Correlated Firing of Cat Retinal Ganglion Cells. 
II. Responses of X- and Y-Cells to 
Single Quantal Events 

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SUMMARY AND CONCLUSIONS

1. The correlated firing of neighboring cat retinal ganglion cells was examined at low background levels and in darkness. At these backgrounds, cross-correlograms showed a strong, broad peak for a pair of on-center cells, a weak, broad peak for a pair of off-center cells, and a broad well for two cells of opposite center sign. The much narrower peaks and wells described in the preceding paper (30) were superimposed on these broad peaks and wells.

2. An examination of these correlations and of the firing patterns of individual cells provides evidence for the following conclusions. The broad peaks and wells are caused by a single type of common input event lasting 40–50 ms. This input event is excitatory to on-center cells and often causes 2 or 3 spikes in a single on-center cell, giving these cells a tendency to fire in bursts at low backgrounds. The input event is inhibitory to off-center cells and causes visible gaps in their maintained discharge at low backgrounds. Two neighboring on-center cells have a strong tendency to fire together because they are both excited by shared input events. Two off-center cells show a weak tendency to fire together because they are both restricted to firing at the times when they are not inhibited together by shared input events. An on- and an off-center cell have a tendency not to fire together because the off-center cell is inhibited when the on-center cell is excited by their shared input events.

3. The rate of the input events received by ganglion cells at the lowest backgrounds was linearly related to the quantity of background light and matched the calculated rate of effective photon absorptions in the rods. The events appear to originate as individual quantal events in the rods.

4. The characteristic shapes of the correlograms for the different types of pairs at low light levels imply that the average excitatory response of on Y-cells to these quantal events is more transient than that of on X-cells, and that the average inhibition of off Y-cells by quantal events is more transient than that of off X-cells.

5. Further analysis of the correlograms indicates that the primary source of the more sustained response of on X-cells to quantal events is a relatively sustained excitation that does not pass through the active inputs described in the preceding paper and that is considerably stronger for on X-cells than for on Y-cells. The analysis provides evidence for an analogous sustained inhibition of off-center cells that is considerably stronger for off X-cells. The analysis suggests that on active inputs have a rather transient response to quantal events and provide an initial transient excitation of on-center cells and inhibition of off-center cells.

6. It is proposed that cone bipolar cells are the final pathway for the relatively sustained effects of quantal events and that tonic signals from these bipolar cells are a major source of maintained activity for X-cells and a minor source for Y-cells at higher backgrounds.

7. Two hypotheses are discussed as follows: that light-adapted X-cells show a greater
sustained response than Y-cells to a central stimulus because they receive stronger input from cone bipolar cells and that active inputs serve as a "final common pathway" to ganglion cells for the signals responsible for some of the distinctive properties of Y-cells.

INTRODUCTION

The preceding paper (30) deduced the properties of some of the inputs to retinal ganglion cells by examining the correlated firing of neighboring ganglion cells during maintained activity. This correlated firing revealed the activity of a restricted set of inputs to ganglion cells, namely, the inputs that are themselves spontaneously active under a high, constant background illumination. The present paper examines the correlated firing of neighboring ganglion cells under very low background illumination. At these light levels, the capture of a single photon can cause 2–3 spikes in dark-adapted ganglion cells (6) and is thus a strong time-varying stimulus to the neurons driving the ganglion cells. One might expect the correlated firing of cells under these conditions to reflect the activity of other ganglion cell inputs as well as that of the spontaneously active inputs. Indeed, at very low light levels, cross-correlograms between neighboring ganglion cells acquire features much more extended in time than the narrow peaks and wells appearing in the correlograms of the previous paper. Evidence on the source of these features is presented in DISCUSSION to obtain further information on inputs to ganglion cells.

METHODS

This paper presents results obtained at low background levels from 44 pairs of ganglion cells in 10 cats. In nine cats, pairs of cells were recorded as described in the preceding paper (30), with one electrode in the retina and a second electrode in the optic tract. In one experiment, pairs of cells were recorded with two independent intraocular electrodes introduced through two different holes in the sclera.

Backgrounds

A background of 10 cd/m² at the tangent screen was produced by indirect illumination of the whole experiment room using ordinary incandescent bulbs. Lower background levels were generated by shining an appropriate incandescent light onto the rear wall of the experiment room, which resulted in fairly even illumination of the tangent screen at the front wall. Wratten neutral-density filters were interposed to produce backgrounds below 0.01 cd/m². Total darkness was imposed by placing the experimenter and oscilloscopes behind a light-tight curtain; all other sources of light in the experiment room were carefully shielded.

Light levels were measured with a calibrated Spectra Brightness Spot Meter (Photo Research Corp.) and an SE1 photometer. Changes in background were accurate to 0.1 log unit, while absolute light levels might have been in error by as much as 0.2 log units. The effective background level also depended on the diameter of the pupil, which was usually near 11 mm but varied between about 9 and 13 mm. Assuming a pupil size of 11 mm, a color temperature of 2,854 K and the human scotopic luminous efficiency function, 1 cd/m² is, for the rods, equivalent to $6.3 \times 10^7$ quanta (507 nm) deg⁻²·s⁻¹ at the cornea (42).

Correlation procedure

The recording search was carried out at a background of either 10 or 0.1 cd/m². Preliminary experiments monitored the recovery of sensitivity of a ganglion cell to a flashing spot during dark adaptation. From 10 cd/m², sensitivity took slightly over an hour to reach its fully dark-adapted value, while from 0.1 cd/m² dark adaptation took about 10 min. Typically, if a pair of cells was obtained at 10 cd/m², the background was extinguished and spikes recorded through most of dark adaptation. Intermediate backgrounds were then imposed and the lowest backgrounds imposed only when complete dark adaptation was assured. If a pair of cells was found at 0.1 cd/m², the background was reduced in 0.5- or 1-log unit steps; when the lowest backgrounds were reached, the retina was fully dark adapted.

Correlograms and autocorrelograms

The construction and interpretation of cross-correlograms was described in the preceding paper (30). In this paper the bin width is generally 1 ms, as in the previous paper, but the correlograms extend 100 rather than 50 ms from the center in order to show a larger portion of the base line for reference. The value of the base line was obtained by averaging bins 60–100 ms from the center; but, if that portion of the correlogram was still not flat, the base line was obtained from bins 100–200 ms from the center.

For pairs in which one cell (B) was recorded on an optic tract electrode, the correlogram appears shifted to the right by an amount equal to B's conduction latency, as explained previously (30) (pairs in Figs. 1, 2D, 2G, 4D, 4G, and 4J).
is no such shift for pairs recorded on two intraocular electrodes (pairs in Figs. 2A, 3A, 3D, 3G, and 4A).

An autocorrelogram indicates the average firing probability of a single cell at all times after a firing of that same cell. It is constructed like a cross-correlogram except that the spike train of the single cell is the source of both the reference and target events. Each spike is considered as a reference event occurring at time 0, and all succeeding spikes (the target events) are counted in bins corresponding to the intervals from the reference spike to those following spikes.

RESULTS

Slow and fast components

The left side of Fig. 1 shows cross-correlograms from a pair of on X-cells at progressively lower backgrounds. At -2 log cd/m² (Fig. 1A), the cross-correlogram contained a peak involving 12.3% of each cell's spikes; this peak was the same in width (11 ms) but twice as strong as the one obtained at 1 log cd/m² (Fig. 1E of the preceding paper (30)). As the background was lowered to -3 log cd/m² (Fig. 1D), the central peak became even stronger (up to 17.4%) and appeared superimposed on a much broader hump. At still lower backgrounds (-4 and -4.5 log cd/m², Fig. 1G and J), this broad hump grew progressively larger. At -5.5 log cd/m² (Fig. 1M), the correlogram was strongly elevated above the base line for about 45 ms from the center and the correlation was qualitatively quite similar to that obtained in total darkness (not shown). Throughout these progressive changes in the broad hump, the sharp peak continued to appear superimposed on the broader one and changed only slightly in size and shape.

Analogous changes with background were seen for all other types of correlations. As the background was reduced for a pair of neighboring ganglion cells of opposite center sign, a broad well gradually appeared on which was superimposed the sharper well normally seen at higher backgrounds (see correlograms of dark-adapted pairs in Fig. 4). Thus, for all pairs, the correlations at the lowest light levels can be usefully described in terms of two distinct components: a slow, or long duration, component, typically extending 30-50 ms to either side of the center of the correlation, and a fast component, which appeared at all backgrounds and was attributed in the preceding paper (30) to spontaneously active inputs to the ganglion cells.

Relative strengths of slow components for different cell types

Figures 2, 3, and 4 show correlograms obtained from the various types of pairs either in darkness or at backgrounds up to -5.5 log cd/m². These examples illustrate that a simple pattern appears in correlations between cells of differing type: each of the four cell types has associated with it a slow component of a characteristic relative magnitude; and a correlation between a pair of cells shows a component on the left side characteristic of the reference cell A and a component on the right side characteristic of the target cell B. The relative strengths of the slow components are as follows: on X, strongest; off X, moderately strong; on Y, about half as strong as on X; off Y, weakest, about half as strong as off X. To a rough approximation, the absolute magnitudes of the slow components in all types of correlations also follow the above ordering.

A slow component's strength can be measured by the level, relative to the base line, of its intersection with the fast component of the central peak or well. The slow components in the examples are generally somewhat but not greatly stronger than the average found for each type of pair. Slow components were fairly constant in strength from darkness to -5 log cd/m² except as noted below. Two factors in addition to cell type and background influenced the strength of slow components. First, slow components decreased in strength with increasing separation between receptive-field centers in roughly the same way as did the fast components (the correlations described in the preceding paper (30)). Results from 7 of the 10 types of pairs support this conclusion. Second, during dark adaptation, slow components gradually increased in magnitude as if background were being progressively lowered. These two factors were taken into account in formulating the summaries given next. For each type of pair, the numbers of fully dark-adapted (FDA) and partially (insufficiently) dark-adapted (PDA) pairs are given.

ON X ON X (n = 4 FDA). Correlations between
FIG. 1. Cross- and autocorrelograms for a pair of on X-cells at various backgrounds. In this figure, as in Figs. 2, 3, and 4, the correlogram between A and B is on the left and the autocorrelograms of A and B are in the middle and on the right, respectively. Bin width is 1 ms, N_A and N_B are the number of spikes from A and B. These cells were at an eccentricity of 12° and were separated by 0.3° or 1.6 spacings. A, B, C: background of -2 log cd/m², N_A = 9,756, N_B = 9,489. D, E, F: -3 log cd/m², N_A = 9,633, N_B = 8,443. G, H, I: -4 log cd/m², N_A = 8,351, N_B = 8,375. J, K, L: -4.5 log cd/m², N_A = 8,468, N_B = 8,973. M, N, O: -5.5 log cd/m², N_A = 4,320, N_B = 4,444.
on X-cells (Fig. 1 and another pair in Fig. 2A) showed the strongest slow components of any type of pair. The slow components were essentially equal on both sides; that is, the correlations were symmetrical.

ON X-ON Y (n = 5 FDA). In correlations between on X- and on Y-cells (e.g., Fig. 2D), the slow components associated with the on X-cells were moderately strong and 1.8–2.8 (mean, 2.1) times as large as those associated with the on Y-cells.

ON Y-ON Y (n = 2 FDA). Correlations between on Y-cells (e.g., Fig. 2G) were essentially symmetrical with slow components of medium strength.

OFF X-OFF X (n = 1 FDA, 2 PDA). The correlation in Fig. 3A from a pair of off X-cells in the dark has symmetrical and rather weak slow components; two other partially dark-adapted pairs gave similar results. For the pair in Fig. 3A, and for other types of pairs of off-center cells, the slow components became substantially stronger with increasing backgrounds up to −4.5 log cd/m².

OFF X-OFF Y (n = 3 FDA, 1 PDA). In correlations between off X- and off Y-cells (e.g., Fig. 3D), the
slow components associated with the off X-cells were rather weak and 1.4–2.8 (mean, 1.9) times as large as those associated with the off Y-cells. 

OFF Y-OFF Y (n = 3 FDA). Correlations between off Y-cells showed quite weak and symmetrical slow components. In the example of Fig. 3G, the slow components are partially masked by the dips (due to refractoriness) that typically appear on both sides of a strong, sharp central peak (30).

ON X-OFF X (n = 3 FDA, 3 PDA). As Fig. 4A shows, on X-off X correlations can have quite strong slow components on both sides. Here, the intersections between slow and fast components cannot be discerned, possibly because of the strength of the slow components combined with the broadness of the fast components (observed in isolation at 0.1–1.0 cd/m²). For five of the six pairs, those intersections could be discerned, at least under certain circumstances (e.g., at −4 to −3 log cd/m² or during dark adaptation), and the slow components of the on X-cells were 1.2–2.0 times stronger than those of the off X-cells.

ON Y-OFF X (n = 3 FDA). In correlations between on Y- and off X-cells (e.g., Fig. 4D), the slow com-
FIG. 4. Cross- and autocorrelograms from pairs of on- and off-center cells. Bin width is 1 ms. A, B, C: on X-off X pair in the dark. A = on X, B = off X, eccentricity 8°, separation 0.15° or 1.9 spacings. N_a = 6,077, N_b = 30,225. D, E, F: off X-on Y pair, sum of results obtained in the dark and at -6 log cd/m². A = off X, B = on Y, eccentricity 4.5°, separation 0.3° or 4.1 spacings. N_a = 39,512, N_b = 15,194. G, H, I: off Y-on X pair, sum of results obtained in the dark and at -6 log cd/m². A = off Y, B = on X, eccentricity 3°, separation 0.2° or 2.3 spacings. N_a = 37,154, N_b = 7,935. J, K, L: off Y-on Y pair at -5.5 log cd/m². A = off Y, B = on Y, eccentricity 10°, separation 0.35° or 2.8 spacings. N_a = 10,200, N_b = 4,745.

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ponents of the off X-cells were moderately strong and only 1.1–1.4 (mean, 1.2) times stronger than those of the on Y-cells.

**ON X-OFF Y** (*n* = 5 FDA, 2 PDA). For fully dark adapted on X-off Y pairs (e.g., Fig. 4G), the slow components of the on X-cells were fairly strong and about 2.5–4.0 (mean, 3.0) times stronger than those of the off Y-cells. Partially dark-adapted pairs showed qualitatively similar differences between the two slow components.

**ON Y-OFF Y** (*n* = 5 FDA). For on Y-off Y pairs (e.g., Fig. 4J), the slow components of the on Y-cells were of medium strength and at least 1.2–1.4 times stronger than those of the off Y-cells. Slow components of off Y-cells were weaker in darkness than at −5.5 to −4 log cd/m².

**Bursty discharge and autocorrelogram overshoots**

In functional terms, the broad peak in a correlation between two on-center cells (Fig. 2) means that each cell has, on the average, an enhanced firing probability for about 45 ms after a firing of the other. As argued in the preceding paper (30), this implies that the two cells are responding together to common input events whose effect on each cell alone lasts about 45 ms. The following examination of the firing patterns of on-center cells provides evidence that these input events have an excitatory effect (i.e., tend to increase the firing rates of the cells).

Figure 5A is a representation of the spikes from the two on X-cells of Fig. 1 at −5.5 log cd/m². Each cell tended to fire bursts of typically 2–4 spikes in addition to numerous relatively isolated single spikes.

For more detailed information on the nature of this bursty discharge, consider Fig. 1N, the autocorrelogram of one of the two on X-cells at −5.5 log cd/m². If successive spikes from the cell occurred independently, the autocorrelogram would be flat at a level equal to the cell's mean firing rate. Figure 1N indicates that the cell's firing probability rose very quickly out of the refractory period after a spike and within 10 ms reached a peak, or overshoot, about 4 times higher than the mean firing probability. This overshoot is
largely over in about 45 ms. The strength of the overshoot can be measured by the number of counts in the overshoot above the autocorrelogram’s base line, expressed as a fraction of the number of spikes from the cell. (The base line was taken as the average of the bins from 150 to 200 ms after a spike.) This measure reflects the frequency with which a spike is followed by an “unexpected” or extra spike during the duration of the overshoot. For the cell of Fig. 1N, 62% of the firings were followed by an extra spike within 45 ms. Thus, when the cell fired, it had a strongly enhanced firing probability for up to 45 ms thereafter and it often fired at least 1 more spike, resulting in a burst. These facts suggest that the bursts in the on X-cell firing and the corresponding overshoot in the autocorrelogram were caused by excitatory input events lasting about 45 ms.

The spike records of on-center cells in the dark and at low backgrounds generally revealed a tendency to fire in bursts, as previously noted by Barlow and Levick (4) (Fig. 7A shows an example of on Y-cell firing). This tendency is confirmed by the presence of overshoots similar to the one just described in the autocorrelograms of all on-center cells (see autocorrelograms in Figs. 1, 2, and 4). Overshoots lasted 40–50 ms. Overshoots were about twice as strong for on X-as for on Y-cells. For 13 on Y-cells, the maximum overshoot observed (regardless of background) ranged from 21 to 51%, with a mean of 33.1% (SE = 2.8), while for 14 on X-cells recorded in the same experiments, maximum overshoots ranged from 48 to 110% (except for one low value of 19%, Fig. 2E), with a mean of 65.3 (SE = 6.6). (An additional 5 on X-cells recorded in a cat from which no on Y-cells were recorded had unusually high overshoots from 112 to 165%.)

The size of an overshoot is related to the average number of spikes caused by each of the input events responsible for the overshoot, as shown next.

If a cell fires a spike spontaneously or if it fires only a single spike in response to an input event, any following spikes occur by chance and contribute to the base line rather than to the overshoot. If the cell fires a burst of 2 spikes, the first spike has 1 extra spike following it, while succeeding spikes occur by chance and do not contribute to the overshoot. Thus, if a cell fires only in bursts of 2 spikes, half of its spikes would be followed by an extra spike and the strength of the overshoot in its autocorrelogram would be 50%. In a burst of 3 spikes, there are 2 extra spikes following the first one and 1 extra spike following the second one, for a total of 3 extra counts. For a cell firing only in bursts of 3 spikes, each burst of 3 spikes would add 3 extra counts and the overshoot would be 100%. By the same logic, a discharge consisting only of bursts of n spikes would give an overshoot of strength (n - 1)/2. In practice, the number of spikes varies from burst to burst and there may also be spontaneous spikes. If m is the average number of spikes per occurrence where each burst or single spike is an occurrence, analysis shows that the overshoot should be somewhat greater than (m - 1)/2 and that the deviation from the formula increases with greater variability in the number of spikes per occurrence.

The formula obtained above implies that each of the input events responsible for the overshoots causes 1.5–2 (mean, 1.7) spikes in an on Y-cell and 2–3.2 (mean, 2.3) spikes in an on X-cell, provided these events are the sole cause of on-center cell spikes at low backgrounds. To the extent that some of a cell’s spikes are not due to these events, the number of spikes per input event would be greater.

Slow components in on-on correlations

In Fig. 5A, the two on X-cells sometimes fired bursts simultaneously, a burst in one cell occasionally occurred at the same time as a single, isolated spike from the other, and the two cells sometimes fired such isolated spikes at about the same time (i.e., within 50 ms of each other). If both the bursts and the isolated spikes are considered to represent single events produced by the cells, then Fig. 5A suggests that the events for the two cells tended to occur at the same time and that this tendency caused the broad peak in their cross-correlogram.

To test this point, “event trains” were constructed from the spike trains by algorithms (described in the legend of Fig. 5) that generally selected the first spike from each burst and each isolated spike as an event. The events selected by these algorithms thus largely reflect the occurrence of the underlying input events responsible for the bursts. Figure 5B shows events corresponding to the spikes in the last line of Fig. 5A. The cross-correlation of the event trains for the two on
X-cells, shown in Fig. 6, confirms that the two cells had a strong tendency to produce events nearly simultaneously. The number of counts in the peak of Fig. 6 indicates that the two cells produced 26.6% of their events together, in addition to coincident events expected by chance. Other pairs of on-center cells gave similar results.

The duration of the slow components in Fig. 6, the correlation between events, is only 25 ms rather than the 45 ms found in Fig. 1M, the correlation between spikes. This difference probably arose because many of the extra counts appearing 25-45 ms from the center of the peak in the spike correlogram occurred when an early spike in a burst of one cell was correlated with a late spike in a burst of the other. These occurrences were eliminated when the bursts were replaced by individual events and intervals of 25-45 ms between events in the two cells occurred no more often than by chance.

In fact, the narrowness of the peak in Fig. 6 provides additional support for the hypothesis that the input events responsible for bursty firing are the cause of the slow components. If randomly selected subsets of the spikes from two cells were cross-correlated, a peak in the correlogram of the random subsets would have the same width as the peak in the original correlogram. Thus, if the input events responsible for the bursty firing of the on X-cells were unrelated to the cause of the slow components in their correlogram, then the constructed event trains would be essentially random subsets of the spike trains from the cells, i.e., random with respect to the cause of the slow components. In that case, the event correlogram would be no narrower than the original correlogram.

In sum, it appears that the slow components in correlations between two on-center cells are due to common input events lasting about 50 ms that are excitatory to the two cells and cause their bursty discharge. The alternate explanation, that the 50-ms common input events are inhibitory, appears much less likely, since it is the bursts rather than the gaps in firing for on-center cells that last roughly 50 ms; gaps in firing are much longer and more variable in extent.

Slow components in on-off correlations

The slow components, lasting about 50 ms for off-center cells in correlations with on-center cells (Fig. 4), could arise if these input events excitatory to on-center cells simultaneously inhibited off-center cells for about 50 ms or if other input events excited off-center cells and inhibited on-center cells. Figure 7A, a representation of spikes from the off X-on Y pair of Fig. 4D, supports the first cause rather than the second. The on Y-cell (lower trace in each set) tended to fire in brief bursts. The off X-cell (upper trace) had a high, regular discharge, interrupted occasionally by brief pauses, or gaps, in firing. These gaps often occurred at the same time as a burst or isolated spike from the on Y-cell, suggesting that the off X-cell was often inhibited when the on Y-cell received the excitatory input events causing its bursty firing. To test this point, events were derived from the spike train of the on Y-cell, as described in the legend of Fig. 5, and events for the off X-cell were defined to occur at the midpoint of each interspike interval longer than 50 ms. A sample from the two event trains appears in Fig. 7B. The correlogram of the two event trains showed a peak similar to the one in Fig. 6, verifying that the events of the on Y-cell were correlated with the pauses of the off X-cell. This peak involved 40% of the off X-cell's pauses and 17% of the on Y-cell's events. Analysis of other on-off pairs gave similar results, thus providing evidence that off-center cells are inhibited by the input events excitatory to on-center cells.

Further evidence that off-center cells receive inhibitory events is provided by interval histograms, which indicate that off-center cells have a distinct excess of long interspike intervals. Figure 8 shows interval histograms from the off X-cell of Fig. 4D. For off-center
FIG. 7. A: representation of spikes from the off X-on Y pair of Fig. 4D in the dark. Spikes from the off X- and on Y-cell are shown in the top and bottom trace of each pair. B: event trains derived from the last line of spikes in A. For the off X-cell, an event occurred at the middle of any interval longer than 50 ms; for the on Y-cell events were chosen as described in Fig. 5.

FIG. 8. Interspike interval histograms from the off X-cell of Fig. 4D, plotted on a logarithmic scale. Bin width is 2 ms; the ordinate is the log of the number of times each interval occurs. A: at 0.1 cd/m². B: in the dark. Lines were fitted to the two segments of the histogram by eye.

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335 cells at higher light levels (0.1 or 10 cd/m²), the declining portions (tails) of interval histograms, plotted on a logarithmic scale, either fit a straight line or gradually curved down slightly, as in Fig. 8A. At low or zero backgrounds, the tails of interval histograms always broke into two distinct segments, each one approximately linear, as in Fig. 8B. This break was less distinct for off Y-cells (13 cells) than for off X-cells (14 cells). The appearance of the break at low backgrounds could reflect the appearance either of events causing an excess of short intervals or of events causing an excess of long intervals. Events causing an excess of short intervals would make the cells fire in bursts, leading to a sizable overshoot in their autocorrelograms. Since there were breaks in the interval histograms even for off-center cells that showed virtually no overshoot in their autocorrelograms, the breaks quite probably reflect an excess of long rather than short intervals, caused by events inhibitory to off-center cells for 40 ms or longer.

The rate of these inhibitory events can be estimated by comparing the actual number of long intervals with the number of long
intervals expected by extrapolation of the tail's early segment. (This gives an underestimate because two successive inhibitory events can produce one long interval.) The rate was 2.3/s for the cell of Fig. 8B and ranged from 1.6 to 4.2 for 8 off X-cells in the dark. This is comparable to the rate of occurrence of excitatory events for on X-cells in the dark, about 2-6/s, estimated as described in the legend of Fig. 5. Thus, on X- and off X-cells appear to receive their respective excitatory and inhibitory events at about the same rate, providing further evidence that these events are responsible for the slow components in correlations between on- and off-center cells.

**Slow components in off-off correlations**

If two overlapping off-center cells received inhibition simultaneously from these input events, then their correlogram would show a broad peak; i.e., the two cells would tend to fire together because they were both restricted to firing at the times when they were not being inhibited together (33). Figure 9A, a representation of spikes from the two off X-cells of Fig. 3A, suggests that the cells did receive common inhibition. Both cells had a high, irregular discharge interrupted by the usual pauses lasting 40–100 ms. The cells apparently tended to pause together. To test this point, events were defined to occur at the midpoint of any interspike interval greater than 40 ms (see Fig. 9B). The cross-correlogram of the two event trains showed a peak similar to the one in Fig. 6, confirming that the two cells tended to pause together. This peak involved 27.4% of each cell’s pauses.

Can this apparent common inhibition of the two off X-cells account for the slow components in their correlogram, Fig. 3A? Computer simulations indicate that it can. In simulations, two cells firing at the same rate as those of Fig. 3A had slow components as strong as in Fig. 3A when they received common input at a rate of 3/s from an input that inhibited them totally for 40 ms and partially for another 10 ms. This is a reasonable rate of common inhibition, given that the receptive-field centers of these adjacent off X-cells overlapped by at least 50% and that they each received apparent inhibitory events at a rate of at least 3/s, based on estimates from interval histograms. Therefore, the weak slow components in correlations between off-center cells can be explained easily by common inhibition from the events that are excitatory to on-center cells; there is no need to invoke the alternate explanation, 50-ms events excitatory to off-center cells.

![Figure 9](http://jn.physiology.org/)

**FIG. 9.** A: representation of spikes from the two off X-cells of Fig. 3A in the dark. B: event trains derived from the last line of spikes in part A. An event was chosen as the middle of any interspike interval greater than 40 ms.
Autocorrelogram overshoots for off-center cells

The autocorrelograms of off-center cells at low backgrounds showed weak overshoots lasting 30–50 ms (e.g., Fig. 3B, C), which could reflect either excitatory or inhibitory input events of that duration. An inhibitory effect produces an overshoot with the same duration because the firing rate soon after a spike (i.e., in the overshoot) tends to reflect the mean firing rate during times when the cell is not being inhibited, while the firing rate long after a spike (i.e., in the base line) reflects the lower, overall mean firing rate. As discussed above, interval histograms provided evidence that off X-cells in the dark received inhibition at rates up to 4/s. In simulations, cells receiving 50-ms inhibitory events at 4/s had overshoots similar in shape to the actual ones and as strong as the strongest seen for off X-cells in the dark (Fig. 3C). The actual overshoots did become substantially stronger with increasing background light (e.g., Fig. 4C, off X-cell in dark, versus Fig. 3F, same cell at −5.5 log cd/m²). However, the rate of inhibitory events apparently increases with background (see below), and simulations indicate that this increase in inhibitory rate is sufficient to account for the larger overshoots observed at higher backgrounds. Thus, the overshoots for off-center cells can be accounted for by the events previously identified as inhibitory to off-center cells for about 50 ms; they do not provide any evidence of input events excitatory to off-center cells for that duration.

The most prominent overshoot seen for an off-center cell, the sharp peak in Fig. 4E, probably reflects a tendency to fire periodically and does not indicate that the off X-cell received excitatory input events. Figure 4E shows that this cell’s firing probability stayed near zero for 7 ms after a spike, then rose to a peak at 16–17 ms (which was also the most common interspike interval), then fell from the peak in 8 ms, about as fast as it rose to the peak. These features suggest that the peak arises from a collection of interspike intervals approximately evenly distributed around the most common interval and reflects a tendency for the cell to fire periodically at a preferred interval of 16–17 ms. The regularity of the maintained discharge in Fig. 7A supports this conclusion. A statistical measure of irregularity, the ratio of mean to variance for the pulse number distribution (4), also supports this sharp distinction between an overshoot due to a tendency to fire periodically and one due to a tendency to fire in bursts. Such overshoots appeared more weakly for other off-center cells (e.g., Figs. 3I, 4II).

Duration of effects

The duration of a slow component in a correlation between two cells is the interval from the time of coincident firing of the two cells to the time when the component returns to the base line. For both on X- and off X-cells, durations were 40–50 ms, while for on Y-cells they typically ranged from 35 to 45 ms. For off Y-cells the duration was often noticeably shorter, 25–40 ms, and the slow component often crossed to the other side of the base line before returning to it (e.g., Fig. 4G). These measurements are too few and too inaccurate to warrant statistical treatment. However, for each particular cell type, there was no obvious difference between the duration of its slow components in correlations with cells of the same center sign and its durations in correlations with cells of the opposite center sign. Such a difference would suggest the existence of two types of slow input events just as differences in durations of fast components indicated the existence of two types of active inputs in the preceding paper (30). Thus, the durations of slow components are consistent with their being caused by only the one type of input event previously identified.

Adaptation and slow components

An examination of how background level affects slow components, autocorrelogram overshoots, and mean firing rates provides further information on the nature and origin of the slow input events. In Fig. 10, the crosses show the strength of slow components, normalized and averaged for 12 pairs of cells (on-on and on-off); the squares are the average of normalized values for the autocorrelogram overshoots of 10 on X- and 7 on Y-cells (details in figure legend). The relative strength of slow components began to decrease noticeably as the background luminance was raised above −5 log cd/m²; slow components were gone when the background reached −3 log cd/m². The overshoots underwent the same decline but at slightly higher light levels. The overshoots fell to a residual level rather than to zero because many autocorrelograms at higher back-
FIG. 10. Decline in strength of slow components and overshoots with increasing background. Crosses for slow components were obtained as follows. For each pair of cells, the maximum deviation of the slow components from the base line was measured as a fraction of the base line, for each background at which the pair was correlated. These measures were then normalized to give a maximum value of one at whichever background had the strongest slow components. Crosses are the average of these normalized measures for 12 pairs of cells. Strength of autocorrelogram overshoots was measured by the method described in the text for 7 on Y-cells and 10 on X-cells. For each cell, measures were normalized to give a maximum value of 1. Squares are the average value of these 17 normalized measures. Curves are based on a visual sensitivity curve; see text for details.

grounds showed a small narrow peak (e.g., Fig. 1B, C). This peak probably reflected a tendency to fire periodically rather than in bursts; thus, only values above the residual level are of interest here.

The continuous curves in Fig. 10 are based on one typical curve of ganglion cell sensitivity to a visual stimulus versus background, from Fig. 5B of Enroth-Cugell and Lennie (14). (Other curves from reports by Enroth-Cugell and co-workers (e.g., Ref. 16) gave similar results.) The curve expresses relative sensitivity on a linear, not logarithmic, scale; it has been scaled vertically to match the total change in relative slow-component magnitudes. It has not been scaled horizontally, only shifted so as to give the best fit by eye. Clearly the curves fit rather well. Perhaps more important, when published reports on the background dependence of visual sensitivity (3, 5, 13, 16, 24, 29, 38, 43) are considered together with the factors governing that dependence (17), it becomes evident that the slow components and overshoots began to decline at about the same background as does visual sensitivity. These two aspects of the fit suggest strongly that the decreases in slow components and overshoots with increasing background reflect primarily an attenuation of the effects of slow input events on ganglion cells by the same adaptive mechanisms responsible for the decline in visual sensitivity.

Firing rates, event rates, and background level

Mean firing rate increased linearly with background light for 4 on X- and 4 on Y-cells up to rates of 25–30 spikes/s, which were reached at 3–10 μcd/m² for the X-cells and 1–3 μcd/m² for the Y-cells. Figure 11 shows an example of each type of cell. (For other cells, mean rate data were not taken at enough low backgrounds or were inadequate due to drift or oscillations in firing rates.) Mean firing rate can be related to the rate of slow input events by using the strength of autocorrelogram overshoots as an estimate of the number of spikes per event, as discussed earlier. Based on the results in Fig. 10, the number of spikes per event was nearly constant (increased by only 10%) from darkness to −5 log cd/m² (10 μcd/m²). Thus, the rate of input events as well as of spikes increased approximately linearly with background.

Two quantities are of interest in this linear relation. First, “dark light” is the background level that would produce additional activity equal to the intrinsic level of activity found in darkness (6). Dark light was estimated from the abscissa intercepts of lines such as in Fig. 11; at that value of dark light, the firing rate was directly proportional to the
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The mean value of dark light was 3.0 \( \mu \text{cd/m}^2 \) \((n = 8, \text{SE} = 0.72);\) there was no apparent difference between the values for X-cells and Y-cells. Second, the slopes of lines such as in Fig. 11 give the number of additional spikes per increment of background light: 1–2.5 spikes \( \cdot \text{s}^{-1} / \mu \text{cd} \cdot \text{m}^{-2} \) for X-cells (mean = 1.88, SE = 0.29, \( n = 4 \)) and 3.1–9.2 spikes \( \cdot \text{s}^{-1} / \mu \text{cd} \cdot \text{m}^{-2} \) for Y-cells (mean = 5.92, SE = 1.63, \( n = 4 \)).

The rate at which pauses occurred in off-center cells (as estimated from interval histograms) also increased with background light. For the cell of Fig. 4E, this increase was linear up to 3 \( \mu \text{cd/m}^2 \) and yielded a dark light of 2.5 \( \mu \text{cd/m}^2 \), comparable to that found for on-center cells. Less complete data from other off X-cells also suggested that the rate of pauses had a background dependence similar to that of the rate of events for on X-cells.

**DISCUSSION**

At very low light levels, correlations between neighboring ganglion cells showed long-duration components in addition to the fast components that appeared at all backgrounds. This discussion will first summarize the evidence that these slow components are caused by a single type of input event, then consider the evidence that this event originates in a quantal event; i.e., the breakdown of a single rhodopsin molecule in a rod. Then, the relative magnitudes of the slow components for the different cell types will be related to the time course of the average response of each cell type to these quantal events. An analysis of the inputs participating in these responses will lead to an extended model of retinal circuitry (Fig. 13). Finally, the sources of some differences in the visual properties of X- and Y-cells will be discussed.

**Evidence that slow components are caused by a single type of input event, excitatory to on- and inhibitory to off-center cells**

The slow components lasting about 50 ms in correlations at low light levels imply that neighboring ganglion cells receive common input events lasting about 50 ms.

On-center cells tended to fire in bursts lasting roughly 50 ms, this tendency was reflected by peaks, or overshoots, in the autocorrelograms of these cells. The strength and duration of these overshoots indicate that the bursty firing was caused by excitatory input events lasting about 50 ms and often causing 2–3 spikes in an on-center cell.

Neighboring on-center cells tended to fire bursts simultaneously, indicating that the slow components in their cross-correlograms were caused by common excitation from the input events responsible for their bursty firing.

The discharge of off X-cells appeared to be interrupted by pauses lasting 50–100 ms. Interval histograms confirmed that off-center cells had a distinct excess of intervals greater than 40 ms. These long intervals tended to occur at the same time as bursts in neighboring on-center cells. This indicates that off-center cells were inhibited for about 50 ms by the input events excitatory to on-center cells, thus producing the slow components in on-off correlations.

The pauses in firing of neighboring off X-cells tended to occur simultaneously, indicating that two such cells received common inhibition from these slow input events.

This common inhibition of neighboring off-center cells is sufficient to account for the slow components in their cross-correlograms. Inhibition by these input events is also sufficient to account for the weak overshoots lasting about 50 ms in autocorrelograms of off-center cells. The duration of the slow components for each particular cell type did not depend on whether the cell was being correlated with an on- or an off-center neighboring cell. These facts are consistent with there being only one type of slow input event and fail to provide evidence for a second type of event excitatory to off-center cells.

**Evidence that slow input events are quantal events in rods**

The events, like a light stimulus, are excitatory to on-center cells and inhibitory to off-center cells.

Their rate of occurrence is linearly proportional to the amount of background light plus a constant dark light, at least at the lowest backgrounds.

The strength of slow components caused by slow input events decreases over roughly the same range of backgrounds and at about the same rate as does visual sensitivity. This result is consistent with the input events originating in the photoreceptors.
The rate of effective photon absorptions matches the slow input event rate, as shown in the following calculation.

a) For 4 on X-cells, the increase in firing rate per increment of background light averaged 1.9 spikes $\cdot$ s$^{-1}$ $\cdot$ $\mu$cd$^{-1}$ $\cdot$ m$^{-2}$. b) The strength of autocorrelation overshoots suggested that on X-cells fire 2.3 spikes per input event. c) The measurement of 1 $\mu$cd/m$^2$ is equivalent to 63 quanta $\cdot$ deg$^{-2}$ $\cdot$ s$^{-1}$ at the cornea (see METHODS). d) Twenty-four percent of photons incident on the cornea are absorbed by rods at eccentricities of 5-10°, from microspectrophotometry (8). e) Fifty percent of absorbed quanta are effective in producing a rod response, from recordings of toad rods (7). f) The average X-cell has an equivalent center diameter of 0.44° or an area of 0.15 deg$^2$ (11; based on 39 cells recorded intraocularly, which were probably comparable to the cells recorded here in their distribution of receptive-field positions). g) Thus, on X-cells received $1.9/(2.3 \times 63 \times 0.24 \times 0.5 \times 0.15) = 0.73$ events per effectively absorbed quantum. This value is not much different from 1, considering the uncertainties in this calculation.

Barlow, Levick, and Yoon (6) found that on-center ganglion cells can respond to a single effectively absorbed photon with an average of 2-3 spikes, in good agreement with the estimate here of the 2.3 spikes per event, from the autocorrelogram overshoots of on X-cells. (Their sample of cells, recorded intraocularly near the area centralis, probably consisted predominantly of X-cells, based on my own recording experience.)

The statistical analysis of Barlow et al. (6) showed that their cells experienced a dark light equivalent to 5.5 quanta/s, with values for 9 of the 11 cells ranging from 2.2 to 6.5 quanta/s, in good agreement with the values of 2-6 events/s found here for on X-cells in the dark. Barlow et al. (6) showed one cell (their Fig. 1A) with a 70-ms response to a 10-ms flash providing an average of one absorbed quantum per flash. This indicates that the average response of a dark-adapted on-center cell to an instantaneous single photon flash would last 60 ms. This value includes flash-to-flash variability in the intraretinal conduction time and so should be longer than the effect of a single photon on a ganglion cell. It is thus in reasonable agreement with the duration of the effect of slow input events on ganglion cells, 40-50 ms.

In view of the above evidence, the slow input events will be referred to as quantal events in the analysis below; however, the validity of that analysis does not depend on whether the input events are in fact quantal events. No distinction will be made between the quantal events occurring in the dark and those due to effective photon captures. Indeed, no such distinction is justified for the great similarity between the correlograms obtained in total darkness and those obtained at the lowest backgrounds implies that the average effect of a dark-light event on a ganglion cell is similar in shape and duration to the effect of a photon capture.

**Retina as quantum detector**

Although an on X-cell fires an average of 2-3 spikes in response to a quantal event, a system analyzing the output of a single on X-cell would not be a very reliable detector of quantal events because of variability in the cell's response and the possibility that some of the cell's spikes are not caused by quantal events. However, any small region of the retina communicates with 2-4 overlapping ganglion cells of each type, on X, off X, on Y, and off Y (based on cell densities and dendritic-field sizes (9, 22, 39)). A system capable of analyzing the output of many of those cells together could fairly reliably detect a quantal event within that region: the system could detect almost all quantal events by using the signals from the individual on X-cells, which respond strongly when quantal events do occur, and it could avoid false reports of quantal events by requiring a coincident response from many of the cells, which occurs only when there is a quantal event. Thus, the output of the retina as a whole at very low light levels may contain a fairly reliable picture of the occurrence of every quantal event, whether due to dark light, background light, or a visual stimulus. In this view, the X- and Y-cells of the retina do not make any effort to distinguish the "signal" (a visual stimulus) from the "noise" (background and dark light) but merely transmit the signal with the noise as well as they can.

**Relative transience of X and Y responses to quantal events**

A cell's "response to quantal events" refers to the cell's change in firing probability for various times after the beginning of a quantal event's effect on the cell (e.g., the curves in Fig. 12A, B depict possible responses to quantal events). This response should not be con-
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A

\[ QE \rightarrow \text{On X} \]

Time after quanta\text{1 event (msec)}

0 50

B

\[ QE \rightarrow \text{On Y} \]

Time relative to X-cell spike (msec)

0 50

FIG. 12. Illustration of the way in which shapes of responses of two cells to quanta\text{1} events determine the shape of their cross-correlogram. See text for details. Cell A represents an idealized on X-cell, cell B an on Y-cell.

Information on the relative transience of X and Y responses to quanta\text{1} events can be obtained by interpreting the slow components in a cross-correlogram as a convolution of two responses. If two cells, A and B, respond to shared quantal events, the slow components in their correlogram should be proportional to the convolution of the time reversal of A's response with B's response (see APPENDIX, section I). The solid curve in Fig. 12E depicts such a convolution of the responses in Fig. 12A and 12B. Section II of the APPENDIX shows that if two cells have unequal slow components, the cell with the smaller slow component has the more transient response. This principle implies that the responses of the four cell types to quanta\text{1} events increase in transience in the same order as their slow components decrease in relative strength: on X-off X-on Y-off Y, as found in the RESULTS. In particular, the excitatory responses of on Y-cells are more transient than those of on X-cells and the inhibitory responses of off Y-cells are more transient than those of off X-cells.

A difference in transience for two on-center cells should, in fact, lead to a difference in slow component strengths for the following reasons. A's slow component reflects how often a spike from A due to a quanta\text{1} event occurs 5 ms or more after a spike from B due to the same quanta\text{1} event. This depends on how often A fires more than 5 ms after the beginning of a quanta\text{1} event (i.e., in the late portion of its response) and on how often B fires 5 ms or more before those spikes from A (i.e., in the early portion of its response). Similar factors govern B's slow component. If B's response is more transient than A's response, then it is relatively stronger early in the response and relatively weaker late in the response. Thus, A's slow component, which reflects the stronger firing of A late in A's response following the stronger firing of B early in B's response, should be stronger than B's slow component, which reflects the weaker firing of B late in B's response following the weaker firing of A early in A's response.

The difference in transience of on X and on Y responses to quanta\text{1} events can be investigated by asking, for a given on X response, how transient the on Y response must be to produce a difference in slow components equal to that observed in on X-on Y correlations. First consider the limiting case of Fig. 12 in which an on X-cell has a perfectly sustained response lasting 50 ms (Fig. 12A), while an on Y-cell has a transient response of the same duration (Fig. 12B). Because the X-cell's response is constant, the convolution of the two responses, and hence the slow components of the cells' correlogram, are simply proportional to integrals of the Y-cell's response over various times (see APPENDIX, section I). Specifically, the X-cell's slow component at some time \(-t\) is proportional to the integral of the portion of the Y-cell's response shaded in Fig. 12C, while the Y-cell's slow component at \(t\) is proportional to the integral of the portion shaded in Fig. 12L. In order for the on Y slow component at \(5-7\) ms to be half as large as the on X slow component at \(-5\) to \(-7\) ms, as...
was found in actual on X-on Y correlations, the integral in Fig. 12D (with $t = 5-7$ ms) must be half as large as the integral in Fig. 12C. This implies that the first 5–7 ms of the Y-cell’s response must contain slightly more than half of the total response.

Essentially the same conclusion was reached when convolutions assumed on X responses of other shapes, including more realistic shapes that resulted in slow components resembling those actually obtained. When fairly sustained on X responses were used, the on Y-cell had to have at least half of its response in the first 5–7 ms, as in Fig. 12B, to produce slow components differing by a factor of 2. When on X responses with an initial transient component were used, the on Y response had to be even more transient.

Since the actual on Y-cell response to quantal events appears to be at least as transient as that of Fig. 12B, it can be usefully described as having an initial transient component. The remainder of the response will be referred to as the sustained component, even though it may decay over time. Since the inhibitory responses of off Y-cells to quantal events are even more transient than on Y responses, they can also be described as having an initial transient inhibition followed by a sustained component.

The analysis that follows will consider the possible role of the active inputs described in the preceding paper (30) and of other inputs to ganglion cells in generating these transient and sustained components of responses to quantal events.

**On active inputs as source of initial transient**

The durations of the initial transients in Y-cell responses could be measured directly if correlograms consisted only of slow components (i.e., reflected only the convolution of the two cells’ responses). The duration of an initial transient would then equal the time when the Y-cell’s slow component completed its transition from an initially steep slope to a shallower slope that it maintained for the rest of its duration, as illustrated by the heavy arrow in Fig. 12E. However, the presence of fast components (the dashed peak in Fig. 12E) potentially obscures this transition point. In actual on X-on Y correlations (e.g., Fig. 2D), a shallow slope was already fully attained at the end of the fast component of the on Y-cell; thus it is possible to state only that the duration of the transient effect on the on Y-cell was no greater than that fast component’s duration. Similarly, the slow component fully attained a shallow slope by the end of the fast component for off Y-cells in correlations with on X and on Y-cells; thus, the duration of the transient inhibition of off Y-cells was no greater than that fast component’s duration. Now, the fast components in these correlations appeared continuously from 10 cd/m$^2$ to darkness and changed little in duration over this range of backgrounds (e.g., Fig. 1). These facts indicate that the fast components were caused by the active inputs identified in the preceding paper (30) as the source of correlations at 10 cd/m$^2$. That paper concluded that the duration of the on Y-cell sides of peaks in on-on correlations reflected the duration of the excitation of on Y-cells by on active inputs, while the duration of the off Y-cell sides of wells in on-off correlations reflected the duration of the inhibition of off Y-cells by on active inputs. Applying that conclusion to the fast components at low backgrounds, the durations of the initial transient effects on on Y- and off Y-cells are at least as brief as the durations of the excitatory and inhibitory effects of on active inputs, respectively. Thus, there is a mechanism transiently excitatory to on Y-cells and a mechanism transiently inhibitory to off Y-cells, each of which must satisfy rather stringent limits on duration (typically 5–7 and 6–8 ms, respectively). Since on active inputs alone can easily serve both of these functions within these limits, they appear to be a likely source of the initial transients of Y-cell responses (as a final intermediary if not as the origin of the effect). This conclusion is intrinsically reasonable: as a major excitatory input to on Y-cells, the on active inputs would be expected to be excited by quantal events and to play a major role in the response of the on Y-cell to such events.

**Evidence for a sustained effect**

Could the on active inputs be the sole mediator of the effects of quantal events? If they were, then the response to quantal events for each cell type would tend to have the same shape as the response of on active
inputs, which would be inconsistent with the large differences in response shapes for the four cell types implied by differences in slow component strengths. Thus, the effects of quantal events must pass through inputs other than or in addition to on active inputs.

The relative strength of the fast and slow components in correlations between on X-cells provides evidence on the existence and nature of such inputs. Computer simulations described in the APPENDIX (section III) show that if shared active inputs were the sole pathway for the common effects of quantal events on two on X-cells, then those inputs would often fire the cells nearly simultaneously, producing much stronger fast components, relative to the slow components, than were observed. This finding indicates that on X-cells receive other inputs that (unlike the active inputs) contribute more to the slow than to the fast components of their correlogram. In order to do so, those inputs must contribute to the sustained portion of the X-cell's response rather than being confined to the first few milliseconds of the quantal event. Moreover, those inputs must not provide brief, strong, spikelike input events, like the active input events, since that would often fire the X-cells nearly simultaneously and contribute strongly to the central peak. The latter requirement would be satisfied by inputs operating by graded potentials.

In the APPENDIX (section IV), the sustained portion of the on X response to quantal events is estimated to be at least 2.3 times stronger than that of the on Y response. Since the above considerations indicate that inputs other than active inputs cause the bulk of these sustained responses, this estimate suggests that those other inputs have an effect at least 2.3 times stronger on X-cells than on Y-cells. This difference appears to be the primary source of the difference in transience of on X and on Y responses.

Possible sustained inputs inhibitory to off X-cells

As seen above, the inhibition of off Y-cells by quantal events is more transient than that of off X-cells. This difference quite probably reflects a stronger sustained inhibition of off X-cells, rather than a weaker initial transient inhibition, for three reasons. 1) The breaks in interval histograms, pauses in maintained discharge, and autocorrelogram overshoots were all more apparent for off X-cells than for off Y-cells. These differences indicate that off X-cells are more strongly inhibited for an extended period of time by quantal events, producing a more prominent population of long interspike intervals. 2) It was seen above that a likely source of the initial transient inhibition is the on active inputs, and the preceding paper indicates that these inputs should be strongly inhibitory to both off X- and off Y-cells. 3) Slow components for on Y-cells were about 10% stronger in on Y-off X than in on Y-off Y correlations. The strength of on Y slow components depends on the following: a) the fraction of the quantal events excitatory to the on Y-cell that also inhibit the off-center cell, which should depend on the area of overlap of the cells and be smaller for on Y-off X pairs; b) the strength of the later (sustained) portion of the on Y response, which should be the same in the two cases; and c) the strength of the early portion of the off-center cell's response, which is dominated by the strength of the initial transient portion of the inhibition. The last factor must be greater for off X-cells in order to counteract the contribution of the first factor; thus it is unlikely that the initial transient is any weaker for off X-cells.

If the initial transient inhibition is essentially the same for off X- and off Y-cells, as just argued, then simulations can be used to assess the difference in the sustained inhibition of the two cell types. These simulations, described in the APPENDIX (section V), indicate that the sustained inhibition is about 3 times stronger for off X- than for off Y-cells during at least some portion of the inhibitory effect. Quantal events could inhibit off-center cells by exciting on active inputs that inhibit off-center cells or by inhibiting off active inputs that would otherwise excite the off-center cells. However, it seems unlikely that either mechanism would lead to a difference of a factor of 3 in the sustained inhibition by quantal events, since both X- and Y-cells appear to receive input of roughly comparable magnitude from active inputs (30). This suggests that there are inputs other than active inputs that contribute to the sustained inhibition of off X-cells by quantal events and have about a 3 times stronger effect on off X- than on off Y-cells.
One final point enters into the model presented next. Calculations presented in the APPENDIX (section VI) show that sources other than off active inputs account for about 50% of the maintained activity of off X-cells in the dark but only a third as much for off Y-cells.

**Model and retinal circuitry**

Figure 13 presents the model suggested by this discussion. The curves in Fig. 13 depict the responses of ganglion cells to quanta1 events and the average effects transmitted along the various pathways, based on computer simulations of the model using plausible parameters. Those simulations reproduced most of the essential features of the observed cross-correlograms in Figs. 2, 3, and 4; thus, the ganglion cell responses shown do accurately reflect the degree to which Y-cell responses are more transient than X-cell responses.

In the model of Fig. 13, quantal events have a predominantly transient, excitatory effect on on active inputs, which leads to a strong transient excitation of on-center cells and inhibition of off-center cells. Quantal events also have two relatively sustained effects that do not pass through active inputs: an excitatory effect that is considerably stronger for on X- than for on Y-cells and an inhibitory effect that is considerably stronger for off X- than for off Y-cells. As a result, the responses of the X-cells are substantially sustained, while those of the Y-cells are rather transient. Off active inputs have a high level of maintained activity, which causes most of the maintained firing of off Y-cells in the dark. The remainder of the maintained activity of off-center cells is attributed to a "bias" signal that is considerably stronger for off X-cells. The same signal may be responsible for the maintained activity of off active inputs. Both the bias signal and the sustained inhibition by quantal events are stronger for off X-cells, suggesting that the sustained inhibition may operate by inhibiting a tonic excitatory signal.

Three findings in the cat retina suggest an anatomical basis for the model. 1) Kolb (26) has reconstructed the synaptic input to three ganglion cells: type Iia (off X) with 30% amacrine input and 70% input from cone bipolaris branching in sublamina a of the inner plexiform layer; type IIB (on X) with 30% amacrine input and 70% input from cone bipolaris branching in sublamina b; and type Ib (on Y) with 80% amacrine input and 20% input from sublamina b cone bipolaris. 2) Cone bipolar cells branching in either sublamina contain rod signals comparable in magnitude to signals that arise from cone activity (35). 3) Rod bipolar cells do not contact ganglion cells (26, 27). These facts suggest that 1) sublamina b cone bipolar are the final pathway for the relatively sustained excitation of on-center cells by quantal events in the rods, and sublamina a cone bipolar are the final pathway for the sustained inhibition of off-center cells by quantal events. Thus, these sustained effects appear to be 2.5–3 times stronger for X-cells than for Y-cells. 2) At high backgrounds, cone bipolar making excitatory contacts onto ganglion cells provide a tonic input that is the major source of ganglion cell maintained activity aside from active inputs. This mechanism would explain why sources other than active inputs account for the large majority (about 85%) of X-cell maintained activity and only about a quarter as much (roughly 20%) of Y-
cell activity at 10 cd/m² (30). 3) At low backgrounds, at least some of the sublamina a cone bipolars are depolarized in the absence of quantal events and provide tonic excitation of off-center cells. Thus, sources other than active inputs account for about 50% of off X-cell activity in the dark and less than a third as much off Y-cell activity.

Two pathways from rods to cone bipolars have been identified. First, rod signals appear in the cones themselves (34), presumably via gap junctions between cone processes and rod spherules (25). Second, one principal output of rod bipolars is to the depolarizing AII, or rod, amacrines, which have extensive gap junctions with sublamina b cone bipolars and synapses onto sublamina a cone bipolars as well as onto off-center ganglion cell dendrites (18, 26, 27). Three points suggest which of these two pathways might be the primary one for the transmission of quantal events: 1) the AII amacrines show a transient response of about 70 ms to a strong stimulus (36), which is close to the 50-ms duration of the slow input events described here; 2) the autocorrelograms of most X-cells indicate that the cells received not only the 50-ms events but also weak input events lasting several hundred milliseconds; and 3) it seems likely that the latter duration, several hundred milliseconds, rather than the 50-ms duration, more closely reflects the duration of quantal events as they occur in rods (e.g., quantal events in toad rods last about 5 s (7)). These points suggest that the 50-ms slow input events but also weak input events lasting several hundred milliseconds, rather than the 50-ms duration, more closely reflects the duration of quantal events as they occur in rods (e.g., quantal events in toad rods last about 5 s (7)).

Distinctive properties of Y-cells

Y-cells show a number of response properties that appear to be due to inputs that drive Y-cells more effectively than X-cells. These “Y-like” properties include nonlinear spatial summation, the periphery effect, on-off surround responses, and strong responses to large fast-moving targets that can stimulate the surround (10, 15, 41). The stimuli eliciting these properties seem to cause a 2.5–5 times stronger effect on Y-cells than on X-
cells (1, 10, 12, 19, 20, 23, 32, 41, and unpublished observations). When one property, an unmodulated periphery effect, is elicited to its fullest extent, other Y-like properties become harder to elicit (28), suggesting that signals for a number of Y-like properties may at some point pass through a single pathway to Y-cells. Could active inputs, which are a major source of excitatory input to Y-cells (30), provide a final common pathway for some or all of the Y-like properties, even though they also drive X-cells? The stimuli eliciting Y-like properties either directly generate signals in an area that includes the entire receptive-field center of a Y-cell or must involve lateral conduction of signals from their origin some distance from a Y-cell; this lateral conduction would be expected to spread signals evenly and present those signals to the entire dendritic field of the Y-cell. In either case, stimuli using the active inputs as a final pathway would activate those inputs throughout a Y-cell field and a Y-cell would be considerably more excited than an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell (as estimated in the preceding paper (30) for active inputs during maintained activity at 10 cd/m²) and could cause up to 5 times more activity if the elevated mean firing rate of the Y-cell, typically found with these stimuli, increased the effectiveness of the inputs to the Y-cell. Thus, a stimulus using active inputs as a final pathway would activate those inputs throughout a Y-cell field and a Y-cell would be considerably more excited than an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs. Such an activation of an entire field of inputs would cause at least 3 times more activity in a Y-cell than in an X-cell, which, being smaller, has access to many fewer active inputs.

APPENDIX

This APPENDIX presents further justification for some points made in the DISCUSSION.

I. Relation between cross-correlogram and convolution of responses

For two cells, A and B, let $T =$ total duration of a single input event (IE), $R_a(s), R_b(s) =$ A or B's response (change in firing rate) at time $s$ after the beginning of an IE. These are 0 for $s < 0$ and $s > T$. $M_a, M_b =$ overall mean firing rates of the two cells, A and B. $M_i =$ mean rate of IEs. $C(t) =$ cross-correlogram of A and B at time $t$. Assuming that the effects of several IEs combine by simple addition and that IEs occur independently of each other and form a Poisson process, it can be proved that

$$C(t) = M_b + M_i \int_0^t R_a(s) \cdot R_b(s + t) \cdot ds \quad (A1)$$

The integral in equation $A1$ can also be written

$$\int_0^t R_a(-s) \cdot R_b(t - s) \cdot ds \quad (A2)$$

which is the convolution of $R_a$ with the time reversal of $R_b$. Thus, the base line of the correlogram ($|t| > T$) is the mean firing rate of B; deviations from the base line are proportional to the convolution of the time reversal of the input’s effect on A with the input’s effect on B.

For two cells excited by a common input, it is possible to explain why their correlogram should be a convolution of the average responses of the two cells. A count occurs in the peak of the correlogram when an IE succeeds in firing both A and B. Given that A and B both fire due to an IE, the probability that A fires at time $s$ in the IE is proportional to $R_a(s)$. The probability that B fires at time $t$ after that A spike is proportional to $R_b(s + t)$. Thus the probability that A fires at time $s$ in the IE and that B fires at time $t$ after that, given that both fire due to an IE, is proportional to $R_a(s) \cdot R_b(s + t)$. The overall probability of B firing at time $t$ after A, given that both fire due to an IE, is the sum over all the times that A can fire during the IE and is thus proportional to the integral in equation $A1$. However, it is more difficult to establish this point for inhibitory as well as excitatory responses.

II. Relation between transience of responses and centroids of correlograms

A response is termed transient if a disproportionately high fraction of the response occurs early in the response. The degree of transience may be quantified by the difference between the fraction of properties, it is possible that these nonlinear subunits are the active inputs.
of the total response obtained by some given time \( t \) and the fraction of time elapsed

\[
\frac{\int_0^t R(s) \, ds}{\int_0^T R(s) \, ds} = \frac{t}{T} \quad (A3)
\]

The average of this quantity for all times \( t \) would give an overall measure of transience \( D_R \)

\[
D_R = \frac{1}{T} \int_0^T \left( \frac{\int_0^t R(s) \, ds}{\int_0^T R(s) \, ds} - \frac{t}{T} \right) dt \quad (A4)
\]

Transforming equation \( A4 \) with an integration by parts gives

\[
D_R = 0.5 - \frac{1}{T} \int_0^T \frac{t \cdot R(t) \, dt}{\int_0^T R(t) \, dt} = 0.5 - \frac{\bar{t}_R}{T} \quad (A5)
\]

where \( \bar{t}_R \) is the centroid of the function \( R \). Thus the overall transience of the response is simply related to its centroid by equation \( A5 \).

It can be shown that the centroid of a convolution of two functions is the sum of the centroids of the two functions. Thus, for \( A \) and \( B \) affected by a common input, the centroid of the part of their correlogram deviating from the base line is the centroid of \( R_A \) plus the centroid of the time reversal of \( R_B \), i.e., the centroid of \( R_B \) minus the centroid of \( R_A \).

For correlograms at low light levels, the evidence indicated that the slow components reflect the common response of the two cells to quantaI events. The deviation of the slow components from the base line should then be proportional to the convolution of the two responses given in equation \( A2 \). If the slow component of \( A \) is larger than that of \( B \), this imbalance indicates that the centroid of that convolution has a negative value; i.e., it is located on the side of the correlogram with the larger slow component. In that case, the centroid of \( A \)'s response to quantaI events must be larger than that of \( B \)'s response and, by equation \( A5 \), the transience of \( A \)'s response must be less than that of \( B \)'s response. In other words, in a correlogram with unequal slow components, the cell with the smaller slow component has the more transient response to quantaI events.

There is some uncertainty in the location of the centroid of the convolution for the correlograms presented here because the central part of the convolution is masked by the fast components. However, the fact that this central part is generally masked by a fairly symmetrical peak or well indicates that the masked part of the convolution either is fairly symmetrical itself or is imbalanced in the same way as the slow components are (e.g., Fig. 12E). Thus, an imbalance in the visible part of the convolution (the slow components) indicates accurately whether the centroid is located to the left or right of zero.

III. Simulation of excitatory effects passing solely through active inputs

Computer simulations were used to determine how the correlation between two on \( X \)-cells would appear if the effects of their shared quantaI effects passed solely through their shared on active inputs. Simulations used the algorithms described in Appendix I of the preceding paper (30). In addition, one input (representing the quantaI events) could have a specified effect on two other inputs (on and off active inputs) as well as on the ganglion cells. In the simulated correlograms, the ratio of the fast to slow component strengths was greater for more transient active input responses to quantaI events and for higher active input effectiveness in firing the \( X \)-cells. To obtain a ratio of fast to slow components as low as that of the on \( X \)-on \( X \) pair of Fig. 1, the active input responses had to be made perfectly sustained and the active input effectiveness had to be set at 0.25. At 10 cd/m², effectiveness is at least 0.69 (30). Since strength of correlation depends on the product of the effectiveness of active inputs in firing the two cells, a decrease in effectiveness from 0.69 to 0.25 would tend to decrease the strength of the fast peak (the correlation due to active inputs) by a factor of 7.6. In reality, correlations more than doubled in strength as background was reduced below 10 cd/m² (see discussion of Fig. 1), indicating that effectiveness could not have decreased greatly. Thus it is quite unlikely that the effects of quantaI events pass solely through active inputs.

IV. Difference in sustained responses of on \( X \)- and on \( Y \)-cells

A minimum estimate of the difference in the sustained portions of the on \( X \)- and on \( Y \)-cell's responses to quantaI events is obtained as follows: 1) The mean magnitude of autocorrelogram overshoots suggests that the total responses of \( X \)- and \( Y \)-cells are 2.3 and 1.7 spikes per quantaI event, respectively. 2) The transient portion of the on \( Y \)-cell's response is taken to be 1.0 spike. If it were significantly higher (as much as 1.1 spike), the \( Y \)-cell would often fire twice within 5 ms and its autocorrelogram would contain a much stronger sharp peak in the first 5 ms than is ever observed. If it were lower, the estimated difference in \( X \) and \( Y \) sustained responses would turn out higher. 3) The \( Y \)-cell sustained response is then 1.7 - 1.0 = 0.7 spikes. 4) Simulations indicate that the on \( X \)-cell must then have transient and sustained responses of 0.7 and 1.6 spikes, respectively, in order to achieve the appropriate disparity of slow com-
ponents in an on X-on Y correlation. 5) Thus the sustained responses differ by at least a factor of $1.6/0.7 \approx 2.3$.

V. Difference in sustained inhibition of off X- and off Y-cells

The difference in the degree of sustained inhibition of off X- and off Y-cells was assessed with simulations. Off Y- and off X-cells were assumed to be inhibited totally for the first 8 and 10 ms, respectively, of a quantal event to reflect the conclusions in the DISCUSSION that the two cells do not differ much in their initial transient inhibition and that on active inputs may cause this inhibition. The strength of off X sustained inhibition was adjusted so that two off X-cells receiving common inhibitory events at 4/s would have slow components 9% above the base line (as in Fig. 3A). The strength of off Y sustained inhibition was then adjusted so that an off X-off Y correlation would have slow components differing by a factor of 1.9, as found in the RESULTS. Since a single X-cell receives quantal events at only 2-4/s in the dark (see RESULTS), 4/s is an overestimate of the rate of common inhibition to two off X-cells; thus this method underestimates both the strength of off X sustained inhibition and the difference in X and Y inhibition. When the sustained inhibition was assumed to have the same shape for the two cells, it had to be about 3 times weaker for the off Y-cell than for the off X-cell. When the inhibition of the off Y-cell was assumed to last only 30 ms after a quantal event, as suggested by the duration of off Y slow components, the early portion of the off X sustained inhibition was still at least 2 times stronger than that of the off Y-cell, while the inhibition of the off X-cell after 30 ms was infinitely stronger than that of the off Y-cell.

VI. Fraction of off X- and off Y-cell maintained activity not due to off active inputs in dark

This fraction is estimated as follows. Let $S_x$ and $S_y$ be the fraction of off X and off Y spikes due to off active inputs, $M_x$ and $M_y$ be the mean firing rates of off X- and off Y-cells, $I_x$ and $I_y$ be the rate of off active input events affecting the X- and Y-cells, and $E_x$ and $E_y$ be the average effectiveness of those inputs in firing the X- and Y-cells, then

$$S_x = I_x \times E_x / M_x$$
$$S_y = I_y \times E_y / M_y$$

Thus $S_y/S_x$ is at least 4.6 at 10 cd/m² (30). Its value in the dark depends on how each of the terms in equation A6 changes on going into the dark. The ratio of input rates, $I_x/I_y$, depends on the size of X- and Y-cells and the spatial extent of the output of off active inputs; it should be constant. The ratio of mean rates, $M_x/M_y$, decreases from about 1.9 to about 0.85. The ratio of effectivenesses, $E_y/E_x$, probably increases since the Y-cell mean rate increases so much more than the X-cell rate. Thus $S_y/S_x$ should be at least 2.0 in the dark. $S_x$ can be estimated from the depth of central wells in on Y-off X correlations, which changed from about 25% at 10 cd/m² to about 60% at 0.01 cd/m² and lower backgrounds. This depth depends on the fraction of Y-cell spikes due to on active inputs inhibitory to the off X-cell, which cannot increase much because it is limited by the relative size of X- and Y-cells, and on the fraction of X-cell spikes due to off active inputs inhibitory to the Y-cell, which should equal $S_x$. The methods of the preceding paper yield the conclusion that the increase in the depth of the well implies an increase in $S_x$ from 15% at 10 cd/m² to at least 40% in the dark. Thus $S_x$ is at least 80% in the dark, and the fraction of spikes not due to off active inputs is at least 3 times greater for off X- than for off Y-cells.

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