar leads with the electrodes placed at O_Z and 5 cm to the right and left (these electrodes were designated O_R and O_L). Linked earlobes served as reference. After amplification (Tektronix AM 502) in the 0.1–100 Hz band, 100 epochs of 400 msec duration were averaged with a sampling rate of 500 Hz on a PDP-11/03 microcomputer or an IBM compatible 386 PC computer with a 12-bit A/D converter (Data Translation).

For a further seven children checkerboards with 40 min arc checks, of contrast and mean luminance identical to those in the main experiments, were generated on a computer monitor (ViewSonic 21; 100 frames/sec; total display size 30 × 40 deg) under computer control (IBM compatible 486 PC). In these experiments, responses to full-field stimulation were compared with those for stimuli restricted to the central 5 and 2 deg of the field, and with responses from the peripheral field alone (excluding the central 5 deg). In all conditions, the fixation point appeared in the middle of the display. For motion-onset stimulation using this television display, the pattern was displaced at a velocity of 5 deg/sec and the direction of displacement varied randomly from trial to trial (left, right, up or down). Otherwise the stimulus conditions were the same as for the experiments with projected stimuli.

RESULTS

Figure 1 shows representative pattern-reversal and motion-onset VEPs for stimulation of the normal eye of an amblyopic child, with the major peaks designated. For pattern-reversal VEPs we determined the latency of the first positive peak, P_1, and its amplitude [measured as $(N_1P_1 + P_1N_2)/2$] for the O_Z-C_Z lead.

For motion-onset VEPs we measured the latency and amplitude of the major negative peak, N_2 (the most distinct and constant peak of such VEPs). This motion-onset VEP [measured as $(P_1N_2 + N_2P_2)/2$] often differs in amplitude between the two sides of the brain (Kuba & Kubová, 1992), being clearly larger over the right hemisphere in about 50% of cases and over the left in about 30%. The parameters of the N_2 peak given below are always for the channel with the largest amplitude.

Figure 2 shows examples of pattern-reversal and motion-onset VEPs for stimulation of the normal and amblyopic eyes of six children, selected to be representative of the entire group. Although there was some variability between individuals in the overall amplitude and general form of the signals, especially the later components, the major early peaks described in Fig. 1 could always be distinguished. Comparison of responses through the two eyes shows that in every case the pattern-reversal (P_1) VEP was clearly reduced in amplitude through the amblyopic eye and it was usually somewhat delayed in latency, while the motion-onset (N_2) VEP did not differ consistently between the eyes. Note that a small positive peak at about 110–120 msec, probably equivalent to the pattern-offset P_1 component (Kubová *et al.*, 1995), is also discernible in the VEPs to motion onset through the normal eyes. The reduction or virtual absence of this peak for the amblyopic eye is presumably responsible for the broadening of the N_2 peak through that eye.

For the whole set of amblyopic children ($n = 30$), Table 1 gives mean values of latency and amplitude of pattern-reversal (P_1) and motion-onset (N_2) VEPs through non-amblyopic and amblyopic eyes, and average interocular differences calculated from *individual* measurements for each child.

While the pattern-reversal VEPs had significantly longer latencies and reduced amplitudes through the amblyopic eye ($P < 0.001$ for both), motion-onset VEPs were always very similar in both amplitude and latency through the two eyes of individual subjects. This was true for all three subgroups of patients, classified according to the origin of their amblyopia (i.e. anisometropia, strabismus and anisometropia combined with strabismus). These three subgroups could not be distinguished in any of the parameters estimated (Kruskal–Wallis non-parametric test).

Correlation of the deficits in pattern-reversal VEP with the loss of acuity

We were interested to know whether the deficits in pattern VEPs correlated with the depth of amblyopia. Even among the 11 children with relatively mild

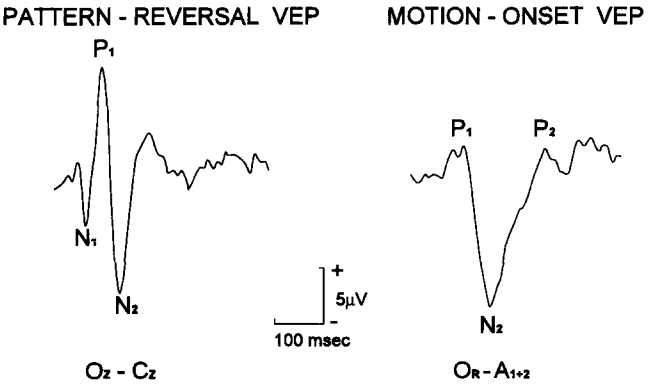

FIGURE 1. Typical examples of the pattern-reversal VEP (with its main positive peak, P_1) and the motion-onset VEP (with its dominant N_2 peak) for stimulation through the normal eye of an amblyopic child. The recording leads are indicated.

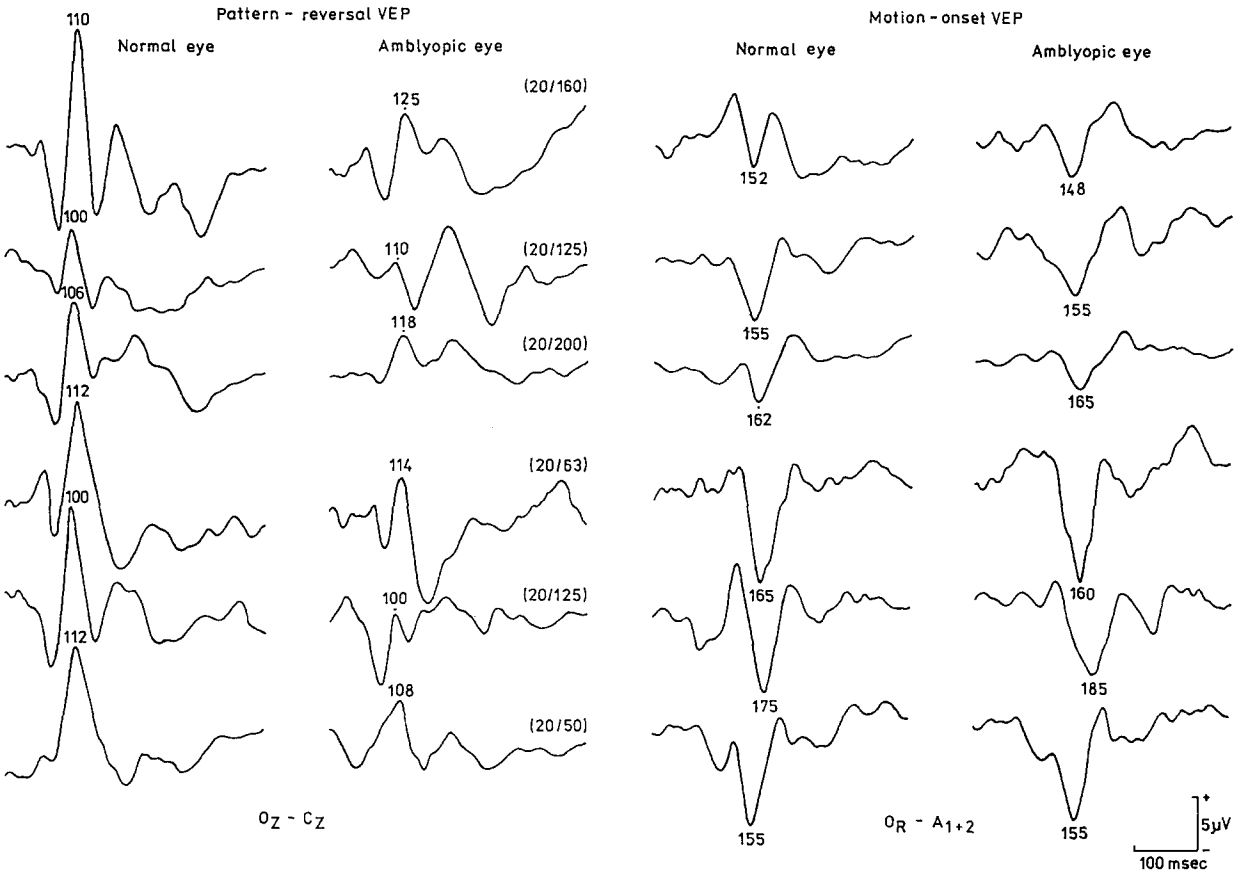

FIGURE 2. Pattern-reversal and motion-onset VEPs through each eye for six of the amblyopic children, selected to be representative of the variation in the general patterns of response among the entire group. Each row of recordings was taken from one child in a single recording session. Latencies in msec are indicated above the P_1 peaks of the pattern-reversal VEPs and below the N_2 peaks in the motion-onset responses. Pattern VEPs all show a sharp positivity (P_1) at about 110 msec. Its amplitude varies considerably from subject to subject but in every case it is considerably larger through the normal than through the amblyopic eye. The visual acuity of the amblyopic eye is indicated above the pattern VEP for each child. The amplitudes and the latencies of the N_2 peaks of the motion-onset VEPs also vary somewhat from child to child but are very similar through the two eyes in every individual case. Note that, for several of the children, the N_2 component appears broader through the amblyopic than the normal eye. This was the case in two-thirds of our subjects and it is probably due to the reduction in the preceding small positive peak, seen in the responses through the normal eyes, which may be equivalent to the P_1 pattern-offset component (Kubová *et al.*, 1995).

amblyopia (visual acuity of 20/50 or 20/63) the mean values of latency and amplitude of the pattern-reversal VEPs differed significantly between the two eyes ($P < 0.001$; Kruskall–Wallis test). Nevertheless, the abnormalities in the P_1 component in the pattern-reversal response were, on average, even more pronounced in the children with more severe amblyopia. Interestingly, no clear P_1 peak could be detected in motion-onset VEPs from those amblyopic eyes with particularly poor visual acuity (below 20/125).

TABLE 1. Mean latency and amplitude ($\pm$ 1 SD) of pattern-reversal and motion-onset VEPs for non-amblyopic and amblyopic eyes in all the children ($n = 30$)

	Non-amblyopic eye	Amblyopic eye	Interocular difference
Pattern-reversal VEPs			
Latency	105.5 $\pm$ 4.5 msec	117.7 $\pm$ 8.8 msec	12.2 $\pm$ 7.7 msec
Amplitude	17.9 $\pm$ 6.0 μV	10.9 $\pm$ 4.4 μV	6.9 $\pm$ 4.6 μV
Motion-onset VEPs			
Latency	157.7 $\pm$ 9.1 msec	158.2 $\pm$ 8.4 msec	7.2 $\pm$ 5.5 msec
Amplitude	8.2 $\pm$ 2.9 μV	7.3 $\pm$ 2.3 μV	1.8 $\pm$ 2.0 μV

Note that the interocular differences were calculated as the means of the differences in *individual* children. Since these differences were not always consistent in direction, especially for motion VEPs, these means are not equal to the differences between the pooled means for each set of eyes.

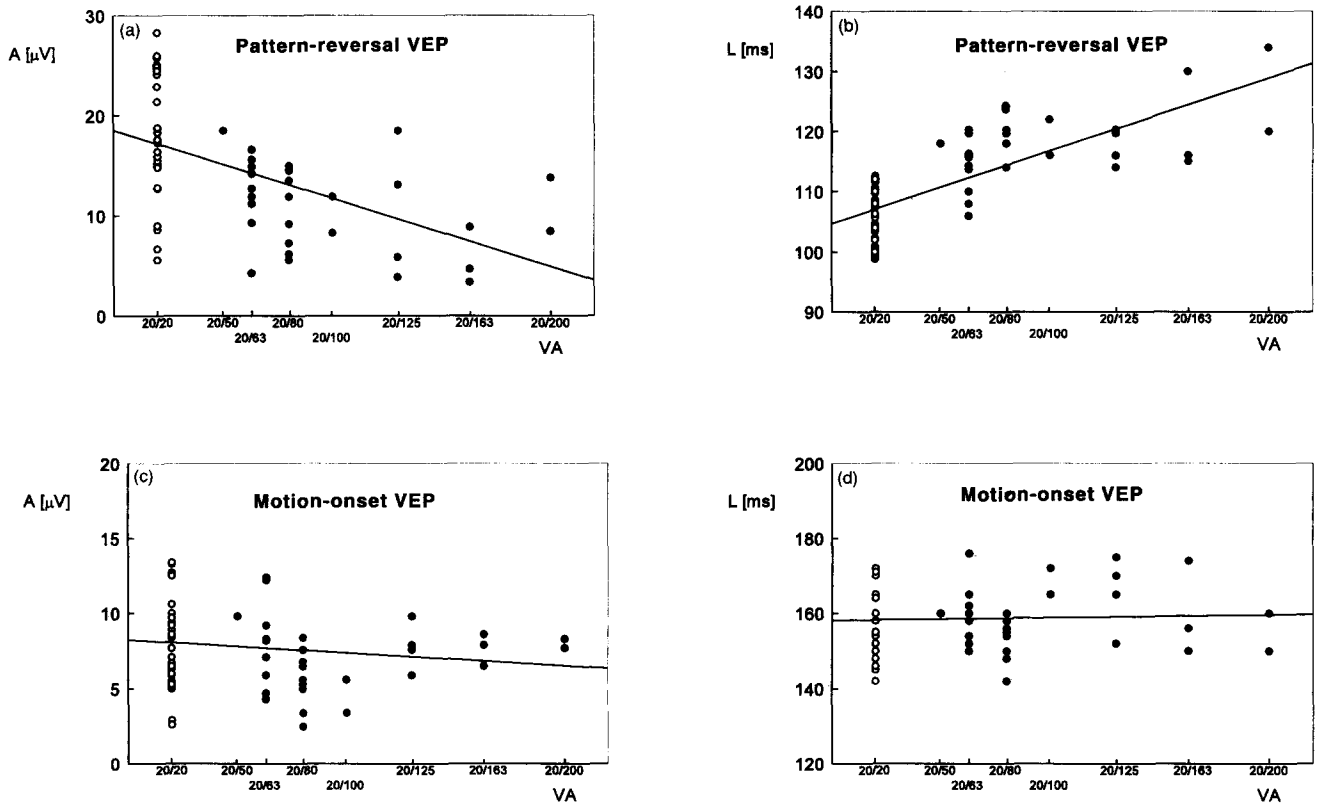

FIGURE 3. The graphs plot the amplitude, A (a, c) and latency, L (b, d) of pattern-reversal (a, b) and motion-onset VEPs (c, d), as a function of visual acuity (VA) (on a linear scale for the decimal value of acuity). Data from the normal eyes are plotted as open circles, above a visual acuity of 20/20, those from the amblyopic eyes as solid circles. Linear regression lines are plotted. Although there is considerable scatter of values at each acuity value, especially for amplitude, the amplitude of the pattern VEP clearly tends to decrease and its latency to increase with decreasing visual acuity ($P < 0.001$ for both), but there are no obvious changes in the motion VEP.

The scatter diagrams in Fig. 3 plot the amplitudes and latencies of pattern-reversal [Fig. 3(a, b)] and motion VEPs [Fig. 3(c, d)] as a function of visual acuity for all 30 children. The open circles represent results through the normal eyes, all of which had corrected visual acuity of 20/20 (or slightly better). The solid circles show data for the amblyopic eyes. Linear regression lines (calculated from decimal values of acuity) are shown. At each acuity level there is considerable variation from eye to eye in the absolute amplitude of signals and some scatter of latencies (cf. Fig. 2). However, despite this variability, the average amplitude of pattern VEPs clearly decreased and the latency increased progressively with decreasing visual acuity (correlation coefficients 0.57 and 0.65 respectively; $P < 0.001$ for both). These trends for pattern VEPs were evident for all three classes of amblyopes (anisometropic, strabismic and mixed). In contrast, neither the amplitude nor the latency of the N_2 motion-onset VEPs showed any obvious variation with visual acuity.

We also considered the results in relation to the pattern of fixation of the amblyopic eye. The amplitudes and latencies of pattern VEPs and also the latencies of motion VEPs did not differ between the amblyopic eyes with central fixation ($n = 18$) and those with eccentric fixation ($n = 12$). Curiously, motion-onset VEPs were, on average, slightly larger for amblyopic eyes with parafoveal or peripheral fixation than for those eyes with central fixation, and this difference just reached statistical significance ($P < 0.05$).

Responses from central and peripheral visual field

In seven additional amblyopic children (six anisometropic and one strabismic), whose visual acuity through their amblyopic eyes was in the range of 20/80 to 20/200, we recorded pattern VEPs and motion VEPs not only with full-field checkerboard stimulation (30 × 40 deg; generated on the television display) but also with the pattern limited to a circular central patch of 5 or 2 deg diameter, centred on the fixation point. In addition, responses were recorded for peripheral stimulation alone, produced by covering the central 5 deg diameter of the screen with a mask. In every condition the child was instructed to hold fixation on the point in the centre of the screen.

Typical results for one anisometropic subject are shown in Fig. 4. The amplitude of the P_1 pattern-reversal component through the normal eye was clearly dependent on the area of the field stimulated. The response appears to originate disproportionately from the macular visual field since occlusion of only the central 5 deg roughly halved its amplitude, while it remained clearly detectable, with about one-third of the full-field amplitude, even for stimulation of the central 2 deg alone. Under *all* conditions the response was

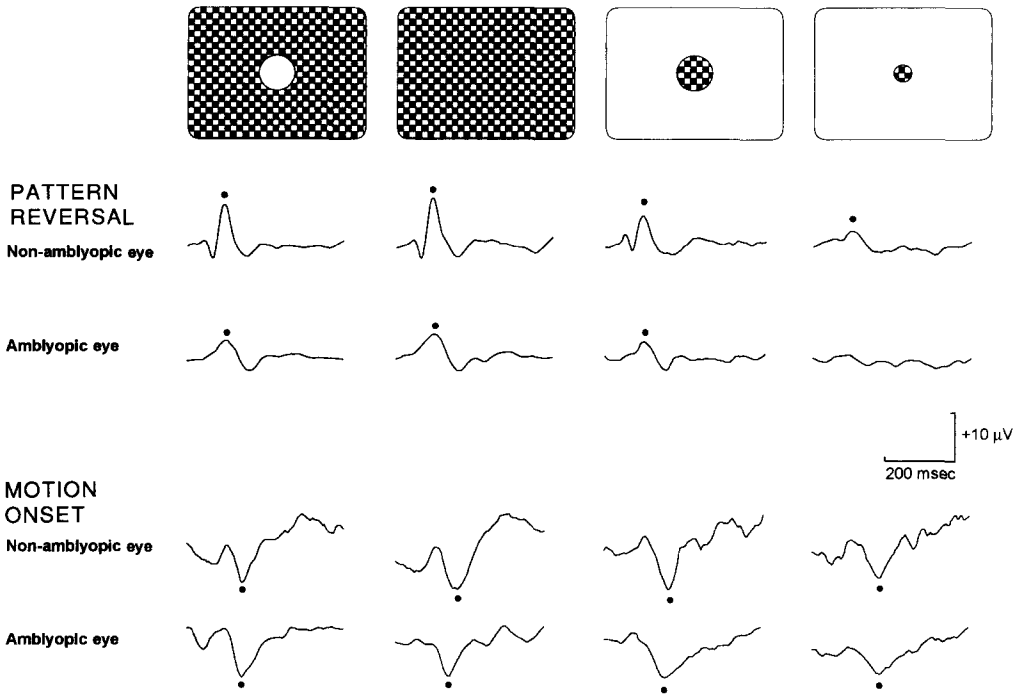

FIGURE 4. Typical examples of pattern-reversal and motion-onset VEPs, through the two eyes of one child, with stimulation restricted to various parts of the visual field. Visual acuity was 20/20 through the normal eye and 20/125 in the amblyopic eye. For these experiments the checkerboards were generated on a computer monitor of 30 × 40 deg (see Methods). The four stimulus conditions are illustrated schematically (not to scale) above the traces: peripheral stimulation alone, with the central 5 deg diameter occluded; full-field stimulation; central stimulation with a 5 deg diameter patch; foveal stimulation with a 2 deg patch. The P_1 peak in the pattern-reversal VEPs and the N_2 component in the motion-onset responses are indicated by small dots above or below the records. Note that the pattern response is consistently smaller through the amblyopic eye for all stimulus configurations. While covering the central field has no effect on the motion-onset response, stimulation of the central field alone produces a clear motion signal. Indeed even for foveal (2 deg diameter) stimulation there is a distinct N_2 component. Moreover, the motion response is indistinguishable in amplitude between the two eyes under all conditions.

consistently smaller (about half the amplitude) through the amblyopic eye compared with the normal.

The N_2 component of the response to motion onset was also detectable for all stimulus areas, though its behaviour across these stimulus conditions was quite different from that of the pattern VEP. Covering the central 5 deg of the stimulus had no effect on the N_2 amplitude. Although the N_2 component became smaller with decreasing field size, its decline in amplitude was less dramatic than for the P_1 pattern VEP. Most important, the similarity in amplitude through the two eyes of the N_2 component for motion onset with full-field stimulation was maintained at all field configurations. Even with a stimulus patch restricted to the central 2 deg, N_2 motion-onset responses were very similar through the two eyes (except for the broadening of the response through the amblyopic eye, presumed to be associated with attenuation of the initial small P_1 component; see above).

The results were similar for all seven children tested and the results are pooled in Fig. 5 in the form of histograms plotting the mean amplitude of P_1 and N_2 components in the pattern and motion VEPs respectively, through the normal and amblyopic eyes, under these four stimulus conditions. For every field configuration the pattern response was significantly smaller through the amblyopic eye than through the normal eye ($P < 0.001$), while there were no significant differences between the eyes in the amplitude of the motion-onset response, even for foveal stimulation alone.

DISCUSSION

Our findings fully confirm the results of numerous previous studies in showing that the amplitude of the major positive component of the pattern-reversal VEP is reduced and its latency increased through amblyopic eyes compared with normal eyes (e.g. Arden & Barnard, 1979; Wanger & Persson, 1980; Mayeles & Mulholand, 1980; Sokol, 1983, 1986; Levi & Manny, 1986; Odom, 1991). Furthermore, these abnormalities clearly correlate with the severity of amblyopia (Fig. 3).

Neural correlates of amblyopia

The acuity deficit in amblyopia could be due to neural under-sampling, neural "blurring" or positional uncertainty in the representation of the fine detail of the image in the visual pathway (e.g. Levi & Klein, 1986; Hess, Field & Watt, 1990). In monkeys reared with one eye closed (e.g. Baker, Grigg & Von Noorden, 1974; LeVay, Wiesel & Hubel, 1980; Swindale, Vital-Durand & Blakemore, 1981; see Blakemore, 1988) or even unilaterally defocused (Movshon, Eggers, Gizzi, Hendrickson, Kiorpes & Boothe, 1987), the ocular dominance of

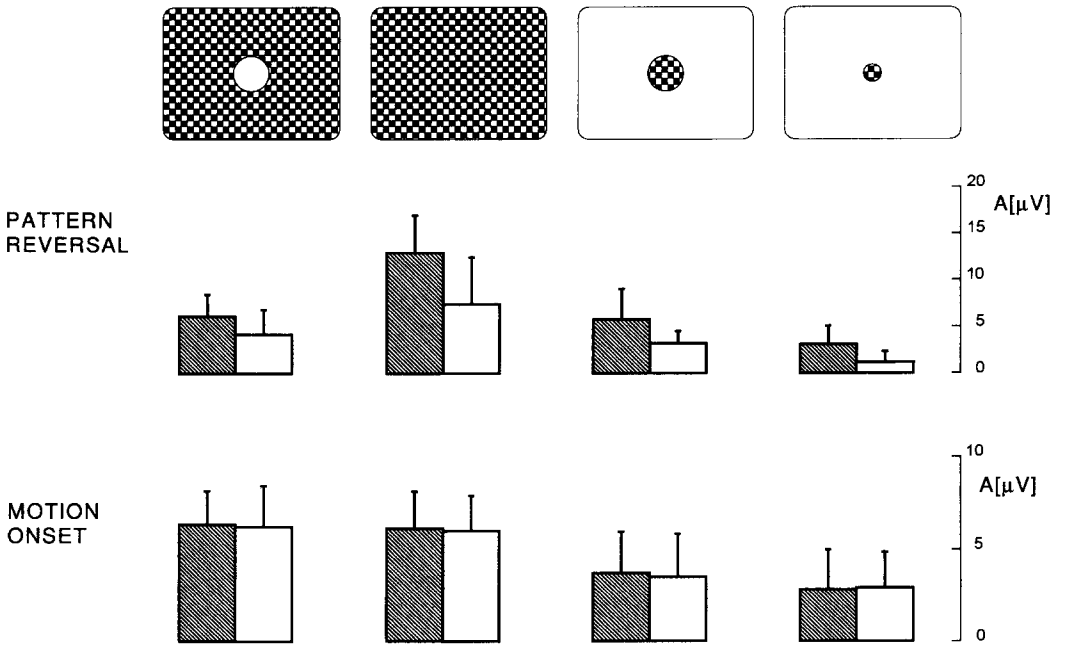

FIGURE 5. Pooled results for all seven children in which responses were measured with stimulation restricted to different parts of the visual field. The stimulus configurations are illustrated schematically as in Fig. 4. The histograms plot the mean (and SD) amplitude (A) through the non-amblyopic eyes (solid blocks) and the amblyopic eyes (open blocks), under these different stimulation conditions, for pattern-reversal responses and motion-onset signals. In every condition the amplitude of the pattern response differs significantly between the two eyes ($P < 0.001$) while there are no significant differences in the motion-onset responses through the two eyes with any stimulus configuration.

individual neurons in the striate cortex becomes biased in favour of the normal eye, with only a small proportion of cells responding through the amblyopic eye. Presumably such a gross loss of input could result in undersampling of the image, hence contributing to the letter-acuity deficit and the reduction in amplitude of the P_1 component of the pattern-reversal VEP, which almost certainly derives from the striate cortex (Maier *et al.*, 1987).

In addition to these dramatic changes in ocular dominance, the spatial characteristics of the receptive fields of individual neurons in the striate cortex are degraded through the amblyopic eye. In monkeys made amblyopic by deprivation of vision (Blakemore & Vital-Durand, 1984; Blakemore, 1990) or by unilateral defocus (Movshon *et al.*, 1987), striate cells driven through the amblyopic eye have lower spatial resolution and contrast sensitivity for grating stimuli than those driven through the normal eye. Such neural "blurring" at the level of the striate cortex could also presumably contribute to the decrease in amplitude of the pattern-reversal VEP, because neurons produce smaller responses for stimuli of any particular spatial frequency and contrast through the amblyopic than through the normal eye.

Foveal and peripheral-field contributions to pattern-reversal VEPs

It is well known that responses from the fovea, which is of course normally specialized for fine spatial resolution, contribute disproportionately to the pattern-reversal VEP (Blumhardt, Barrett, Halliday & Kriss, 1989). In the present study the P_1 component for full-field stimulation (through normal eyes) was reduced in amplitude by about 50% if either the central 5 deg was masked or the stimulus was restricted to the central 5 deg (Figs 4 and 5), which implies approximate additivity of pattern-reversal signals from central and peripheral field. However, this result shows that the amplitude was far from linearly related to the area of field stimulated. Spatial summation was non-linear even within the central 5 deg, since a reduction in field area by 84% (from 5 to 2 deg diameter) only halved the amplitude.

Most important for the present study, the deficit in the pattern response through the amblyopic eye was evident for all field sizes and configurations. Even for peripheral stimulation alone, with the central 5 deg covered, the P_1 component was significantly smaller through the amblyopic eye (Fig. 5). This is somewhat surprising in view of the finding that peripheral vision is often less dramatically affected in amblyopia than is central vision (Hess, 1978; Hess & Howell, 1978; Hess & Pointer, 1985). Hess, Campbell and Zimmern (1980) reported that vision through an amblyopic eye can be similar to that in a normal eye with a simulated central scotoma or with the luminance reduced sufficiently to compromise the fovea, but that strabismic and anisometropic amblyopes perform differently under reduced luminance. In strabismic amblyopia the anomaly of vision is predominantly central (within the central 5 deg), whereas anisometropic amblyopes have an equally severe abnormality for centrally or peripherally located stimuli. It is, then, important to note that of the seven children for whom responses from the peripheral field were studied, six were anisometropic. It would be interesting to repeat this

experiment in a cohort of strabismic amblyopes to see whether their deficits in the pattern response are restricted to the central field.

When the central 5 deg is masked, the amplitude of the P_1 pattern-reversal VEP through normal eyes is reduced to about the same extent as the mean difference between normal and amblyopic eyes for full-field stimulation (Fig. 5). This result, which is in agreement with findings of Levi and Manny (1982), might on its own be taken to imply that the central field of an amblyopic eye generates virtually no pattern component. However, definite pattern responses could be elicited with stimuli restricted to the central field of the amblyopic eye (at least with a field diameter of 5 deg), although consistently smaller in amplitude than through the normal eye.

Srebro (1984) reported pattern-reversal VEPs to be relatively less affected for eccentrically fixating than centrally fixing amblyopic eyes. However, in our amblyopes we saw no difference in the amplitudes of pattern-reversal VEPs between the groups exhibiting central and eccentric fixation with their amblyopic eyes. We have no ready explanation for this contradiction and can only suggest that there might have been differences between our group and Srebro's in the average angle of eccentric fixation in the sample.

Motion-onset VEPs are relatively unaffected in amblyopia

Our most interesting finding is that neither the amplitude nor the latency of the motion specific N_2 peak of motion-onset VEPs is obviously affected in amblyopia, regardless of the reduction in visual acuity, at least for the high contrast and relatively low spatial frequency of our stimuli. This was the case not only for full-field stimulation but also for patterns restricted to the periphery or centre alone (Figs 4 and 5).

Psychophysical studies have led to a confusing variety of interpretations about motion perception in amblyopia. Some suggest that the visibility of dynamic stimuli is selectively reduced (e.g. Schor & Levi, 1980) while others report that form and motion detection are often equally affected (e.g. Woods & Kulikowski, 1978; Steinman, Levi & McKee, 1987; Banton & Levi, 1991; Hess & Anderson, 1993). However, several reports suggest that motion processing is selectively spared in amblyopia (Hess *et al.*, 1978, 1981; Levi, Klein & Aitsebaomo, 1984; Rentschler *et al.*, 1981). Recently Hess and Anderson (1993) pointed out that some claims that motion sensitivity is differentially affected in amblyopia are based on the assumption that the motion system mediates the detection of "flicker" in contrast-reversing stimuli; but perceptual judgements of flicker vs pattern are not easy.

Unlike the pattern response, the N_2 component does not show even approximate additivity between different parts of the field. It is similar in amplitude for whole-field and peripheral stimulation (Fig. 4). This could be taken to indicate that it is generated only by the peripheral field, but stimulation restricted to the central field generate good motion-onset responses: indeed; they are less reduced in amplitude than are pattern responses (Fig. 5). Clearly the central fovea alone is capable of generating distinct motion-onset signals and, significantly, even these central field responses are indistinguishable in amplitude between normal and amblyopic eyes.

We have recently shown that, in normal observers, the motion VEP maintains virtually constant amplitude as contrast is reduced, down to $<2\%$ (Kubová *et al.*, 1995). This, together with the non-additivity seen in the present study, might all be interpreted to mean that the N_2 signal is normally saturated. Such saturation could render any small deficits in motion-onset signals associated with amblyopia impossible to detect. Further work will be needed to see whether there are conditions, perhaps of very low contrast or high spatial frequency, under which deficiencies in the motion response appear through amblyopic eyes. Certainly amblyopic vision is most severely compromised at high spatial frequencies and it would be very surprising if deficits in the N_2 component did not appear with moving stimuli close to the acuity or contrast sensitivity limits of the amblyopic eye. However the fact remains that pattern-reversal VEPs are consistently abnormal through the amblyopic eye, even for stimuli of low spatial frequency and high contrast that are clearly visible through that eye, whereas motion-onset signals are apparently unaffected. Perhaps we should be more surprised by the fact that the P_1 pattern-reversal component is always so clearly reduced in amplitude, even for visible stimuli, than by the fact that the motion VEP is not!

Moving patterns selectively activate an extrastriate area in the human cortex anterior and lateral to the calcarine fissure, which may be the equivalent of area MT (or V5) in the macaque (e.g. Mora *et al.*, 1989; Zeki *et al.*, 1991; Watson *et al.*, 1993). This motion area may well be the source of the negative component of the motion-onset VEP (Probst *et al.*, 1993). Thus our finding that the motion-specific N_2 component is not obviously affected in amblyopia suggests that the motion "pathway" may be relatively spared.

Perhaps conditions that cause amblyopia have less effect on the receptive field properties of cells in the magnocellular-dominated motion pathway than on those of the cells in V1, which are presumed to mediate resolution acuity. Movshon and Kiorpes (1992) have recently reported that early strabismus does cause a reduction in the binocularity of cells in area MT of the macaque but "does not seem to affect motion signals". More work is needed to determine whether monocular deprivation and defocus have any effect on the ocular dominance and the spatial properties of neurons in MT, in order to discover the extent to which the motion system is spared under conditions that lead to amblyopia.

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