
The human visual evoked potential was recorded during 9 hr. of monocular occlusion or deprivation of fine detail by anisometropia. Some decrement in the response from the occluded eye was evident, but the major result was a sustained increase in the response from the nonoccluded eye. The anisometropic condition produced no decrement for stimulation of the deprived eye, but again there was an increase in the response from the nondeprived eye. Under some conditions, the changes became apparent after only 6 hr. of deprivation. The data may be interpreted in terms of binocular competition or reciprocal inhibition at the level of the visual cortex or lateral geniculate nucleus.

The nature of binocular interaction, although intriguing as a fundamental problem of the organization of the visual system, is of major importance in the etiology of ophthalmological disorders involving abnormal or unequal stimulation of the two eyes. For example, abnormal stimulation occurs in strabismus and often leads to strabismic amblyopia. Unequal stimulation can be caused by unequal refraction (anisometropia), unequal magnification (aniseikonia), clouding of the ocular media, or other impairment of the image (anopia) or can occur as a secondary effect of organic pathological conditions in one eye.

The first point in the visual system where binocular interaction takes place is the lateral geniculate nucleus (LGN). This is arranged in layers of neurons which are excited only by monocular stimulation but which do show binocular inhibition between the monocular layers. A second point at which binocular interaction is present is at the synaptic input to cortical neurons.

Fig. 1. Changes in the VEP during 9 hr. of normal visual stimulation (A) and monocular deprivation (B). Each point represents the mean amplitude of the VEP during two experimental sessions. The responses for each eye were normalized to the mean of the two eyes at 0 hr. The control condition produced no change in the VEP amplitude throughout the day. Monocular deprivation resulted in a significant increase in the amplitude of the VEP evoked by stimulating the nondeprived eye after 2 hr. of deprivation (B, solid line, J. Z.) or after 6 hr. (B, solid line, M. K.). Stimulation of the deprived eye resulted in a decrease in response amplitude from the occluded eye at 6 hr. (B, dashed line, J. Z.) or 3 hr. (B dashed line, M. K.).

In humans, it is possible to investigate binocular interactions either in patients with naturally occurring pathologies or in normal observers under experimental conditions. Little is known about the psychophysics of binocular interaction in pathological conditions, but some interesting observations have been imparted by electrophysiological techniques. Several authors have found no consistent difference between the electoretinograms of normal and amblyopic eyes, suggesting that little change occurs at the retinal level.

At higher levels, however, binocular interactions have been reported consistently. In a study on strabismic amblyopia, Shipley observed that although the visual evoked potential (VEP) from stimulation of the amblyopic eye was generally reduced in comparison to that of the normal eye, steady diffuse illumination of the amblyopic eye markedly reduced the response from the normal eye. Illumination of the normal eye had little effect on the response from the amblyopic eye. One possible explanation offered for this result is reciprocal inhibition between the two monocular pathways. It is possible that the lack of inhibition...
of the amblyopic response\textsuperscript{10,11} occurs because there is maximum tonic inhibition from the normal eye pathway at all times and hence no change is seen with change in illumination. Shipley's result is corroborated by Tsutsumi et al.,\textsuperscript{12} who found that in a number of amblyopes the response to binocular pattern alternation was reduced in comparison with the response from the normal eye.

One type of binocular interaction is binocular rivalry, which can produce perceptual image suppression with a time course of seconds. Suppression of the monocular VEP to a flickering checkerboard by presentation of a high-contrast stationary checkerboard in the other eye can occur with a time course of milliseconds.\textsuperscript{13} Similar suppression is obtained when the static pattern in the other eye is of different spatial frequency from the pattern producing the monocular evoked potential.\textsuperscript{14} The time course with which this immediate suppression during binocular stimulation becomes transposed into a tonic suppression is not known.

An important study which has addressed this question is that of Zubek and Bross,\textsuperscript{15} who examined critical flicker-fusion frequency (CFF) of each eye during short-term monocular deprivation. They found that CFF in the nondeprived eye was reduced during the first 12 hr. of monocular deprivation and then enhanced following a night's rest (binocular deprivation). A control study on the deprived eye showed no change in CFF. This result can only be explained in terms of binocular interaction rather than direct effects of deprivation. However, it is difficult to evaluate their results, since the control study on the deprived eye was conducted on a different group of observers. Furthermore, the role of circadian rhythms and sleep vs. wakefulness in the two segments of deprivation is obscure.

We therefore decided to examine the effect of short-term monocular deprivation on the VEP in order to replicate the general result of the effects of monocular deprivation and to further understand the role of binocular interactions in the determination of visual response.

**Methods and materials.** VEP's were recorded from human observers asked to fixate a periodically changing target, of which two different types were used in different experiments. The stimulus target lay within a square subtending 10 degrees on a side, with a dark surround. In one of the stimulus patterns, the upper half-field contained a checkerboard pattern moving in counterphase, and the lower half-field a stationary checkerboard pattern. The checks in both half-fields subtended 23 minutes of visual angle. In the other stimulus configuration, the field contained a random array of dots which were shifted by 10 degrees to replace them with a new set of dots for every stimulus event. Both stimuli were alternated at a rate of 4 Hz. The mean luminance of the stimuli was 150 cd/m\textsuperscript{2} with a contrast of 90 percent. To generate the stimulus light from a tungsten ribbon-filament lamp collimated by achromatic lenses was passed through an appropriate photographic transparency, reflected from a mirror surface, and presented to the observer's eye in a well-well view. Voltage applied to a Brush pen motor produced a rotation of the mirror and consequent modulation of the checkerboard or change of position of the random dot pattern.

The observers steadied their heads with a bite-board, and the nonstimulated eye was covered by an eye patch during testing.

Conventional monopolar techniques were used for recording the VEP. The active electrode was placed 2.5 cm. above inion, with a reference on the right earlobe and a ground on the left earlobe. Electrode resistance was maintained at below 1K\textsuperscript{2} prior to each recording session. The active and reference electrodes were connected to the differential input of low-level preamplifiers (Tektronix type 122). The total gain of the amplifiers was 10\textsuperscript{2}, and signals were attenuated below 2 and above 50 Hz. The output was monitored on an oscilloscope to avoid muscle artifacts and fed to a computer of average transients. Averages of
198 responses constituted a trial, and responses were obtained in an alternating series of three trials for each eye. Each test lasted approximately 10 min., and tests were made at the start of the deprivation period and at 1½, 2, or 3 hr. intervals thereafter (as indicated in the datum points of the figures). Testing time was therefore small relative to the deprivation time and was presumed to produce a negligible alteration of the deprivation effect.

VEP was measured under three conditions: no deprivation (as a control for circadian rhythms), monocular occlusion for 9 hr., and monocular deprivation of detail vision by monocular removal of an observer's 4.50 D. contact lens for 9 hr. During deprivation the observers continued their normal functions, so that input to the nondeprived eye consisted of the normal range of stimulation.

The amplitude of the response was measured from P1 to N1 at fixed latencies (40 to 70 msec.) determined by inspection of the waveforms. The responses were normalized for each eye to the mean of the two eyes at 0 hr., since a baseline difference between the responses for the two eyes is not of interest in this experiment.

Results. In the control condition, the data for two observers, each tested on at least 2 separate days, showed little evidence of fluctuations over a 9 hr. period, although there was a nonsignificant tendency for the response to decline through the day (Fig. 1, A).

Against this steady baseline, we found large variations in VEP amplitude of both eyes during the monocular occlusion condition (Fig. 1, B). Observer J. Z. was tested with the same visual noise-alteration stimulus as used for the control runs. She showed a significant (p > 0.05) decrease in the response amplitude of the occluded eye beginning at about 6 hr. of deprivation (Fig. 1, B, dashed line). More surprising was the significant increase in response of the nonoccluded eye (Fig. 1, B, solid line), which was maximal by 2 hr. of deprivation.

The results were similar for Observer M. F. K., who was tested with the counterphase checkerboard stimulus. In this case, the occluded eye showed a significant decrement beginning at 3 hr. (Fig. 1, B, dashed line), and the nonoccluded eye (Fig. 1, B, solid line) showed a decrease which peaked at about 6 hr. of deprivation.

Selective monocular deprivation of form information also influenced the VEP amplitude, as shown in the third phase of our study. Here a condition of anisometropia was produced by removing one corrective lens in an observer with 4.50 D. myopia. The experiment was repeated on 3 separate days. There was no significant effect on the VEP for the deprived eye (Fig. 2, dashed line). The nondeprived eye (Fig. 2, solid line) showed an increase in response beginning at about 6 hr.

Discussion. These data provide preliminary evidence of a short-term effect of monocular deprivation on the human VEP. With as little as 6 hr. of deprivation, a marked difference arises in VEP amplitude in favor of the nondeprived eye. This difference is due mostly to an increase in response to stimulation of the nondeprived eye than to a decrease for the deprived eye. Therefore it does not depend on functional atrophy of the occluded pathway. Furthermore, the monocular deprivation need not be complete. Monocular reduction in high-spatial-frequency components of the visual image by anisometropia is sufficient. We favor an explanation based on binocular competition either at the level of the LGN or the visual cortex. Such an interaction could involve tonic mutual inhibition between ocular channels and therefore rebound disinhibition upon deprivation of one eye. Alternatively, the affected channel from the nondeprived eye might show increased efficiency in the absence of competitive input from the other eye. The latter mechanism has been proposed to account for much longer-term binocular imbalance following monocular deprivation in developing animals.

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