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SELF-FACILITATION OF GANGLION CELLS IN THE RETINA OF THE TURTLE

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SUMMARY

1. Ganglion cells responses to illumination and to optic nerve stimulation were recorded intracellularly from the retina of the turtle. All ganglion cells were identified by their antidromic responses to optic nerve stimulation.

2. When solitary spikes are produced following antidromic, orthodromic or intracellular stimulation, about 20% of the recorded ganglion cells show an additional depolarization along the falling phase of the action potential (post-spike depolarization, PSD).

3. The PSD following the antidromic action potential disappears upon collision with a direct spike or when the antidromic spike is prevented from invading the cell soma.

4. By pairing two optic nerve stimuli the PSD is depressed with brief interstimulus intervals, but gradually recovers to the control amplitude 600–800 msec after the conditioning shock.

5. The PSD is tentatively interpreted as an e.p.s.p. transmitted by ganglion cell collaterals originating at the level of the soma dendritic complex of the recorded cell.

6. The interspike interval histogram of ganglion cells showing PSD is characterized by a peak at about 10 msec, as opposed to a peak between 12 and 100 msec observed in cells without PSD. It is suggested that the occurrence of PSD facilitate the onset of additional action potentials at brief interspikes intervals, thus potentiating ganglion cell discharges.

INTRODUCTION

In the vertebrate retina, ganglion cells receive afferent connexions from bipolars and amacrine cells (Cajal, 1892; see for ref. Stell, 1972; Rodieck, 1973). Additional inputs may originate from the associational cells of Marenghi (1900) which, however, are rare (Gallego & Cruz, 1965), and from

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centrifugal fibres, whose terminations on ganglion cells (Cajal, 1889; Maturana & Frenk, 1965) has not been confirmed by electron microscopy (see for ref. Cowan, 1970). Because of their well established anatomical connexions, ganglion cells may be assigned the role of 'final common pathways' which integrate and channel to the brain their afferent inputs. In this respect, retinal ganglion cells differ from the majority of neurones in the central nervous system in that the latter possess recurrent collaterals feeding back signals which may influence either directly, or through interneurones, the cell output (Eccles, 1964). With regard to the retinal ganglion cells, there are no anatomical reports indicating the existence of reverberatory collaterals, and the few studies with intracellular recording from these cells have not dealt with the problem.

During the course of a systematic analysis of intracellular activity of ganglion cells, all of which were identified by their antidromic activation by optic nerve stimulation, we have recorded depolarizing waves which may be interpreted as positive feed-back possibly transmitted by reverberatory collaterals. Functionally, the occurrence of these depolarizing potentials facilitates ganglion cells photoresponses, by giving rise to additional firing at short interspike intervals.

METHODS

Preparation and recording

The experiments were performed in retinae of the turtle *Pseudemys scripta elegans*. The dissection of the preparation and the experimental arrangement used for recording has been described in a previous paper (Marchiafava, 1976). Recordings during the experiments were stored on magnetic tape for subsequent analysis. As the frequency cut-off of the tape was -3 db at 700 Hz, the height of the action potentials reproduced below is reduced by 15–20%. The electronics and optical stimulator were similar to those described by Lasansky & Marchiafava (1974).

Data analysis

The spike density of ganglion cells discharge was measured with a Time Histogram Analyzer, Model 4620/4621, made by ORTEC (U.S.A.). By setting the programme at 'A Interval Mode' the time intervals between successive spikes were analysed. The longest interval that could be measured by such procedure was 254 times the Time Base, that is the time interval chosen for the sampling. Each memory address had a capacity of 255 counts. The time base used varied from 2 to 5 msec. Longer time bases were used to analyse intervals distribution over intervals greater than 1 sec.

The Time Histogram Analyzer was connected to an X–Y type recorder to plot the accumulated data or, alternatively, to an oscilloscope where the visual display was photographed with a polaroid camera for subsequent analysis.

RESULTS

The responses of a ganglion cell to flashes of different intensities in the shape of a small circle (120 μm radius) or an annulus (225 and 800 μm inner and outer radius, respectively) centred on the recording microelectrode are shown in Fig. 1. The photoresponse to the lowest light intensity is a transient depolarization at the end of central illumination ('off'- centre) or at the onset of peripheral illumination ('on'-periphery) (Fig. 1, first line, left and right, respectively). With increasing intensities

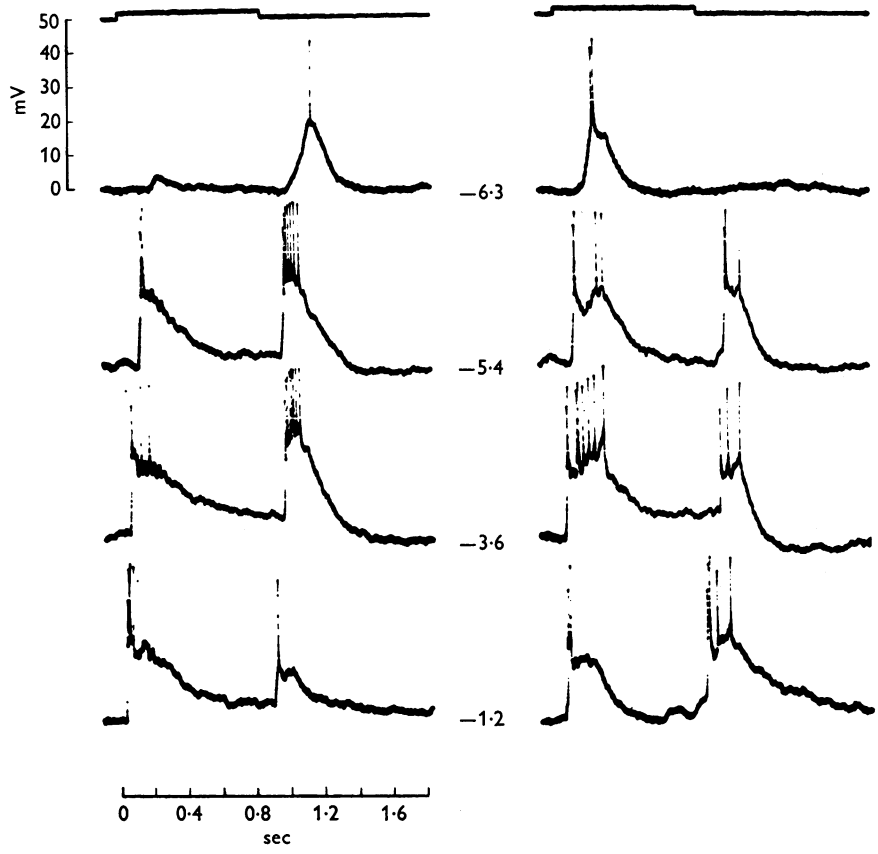

Fig. 1. Intracellular recordings of ganglion cell responses to illumination in the form of a circle (left column) and of an annulus (right column). The steps of light (920 msec duration) are indicated on the upper line. Photoresponses on the same line are evoked by light intensity represented by logarithmic units of attenuation, at the middle of each line. Note the different time course of the responses to central ('off'-type) and to peripheral illumination ('on'-type) with the lower intensity (−6·3). With increasing light intensity both stimuli produce 'on-off' type response.

the cell gives mixed 'on-off' responses to both stimuli which are superimposed on a small, sustained depolarization (< 10 mV) lasting the duration of illumination. This cell was identified as a ganglion cell because it produced an antidromic action potential after single shock stimulation

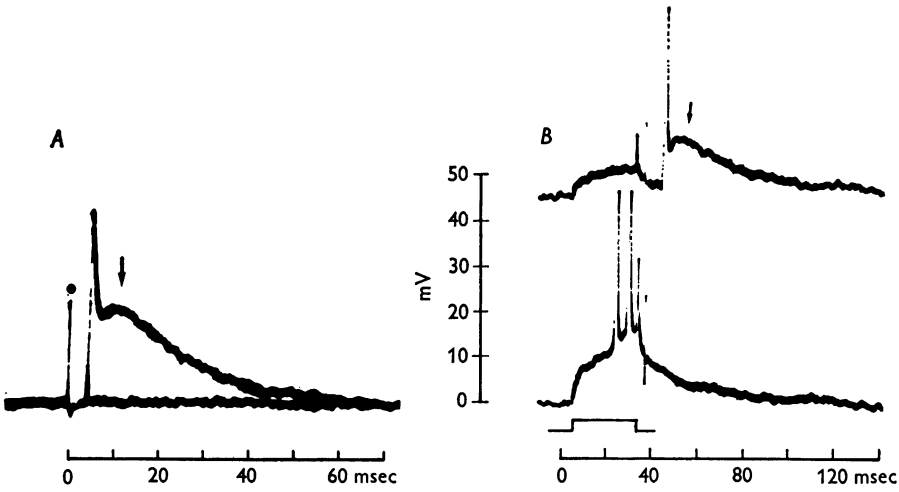

Fig. 2. Ganglion cell responses to stimulation of the optic nerve and to direct stimulation. *A*, the single shock stimulation of the optic nerve (0·1 msec, 2·5 mA) starts at time zero. The stimulus artifact is indicated by a filled circle. The ganglion cell response consists of an antidromic action potential followed by a prolonged depolarizing wave (post-spike depolarization, PSD) marked by an arrow. The latency of the spike is about 4 msec, corresponding to a conduction velocity of approximately 1 m/sec. Two superimposed traces. The flat trace represents a failure of the stimulus at threshold intensity (see text). *B*, the antidromic spike and the PSD (upper line, indicated by the arrow) disappear upon collision with direct spikes (second line). The lower line indicates duration of current injection. The PSD following the second, direct spike shown on the second line is depressed by refractoriness (see text and Fig. 4*B* and *D*). Similar PSD depression is observed in Fig. 3*B*.

of the optic nerve (0·1 msec, 2·5 mA) (Fig. 2*A*). The antidromic nature of the spike is demonstrated by the collision with a direct action potential (Fig. 2*B*) (Marchiafava, 1976). An important observation is that the falling phase of the antidromic spike is followed by a depolarizing deflexion which decays in about 50 msec (Fig. 2*A*). This additional, post-spike depolarization (PSD), however does not seem to be a general property of the ganglion cells because it was observed in only about 20 % of the successfully penetrated ganglion cells.

These late responses might be thought to arise from coactivation, at the level of the optic nerve, of some centrifugal fibres, which are known to

produce e.p.s.p.s in both amacrine and ganglion cells (Marchiafava, 1976). This hypothesis, however, is not consistent with the observation that the PSD is abolished along with the antidromic spike in collision experiments (Fig. 2*B*). In addition the threshold for evoking both the antidromic spike and the PSD was always the same, and the PSD was never obtained in the absence of a spike.

On the basis of these results, the hypothesis may be advanced that the PSD is dependent on a prior action potential in the impaled ganglion cell itself. This idea was supported by intracellular injection of extrinsic current. In this condition excitation is limited to the recorded cell, and both the cell spike and the PSD might be expected to arise together. Fig. 3*A* shows a spike which falls just at the end of a brief pulse of depolarizing current at threshold intensity. The direct action potential was systematically followed by a prolonged depolarizing tail with a time course comparable to the PSD shown in Fig. 2*A*. A more prolonged artificial depolarization of the ganglion cell at threshold always produced two action potentials (Fig. 3*B*). Here the second spike might have originated from the PSD, subsequent to the first spike, which was brought to threshold by the artificial depolarization. Furthermore, a slow depolarizing after-potential accompanied also the spike induced by post-anodic excitation following a pulse of strong hyperpolarizing current (Fig. 3*C*, first line). At times these after-potentials, which may be identified as PSDs, developed into a full spike (Fig. 3*C*, second line).

The data indicate, therefore, that the depolarizing after-potential is uniquely dependent on an immediately antecedent activation of the impaled cell. A possible mechanism for these responses could be recurrent collaterals that originate and terminate upon the same ganglion cell, operating a positive feed-back in the form of an e.p.s.p.

Further support for this conclusion is the fact that the PSD following the antidromic spike is depressed by a conditioning shock to the optic nerve. Fig. 4*A* shows a series of ganglion cell responses to a threshold, double electric shock applied to the optic nerve. When the first shock failed to elicit a response, the second shock produced an antidromic spike followed by the PSD (Fig. 4*A*, first line). However, when both shocks were effective, the PSD after the second shock was decreased (Fig. 4*A*, second line). The effect of the conditioning shock on the test PSD is clearly seen when the first and second lines are superimposed (Fig. 4*A*, third line).

The time course of the PSD depression was studied by progressively decreasing the interval between the two optic nerve shocks. In Fig. 4*C* the amplitude of the PSD following the test shock is plotted against the interstimulus intervals. The experimental points were fitted by the

following equation:

$$V_{\Delta t} = 18 + \frac{5{\cdot}8}{1+\exp -\left(\frac{(T_T - T_c) - 0{\cdot}29}{0{\cdot}045}\right)},$$

and indicate that the half recovery time of the PSD is about 0·3 sec and full recovery occurs in about 1 sec.

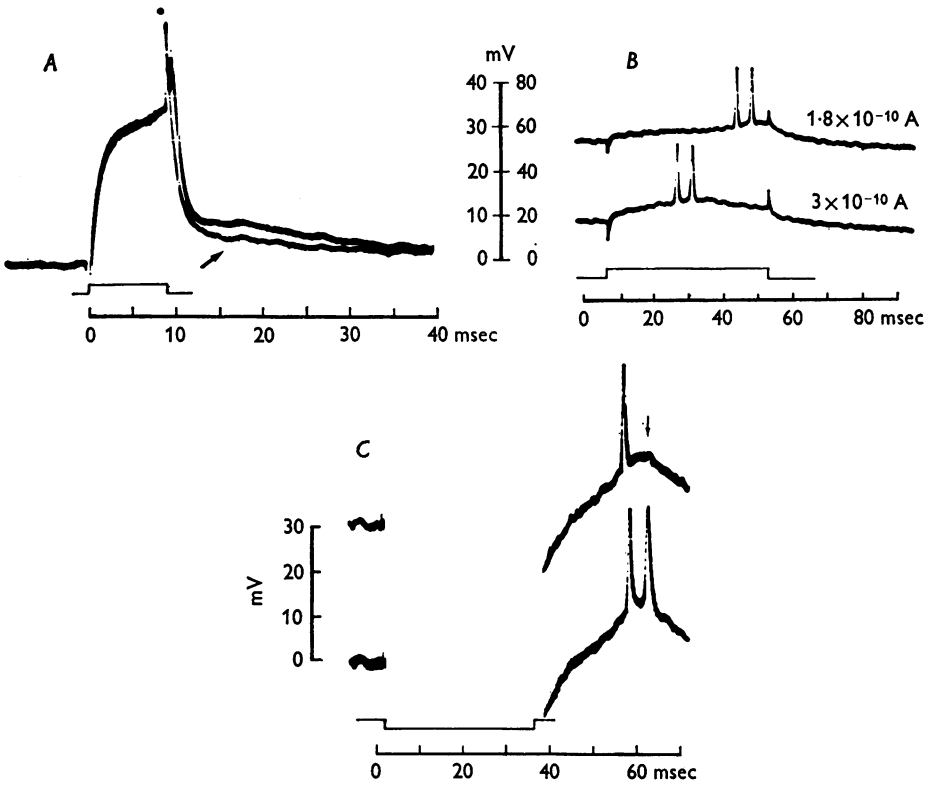

Fig. 3. Action potentials and PSD produced by direct stimulation of a ganglion cell. The current injection is indicated by the lower line on each photograph. *A*, three superimposed traces. A brief artificial depolarization, starting at time zero is followed by an action potential and a PSD. Current injection is at threshold for spike generation and in one trace (indicated by the arrow) the depolarization fails to induce both spike and PSD. The filled circle marks the artifact at the end of depolarization, followed by the spike. *B*, direct action potentials produced during prolonged, depolarizing steps of current of increasing intensity. The second spike may be the result of the PSD brought to threshold by the artificial depolarization. The current injected is indicated at the upper right. *C*, following a strong artificial hyperpolarization (the displaced membrane potential is not illustrated) a post-anodic depolarization develops into a single spike followed by a PSD, pointed by the arrow (upper trace), which may give rise to a second action potential (lower trace).

The graded modulation of the response amplitude and the recovery time following the conditioning shock suggest that the PSD is a synaptic event, as synaptic depression with similar time course is observed at the neuromuscular junction of amphibians, birds and mammals (for reference see Discussion).

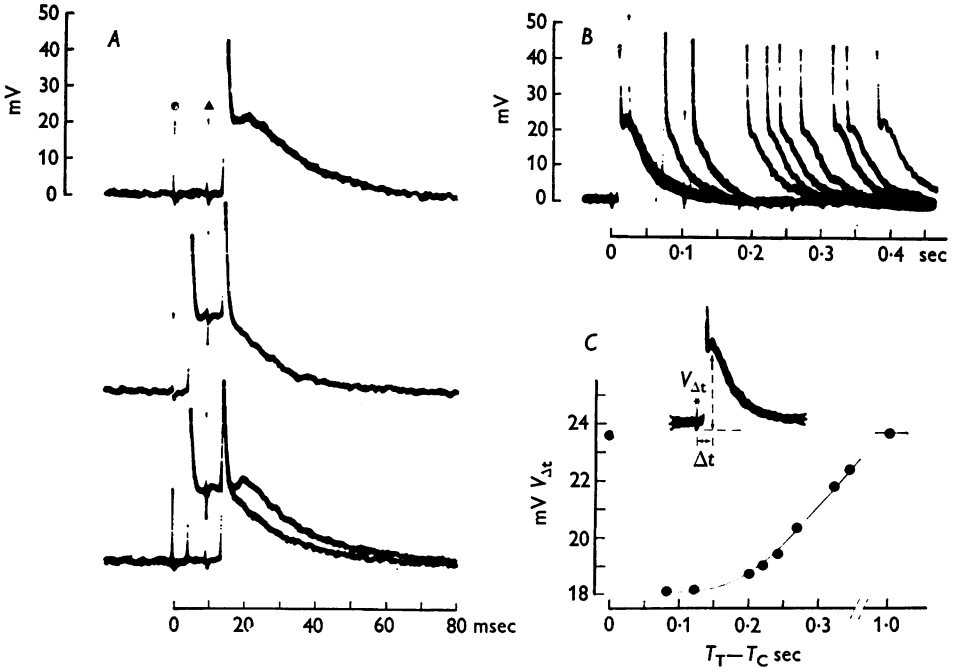

Fig. 4. Effect of a conditioning antidromic response on the amplitude of the PSD. *A*, ganglion cell responses to threshold, double stimulation of the optic nerve. The stimuli are indicated, on the first trace, by a filled circle and triangle. On the upper line, the first shock fails, but the second produces a response similar to that described in Fig. 2*A*. On the second line, both shocks produce antidromic action potentials; however the PSD following the second spike has got smaller. In the third line, the same responses are superimposed for comparison. *B* and *C*, time course and magnitude of PSD depression induced by progressively decreasing the interval between two optic nerve shocks. In *B*, responses with decreasing interstimulus intervals are superimposed. The response at the extreme right is obtained with 1 sec interval. In *C* plot of the PSD amplitude as a function of the interstimulus interval. $T_T - T_C$ = time difference between the test (T) and the conditioning (C) shocks. The insert shows how PSD is measured as $V_{\Delta t}$, where Δt is a fixed time interval from the nerve shock, corresponding to the PSD peak amplitude. The artifact of the nerve stimulation is indicated by the small asterisk. By this definition $V_{\Delta t}$ may also include some transient depolarization which constantly adds to PSD, but not dependent on it.

The possibility of finding an equilibrium potential of PSD close to that of excitatory synaptic potentials may provide further evidence in favour of the synaptic nature of PSD. The effect of injecting steps of depolarizing and hyperpolarizing current on the ganglion cell response to single shock

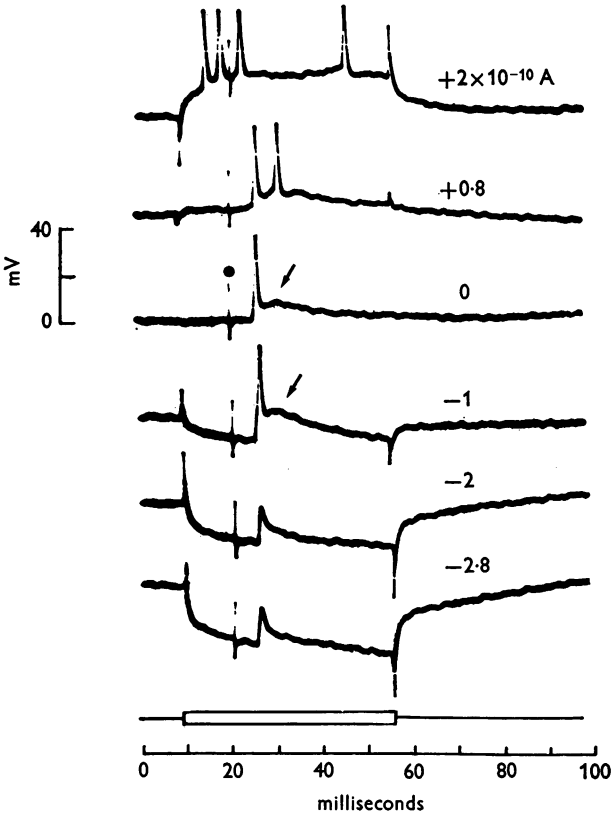

Fig. 5. The effect of artificially varying membrane potential on both antidromic spike and PSD. Direct injection of inward current (marked by the line at the bottom) reduces the antidromic spike, in an all-or-none fashion, to a smaller response which can be identified as an action potential blocked at some distance from the recording site (see text for further explanations). Note that the PSD (marked by the arrow on the third and fourth lines), disappears together with the highest portion of the spike. The artifact of the nerve stimulation is marked by the dot on the third line. The current injected is indicated above each line, at right.

stimulation of the optic nerve is shown in Fig. 5. During artificial hyperpolarization the PSD amplitude following the antidromic spike has almost doubled suggesting a conductance increase toward an ionic species with an equilibrium potential more positive than membrane resting potential.

Unfortunately, measurement of PSD amplitude is impossible during artificial depolarization because the latter initiates a burst of direct spikes which, *per se*, depresses the PSD (cf. Fig. 4) or even abolishes the antidromic spike by collision (Fig. 5, first line). With the data available the

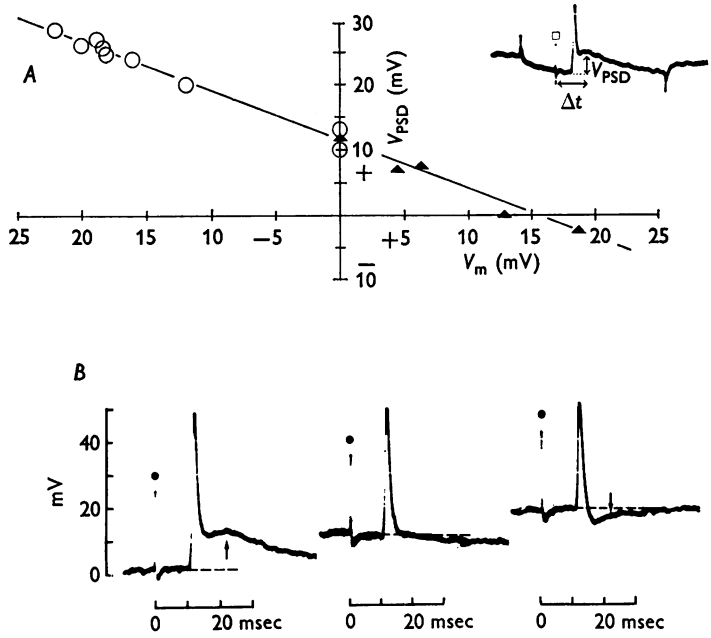

Fig. 6. The effects of varying membrane potential on PSD amplitude. *A*, diagram of PSD amplitude (see insert showing the same criterion used to measure V_{PSD} as in Fig. 4) *vs.* membrane potential. Zero in the abscissae represents the resting potential in darkness (V_m). Open circles represent V_{PSD} during hyperpolarization induced by intracellular current injection, as in Fig. 5. Filled triangles represent V_{PSD} during depolarization obtained as shown in *B*. All measurements were taken from the same ganglion cell. *B*, responses to single shock stimulation of the optic nerve recorded during progressive depolarization of the membrane, perhaps due to electrode damage. The antidromic spike apparently maintains the same characteristics but the PSD (pointed by the arrows) reverses at about 17 mV above resting potential in darkness.

PSD equilibrium potential can be tentatively indicated only by extrapolation as shown in Fig. 6*A*. The value thus obtained is not far from the equilibrium potential of end-plate currents (Takeuchi & Takeuchi, 1960). Further indication of the PSD reversal potential was obtained, in the same cell shown in Fig. 6*A*, at the end of the experiment when the cell was depolarized by damage caused by slight movement of the recording electrode. As shown in Fig. 6*B*, the antidromic spike maintained the same

absolute peak voltages, but the PSD reverses with membrane depolarization of about + 20 mV. The amplitudes of the PSD, measured at different levels of depolarization (Fig. 6*B*) are almost superimposable on the extrapolated line (Fig. 6*A*).

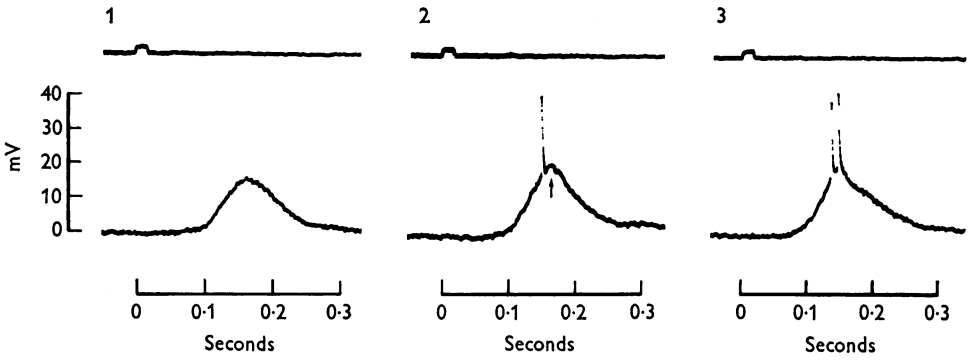

Fig. 7. Ganglion cell photoresponses to brief flashes of light (20 msec), indicated on the first line. The illumination was in the form of an annulus. The same light intensity (6·0 log units of attenuation) was used in 1, 2 and 3. Note that when a spike is produced, as in 2, this is followed by a PSD (see the small depolarizing wave marked by the arrow) which may develop into a full spike, as in 3.

It is interesting to define the site of origin of the postulated cell collateral(s), and the following test seems to provide an approximate indication. When the antidromic response is elicited during the simultaneous injection of a pulse of hyperpolarizing current, the action potential is reduced, in all-or-none fashion (Fig. 5, fifth line). The resulting response may be interpreted as an action potential blocked at some distance from the recording site (Eccles, 1955; Fuortes, Frank & Becker, 1957). This reduced response cannot be a chemically transmitted post-synaptic potential because it does not increase in amplitude with stronger hyperpolarizing currents (Fig. 5, lower line). It is further evident in Fig. 5 that, whenever the largest portion of the spike, i.e. the soma-dendritic component (Eccles, 1955; Fuortes *et al.* 1957) is abolished, the depolarizing after-potential also disappears. Thus the collateral(s) responsible for the PSD cannot originate at the level of the cell axon.

PSD may also occur after orthodromic excitation of ganglion cell. Fig. 7 shows three ganglion cell responses to peripheral illumination of threshold intensity for eliciting spikes. Whenever a spike occurred, it was followed by a PSD (Fig. 7, 2), which in turn could drive the membrane potential to the firing level giving rise to the late spike shown in Fig. 7, 3. Here, the time interval between the first and the second spike is about

10 msec, corresponding to the delay from the onset of the action potential to the peak of the PSD shown in Fig. 2*A*.

It is then possible that also in the case of photoresponses characterized by a more conspicuous firing, each of the component spikes, by virtue of the PSD they produce, may give rise to an additional action potential, at the observed interval of about 10 msec. If this were the case the intervals histogram of the discharge of cells producing PSD should reveal an increased probability of firing at about 10 msec interval, as compared to other ganglion cells where the membrane firing is directly dependent upon illumination alone. Interspike interval histograms were obtained during photoresponses from ganglion cells showing PSD and compared with those obtained from ganglion cells without PSD. The illuminated area was either a circle or an annulus. The following results apply equally to histograms of photoresponses obtained with either of these stimuli. Cells not showing PSD showed a peak probability of firing distributed along a wide range of intervals varying from 12 to 100 msec. Fig. 8*A* (first line) shows a typical interval distribution in a ganglion cell without PSD. Here the maximal probability of firing corresponds to intervals of about 25 msec. Ganglion cells without PSD may show widespread interspike interval distributions, with histogram peaks only slightly elevated above the rest of the curve, as shown in Fig. 8*A*, second line. Histograms of this form, as will be described below, were never obtained from ganglion cells with PSD.

The typical histogram obtained from ganglion cells with PSD shows that the highest probability of firing occurs at interspike intervals of about 10 msec (Fig. 8*B*). Here a great number of intervals are concentrated within a narrow range around the peak value, contributing to the characteristic pointed shape of the histograms, a frequent observation in cells with PSD.

The predominance of short intervals in cells with PSD with respect to cells without PSD cannot be attributed to light intensity, because the absolute values of light intensities used for both types of cells were alike. Furthermore, the histogram form of the cells with PSD does not change significantly by varying the intensity of illumination from 0·6 to 1·8 log units above threshold to the maximal intensity available (6·6 log units above threshold) producing saturated responses. The histograms of photoresponses to dim and bright illumination obtained from ganglion cells with PSD are shown for comparison in Fig. 8*D* and *E* (upper and lower lines, respectively). Similarly, ganglion cells without PSD showed no significant changes in the histogram form as a function of light intensity. These results contrast with the shortening of the interspike intervals obtained by Barlow & Levick (1969) in the cat by increasing the intensity

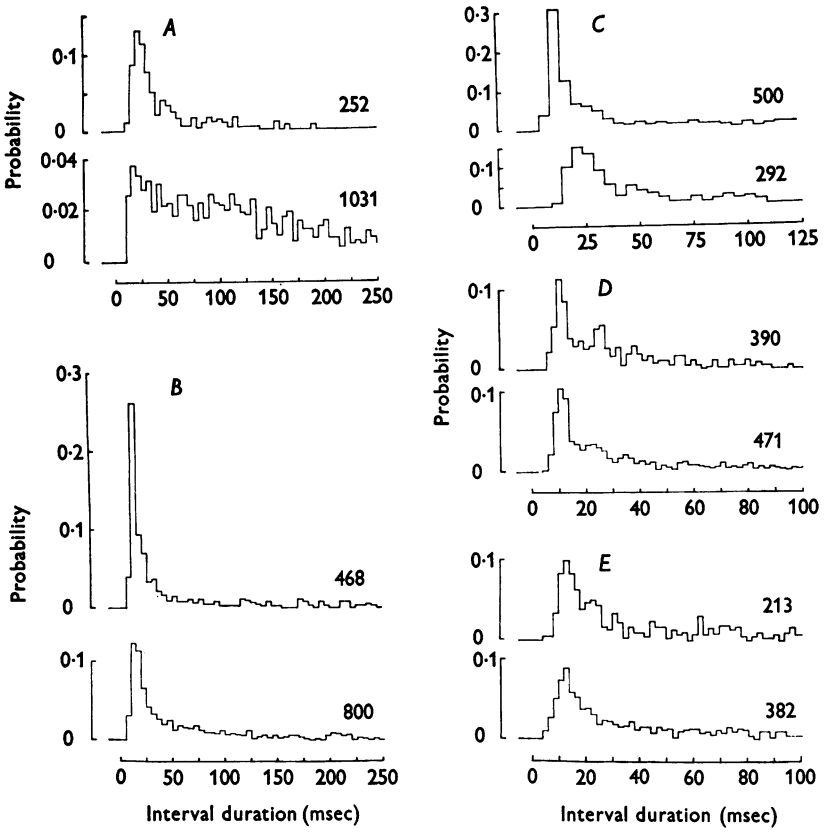

Fig. 8. Histogram of the interspike intervals obtained during ganglion cells photoresponses. *A*, histograms obtained from two ganglion cells, first and second line respectively, which did not show PSD. *B*, histograms from two ganglion cells, first and second line respectively, showing PSD. *C*, histograms from a cell with PSD, first line, and without PSD, second line, are shown for comparison on an expanded time scale. Note the increase in the relative probability of firing at shorter time intervals in the cell with PSD with respect to the other cell. *D* and *E*, histograms obtained from two ganglion cells with PSD during illumination at low and high intensity (first and second line, respectively). Low intensity refers to the range between threshold and 1·8 log units above threshold. High intensity spans from 2·1 log units above threshold to the maximal intensity, producing saturated responses (6·0 log units above threshold). In *A*, *B* and *C* histograms refer to photoresponses obtained throughout the whole range of intensities. The duration of a single bin was 5 msec in *A*, *B* and *C*, and 2 msec in *D* and *E*. The ordinates refer to the probability of occurrence of interspikes intervals. Probability values were computed by dividing the number of time intervals occurring in a given bin by the total number of intervals embodied in the histograms. The total number of intervals are indicated at the upper right of each histogram.

of illumination. It should be pointed out, however, that in the other work the stationary ganglion cells responses to maintained illumination were analysed, while in the present report on the turtle only transient ganglion cell photoresponses have been found. A possible interpretation of the absence of a characteristic 'low intensity' histogram in turtles is that in these ganglion cells, contrary to cats, there is no spontaneous activity. Thus, a low intensity illumination producing only subthreshold, graded depolarizing responses in turtle ganglion cells, would however be effective in producing at least some ganglion cells discharge in cats because it summates to the maintained background of spontaneous depolarization.

TABLE 1. Peak interspikes interval (t_{peak}) and probability of occurrence ($P_{t_{peak}}$) during ganglion cell photoresponses

Ganglion cells with PSD				Ganglion cells without PSD			
Cell	t_{peak} (msec)	$P_{t_{peak}}$	No. of inter-vals	Cell	t_{peak} (msec)	$P_{t_{peak}}$	No. of inter-vals
7-6 76/1	9–10	0·06	128	8-1-76	20–25	0·023	252
23-2-76/2	10–12	0·21	468	31-6-76/2	14–25	0·04	128
21-1-76/4	10–12	0·15	595	10-6-76	12–14	0·06	500
11-6-76	10–12	0·19	800	31-5-76/3*	20–25	0·08	103
31-5-76/1	7–9	0·07	128	9-6-76	50–70	0·09	128

The data refer to spike activity during ganglion cell responses to stationary spots of light, except in cell 7/6/76 where a moving spot was used.

* This cell showed a sustained injury discharge producing a second peak in the histogram at about 100 msec.

A comparison between the histograms obtained from ganglion cells with and without PSD is shown in Table 1.

Conduction velocity of ganglion cells with and without PSD have been computed from the latency of the soma spike produced by optic nerve stimulation. Possible errors due to stimulus current spread far from the stimulating point was avoided by considering only those cells which did not show latency variations by increasing the stimulus current from threshold (T) to maximal (about 5–8T). It has been found that in fifty ganglion cells conduction velocity may vary from 0·4 to 2·7 m/sec, independently of the presence, or not, of PSD.

DISCUSSION

The simultaneous abolition of both the PSD and the antidromic spike during the collision test and, conversely, the occurrence of an evoked PSD after a directly initiated spike are strong evidence that a prior firing of the

recorded cell is uniquely required for the generation of the PSD. These results rule out the possibility that the PSD is produced by costimulation, at the level of the optic nerve, of centrifugal fibres to the retina.

At least three other hypotheses could explain the origin of the PSD. Firstly, it may be a true 'after-potential' originating, as in peripheral nerve fibres (Frankenhauser & Hodgkin, 1956; Greengard & Straub, 1958) from a transient extracellular accumulation of potassium. If this were the case one should observe a summation of the PSDs during a train of impulses. But Fig. 4 shows that such a mechanism is not consistent with the PSD because it is depressed instead of enhanced by repeated stimuli. By the same argument one also excludes that PSD may be analogous to the 'after-potential' observed in hippocampal neurones (Kandel & Spencer, 1961).

A second hypothesis is that the PSD represents the sequential invasion of remote (dendritic) portions of the cell, as observed in other preparations (Spencer & Kandel, 1961; Kuno & Llinás, 1970; Zucker, 1972). Dendritic spikes, however, fire at high repetition rates (up to 100 impulses/sec) and when they fail, they do so in an 'all-or-none' fashion. Such characteristics are clearly in contrast with the graded amplitude modulation of the PSD, as illustrated in Fig. 4. Nevertheless the dendrites might play a fundamental but different role in the generation of the PSD to transmit a positive feed-back, in the form of a PSD.

The synaptic nature of PSD is suggested by its graded amplitude modulation and by the extrapolated reversal potential which approaches that of end-plate potentials (Takeuchi & Takeuchi, 1960). The graded depression of the PSD following a conditioning shock to the optic nerve correlates well with similar phenomena observed in both invertebrate and vertebrate neuromuscular junctions (Eccles, Katz & Kuffler, 1941; Lundberg & Quilisch, 1953; Liley & North, 1953; Takeuchi, 1958; Thies, 1965; Betz, 1970; Zucker, 1972). The depression of the PSD could be explained by a reduction in the release probability combined with a partial depletion of transmitter available for release at the dendritic terminals (cf. Betz, 1970).

It should be pointed out, however, that the present results do not provide unequivocal evidences in favour of the synaptic nature of PSD. Thus, even if unlikely, the possibility still exists that PSD represent a late component of a few ganglion cells spike, with ionic mechanism and refractoriness different from those of the earlier portions of the spike.

An important question is whether ganglion cells transmit also to other retinal cells. The possibility of a ganglion cell synapses with receptor, horizontal or bipolar cells has already been excluded in previous work (Marchiafava,1976, and unpublished results). A ganglion to amacrine cell synapse may be postulated, instead, on the basis that amacrine cells

generate an EPSP in response to optic nerve stimulation (Marchiafava, 1976). However, the amacrine cell e.p.s.p. follows high frequency stimulation of the optic nerve up to 80/sec, while the characteristics of the PSD are incompatible with synaptic transmission faster than 1–1·5/sec. Thus the different properties of the amacrine synaptic response and the ganglion cell PSD may be referred to two distinct synaptic pathways.

With regard to possible ganglion-to-ganglion cell synapses, it is important to note that the PSD cannot be confused with the late ganglion cell synaptic response to optic nerve stimulation, described in a previous report (Marchiafava, 1976). Thus in contrast to the PSD, the ganglion cell, late e.p.s.p., (i) is not affected by collision, (ii) it has a higher threshold than the antidromic response, (iii) it increases with the nerve stimulus intensity, and finally, (iv) it follows higher stimulus repetition rates than the PSD. However, on the basis of the present results, one cannot rule out that at least a portion of the ganglion cell e.p.s.p. reported in the previous paper is actually transmitted by collaterals originating from a different ganglion cell.

The functional role of PSD is to potentiate the visual signal transmitted to post-retinal neurones in such a way that each of the spikes produced directly by illumination may be followed, at about 10 msec, by an additional action potential. These supplementary spikes are thought to arise from PSD, since both events occur with the same delay of about 10 msec after the preceding spike. This conclusion is supported by the fact that the probability of such short interspikes intervals is extremely low in cells which did not produce PSD (Fig. 8). Functionally, such potentiation seems to be very relevant if one considers that in cells with PSD the great majority of spikes characterizing the photoresponse actually occurs at interspikes intervals of about 10 msec. Thus, PSD seems to play a major role in characterizing the cell discharge.

The fact that during high frequency discharge, such as during photoresponses (cf. Fig. 1), the PSD still produced the additional firing apparently contrasts with the evidence of a long PSD refractoriness, as illustrated in Fig. 4. However, by analogy with similar events at the neuromuscular end-plate (Thies, 1965), the depression should not involve more than 20–30 % of the PSD, thus leaving a substantial portion of PSD to operate the cell facilitation.

The finding of a distinct class of retinal ganglion cells which fire at short interspike intervals suggests the possibility that these cells may have specialized prerogatives in the transmission of visual information to the central nervous system.

Note. In a very recent work on the cat Baldissera (1976), recorded intracellular action potentials closely followed by a depolarizing wave ('delayed depolarization', or DD; see Granit, Kernell & Smith, 1963) which, under some experimental conditions may share some of the features of PSD. However, PSD' duration and time course, its exclusive dependence on the soma spike and finally, its monotonic depression during repetitive activity rule out that PSD and DD might have the same origin.

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