SEPARABLE PHASES OF LIGHT-EVOKED DEPOLARIZATIONS IN THE RETINA OF *STROMBUS*

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SUMMARY

The waveforms of light-evoked depolarizations in *Strombus* retinal neurones can exhibit two sequential peaks or phases, the relative amplitudes of which vary with changes in stimulus intensity and interstimulus interval. Experiments employing either the passage of constant intracellular current or voltage clamp techniques indicate that both phases reverse polarity at intracellular potentials less negative than the resting potential. The potential at which the first phase reverses its polarity is considerably more positive than that of the second phase. The results indicate that the light-evoked depolarizations are generated by at least two different processes; these appear to be separate conductance changes, neither of which is voltage dependent. Under certain conditions, the second phase was inhibited by high extracellular concentrations of Mg^{2+}, indicating that it may arise as a result of chemically mediated synaptic transmission. The first phase did not show such inhibition and appears to be caused by the direct action of light on the cell.

INTRODUCTION

In a previous paper we reported that the retina of *Strombus luhuanus* contains several classes of light-sensitive cells, distinguishable according to their intracellular electrophysiological properties in the dark and the waveform of their light-evoked potentials (Quandt & Gillary, 1979). One type of cell ('type II') exhibits a resting potential of about -75 mV and a relatively prolonged light-evoked depolarization. In that paper it was also emphasized that under certain conditions the waveform of the light-evoked depolarization could exhibit two peaks. Such a waveform is similar to the cornea-negative phase of the extracellularly recorded electroretinogram (ERG), which apparently reflects such light-evoked retinal depolarizations (Gillary, 1974; Quandt & Gillary, 1979). The differential dependence of the amplitudes of the ERG peaks on light adaptation and temperature suggests that they arise from separate cellular processes (Gillary, 1974). Similarly, one might expect the waveform of the

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intracellularly recorded light-evoked depolarization to be generated by more than one process. The present study concerned the origin of the light-evoked depolarization of the 'type II' cell, and particularly the cellular processes which generate the relatively complex two-peaked waveform.

METHODS

In all experiments, the retina was isolated in sea water, impaled with KCl-filled microelectrodes for intracellular recording, and presented with stimuli of white light from a tungsten source. These procedures, as well as those for minimizing light adaptation, were carried out as previously described (Quandt & Gillary, 1979).

For experiments involving the simultaneous passage of current and measurement of membrane potential, dual channel microelectrodes were used. These were constructed from 'theta' glass tubing (R & D Optical Systems, Spencerville, Maryland), filled with 3 M-KCl, and bevelled prior to use to lower the coupling resistance between the two channels (Werblin, 1975). This resistance measured in the bath was less than 100 KΩ for currents within the range of $\pm$ 10 nA, which exceeds the range of currents passed intracellularly. Since a value of 100 KΩ was calculated to produce less than 1 mV error in the measurement of the membrane potential during the application of current, it is unlikely that electrode coupling resistance significantly affected the experimental results. Although capacitative transients occurred at the onset and termination of the current steps (apparently due to the capacitance between the electrode channels), these were less than 1 ms in duration and did not obscure the light-evoked potentials and currents, the time courses of which were considerably slower.

For certain experiments a constant current source was used to pass current (Model M701, W-P Instruments, New Haven, Conn.). For voltage clamp experiments, a circuit similar to that described by Werblin (1975) was used, which enabled the membrane potential to be controlled to within 1 mV in less than 1 ms. Currents were measured by a current-to-voltage converter connected to the reference electrode.

In one series of experiments, a gravity-feed perfusion system was used to change the medium bathing the preparation during intracellular impalement. The bathing solutions included normal sea water, artificial sea water (NaCl 470 mM, MgCl$_2$ 30 mM, KCl 10 mM, CaCl$_2$ 10 mM, MgSO$_4$ 20 mM, NaHCO$_3$ 3 mM; pH 7·5), and 'high Mg^{2+} sea water', which differed in composition from artificial sea water only with respect to NaCl (120 mM) and MgCl$_2$ (250 mM). The experimental procedures used in the present studies have been described elsewhere in greater detail (Quandt, 1976).

RESULTS

Effects of interstimulus interval and stimulus intensity on the light-evoked potential

Typically a dark-adapted cell exhibited a resting membrane potential of about -75 mV and, in response to retinal illumination, a depolarization as large as 40 mV in amplitude (Quandt & Gillary, 1979). Under appropriate conditions, the response exhibited two peaks, separated by a brief period of repolarization (Fig. 1A). These two peaks, or phases, will be referred to as the 'early' and 'late' phases of depolarization.
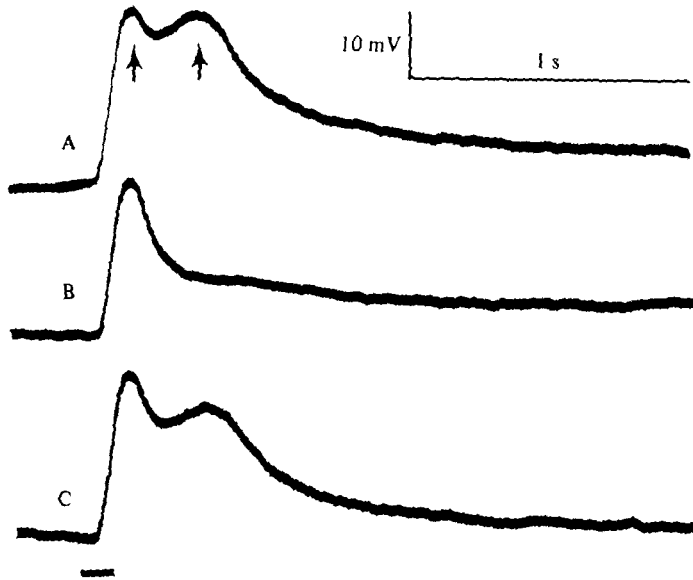
Fig. 1. Waveform characteristics of the light-evoked depolarization. The bar beneath the lowest record monitors for all three traces a o·t s photic stimulus of constant intensity. In this and all subsequent figures, membrane depolarization is upwards. (A) Repose to one of a series of repetitive stimuli delivered at 3 min intervals. Arrows indicate two peaks or phases (respectively termed the 'early depolarization' and 'late depolarization') separated by a brief period of repolarization. (B) Decreasing the interstimulus interval to 10 s caused a greater decrease in the late depolarization. (C) Increasing the interval back to 3 min restored the late depolarization.

Decreasing the interval between repetitive stimuli could selectively and reversibly abolish the second peak (Fig. 1). The relation of the respective amplitudes of the two phases to the interstimulus interval is illustrated in Fig. 2. In general, the amplitudes of both peaks decreased as a function of decreasing interstimulus interval but, as illustrated in Fig. 2, the rate of decrease of the second tended to be greater. This observation suggests that the two phases are generated by separate processes.

The relative amplitudes of the two phases also varied as a function of stimulus intensity (Fig. 3). In general, as the intensity was increased the amplitude of the second phase increased at a greater rate than that of the first and attained a substantially larger value at the highest intensities. These results also suggest that the two phases reflect more than a single underlying process.

Effects of constant intracellular current on the light-evoked potential

The effects of passing different amounts of constant intracellular current on the light-evoked potential are illustrated in Fig. 4. In such an experiment, repetitive photic stimuli of identical intensity and duration were delivered at fixed intervals to an initially dark-adapted preparation. After the light-evoked potential ceased to change, pulses of constant current (initiated just prior to the photic stimulus and maintained throughout the light-evoked potentials) were delivered via one channel of a dual-channel microelectrode. The records in Fig. 4 A show that hyperpolarization of the
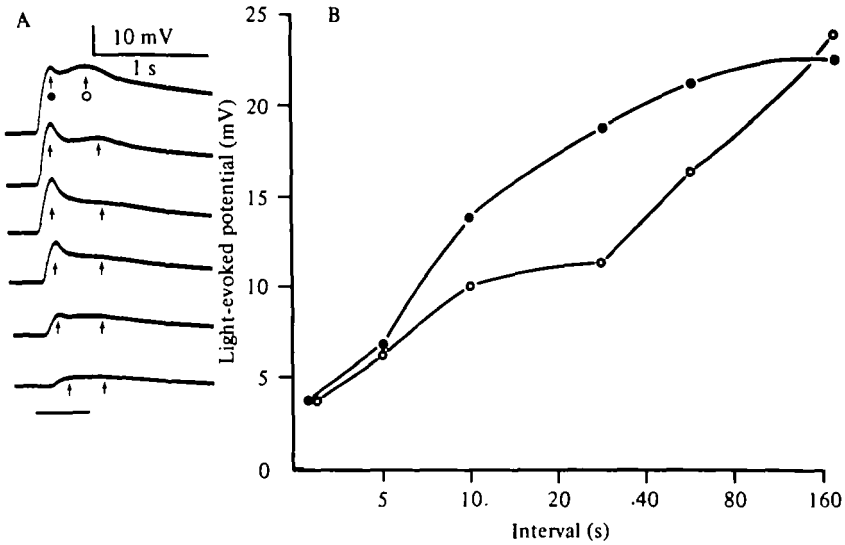
Fig. 2. Effects of interstimulus interval on the light-evoked potential. (A) Each of the six records, obtained from the same cell, represents a typical 'steady state' response after it ceased to change to identical repetitive stimuli delivered at constant frequencies. From top to bottom, the respective interstimulus intervals were 180, 60, 30, 10, 5 and 2 s. The bar below the bottom trace monitors, for all traces, a stimulus the duration and intensity of which were identical for all intervals. The arrows indicate points on the waveform selected to correspond to the phases of early depolarization (filled circle) and late depolarization (open circle). (B) A plot of the amplitudes of these two phases (measured as the displacement from resting potential in the dark) as a function of the interstimulus interval. The values were obtained from the records in A. Note that the ratio of the amplitudes of the two phases is not constant.
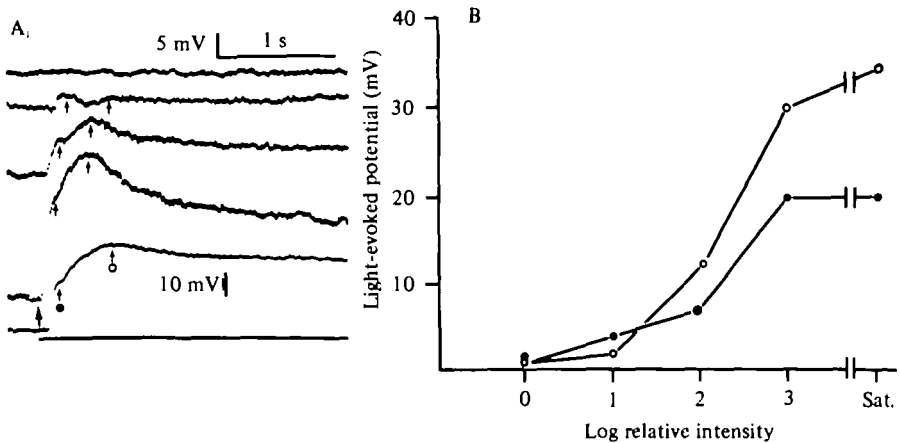

Fig. 3. Effects of light intensity on the light-evoked potential. (A) Five sequential responses from an initially dark-adapted cell stimulated at 3 min intervals with increasing intensities. The large arrow indicates, for the four uppermost records, the onset of a 0·1 s flash, the intensity of which was increased, from top to bottom, in unit log increments. The lowermost record is the response to a stimulus of long duration (monitored by the bar below it), and of the same intensity as that of the preceding brief stimulus. The small arrows indicate points on the waveform which correspond to the phases of early depolarization (filled circle) and late depolarization (open circle), the respective amplitudes of which are plotted in B. The uppermost vertical calibration mark applies to the upper four traces. (B) Plot of the amplitudes of the two phases of the records in A as a function of stimulus intensity. Note that the amplitude of each phase is graded with intensity but is not related to that of the other phase by a fixed ratio.
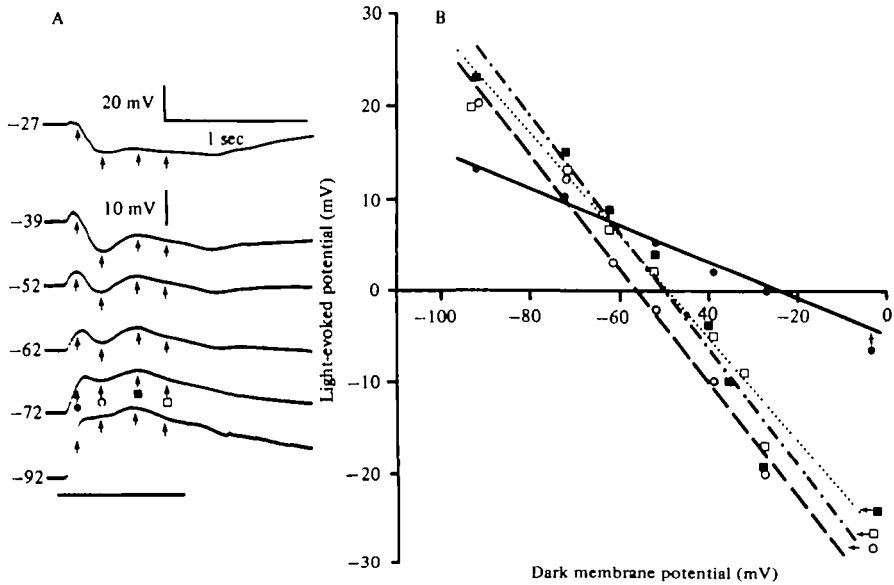
Fig. 4. Relation of the light-evoked potential to the dark membrane potential, varied by passing constant current. (A) 'Steady state' responses (see Fig. 2) from a single cell evoked by photic stimuli of equal intensity and duration (monitored for all records by the bar below) and presented at constant 3 min intervals. The dark membrane potential was shifted from the resting potential (-72 mV) by applying a constant current (in the range of o to several nA) which was initiated just prior to the photic stimulus and maintained throughout the light-evoked potential. The dark potentials for each trace are indicated in the figure. (The actual experimental sequence was -72, -27, -62, -52, -39 and -92 mV.) The arrows mark points on the waveform at fixed latencies from the onset of the photic stimulus; the first and third of these correspond approximately to the peaks of the early depolarization (filled circle) and late depolarization phase (filled square), respectively. The vertical calibration for the second trace applies also to the traces below it. (B) Plot of the amplitude of the light-evoked potential for the points obtained from the records in A, as a function of the initial, dark potential. The lines are regression lines. Note that the apparent reversal potential (i.e. the dark potential at which the receptor potential extrapolates to zero) for the early depolarization is considerably more positive than that of the late depolarization.

cell with extrinsic current increased with amplitude of the light-evoked depolarization, while depolarization with extrinsic current reduced its amplitude and could reverse its polarity.

In Fig. 4B, the amplitude of the light-evoked potential at each of four points of fixed latency from the stimulus is plotted as a function of the membrane potential in the dark (dark potential) immediately preceding the photic stimulus. Each straight line (which approximates the data for a given point in time during the waveform) intersects the abscissa at a value corresponding to the dark potential at which that point on the light-evoked potential reduces to zero and reverses polarity. These reversal potentials were always more positive than the resting potential but were never the same for all points in time during the response. The early phase of depolarization always reversed polarity at a value more positive than the reversal values for the rest of the response. The potential at which the brief intervening phase of repolarization reversed polarity was always slightly more negative than that of the late phase of depolarization.

One hypothesis compatible with the above results is that the light-evoked depolarization arises as a result of light-induced increases in ionic conductance, which would bring the membrane potential towards the reversal potential. If this were the case, one might expect extrinsic alteration of the membrane potential to produce such changes in light-evoked potential, by altering ionic driving forces, which depend on the differences between the membrane potential and their equilibrium potentials (Brown et al. 1971). One could account for the differences in reversal potential for different phases of the response by postulating the involvement of two or more conductance increases, which could differ with regard to the type of ion, or possibly the site that the change occurs in the cell.

Results from another type of experiment are compatible with the hypothesis that the light-evoked depolarization is effected by an increase in conductance of the cell membrane. In these experiments, repetitive brief pulses of constant current (usually ca. 100 ms in duration, -1 nA and $3/s$) were initiated prior to photic stimulation and maintained throughout the light-evoked response. The voltage displacement for each pulse was seen to decrease during the light-evoked depolarization, which is consistent with the hypothesis that photic stimulation evokes an increase in the membrane conductance of the impaled cell. Depolarization with extrinsic current in the dark does not result in a decrease in input resistance (Quandt & Gillary, 1979).

Effects of voltage-clamped potential on the light-evoked current

In one series of experiments the voltage clamp technique was used to investigate the possible contribution to the light-evoked response of voltage-dependent changes in membrane conductance. By holding membrane potential constant during the response, this technique should in principle be able to eliminate such voltage-dependent changes. In Fig. 5A, the waveform of the light-evoked potential is compared with that of the light-evoked current while the transmembrane potential was clamped at its 'resting' value (-82 mV). The occurrence of two peaks of light-induced inward current that appear to correspond to the early and late phases of the light-evoked depolarization indicates that changes in the recorded membrane potential are not required for the phases to occur. Two other findings reported previously (Quandt & Gillary, 1979) also indicate that voltage-dependent conductances do not contribute to the light-evoked depolarization of these cells. First, the current–voltage relationship for the cell membrane in the dark showed no evidence of rectification, for the input resistance measured by the application of extrinsic current was constant over a large range of currents. Secondly, the time course of the change in membrane potential in response to current steps appeared to reflect only simple membrane charging.

The voltage clamp technique was also used to determine the values cf membrane potential at which the early and late phases of the light-evoked current reversed polarity. These 'reversal potentials' were expected to reflect the actual equilibrium potentials of the ions which might be involved more accurately than those values obtained by passing constant current, since the clamped membrane potential and consequent ionic driving forces should remain constant throughout the response. As illustrated in Fig. 5B and C, the membrane potential at which the light-evoked current reversed polarity was not constant throughout the response. The current
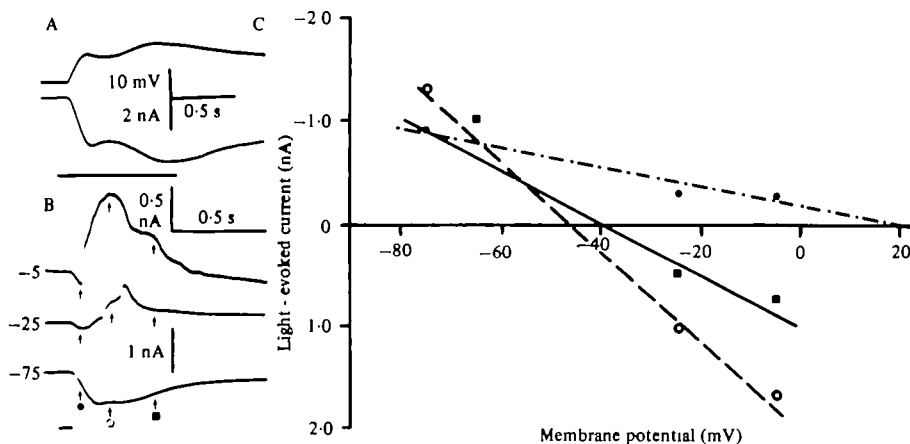
Fig. 5. Effects of the voltage-clamped membrane potential on the light-evoked current. (A) Comparison of the waveforms of the light-evoked potential and light-evoked current. Records were obtained from the same cell for identical conditions of photic stimulation, after the light-evoked potential to repetitive stimuli, presented every 2 min, attained a 'steady state' (see Fig. 2). The bar monitors, for both traces, the duration of the photic stimulus. Top trace, light-evoked potential; bottom trace, light-evoked current with the cell held at the resting membrane potential (−82 mV). Inward current is downward. Note that both traces exhibit similar waveforms. (B) Light-evoked currents from a single cell elicited under stimulating conditions as for B, above, except that just prior to the photic stimulus the membrane potential was clamped to a different value; these were respectively, for the bottom trace upward (which was the actual experimental sequence), −75 mV (resting potential), −25 mV and −5 mV. The bar below monitors the stimulus for all three traces. The lower vertical calibration mark in B applies to the bottom two records. Arrows mark points in the waveform (the amplitudes of which are plotted in C) at fixed latencies from the onset of the photic stimulus; these points were selected to correspond to the peaks of the early depolarization (filled circle) and late depolarization (square) of the light-evoked potential, and the period of repolarization between them (open circle). (C) Plot of the amplitude of the light-evoked current at different times after stimulation versus the clamped membrane potential. Inward ionic current is negative. Contrary to convention, inward currents are plotted above the abscissa, in order to facilitate comparison with Fig. 4B. The points plotted represent amplitudes measured from the records in A, at the post-stimulus times indicated by the arrows. The lines are regression lines. Note that the dark potential at which the light-evoked current extrapolated to zero was different for each point in time during the waveform.

corresponding to the late phase of depolarization reversed polarity at approximately −40 mV, and that corresponding to the preceding, brief phase of repolarization reversed polarity at a slightly more negative potential. Both of these reversal potentials were substantially more positive than the resting potential. Sufficient current could not be passed actually to reverse the polarity of the current corresponding to the early phase of depolarization; however, the data extrapolate to a reversal potential that is markedly more positive than the values for the rest of the response. These general findings are similar to those of the experiments in which constant intracellular current was passed during the response (cf. Fig. 4).

It is possible that the experimentally determined values of reversal potential differ from the actual values, as a result of such factors as altered recording conditions following cellular impalement (Wardell & Tomita, 1967; Werblin, 1975), non-uniform distribution of potential over the surface of the impaled cell, or electrical coupling of the impaled cell to other cells. However, these factors cannot explain the
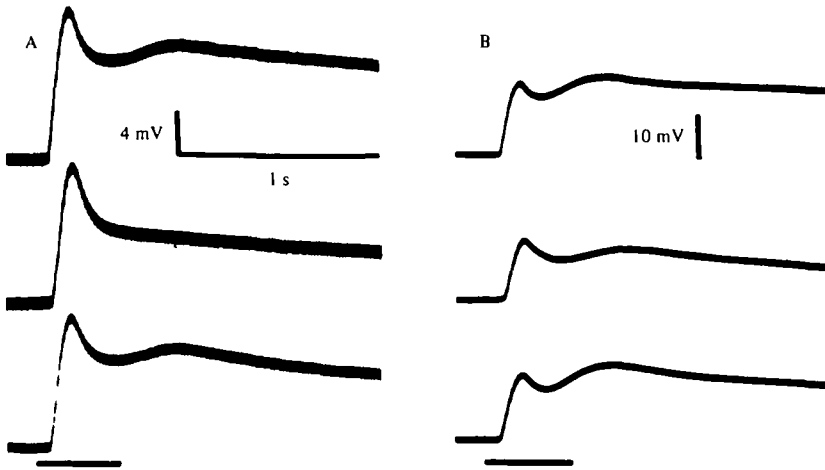
Fig. 6. Effects of magnesium chloride on the light-evoked potential. (A) Three sequential records from a single cell stimulated at 1 min intervals by flashes of equal intensity and duration (indicated for all traces by the bar below), while the perfusing solution was varied (see text for more details). For the top record the solution was sea water, for the middle record it was sea water with high Mg^{2+} and low Na^+ (see Methods), for the bottom record it was again normal sea water. (B) Records from the same cell under conditions that were identical to those for A except that interstimulus intervals of 2 min were used. Note that the late depolarization was reversibly abolished in the high Mg^{2+} solution when interstimulus intervals of 1 min were used, but was only partially decreased when intervals of 2 min were used.

relative differences in the reversal potentials determined for the different phases of the response. The existence of these differences accords with the hypothesis that the different phases originate from separate conductance changes. Some of the implications of the possible sources of error mentioned above and of the observed changes in reversal potential during the course of the response are considered at greater length in the Discussion.

Effects of magnesium ions on the light-evoked potential

To explore the possible contribution of chemically mediated synaptic input to the waveform of the light-evoked depolarization, the effect on the response of high Mg^{2+} concentration, known to inhibit transmission at certain chemical synapses (del Castillo & Engbaek, 1954; Takeuchi & Takeuchi, 1962) was examined. In these studies the responses of preparations perfused with normal or artificial sea water (no differences in response were seen for these two media) were compared with the responses obtained when the perfusion medium was 'high Mg^{2+} sea water' (see Methods). The concentration of Mg^{2+} in this solution (270 mM) is sufficient to inhibit reversibly in *Strombus* other responses apparently mediated by chemical transmission, such as 'off' activity in optic nerve fibres and light-evoked retinal movement (Gillary, 1977).

The results of a typical experiment are illustrated in Fig. 6. Each record represents a 'steady state' response to photic stimuli presented at regular intervals, after it ceased to change following perfusion of the preparation with either normal sea water or a

solution with high Mg^{2+} concentration. Although high Mg^{2+} concentration had very little if any effect on the early phase of depolarization, it very noticeably inhibited the late phase. When the interstimulus interval was 1 min, the second peak appeared to be completely abolished (Fig. 6A). However, when the intervals were increased to 2 min (as a result of which the preparation became more dark-adapted), the inhibition of the late phase was only partial. Thus it appears that Mg^{2+} alone, at the concentrations used, is insufficient to block completely the second phase of depolarization, and that other conditions, such as interstimulus interval (cf. Fig. 2), are required to bring about complete inhibition.

DISCUSSION

The light-evoked depolarization examined in the present study is similar to those of rhabdomeric photoreceptors of other species, including molluscs (McReynolds & Gorman, 1970b; Detwiler, 1976) and arthropods (Millechia & Mauro, 1969; Brown *et al.* 1970; Wulff, 1971; Nolte & Brown, 1972). In these other species, the responses were associated with reversal potentials which were more positive than the resting potential and were apparently generated by light-induced, inward current produced by an increase in membrane conductance to one or more ions driven down an electrochemical gradient. Morphological studies on the retina of *Strombus* (Gillary & Gillary, 1979) indicate that it contains a predominance of cells bearing rhabdomeric structures similar to those of other molluscan photoreceptors which depolarize in response to illumination (Hagins *et al.* 1962; Tomita, 1968; Mauro & Baumann, 1968; Jacklet, 1969; McReynolds & Gorman, 1970a; Mpitsos, 1973; Alkon & Fuortes, 1972; Chase, 1974; Pynsent & Duncan, 1977; see also Quandt & Gillary, 1979). These observations suggest that the type of cell studied here is a photoreceptor. However, the present evidence does not support this view unequivocally. For example, it is possible that the light-evoked depolarization is generated not in the impaled cell but in a different type of retinal cell (e.g. a photoreceptor) to which it is electrotonically coupled, as is the case for the eccentric cell in the lateral eye of *Limulus* (Smith *et al.* 1965; Behrens & Wulff, 1967).

The light-evoked depolarization studied here appears to consist of at least two separable phases (referred to as the 'early' and 'late' phases of depolarization, respectively). The amplitudes of these differ with regard to their dependence on interstimulus interval and light intensity and, most notably, the dark potential at which the light-evoked potential or concomitant light-evoked current reverses polarity. It is possible that these experimentally determined reversal values differ substantially from the actual values for the ions involved in carrying the light-induced current, if the recorded potential differed significantly from the membrane potential at the site of generation of the light-induced current (i.e. the cell membrane potential was not uniform). If such were the case, current injected via the microelectrode would have caused a smaller displacement in potential at the relatively remote generation site and led to an overestimation in reversal potential (i.e. one that was more positive than the 'true' value, which should reflect the equilibrium potentials of the ions involved). A similar overestimation in reversal potential would have occurred if the cell was electrically coupled to other cells undergoing similar, synchronous conductance changes. The above conditions cannot, however, account for the relative

differences in the reversal values determined for the two phases, which appear to be generated by at least two separate processes involving changes in membrane conductance. These may reflect differences regarding the types of ions involved (which could have different equilibrium potentials) or, if the membrane potential need not be identical for all points along the cell, differences in the cellular locations of the conductance changes, for either different ions, or for the same ion (in which case the phase arising at the more remote site would reverse polarity at a more positive recorded potential).

Variation in reversal potential with time during a light-evoked response has been found in the photoreceptors of other gastropods (Chase, 1974; Detwiler, 1976). In the retinal photoreceptors of *Hermissenda* studied by Detwiler (1976), the reversal potential associated with the first phase of depolarization was around 0 mV, whereas that of the subsequent repolarization and second depolarizing phase was about -70 mV (which was more negative than the resting potential, about -50 mV). The results of experiments in which the effects of Na^+-free and K^+-free media were examined were consistent with the interpretation that the phases were generated by an initial light-evoked increase in Na^+ conductance followed by a K^+ conductance increase and subsequent decrease. The reversal potentials for the different phases of the light-evoked potential in *Strombus* described in this report are all more positive than the resting potential. If the K^+ equilibrium potential is equal to or more negative than the resting potential, it is unlikely that in these cells the late phase of depolarization and preceding brief repolarizing phase are mediated exclusively by changes in K^+ conductance. However, the phases of the light-evoked response in *Strombus* could well be generated similarly by other ionic conductance changes.

Another alternative, already mentioned, which could explain the observed variations in the reversal potential during the response is that the locus of current generation changes with time during the response. For example, if the light-evoked current during the early phase was generated at a site more remote from the recording electrode than that of the late phase, this could give rise to an apparent reversal potential for the early phase that was more positive than that of the late phase, even though both were mediated by the same ion.

The observation that the early phase of depolarization is resistant to Mg^{2+} inhibition, a known synaptic inhibitor, indicates that it is not dependent on chemically mediated transmission. This, along with its relatively short latency and the observation that changes in membrane potential are not required for its initiation, suggests that it arises as a direct result of the action of light, either on the impaled cell or on a cell to which it is electrically coupled. This direct action is presumably a light-induced increase in membrane conductance to one or more ions.

The origin of the late phase of depolarization seems more puzzling. Evidence already presented indicates that it is most probably not a potential generated by a voltage-dependent change in conductance triggered by the early phase of depolarization. Its susceptibility to Mg^{2+} inhibition suggests synaptic mediation, and in fact the retina of *Strombus* exhibits synaptic-like structures in the retinal neuropile (Gillary & Gillary, 1979) and probable synaptic activity (Gillary, 1974, 1977), as do other gastropod retinas (Alkon & Fuortes, 1972). However, the failure of the high concentration of Mg^{2+} used to inhibit totally the response in the relatively dark-adapted eye leaves some doubt regarding the synaptic origin of this phase.

It is also possible that the late phase of depolarization arises, as the early phase apparently does, as a direct response to illumination, by either the impaled cell or cells to which it is electrically coupled, but that this response displays a slower time course than that of the early phase. Other invertebrate photoreceptors exhibit light-evoked potentials which apparently are generated by two or more processes mediated directly by light. Examples of these include the multiple membrane conductance changes postulated to underlie the receptor potentials of *Hermissenda* photoreceptors (Detwiler, 1976), and the generation of current in the ventral photoreceptors in *Limulus* by two light-induced processes (Lisman & Brown, 1971). Two antagonistic processes related to the state of the photopigment apparently affect the light-evoked potential in barnacle photoreceptors as well as those in the ventral eye of *Limulus* (Hochstein *et al.* 1973; Lisman & Sheline, 1976). A Ca^{2+}-activated K^+ conductance also contributes to the response of barnacle photoreceptors (Hanani & Shaw, 1977). Further experimental studies should be useful in testing the various alternatives regarding the origin of the light-evoked potential described here. These include ion substitution studies and the examination of the effects of specific inhibitors on the response, as well as the morphological description of the impaled cells and those with which they may interact.

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