A Nonlinearity in the
Inhibitory Interactions in the
Lateral Eye of *Limulus*

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ABSTRACT Receptor units in the eye of the horseshoe crab are more sensitive
to lateral inhibition at some levels of excitation than they are at others. As a re-
result, the steady-state inhibition of the response of a given unit is not directly
proportional to the response levels of neighboring units. This effect may be
represented by the introduction of a nonlinearity in the Hartline-Ratliff system
of equations. The nonlinear inhibitory effect appears to increase the operating
range of the receptor units.

INTRODUCTION

The processing of visual information begins in the retina. Nerve cells integrate
excitatory and inhibitory inputs and interact with other neurons in the retina
to shape the signals that pass on to the higher visual centers. The mechanisms
underlying these events have been described in detail for the lateral eye of
*Limulus* by Hartline, Ratliff, and their coworkers (for a review, see Hartline
and Ratliff, 1972). In brief, they found that the interactions among receptor
units (ommatidia) in the *Limulus* eye are predominantly inhibitory and obey
simple linear relationships. For two interacting receptors, the inhibition on
one is linearly related to the concurrent activity of the other (Eq. 1 in Table I).
For *n* interacting receptors, the activity of each unit is given by a set of linear
equations (Eq. 2). Hartline and Ratliff derived these relationships from direct
physiological measurements and found them to be valid within the range of
their experiments.

We further investigated the lateral interactions in the *Limulus* eye using both
antidromic and light-evoked inhibition. In effect, our work extends the range
of the original experiments by Hartline et al. (1956). Our interpretation of the
results differs significantly from theirs. Whereas they concluded that the
amount of inhibition exerted on a receptor was determined only by the ac-
TABLE I

STEADY-STATE EQUATIONS FOR INHIBITORY INTERACTIONS

Hartline-Ratliff formulation:

\[
\begin{align*}
    r_1 &= [c_1 - k_{11}(r_2 - r_{12})^+]_+ \\
    r_2 &= [c_2 - k_{21}(r_1 - r_{21})^+]_+ \\
    r_p &= [r_p - \sum_{j \neq p} k_{p j}(r_j - r_{p j})^+]_+, \quad p = 1, \cdots, n.
\end{align*}
\]  

(1)

Modified Hartline-Ratliff formulation:

\[
\begin{align*}
    r_p &= [r_p - (1 + a e_p) a k''_j(r_j - r''_j)^+]_+, \quad p = 1, \cdots, n
\end{align*}
\]  

(2)

Nonnegativity restrictions for Eqs. 1, 2, and 3:

- \(e_p, k_{p j}, k''_{p j}, r''_{p j}, \) and \(a\) are non-negative
- Define \(\alpha_p = \begin{cases} \alpha & \text{if } \alpha \geq 0 \\ 0 & \text{if } \alpha < 0 \end{cases}\)
- Then \(r_p \geq 0\).

The response \(r_p\) in impulses per second of a particular ommatidium \(p\) is equal to its uninhibited response \(e_p\) minus the sum of the inhibitory influences exerted on it by the other \(j\) ommatidia. The term \(r_j - r''_j\) represents the amount by which the response of the \(j\)th ommatidium exceeds the threshold \(r''_j\) of its inhibitory effect on the \(p\)th unit, and \(k_{p j}\) is the inhibitory coefficient for the action of \(j\) on \(p\). Eq. 1 gives the case for two interacting ommatidia as indicated by the subscripts. Eq. 2 is the generalized formulation proposed by Hartline and Ratliff (1958), and Eq. 3 is the modified version (Lange, 1965). The constant \(a\) can be determined experimentally as described in this paper.

The amount of inhibition is also influenced by the level of excitation of the receptor itself. Apparently, the receptor's excitatory level \((e_p\) in Eq. 2) governs to some extent its sensitivity to lateral inhibitory inputs. As will be seen below, this effect constitutes an essential nonlinearity in the resulting formulation (Eq. 3).

It is the purpose of this paper to describe quantitatively the nonlinear effect in the inhibitory interactions in the Limulus eye, and to show that the Hartline-Ratliff equation (2) can be modified to include the nonlinearity. A following paper will show that computations with the modified equation describe accurately the Mach-band response recorded from the eye (Barlow and Quarles, in preparation).

METHODS AND RESULTS

This study of lateral inhibition in the excised eye of Limulus is based on the measurement of the frequency of the steady-state impulse discharge from single receptor units (termed test units or test receptors). Two methods were used to inhibit the activity of the test receptors: illumination of neighboring retinal regions and antidromic electrical stimulation of the optic nerve trunk. Three types of experiments were performed.
Experiment I

This experiment investigated the inhibition exerted on a test receptor by a small cluster of neighboring units over a wide range of light intensities on the test unit. The small cluster of receptors (usually four in number) provided levels of inhibition that could be measured with precision. The size of the cluster was otherwise arbitrary. Both the test unit and the cluster were illuminated via fiber optics (Barlow, 1967, 1969). Techniques for excising the Limulus eye and for recording from its optic nerve fibers follow in general those developed by Hartline and his colleagues (Hartline et al., 1956; Hartline and Ratliff, 1957).

In a typical experiment, the response of the test unit to a 10-s light stimulus was recorded, while the rest of the eye remained in darkness. 2 min later the stimulus to the unit was repeated with a concurrent light stimulus delivered to the small cluster of neighboring units. The response of only one of the four units within the cluster was recorded, on the assumption that all four responded alike (Barlow, 1969). Runs with and without inhibition were alternated and sets of runs were made with different intensities of illumination on the test unit and on the cluster. The light intensity incident on the test unit at the cornea was varied from $10^7$ to $10^{12}$ photons/s in the wavelength range of 400-630 nm. Inhibited and uninhibited response rates were recorded from the test unit during the last 5 s of the 10-s light stimulus after all transient effects had died out.

The raw data in this and in the following two experiments consisted of many trains of nerve impulses recorded from one or more optic nerve fibers. A computer (CDC, 160A or DEC, Linc-8), a programmed timer (Milkman and Schoenfeld, 1966), and associated equipment (Schoenfeld, 1964; Kletsky, 1971) were integrated to control and monitor an experiment, and to collect, preserve, and process the data. For a detailed description of these methods, see Lange (1965), and Lange et al. (1966).

In Fig. 1 A the inhibition exerted by a small cluster of ommatidia on a single test unit is plotted on the ordinate as a function of the uninhibited firing rate of the test unit ($e_p$) plotted on the abscissa. The inhibitory effects were measured over a wide range of light intensities on the test unit and for four levels of illumination on the cluster (each level indicated by a different symbol). For each level of illumination on the cluster in this particular experiment, the amount of inhibition exerted on the test unit was relatively small at low firing rates of the test unit (2–20 impulses/s), large at intermediate rates (20–40 impulses/s), and then small again at high rates (40–60 impulses/s). Other experiments of this type gave similar results for uninhibited rates of up to about 30 impulses/s. However, different results were sometimes observed for rates greater than 30 impulses/s. For example, in several experiments the inhibition exerted on the test unit continued to increase beyond 30 impulses/s, and in other experiments the inhibition on the test unit remained constant above 30 impulses/s. Some of the data from this experiment are summarized in Fig. 4 and discussed further in the associated text.

The significant result of this experiment is that the reduction of the frequency of discharge from the test receptor, caused by a constant inhibitory input, was dependent on its level of excitation ($e_p$). If the inhibitory effects on the unit were not dependent on its level of excitation as reported by Hartline et al. (1956), then the individual sets of points in Fig. 1 A would describe straight lines with slopes of zero. The measurements by Hartline et al. were made for small values of $e_p$ (up to 14 impulses/s). Apparently for larger values of $e_p$ (up to 60 ips), the inhibitory interactions do not obey the Hartline-Ratliff formulation where the inhibition on the $p$th unit (term on extreme right in Eq. 2) is independent of $e_p$. Replotting the data for several values of $e_p$ (Fig. 1 B) gives
Figure 1. The dependence of inhibition on the excitatory level of an ommatidium. A (from Barlow, 1967) gives the amount of inhibition of the test unit (decrease in frequency of discharge) on the ordinate as a function of the excitatory level of the test unit (uninhibited firing rate) on the abscissa, for four different intensities of inhibitory illumination. The table indicates the response levels of the cluster of inhibiting ommatidia which was located about 0.7 mm (three receptor diameters) away from the test unit. The dashed line indicates the uninhibited firing frequency and therefore represents the "maximum decrease possible." B replots data from A for three levels of excitation of the test ommatidium. The magnitude of the inhibitory effect exerted on the test unit is plotted as a function of the concurrent activity of the source of inhibition. The lines were fitted by eye. The slopes of the lines give the values of the inhibitory coefficients, \( k_{ij} \), Eq. 2, for the various levels of excitation as indicated by \( \epsilon_p \) (impulses per second). The fact that the slope changes as \( \epsilon_p \) is changed indicates that the generalized Hartline-Ratliff equation (2) does not apply.
linear relationships similar to those reported by Hartline and Ratliff (1957). This indicates that Eq. 2 provides an adequate description of the inhibitory interactions when \( \epsilon_p \) is held constant. This result is further supported by Experiments II and III.

**Experiment II**

Inhibitory influences on a single receptor were again studied over a wide range of incident light intensities; however, in this experiment the effects were maximized by all receptors in the eye acting together. The optic nerve discharge of just a single receptor, the test receptor, was recorded. A mask with an aperture 300 \( \mu \)m in diameter was placed in contact with the cornea to restrict the incident illumination to the test unit. A flat Teflon diffusing screen was placed directly behind the mask and light from a tungsten filament source was projected on the screen. Under these conditions, steady-state uninhibited responses \( \epsilon_p \) to 10-s light stimuli of various intensities were recorded from the test unit. The mask was then removed and the experiments were repeated for the same range of light intensities \( (10^2-10^{11} \text{ photons/s}) \) incident on the single unit at the cornea between 400 and 650 nm). Sufficient time elapsed between runs to allow the sensitivity of the test unit to return to its dark-adapted state. With the mask removed, the diffused light beam illuminated with equal intensity all but the most peripherally located ommatidia. We therefore assumed that all the receptors, including the test receptor, responded at the same rate \( \epsilon_p \). This assumption seems reasonable since, in control experiments, it was found that equal light intensities evoked nearly equal firing rates from a number of ommatidia in a given eye.

Fig. 2 gives the discharge rates recorded from a single receptor unit for several intensities of illumination on the unit \( \epsilon_p \) and on the whole eye \( \epsilon_p \). As explained above, when the eye is uniformly illuminated all receptors including the test unit respond at

![Figure 2](image-url)

**Figure 2.** Intensity characteristics for the steady-state response from a single ommatidium with and without inhibition. The unit's response is plotted on the ordinate as a function of the log of the relative light intensity plotted on the abscissa. Responses with inhibition \( \epsilon_p \) were recorded from the test unit with the whole eye uniformly illuminated. Responses without inhibition \( \epsilon_p \) were recorded with a small aperture placed on the cornea to restrict illumination to the test unit. At log I = 0 approximately \( 10^{11} \) quanta/s are incident on a single ommatidium at the cornea from 400 to 650 nm.
nearly the same rate \( (r_p) \). Assuming that Eq. 2 describes adequately the inhibitory interactions for such stimulus conditions (as indicated by the results of Experiment I), then \( r_j \) will equal \( r_p \) for all values of \( j \) and Eq. 2 becomes

\[
r_p = e_p - \sum k_{pj}(r_p - r_{pj}).
\]

(4)

The summation limits and nonnegativity restrictions are the same as for Eq. 2. The value of \( \Sigma k_{pj} \) measures the efficacy of the inhibitory effect exerted on the test unit. To facilitate the solution of Eq. 4 for \( \Sigma k_{pj} \), we assumed that the thresholds \( (r_{pj}) \) were zero, giving

\[
\Sigma k_{pj} = \frac{e_p - 1}{r_p}.
\]

(5)

This assumption appears reasonable, since in our experiments all pertinent thresholds were exceeded. Values of \( \Sigma k_{pj} \) were computed from Eq. 5 for various levels of \( e_p \).

Nine experiments of this type were performed, each with a different eye. The common finding was that the value of \( \Sigma k_{pj} \) depended on the level \( e_p \). However, for a given level of \( e_p \) the absolute value of \( \Sigma k_{pj} \) may differ for units in different eyes. For example, in the experiment illustrated in Fig. 2, \( \Sigma k_{pj} \) was 1.5 for \( e_p \) equal to 25 impulses/s. For the same level of excitation, values of \( \Sigma k_{pj} \) in other preparations ranged from 0.4 to 2.3. Such variability is not usually found for units within the same eye. For the purpose of comparison, the data from each of the nine experiments were normalized to the value of 1.0 at 27 impulses/s. The results are summarized in Fig. 4.

**Experiment III**

Inhibition in the *Limulus* eye can also be produced by antidromic volleys in the optic nerve (Tomita, 1958). The preparation was similar to that described above in that the eye was excised and a single active nerve fiber was isolated by dissection. The receptor from which the nerve fiber arose was taken as the test unit. It was illuminated by a narrow beam of light focused on its corneal facet. Inhibition of the test unit was produced by stimulating the remaining portion of the optic nerve with repetitive current shock to generate volleys of antidromic impulses. The experimental methods employed here have been described fully elsewhere (Lange, 1965 and Lange et al., 1966). Antidromic inhibition destroys the mutuality of the retinal interactions and thereby allows a precise control of the inhibition exerted on the test unit. The response rates of all receptors except the test receptor are equal to the rate of antidromic current stimulation. In terms of Eq. 2, \( r_j \) equals the impressed antidromic rate for all values of \( j \) except \( p \).

Fig. 3 shows the results of an experiment with antidromic inhibition. This experiment is directly analogous to Experiment 1 (Fig. 1 A), except here antidromic stimulation of the optic nerve drives most of the retinal units together to elicit the inhibitory effects, whereas in Experiment 1 only a small cluster of neighboring receptors participated. Also the range of this experiment was limited to values of \( e_p \) less than 20 ips. Nevertheless, note that the highest antidromic frequency in this experiment and the brightest inhibiting light in Experiment I produced qualitatively similar effects for the same values of \( e_p \). As one might expect, the antidromic stimulus to the whole optic nerve produced larger inhibitory effects than did the illumination of a small cluster of ommatidia.
Following the procedure used in treating the data of Experiment I, the results given in Fig. 3 were replotted and sums of the inhibitory coefficients ($\Sigma k_{ij}$) were computed for several uninhibited response rates of the test receptor. The resulting $\Sigma k_{ij}$'s ranged from 1.7 at an $e_p$ of 20 impulses/s to 0.6 at 5 impulses/s. These values are comparable to those obtained in Experiment II, as one might expect, since in both experiments inhibition was elicited from large regions of the retina. Normalized values of $\Sigma k_{ij}$ are plotted in Fig. 4.

**SUMMARY OF RESULTS**

Experiments I, II, and III were designed to investigate lateral inhibitory effects over wide ranges of excitation. All three experiments gave the same result: The reduction of the response from an ommatidium caused by a constant stimulus on neighboring units is influenced by its level of excitation. This result can be described in quantitative terms by comparing the values of $\Sigma k_{ij}$ for different levels of excitation ($e_p$) of the test receptor. Fig. 4 summarizes the results of all three experiments for values of $e_p$ up to 27 impulses/s. Above this level of excitation, the results varied considerably from one receptor to another within the same eye and no general rule could be given for a population of receptors. According to the Hartline-Ratliff formulation, the data in Fig. 4 should describe a straight line with a slope of zero. However, the slope is positive, indicating that $\Sigma k_{ij}$ is not constant but instead increases signifi-
Fig. 4. Estimated values of the normalized sum of inhibitory coefficients based upon experimental data and the Hartline-Ratliff formulation in Eq. 2. The filled circles are mean values of these sums, normalized by a constant factor to the value 1.0 at 27 impulses/s for the uninhibited response rate, $e_p$. The data were obtained from nine experiments, each with a different eye, using light-evoked inhibition. The vertical lines indicate the standard deviations of the means. The standard deviations increased substantially for uninhibited response rates greater than 27 impulses/s. The slanted line is a least squares fit to the data. The four unfilled circles show the results (after normalization) of an experiment using antidromic inhibition evoked by repetitive current shocks to the optic nerve. In each experiment an approximate 10-fold increase in light intensity was required to increase the uninhibited response rate from 10 to 20 impulses/s.

stantly with $e_p$. In other words, it is the receptor's sensitivity to inhibition that increases with increasing levels of excitation.

This effect can be included in the Hartline-Ratliff formulation by replacing each inhibitory coefficient ($k_{pi}$) in Eq. 2 by the term $(1 + ae_p)k_{pi}^\prime$. The values of $a$ and $\Sigma k_{pi}^\prime$ can be calculated from the slope and the y-intercept of the line in Fig. 4. Eq. 3 in Table I gives the revised formulation. We refer to the dependence of $\Sigma k_{pi}$ upon $e_p$ as a "nonlinear" inhibitory effect because the inhibition of a given unit ($e_p - r_p$) is not linearly related to the responses of neighboring units. Rather, in experiments like II, there is a second order dependence of inhibition on the mean response rate.

DISCUSSION

The fact that Hartline et al. (1956) found the sensitivity of a receptor to lateral inhibition to be relatively unaffected by its level of excitation, and we did not, can probably be attributed to the different ranges of light intensity used in each study. They employed an intensity range of about 2.5 log units which produced uninhibited firing rates ($e_p$) of up to 14 impulses/s. We used a 5.0-log unit range which gave $e_p$'s of up to 60 impulses/s. At the lower light intensities, where the two studies overlap, the results are in general agreement
(compare Fig. 1 A in this paper to Fig. 8 in Hartline et al., 1956). In this range of intensities it appears that a receptor's sensitivity to inhibition is not strongly influenced by the incident light level. This observation was one of the earliest made by Hartline and his coworkers on inhibition in the *Limulus* eye and it forms the basis of the general theory given by Eq. 2. Although at the lower intensities, the effect may indeed be weak, at higher intensities it is not. Increasing the light intensity by a factor of 25 doubles the sensitivity of a receptor to lateral inhibition. This represents a deviation from the linearity of Eq. 2 which becomes significant when the illumination of the eye covers a relatively large range of intensities.

We should point out that the activity of a single receptor unit under constant illumination ($e_p = \text{constant}$) can be described by the original Hartline-Ratliff formulation, that is, the revised equation (Eq. 3) reduces to Eq. 2 for constant $e_p$. As a result, studies which applied linear systems analysis to the dynamics of the inhibitory process (Knight et al., 1970) were unaffected for cases of constant or nearly constant levels of excitation ($e_p$).

Although much is known about the inhibitory process in the *Limulus* eye, the mechanism underlying the nonlinear effect has not yet been established. Purple (1964) qualitatively predicts results similar to ours from arguments based on his work on the electrical properties of the impulse-firing cell (eccentric cell) in the ommatidium. For example, Purple shows that as the cell's membrane is depolarized and the membrane potential displaced from the inhibitory equilibrium potential ($-70 \text{ mV}$), the potential drive for the flow of inhibitory current increases. For a fixed inhibitory input, i.e. constant conductance change, the inhibitory current is large for a large membrane depolarization which occurs when the cell is maximally stimulated. The inhibitory current sums with the excitatory current at the pacemaker region of the eccentric cell and the resulting "net" ionic current determines the cell's firing rate. As a result, inhibition would influence most strongly the responses of maximally stimulated cells.

However, Purple also points out that it is possible for the inhibitory current to be shunted away from the pacemaker region of the cell. Possible shunt pathways could be patches of membrane that are highly conductive because of large excitatory inputs. If such shunting occurred, the expected result would be a decreased inhibitory effect at high levels of excitation as was the case for the experiment shown in Fig. 1. As we have noted, other experiments showed an increase in sensitivity to inhibition at high light intensities and still other experiments indicated a leveling off of the effect. If shunting exists, these results suggest that the degree of shunting may depend on the relative location of the excitatory and inhibitory current pathways and that the relative location of the two current pathways may change from cell to cell. It is possible, therefore, to explain various features of the nonlinear effect in terms of ionic currents within the eccentric cell of the ommatidium.
What role, if any, the nonlinear effect may play in processing visual information is not known. The results of our experiments suggest that it may serve to increase the operating range of the receptor units. Dimly illuminated ommatidia continue to respond under large inhibitory inputs because of their lower sensitivity to inhibition. The capability of responding under such stimulus conditions is probably achieved at the expense of the normal benefits of lateral inhibition, although this possibility has not been directly tested. At higher levels of illumination the ommatidia regain their sensitivity to inhibition. Analogous effects have been described by H. B. Barlow et al. (1957) for the cat retina. By studying the influence of spatial summation on the response of ganglion cells, they concluded that lateral inhibition was weak at the low, scotopic levels of illumination and strong at the higher photopic levels. The possible role that this property of inhibition may play in extending the operating range of retinal units has been discussed by H. B. Barlow and Levick (1969).

The dependence of a receptor's sensitivity to lateral inhibition on the incident light level may actually reduce physiological contrast, that is, it may decrease the efficacy of inhibition to accentuate borders and contours in the visual field. This and other possible effects of the nonlinearity on coding spatial information are discussed in detail in the following paper.

The nonlinear inhibitory affect we have described is probably not unique to the *Limulus* eye. As indicated above, the cat retinal ganglion cells exhibit similar properties. Also, a nonlinear effect of the type we have described may form the basis of a motion-detecting system such as the one responsible for the optomotor response of insects (Thorson, 1966).

Presumably lateral inhibition filters the information falling on the retina, selecting and enhancing certain features and suppressing others. The main characteristics of the filtering process in the *Limulus* eye are adequately described by the classical Hartline-Ratliff equation (2); the more subtle features described in this paper require a somewhat modified version (Eq. 3). It is entirely possible that the results of future experiments will lead to further modifications.

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