The Effect of Tetrodotoxin, Veratridine and Tetraethylammonium Chloride on the Receptor Potential of the Crayfish Photoreceptor Cell

H. STIEVE and T. MALINOWSKA

Institut für Neurobiologie der Kernforschungsanlage Jülich GmbH.

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The effect of three drugs on the receptor potential (ReP) of the crayfish retina measured with external electrodes was tested.

1. Tetrodotoxin (TTX) ($1 \cdot 10^{-6}$ g/ml and $1 \cdot 10^{-5}$ g/ml) showed only slight effects on the receptor potential (Table I and II).
2. Veratridine (VERA) ($1 \cdot 10^{-5}$ g/ml) showed only small effects of the same type as TTX but a little stronger on the receptor potential which became obvious especially in the period after washing out the drug (Table III).
3. Tetraethylammonium (TEA) (5 mM/l) caused a decrease of the height of the receptor potential without influencing the repolarizing phase (decrease-time t_2) markedly (Table IV).

The results can be interpreted as follows:

1. The permeability increase for sodium, calcium and magnesium, which causes the rising phase of the receptor potential in the crayfish photoreceptor cell is of different nature than the increase of the sodium permeability in the squid nerve, or the sites of the sodium channel are not accessible for the TTX in the photoreceptor cell. This is shown by the experiments with TTX and VERA.
2. All the changes of the receptor potential which are caused by TEA can be explained by assuming that the permeability for potassium in the dark is lowered by TEA and that in the course of the normal receptor potential an increase of potassium permeability, which causes the decreasing repolarizing phase of the receptor potential, does not occur.

Introduction

It is known that the cell membrane of the photoreceptor is selectively permeable for different ions, e. g. HAGINS 1962, 1972[1,2]; de PONT et al.[3]; DUNCAN and WEEKS[4]; KORENBROT and CONE[5] and that during light excitation the permeability of the membrane changes (MILLECCHIA and MAURO[6], TOYODA et al.[7], BROWN et al.[8]).

In the case of the crayfish photoreceptor cell there occurs a transient influx of sodium, calcium, and magnesium ions in the course of the excitation, which causes the depolarization of the cell membrane. The steady state value of the receptor potential (ReP) is also influenced by active transport phenomena and perhaps by a chloride permeability (STIEVE et al.[9]). Very little is known about the nature of this permeability change in the photoreceptor cell membrane.

In the case of nerve and muscle membrane the usage of certain drugs such as puffer fish poison tetrodotoxin (TTX), alkaloid veratridine (VERA) and tetraethylammonium chloride (TEA) could help very much to clarify our knowledge of the mechanisms of the permeability changes involved in this excitation.

There are only very few data published about the action of these drugs on the receptor cell membrane. Only the action of TTX on the photoreceptor cell was investigated as e. g. by BENOLKEN[10,11], who found that TTX influences the transient of the ReP but not the plateau-value.

Therefore we quantitatively investigated the action of three well known permeability changing drugs on the receptor potential of the crayfish photoreceptor in the hope of finding specific effects.*

Material and Methods

The present paper deals with three series of experiments during which the retina of the crayfish *Astacus leptodactylus* Eschscholz was treated with the drugs

* The results of these experiments are briefly mentioned elsewhere (STIEVE[12]).

tetrodotoxin, TEA and veratridine. The effect of these drugs on the ReP of the crayfishretina, measured by extracellular electrodes, was investigated.

Preparation of the retina and experimental procedure including measuring apparatus have been described in detail in previous papers (STIEVE et al.[13,14]). The temperature of the retina during the experiments was always close to 15 °C, except for the experiments with VERA which were performed at 10 °C.

Solutions:

Van Harreveld's solution (VAN HARREVELD[15]) with a pH of about 7.6 was used as saline.

TEA

Van Harreveld's solution with addition of tetraethylammonium chloride 5 mM/l (Fa. Schuchardt, München).

TTX

Van Harreveld's solution with addition of tetrodotoxin $1 \cdot 10^{-6}$ g/ml and $1 \cdot 10^{-5}$ g TTX/ml (Fa. Sankyo Co. Ltd., Tokyo).

VERA

Van Harreveld's solution with addition of $1 \cdot 10^{-5}$ g/ml veratridine (Fa. EGA-Chemie KG, Steinheim/Olbuch F.R.G.) TTX, VERA and TEA were added from stock solutions in appropriate amounts.

The pH-values of all test solutions used were between 7.6 and 7.8.

Procedure

The retina was illuminated by white light at regular time intervals, and the electrical response to light, the ReP of the visual cells, was registered.

Every 10 min the retina was stimulated for about 6 ms and every 30 min a stimulus·of about 1 s duration was applied.

Apart from these light stimuli the preparation was kept in the dark during the whole experiment. The duration of the experiments was 3 to 5 hours. The time during the experiment was counted from the beginning of the pre-period. All potentials occurring in the retina were expressed as polarity of the distal against the proximal electrode. In all figures the negative voltages are plotted in upward direction.

The following measured values were determined for the characterisation of the ReP's and served as basis for the evaluation:

a) For short stimuli (τ about 6 ms):

h_{max} — The amplitude of the maximum (mV),
t_1 — The latency — the time at which stimulus begins until the first visible increase of the ReP (ms),
t_{max} — The peak-amplitude-time — the time from beginning of the stimulus until the maximum of the ReP is reached (ms),
t_2 — The time in which the ReP decreases from h_{max} to $h_{max}/2$ (ms).

b) For long stimuli (τ about 1 s):

h_{max} — The amplitude of the maximum (mV),
h_e — The plateau-value — the amplitude at stimulus end (mV),
t_{max} — The peak-amplitude-time (ms),
h_a — The amplitude 500 ms after stimulus' end (mV).

The shape-quotient h_{max}/h_e was also determined.

In the following the stimulus duration τ will be quoted as a subscript of the respective measured quantity (e. g. $h_{max_6} = h_{max}$ after a stimulus of 6 ms duration).

For the evaluation of all experiments of a series, the measured quantities of the RePs were compared as relative values, i. e. they were expressed in per cent of the value of a reference potential recorded in the same experiment at the end of the pre-period (immediately before addition of the test saline).

After a pre-period of 60 min, during which the test vessel containing the retina was perfused with normal saline, the preparation was exposed to the test saline for 60 or more minutes in the main period. The experiments ended with another 60 min perfusion with normal saline in the after-period. For the evaluation, the last ReP of the pre-period (a-value) was compared with the ReP after 60 min perfusion with test saline (b-value) and with the ReP after 60 min perfusion again with normal saline (c-value). If main period or after-period lasted longer than 60 min, the RePs after 120 min and 180 min (e. g. for the main period: values b_2 and b_3) were also evaluated.

Results

TTX-response

In 6 experiments the responses of the retina were investigated in physiological solution containing $1 \cdot 10^{-6}$ g TTX/ml.

Fig. 1 shows RePs recorded after long and short stimuli during one experiment of this series and Fig. 2 the course of the experiment.

In Table I the results of this series are compiled. After application of TTX the amplitude h_{max} of the ReP resulting from 6 ms stimuli changes insignificantly. The times t_1 and t_{max} also show no significant change, only the t_2-value increases significantly to about 125 per cent, decreases at the beginning of the c-period and increases again to 130 per cent until the end of the experiment.

For long stimuli (duration about 1000 ms) the maximal amplitude h_{max} of the RePs after application of TTX is slightly but not significantly increased.

The plateau-value h_e and the h_a-value increase irreversibly during the experiment: h_e to about 120
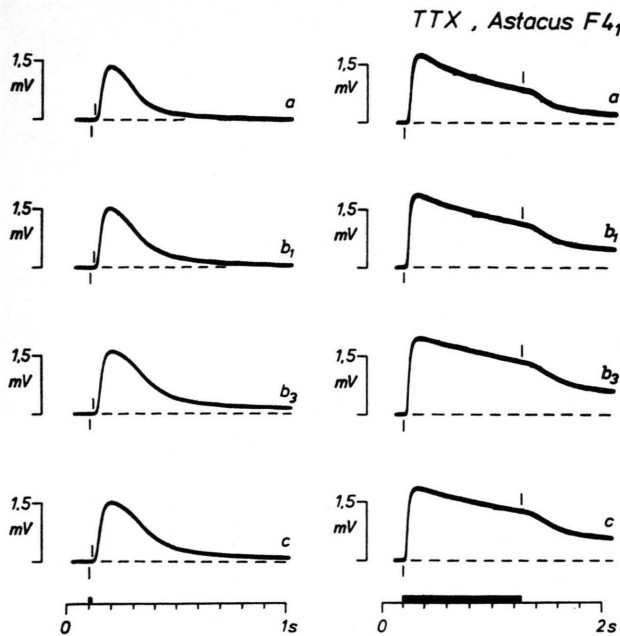
Fig. 1. Receptor potentials of an isolated crayfish retina in TTX-saline ($1 \cdot 10^{-5}$ g TTX/ml) recorded after short (τ about 6 ms) and long (τ about 1000 ms) stimuli (F 4_1). Times of photographs: a-Values: Pre-period; b-values: Perfusion with test saline; b_1 — 85 min, b_3 — 205 min; c-values: After-period. Temperature 15 °C.

Fig. 2. Plot of measured quantities (peak-amplitude h_{max_6} for short stimuli and $h_{max_{1000}}$ for long stimuli; shape-quotient h_{max}/h_e; decrease-time t_2) of the ReP recorded during perfusion with TTX-saline. Last value of pre-period: 100 Per cent-reference value. (F 4_1).

per cent, h_a to about 170 per cent. The shape-quotient h_{max}/h_e decreases to about 80 per cent, at the beginning of the c-period slightly increases (to about 90 per cent) and reaches again the 80 per cent-level at the end of the experiment. It seems that TTX influences the amplitudes of the ReP the more the later they
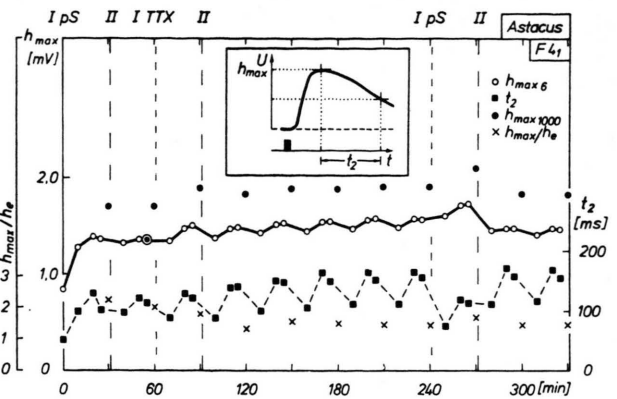
occur: h_{max} less than h_e, h_e less than h_a. The peak-amplitude-time t_{max} remains almost unchanged.

As we could not observe marked effects of TTX on the ReP by concentration of the drug $1 \cdot 10^{-6}$ g/ml we tried a series of 3 experiments with higher concentration.

We obtained by the concentration of TTX $1 \cdot 10^{-5}$ g/ml similar results (Table II).

BENOLKEN and RUSSEL (1966, 1967)[10,11] reported that the transient component of the generator potential of the *Limulus* lateral eye (intracellularly recorded) is selectively and reversibly inhibited by TTX (10^{-7} g/ml), while the steady-state component of this intracellular light response is either unaffected or only slightly affected by the drug. At low concen-

Table I. Measured quantities of the RePs in TTX-saline ($1 \cdot 10^{-6}$ g/ml). Peak amplitude (h_{max}), plateau-value (h_e), shape-quotient (h_{max}/h_e), peak-amplitude-time (t_{max}), and amplitude 500 ms after stimulus end (h_a) for long stimuli (τ about 1000 ms), and peak-amplitude, latency (t_1), peak-amplitude-time, and decrease-time (t_2) for short stimuli (τ about 6 ms). a-Values: Pre-period; b-values: Perfusion with TTX-saline; c-values: After-period. Temperature 15 °C. (F 3)

| | \multicolumn{5}{c}{1. Stimulus duration τ = ca. 8 ms} | | \multicolumn{5}{c}{2. Stimulus duration τ = ca. 1000 ms} |
	time [min]	h_{max} [mV]	t_1 [ms]	t_{max} [ms]	t_2 [ms]	time [min]	h_{max} [mV]	h_e [mV]	h_a [mV]	h_{max}/h_e	t_{max} [ms]
a	55	1.0±0.1	19.5±1.6	89.5±5.8	135.8±13.2	60	1.3±0.1	0.8±0.1	0.5±0.1	1.6±0.1	132.6±15.8
		[%]	[%]	[%]	[%]		[%]	[%]	[%]	[%]	[%]
b_1	85	112±5	84±3	93±3	107±2	90	110±3	119±3	141±11	92±1	101±4
b_2	145	113±7	90±4	99±4	127±6	150	106±3	129±6	166±17	83±3	105±4
b_3	205	110±8	92±6	103±5	126±8	210	104±4	132±7	178±21	79±3	113±3
c_1	235	115±10	75±8	79±3	93±6	240	109±5	123±8	160±24	89±3	89±3
c_2	265	103±8	108±6	106±3	128±9	270	98±4	125±7	169±21	79±3	112±3
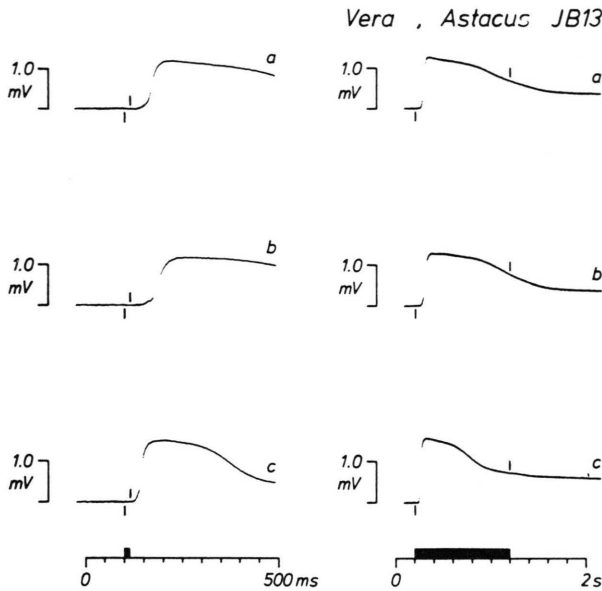
Fig. 3. Receptor potentials of an isolated crayfish retina in VERA-saline $(1 \cdot 10^{-5}$ g/ml) recorded after short (τ about 8 ms) and long (τ about 1000 ms) stimuli (JB 13). Times of photographs: a-Values: Pre-period; b-values: Perfusion with test saline; c-values: After-period. Temperature 10 °C.

Fig. 4. Plot of measured quantities (peak-amplitude h_{max_s} for short stimuli and $h_{\mathrm{max}_{1000}}$ for long stimuli; shape-quotient h_{max}/h_e; peak-amplitude-time t_{max_s} for short stimuli and $t_{\mathrm{max}_{1000}}$ for long stimuli) of the RePs recorded during perfusion with VERA-saline. Last value of pre-period: 100 Per cent-reference value. (JB 13).

trations (100 times lower) TTX inhibits only optic nerve activity producing no effect on the generator potential.

LOEWENSTEIN, TERZUOLO and WASHIZU[16], trying action of tetrodotoxin on two sensory systems, the crustacean stretch receptor and the mammalian Pacinian corpuscle, reported that the drug selectively blocked the propagated nerve impulse of the sense organs but that it had no effect on the graded generator potential.

Table II. Measured quantities of the RePs in TTX-saline $(1 \cdot 10^{-5}$ g/ml). a-Values: Pre-period; b-values: Perfusion with TTX-saline; c-values: After-period. Temperature 15 °C. (F 4)

	1. Stimulus duration τ = ca. 6 ms				2. Stimulus duration τ = ca. 1000 ms					
	time [min]	h_{max} [mV]	t_1 [ms]	t_{max} [ms]	t_2 [ms]	time [min]	h_{max} [mV]	h_e [mV]	h_a [mV]	h_{max}/h_e	t_{max} [ms]
a	55	1.1±0.1	14.9±1.0	82.5±6.3	129.5±10.5	60	1.4±0.2	0.7±0.1	0.3±0.1	2.1±0.1	132.6±20.0
		[%]	[%]	[%]	[%]		[%]	[%]	[%]	[%]	[%]
b_1	115	107±7	96±11	102±3	120±2	120	103±4	108±17	120±30	100±13	99±6
b_2	175	103±7	93±7	105±3	125±6	180	101±6	117±17	135±38	88±8	96±2
b_3	235	102±8	115±12	112±3	132±2	240	99±8	117±21	137±40	88±9	104±6
c	295	99±8	104±8	103±7	130±5	300	95±8	110±24	131±42	93±15	101±3

Table III. Measured quantities of the RePs in VERA-saline $(1 \cdot 10^{-5}$ g/l). a-Values: Pre-period; b-values: Perfusion with VERA-saline; c-values: After-period. Temperature 10 °C. (IB 11—15)

	1. Stimulus duration τ = ca. 8 ms				2. Stimulus duration τ = ca. 1000 ms					
	time [min]	h_{max} [mV]	t_1 [ms]	t_{max} [ms]	t_2 [ms]	time [min]	h_{max} [mV]	h_e [mV]	h_a [mV]	h_{max}/h_e	t_{max} [ms]
a	55	1.1±0.3	32.1±5.6	100.5±17.0	304.9±53.3	60	1.2±0.5	0.6±0.1	0.3±0.1	2.0±0.2	120.0±27.4
		[%]	[%]	[%]	[%]		[%]	[%]	[%]	[%]	[%]
b	115	103±4	119±7	117±9	130±16	120	103±5	110±10	121±9	94±6	114±9
c	175–185	145±6	75±16	77±11	73±8	180	139±5	121±9	166±9	115±7	85±10

TTX does not affect the action potential of *Helix pomatia* neurones, even in high concentrations (5 · 10^{-6} g/ml) (MEVES[17]).

Veratridine

In 5 experiments retinae were perfused with a saline containing $1 \cdot 10^{-5}$ g/ml veratridine.

Fig. 3 shows RePs recorded after long and short stimuli during one experiment of this series and Fig. 4 the course of the experiment. In Table III the experimental data are presented.

After application of veratridine the amplitude h_{max} of the ReP caused by short stimuli remains unchanged during the drug-action period; upon washing out the test saline h_{max} increases to 140-150 per cent.

The latency t_1 as well as the values of t_{max} and t_2 increase to about 120 per cent during perfusion with veratridine.

At the end of the after-period t_1, t_{max} and t_2 were about 75 per cent of the respective reference values.

For long stimuli the course of the amplitude $h_{max_{1000}}$ is parallel to the amplitude h_{max_6}; we observed no change during perfusion with the test saline and an increase of the after-period-value (to about 140 per cent). The plateau-value h_e increased slightly and remained at about 120 per cent in the after-period. So did the h_a-value; its c-value was about 165 per cent of the reference value.

The shape quotient h_{max}/h_e was not significantly decreased in the main-period but slightly increased in the after-period (115 per cent). The same is valid for the peak-amplitude-time t_{max}, which was not much increased in the main-period (b-value: 114 per cent) and slightly decreased in the after-period (c-value: 85 per cent).

In our experiments VERA shows a slight effect on the ReP which becomes obvious and statistically significant only by comparison of the b- and the c-values: the time course of the ReP is slowed down a little bit by veratridine and after washing out it is enhanced above the reference values.

The amplitude values h_{max}, h_e and h_a are changing from a to b and from b to c in the same direction. This means that the effect of washing out does not reverse these effects of the VERA. This is especially obvious by comparison of the values of h_a. As a general rule VERA like TTX is influencing the amplitudes of the receptor potential the more the later they occur (h_{max} less than h_e, h_e less than h_a).

Fig. 4 shows the opposite influence of VERA on amplitude and time course (t_{max}) of the ReP.

TEA

Two concentrations of TEA-solution (1 mM/l and 5 mM/l) were tested and the 5 mM-solution of TEA was used. The total number of experiments with 5 mM/l concentration of TEA was eight.

Fig. 5 shows RePs recorded after short and long stimuli in one experiment of this series and Fig. 6 the course of the experiment. In Table IV the measured values of the experiments after both short and long stimuli are compiled.

After application of TEA the ReP amplitude h_{max} caused by short stimuli diminishes to about 70 per cent; upon washing out the test saline, the ReP-maximum rises to the initial value.

The latency t_1 shows no significant change, though it appears to decrease slightly in the first period of perfusion of the retina with the test solution, just as the t_{max}-value which decreases to about 90 per cent only during the first 60 min of perfusion with test saline. The t_2-value remains almost unchanged.

For long stimuli (about 1000 ms) the maximal amplitude h_{max} of the RePs after application of TEA decreases to about 60 per cent. The plateau-value h_e decreases more than h_{max} in the presence of the drug to about 40 per cent, but increases again to about

Table IV. Measured quantities of the RePs in TEA-saline (5 m M/l). a-Values: Pre-period; b-values: Perfusion with TEA-saline; c-values: After-period. Temperature 15 °C. (F 2)

	time [min]	h_{max} [mV]	t_1 [ms]	t_{max} [ms]	t_2 [ms]	time [min]	h_{max} [mV]	h_e [mV]	h_a [mV]	h_{max}/h_e	t_{max} [ms]
		1. Stimulus duration τ = ca. 6 ms					2. Stimulus duration τ = ca. 1000 ms				
a	55	0.8±0.2	17.3±1.4	90.5±4.3	122.5±8.1	60	1.1±0.3	0.7±0.1	0.26±0.1	1.7±0.1	156±12.7
		[%]	[%]	[%]	[%]		[%]	[%]	[%]	[%]	[%]
b_1	115	72±3	94±3	87±3	104±5	120	61±2	38±2	64±8	166±10	63±3
b_2	175	69±4	106±7	96±4	107±6	180	59±3	38±3	51±7	158±9	66±4
c	235	98±6	97±4	103±3	117±5	240	98±4	121±6	145±17	82±2	112±5

Discussion

Tetrodotoxin

Pharmacologic agents have long served as useful aids in the study of physiological mechanisms. It has been shown that tetrodotoxin acts on the nerve membrane of the squid and crayfish, on the muscle membrane of the frog and on the electroplaques of the electric eel. TTX acts specifically in blocking the sodium channels which are activated (opened) in the initial phase of the excitation thus causing the rising phase component of the action potential (spike) (Nakajima et al.[18], Narahashi et al.[19,20], Nakamura et al.[21], Moore and Narahashi[22,23], Yamagishi and Grundfest[24]).

In those membranes there are at least two different channels for the movement of sodium and potassium ions across the cell membrane (Hille[25]). In rat cerebral tissues TTX (10^{-8} M) inhibits the increases in sodium and potassium fluxes which normally accompany electrical excitation (McIlwain et al.[26], Ramsey and McIlwain[27]). In the same tissues TTX (10^{-7}–10^{-8} M) also blocks the influx of calcium which occurs in the absence of electrical stimulation (Bull and Trevor[28]).

At present we do not know much about the molecular structure of the membrane of the photoreceptor cell of the crayfish, but evidence exists that the photoreceptor cell membrane is selectively permeable for different species of ions and that the ionic permeabilities change specifically during excitation (see: Introduction). The transient of the receptor potential is mainly caused by an influx of sodium ions. But also calcium and magnesium fluxes contribute to it to a certain degree. The plateau-value is determined by the fluxes of sodium, calcium and magnesium and active transport processes. Perhaps also chloride movement contributes to the plateau-value (Stieve et al.[9]).

The experiments reported here did not show any marked effect of the action of TTX on the ReP of the crayfish. In respect to the transient of the ReP our experiments differ from those of Benolken[10,11] in the *Limulus* photoreceptor cell (see: Results, page 151). They reported selective and reversible inhibition of the transient by TTX. Their data seemed to support the hypothesis that the transient component of graded response includes a regenerative sodium dependent component.

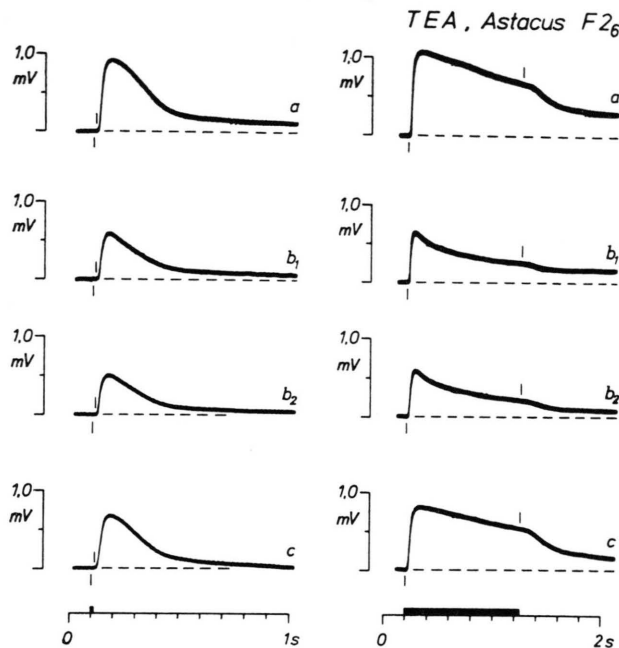Our experiments show that the crayfish photorecep-

Fig. 5. Receptor potentials recorded after short (τ about 6 ms) and long (τ about 1000 ms) stimuli in TEA-saline (5 mM/l). For time of photographs see Fig. 1. (F 2_6). Temperature 15 °C.
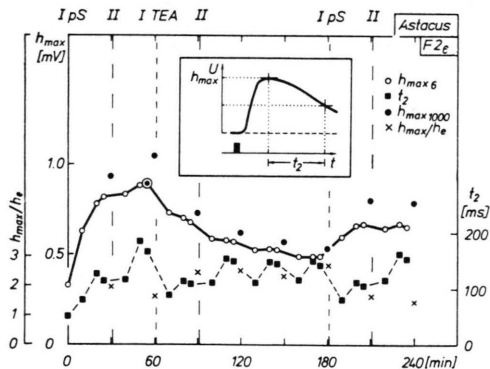

Fig. 6. Plot of measured quantities of the RePs recorded during perfusion with TEA-saline. Last value of pre-period: 100 Per cent-reference value. (F 2_6). For measured quantities see Fig. 2.

120 per cent in the after-period. The shape quotient h_{max}/h_e rises to about 160 per cent. These changes are reversible in the after-period. Only the h_a-value decreases (to about 60 per cent) under the influence of the drug but arrives at the 150 per cent level by the end of the experiment (*c*-value). The *c*-value of the plateau-amplitude h_e reaches about 120 per cent.

The t_{max}-value becomes significantly shorter (to about 60 per cent) but increases again in normal saline (*c*-value: 112 per cent).

tor potential has a different mechanism than that of the *Limulus* photoreceptor potential. They also show that ionic fluxes (the sodium flux and possibly calcium and magnesium) causing the transient phase of the ReP in the crayfish are not specifically blocked by TTX. In this connection one should not forget that the *Limulus* photoreceptor has different sensitivity to Na-ions.

There is another effect we observed. We obtained in TTX-solution a marked increase of the plateau-value and h_a value; TTX obviously acts by retarding the repolarization of the ReP but we do not know which mechanisms are causing this effect. One possibility among others could be an influencing of the active transport. This all means that the increase of the permeability of the crayfish photoreceptor membrane for sodium-, calcium- and magnesium-ions is distinctly different in its nature from that observed in the investigated nerve and muscle membranes.

There is also another possibility:

If one assumes heterogeneity of the structure of photoreceptor membrane, it seems reasonable to assume that TTX interacts somehow with specific membrane components (Na-channels structure).

In the case of the specific photoreceptor membrane these channel structures in the membrane could either be of different chemical character than in the nerve membrane or of similar nature like in the nerve membrane, but not easily accessible to the TTX-molecule, and therefore not causing any marked effects.

Veratridine

In the membrane of the node of *Ranvier* VERA is assumed to change a fraction of the sodium channels into ones which open much more slowly than the normal sodium channels during excitation (ULBRICHT[29]). The same action seems to be true in the muscle membrane (ULBRICHT[30]). This results in a slowly developing increase in sodium permeability following the only slightly changed spike of the nerve fibre. Veratridine has the same effects on snail neurones as on myelinated nerve fibres. It lowers the resting potential, changes the current-voltage relation and causes increased long-lasting after-depolarizations (LEICHT et al.[31]).

In our experiments VERA has just a very small effect which is not conclusively interpretable. If the sodium channels in the crayfish photoreceptor cell membrane are different to those of the nerve membrane, as suggested by the TTX experiments, one should expect that also VERA acts differently on this membrane. But similar to the effect on the nerve membrane the kinetics of the action potential is influenced. There is no indication in our experiments to assume that VERA creates the new sodium channels in the visual cell membrane and if it changes existing sodium channels, it does not change them very much.

We tried to understand the late changes of the ReP induced in the after-period. It is possible that traces of the alkaloid remaining in the retina cause increased permeability (possibly interfering with a closing of the Na-channels). For long stimuli plateau-value h_e and h_a-value remain increased in the after-period too.

ULBRICHT[32] discusses a similar effect (not full recovery after prolonged alkaloid treatment). It is possible that externally applied veratridine penetrates the membrane, accumulates in some intracellular compartment and even during washing periods this internal pool, supplying drug molecules to the membrane, causes an "irreversible" effect.

Tetraethylammonium chloride — TEA

It is shown that in nerve and muscle membrane TEA blocks specifically the permeability increase for potassium ions which enhances the repolarizing phase of the action potential following the maximum depolarization. TEA also diminishes the potassium permeability of the unexcited nerve and muscle membrane (STANFIELD[33], SCHMIDT[34], SCHMIDT and STÄMPFLI[35], KOPPENHÖFER and VOGEL[36], HILLE[37], ARMSTRONG[38]).

If the mechanism of the receptor potential of the crayfish photoreceptor involves an increase in potassium permeability, resulting in an enhancement of the repolarization, one could expect the action of TEA to produce a prolongation of t_2. This is obviously not the case. TEA affects the receptor potential considerably but the t_2-value is not affected. This leads to the conclusion that either there is no increased potassium permeability in the repolarizing phase of the ReP or that the opening of additional potassium channels is not blocked by TEA in the crayfish visual cell. For several reasons the first possibility seems to be more probable.

All the changes of the ReP: decrease of h_{max}, stronger decrease of h_e, decrease of t_{max} and no marked change in t_2 could be explained by assuming a reduction of the dark potential of the photoreceptor cell due to a decrease in the dark permeability of potassium. These changes of the receptor potential

are similar to those observed during depolarizing the cell by increased outer potassium concentration (STIEVE[39], STIEVE and WIRTH[13]) or by ouabain application (STIEVE et al.[14]). In both cases we observed effects similar to those of TEA on the ReP except those on the t_2-value, which became markedly shortened.

If one assumes that the potassium permeability is decreased in the dark by TEA, the result should be that t_2 is prolonged, for it takes longer for a smaller potassium current to repolarize the membrane. This prolongative effect could possibly compensate for the decrease in t_2 which should be produced by decreased resting potential.

The affinity of TEA to the potassium channels in the crayfish photoreceptor membrane should therefore be similar to that in the squid nerve membrane in contrast to the mechanism of the sodium permeability.

This would be consistent with the assumption that TEA acts on the photoreceptor cell membrane similarly as on the nerve membrane but that in the crayfish photoreceptor cell there does not occur an increased potassium permeability in the course of the excitation.

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