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## Enhancement of Flicker by Lateral Inhibition


Abstract. *Sinusoidal modulation of illumination on the compound eye of the horseshoe crab,* Limulus, *produces a corresponding variation in the rate of discharge of optic nerve impulses. Increasing the area of illumination decreases the variation at low frequencies of modulation, but unexpectedly enhances—or "amplifies"—the variation at the intermediate frequencies to which the eye is most sensitive. Both effects must result from inhibition since it is the only significant lateral influence in this eye.*


The appearance of flicker in a light that varies periodically depends upon several variables (*1*). For example, whether a flicker will be observed in a light with sinusoidally varying luminance depends on both the amplitude and frequency of the variation. Results typical of those obtained by DeLange in 1957 from a human observer viewing a small sinusoidally modulated 2-deg field of white light are represented by the solid curve in Fig. 1A. Increase in frequency, up to about 10 cycle/sec, decreases the percentage of modulation about the mean luminance required for the observer to see flicker. At this point the modulation to reach threshold is minimal; that is, flicker sensitivity is maximal. With additional increases in frequency, the percentage of modulation required to reach threshold increases rapidly, and at some point between 50 to 75 cycle/sec even a light modulated 100 percent does not appear to flicker.

The size of the retinal area stimulated has a marked effect on the threshold of flicker. Measurements made by Kelly in 1960, using a large, 85-deg field, are represented by the dashed curve in Fig. 1A. The dash-dot curve, extending from 1 to 10 cycle/sec, represents some results obtained by Thomas and Kendall in 1962 in an experiment in which a sinusoidal modulation was applied to the illumination of the entire room in which the subject was seated.

The increase, at low frequencies, in the percentage of modulation required to reach threshold that results from an increase in the area of the stimulus has been attributed to the effects of lateral inhibition (*1*). In order to test this hypothesis by direct electrophysiological observations we have carried out some comparable experiments with sinusoidal stimuli (*2*) on the compound eye of the horseshoe crab, *Limulus,* in which lateral influences are predominantly inhibitory (*3*).

Modulation of the intensity of light shining on an ommatidium of the *Limulus* eye causes a modulation in the rate of discharge of impulses in the optic nerve fiber from that ommatidium. The amplitude of this modulation of the discharge may be taken as a measure of the "flicker response" of the receptor. Figure 1B shows that this response varies in a characteristic manner with frequency of modulation, and that it is affected by varying the area of the eye illuminated by the modulated light.

In order to obtain the results illustrated in Fig. 1B, a *Limulus* eye was excised and mounted in a moist chamber. A single active optic nerve fiber, arising from an ommatidium near the center of the eye, was dissected from the optic nerve and placed on wick electrodes in the input circuit of an amplifier. A spot of light was centered on the ommatidium from which the nerve fiber arose. The mean intensity of this spot was set by neutral density wedges inserted in the beam. The intensity was then varied sinusoidally, with a constant modulation about this mean, by a rotating polarizer and a fixed analyzer. (Other experiments showed that the polarization itself played no role in the effects reported here.)

The modulated light was turned on for 20 seconds every 3 minutes. The discharge of impluses by the single optic nerve fiber was recorded on line by a small digital computer (*4*). In order to avoid the transient response at the onset of the illumination, only the final 15 seconds of each record were processed. The sum of a constant plus a sine was fitted, by the method of least squares, to the reciprocals of the intervals between successive impulses. In this way average discharge rate (impulses per second) and amplitude and phase of the response modulation were determined (*5*).

The results for a small field (a spot of light about 0.25 mm in diameter which is slightly larger than the facet of one ommatidium) are represented by the solid curve in Fig. 1B. The results that were obtained with a large field (a spot about 1.5 mm in diameter that covered about 20 ommatidia) are represented by the dashed curve. In Fig. 1B *modulation* refers to the various peak-to-peak amplitudes of the neural flicker responses to stimuli of constant amplitude; in Fig. 1A *modulation* refers to the various amplitudes of the stimuli required to produce a constant response—that is, to reach the observer's threshold of flicker. Note that the scale of frequency in Fig. 1A is three times that in Fig. 1B.

Increasing the area of illumination produced two major effects. (i) There was the expected decrease in the amplitude of the flicker response to low-frequency modulation of the light. (ii) At its maximum, the dashed curve in Fig. 1B actually exceeds the maximum of the solid curve. This enhancement or "amplification" of the amplitude of the flicker response to intermediate modulation frequencies was unexpected. Both effects are the result of lateral inhibition.

Previous experiments on single ommatidia have shown that the transfer functions relating intensity of illumination to generator potential (*6*) and intensity of illumination or of generator potential to frequency of discharge of impulses (*5, 7*) have characteristic shapes. In brief, there is some frequency of modulation of the light to which the ommatidium is most sensitive. Typically, there is a span of lower frequencies over which the visual sensitivity is somewhat reduced, and when the optimal frequency is exceeded, there is a very pronounced high-frequency cut-off. In general, the crest of the flicker response leads the crest of the modulation of the stimulus at frequencies below the optimum. As the frequency of the modulation of the stimulus increases, the phase lead turns into a phase lag, generally reversing somewhere near the optimum. Thus, the photoreceptor and its axon are a "tuned" system which responds maximally to frequencies of modulation of the illumination of about 3 cycle/sec.

In this experiment, a further depression of the low-frequency portion of the transfer function resulted from increasing the area of illumination. Lateral inhibition must account for this effect, because the only significant neural effect of increasing the area of illumination is to increase the amount of inhibition exerted on the ommatidium under observation. Lateral inhibition must therefore also account for the unexpected amplification.

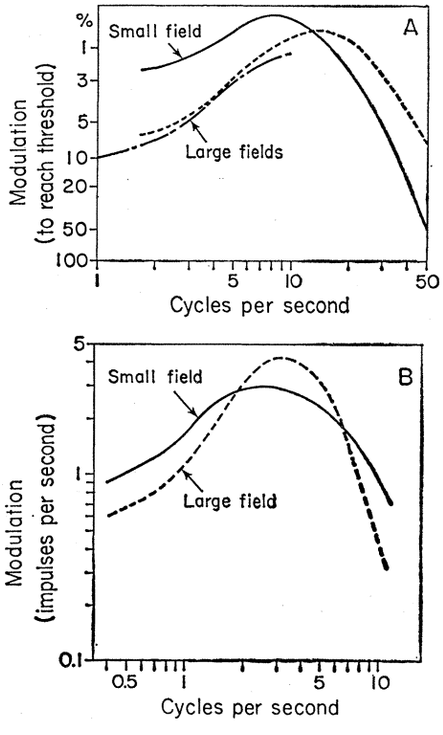


Fig. 1. Modulation sensitivity curves showing effects of size of retinal area stimulated. (A) Psychophysical measurements on human subjects [redrawn after Kelly (*1*)]. (B) Electrophysiological measurements on the compound eye of the horseshoe crab, *Limulus*.

This amplification appears to result mainly from a substantial delay between the generation of an impulse in one ommatidium and the production of its inhibitory effect in a neighboring ommatidium. Because this delay is about 150 msec (*8*), the opposed excitatory and inhibitory influences are about ½ cycle out of phase at stimulus frequencies of about 3 cycle/sec (period of 333 msec). The transfer function for the large field passes through its maximum at the frequency at which the opposed excitatory and inhibitory influences are most out of phase, because that is the frequency at which the greatest inhibitory influence coincides with the smallest excitatory influence. This general conclusion is borne out by extensive theoretical calculations which will be reported elsewhere (*9*). Whether a similar amplification of the maximal flicker sensitivity occurs in the human visual system has yet to be determined.

"Amplification" by lateral inhibition is not necessarily restricted to the responses to stimuli that vary in time. Responses to variations in the spatial distribution of stimuli may be similarly affected. It has been known, since Mach's work in 1865 (*10*), that inhibitory interaction can produce maxima and minima in the neural response where there are no corresponding maxima and minima in the spatial distribution of illumination—only steps or flections. Thus inhibition accentuates the neural responses to certain features of the stimulus pattern. It has been generally assumed, however, that the peak-to-peak distance between the maxima and minima, such as those produced by a step, cannot exceed the step that would appear in the response without inhibition. But, according to theory, lateral inhibition can amplify the peak-to-peak distance above that found in the uninhibited response. These spatial amplification effects, resulting from particular spatial distributions of the inhibitory influence, have been demonstrated in calculations by von Békésy (*11*) for a sinusoidal distribution of illumination and by Barlow and Quarles (*12*) for a step pattern of illumination. A more general theoretical treatment by Ratliff, Knight, and Graham is in preparation (*9*). None of these spatial amplification effects have been demonstrated experimentally as yet.

In the experiment reported here, the amplification of the variations in the response by lateral inhibition is accomplished at the expense of a reduction in the mean level of response. The same is also true in all the theoretical calculations mentioned earlier for either temporal or spatial modulation. Lateral inhibition provides a mechanism for enhancing significant variations in both spatial and temporal patterns of illumination.


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