From the Departments of Ophthalmology and Pharmacology, College of Physicians and Surgeons, Columbia University, New York, N. Y. This investigation was supported by U.S. Public Health Service Research Grants EY 01977, EY 02861, and EY 00457. Submitted for publication May 5, 1980. Reprint requests: Dr. B. D. Srinivasan, Columbia University, 630 W. 168th St., New York, N. Y. 10032.

Key words: re-epithelialization, cornea, conjunctiva, inflammatory drugs in ocular conditions.

REFERENCES


An ever-changing retinal stimulation is necessary for the proper maintenance of vision. The reason that steady visual scenes do not fade away is because a variety of physiologic mechanisms such as those of eye and head movement exist to continually produce fluctuations in the local stimulation of the retinal receptors.¹ ² One of these mechanisms, that of blinking,³ has received little experimental attention. Blinks briefly interrupt the light falling on the retina and thus introduce transient stimulation. This report outlines a method for recording evoked potentials that are produced as a result of blinking and describes some of their salient properties.

Methods. Most aspects of the recording situation, except for those uniquely associated with blinking, were the same as those adopted previously to record the potentials that accompany saccadic eye movement.⁴ A Maxwellian view stimulator presented striped patterns of light to the subject's eye. The stimulus field subtended a visual angle of 20 degrees; the stripes subtended an angle of 1 degree and had a contrast of 99%. The maximum luminance of the bright areas was 1000 trolands. Central fixation was used. There were two recording channels. One registered the evoked potential, and the other the electro-oculogram (EOG). The latter was used to synchronize the computer with the subject's blinks.

The recording amplifiers were operated to have a "flat" response over the range of 0.2 to 55 Hz. Isolation amplifiers and "isofuses" were used in all leads to protect the subject. During amplification the physiologic potentials were recorded on magnetic tape and subsequently were played into a computer averaging system. The potentials were picked up with standard electroencephalographic electrodes of the silver cup variety. These were mounted just above and below the eye for the EOG and in the special way described below for the evoked potential.

In the analysis of the data the computer was programmed to average the response activity that followed consecutive eye blinks. To accomplish this, the EOG was used to obtain a synchronizing signal. The large blink potentials that appeared were sent to a trigger circuit, previously used to investigate saccadic eye movement.⁵ Its output pulse signaled the onset of a blink to the computer. The number of responses in each average depended on the frequency of blinking and was in the order of 100.

A problem that is encountered when recording these responses is that they are necessarily time-locked to the blinks that are responsible for them. Thus they may be confounded with motor artifacts


Visual activity is initiated whenever there is a change in the light falling on the retinal receptors. In the present experiment, visually evoked cortical potentials, elicited by the light transients that accompany blinking, were recorded with an electrode array that minimized artifact pickup. Although these evoked potentials were roughly similar to those obtained by more conventional recording procedures, specific waveform features were observed.
that are produced by movement of the eyes and lids. An electrode array, diagrammed in Fig. 1, A, was adopted to minimize the effects of such artifacts. It was designed to measure the signal under an active electrode with respect to the average of a ring of surrounding electrodes. It was based on the average reference described by Offner as developed by Perl and Casby to record localized potentials. They termed it a "Laplacian" electrode. In the present application the central electrode was placed on the scalp in the occipital region (Oz) where the response potentials were believed to arise most strongly. The potential spread laterally at reduced amplitude and as a result was smaller at the four peripheral electrodes (located 3 cm away) that acted as reference points. Hence, the evoked potential was recorded with high sensitivity. Potentials that arose some distance from the recording site, particularly eye movement artifacts, reached it with low amplitude. Furthermore they tended to be distributed so as to be canceled. Let us assume that the artifacts from the eyes spread in such a way that they appear with greatest amplitude at the outer electrodes (say, Ep1 and Ep2) that are nearest to the eyes, that they arrive with lower amplitude at the central electrode, and that they will reach the farthest electrodes (say Ep3 and Ep4) with still lower amplitude. Then, when the potentials of the peripheral electrodes are combined, they match the potential of the central electrode and no difference is recorded. Of course there is no general assurance that the potentials are distributed as assumed, and thus the value of the electrode array must be demonstrated experimentally.

**Results.** Responses obtained with the electrode array...
Fig. 2. Evoked potentials (left column) and accompanying EOGs (right column) obtained over a range of stimulus intensities. Subject J. A.

array are shown in Fig. 1, B, for three subjects. Prominent activity was present when the stimulus was present, whereas little could be seen when it was switched off. Clearly the records are relatively free from blink related artifacts. Fig. 2 illustrates the essential features of the response wave over a range of intensities. It is convenient to consider the response as having two sections. The first was an initial series of fluctuations that followed the blink onset after a short delay. Its most prominent feature was a positive peak that appeared at high luminances with a delay of about 85 msec. The second section appeared after the blink had ended. It had an initial positive wave that gave way to a large negative deflection peaking 225 msec after the onset of the blink. These latency values are approximate, however, and depend on the accuracy of triggering the computer at blink onsets, on blink duration, and on stimulus luminance.

The effect of stimulus intensity is clear. As intensity was reduced the amplitude of the components dropped off systematically, and latency increased over the same range.

Fig. 2 also shows the average waveforms of the blink potentials used as triggers. These recordings point to the genuineness of the response potentials, since the blink signals did not change with stimulus luminance in any systematic way whereas the response waveforms did. Furthermore there was no apparent relationship between the blink waveform and that of the responses.

The response waveforms of evoked potentials depend on a multiplicity of parameters, many of which are not yet well understood. Thus a variety of waveforms appear in the literature. In the present case the electrode array is crucial in determining the recorded waveform. Thus it becomes important to see whether the waveform accompanying blinking is similar to that produced by actual stimulus transients. To do this, recordings
Fig. 3. Comparison of evoked potentials elicited by blinks with those produced with shuttered decrements in retinal illumination (negative flashes). Subject J. A.

were made with a shutter in the optical path. The shutter produced negative flashes by interrupting the stimulus for 130 msec once every 3 sec. Recordings were made on magnetic tape so that the data samples could be averaged by triggering at the onset of the negative flashes and at the onset of blinks. The result is shown in Fig. 3. Considering the second section of each response, there is a striking similarity between the two columns. The prominent long latency negative peak seen in the earlier figures was present in both series of recordings. There is also, a decrease in amplitude and an increase in latency with lowered stimulus luminance. However, the early section of the responses did differ, particularly at high intensities. The positive peak, seen at high luminances with blinking, was substantially reduced with negative flash stimuli. Thus, although the responses were similar, it cannot be concluded that those produced by negative flashes were identical to those produced by blinking.

Discussion. The results of this experiment suggest that blinks coexist with other mechanisms such as head movements and the various forms of eye movement to produce changes in retinal stimulation when the eye is viewing a steady scene. These changes maintain the activity of the receptors by preventing them from becoming completely adapted to the stimulus, and they initiate visual response. Because blinks occur rather infrequently, however, their effect on vision must be small in relation to other inducing mechanisms. There are two possible reasons that the responses to negative flashes might differ from those of blinks. (1) The actual retinal stimulation generated by the two methods is not the same. (2) Blinking produces some specific change in the response system. Although the negative flashes were intended to approximate blinks, there were inevitable differences in the stimulation produced. Eye movement accompanies blinks, and it could displace the image just before the light is interrupted. Blinks come at an irregular rate and vary in their amplitude and duration. They do not in-
interrupt the light quite as abruptly as a mechanical shutter. There is reason to believe, however, that the differences in waveform, particularly in the first section of the responses, cannot be attributed only to stimulus factors. The differences seem too large. Furthermore it is now recognized that the role of blinks is not merely to periodically interrupt the light. Psychophysical experiments have demonstrated a transient increase in visual threshold that is time-locked to blinks. The importance of blinks and their relation to vision deserves further investigation.

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From the Department of Psychology, Northeastern University, Boston. This research was supported by grant EY 00589 from the National Eye Institute. Submitted for publication Aug. 20, 1980. Reprint requests: John C. Armington, Department of Psychology, Northeastern University, Boston, Mass. 02115.

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Amblyopic abnormality involves neural mechanisms concerned with movement processing. INGO RENTSCHLER, RUDOLF HILZ, AND HANS BRETEL.

In strabismic amblyopia the detection of apparent movement for counterphase gratings is considerably more impaired than the detection of pattern. No such anomaly is found for the detection of changes from a blank field when gradual or abrupt onsets or offsets of the temporal grating presentation are used. Similarly, normal peripheral vision is relatively poor in detecting alternation of spatial phase. It is concluded that the observed movement abnormality does not reflect a loss in sensitivity of transient mechanisms but rather a visual insensitivity to spatial phase. This would explain why perceptual distortions and low optotype acuity occur in amblyopes with normal contrast sensitivity.

The dramatic loss of visual function in amblyopia is conventionally characterized by reduced acuity and spatial contrast sensitivity. More specifically, it is assumed that high spatial frequencies in the contrast sensitivity function would be most affected by the visual anomaly. This acuity interpretation of amblyopia meets a difficulty: perceptual distortions of suprathreshold grating stimuli and strongly reduced acuity for single optotypes have been reported from amblyopes with normal contrast sensitivity. In this study we provide evidence that spatiotemporal aspects of amblyopic vision may account for the discrepancy.

Two distinct thresholds may be found for a temporally modulated grating pattern. At one contrast the spatial structure of the pattern is recognized and at another contrast flicker or movement just becomes visible. This psychophysical dichotomy possibly reflects the activity of two neural subsystems. Indeed, as Enroth-Cugell and Robson have shown, there is neurophysiologic evidence for the existence of parallel X- (sustained) and Y- (transient) subsystems in the visual system. Compared with Y-cells, X-cells generally have smaller receptive fields and are more sensitive to stationary stimuli, whereas they respond less strongly to quickly moving targets.

The authors of recent studies agree that both flicker (or movement) and pattern detection are similarly affected in amblyopic vision. In the presence of sharp temporal transients, however,