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# THE EFFECT OF POLARIZATION ON THE EVOKED AND SEIZURE POTENTIALS IN THE CAT

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**Abstract**

The effects of polarization was studied on cortical auditory evoked and strychnine potentials. Hyperpolarization augmented the acoustic evoked potentials in deep leads, depolarization depressed them. Polarization resulted on strychnine potentials in changes of proportions of different phases without altering the peak-to-peak amplitude. The authors assume, that modifications in transmitter output from presynaptic endings may play a considerable role in the mechanism of polarizational effects.

The mechanism of interneuronal transmission of impulses at the chemical synapses is in many aspects uncleared, up to now. This problem has aroused an intensive interest among morphologists, biochemists, and even physicists, too.

The interneuronal transmission as a whole can be divided into four distinct moments as follows: i) invasion of the presynaptic ending by the propagating axonal spike, ii) the liberation of transmitter substance from its place of storage, iii), the diffusion of the transmitter through the synaptic gap to the postsynaptic membrane, if condunctance changes of the postsynaptic membrane.

The aim of our work was to study the finer details of these processes, especially those taking place on the presynaptic side in cortical synapses.

The experiments were performed on the acoustic cortex of the cat. Primary evoked potentials were elicited by application of click-stimuli to one ear. The frequency dependence of the evoked potentials and the effect of electric polarization was studied. On the basis of the data obtained in this way we draw conclusion as to the quantitative aspects of transmitter dynamics in cortical synapses.

## Methods

The experiments were performed on adult cats of 2,5—3,0 kg weight, anaesthetized by 40 mg/kg pentobarbital-Na given intraperitoneally. The femoral vein and trachea were cannulated. For maintaining the level of anaesthesia 5—10 mg/kg pentobarbital-Na was given additionally.

The head of the animal was fixed in a stereotaxic instrument (type-Kovách), the skin and the muscles of the scalp were removed bilaterally. By use of a dental drill we opened the skull and exposed the gyri ectosylvius and suprasylvius on both sides. The dura was cut with fine scissors. Warm paraffin oil protected the brain surface from cooling and drying. Bone-bleeding was prevented by wax. After surgery the animal remained in rest for 1—2 hours. Body temperature was kept constant by an electric heater.

Recording.

Acoustically evoked potentials were led off from the primary auditory area, from a depth of 1500—2000 micra. The recorded site was always at the *punctum maximum* of these potentials. As a

recording electrode we used a steel-needle of 0,3 mm in diameter, isolated up to the tip. The electrode, fixed to the holder of the stereotaxic instrument could be moved in three dimensions. Depth measurements were possible with 0,05 mm accuracy. As the introduction of the electrode inevitably compressed the cortex and falsified depth measurement, the electrode was introduced somewhat deeper and the depth wanted was attained during withdrawal. The tip of the electrode remained at the depth where deep negative evoked potentials appeared to be maximal. After having inserted the electrode at the appropriate depth, 15—20 minutes rest was given for restitution of the cortical circulation. A silver wire was attached to the skin of the head as an indifferent electrode. The signals to be amplified were led into a DISA two-channel Electromyograph with a time constant of 120 msec. For data storage we used a magnetic tape recorder.

*Stimulation.* For evoking primary acoustic potentials we used click stimuli through an earphone applied to the right ear. Clicks were produced by 35 V, 1 msec impulses of a square-wawe stimulator. Stimulation was always supramaximal.

*Polarization.* We applied polarizing electrodes on the anterior ectosylvian gyri on both sides. In this way the polarization was transcortical. The polarizing circuit included a battery, a microammeter, a potentiometer and a polarity-switch. As polarizing electrodes we used chlorided silver plates of 0,6 cm surface area. Thus the current loops traversed the cortex, the underlying white matter and the commissural fiber system (Fig. 6).

The intensity of the polarizing current was adjusted to cause a voltage-gradient of 30 mV/mm extracellularly. This voltage-gradient could be maintained in most cases by a current of about 400 $\mu$A. The contact of the polarizing electrode to the cortex did not disturb the cortical circulation. On the polarizing electrode of the left side there were small holes for the recording electrode. When the positive electrode was on the recorded side, we regarded it as an anodal or hyperpolarization. The polarization started 1 minute before stimulation and acted till the end of it. After each polarization period 5 minutes rest followed for restitution from polarizational changes.

*Evaluation of data*

The storage of the signals on magnetic tape enabled us to make amplitude averaging. This was done by means of the 512 channel amplitude analyser (KFKI typ. NTA 512) placed kindly at our disposal by the Dept. of Physiology of the Medical School in Szeged. At every stimulation frequency 100 signals were averaged. Channel width was 128 msec at frequencies 1—5, analysis time 0,5 sec. At 7— to 10 cps channel width was 64 msec. An X—Y recorder fed from the amplitude averager delineated the experimental curves.

## The effect of DC polarization on the evoked potentials

The polarizing current, whose loops extended between the ectosylvian gyri of the two sides, resulted in a voltage change of 30 mV/mm at 400 $\mu$A current strength. The effect of polarization on the frequency dependence curves can be seen in Fig. 1. The diagrams include the results of six experiments. As it is apparent in this Figure, hyperpolarization shifts the whole frequency dependence curve toward higher amplitudes; depolarization has the opposite effect. In the first two experiments depolarization causes the appearance of a new inflexion point; this may be the consequence of a shift of the upper inflexion point toward higher frequencies.

In course of the evaluation special attention was paid to the question: how the polarization induced changes of the frequency dependence curves could be fitted to the curves computed on basis of our theory about transmitter dynamics (FEHÉR, HUNYA, 1973). A further question was: which parameters of the model could be made responsible for changes observed during polarization.

The block diagram clearly demonstrates, that the proportion of transmitter liberated by one iinpulse was decreased by depolarization and increased by hyperpolarization. The extent of these changes could be estimated quite accurately by analysing the frequency-dependence curves. This shows a good agreement with

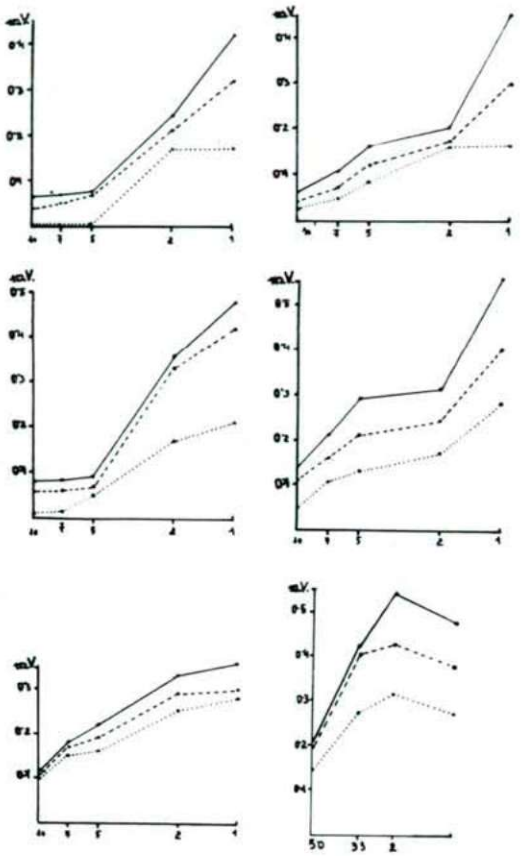

*Fig. 1.* Effect of polarization on the amplitude of evoked potentials. The diagrams represent the results of six experiments. Ordinates: amplitudes in μV. Abscisses: stimulation frequencies on logarithmic scale. Broken lines: control series, continuous lines: frequency dependences curves taken during hyperpolarization, dashed lines: the same during depolarization. Polarizing current intensity was 400 μA in each case.

conclusions drawn from experiments on muscles; according to these, the amplitude of the spike potential is the main factor determining the liberated transmitter quantities from synaptic endings. One parameter of our model (FEHÉR, HUNYA, 1972) reflects the proportion of transmitter liberated by one impulse quite accurately and therefore conclusions can be drawn from changes of frequency dependence curves as to the mechanism of action of any factor acting upon transmitter dynamics. Unfortunately it is not easy to judge which one of the factors can be made responsible for changes in transmitter proportions liberated by one impulse: the duration of action potentials or modification of permeability of the presynaptic endings. Presumably, the form er more constant and thus permeability changes lead more likely to changes in transmitter output.

As it was mentioned above, in experiments with polarization the upper inflexion point on the frequency dependence curve could not be found. Its situation on the curve is influenced by transmitter synthesis. No data are, however, available so far according to which polarization would exert any effect upon this process. Thus, the lack of the upper inflexion point does not curtail the usefulness of our model.

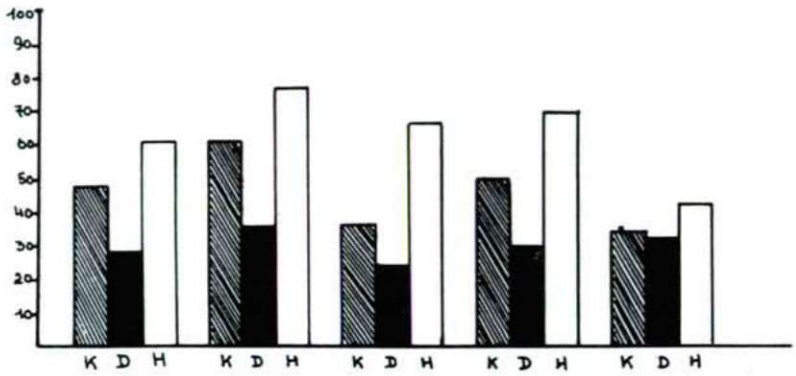


*Fig. 2.* The evaluation of five experiments illustrated in Fig. 1. Ordinate: The percentual proportion of the transmitter store, liberated by one impulse. Designations of blocks: K: control, D: Depolarization, H: hyperpolarization.

### The effect of polarization on strychnine potentials

Strychnine potentials were evoked by applying 1 percent strychnine solution on the cortex by a 2×2 mm filter paper soaked in it. As the site of origin of strychnine potentials lies in the superficial layers, we examined the effects of polarization in surface leads. The strychnine potentials follow stimulation frequencies over 3 cps rather poorly. Therefore, their frequency dependence could not be examined. The lack of these curves as to the strychnine potentials did not prove to be disadvantageous because polarization failed to exert any effect on them at 1 and 2 cps in surface leads. This is illustrated in Fig. 3 by synchronized records in control situation, at hyper- and depolarization, respectively.

Our findings in deep leads were seemingly contrary (Fig. 4). The strychnine potentials exhibiting a negative-positive-negative sequence in depth records, underwent characteristic changes during polarization; depolarization enhanced their negative phase while the positive wave decreased or disappeared completely. Hyperpolarization, on the contrary, depressed the negative phase and enhanced the deep positive waves. The peak-to-peak amplitude, remained, however, unchanged. The effect of polarizing current consisted in a shift of the DC-level and the strychnine potentials appearing on different background potential level showed variations only in proportions of their positive and negative phases. Depolarization emphasized their negative, hyperpolarization augmented their positive components.

A similar phenomenon could be observed also at the evoked potentials, but it was joint always with changes of the peak-to-peak amplitude described above.

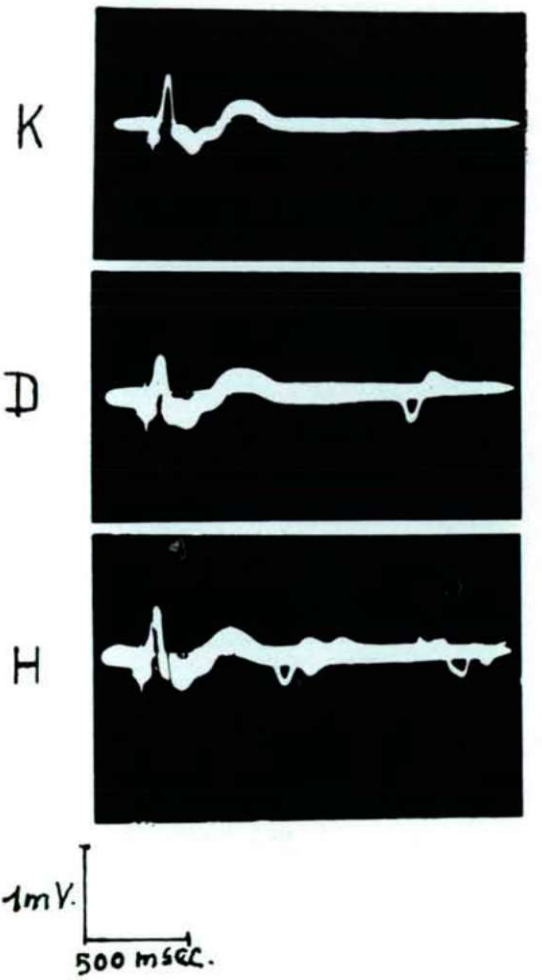


*Fig. 3.* Effect of polarization on strychnine potentials in surface leads. Ten superimposed traces photographed from the screen of an oscilloscope. Negativity upwards. Calibration: 1 mV and 10 msec. K: control, H: hyperpolarization, D: depolarization.

Analysing the effects of the polarizing current on cortical structures, the first question, that arises concerns their explicability in terms of the electrotone observed on peripheral nerves. As it is well known, the membrane potential of peripheral nerves decreases in catelectrotone with a concomitant increase in excitability and conductivity. Anelectrotone causes changes of opposite sign.

No doubt, the cortical polarization phenomena show remarkable similarities to those observed in peripheral electrotone. The conditions under which the polarizing current acts are, however, different in many respects in the two structures. i) The polarizing current traversing the cortical tissue makes its way through the intercellular

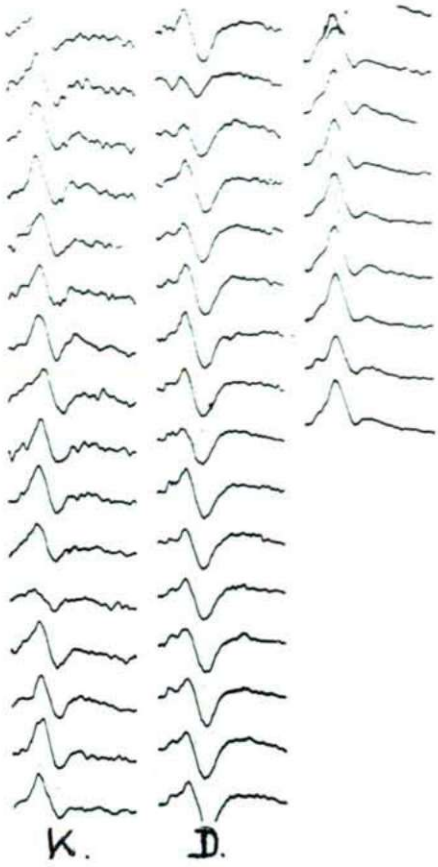


*Fig. 4.* Effect of polarization on strychnine potentials lead off from a depth of 1500 μ. K: control, D: depolarization, H: hyperpolarization. Although peak-to-peak values do not vary significantly, deep negative phase is augmented during hyperpolarization, and deep positive phase changes in the same direction during depolarization.

space and thus the current intensity entering the neural elements is rather low. ii) In the cerebral cortex one must reckon not only with effects on conductivity but also with those directed to transmission of impulses, iii) In peripheral nerves propagated spikes of "all, or nothing" type serve as indicators for polarizational effects; in the cerebral cortex evoked potentials, composed of PSP-s were subjected to examination.

In the following discussion we make an attempt to explain the mechanism of the polarizational changes and to answer the question: to what extent they can be attributed to shifts of membrane potential and to what extent one has to take into account actions exerted on interneuronal transmission.

Our analysis starts from the electric membrane model constructed on the basis of the cable theory and described in detail by COLE (1968).

The physical system modelling the nerve membrane is illustrated in Figure 5. In this $R_a$ represents the resistance of the external medium of unit length (200 Ohm cm); $R_i$ is the resistance of the internal medium of unit length (60 Ohm cm); $R_m$ is the resistance of the membrane of unit area (2000 Ohm cm²); $I_a$ represents the current flowing in the external medium, $I_i$: the current flowing within the nerve fiber; $I_m$ is the membrane current. (The electric parameters were taken from Aidley:

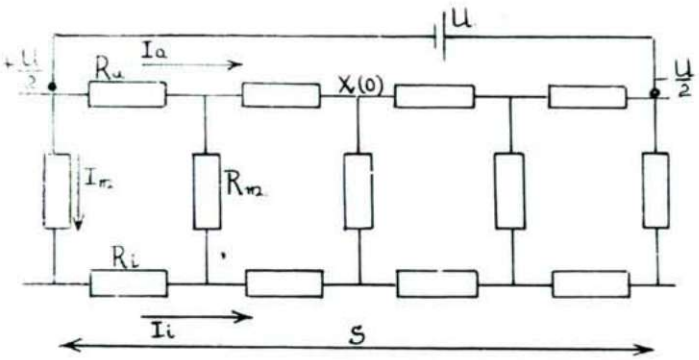


*Fig. 5.* The electric model of nerve cell membrane, simplified for stationary DC relations. Explanations see in the text.

"The physiology of excitable cells".) The equations describing the respective voltages appearing on the outer and inner membrane surfaces are according to Beier:

$$(1) \qquad U_a = \frac{U}{K + \coth\frac{s}{2\lambda} + 2\frac{\lambda}{s}\frac{R_a}{R_i}} \cdot \left[ -\frac{\lambda}{s} \cdot \frac{R_a}{R_i} \cdot \frac{\sinh\frac{x}{\lambda}}{\sinh\frac{s}{2\lambda}} - \left(K + \coth\frac{s}{2\lambda}\right)\frac{x}{s} \right]$$

$$(2) \qquad U_i = \frac{U}{K + \coth\frac{s}{2\lambda} + 2\frac{\lambda}{s}\frac{R_a}{R_i}} \cdot \left[ \frac{\lambda}{s} \frac{\sin\frac{x}{\lambda}}{\sin\frac{s}{2\lambda}} - \left(K + \coth\frac{s}{2\lambda}\right)\frac{x}{s} \right]$$

The difference $U_a$—$U_i$ represents the membrane potential change during polarization from a battery of voltage U. K is a constant depending on the shape of the electrodes. $\lambda$ is the length constant and equals, $\lambda = \sqrt{\frac{R_m}{R_a + R_i}}$ · x is the distance of the nerve portion under examination from the middle of the intrapolar zone. The validity of the equations is restricted to stationary DC potentials.

The physical relations described above are realized among the special conditions of the cat's cerebral cortex as follows (Fig. 6a).

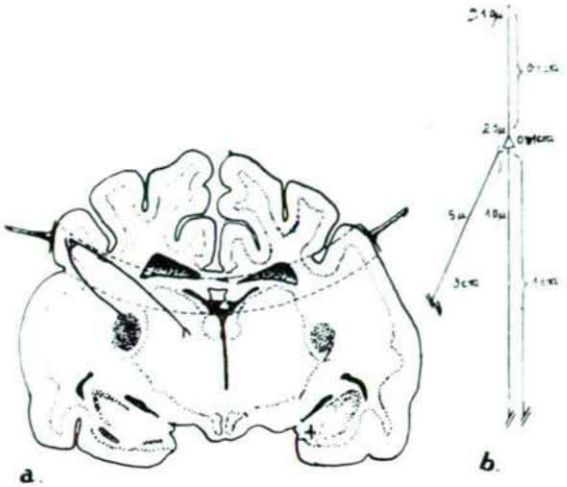

*Fig. 6a.* The anatomical circumstances of polarization. (Explanation see in the text.)
*Fig. 6b.* An idealized pyramidal cell with thalamocortical afferent fibers. Numbers on the right refer to length, those on the left refer to diameters of fibers. These sizes served as basis for calculations of changes in membrane pontetial.

The two polarizing electrodes were situated on the ectosylvian gyri of both sides. Their shortest distance was 6 cm, from each other and 3 cm from the medial geniculate body of the same side. The geniculocortical pathway fell presumably at least in 1 cm length into the path of the polarizing current.

As to the geometric parameters of the vertically oriented cells (pyramidal neurones of different layers) the following assumptions were made (Fig. 6b).

The hypothetic pyramidal neurone is situated in 1 mm depth; its apical dendrite reaches the cortical surface and makes an arborization parallel with it. The radius of the dendrite is 3 μ at its origin while at the final branches 0,1 μ. The thalamocortical axon runs about 1 cm parallel with the current loops. Its radius diminutes gradually from 5 μ to 0,1 μ at the terminal branching. Its terminal arborization was supposed at the final portion 100 μ in length. The soma was regarded as a cone of 100 μ height with a lower diameter of 30 μ and an upper one of 6 μ. The longitudinal axis of the soma was assumed to be parallel with the direction of current.

By making use of the equations (1) and (2) at different sites of the pyramidal cell and at the thalamocortical endings, the following membrane potential changes were calculated at an extracellular potential gradient of 30 mV/mm.

at terminal branching of apical dendrites $\pm 6{,}7$ mV;
at terminal branching of thalamic afferents $\pm 16{,}0$ mV;
at the soma of pyramidal neurones $\pm 20{,}0$ mV.

The bias of membrane potential changes listed above is negative at depolarizing and positive at hyperpolarizing currents. Because of the complicated geometry of the basal dendrites, the assumptions made for the soma region could only be approximate. As it turned out of the calculations, the polarization changes depended mainly on the length of the nerve portion parallel to the current flow, but the diameter of the nerve fibres plays a considerable role as well.

Considering the main results of our experiments several observations deserve special attention and discussion.

It was noteworthy that strychnine potentials did not undergo in surface leads any change during polarization as regards their amplitude, or in proportions of their component waves, either. In deep leads, however, hyperpolarization enhanced the deep positive and depolarization the deep negative waves, without altering the peak-to-peak amplitude. These seemingly contrary findings might have been resulted by different conditions for electric conduction.

In the neuropile of the superficial layers consisting of very fine dendritic branching one has to reckon with the high internal resistance of the axoplasm. Therefore the polarizing current intensity and the voltage gradient across the membrane remained relatively low. In an apical dendrite 1 mm length of which falls in the direction of the polarizing current, the polarization voltage gradient was calculated to be about 6,7 mV. Its effect on the amplitude and phase relations of the surface phenomena could only be moderate.

The obvious changes of phase relations in deep leads resulting from polarizational effects may be explained by the modifications of the membrane potential in the soma and basal dendrites. The hyperpolarization of the soma and the basal dendrites will augment the EPSP-s, while depolarization should act in the opposite direction. This seems to be valid also for hyperpolarizations arising from real surface depolarizations by means of currents carried by the volume conductor of the cortex. The surface negative spike of the strychnine potential is corresponded by a deep positive wave; this latter is suppressed by hyperpolarization, because of the elevation of the membrane potential.

The question may now be raised, whether polarizational effects on thalamocortical fibers acted upon liberation of transmitters as it was shown by ECCLES and coll. (1962) in spinal cord, by HUBBARD (1962a, b) at the neuromuscular junction. Any significant change in amplitude of strychnine potentials could not be seen during polarization. In explaining this, we assume, that the enormous irradiation of excitation represented by the strychnine potentials takes its origin from a widespread excitation of the superficial dendritic mass and a great number of short-axon interneurones which are influenced relatively weakly by the polarizing current. Based on equations (1) and (2) one can calculate, that a polarizing current of 400 μA causes a membrane potential change of only 0,5 mV in an axon, having a diameter of 5 μ

The polarizational changes of the evoked potentials are of quite different origin. Their most significant feature is a change in peak-to-peak amplitude. Hyperpolarization causes an increase in amplitude without altering the proportions of the negative and positive phases. Depolarization acts in the opposite sense. These modifications may find their explanation in variations of transmitter release from the thalamocortical endings. This latter assumption was based on the analysis of the frequency dependence curves obtained experimentally and by means of computer calculations. According to equations (1) and (2) a gemuculocortical afferent fiber suffers a membrane potential change of 16,0 mV, if it has a portion of at least 1 cm in length prallel with the polarizing current. Assuming that membrane potential changes influence the transmitter output at about the same extent as they do at peripheral terminals, a hyperpolarization of 16,0 mV may be able to treble transmitter release. Presumably, depolarization is as effective in the opposite direction. As

measurements concerning the effect of polarizing current upon transmitter release in the cortex are lacking, we quote data of HUBBARD (1962a), about the correlation between the amplitude of potsynaptic EPSP-s and the membrane potential of he-presynaptic fibres causing them to appear. The change in transmitter output ctal culated by use of the model finds its explanation, at least qualitatytively, in the presumed modifications of the presynaptic membrane potential.

The next question, that may be raised, concerns the lack of changes in the proportion of the negative and positive phase of the evoked potentials despite the considerable influence of the polarizing current on the soma and basal dendrites. As it was mentioned earlier, an effect of this type was observed at 1 cps frequency but not at higher ones, producing evoked potentials of smaller amplitude. The reason for this seems to be, that membrane potential changes affect large evoked potentials at a greater extent than the smaller ones. As an example for this, strychnine potentials can be quoted, which represent nearly maximal depolarization of the neuronal membrane.

The fundamental data regarding the effect of DC polarization upon nerve endings have come from experiments on the neuromuscular junction. On a relatively simple frog nerve-muscle preparate del CASTILLO and KATZ (1954) found, that by applying current on the motor axon, considerable changes occurred on the miniature endplate potentials and on those elicited by motor axon stimulation. Cathodal polarization caused an increase in the amplitude of the miniature EPP's, while anodal polarization remained ineffective. In these cases electrotonic changes did not extend to the postsynaptic side. EPP's elicited by motor axon stimulation were augmented during anodal polarization.

The effect of repetitive synaptic stimulation on the EPSP-s of spinal motor neurons was studied by CURTIS and ECCLES (1960). They presumed, that the EPSP amplitudes are proportional to the transmitter action evoking them. In their opinion, a motor volley initiates two processes in the junctional apparatus: i) an enhancement of 200 msec in duration and ii) a subsequent depression which may last for seconds. By applying repetitive stimuli a constant level of EPSP amplitude could be attainel which, usualy, is below the normal one. If the stimulus frequency was between 4 and 20 cps, the depression became more intensive by 0,3 sec after the beginning of stimulation and the EPSP amplitudes decreased to 70—80 percent of the initial value. At higher frequencies a potentiation appeared that reached its maximum between 30 and 100 cps. This points to the fact, that the charge of the transmitter store is not of constant intensity and depends on the stimulation frequency. This is contrary to the findings of ZABLOCKA—ESPLIN (1972). A good correlation was found between the amplitude of the second EPSP in a series and the average amplitude of the same series. This fits well with our experiences. At very high frequencies, the EPSP amplitude decreases progressively and over 250 cps, becomes inversely proportional to the time separating the impulses. This means that the transmitter output had attained its maximal rate. For this and other phenomena, not reported here, four basic mechanisms can be held responsible: i) a partial blockage of the presynaptic volley near to the final axon branchings, ii) hyperpolarization with consecutive augmentation of the spike potential, iii) modifications of the transmitter output from presynaptic endings and iv) changes in sensitivity of the postsynaptic membrane against the transmitter substances. Possibilities listed under i) and iv), could be excluded by appropriate control experiments. Thus all phenomena of facili-

tation and depression could be ascribed to respective changes of the presynaptic spike and transmitter output. The effects of polarization on the EPSP's of rats phrenic diaphragm preparation were studied by HUBBARD and WILLIS (1962). During hyperpolarization the EPSP-amplitudes rose to ninefold, while depolarization resulted in a considerable decrease. In explaining these phenomena they took into account mainly the variations of the transmitter output. In another series of experiments HUBBARD and SCHMIDT (1963) confirming their earlier results, observed that rhythmic stimulation enhanced presynaptic spike amplitude considerably and the heights of the postsynaptic potentials seemed to depend on them in a logarithmic manner. In case of paired stimuli, the second EPP proved to be higher. The mechanism of the potentiating action had to be sought in a different way, because the second presynaptic spike was usually smaller in these cases.

Our specific field of interest is more directly concerned by the extensive literature dealing with modifications of the evoked potentials during the passage of polarizing currents. In this place only data of immediate importance will be cited. BISHOP and O'LEARY (1950) has described essentially the same effects of polarization on the cerebral cortex as we did. In explaining their experimental findings they take into account only postsynaptic processes and neglect presynaptic ones at all. According to them, different phases of the evoked potentials can be deduced from activity of different cell groups. Their interpretation however does not reckon with the effect of the volume conductor of the cerebral tissue. The effects of polarization on cortical responses produced by epicortical stimulation were subjected to investigation by CASPERS (1960). In his experiments anodal polarization enhanced dendritic potentials negative in sign; cathodal current depressed and, later, inverted them. Interestingly, the actions of GABA were counteracted by hyperpolarization, while depolarizing currents proved to be synergistic. Considering that the mode of provoking surface dendritic potentials was quite different from that applied by us, analogies can be drawn only with caution. It seems to be sure, that the current intensities used by CASPERS (1960) were high enough to cause membrane potential changes in the superficial dendritic mass, too.

Basing on earlier works of RUSINOW MORREL (1961) has shown that surface anodal polarization facilitated the discharges of cortical neurones recorded intracellularly by microelectrodes. Another highly important observation was in his experiments, that cortical neurones retained their newly acquisited conditional-connections better, if they stood under the effect of hyperpolarizing currentat the time of the conditioning procedure. On conditional stimuli they exhibited positive responses at times, when other cells failed to respond at all.

HERN and coll. (1962) observed, that the electric threshold of the motor cortex was much lower to anodal stimuli, than to cathodal ones. They assume, that anodal stimuli excite pyramidal cells directly, the cathodal impulses do it only in in tnraspresynaptic way. According to LIPPOLD and coll. (1962) a hyperpolarization applied to the somato-sensory cortex augmented the positive phase of the evoked potential and enhanced the unit discharges coinciding with it. Depolarization acted in the opposite sense. The polarizational after-effects lasted about 20 minutes. DENNEY and BROOKHART (1962) subjected recruiting and augmenting responses to polarizion currents of 250—1000 μA and 200—300 μA intensity. Their leads were made from the surface. Hyperpolarization enhanced mainly the surface negative waves, depolarization depressed them. According to their interpretation, the polarizational changes

mentioned above are due to membrane potential changes of neural elements producing graded electric responses. This view has been confirmed by BINDMAN et ol. (1964) in commenting their studies carried out by means of polarization through microelectrodes.

PURPURA and SHOFER (1964) were able to induce membrane potential changes in cortical units through microelectrodes. They brought clear evidences according to which the height of EPSP's or IPSP's was modified depending on the direction of the membrane potential shift as compared to the respective equilibrium potentials of the two postsynaptic potentials. Their evidences favour the view that evoked potentials represent the sums of local potentials in cortical neurones.

LANDAU and coll. (1964) applied polarizing currents at the cortical surface and at different depths. The modifications of the evoked and strychnine potentials observed by them are essentially the same as those seen in our experiments. However, at variance with our findings they did obtain changes in the phase relations of the surface strychnine potentials. The current intensities, applied by them exceeded those used by us 2—3 fold. As to the effect of polarization upon the presynaptic endings most direct evidences can be derived from the experiments of PURPURA and MCMURTRY (1965). They recorded the membrane potential changes of PT and non-PT-cells of the cat's motor cortex intracellularly ensuing on orthodromic an antidromic stimuli during polarization of both signs. Anodal polarization enhanced the activity of PT-cells, cathodal currents depressed it. The membrane potential changes provoked in this way were not very significant. On non-PT-cells, at variance, surface anodal polarization caused hyperpolarization with corresponding modification of activity. The strong polarizational effects obtained on surface augmenting responses migth be due to the circumstance that their current intensities were several times higher than our ones. According to the experiences of RABINOVICH and KOPYTOWA (1969) neurones of the rabbit's motor cortex retained a long lasting higher reactivity after a period of hyperpolarization while the responsiveness of those which had not been hyperpolarized previously was lost rapidly. Thus, there appears, that hyperpolarization exerts an overt facilitatory effect obn the quasi-conditioned responses of cells stimulated from different sources. Similar positive after-effects were observed by VORONIN and SOLNTZEWA (1969) on the rabbit's sensory-motor cortex after surface positive polarization. This lasted up to 10 seconds without significant alterations in the membrane potential. The main conclusion that can be drawn from their observations is, that the effect of surface positive polarization is directed mainly to the transmitter output of the presynaptic endings.

In comparing our experimental results with the data of the extensive literature referring to our problem, the conclusion seems to be justified, that DC polarization of the cerebral cortex may not only be effective by altering the membrane potential of postsynaptic elements but also through modification of the transmitter release from presynaptic endings. Its seems very likely, that this latter type of action may play an important role both in the establishment of new conditional connections and in preservation of memory traces. The polarization and especially the anodal polarization of the cerebral cortex creates therefore an excellent model situation for the study of fixation of memory traces at the cellular level.

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